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Abstracts: Case Reports



CASE REPORT

How to Manage Mitral Stenosis Patient with Ongoing Atrial Fibrillation and Atrial Flutter Episode in Remote Cardiac Clinic

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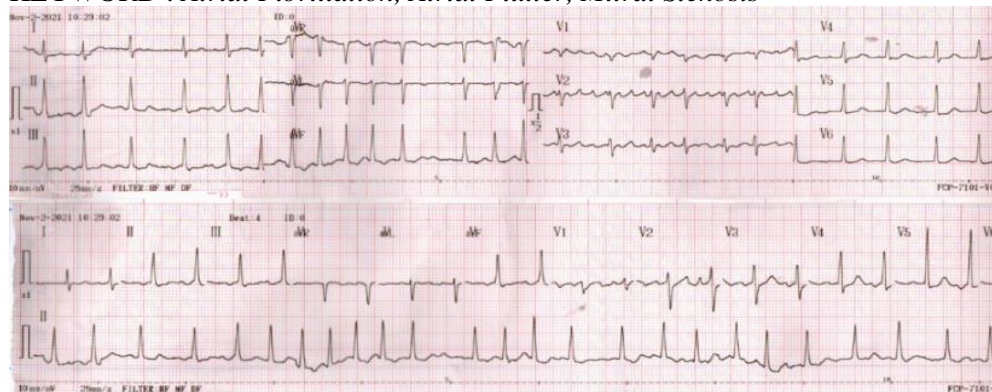
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Background: Atrial fibrillation is the most common type of cardiac arrhythmia and often seen in patients with mitral stenosis (MS). The combination of MS and AF increases the risk of stroke more than AF without MS. Thus, management of anticoagulants for atrial fibrillation or atrial flutter is important to reduce the risks for stroke and bleeding. Management of antiarrhythmic drugs (AAD) AF patients with MS is pretty similar to management for AF of any cause. However, the challenge lay in how to restore and maintain the sinus rhythm.

Case Illustration: A-57 year-old woman was admitted to the ER with a chief complaint of palpitation two days before admission. She also came with shortness of breath, gastric pain and slight dizziness. The patient had a history of HFrEF (EF 34%), severe mitral stenosis, and AF on rate control with bisoprolol 10 mg od since 9 months ago. The patient's heart rate is about 130-140 bpm with normal blood pressure. The patient's CHA2DS2-VASc score is 3. From ECG was found atrial fibrillation with an episode of atrial flutter in precordial leads. The laboratory examination still in normal limit with potassium level was 3.6 mEq/dL. We administered digoxin 0.25 mg iv every 2 hours to control the rate and correct the potassium level to a higher limit but the rate is still in rapid response. We decided to administer amiodarone to replace digoxin followed by bisoprolol 10 mg od (previous medication) with closed heart rate monitoring. Fortunately, the rate can be controlled, and the patient was discharged with bisoprolol 10 mg od and digoxin 0.25mg od to control the rate with warfarin for stroke prevention and diuretic and ace inhibitor to treat the heart failure. Patient received digoxin as her rate control as digoxin can be used in AF patients with HFrEF. Amiodarone was administered after digoxin failed to control her heart rate.. Patient received warfarin 2 mg as her anticoagulant as warfarin is currently the only treatment with established safety in AF patients with rheumatic mitral valve disease and/or an artificial heart valve.¹We also administered KCL 25 meq in 500 cc NaCl 0.9% to correct the potassium level as hypokalemia is a well-recognized risk factor for VT, and often associated with worse clinical outcomes in HF patients. Based on expert opinion, patients with a history of congestive heart failure should maintain at least 4 mEq serum potassium per L (4 mmol/L).

Conclusions: Controlling the heart rate and rhythm in The MS patient with Atrial Fibrillation was a really challenging case. In some circumstances, a single antiarrhythmic drug was not enough. Combination of AAD with electrolyte correction should be considered with careful monitoring of heart rate and rhythm.

KEYWORD : Atrial Fibrillation, Atrial Flutter, Mitral Stenosis





CASE REPORT

Poor Outcome of Acute Coronary Syndrome in Premature Coronary Artery Disease Patients with Coronary Artery Calcification: A Case Series

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Introduction: Coronary artery disease (CAD) is one of the highest contributors of mortality rate and health burden in the developing world. Patients diagnosed with Premature CAD (PCAD) were associated with poorer outcomes in terms of higher incidence of acute coronary syndrome (ACS) and total occlusive lesion, especially if accompanied with other risk factors. Among those risk factors are smoking, diabetes, and family history of PCAD. Major studies have shown Coronary Artery Calcification (CAC) is one of the important mechanisms in PCAD progression. This study presents a series of cases to elaborate the factors contributing to the poor outcome of PCAD patients, and benefits of proper diagnostic and management.

Case illustration: Three male patients aged 48, 52, and 55 years old came to the ER at Saiful Anwar General Hospital, Malang, Indonesia. All patients came with angina pain and shortness of breath. The first and second patient had risk factors of smoking, hypertension, diabetes, and family history of coronary artery disease, and the third patient with long term heavy smoking. ECG showed ST-elevation in the first and second patient and T-inversion in the third patient. Increase in serum Troponin and CKMB level supported the diagnosis of Acute Coronary Syndrome. Primary percutaneous coronary intervention attempts were performed in both patients. In the third patient, fibrinolytics failed, so rescue PCIs were performed. Poor outcomes were observed in all patients. From angiography, suspicion of coronary artery calcification was found in all patients.

Conclusion: ACS in PCAD patients with Coronary Artery Calcification (CAC) was still a major challenge for cardiologists. A careful observation of risk factors might be beneficial in order to properly diagnose a patient with CAD, so that earlier management could be given to prevent a poorer outcome.

KEYWORD: PCAD, Poor Outcome, PCI, Calcification



CASE REPORT

Misdiagnosis of Acute Aortic Dissection in Emergency Room

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Background: Acute aortic dissection is a fatal condition that occurs when the inner layer of aorta (intima) tears and it causes separation with the middle layer (media). The mortality rate of aortic dissection is about 1% per hour. The most frequent symptom is the sudden onset of severe chest pain (80%), followed by back (40%) and abdominal pain (25%) respectively. Other symptoms may appear because of the complications such as aortic regurgitation, myocardial ischaemia or infarction, neurological deficits, and cardiac tamponade. Diagnosis of aortic dissection in atypical presentation such as neurological symptoms is often missed. We present a case of acute aortic dissection patient with stroke presentation.

Case Presentation: A 67 years old woman came down with a loss of consciousness one hour before. She experienced epigastric pain during the last 2 weeks and there was no chest pain. She had no hypertension, diabetes, stroke and cardiovascular diseases before. At arrival, Glasgow Coma Scale was 7. Initial vital sign: blood pressure 167/89 mmHg, pulse rate 104x/min, respiration rate 34x/min, temperature 36.6 °C, saturation 99% room air. Physical examination showed left hemiparesis. Cardiac examination showed regular heart sound and no murmur. The electrocardiogram showed normal sinus rhythm. Laboratory findings were normal. From the brain ct-scan without contrast there was no abnormality. The chest radiograph showed wide mediastinum. Then we reevaluated the patient. We found a pulse deficit in the right arm and there was a blood pressure differential between right arm and left arm. Bedside echocardiography showed aortic regurgitation. The chest ct-scan with contrast demonstrated Stanford type A aortic dissection extending from ascending aorta to descending aorta.

Conclusion: As practitioners we should consider aortic dissection in patients with atypical symptoms. We can do physical examinations such as pulse deficit checking and blood pressure measurement in both arms and legs. Also we can suspect aortic dissection from chest x-ray and bedside echocardiography before confirming the diagnosis with chest ct scan with contrast.

KEYWORDS: *Misdiagnosis, Aortic Dissection*



CASE REPORT

Effectiveness of Temporary Pacemaker Installation As The Main Choice in Controlling Heart Rate in Atrial Fibrillation with Slow Ventricular Response : A Case Report

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Background: Atrial fibrillation (AF) is the most prevalent supraventricular tachycardia encountered in current hospital practice. Over the past 20 years there has been a 66% increase in hospital admissions for AF, and this growth is expected to continue due to the aging population, rising prevalence of chronic heart disease, and improvements in monitoring and diagnostic devices. According to the American Heart Association, heart disease is the most common risk factor for AF, while hypertension and other cardiac comorbidities have also been shown to play a role.

Case Illustration: Woman, 58 years old, came with complaints of shortness of breath accompanied by cold sweats since 2 weeks ago. The patient had experienced fainting, felt very weak and often complained of left chest pain. Other complaints are swelling in both legs, nausea and vomiting. Past medical history, a patient has a history of heart disease, but is not routinely monitored. Examination of vital signs and physicals showed a decreased heart rate, 38x/minutes and edema in both lower extremities. The electrocardiogram revealed atrial fibrillation slow ventricular response with extreme bradycardia and right bundle branch block pattern. Laboratory examination showed a decrease of sodium 131, and calcium 8.3. The treatment of choice at the time of the emergency department is the administration of 4 tabs of Aspilet and Clopidogrel, 5 mg of Nitrate. Then proceed with the installation of Transcutaneous Pacing to temporarily increase the heart rate due to an emergency but there was no significant difference in the patient's heart rate compared to before transcutaneous pacing was installed. So it is planned to do a temporary pacemaker installation in the Cath lab room with a heart rate setting of 70 bpm. After the treatment, the patient showed adequate response, the heart rate increased and changed to sinus rhythm suggesting successful pacemaker installation.

Conclusion: Pacemaker therapy in atrial fibrillation means the best therapy for patients with bradycardias with the aim to avoid the onset of atrial fibrillation. Patients with slow ventricular rates and permanent atrial fibrillation should receive a pacemaker, if the bradycardias causes syncope, dizziness or a decrease of their exercise tolerance.

KEYWORDS: *Atrial fibrillation, pacemaker, Bradycardia*



CASE REPORT

Acute Myocardial Infarction After Inactivated Virus Covid-19 Vaccination, Is There a Relationship?

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Background: Covid-19 vaccination is one of the many efforts that are expected to solve the Covid-19 pandemic in the world, including in Indonesia. The inactivated virus is one of the covid-19 vaccine groups that are widely used in Indonesia. Side effects of using covid-19 vaccine are still being researched, one of the important things that must be researched is the effect of covid-19 vaccine on the cardiovascular system. In this case reports, we have a patient who comes to the emergency department with an acute myocardial infarction after receiving a covid-19 vaccination. This is an interesting case, because we don't know if it is related or something that works independently.

Case Illustration: A 47 year old man comes to the emergency department with a main complaint, typical chest pain since 30 minutes before admission. The patient never felt the same complaint before, and he was a heavy smoker, had no history of hypertension, diabetes mellitus, allergy, or asthma. One hour before admission to ED, the patient had received the first dose of the covid-19 vaccination with the inactivated virus group vaccine. He was stable and remained alert, afebrile, BP :110/80 mmHg, HR 108 BPM, and physical examination was within normal limit. Laboratory blood examination shows normal values of cardiac enzyme (CKMB) on the first examination, and increases up to about 30-fold on the second examination; other laboratory examinations are within normal limits. ECG examination shows ST segments elevation in anterolateral leads, with reciprocal change ST-depression on inferior leads. The patient received initial management of acute myocardial infarction in the emergency department, and was immediately referred to the cardiac catheterization laboratory for primary PCI strategy. Primary PCI with drug eluting stent (DES) was success performed on total occlusion in proximal of left anterior descending artery (LAD), and contrast injection shows flow TIMI 3. After the procedure, the patient is transferred to a high care unit for observation and care. The following day, transthoracic echocardiography was performed and showed anteroseptal regional wall motion abnormality with 42% of LVEF. And then the patient was discharged from hospital without any complaint and received dual antiplatelet therapy, statin, and ACE inhibitors. One week after hospital discharge, the patient has no symptoms and the echocardiography evaluation shows improvement in wall motion and the LVEF increased to 55%.

Conclusion: The interesting thing about the illustration of the case above is a patient with acute myocardial infarction, after getting the first dose of inactivated virus covid-19 vaccine. *Ozdemir et al* in 2021 reported cases of type 1 kounis syndrome induced by inactivated SARS-COV-2 vaccine, is our case a type 2 kounis syndrome induced by inactivated SARS-COV-2 vaccine?

KEYWORDS: AMI, Covid-19 Vaccination, Inactivated Virus Vaccine, Kounis Syndrome



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Fig.1 Total occlusion proximal of LAD



CASE REPORT

A 68-years-old Man with Unstable Ventricular Tachycardia in Recurrent Ventricular Tachycardia after Myocardial Infarction: A Case Report

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Background: Ventricular tachycardia (VT) can be associated with an increased risk of sudden death. The most common cause of VT is myocardial infarction (MI), whereas a myocardial scar from prior infarct can lead to intramyocardial reentry. Patients who survive a MI are at risk, with the incidence of sudden death 1.40% in the first month after MI and decreasing to 0.14% per month at 2 years after MI. Emergent cardioversion is warranted for unstable VT that is causing symptomatic hypotension, pulmonary edema, or myocardial ischemia.

Case Illustration: A 68-years-old man with a history of CAD post PCI five years ago came to hospital with chest discomfort, diaphoresis, and vomiting. His blood pressure was 108/69 mmHg, HR 150 bpm, RR 22 x/m and 96% oxygen saturation. Physical examination found vesicular in bilateral lungs. From the Verecke and Brugada algorithm the electrocardiography showed VT. Chest x-ray suggested cardiomegaly and bronchopneumonia. His HS-Troponin was 21.0 and leukocytosis. Because the VT is stable, the patient is treated with amiodarone and the rhythm converted to Atrial Fibrillation NVR. In ICCU, he had one episode VT while on amiodarone drip, the heart rate is slower but the rhythm remains VT and never converts to sinus rhythm. On the next two days, he had unstable VT with 66/50 mmHg blood pressure, cold extremities and chest discomfort. Electrical cardioversion was done with 100 joule and converted to Atrial Fibrillation NVR. The next VT episode, because of the suspicion of a scar VT, no amiodarone bolus was given but electrical cardioversion, although it was stable. The patient then referred to the referral center hospital for further management.

Conclusion: MI have an important role in VT in patients with an infarction scar that causes disturbances in automaticity and intramyocardial reentry. Early recognition and prompt treatment for VT can prevent the patient from failing in hemodynamic instability and reduce mortality. Even though the VT is stable, if you leave it not converting it will fall into an unstable condition.

KEYWORD: *Unstable ventricular tachycardia, myocardial infarction, cardioversion*



CASE REPORT

Atrial Fibrillation Rapid Ventricular Response in A 55-years-old Woman Followed by ST Elevation Myocardial Infarction: A Case Report

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Background: Atrial fibrillation (AF) is one of the most common supraventricular arrhythmias with an incidence of 5–18% in patients with acute myocardial infarction (MI). ARIC and REGARDS study showed that AF is a risk factor for myocardial infarction. AF increase in peripheral prothrombotic risk through systemic platelet activation, thrombin generation, endothelial dysfunction, and inflammation.

Case Illustration: A 55-years-old woman with a history of uncontrolled hypertension came to hospital with chest discomfort, palpitation and dyspnea. Her blood pressure was 150/89 mmHg, HR 131 bpm irregular, RR 28 x/min and 96% oxygen saturation. Physical examination found minimal rales in bilateral lungs. The ECG showed AFRVR and LVH. Chest X-ray suggested cardiomegaly. She was treated with furosemide and digoxin, the rhythm converted to AFNVR and she was transferred to ICCU. On the next day, she was stable even though the rhythm never converted to sinus rhythm and she was transferred to the ward. In the ward, she complained of typical chest pain and diaphoresis, an ECG was performed and showed anterior STEMI. Loading of aspirin, clopidogrel, atorvastatin and thrombolysis with streptokinase 1.5 million units was done and she had cardiogenic shock, bradycardia and ROSC after cardiac arrest during thrombolysis. The thrombolysis failed so rescue PCI was performed and showed 85% mid stenosis in LAD and 1 drug eluting stent placed on LAD. In the next three days, her hemodynamic was stable and no longer using inotropic support and vasopressors and she was discharged from hospital.

Conclusion: AF was associated with a 70% increased risk of incident MI after adjustment for several cardiovascular risk factors and potential confounders, and the risk was significantly higher in women and in blacks. AF seems to be associated with an increased risk of subsequent myocardial infarction in patients without coronary heart disease and an increased risk of, all-cause mortality and heart failure in patients with and without coronary heart disease.

KEYWORD: *Atrial fibrillation, ST elevation myocardial infarction*



CASE REPORT

A Successful Thrombolysis in a Patient with Concomitant Diagnosis of ST Elevation Myocardial Infarction (STEMI) and COVID-19

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Background: COVID-19 is a disease that is caused by severe acute respiratory syndrome coronavirus 2 (SARS CoV-2) infection. COVID-19 also has several manifestations. Thrombosis and coagulopathy are frequent complications in patients with COVID-19. Although it is plausible that the COVID-19 can increase the rates of atherothrombotic events, a global increase in myocardial infarction rates (in example, STEMI as a type 1 myocardial infarction) has not been described yet. The management of STEMI on COVID-19 infection during pandemic has been discussed among medical professionals. The issues are providing timely treatment to get the best outcome and implementing infection control procedures to prevent the spreading of COVID-19 infection.

Case Illustration: A 56-year-old female was admitted to the emergency department with chest pain for 12 hours of onset, accompanied with diaphoresis and nausea. The pain was dull, radiated to the back and jaw. No complaints of fever, cough, or shortened breath. The patient was fully alert, blood pressure was 140/70 mmHg, heart rate was 102 bpm, afebrile, SpO₂ 99%, VAS 9, no rales on both lungs, and no signs of shock. The echocardiography (ECG) showed ST elevation in leads II, III, aVf, Troponin I level increased to 3375 ng/L, D-Dimer 430ng/L, antiSARS-Cov-2 reactive, no abnormality on chest x-ray, and seven days after admission the reverse transcription polymerase chain reaction came out positive. The patient was given a loading dose of antiplatelet, then decided to give thrombolysis with streptokinase because the onset had been 12 hours and the hospital was not capable of doing the catheterization procedure. After thrombolysis, the patient had significant relief of pain (VAS 2), the ECG showed > 50% reduction of ST elevation level and Troponin I spiked to 10468 ng/L.

Conclusion: Thrombolysis can be a therapeutic option to manage STEMI in a COVID 19 patient during the pandemic in a hospital with no catheterization facility. The pathophysiology of STEMI in COVID 19 has not been clearly understood. Further research and reports are needed to get the best management recommendation.

KEYWORDS: COVID-19, STEMI, Thrombolysis



CASE REPORT

Extensive Aortic Dissection Stanford Type A, De-Bakey I Presented As Atypical Angina : A Rare Case Report

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Background : Aortic dissection is one of the acute emergency cases with high mortality. Not all of the patients show the most common symptoms, such as sudden tearing pain in the chest. Therefore, we must include the possibility of aortic dissection presented as atypical angina.

Case Illustration : A case of 37 years old obese male with uncontrolled hypertension history, complained of chest discomfort since 2 days prior, that radiated to the back, with associated symptoms of sore back muscle and sweating. Complaints of epigastric pain and burping were also reported. Blood pressure was 170/110 mmHg, while physical examination revealed diastolic murmur grade II-IV on the intercostal space II. ECG showed sinus rhythm, normoaxis, left ventricular hypertrophy, T inverted in lead III and aVF. Initial laboratory results showed low platelet count, high CRP, high LDL, however normal serial CKMB and Troponin T hs. From echocardiography, there was left ventricular concentric hypertrophy, grade I diastolic dysfunction, mild AR, and dilatation of ascending aorta, global normokinetic with LVEF 64%. MSCT cardiac (triple rule out) was done and intramuscular bridging at mid LAD along with aortic dissection were found. Calcium scoring Agatston score was negative. Subsequent CTA thoraco-abdominal aorta showed extensive aortic dissection (Stanford type A, De Bakey type I) from sinotubular junction that passes ascending aorta, aortic arch, descending aorta, aorta abdominalis, to the right illiaca communis (+- 6 cm from bifurcation). The patient then underwent Bentall procedure and total arch replacement at tertiary hospital. After the procedure, he is currently doing phase II cardiac rehabilitation and he is being prepared to return to work.

Conclusion : This case describes an important role of clinical reasoning and comprehensive examinations in evaluating patients with atypical angina, in order not to miss the possibility of aortic dissection.

KEYWORDS : *extensive aortic dissection, Stanford classification, De Bakey classification, atypical chest pain, triple rule out*



Figure 1. CTA thoraco-abdominal aorta showing extensive aortic dissection from sinotubular junction to right iliaca communis.



CASE REPORT

Diagnosing Myocardial Infarction in A 20-Years-Old Athlete in Rural Setting without Cardiac Enzyme Studies or Angiography: A Case Report

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Background: Exercise controls risk factors for cardiovascular disease, reduces the incidence of obesity, diabetes, improves lipid profile and blood pressure, thereby reducing coronary and acute events. However, as technology and diagnostic capabilities for heart disease improve, the number of deaths from heart disease at a young age also increases, including among young athletes.

Case illustration: A 20-years-old athlete from a local football club, was brought to the hospital complaining of chest pain as if he was under heavy weight pressure. ECG showed sinus rhythm (77bpm), no cardiac chamber enlargement, and T-inversion in anterior and inferior leads, and ST-elevation in anterior leads (V2-V3), which was considered normal variant or early repolarization in initial assessment. We diagnosed this patient as NSTEMI-ACS. Myocardial infarction with non-obstructive coronary arteries (MINOCA) such as myocarditis was one of the differential diagnoses, but cardiac enzyme studies and angiography were not available.

Standard initial NSTEMI-ACS treatment was given such as acetylsalicylic acid 320 mg, clopidogrel 300 mg, atorvastatin 40 mg, and isosorbide dinitrate 5 mg sublingually, continued to 1 mg / hour via syringe pump. Fondaparinux 2.5 mg for 5 days was administered after the patient's kidney function was considered normal. With no evolution in the ECG, our clinical diagnosis was NSTEMI-ACS. The pre-discharge echocardiography was considered normal. The patient was discharged without symptoms after being treated for 2 days in the ICU and 3 days in the ward.

The risk of ACS in the first 2 hours when doing moderate exercise is 1,6 times, and 5,7 times in the first 2 hours when doing vigorous exercise, drastically increasing the blood pressure, pulse pressure, heart contraction, spasm, and fluid dynamic stress. All of which are known to cause rupture of plaque, increasing platelet reactivity, blood coagulation, and causing much damage to non-coronary disease such as Takotsubo cardiomyopathy or myocarditis as well.

Conclusion: Myocardial Infarction is not a > 40-years-old disease anymore, and it may occur to anyone, even young athletes who have an active lifestyle.

KEYWORDS: *Acute coronary syndrome, young athlete, vigorous exercise, limited diagnostic capability.*



CASE REPORT

A Case Report: Prompt Treatment for Rapid Ventricular Response Atrial Fibrillation and Hypertensive Crisis in Severe Acute Exacerbation of Chronic Obstructive Pulmonary Disease, ABC or CAB Strategy?

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Background: Atrial fibrillation (AF) is the most common cardiac arrhythmia, and its prevalence is 60x in COPD patients. Atrial fibrillation with rapid ventricular response (AF RVR) potentially transformed into hemodynamic instability. Prompt treatment of AF RVR in severe acute COPD exacerbation is essential.

Case Illustration: A 70 years old man came to the emergency department with dyspnea for 10 hours. He felt palpitation, without any chest pain and history of cardiac disease. He had COPD history and routinely used fenoterol inhalers and salbutamol tablets. The patient was only able to speak 1-2 words, and the wheezing was heard in all fields of the lung. He had already nebulized with albuterol in a previous health facility, but no significant improvement was achieved. The BP was 200/120, HR 190 bpm, RR 30x/m, SpO₂ 86% and temperature 37.9C. Electrocardiography reveals AF RVR with 200bpm. Blood gas analysis showed hypercapnia and respiratory acidosis. Ten lpm oxygen by NRM and albuterol nebulization were given but no improvement observed. Aggressive therapy for COPD by intravenous terbutaline, along with intravenous bolus diltiazem for AF and hypertensive crisis, was followed by patient's improvement in some 5 minutes. The ventricular response and BP were 120 bpm and 170/100mmHg respectively. Diltiazem was continued by intravenous pumping and the patient was treated for respiratory acidosis. Chronic obstructive pulmonary disease is an independent risk for incident, recurrence, progressivity and mortality in AF. Unintended pharmacological arrhythmogenic drugs for COPD, hypoxia and respiratory acidosis in severe acute COPD exacerbation commonly precipitate AF by direct mechanism. Indirect mechanisms, such as increasing sympathetic activity, also intensify the occurrence of AF. First line therapy for AF in COPD is to treat pulmonary disease, hypoxia and respiratory acidosis. In addition, intravenous non-dihydropyridine CCB may be beneficial to improve the ventricular response and hypertensive crisis.

Conclusion: First line therapy of AF RVR and hypertensive crisis during severe acute COPD exacerbation is treating the pulmonary disease, hypoxia and respiratory acidosis. Intravenous non-dihydropyridine CCB may give some additional benefit.

KEYWORDS: *Atrial Fibrillation, Chronic Obstructive Pulmonary Disorder, Severe Acute Exacerbation, Hypertensive Crisis, Rapid Ventricular Response*



CASE REPORT

Brady-Arrhythmias with Prolonged QTc in Gitelman Syndrome Patient

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Background: Gitelman syndrome is an autosomal recessive tubular disorder induced by mutations of some genes characterized by hypokalemic metabolic alkalosis, hypomagnesemia and high urinary chloride excretion but low calcium excretion. Bettineli et al demonstrated prolongation of QTc in Gitelman syndrome, suggesting that there is an increased risk for development of dangerous arrhythmias.

Case Illustrations: A 40 years old man was referred from a private hospital with total AV block (TAVB) and history of cardiac arrest (mode VT/VF). His chief complaint was syncope with symptoms of orthostatic hypotension. He also complained of dizziness for a year, but never got treated before. He denied taking any drugs and symptoms of paralysis, dyspnea and chest pain. In the emergency department of our hospital, vital signs and physical examination were normal. ECG showed atrial bradycardia with prolonged QTc. As the laboratory result showed hypokalemia (2.3 mmol/L) and hypomagnesemia (1.56 mmol/l), we suspected that imbalance electrolyte as the reversible causes. Despite repeated correction with intravenous potassium, he got persistent hypokalemia. Increased random urinary potassium and urinary creatinine ratio (37,96 mmol/gr) suggests renal cause. On gas analysis, he presented a metabolic alkalosis (pH 7.501, HCO₃ 32 mmol/L, PCO₂ 40,9 mmHg). Further investigations showed increased urinary excretion of chloride (136,6 mmol/L) and normotensive. Based on the association of hypomagnesemia, hypokalemia, metabolic alkalosis and normotensive, the diagnosis of Gitelman Syndrome was established. The patient began oral supplementation 600mg per 8 hours, spironolactone 50mg per day and he was encouraged to maintain a high potassium diet. The potassium became increased but the electrocardiogram evaluation showed persistent atrial bradycardia with prolonged QTc (506 msec), so we did implantation of VVIR permanent pacemaker.

Conclusions: Gitelman syndrome related persistent hypokalemia could contribute to persistent prolonged QTc and arrhythmias, careful holistic management should be applied for avoiding life threatening cardiac events.

KEYWORDS: Persistent hypokalemia, TAVB, atrial bradycardia, Gitelman syndrome



CASE REPORT

Immune-mediated Myocarditis associated with Rheumatic Fever in a Young Male adult: A case report

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Background: Myocarditis is a form of cardiac muscle disease due to an inflammatory that can result from various causes. While viral infections are the most common causes, some cases are associated with immune-mediated activation or secondary due to bacterial infection, parasite, fungi, or drug reaction. Myocarditis initially has such non-specific clinical manifestations. It can manifest like a myocardial infarction with arrhythmias, dyspnea, sudden-onset angina pectoris, and heart failure developing within days. Here, we presented an unusual case of immune-mediated myocarditis associated with rheumatic fever in a young male adult.

Case Presentation: A 19-year-old male presented with substernal chest pain, dyspnea, palpitations, cough, nausea, and dizziness for three days. Previously, the patient had a history of fever for two days. Physical examination revealed fine crackles on auscultation. Electrocardiogram showed ST elevation in leads II, III, aVF, and V5, V6. Echocardiography showed mild mitral regurgitation and segmental wall motion abnormality. Laboratory findings showed monocytosis 13%, elevated NLR 3.25%, elevated CK-MB (76 U/L), and Troponin T levels (425.70 ng/L) along with elevated liver enzymes levels, AST (79.3 U/L), ALT (46.4 U/L). Serology test showed ASTO positive (>200 IU/mL) meanwhile RF negative (<200 IU/mL). Cardiac Computed Tomography showed inhomogeneous enhancement within apico medial myocardial wall with 52% ejection fraction. ANA Profile test was +2 on Anti-Ro-52 antibodies. He was administered with beta-blockers, acetylsalicylate acid, corticosteroids, diuretic, atorvastatin, IVIG and was discharged without any complications on day 4. At follow-up, three weeks after discharge, the ejection fraction was improved up to 58%, the CK-MB (37 U/L) and Troponin T (9.75 ng/L) levels had lowered, and the symptoms resolved.

Conclusion: Immune-mediated myocarditis is a rare condition with diverse clinical patterns and presentation. It remains challenging for physicians to differentiate between the causes of myocarditis, especially in the acute phases. A diagnosis should be made based on a wide evaluation of the evidence, including medical history, clinical presentation, and results of the available additional tests, to provide early treatment and avoid jeopardizing outcomes.

KEYWORDS: Myocarditis, Immune-mediated, Infection

CASE REPORT

Patent foramen ovale associated with cryptogenic stroke a case report: Should *it* be closed?

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Background: Patent foramen ovale (PFO) is a common abnormality, present in almost 20-30% of the population. The presence of PFO was associated with cryptogenic stroke. We describe a patient that came with PFO associated with a cryptogenic stroke that came to our hospital.

Case Description: A 46 years old man came with a history of a brief period of loss consciousness. There was a history of brief periods of loss consciousness in 2010, 2014 and 2020. He appeared well with GCS E4V5M6, his vital sign is in normal range. Echocardiography showed EF 74%, global normokinetic, normal valve, and interatrial septum showed possibility of PFO or ASD (Atrial Septal defect). Patient then underwent transesophageal echocardiography (TEE). TEE showed interatrial septum gap with stretch PFO (0.2 cm – 0.5 cm) left to right shunt. The bubble test was positive with grade II (5-25 bubbles). Brain CT scan showed acute lacunar infarct in left basal ganglia. A diagnosis of PFO was made with RoPE (risk of paradoxical embolism) score 7 and the patient was planned to undergo closure of PFO with amplatzer 25 mm. Post PFO closure his echocardiography showed there's no leakage and stable APO (amplatzer occluder). Patient was being monitored from the outpatient clinic. Patent foramen ovale is a common abnormality and it is found in almost 20-30% of the population. The RoPE (risk of paradoxical embolism) score can be used to determine probability that a PFO is responsible for cryptogenic stroke. A high score is associated with increased likelihood that PFO is responsible for the cryptogenic stroke. Several trials about PFO closure for recurrent cryptogenic stroke prevention have been done. the Gore-REDUCE (Gore Helex Septal Occluder/Gore Septal Occluder for Patent Foramen Ovale Closure in Stroke Patients). This trial concludes closure is superior to antiplatelet therapy. The DEFENSE-PFO (Device Closure Versus Medical Therapy for Cryptogenic Stroke Patients with High-Risk Patent Foramen Ovale) this trial concluded closure in patients with high risk PFO characteristics resulted in lower rate of ischemic stroke versus medical therapy.

Conclusion: PFO closure is indicated when a patient has a cryptogenic stroke. Other factors including hypercoagulable states, atherosclerotic lesion, cardioembolic sources and arterial dissection should be excluded before considering PFO closure.

KEYWORDS: *Patent foramen ovale closure, cryptogenic stroke, paradoxical embolism.*

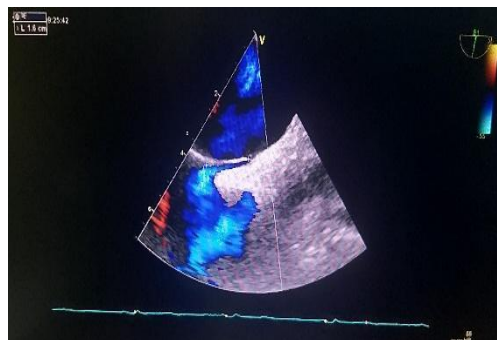


Fig 1. Transesophageal echocardiography (TEE) showed PFO with left to right shunt.



CASE REPORT

Atrial Myxoma Case Series:

Atrial Myxoma Presenting with Neurological Problem

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Background: Atrial myxoma is a rare disease with an incidence of surgically resected cases of 0.5–0.7 per million population. We describe two patient cases with atrial myxoma that came to our hospital.

Case Description: First case, a 64 years old female came to our hospital with chief complaint weakness on the right side of extremities started three days ago. There was a history of CVD and HNP in 2015. She looked ill with GCS E4VxM6. Physical examination showed NVII paralysis. Motoric strength was 1 point for right sided extremities. Brain MRI showed acute ischemic infarct on the left temporal-parietal lobe. The echocardiography showed a large mass in the left atrium, moving in and out through MV. Patient then successfully underwent resection of atrial myxoma. Second case, a 16 years old man came with chief complaints of loss of consciousness and weakness on the right side of extremities starting one days ago. He looked ill with GCS E4V5M6. Physical examination showed NVII paralysis, motoric strength was 2 points for right sided extremities. Brain MRI showed multiple hyperacute lacunar infarct on the left cerebellum. From echocardiography showed a large mass moving in and out through MV. Patient then successfully underwent resection of atrial myxomas. Most cardiac tumors are benign tumors, mainly myxoma. The classic triad of cardiac myxoma are symptoms due to cardiac obstruction, cerebral or peripheral embolism and constitutional symptoms like fever and fatigue. Transthoracic echocardiography had up to 95% sensitivity on diagnosing atrial myxoma. Once a patient is diagnosed with cardiac myxoma urgent surgical resection is recommended due to risk of embolization.

Conclusion: Most primary cardiac tumors are atrial myxoma. The Signs and symptoms can vary. Echocardiography is important in diagnosing atrial myxoma. Resection of atrial myxomas is curative with low chance of recurrences.

KEYWORD: *Atrial myxoma, Cardiac tumors, Transthoracic Echocardiography*



CASE REPORT

ST-Segment Elevation Myocardial Infarction in Critically Ill COVID-19 Patient : The Double Trouble

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Background: Coronavirus disease-2019 (COVID-19) has been shown to result in coagulation abnormalities and predispose patients to thrombotic disease, one of which is ST-segment elevation myocardial infarction (STEMI). Here, we report a case of a critically ill covid-19 patient who developed STEMI during treatment.

Case Illustration: A 65 year old man was admitted to intensive care unit (ICU) with critically ill confirmed COVID-19 pneumonia in the inflammatory phase. Patient was intubated on day 3 of ICU admission. He was consulted by a cardiologist due to haemodynamic instability. The blood pressure was 70/40 mmHg, ECG showed ST-segment elevation in leads II, III, and aVF and the troponin I level was >50 µg/L. Subject was then given 1 liter of fluid, loading dose of 160 mg aspirin, 300 mg clopidogrel, and 80 mg of atorvastatin. Patient has previously been given enoxaparin 0.6 cc/24 hour for 5 days. Fibrinolytic therapy could not be administered because of a history of gastrointestinal bleeding within the past month. Currently the patient is receiving conservative therapy.

COVID-19 has a contributory factor to an increase in procoagulant activity and destabilization of vascular plaques, thereby increasing the risk of STEMI. A meta-analysis involving 1.527 COVID-19 patients revealed at least 8% of the patients have acute myocardial injury. Some studies suggest that inpatients with known COVID-19 and severely ill in ICU are not suitable for Percutaneous Coronary Intervention (PCI) and should be considered for fibrinolytic therapy.

Conclusion: Revascularization with either fibrinolytic or percutaneous coronary intervention remains the choice in STEMI patients with COVID. However, for some patients which are not eligible for revascularization, optimal medical therapy is an option.

KEYWORDS: *ST-segment elevation myocardial infarction, Critically ill patient, Coronavirus disease-2019*



CASE REPORT

Unusual Cases of Acute Limb Ischemia as a Complication of Infective Endocarditis : a Case Series

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Background: Infective endocarditis (IE) has a high morbidity and mortality despite improved diagnostic and treatment options. Among many complications, systemic embolism has the potential of causing devastating sequelae and even life-threatening clinical situations. The incidence of embolization causing acute limb ischemia (ALI) remains unknown. We report unusual cases of ALI which are complications of IE.

Case Illustration: Patient A, female, 52 years old was referred to the emergency department because of acute pain in the right leg. Examination with doppler ultrasound showed a thrombus at the right common femoral artery then diagnosed with ALI Rutherford stage IIb. In addition, by transthoracic echocardiography we found an oscillating mass at the posterior mitral leaflet. Patient then underwent an emergency embolectomy. During surgery, emboli were removed to be examined histologically. Post operative exam revealed a palpable pulse and warm leg. The cultures of blood and emboli were positive for *Staphylococcus pyogenes*. Patient B, a 68 years old woman was admitted to the emergency department due to acute pain in the left leg. Thrombus was seen at the left tibialis anterior et posterior artery with doppler ultrasound, she was later diagnosed with ALI Rutherford Stage IIb. An oscillating mass was found in the aortic valve. The blood culture was positive for *Staphylococcus sciuri*. She also underwent an embolectomy. The pulse in the left leg was palpable postoperatively. However, she had respiratory failure, was intubated, and died on the fourth day of hospitalization. Both patients received empiric antibiotics in the emergency department then continued according to culture results.

Conclusion: Our case series shows an uncommon case of infective endocarditis complicated with acute lower limb ischemia. Although both patients undergoing colectomy and received aggressive antibiotics, they had different outcomes. However, peripheral arterial embolism that results from bacterial endocarditis may be silent or catastrophic.

KEYWORDS: *Infective endocarditis, acute limb ischemia, embolectomy*



CASE REPORT

Dressler's Syndrome As A Late Complication In Failed Fibrinolytic Stemi Patients

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Background: One of complications in ST-segment elevation myocardial infarction (STEMI) is pericarditis. Dressler's syndrome is a form of secondary pericarditis with or without a pericardial effusion, that occurs as a result of injury to the heart or pericardium. Dressler's syndrome was first described in 1956. Its incidence has decreased in the reperfusion era. We present a case of pericardial effusion in a failed fibrinolytic STEMI patient.

Case Presentation: A 38 years old male came with typical chest pain 8 hours ago. Complaints of shortness of breath, palpitations, cough, and fever were denied. The patient had STEMI and received fibrinolytic with a failed response. He did not receive rescue percutaneous coronary intervention (PCI) because of insurance issues. Past History: Hypertension (+) controlled and Stroke SNH 5 years ago. Patient complained of pleuritic chest pain and dyspnea on day 6 of admission, which improved with a change of position. On the seven days of care, the problems began to worsen. Vital signs showed elevated body temperature: 38⁰C. Electrocardiography showed anterior ST segment elevation, and echocardiography revealed a 2.1 cm pericardial effusion at superior RA and RA collapse. We diagnosed the patient with Dressler's syndrome, pericardiocentesis was then conducted, although it was unsuccessful. Then we did a pericardial window to drain the fluid with hemorrhagic fluid 500 cc. The cytology of the pericardial fluid showed exudative. We gave the patient standard therapy for pericarditis and planned coronary angiography. His condition clinically improved after treatment and he was discharged 21 days after admission. Combination therapy of colchicine and acetaminophen could be a treatment option for Dressler's syndrome. We report a patient with Dressler's syndrome successfully treated with colchicine and acetaminophen. Dressler's syndrome, which is a secondary form of pericarditis that is typically demonstrated weeks to months after MI, is presumed to be mediated by an autoimmune mechanism.

Conclusion: Dressler's syndrome came as a late complication because there was no reperfusion therapy in this patient.

KEYWORDS: *Dressler's syndrome, Fibrinolytic, STEMI, Pericardiocentesis.*



CASE REPORT

Myocardial Bridging: A case report series of two patients with a different entity

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Background: Myocardial bridging is an anatomical abnormality in which a muscle overlies the intramyocardial segment of an epicardial coronary artery. This condition allows the myocardium to compress the tunneled artery on the systolic phase, or diastolic phase in severe conditions and disturbs blood flow to the myocardium. Symptoms may present as atypical chest pain or even typical anginal chest pain that can mimic acute coronary syndrome.

Case Illustration: Case 1. A 62 years-old male, visited our clinic with typical angina. He also experienced dyspnea on effort. This patient has a history of dyslipidemia, uncontrolled hypertension. He is an active smoker and had a prior history of cerebrovascular accidents. ECG showed signs of left atrial enlargement, left ventricular strain, and ischemic in the high lateral lead. Echocardiography showed concentric LVH, with preserved ejection fraction. The patient was then scheduled for angiography. We found myocardial bridging at mid LAD and no significant stenosis. The patient was then treated with optimal medications for angina. Symptoms were relieved and patients were in good condition and remained asymptomatic after treatment. Case 2. A 49 years old male with crushing chest pain 12 hours before admission, radiated to the left arm and was accompanied by diaphoresis. He has a history of hypertension and well-controlled asthma. An electrocardiogram showed pathological Q-wave in the inferior lead and no further evolution was found upon observation. The cardiac enzyme was normal. The patient was diagnosed with unstable angina pectoris and then scheduled for coronary angiography. We found no significant lesions on CAG, but we found myocardial bridging at mid LAD. The patient was then treated with optimal medical therapy with adequate anti-ischemic medication. No residual symptoms upon discharge until follow-up control.

Conclusion: Among patients with non-obstructive coronary artery disease, myocardial bridging may not be an uncommon presentation in daily practice. Precise diagnosis and therapy help patients to achieve a better quality of life and reduce major adverse cardiac events.

KEYWORDS: *Myocardial bridging, coronary angiograph*



CASE REPORT

Case Report of Repetitive Sustained Monomorphic Ventricular Tachycardia: A Challenge To Find The Origin of The Disease

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Background: The cause of monomorphic ventricular tachycardia (VT) consists of Scar-related reentry, Purkinje disease, or an Idiopathic VT. One type of scar-related reentry, Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy (ARVD/C), and one of Idiopathic VT, Right Ventricular Outflow Tract (RVOT) Tachycardia may share same QRS morphology, and differentiation between these 2 distinct disease states is paramount.

Case Presentation: A 59-years old male was admitted with progressive chest pain 4 hours prior. It felt like a burning sensation followed by diaphoresis. The patient was a heavy smoker with a history of uncontrolled hypertension. Initial EKG showed sinus rhythm with right axis deviation and inverted T wave on V2-V6. Troponin I level of 160 ng/l. In a phenotype of an acute coronary syndrome (ACS), standard regimental therapy was given and the patient was admitted to ICU. At midnight, the EKG showed stable sustained monomorphic ventricular tachycardia with inferior axis, LBBB morphology. Patient was managed with iv bolus of 150 mg Amiodarone, and 15 minutes later the heart rhythm was returned to sinus, thus maintenance doses were given. Twelve hours after dose completion, the same pattern of ventricular tachycardia was reoccurred accompanied by hemodynamic instability. We attempted 100 J of synchronized cardioversion but it failed, so a second loading dosage of amiodarone was administered, and the heart rhythm reverted to normal. We did a further investigation to find the source of VT as we suspect ARVD/C or RVOT-VT. Electrolyte levels were normal, coroangiography showed normal coroner with Troponin I level reduced to 70 ng/L after 3 days of treatment. On echocardiography, we found dilated right ventricular with the diameter of RVOT was 38 mm on PLAX, with systolic function of left and right ventricle tend to be normal, reflecting partial fulfillment of major echocardiographic criteria for ARVC. Based on these findings, we discontinued the ACS drug and focused on ventricular arrhythmia. Further investigation with MRI is needed to justify potential structural heart disease that causes the VT.

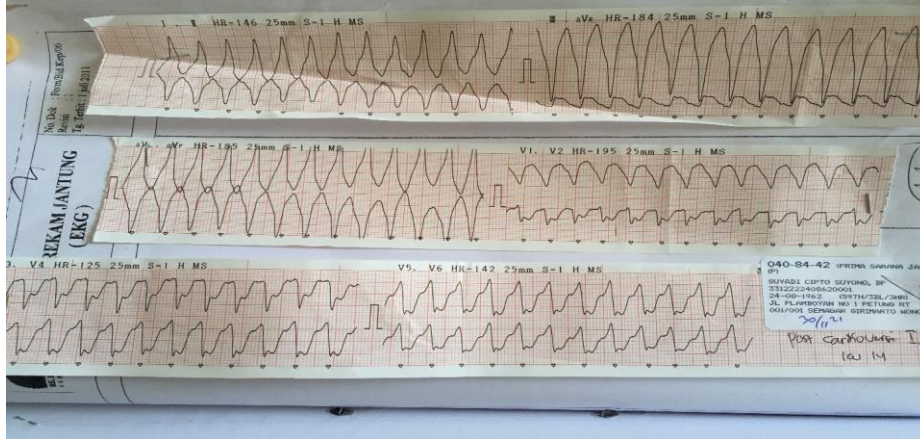
Conclusion: The RVOT is the most prevalent source of idiopathic VT. The EKG has distinct structural characteristics, including LBBB with inferior axis and right axis deviation. Ventricular arrhythmias from ARVD/C may share same QRS morphology and T inversion in V1-V3 may aid the diagnosis of ARVD/C. Given the differences in illness origin and care, it's vital to distinguish between RVOT-VT from ARVD/C with a more advanced diagnostic approach.

KEYWORDS: *Ventricular tachycardia, RVOT, ARVD/C, Emergency*



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CASE REPORT

**Successful Resolution of Total Atrioventricular Block in
Infero-posterior ST-Elevation with Acute Right Ventricular Myocardial Infarction
a Case Report**

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Background: Total atrioventricular block (TAVB) is a common complication of inferior myocardial infarction (MI). An occlusion in the right coronary artery (RCA) results in a decreased oxygen perfusion to the inferior wall of the heart, leading to inferior myocardial infarction. The RCA that supplies blood to the inferior wall of the heart also supplies blood to the (AV) node. So, an occlusion in the RCA can also cause impedance to the cardiac conduction system, such as total AV block.

Case Illustration: A 50 years old man was admitted to our emergency department with typical chest pain accompanied by diaphoresis starting 13 hours before admission. Neither dyspnoe nor palpitation was found and had clinically stable hemodynamic. He had several risk factors such as hypertension, diabetes mellitus, and was smoking two packs per day. Electrocardiography (ECG) showed inferoposterior ST-elevation with right ventricular myocardial infarction (STEMI) and Q wave at anterior lead. On the second day of care, the patient still suffered with chest pain and his ECG became a total AV block. Implantation temporary pacemaker (TPM) with coronary angiography was performed, showed total occlusion of the mid RCA, and no significant coronary artery disease in other coronary arteries. One stent placement in the mid RCA was done with good results (TIMI Flow III). Two days after revascularization of the RCA, symptoms of angina disappeared and were clinically stable. His ECG returned to sinus rhythm even when the temporary pacemaker was off. He was discharged from hospital three days later.

Conclusion: With appropriate medical treatment, AV block caused by inferior wall ischemia may be reversible. The treatment involves a reperfusion strategy to restore blood flow and an application of transcutaneous or transvenous pacing for rhythm stabilization. Patients with reversible causes of AV block should consider temporary pacing before determining permanent pacing. For this patient, the preferred reperfusion strategy is urgent PCI and implantation of a temporary pacemaker for rhythm stabilization hemodynamic.

KEYWORDS: *Total atrioventricular block, Inferior ST-elevation myocardial infarction, Percutaneous coronary intervention, Temporary pacemaker*



CASE REPORT

Preservation of Cardiac Contractility and Rhythm in Recurrent Cardiac Arrest in ST-Elevation Myocardial Infarction (STEMI) After Successful Reperfusion Therapy: STEMI Guideline Revisited

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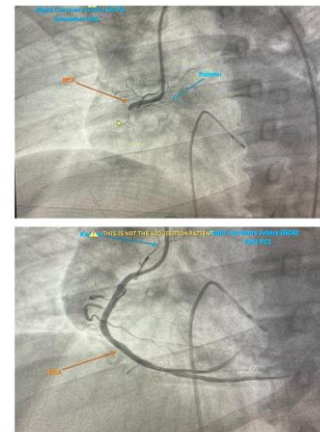
Background: ST-Elevation Myocardial Infarction (STEMI) is one of the main causes of mortality. Reperfusion strategies for STEMI were divided based on the time from symptoms onset according to guidelines. Current guidelines show time is important in choosing reperfusion therapy for preserving cardiac contractility in STEMI.

Case Presentation: A 41-year-old man came to ER with acute severe retrosternal pain radiating to the left arm and jaw, 30 minutes before admission. The vital sign was stable at that time. He had histories of hypertension and smoking. Electrocardiography (ECG) showed an ST-Elevation in the inferior lead and suggestive right ventricle involvement with 1st-degree AV-block. While waiting for the family's decision, the patient went through cardiac arrest. Monitor ECG showed ventricular fibrillation. The patient received ACLS Protocol and was defibrillated two times and successfully back into the return of spontaneous circulation (ROSC) state. The cardiac arrest happened several times, so we decided to give thrombolytic therapy. After thrombolytic was given, He was awake but still complained of chest pain and the monitor showed intermittent complete AV-block. So, we activate the Cath lab, to do a percutaneous coronary intervention and temporary transvenous pacemaker insertion. Patients with inferior infarction often have concomitant ischemia or infarction of the AV node secondary to hypoperfusion of the AV node artery and may develop ventricular arrhythmias, AV-block, mechanical complications, low cardiac output, and shock. ⁽²⁾ Hyperstimulation of the myocardium also can activate the Bezold-Jarisch reflex and promotes parasympathetic activity, leading to bradycardia, vasodilatation, hypotension, or even asystole. ⁽³⁾ This mechanism was thought to be the cause of simultaneous arrest in this patient.

Coronary angiography showed a total occlusion in the right coronary artery after thrombolytic therapy. After the rescue PCI, ECG showed a resolution of ST-segment and sinus rhythm. The pacemaker was released and the patient was sent to the Cardiac ICU. Echocardiography evaluation showed a preserved ejection fraction after successful revascularization.

Conclusion: Revascularization is recommended in patients with STEMI and AV-Block. AV-Block may require temporary pacing in the presence of refractory symptoms or hemodynamic compromise, but most often resolves within a few days. Choosing the right reperfusion strategy in STEMI can lower mortality rates, restore cardiac contractility and rhythm without complications.

KEYWORDS: STEMI, Reperfusion Therapy, Cardiac





CASE REPORT

Optimal Medical Therapy For A Life-Threatening Acute Inferior Myocardial Infarction With A Persistent ST-Segment Elevation: What If We Cannot Revascularize?

A Perilous Premise In A Type C Rural Hospital

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Background: Inferior transmural myocardial infarction is a type of acute CAD indicated by an ST-elevation in inferior leads due to RCA occlusion, resulting in a full-thickness inferior wall ischemia, putting the patient at a great risk of fatal complications such as atrioventricular blocks, RVMI, and cardiogenic shock. Inferior STEMI ideally requires an immediate coronary stenting, with a prior fibrinolysis if cathlab is unreachable within two-hours. This study aims to elaborate on what can physicians do to manage this life-threatening condition in a vastly rural region hospital without any methods of revascularization, and the closest accessible cath lab is twelve-hours away.

Case Description: A 47-years old male was admitted to the ER, in a rural hospital without any cath lab nor fibrinolytics, with a crushing chest pain since four hours prior. The pain radiates throughout the left arm, accompanied by nausea and sweating. Vital signs show BP 137/90 mmHg, HR 63 BPM, RR 20x/m, SpO2 99%. Upon ECG, a prominent ST-elevation in leads II,III,aVF was found. Cardiac biomarker test was unperformable. The patient was then transferred to ICU after initial loading of aspirin and clopidogrel (320 mg/300 mg) and high-dose statin. The patient experienced ongoing angina during the first 24-hours, despite treatment with subcutaneous fondaparinux 2,5mg sid, nitrate, antiplatelets, and statin. Serial ECG shows a persistent inferior ST-elevation, gradually de-evolving after the first 24-hours. The patient was initially minimally responsive, but was eventually stabilized and discharged. The patient denied further plans for revascularization. The clinical and ECG findings are consistent with inferior STEMI. In a rural hospital without reperfusion methods, optimal medical therapy (OMT) which includes DAPT (Aspirin sid, P2Y₁₂ inhibitor sid), statin sid to induce plaque stabilization and regression, and anticoagulants such as fondaparinux, must be fully optimized. Nitrates must be judiciously administered in inferior STEMI, due to the possibility of RV involvement. Despite the optimization of medical therapy, invasive therapy is still arguably the only definitive treatment. Eventually, transfer to a cath lab facility must be managed.

Conclusion: Although OMT might seem initially sufficient, invasive measures are still the definitive treatment for patients with transmural inferior MI. OMT will only delay, not stop, the progression of inferior MI.

KEYWORDS: *Inferior STEMI, Optimal Medical Therapy, Revascularization Unavailable, Rural Hospital.*

CASE REPORT

Stroke During Primary Percutaneous Coronary Intervention Procedure in Inferior ST-Elevation Myocardial Infarction with Total Atrioventricular Block: What Should be Evaluated?

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Background: Inferior ST-Elevation Myocardial Infarction (STEMI) with a total atrioventricular block (AV block) needs early reperfusion to achieve a better outcome. Primary Percutaneous Coronary Intervention (PCI) is considered safe as the strategy for inferior STEMI complicated by total AV block. However, there are still inevitably complications during primary PCIs, such as stroke although it is rare.

Case presentation: A 58-year-old male was referred to our hospital with inferior STEMI complicated by total AV block. Coronary angiography demonstrated a large thrombus burden in the proximal right coronary artery (RCA). The patient undergoes a temporary pacemaker (TPM) followed by primary PCI. However, during the primary PCI, he developed a delirium followed by sudden onset of loss of power to the right upper and lower extremities. From our investigation, it was supposed to be a stroke as a neurological complication during the primary PCI procedure. There must be several points to be evaluated to prevent a similar complication occurring.

Conclusion: Stroke during primary PCI was very rare. However, it can be catastrophic and associated with prolonged hospitalization and patient morbidity. There were several points to be evaluated from this case. First, optimize pharmacologic treatment with Dual Antiplatelet Therapy (DAPT) and anticoagulant (with consideration) before undergoing primary PCI. Second, during primary PCI, a thrombus aspiration procedure must be used cautiously in case of a large thrombus burden, although, recent STEMI guidelines do not recommend regular use of thrombus aspiration. Third, we must consider and be well-prepared for the management of the periprocedural stroke as complications. Fourth, consider collaborating with neurologists to manage the complication after the primary PCI procedure.

KEYWORDS: *Inferior STEMI, Stroke, Primary PCI, A large thrombus burden*

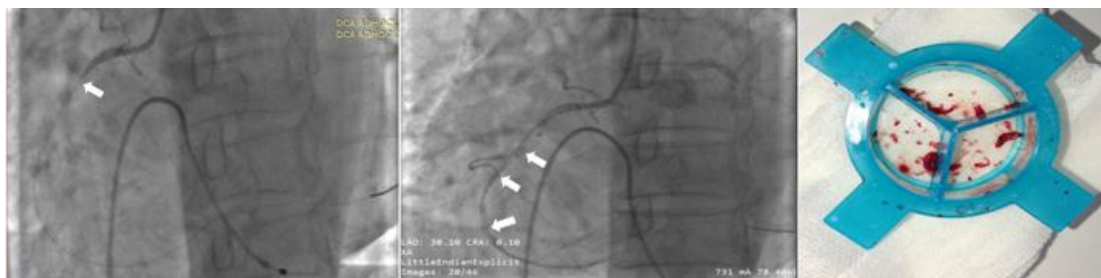


Figure 1. Coronary angiography of the patient and a large thrombus burden after thrombus aspiration.

CASE REPORT

Ultrasound for Guiding Therapy in Cardiogenic Shock: How Important?

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Background: Cardiogenic shock-associated mortality was declining over time. However, cardiogenic shock still possesses an ultimate risk of mortality, ranging from 35-40% in recent studies. Unfortunately, sometimes it's difficult to decide the early appropriate management based solely on physical examination. The use of ultrasound may help in guiding therapy according to the underlying cause.

Case Illustration: Female, 46-years old, came to ED due to worsening dyspnea a few hours ago accompanied with fatigue. Her blood pressure (BP) was 72/54 mmHg and heart rate (HR) was 150 bpm. Her extremities were cold and the capillary refill time was slow. On physical examination, she had rales on her both lungs, irregular heart sound, diastolic murmur 3/4 on the apex, elevated jugular venous pressure, and hepatomegaly. Her blood glucose was detected very low. Thus, she was given dextrose 40% and norepinephrine drip started at 0.05 mcg/kg/min. Her ECG showed atrial fibrillation with rapid ventricular response and right ventricular hypertrophy. The chest x-ray showed cardiomegaly with pulmonary edema and pneumonia. Her laboratory result came with leukocytosis, elevated liver enzyme, and hypoglycemia (Table 1). After titration of norepinephrine up to 0.15 mcg/kg/min, her BP was dropped to 58/39 mmHg. Dobutamine 10 mcg/kg/min along with furosemide infusion 5 mg/hour was given and she was transferred to ICU. In the ICU, bedside echocardiography was performed and the result was reviewed by a cardiologist via telemedicine. Chambers dilatation with severe mitral stenosis was noted (Figure 1). It's presumed that the patient might have a mixed shock from septic and cardiogenic shock. Therefore, an extra intravenous digoxin 0.5 mg followed by oral digoxin 0.25 mg once daily was given in addition to furosemide and dobutamine infusion, tapering off norepinephrine to 0.05 mcg/kg/min, and antibiotic. After switching the therapy, her condition improved. Rales and dyspnea were diminished. Her BP was 109/72 mmHg with minimal support. After 3 days of observation in the ICU, she was transferred to the inpatient ward. She was safely discharged on the sixth day.

Conclusion: Ultrasound in addition to clinical judgment may help in composing a tailored therapy for cardiogenic shock.

KEYWORD: *Ultrasound; Cardiogenic Shock; Telemedicine*

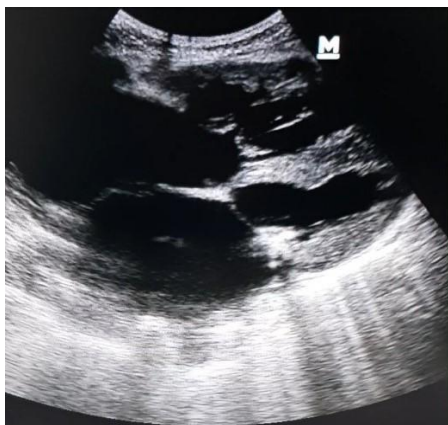


Figure 1. Bedside Echocardiography: LA, RA, and RV Dilatation with Mitral Valve Calcification

CASE REPORT

Timely Fibrinolytic Therapy in Anterior ST-Elevation Myocardial Infarction with Non-Sustained Ventricular Tachycardia in Rural Area

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Background: ST-elevation myocardial infarction (STEMI) can lead to devastating outcomes. Recent guidelines recommended primary percutaneous coronary intervention (PCI) as the first-line option for reperfusion. However, in a rural area, the availability of a PCI center is very limited, difficult to access, and yet costly for the patients. Thus, timely fibrinolytic therapy may provide an alternative treatment for STEMI in a rural area with comparable outcomes.

Case Illustration: A 54 years-old male, came to ED with chest pain 1-hour ago during activity. The chest pain was described as heaviness that radiated to his back. He denied other complaints such as shortness of breath, palpitation, nausea-vomiting, or diaphoresis. He was an active smoker with a history of diabetes and inferior STEMI two years in advance with poor compliance to medication. Vital Signs showed a Numeric Pain Scale of 8 on the admission. On examination, an irregular heart sound was found and ECG recording showed ST-elevation in V1-V5 with non-sustained ventricular tachycardia (Figure 1). The Troponin T level was still within the normal limit. The chest x-ray showed cardiomegaly with pulmonary edema. His TIMI Risk Score was 4/13 and GRACE Risk Score was 102. Aspirin 320 mg, clopidogrel 300 mg, atorvastatin 40 mg, isosorbide dinitrate 5 mg, ranitidine 50 mg, and fibrinolytic therapy using streptokinase 1.5 million units were given. Careful observation was done during the fibrinolytic administration. Meanwhile, the ECG showed frequent bigemini premature ventricular contraction. The chest pain subsided after the fibrinolytic. An evaluation ECG was taken and showed a more than 50% reduction of ST-elevation (Figure 2). The fibrinolytic was considered successful. The patient was then admitted to the ICU. After 2 days of observation, with the disappearance of chest pain and no major adverse cardiac events (MACE) was observed, he was transferred to inpatient ward and then discharged safely on the fifth day.

Conclusion: In absence of contraindication, fibrinolytic therapy should be done timely as a salvage procedure for STEMI patients in a rural area who can't access a PCI center.

KEYWORD: *ST-Elevation Myocardial Infarction (STEMI); Fibrinolytic; Reperfusion; Rural Area*

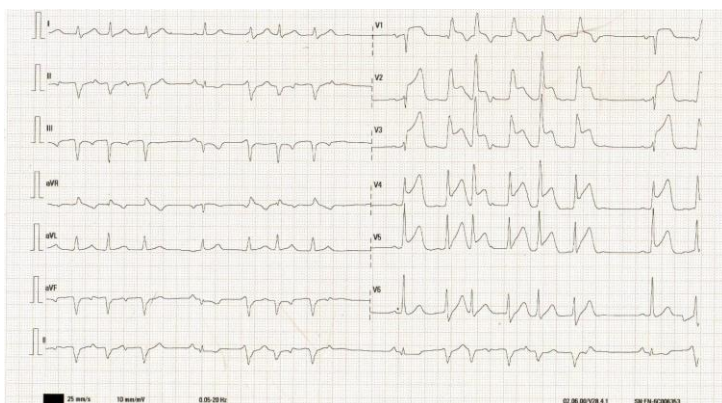


Figure 1. The ECG on Admission



CASE REPORT

Severe Thrombocytopenia on Disseminated Intravascular Coagulation State with Unexpected Presenting ST-Elevation Myocardial Infarct in Young Male Patient: The Dilemma in High Bleeding Risk Situation

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Background: STEMI clinically overt DIC and thrombocytopenia seems to be a very rare and dilemma situation. systemic activation of blood coagulation and inflammation may play a potential role in plaque disruption. Management of this clinical situation has not been an easy task for physicians due to associated bleeding risks.

Case Illustration: A 39-years-old male was presented to the ER with dyspnoea and high fever for 3 days before admission, he also complained of perirectal pain with recurrent purulent drainage that occurred during the last 7 years. Physical examination showed BP: 70/40 mmHg, HR: 130 bpm, RR: 30tpm, T: 39°C SpO₂: 97%. Laboratory findings showed high leukocytosis ($31,5 \times 10^3/\mu\text{L}$), severe thrombocytopenia ($13 \times 10^3/\mu\text{L}$), prolonged PT, and aPTT. Patient was diagnosed with septic shock due to perianal fistula and DIC. After 24-hours of treatment in ICU, the patient suddenly complained of typical chest pain accompanied by diaphoresis. ECG showed ST-segment elevation at inferolateral lead with increased troponin levels. Echocardiography showed regional wall motion abnormality and decreased LV function. The patient was planned for thrombocytes-transfusion and postponed dual antiplatelets, also anticoagulants because of high bleeding risk. On the second day after thrombocytes-transfusions and antibiotics, thrombocytes level was slightly increased ($32 \times 10^3/\mu\text{L}$) and we considered giving a single antiplatelet, clopidogrel 75mg once daily with close monitoring of bleeding signs. On the third day, after 5 bags of thrombocytes-transfusions, thrombocytes level was increased ($97 \times 10^3/\mu\text{L}$) and initiation of dual antiplatelets and anticoagulants was considered. During hospitalization, the patient was relatively stable and there was no bleeding episode. Patient was discharged after 8 days of hospitalization and planned for elective PCI. This is a challenging case because STEMI with DIC and thrombocytopenia are at high risk of both bleeding and ischemic events with significantly worse outcomes. Unfortunately, there are no specific guideline recommendations that suggest a treatment approach on this topic. Therapeutic decisions with antiplatelet and anticoagulant were mainly based on clinical situations and deserve particular attention and discussion.

Conclusion: The presented case shows how difficult it is to make therapeutic decisions in situations where the risk of opposing complications co-exists. but prompt decision of treatment is critical point for optimal outcome

KEYWORDS: STEMI, Thrombocytopenia, DIC, Young Male.

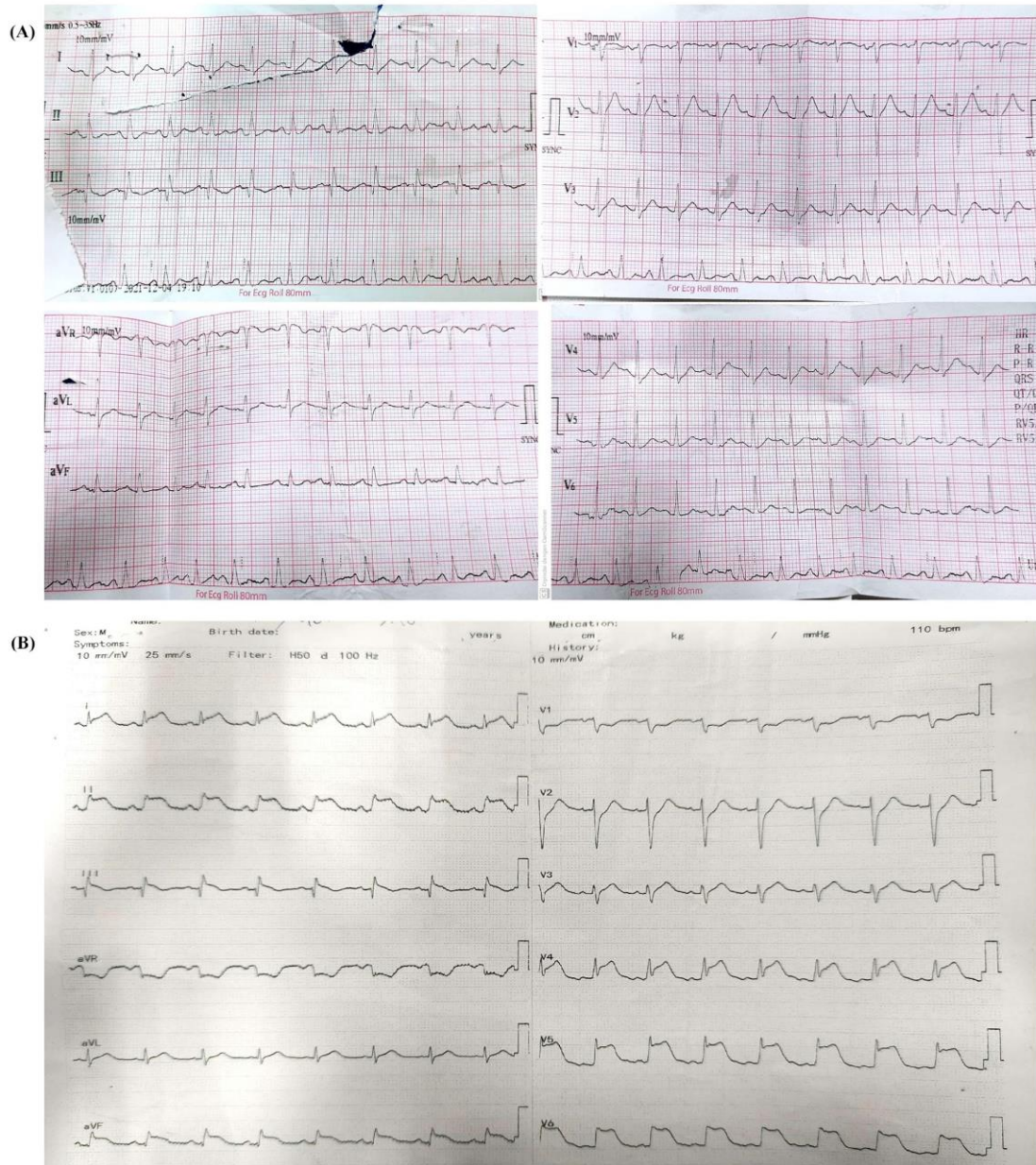


Figure 1. The patient was in severe thrombocytopenia on disseminated intravascular coagulation state with unexpected presenting STEMI, (A) E.R. ECG showed no ST-Segment Elevation, (B) 24-hours ICU admission ECG showed ST-Segment Elevation in inferolateral leads.



CASE REPORT

Repeated Fibrinolysis Therapy For St-Segment Elevation Myocardial Infarction

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Background: Thrombolytic therapy is established as effective treatment of acute myocardial infarction. About 20% of patients eligible for thrombolytic therapy have suffered a previous infarction, and about 9% of patients who receive thrombolytic therapy will suffer another infarction within a year. It is therefore important to know whether re-administration of thrombolytic agents is as safe and effective as initial administration.

Case Description: A 70-year old man was admitted to the Emergency Room (ER) due to angina pectoris 2 hours before admission. ECG examination showed anterior ST-segment elevation with right bundle branch block. Patient was given streptokinase 1.5 million units intravenously. ECG and clinical evaluation showed that the patient had failed fibrinolysis and was then referred to Sardjito Hospital for rescue PCI. This patient had a history of anteroseptal STEMI with right bundle branch block onset 6 hours and treated with Streptokinase 24 days from the current STEMI. From the previous STEMI, this patient was referred to Sardjito for rescue PCI due to failed fibrinolysis with two vessel disease stenting 1 DES at LAD and POBA at D1. This patient has not taken routine medication since the last 3 days before the current STEMI. We performed emergency coronary angiography with result stent patent at proximal LAD and an intrastent thrombus with TIMI 3 flow. This patient was decided to optimize medical therapy with heparinization and potent antiplatelet therapy switch from clopidogrel to ticagrelor. After the procedure, the patient was hemodynamically stable and treated in the ICCU. Streptokinase is a widely used thrombolytic agent. Because it is a foreign bacterial protein, it is antigenic. Antibodies to streptokinase develop within 3 to 4 days of administration, reaching a peak about 2 weeks later. Antibody levels then fall slowly, but a number of studies have shown that anti-streptokinase antibody titres remain increased for several years after therapy. If streptokinase is readministered to patients with high antibody levels, there may be a higher incidence of allergic reactions or other complications by immune complex mechanism and efficacy may be compromised due to the possibility that the patient's antibodies inactivate the second streptokinase dose. At 1 year after streptokinase infusion the antibody concentration and anti-streptokinase neutralizing activity were low in most patients tested, suggesting that a second SK dose will not be inactivated.

Conclusion: Repeated fibrinolysis may be considered in cases of reinfarction to minimize total ischemic time in those unable to reach PCI center quickly. Repeated fibrinolysis using streptokinase is associated with a higher incidence of allergic reactions and decreased effectiveness when given at intervals of less than 1 year. Therefore, repeated fibrinolysis is more appropriate when given using different fibrinolytic agents.

KEYWORD: STEMI, Fibrinolysis, Coronary artery disease



CASE REPORT

Acute Coronary Syndrome in Young Adult with Nephrotic Syndrome

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Background: Young patients represent 0.4-19% of all acute coronary syndrome (ACS) cases. Patients with nephrotic syndrome (NS) have long been assumed to be at increased risk for atherosclerosis and heart disease, including in young adults. Thrombosis and atherosclerosis are possible etiology for CAD among patients with NS.

Case Illustration: A 29-year-old man came to the emergency room in Cardiovascular Center of Harapan Kita (NCCHK) with chest pain radiating to left arm followed by sweating, nausea and vomiting 18 hours before admission, that he has never experienced previously, VAS Score was 9/10. Lower extremities examination showed bilateral pitting oedema. Patient was diagnosed with NS in 2009. Electrocardiography showed ST elevation in V1-V6, I, and aVL. Patients undergoing PCI and coroangiography showed thrombus grade V in LAD (high burden thrombus). It was decided to defer further maneuvers and proceeded to medical treatment with intravenous antiplatelet infusion and anticoagulation.

Conclusion: A case of a 29- year-old man patient with ACS and nephrotic syndrome has been reported. The management of ACS in nephrotic syndrome patients is challenging. Controlling the symptoms, correcting hypoalbuminemia and dyslipidemia as well as managing the acute coronary syndrome must be done in this patient.

KEYWORDS: *Acute coronary syndrome, STEMI, Nephrotic syndrome*



CASE REPORT

Left Anterior Descending Artery Total Occlusion Manifesting as ST-Elevation Myocardial Infarction Treated With Primary Percutaneous Coronary Intervention (PCI): A Case Report.

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Background: Coronary artery disease is a chronic disease caused by the gradual deposition of fats in the coronary arteries (atherosclerosis). Among other clinical presentations of coronary artery disease, acute coronary syndromes (ACS) are particularly worrisome as they are prevalent but at the same time portend a worse prognosis. ACS refers to any group of clinical symptoms compatible with acute myocardial ischemia and covers the spectrum of clinical conditions ranging from unstable angina (UA), non-ST-segment elevation myocardial infarction (NSTEMI), to ST-segment elevation myocardial infarction (STEMI). In STEMI, the infarct-related artery is usually totally occluded, and the Left Anterior Descending (LAD) artery is the most common cause of the occlusion. Immediate pharmacological reperfusion or Percutaneous coronary intervention (PCI) is the initial approach to obtain normal coronary blood flow.

Case Illustration: We report a case of a 40-year-old man with a history of uncontrolled hypertension and type 2 Diabetes Mellitus transferred to the emergency unit from another hospital without a PCI facility. He suffered a persistent typical chest pain for 6 hours and nausea. Physical examination showed blood pressure 140/80 mmHg, heart rate 89x/minute, and 97% of oxygen saturation. The Electrocardiogram (ECG) revealed ST-segment elevation in V2-V4, laboratory examination showed Troponin T level >2000 ng/L, non-fasting blood glucose 251 mg/dL. The patient has been treated with 5mg sublingual isosorbid dinitrate, 160mg aspirin loading dose, 300mg clopidogrel loading dose, 2.5 mg subcutaneous fondaparinux, 20 mg atorvastatin, and 0,5 mg/hour novorapid infusion. The patient underwent PCI within 15 minutes after hospital admission. Angiography revealed total occlusion in mid LAD, Drug-Eluting Stent (DES) implantation was successfully performed with the optimal angiographic result. Bisoprolol 2,5mg and ramipril 5mg were given afterward. His condition was stabilized and he was discharged on the third day of hospitalization.

Conclusion: The PCI revealed total occlusion of the mid LAD with a thrombus which was stented and the patient regained his cardiovascular stability. The time management of transferring patients and primary PCI procedures has become a great concern in this article.

KEYWORDS: Coronary artery disease, STEMI, PCI



CASE REPORT

Wellens' Syndrome in Rural Areas: What Can We Do?

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Background: Wellens' syndrome is pre-infarction stage characterized by an electrocardiographic (ECG) pattern characterized by biphasic (type A) or deep (type B) T-wave inversions in precordial leads result during chest pain free. ECG changes are highly specific for critical proximal stenosis of the left anterior descending (LAD) coronary artery. Recognition is important because it can lead to a massive anterior wall myocardial infarction.

Case Presentation: A 39-year-old man was referred to our ER complaining of typical cardiac chest pain and diaphoresis one day before admission. Patient felt chest pain for 2 hours the night before. Previously patients had intermittent chest pain for 5 days with the longest duration being 30 minutes and relieved by sublingual nitrate. The patient was an active smoker as a risk factor. On physical examination he was vitally stable, and cardiac auscultation revealed normal. Other systemic examinations were normal. ECG examination taken during chest pain free period showed normal sinus rhythm with biphasic T wave starting from V2 – V4 precordial chest leads indicating Wellens' syndrome type A. Routine laboratory results were within normal ranges. Cardiac enzyme testing wasn't available. Chest X-ray was normal. He was diagnosed with Wellens' syndrome and treated similarly to an acute myocardial infarction with double antiplatelet therapy, nitrate, statin and anticoagulant. The patient's condition was stable and chest pain free during treatment in ICU for 3 days and 2 days in the ward. Early revascularization therapy should be given after coronary angiography (CAG) to confirm the occlusion, but unfortunately in our rural area the facility is unavailable. Thus, the patient referred to the PCI center.

Conclusion: Identification of wellens' syndrome by its characteristic T-wave changes is important. ECG should be combined with coronary angiography prior to coronary intervention. Failure to recognize this clinical syndrome can result in significant morbidity and mortality because of immediate acute myocardial infarction.

KEYWORDS: Wellens' syndrome, biphasic T-wave inversions, proximal left anterior descending stenosis, acute myocardial infarction.



CASE REPORT

Acute Left Ventricular Dysfunction during Peripartum Period in Patient with Eisenmenger Syndrome

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Background: Heart failure is a common cause of morbidity and mortality in congenital heart disease (CHD). The interaction between genetic factors and postnatal factors found in CHD may play a major role in the development of heart failure. Cardiomyopathy is a rare condition but can cause serious complications and has a major contribution to high morbidity and mortality if it occurs in pregnancy. Peripartum cardiomyopathy (PPCM) can have serious complications and cause difficulty in management if it occurs in patients with Eisenmenger syndrome.

Case Illustration: A 22 years old female was consulted from an obstetrician colleague with a chief complaint of weakness in a patient with G1P0A0 with premature rupture of membrane and a history of congenital heart disease (CHD) with ventricular septal defect (VSD) with Eisenmenger. The patient complained of shortness of breath, dyspnea on effort, and orthopnea when entering the third trimester with no previous complaints during early pregnancy and the second trimester. The patient underwent cesarean section and during the peripartum period, there was a decrease in left ventricular function from around 60% to 30%. She also suffered from hypotension and improved with inotropes. It was found that there was a picture of tricuspid regurgitation with a TVG parameter of around 100 mmHg. The patient's saturation and blood pressure had decreased, so it was decided to give milrinone, sildenafil, iloprost, and delayed vasodilators. A few days later the patient's condition gradually improved and with the patient's ejection fraction at that time still persisting so that after the vital sign improved.

Conclusion: Acute ventricular dysfunction thought to originate from cardiomyopathy that occurs in pregnant women or early postpartum in most cases of cardiomyopathy will occur progressively rather than acutely. The acute LV dysfunction in these patients was a manifestation of the PPCM that occurred concomitantly and resulted in worsening of hemodynamic with the pre existing Eisenmenger physiology.

KEYWORDS: *peripartum cardiomyopathy, acute left heart failure, congenital heart disease, ventricular septal defect, Eisenmenger*



CASE REPORT

Primary Percutaneous Coronary Intervention in Dextrocardia and Mirror Image Patient, The Anatomical Challenge: A Case Report

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Background. ST-Elevation Myocardial Infarction (STEMI) in situs inversus dextrocardia or also called mirror-image dextrocardia is a rare case. Percutaneous coronary intervention could be challenging in the patient with dextrocardia because of difficulty in cannulating the coronary arteries, selection of catheters, catheter manipulation, and interpretation.

Case Illustration. A 63 years old woman presented to the emergency room with a sudden onset of typical chest pain. The chest radiograph confirmed dextrocardia. From the right electrocardiogram revealed normal sinus rhythm rate (74bpm) with ST elevation in leads V1-V4. Laboratory test were significant for elevated troponin-I : 15.00 (Normal : <0.01ng/mL). Echocardiography show the aorta abdominal in left side, inferior cava vein in right side and the hepar shows in the right side, which was suggestive of situs inversus. Diagnosis of acute ST segment elevation myocardial infarction was made and the patient sent to cardiac cath lab. Coronary angiography was done via a right trans-femoral approach using a 6F diagnostic catheter. Diagnostic catheter using Judkins Left 4cm and Judkins Right 4cm. The JL4/6F catheter passes through the aorta, which is on the right side of the spine and cannulates the left coronary artery easily. The JR4/6F catheter cannulated the right coronary artery with a counterclockwise torque instead of the usual clockwise torque. The patient was found to have left anterior descending coronary artery occlusion and underwent coronary artery stenting with results TIMI Flow 3.

Conclusion. Coronary angiography and primary percutaneous coronary intervention in patients with dextrocardia were important challenges to the operators. These challenges include arterial access, variation in anatomic, selection of catheters, techniques of torque of catheter.

KEYWORDS: *ST Elevation Myocardial Infarction ,Dextrocardia, Percutaneous Coronary Intervention*



CASE REPORT

High Degree Atrioventricular Block with Non-Sustained Ventricular Tachycardia in Metastatic Breast Cancer: Is It Electrolyte Imbalance or Chemotherapy Induced?

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Background: Zoledronic acid (ZA) is one of the important bisphosphonates that is widely used in bone metastatic cancer patients. The use of ZA has been linked to an increased risk of arrhythmia. The arrhythmogenic potential and ECG findings of ZA are unknown. In patients treated with ZA, there were no unfavourable arrhythmogenic effects during the acute term, but the long-term effects and pathophysiological mechanisms underlying ZA-related arrhythmias still need further research with a large number of patients.

Case illustration: A 35-year-old woman presented to the emergency room with fatigue, low intake, nausea, and vomiting in the last two weeks after administration of zoledronic acid (ZA). She had a history of invasive ductal lobular carcinoma in 2016 and since 2020 has been diagnosed with bone metastases and has been receiving zoledronic acid at 4 mg/30 days. Two days after admission, she had dyspnea and loss of consciousness. The ECG showed high-grade atrioventricular block with non-sustained ventricular tachycardia. She had repeated cardiac arrests in VT/VF mode and return of spontaneous circulation (ROSC) after a series of cardiopulmonary resuscitations. Laboratory findings showed bicytopenia (Hb 10,5 g/dl, Plt 44.000/mm³) and severe hypokalemia (K 1,31 mmol/dl), other findings were within normal limits. Rapid potassium correction and maintenance of amiodarone were given in the intensive care unit. The ECG showed normal sinus rhythm after administration of amiodarone and corrected potassium (K 4,67 mmol/dl). Unfortunately, the patient passed away 3 days later due to severe complications.

Conclusion: Patients with bone metastases treated with zoledronic acid are at risk of developing hypokalemia, which can lead to arrhythmias and, in severe cases, death. Several possible mechanisms, including an activated inflammatory state, altered electrolytes impacting cardiac conduction, and long-term atrial structural changes. To keep the patient from having severe adverse effects, monitoring of symptoms, physical condition, and laboratory tests is required on a regular basis. However, the timing of routine examinations for these patients needs further research to strike a balance between cost and benefit.

KEYWORDS: *high degree atrioventricular block, non-sustained ventricular tachycardia, hypokalemia, zoledronic acid, metastatic breast cancer*



CASE REPORT

Switching Ventilation Strategy in Patient with Mechanical Ventilation

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Background: Mechanical ventilation has become a cornerstone treatment of critical care patients in the last decade. Various ventilation modes are currently available from controlled, synchronized intermittent mandatory and pressure support ventilation. Despite its benefit, mechanical ventilation can cause ventilator induced lung injury as damage. Recognition of patient condition plays an important value in the selection of ventilation mode.

Case Presentation: 74 years old female was referred to RSUP Dr Sardjito with typical chest pain for 4 days before admission. Because of COVID-19 pandemic she didn't go to hospital on the first symptom. She had history of unstable angina pectoris on 2015. She had uncontrolled hypertension, diabetes and menopause. From physical examination shown pulmonary edema with evolution of anterior STEMI and non-sustained ventricular tachycardia. By Cor Angiografi found coronary artery disease three vessel disease (CAD-3VD). On the next day the patient had worsening of symptoms and was diagnosed with re-infarct anterior Killip IV with uncompensated metabolic acidosis. Patient then intubated and undergo emergency PCI in LM-LAD. Patient had mechanical ventilation for acute heart failure with use of volume tidal 6-8 ml/PBW, PEEP 3-5 cmH₂O and pressure support 10-15. Mechanical ventilation weaning can be achieved on the fifth days. On day 8, the patient had acute respiratory distress syndrome (ARDS) that was caused by worsening of pneumonia by *Acinebocater baumani* mixed with fungal infection. Blood gas analysis showed uncompensated respiratory acidosis (pH 7,16, pCO₂ 66,5, pO₂ 76, HCO₃ 24). Patient then had changes of ventilation strategy that used permissive hypercapnia with maintains pCO₂ at 80-100 mmHg, saturation > 90 % and pH > 7.2. Patient was admitted in ICCU for 36 days. This ventilation strategy becomes a choice in ARDS, asthma and COPD. It plays as protective lung strategy that reduces infection, increases immune response and prevent tissue damage

Conclusion: Ventilation mode selection and its changes are based on the patient condition to increase patient survival.

KEYWORD: ARDS, Mechanical Ventilation, Permissive Hypercapnia, STEMI.



CASE REPORT

Libman-Sacks endocarditis manifested as right heart-sided thrombus in systemic lupus erythematosus: a case report

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Background: Libman-Sacks endocarditis (LSE) is a pathological condition where there is sterile vegetation in the heart valve. It is a rare cardiac manifestation of autoimmune disease.

Case summary: A 22-year-old female patient was referred to RSUP Dr Sardjito with the chief complaint of sudden dyspnea while undergoing gallstone surgery. From the physical examination, it was found that oxygen saturation is 80% in room air, lung sound is clear and no rales found. Echocardiography showed a thrombus in the left pulmonary artery and a thrombus in the right atrium, right ventricle, and tricuspid valve. Blood culture is negative. The patient underwent a laboratory examination to establish the cause of thromboembolism. The patient was diagnosed with hypercoagulable stage systemic lupus erythematosus (secondary antiphospholipid syndrome) with cardiac involvement presenting with LSE. Antiphospholipid syndrome is an autoimmune disease that causes arterial and venous thrombosis. This group of autoimmune diseases produces valvular manifestations such as thickening of the valves and formation of vegetation. Our patient is known to have Libman-Sacks endocarditis after manifestation of pulmonary embolism in the form of sudden dyspnea. Echocardiography of the patient, in this case, showed a thrombus in the right heart. The thrombus is thought to be of intracardiac origin associated with an autoimmune process.

Conclusion: In addition to definitive autoimmune therapy, anticoagulation should be considered as secondary prevention for thromboembolic phenomena in patients who have had a thromboembolic event.

KEYWORDS: *Libman-Sacks endocarditis, systemic lupus erythematosus, right-heart sided thrombus*



CASE REPORT

Dancing Thrombus in the Right Atrium: Is It the Final Destination?

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Background: Right atrial thrombus is an underdiagnosed condition with a high mortality rate. Pulmonary embolism is associated with significant mortality especially if compounded by hemodynamic instability, RA thrombus, and other complications. Diagnosis is challenging and sometimes requires advanced modalities. Management of bleeding risk is relevant throughout the course of treatment.

Case Illustration: A-52 years old man presented with difficulty in breathing during exercise which was worsening in 2 days. Diastolic murmur was heard at the apex during physical examination. Echocardiography showed moderate mitral stenosis with left atrial and right atrial thrombus. Enoxaparin bridging with warfarin were given during hospitalization. On the 10th day of treatment, the patient complained of sudden dyspnea accompanied with hemoptysis and diaphoresis. Laboratory tests showed a rise of INR value to 9.79 seconds but D-dimer was elevated to 12.1 µg/ml. Echocardiography evaluation showed disappearance of RA thrombus but no sign of pulmonary embolism. Anticoagulants were discontinued due to bleeding complications despite high risk of pulmonary embolism. Patient was scheduled to undergo computed tomography pulmonary angiography. CTPA found bilateral pulmonary embolism mainly in the right lower lobe with pulmonary hypertension and pulmonary infarction. Patient was treated with warfarin after normalization of INR and was planned for a 3 months evaluation.

Conclusion: A patient with an RA thrombus complicated by pulmonary embolism was reported. Bleeding during treatment is another problem and must be taken into consideration. There is however no evidence supporting the avoidance of long-term anticoagulation in patients with a high estimated bleeding risk. A shared decision-making regarding continuation or discontinuation of anticoagulation should be discussed with the patient.

KEYWORDS: *Pulmonary embolism, right atrial thrombus, computed tomography pulmonary angiography, cardiac thrombus, bleeding complication.*



CASE REPORT

Case Report: Cardiac Tamponade After Removal Temporary Pacemaker in Patient with Acute Inferior Myocardial Infarction and Total Atrioventricular Block

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Introduction: Cardiac tamponade is a medical emergency, which needs early recognition. Cardiac tamponade by pacemaker is a rare but potentially fatal complication. Acute perforation occurring within twenty-four hours after removal can lead to hemopericardium, cardiac tamponade, and death. We report the case of a 72 years old patient with shock cardiogenic after removal temporary pacemaker due to cardiac tamponade

Case Illustration: A 72-year-old man with acute inferior myocardial infarction, right ventricular infarction, and total atrioventricular block came to emergency. Patient then perform primary percutaneous coronary intervention and insertion of temporary pacemaker. The coroangiography showed right coronary artery (RCA) multiple stenosis 60-90% at ostial-proximal and total occlusion in mid, then performed three drug eluting stent at RCA. After 5 days in intensive care unit, the electrocardiogram (ECG) showed sinus rhythm, then decide to remove temporary pacemaker. Before removal pacemaker, the echocardiography did not showed effusion, but after 20 minutes, patient complain dyspnea, with blood pressure 70/30mmHg and tachycardia. Echocardiography showed effusion with diameter at lateral left ventricular 1,1cm and diameter at lateral right ventricular 1.8cm, with right ventricular dan right atrium collapse. Patient then given inotropic and vasopressor, then perform pericardiocentesis emergency. After pericardiocentesis, hemodynamic back to normal. Myocardial perforation is a rare complication following pacemaker implantation that may cause cardiac tamponade. Perforations involve the right ventricle apex, which is thinner, more commonly than the intraventricular septum or the outflow tract. Early recognition and treatment of cardiac tamponade is essential to prevent fatal outcome. The initially treatment is resuscitated with intravenous fluids to promote maximum filling of the heart and the definitive treatment of cardiac tamponade is the removal of cardiac diastolic restriction by either pericardiocentesis or thoracotomy. Several studies have reported various factors that serve as predictors of lead perforation. These include temporary leads, steroid use, active fixation leads, low body mass index, older age, female gender, and anticoagulant.

Conclusion: Though the complications arising from pacemaker insertion are uncommon, they can be life threatening and hence should be considered in all patients with cardiac pacemakers in the appropriate clinical setting

KEYWORDS: *temporary pacemaker, shock cardiogenic, tamponade*



CASE REPORT

ST Segment Elevation Following Major Operative Non-Cardiac Surgery: What Is The Cause?

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Background: Post operative cardiac injury can be caused by myocardial infarction or non-coronary cause. Stress cardiomyopathy or Takotsubo syndrome (TTS) is a condition of severe heart muscle weakness due to intense emotional or physical stress. Whereas, perioperative myocardial infarction (PMI) is an uncommon and life-threatening event with the incidence 0.9%-11% with peak incidence within the first 3 days after surgery. Diagnosing the cause of cardiac injury is crucial for optimizing management of the patient.

Case Description: A 74-year old woman was admitted to Sardjito Hospital with urinary bladder squamous cell carcinoma and vesicocutaneous fistula. The patient then underwent collaborative surgery between Urology, Digestive Surgery, and Plastic Surgery Department with a total of 17-hour -long surgery. The patient underwent radical cystectomy with trans ureterocutaneostomy (TUUC) with defect closure and transverse colostomy. After surgery, the patient was admitted to the surgical intensive care unit for 1 day then moved to the high care unit. Two days post-operative, the patient was consulted with electrocardiography (ECG) showing ST-elevation at anterior and lateral lead. Echocardiography result after surgery revealed a decreased of systolic function from 65% to 40% with akinesia at apical, and hypokinetic at inferoseptal, anterolateral, inferior, and hypokinetic at anterior, anteroseptal and inferolateral. The patient had nonspecific chest pain symptoms, with no risk factor of coronary artery disease (CAD) such as hypertension, diabetes mellitus, and dyslipidemia. The patient had no prior history of cardiovascular diseases with cardiac risk index (CRI) revised class II with low major adverse cardiac event 6%. The patient had hemoglobin level 10.3 g/dL. High-sensitivity troponin I (hsTropI) level increased with level of 3443.6 ng/L, Creatinine kinase (CK) 243 U/L, and normal Creatinine Kinase -MB (CKMB) 23 U/L. InterTAK score from International Takotsubo Registry showed score 50. InterTAK score has cut off 40 score points. Score value of ≥ 50 has sensitivity 94.7% as TTS. Most patients have a small early increase in cardiac biomarkers, in this patient the level of hsTropI was above 99th percentile but normal CKMB. Due to limitation of diagnosis, we try to confirm the reversibility of systolic function. The systolic dysfunction and the regional wall-motion abnormalities in TTS usually are transient and resolve completely within weeks.

Conclusion: Differentiating the cause of perioperative cardiac injury is important for management of the patient. InterTAK score could be applied in post-operative settings to differentiate stress cardiomyopathy and acute coronary syndrome. Preoperative cardiovascular evaluation is recommended to predict cardiovascular events after noncardiac surgery.

KEYWORD: *post operative injury cardiac injury, interTAK score*



CASE REPORT

Double Culprit Lesion ST Elevation Acute Miocard Infarct

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Background: Acute ST-segment elevation myocardial infarction (STEMI) is caused by a complete occlusion of a coronary artery resulting in sudden impairment of perfusion in the territory supplied by the infarct related artery. Simultaneous acute total occlusion of more than one epicardial vessel is quite rare and always life threatening because of rapid hemodynamic deterioration leading to cardiogenic shock

Case Illustration: A 56 year old male patient presented with the main complaint of chest pain. He was hemodynamically stable without arrhythmia or conduction block. Cardiac risk factor in this patient was heavy smoker. He has no history of hypertension or diabetes mellitus. The presentation of his electrocardiography (ECG) was ST elevation in lead II, III, AVF and hyperacute T in V3 and V4. He underwent primary percutaneous coronary intervention (PCI) with result total occlusion in left ascending artery (LAD) and right coronary artery (RCA). Simultaneous thrombosis affecting ≥ 1 coronary artery has been reported to occur in about 4.8% of the cases at the time of primary percutaneous coronary intervention (PCI). The etiology behind simultaneous multiple coronary thrombosis can be broadly categorized into identifiable and unidentifiable causes. In this case, the underlying etiology remains unidentifiable. Multiple plaque rupture has been postulated as the main theory behind the cases without an identifiable cause. Hyper Catecholamine states, which enhance thrombosis and vasoconstriction, may trigger an acute coronary syndrome if they coincide with the exact time of plaque disruption. Of particular importance is the growing evidence of enhanced platelet reactivity and thrombin generation in cigarette smokers, which also may be related to the release of catecholamines.

Conclusion: Simultaneous multivessel coronary thrombosis is a rare presentation of STEMI and is associated with high incidence of complications including cardiogenic shock and ventricular arrhythmias. This patient was admitted to the hospital hemodynamically stable without arrhythmia or conduction block. The causes of acute occlusion of multiple coronary vessels in this patient remain elusive

KEYWORDS: *double culprit lesion, simultaneously thrombosis, multiple coronary occlusion, STEMI, cardiogenic shock*



CASE REPORT

Acute Anterior Extensive et Inferior and Right Ventricle ST-Elevation Myocardial Infarction with Ambiguous Culprit Lesion

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Introduction: Rarely in the case of ST-Elevation Myocardial Infarction (STEMI) there is an unclear culprit lesion. In case of combined ST elevation in the anterior and inferior leads, these changes may be due to distal occlusion of the “wrapped” left anterior descending artery (LAD) or double culprit. Generally, double culprit comes with a clinically unstable presentation. Therefore, a patient with an ECG with two infarct territories and clinically stable, it is very important to determine the culprit for a revascularization strategy.

Case illustration: A 64-year-old female patient referred to RSUP Dr. Wahidin Sudirohusodo Hospital presenting with an anterior extensive et inferior wall STEMI Killip I with 22 hours of onset after failed fibrinolytic. The patient remains chest pain on admission. Rescue PCI was performed and we found 80-90% diffuse stenosis LAD with TIMI 3 flow and 60-70% stenosis in LCx. RCA angiography showed 95-99% distal stenosis with TIMI 3 flow without thrombus. Based on angiographic and clinical characteristics, a double culprit is less likely and we considered LAD could be the culprit referring to ECG presentation and lesion characteristic on the proximal side, even though RCA could still be the culprit because of the very tight lesion. Because of the ambiguity of the culprit lesion, we decided to execute multivessel PCI strategy at the time of index procedure. Angiography evaluation after stent implantation revealed TIMI 3 flow. Echocardiography evaluation showed decreased LV systolic function (EF 33.1% by Biplane) with wall motion abnormality according to infarct territory based on 2D speckle tracking. The patient was hospitalized for 5 days and was discharged with hemodynamically stable without further complication.

Conclusion : We reported an anterior extensive et inferior wall STEMI with ambiguous culprit finding. We decided to open both vessels with good clinical and angiographic results.

KEYWORD : STEMI, Culprit, PCI, Revascularization



CASE REPORT

Different Scenario with Different Approach for Acute Pulmonary Embolism in Pregnancy: A Case Series

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Background: Pulmonary embolism spans a broad spectrum of illness, ranging from asymptomatic to pressor-dependent pulmonary embolism. We present two cases of patients with acute pulmonary embolism in pregnancy and implemented a severity-adjusted management strategy in both cases.

Case presentation: The first patient was a 25-year-old woman in 12th week gestation, admitted to the emergency room due to sudden onset of dyspnea accompanied with unilateral leg swelling. The blood pressure was 130/70 mmhg, the pulse was 120 beats/min, respiratory rate was 26 times/min, and oxygen saturation was 98%. Electrocardiogram revealed sinus tachycardia, RAD, S1Q3T3 pattern, and T wave inversion V1-V4. Echocardiography revealed right ventricular overload. Laboratory studies showed increased D-dimer 1500 ng/ml and normal Troponin I < 0,01 (<0,02). CT Pulmonary Angiogram showed thrombus at distal RPA and LPA. We diagnosed the patient with intermediate low risk acute pulmonary embolism and deep vein thrombosis. She was given subcutaneous injections of enoxaparin 0.6 cc twice a day. She was discharged with self-injection enoxaparin. The second patient was a 42-year-old woman in 8th week gestation, admitted to the emergency room due to sudden onset of severe dyspnea accompanied with unilateral leg swelling. The blood pressure was 90/70 mmhg, the pulse was 115 beats/min, respiratory rate was 30 times/min, and oxygen saturation was 92%. Electrocardiogram revealed sinus tachycardia and S1Q3T3 pattern. Echocardiography revealed right ventricular overload and multiple thrombus in Right Atrium. Laboratory studies showed increased D-dimer 1850 ng/ml and increased Troponin I 0,12 ng/ml. The patient was diagnosed with intermediate high risk acute pulmonary embolism and deep vein thrombosis. We did thrombolysis with streptokinase. Hours after thrombolysis, the dyspnea has improved, the patient's blood pressure increased to 110/70 mmhg, the pulse decreased to 90 beats/min, the oxygen saturation increased to 99%. We did echocardiography evaluation 24 h after thrombolysis, RA thrombus disappeared and right ventricular function improved. She was discharged with self-injection enoxaparin.

Conclusion: Risk assessment should begin upon suspicion of acute pulmonary embolism. The presence of RV strain by both biomarkers and imaging indicates a high-risk, submassive pulmonary embolism and worse prognosis, which need more aggressive treatment.

KEYWORDS: *Acute Pulmonary Embolism, RV failure*



CASE REPORT

Accelerated Idioventricular Rhythm in Suspected Guillain-Barré Syndrome: How Can It Happen?

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Background: Guillain-Barré syndrome (GBS) is an acute immune-mediated disorder in the peripheral nervous system with progressive, idiopathic, symmetrical weakness and loss of deep tendon reflexes. Autonomic dysfunction has been described in GBS may demonstrate edema and inflammation of autonomic ganglia and destruction of peripheral ganglion cells. There is a wide range of clinical cardiac manifestation such as heart rate variability, blood pressure variability, cardiomyopathy, and electrocardiographic changes. The GBS is currently the most frequent cause of acute flaccid paralysis worldwide and has serious complications.

Case illustration: A 38-year-old man presented to the emergency department with paraplegia one day before admission. Five days before, he felt numbness and pain in his right leg followed by left leg the day after. He had a history of hypertensive urgency 2 days earlier and received ketorolac injection for his pain but on the day of admission the vital signs were within normal limits. Physical examination for the superior extremity was normal for motoric, sensory, physiological reflexes, and no pathologic reflexes, but for the inferior extremity was paraplegia, paresthesia, diminished physiologic reflexes, and no pathological reflexes. Laboratory findings showed leukocytosis 20.050/mm³, increased ureum 44 mg/dL, increased creatinine 1.66mg/dL, and hyperglycemia 307 mg/dL, acidosis metabolic incomplete correction. He was diagnosed as paraplegia flaccid cum hypoesthesia segmental L1 suspect Guillain Barré Syndrome, hyperglycemia, acute kidney injury. Eleven hours after admission, he had dyspnea and got intubation because of desaturation. Six hours later, the ECG showed accelerated interventricular rhythm and became cardiac arrest. The patient passed away after 40 minutes of cardiopulmonary resuscitation.

Conclusion: Arrhythmias are hypothesized to be associated with heart-related autonomic afferent demyelination, direct myocardial involvement, or respiratory failure. It is thought that pathological lesions exist surrounding the vagal nuclei, and that the vagal center's involvement in the brainstem leads to autonomic cardiac dysfunction. Direct and polymorphonuclear cells, as well as myocardial necrosis, are further risk factors independent of autonomic cardiac innervation, and these alterations are also related with poliomyelitis. Close monitoring is needed in order to anticipate deteriorating.

KEYWORDS: *accelerated idioventricular ventricular rhythm, Guillain-Barré syndrome, paraplegia, paraesthesia, paraparesis*



CASE REPORT

Synchronized Cardioversion As First Line Treatment In Patient With Stable Supraventricular Tachycardia : An Acute Management In Limited Emergency Unit

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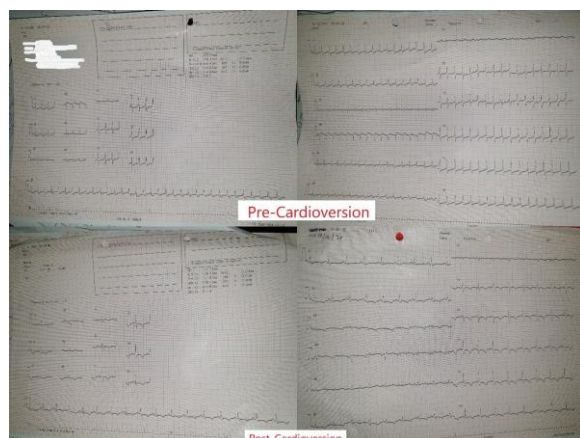
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Background : Cardioversion, especially with synchronized mode, has been widely used as a first line treatment for unstable supraventricular tachycardia (SVT) in tachyarrhythmia algorithm. However, in current guidelines, there is no recommendation to use synchronized cardioversion as a first line treatment for stable supraventricular tachycardia unless pharmacological therapy does terminate the tachycardia or is contraindicated. This case presents the usage of synchronized cardioversion as a first line treatment on patients with stable SVT in a limited emergency unit.

Case Illustration : A 26-year old female patient was admitted to our emergency unit due to worsening epigastric pain along with nausea and profuse vomiting on that day. She also complained of a sudden onset of palpitation relieved by resting within a few minutes without any history of chest pain, syncope or dyspnea. First time in the ER, BP 90/60, HR 205x/minute, RR 20x/minute, mental status was compositus (CM) and glasgow coma scale (GCS) score of 15. Electrocardiogram (ECG) recorded a regular narrow QRS tachycardia rhythm without any visible p wave, a SVT was suspected. Initially, the patient was given symptomatic treatment such as omeprazole and ondansetron for her dyspepsia symptoms. The patient was not administered adenosine due to lack of resources and was not done any vagal stimulation maneuver following the cardiologist advice. As time progressed, the patient's vital sign was monitored and found that BP 118/70, HR 107x/minute, RR 20x/minute, mental status was CM and GCS score of 15. Then, synchronized cardioversion was delivered to the patient in conscious condition at initial dose 100 J following premedication with double dose of fentanyl and midazolam, respectively . SVT was still not terminated and then another cardioversion was given with increased dose to 200 J. After that, SVT was terminated and ECG recorded sinus rhythm with widespread ST depression, a global ischemia was suspected.

Conclusion: In limited emergency units, synchronized cardioversion on patients with stable SVT shows successful conversion to sinus rhythm and is proven to be safe as a first line management once analgesic and sedative are premedicated before cardioversion.

KEYWORDS : Cardioversion, First Line Treatment, Stable SVT, Emergency Unit, Tachycardia



CASE REPORT

Resolution Of Hyperacute T Wave On Anterior Stemi Post Dual Antiplatelet Loading Without Reperfusion Therapy In Limited Health Settings : Does It Still Need Prompt Primary Percutaneous Coronary Intervention ?

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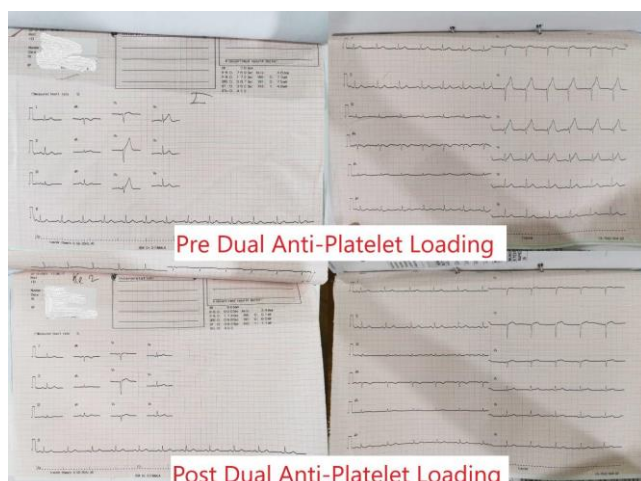
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Background: Tall and broad-based T waves, referred to as hyperacute T wave, may present as the earliest ECG finding of transmural ischemia in ST-elevation myocardial infarction (STEMI). Coronary reperfusion is still widely regarded as the global gold standard of practice for STEMI. However, at times, hyperacute T waves had already resolved even before reperfusion took place attributed to STEMI dual antiplatelet protocol. The aim of this case report was to highlight the dilemma in managing a patient with resolving hyperacute T wave of STEMI in the hospital lacking primary percutaneous coronary intervention (PCI) facility.

Case Illustration: A 60-year-old male was admitted to our emergency department unit due to a persistent 3-hour angina. While driving a car, he reported a sudden onset of a sharp and stabbing sensation of chest pain radiating to the left hand. In the ER, BP 140/60, HR 85x/min, RR 20x/min. First ECG in admission recorded hyperacute T wave in V2-V3 leads with reciprocal ST depression in inferior leads. A serial ECG was done within 10 minutes after loading of dual antiplatelet (160 mg aspirin and 300 mg clopidogrel) and showed a resolution of hyperacute T wave in leads V2-V3. Cardiac enzyme measurement was obtained 3 hours after admission showed a significant raise of standard troponin T level. STEMI protocol, such anti-anginal and dual antiplatelet loading had been done in the ED, yet reperfusion therapy by either fibrinolytic or primary PCI cannot be done due to limited facilities. Currently, studies suggest that primary PCI is still needed after resolution of hyperacute T wave in STEMI following dual anti platelet loading, since that therapy could only reduce the rate of occlusion of the infarct-related artery. Furthermore, current guidelines also recommend that hyperacute T wave as the earliest sign of STEMI is indicated for prompt PCI as long as clinical symptoms are consistent with myocardial ischemia.

Conclusion: In limited health facilities, resolution of hyperacute T wave in STEMI due to dual antiplatelet loading should not delay the patient's referral for reperfusion strategy as primary PCI is a must management following dual antiplatelet loading therapy in STEMI.

KEYWORDS: *Hyperacute T Wave Resolution ; STEMI ; Dual Antiplatelet ; Reperfusion Therapy ; ACS*





CASE REPORT

Management of Patients with Pulmonary Hypertension Crisis and Right Heart Failure

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Background: Pulmonary hypertensive crisis (PHC) occurs when compensatory mechanisms fail, right ventricle (RV) systolic function decompensates and left ventricle (LV) preload acutely decreases resulting in abolished cardiac output and coronary perfusion. RV failure following or in the context of severe rise of pulmonary vascular resistance (PVR) is a challenging complication of PH and is associated with substantial morbidity and mortality.

Case Illustration: A 31 years old female came to the emergency room (ER) of Dr. Sardjito Hospital complaining of shortness of breath with hemoptysis. She had been diagnosed with pulmonary hypertension group 1 since 2015 with routine treatment sildenafil 3x40 mg, spironolactone 1x50 mg, warfarin 1x2 mg, furosemide 1x40 mg, bisoprolol 1x5 mg, and digoxin 1x0.25 mg. In the ER, her blood pressure was 80/50 mmHg, with heart rate (HR) 100 bpm, respiratory rate 28 bpm, and oxygen saturation 87%. On physical examination, we found elevated JVP, cardiomegaly, RV heaving, loud P2, carvallo sign, rales and crackles in both lungs, and also ascites and hepatomegaly. The ECG showed sinus rhythm with HR 110 bpm, right axis deviation, pulmonal P, right atrial hypertrophy, RV hypertrophy, and complete right bundle branch block. Her blood gas analysis showed pH 7.39, pCO₂ 21.7, pO₂ 44, BE -12, HCO₃ 13.2, SO₂ 80%, and lactate 2.44. She was diagnosed with PHC and RV failure. In 2 days, the patient's condition deteriorated. She was intubated and was given dobutamine drip, iloprost nebulization every 8 hours, sildenafil 3x40 mg, digoxin 1x0.25 mg, and codeine 3x10 mg. Despite the effort, the patient deteriorated further and eventually died in the intensive cardiac care unit (ICCU).

Conclusion: Early recognition of patients at particular risk of PHC, and timely establishment of efficient therapeutic actions and hemodynamic monitoring may prevent the development of severe cardiac dysfunction, low cardiac output, and death. Invasive monitoring with an arterial and a central venous line should be established in all patients with cardiopulmonary compromise in whom vasopressor or inotropic therapy may be necessary.

KEYWORD: *Pulmonary hypertension crisis, management, right heart failure*



CASE REPORT

Cardiac Tamponade In Systemic Lupus Erythematosus

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Background. Systemic lupus erythematosus (SLE) is a chronic autoimmune disease that has many clinical manifestations and may involve any organ system. Cardiac involvement is frequent and can be observed in more than 50% of patients with SLE. Cardiac tamponade is a rare but life threatening complication of systemic lupus erythematosus (SLE). In the treatment, timely pericardial puncture can gain time for subsequent treatment, including corticosteroids and DMARD therapy

Case Illustration. A- 41 years old female without any formerly diagnosed disease presented with difficulty of breathing and fatigue for approximately 2 weeks which was worsening a few hours before admitted to the emergency ward. She also had a history of pain in small joints for around 2 months. There was no family history of SLE. Physical examination showed the typical Becks' triad which were the muffled heart sound, jugular distention, blood pressure 80/50 along with body temperature 37.5°C and heart rate of 120 beats/minute. The electrocardiogram showed sinus tachycardia and low voltage. The chest radiograph showed enlargement of the cardiac silhouette. The echocardiography presented a large pericardial effusion with diastolic collapse of the right atrium, dilated vena cava, and the swinging heart, confirmed as cardiac tamponade. Emergency pericardiocentesis was then performed with drainage of 300 cc of amber colour fluid (exudate) with improvement in hemodynamics. Pericardial fluid analysis revealed no sign of significant bacterial infection and blood test showed no markable elevation of cancer marker. During the hospital stay the patient was discovered with a mouth ulcer which led to suspicion of systemic lupus erythematosus diagnosis. The urinalysis showed +3 for protein urine, 10-15 for erythrocytes and ANA IF test was performed which resulted to be positive, confirming the diagnosis of SLE. Patients were treated with high dose corticosteroid, hydroxychloroquine and mycophenolate mofetil sodium. Patient then showed a remarkable improvement on clinical presentation and on the 20th day of treatment the pigtail catheter was alienated.

Conclusion. Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease with worldwide prevalence that affects almost all organs of the body. Pericarditis is the commonest cardiac manifestation of lupus occurring in around 9-54% patients with lupus. Cardiac tamponade is much rarer with incidence <2.5%. In patients with known SLE, cardiac tamponade was more described in women and in patients with anemia, renal disease, pleuritis, higher ESR values and lower C4 levels. In the majority of cases, treatment consists of high doses of glucocorticoids and hydroxychloroquine after urgent pericardial fluid withdrawal.

KEYWORD: *Cardiac tamponade, Systemic lupus erythematosus, Pericardiocentesis*



CASE REPORT

Total Atrioventricular Block in Patients with Chronic Subdural Hematoma: Causality or Coincidence?

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Introduction: Total atrioventricular block (TAVB) is an AV node dysfunction characterized by loss of conduction between the atria and ventricles resulting in atrioventricular dissociation. The etiology of TAVB includes a myriad of causes such as ischemic heart disease, myocardial infarction, increased vagal tone, or drug effects. One of the causes of reversible TAVB that is very rare is the presence of a neurological disorder. Chronic subdural hematoma (CSDH) generally does not cause symptoms until there is an increase in intracranial pressure (ICP). In acute SDH, an increase of ICP triggers a response to bradycardia, hypertension, and respiratory distress. But, there is limited data that convey the relationship between CSDH and TAVB. We highlight the clinical course of the event and whether there were any relationships between TAVB and CSDH.

Case Illustration: A 78 year old man came to the emergency department of Sardjito hospital with a complaint of near syncope accompanied by a history of loss of consciousness. The patient was referred from a previous hospital with a diagnosis of TAVB with suspected stroke. Physical examination of vital signs showed the patient was somnolent with blood pressure (BP) 115/60 mmHg, heart rate 20x/mnts, respiratory rate (RR) 20x per minute, and temperature (T) 36.5⁰C. The ECG showed total AV block with atrial fibrillation rhythm with HR 24 bpm. Patient then underwent head CT and showed a chronic subdural hematoma. Temporary transvenous pacemaker was performed to the patient and admitted to CVCU. Patient was observed in order to determine the etiology of TAVB. Patient then underwent burr holes surgery to remove the hematoma and we would observe whether the TAVB was reversible after surgery. After hematoma evacuation, ECG still showed TAVB, so the patient planned to have a permanent pacemaker implantation.

Conclusion: In the case of TAVB with CSDH, it should be carefully investigated whether there is a relationship between them. In this case, TAVB was most likely caused by a degenerative process, but also affects the risk of chronic SDH (coincidence) occurrence, so pacemaker implantation was the appropriate management for patients.

KEYWORDS: TAVB, CSDH, Causality, Coincidence, Pacemaker

CASE REPORT

Chest Pain On Day One In Covid-19 Isolation Ward: What Do We Know?

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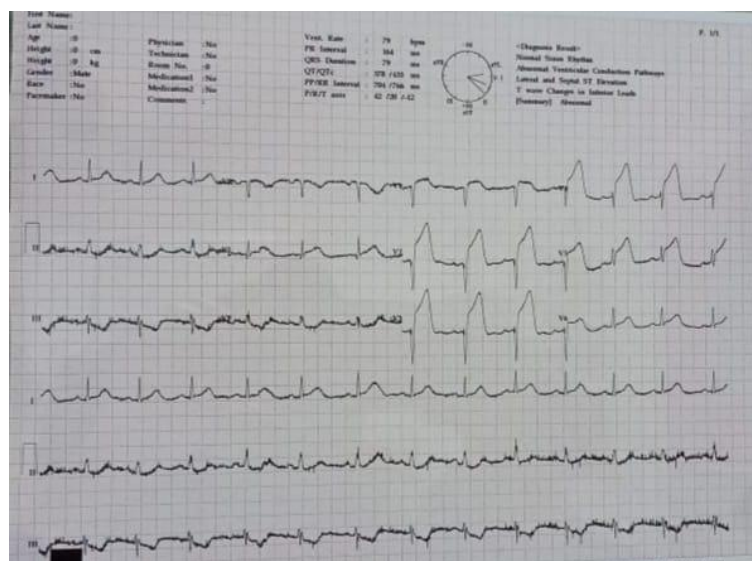
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Background: Coronavirus disease 2019 (COVID-19) first learned as a viral respiratory illness in December 2019, this infectious disease caused by Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-COV-2). However, some patients with COVID-19 may develop thrombotic and coagulation abnormalities.

Case Illustration: A 47-years-old man with a history of hypertension and dyslipidemia came to the emergency room suffering from a headache, cough and nausea for 4 days. Rapid Diagnostic Test (RDT) for COVID-19 resulted positive. One day in the isolation ward he had chest pain and diaphoresis. Electrocardiography (ECG) showed ST-segment elevation in lead V1-V6 with cardiac troponin I 562,5 pg/ml. He was diagnosed with ST-segment elevation ACS (STE-ACS) killip II. In the present case, the patient didn't undergo reperfusion therapy due to late onset of the symptoms. He was given aspirin 80mg, clopidogrel 75mg, nitroglycerin 30 mcg/minute, furosemide 2mg/hour and fondaparinux 2,5mg. Later, PCR test results were positive, D-dimer increased >24000µg/L, CRP 203,37 mg/L, leading to the diagnosis of COVID-19. Therefore, heparin 1000iu/hour and methylprednisolone 125 mg added to therapy. After 14 days of treatment, the patient was in a better condition on hospital discharge. According to Stefanini et. al, 85,7% patients can present ST-Elevation Myocardial Infarct (STEMI) symptoms as the first clinical manifestation of COVID-19, while the rest 14,3% have STEMI during hospitalization.

Conclusion: Since the research concerning the progression of COVID-19 or complication itself is still ongoing, the clinician should be alert of the patient's condition in any phases.

KEYWORDS: COVID-19, ACS, STEMI, chest pain.





CASE REPORT

Hemodynamic Deterioration In Right Atrial Thrombus and Pulmonary Embolism Associated Post-COVID-19 Induced Coagulopathy Accompanied by Acute Renocardiac Syndrome: The Strategy of Anticipating and Managing Coagulopathy

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Background: COVID-19 infection is associated with frequent coagulopathy and thrombotic events. The precise mechanism behind the pro-coagulability of COVID-19 is poorly understood. Dysregulation of clotting mechanisms may play a crucial role in this state.

Case Illustration: A 28-years-old male was presented to the ER with fever, cough, dyspnoea before admission. The patient had a history of COVID-19 infection with moderate symptoms one month ago and he refused to be hospitalized. From last RT-PCR has shown negative results, but the complaints of dyspnea haven't decreased and worsened in the last 4 days. Physical examination showed BP: 100/60 mmHg, HR: 118bpm, RR: 30tpm, T: 39°C SpO₂: 90%. Laboratory findings showed leukocytosis (14,8x10³/uL). ECG in E.R showed sinus tachycardia. CXR showed bilateral pulmonary infiltrates. On the first day of admission, patients experienced progressive dyspnea and hypotension, blood pressure dropped to 70/50 mmHg. He was transferred to the ICU and consulted with a cardiologist. The laboratory-result showed increased ureum (163 mg/dl), creatinine (5,44 mg/dl), D-dimer was highly increased (2112 ng/ml). ECG showed S1Q3S3 suspected pulmonary embolism. CT-Thorax showed a mass suspected tumor or thrombus in the right lung with bilateral consolidation, Echocardiography revealed RA thrombus with diameter about 15-22 mm with mild decrease of RV systolic function. The patient was diagnosed with shock condition in Post-COVID-19 syndrome accompanied with acute renocardiac syndrome, suspected pulmonary embolism, and hypercoagulopathy. After optimal therapy accompanied with 4 times of emergency hemodialysis and heparinization with close monitoring of aPTT, clinical condition also ureum and creatinine levels showed improvement. Heparinization continued for ten days. Evaluation of echocardiography and CT-Thorax showed reduction of RA thrombus size and mass dd thrombus in the right lung had disappeared. The patient was discharged after 16 days of treatment and received oral anticoagulants during outpatient care. After three months of follow-up, echocardiography showed remaining thrombus measuring 0.1 mm and D-dimer level returned to normal.

Conclusion: The role of therapeutic anticoagulation in COVID-19-associated prothrombotic episodes seemed to be reasonable treatment and have better efficacy in decreasing mortality in hospitalized patients due to COVID-19 with coagulopathy.

KEYWORD: COVID-19, Right Atrial Thrombus, Coagulopathy, Acute Renocardiac Syndrome.

CASE REPORT

Posterior Myocardial Infarction, A Challenge to Diagnose

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Background: Acute posterior myocardial infarction (MI) accounts for up to 20% of all MI, with the vast majority occurring along with inferior or lateral acute MI. A true posterior MI is considered more rare, with an incidence of approximately 3.3%. However, posterior MI diagnosis continues to be a challenge, with cases often missed and it can cause delays in treatment.

Case illustration: 63th year old male, referred to ED from primary health care, with typical chest pain since 2.5 hour before admission and smoking as the CAD risk factor. Blood pressure was 123/71 mmHg, pulse 63x/min and normal in general physical examination. 12-lead ECG showed ST depression in leads V2-V4 with an upright T wave and a tall R wave in leads V3-V4. In posterior leads, there was ST elevation 1 mm in leads V8-V9. Laboratory results of cardiac enzymes revealed CKMB 22 U/L (N 0-26 U/L) and Troponin I level 11.9 ng/L (<2 negative; >100 positive). After initial treatments as DAPT and statin, the patient underwent primary PCI. Coronary angiography revealed total occlusion in proximal LCX. 1 DES inserted towards proximal LCX, with angiography evaluation TIMI flow 3. The diagnosis of posterior MI is often under-recognized, largely due to the insensitivity of a standard 12-lead ECG. When there is marked ST depression confined to leads V1 through V4 and accompanied by prominent R waves V1-V3 and upright T waves, indicative of a true posterior injury. Confirmation by concomitant ST elevation recorded in leads V7-V9 ≥ 0.5 mm should be considered as posterior MI. However, in this case we found a tall R wave in leads V3-V4. Posterior lead ECG examination insisted and showed ST elevation, thus confirming for posterior MI. The patient was discharged after 4 days hospitalized with no further complaint.

Conclusion: Identification of posterior MI continues to be a challenge. The addition of posterior leads ECG (V7-V9) can establish the diagnosis. Once the posterior MI confirmed, initiation of reperfusion therapy should not be delayed.

KEYWORDS: *posterior myocardial infarction*

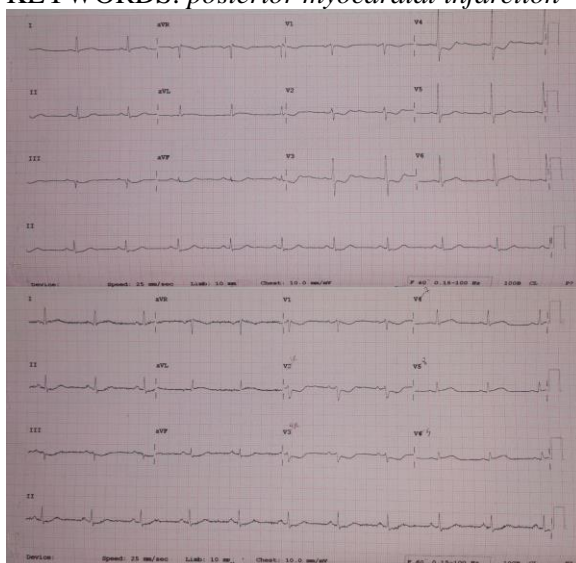


Figure 1. ECG on admission



CASE REPORT

Late Presenting Inferolateral STEMI With Multiple Organ Failure In Critical COVID-19 Patients, How Far Will We Go?

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Background: The COVID-19 pandemic has impacted the management of patients with acute coronary syndrome. The time of reperfusion strategy is important for STEMI patients. Regarding the management of STEMI patients, the exposure risk due to absence of a negative pressure catheterization laboratory may contribute to the relatively secondary role of PCI during COVID-19 pandemic. The decision to perform PCI in late presenting STEMI with critical COVID -19 and multiple comorbidities may be challenging.

Case Summary: A 58-year-old male transferred from another hospital to our emergency unit with breathlessness and severe chest pain started 4 days prior to hospital admission. He was diagnosed with late Inferolateral STEMI with critical COVID-19. ECG showed ST elevation in inferolateral with TAVB. Patients showed the presence of ongoing ischemia and haemodynamic instability with cardiogenic shock. Patient had a progressive respiratory distress due to SARS-CoV-2 infection which led to respiratory failure. The dysregulated immune system due to SARS-CoV-2 and cardiogenic shock induced multi-organ failure. We managed acute renal failure with CRRT. We decided to perform PCI strategy. The angiography revealed subtotal stenosis 90-99% in distal RCA and stenosis 90% in proximal OM1 LCx. We performed PCI in distal RCA with TIMI-3-flow result and inserted a temporary pacemaker. We gave antibiotics with Meropenem 500mg / 8 hours and Azithromycin 500 mg o.d for 10 days. As the hemodynamics improved, renal function ,respiratory function and blood glucose level synergized to improve.

Conclusion: PCI strategy to the patient with late onset STEMI and critical COVID-19 should be prioritized with adequate PPE to improve the haemodynamics. Crucial elements in decision making too late onset STEMI are the presence of haemodynamics or electrical instability. Multidisciplinary approach could save the patient.

KEYWORD: ACS, STEMI, COVID-19, PCI

CASE REPORT

Ventricular Septal Rupture Early Onset After Acute Myocardial Infarction: When Do We Refer?

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Background: A Ventricular Septal Rupture (VSR) is a lethal complication of ST-Elevation Myocardial Infarction (STEMI). We report a case of medically-treated VSR developed on the fourth day of hospitalization.

Case Presentation: A 67-year-old male was referred with the diagnosis of STEMI anterior. He had chest pain 2 days before admission and was referred to our hospital due to worsening chest pain and shortness of breath. S3 gallop was appreciated and rales were heard throughout both lungs. His ECG showed ST Segment Elevation in leads V2-V6 and Q wave in lead II, III, aVF. His troponin T level was elevated. Chest X-ray revealed pulmonary congestion on both lungs. He underwent Percutaneous Coronary Intervention on the third day because he needed to stabilize his condition after an acute heart failure, which showed multivessel stenosis with total occlusion in Left Anterior Descending (LAD) artery, successful stent implantation was performed in LAD. One the next day, he still had shortness of breath, a holosystolic murmur was heard from cardiac auscultation. Echocardiography examination showed a VSR with a left-to-right shunt in the apical region. Has referred to a tertiary hospital for further management. Successful treatment with PCI can reduce the risk for VSR, unfortunately in our case, VSR occurs the next day after PCI. Surgery became dilemmatic because he was already treated with stent implantation.

Conclusion: VSR can still occur despite aggressive treatment with stent implantation in patients with STEMI

KEYWORDS: *ST-Elevation Myocardial Infarction (STEMI), Ventricular Septal Rupture (VSR), Percutaneous Coronary Intervention (PCI)*

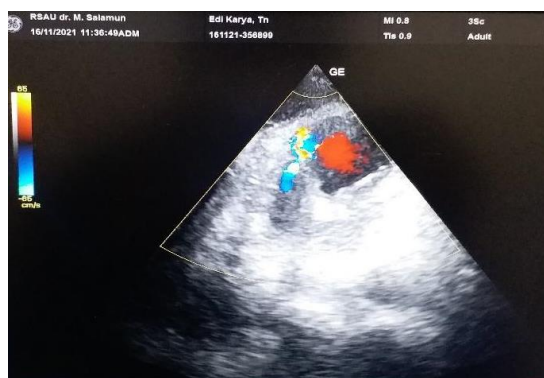


Figure 1. Echocardiography: a VSR with a left-to-right shunt in the apical region

CASE REPORT

Acute Cardiac Tamponade in ST-Elevation Myocardial Infarction: A Rare Case

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Background: Pericardial Effusion (PE) is one of the complications in patients with ST-Elevation Myocardial Infarction (STEMI). The majority of PE in STEMI does not cause hemodynamic instability. We present the case of early-onset PE that causes cardiac tamponade in a patient with STEMI.

Case Presentation: A 40-year-old male presents with acute typically chest pain 12 hours before admission. He was agitated and his hemodynamic was unstable despite two vasopressor supports. Fluid challenge test also failed to increase the blood pressure. His electrocardiogram showed ST-segment elevation in lead V2 through V4 and Q wave in lead III and aVF. He was diagnosed with STEMI anterior and cardiogenic shock. Primary Percutaneous Coronary Intervention (PCI) was performed in this patient. Coronary angiography showed total occlusion at proximal Left Anterior Descending (LAD) artery, and he underwent successful stent implantation in LAD. No complications such as coronary perforation or dissection were observed after PCI. After the procedure, his condition did not improve significantly, he became more agitated, his blood pressure and oxygen saturation suddenly dropped. Immediately echocardiography examination revealed massive pericardial effusion (>20 mm circumferential) with RA and RV collapse, and there was no free wall rupture observed. Urgent pericardiocentesis was performed with fluoroscopy guidance. About 600 ml of pericardial fluid was removed by pig-tail catheter, and his condition was dramatically improved. Follow-up echocardiography on the third day of hospitalization showed minimal pericardial effusion and his catheter was removed thereafter.

Conclusion: In STEMI, most of the patients presented with hemodynamic instability are due to ischemic and heart failure complications. Early PE in a patient with STEMI that causes cardiac tamponade and hemodynamic instability is a rare case and often missed.

KEYWORDS: *Cardiac Tamponade, Acute Myocardial Infarction, Pericardiocentesis*

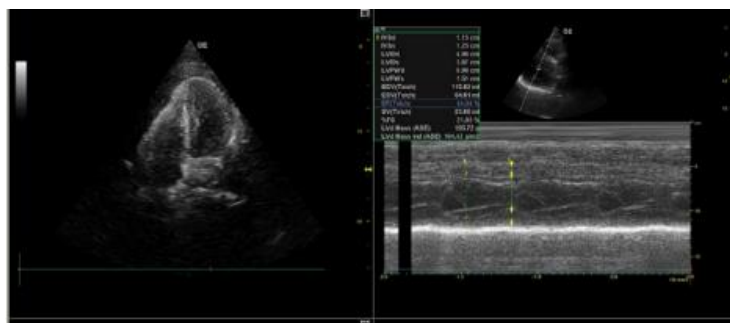


Figure 1. Echocardiography before Pericardiocentesis



CASE REPORT

Serial Case of Acute Pericarditis Patients Early Onset After Coronary Stenting in Salamun Air Force Hospital

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Introduction : Acute pericarditis is an inflammation of the pericardium, which classically presents with progressive, frequently severe, chest pain that is sharp and pleuritic can be triggered by a traumatic procedure. Acute pericarditis is a rare complication of percutaneous coronary intervention (PCI) with typically early-onset which occurred in this serial case of two patients.

Case One. A 58-year-old man was admitted to the hospital for an emergency PCI he was diagnosed with STEMI Anterior. Coronary Angiogram aimed directly to Left Anterior Descending Artery (LAD total occlusion) had successfully to distal lesion passed and a stent was successfully placed to distal LAD. One hour after the procedure, the pain is central, worse with inspiration or when lying down and improved by sitting up and followed by a high fever. ECG demonstrated widespread ST-Segment Elevation in anterior and lateral leads. Troponin I and CRP were highly elevated. Echocardiography showed an LVEF of 35,25%. He was given aspirin 4x500mg, colchicine 3x0,5mg, ticagrelor 2x90mg, diuretic IV, nitrate IV, ace inhibitor, beta-blocker, and high-intensity statin.

Case Two. A man 62-years-old with Stemi Inferior Killip II was immediately undergoing primary PCI. Coronary angiogram showed an attempt to revascularize Right Coronary Artery (Stenosis 80% at distal RCA). Echocardiography showed LVEF 45,42%. One hour after PCI, clinically presents high fever and chest pain worse when lying supine and relieved by leaning forward with unstable hemodynamic. In physical examination, pericardial friction rub and rales were found in both lungs. Inflammatory markers and cardiac enzymes were elevated. He was given aspirin 4x500mg, colchicine 3x0,5mg, clopidogrel 1x75mg, inotropic support, and high-intensity statin.

Conclusion: In our case, pericarditis chest pain, pericardial friction rub, diffuse ST-segment elevation were present which are very typical for pericarditis. The symptom improved dramatically with the treatment of nonsteroidal anti-inflammatory drug treatment and colchicine has been proven effective for the reduction of recurrences in pericarditis. Therefore, it is important for the clinician to differentiate acute myocardial infarction/acute stent thrombosis from this rare complication after PCI.

KEYWORDS: *Pericarditis; STEMI; Percutaneous Coronary Intervention*



CASE REPORT

Simultaneous Acute Myocardial Infarction and Acute Ischemic Stroke: What Should We Do

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Background: Both acute myocardial infarction (AMI) and acute ischemic stroke (AIS) are medical emergencies that require precise and timely treatment. Cardiocerebral infarction (CCI), when AMI and AIS occur simultaneously, is a rare medical emergency. The management of one condition can risk delaying treatment for the other and can cause disability or even death.

Case Illustration: A 57 year-old male was referred to Sardjito Hospital with a chief complaint of weakness and loss of consciousness 15 hours before admission. At the referring hospital, the patient was unconscious, hypotensive, and bradycardic, but later regained consciousness. He was aphasic and had right-sided weakness. The head CT scan showed large infarction in the left frontal, temporal, parietal, and occipital areas. The electrocardiogram (ECG) showed ST-segment elevation in the inferior leads and total atrioventricular (AV) block (TAVB). He was diagnosed with inferior ST-segment elevation myocardial infarction (STEMI) with TAVB that later converted into 1st degree AV block and extensive non-hemorrhagic stroke. He was initially managed conservatively in the intensive cardiac care unit (ICCU) due to concern of risk of hemorrhagic transformation. However, the next day, he went into cardiac arrest. After return of spontaneous circulation (ROSC), he underwent a percutaneous coronary intervention (PCI). The coronary angiogram revealed a total occlusion in the right coronary artery (RCA), and a drug-eluting stent (DES) was placed. Since no-reflow occurred, thrombosuction and intracoronary heparin was given. A transvenous temporary pacemaker (TPM) was inserted due to the high degree AV block. Despite this, the patient's condition worsened and he had recurrent cardiac arrests in the ICCU. He passed away 3 days later.

Conclusion: This case illustrates the dilemma and challenge in the management of CCI. Various case reports of CCI use different approaches in the management of CCI with various results. Until now, there is no clear guideline on the management of CCI. In the case of hemodynamic instability, primary PCI is preferred. The management of patients with CCI requires a collaboration among cardiologist, neurologist, interventional cardiologist, and interventional neurologist to decide the appropriate and comprehensive approach for patients with CCI.

KEYWORDS: *cardiocerebral infarction, acute myocardial infarction, acute ischemic stroke*



CASE REPORT

Unexpected TAVB with Cardiogenic Shock in Young Female Patient: Challenge in Rural Setting

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Background: Total atrioventricular (AV) block is a rather common arrhythmia in the elderly. Although high-grade AV conduction anomalies are uncommon in young or middle-aged people, they create issues when they are discovered.

Case Illustration: We report a case of initial diagnosis and treatment of Total AV Block in a rural hospital with limited settings and facilitation. A 19-year-old female with no known past medical history presented to the emergency department with three days of chest pain spread into torso while sleeping. She felt abdominal pain in one week and a non purulent productive cough. No fever presented. She had no recent travel or contact with covid patients. On physical examination the patient appeared fully conscious. Her blood pressure was 80/60 mmHg, with cold extremities and weak pulse. Auscultation of the heart found a slow heart rate, but it was normal, with no murmurs in the first and second heart sounds. Electrocardiogram was taken and unexpectedly revealed Total AV Block with cardiomegaly obviously seen on chest x-ray. Dopamin Pump 3 mcg/KgW.minutes was administered to stabilize the patient, then referred to a larger health center immediately.

Conclusion: Treatment of symptomatic TAVB with cardiogenic shock in rural hospital settings is challenging. Rapid assessment and early management is needed to stabilize the patient and immediately refer to a facilitated hospital to receive permanent pacemaker and further treatment.

KEYWORDS : *Total AV Block, Cardiogenic Shock, Young Female*



CASE REPORT

Mitral Prosthetic Valve Dysfunction and the Role of Echocardiography to Guide Clinical Decision: A Case report

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Background: More than 4 million prosthetic heart valve (PHV) replacements procedures have been done over the past 50 years. A comprehensive approach that integrates several parameters of valve physiology, morphology, and function assessed with 2D/3D transthoracic (TTE) and transoesophageal (TOE) echocardiography is a key to appropriately diagnose and quantitate PHV dysfunction, and in the end to determine the next clinical step.

Case Illustration: A female, 57 years old, came to the emergency department with shortness of breath from 2 weeks ago, and got worse in 2 days. Patient with history of MVR surgery with mechanical valve in 2019, and history of 1 month discontinuation of anticoagulant. From echocardiogram MVA VTI 0.3-0.45 cm², MVG was high, 11-14 mmHg. TEE confirmed the presence of thrombus. Patient underwent heparinization, pre discharge transthoracic echo showed improvement of prosthetic valve gradient, MVG 4-5 mmHg. MVA 1.5 cm². Patient planned for re-evaluation after 3 months of proper anticoagulation.

Conclusion: Echocardiogram plays an important role as a non-invasive diagnostic tool for prosthetic valve malfunction, whether as an initial examination or confirmatory examination with TEE. Quantification of the standardized degree of dysfunction of either obstruction or regurgitation plays an important role in determining the next clinical step

KEYWORDS: *prosthetic valves, echocardiography, mechanical, acute, prosthetic valve dysfunction, malfunction*



CASE REPORT

Myocardial Stunning After Primary PCI in Patient with Left Main Culprit Lesion : A Case Report

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Background: Myocardial stunning is a state of prolonged, post-ischemic ventricular dysfunction that happens after brief periods of ischemia, and is also associated with prolonged biochemical abnormalities that may take days to resolve following initial resolution of ischemia. Prolonged LV dysfunction will develop cardiogenic shock. Patients with culprit lesions of the left main coronary artery were associated with the worst mortality rate in 30 days.

Cases Illustration: A 54 year old man with an extensive Anterior STEMI 5 hour onset. Patients presented with heavy chest pain, with ECG showed ST elevation at I, AvL, V2-V6. Patient developed Atrial Fibrillation just before Primary PCI. Coronary angiography showed Left Main total occlusion in the ostial and was successfully implanted with 2 stent DES at LM-LAD with TIMI flow III. At CVCU patients developed shortness of breath and signs of hypotension and hypoperfusion which indicate cardiogenic shock. Bedside transthoracic echocardiography showed 26% LVEF with akinetic at segment anterior, lateral, septal at the level of basal until apex and LVH Concentric. This condition of myocardial stunning did not relieve during hospitalization. At CVCU the condition worsened, the patient get intubated, and suffered from persistent cardiogenic shock even with inotropes and vasopressor support. Unfortunately, the patient passed away on the seventh day of treatment.

Conclusion: Myocardial stunning could happen even after revascularization of myocardial infarction. However, prolonged myocardial stunning could lead to cardiogenic shock. In this case, patients with culprit lesions of the left main coronary artery have the highest mortality rate and the worst outcome.

KEYWORDS: *myocardial stunning, myocardial infarction, cardiogenic shock*

CASE REPORT

Reocclusion After Successful Fibrinolytic

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Background: Thrombotic occlusion of an epicardial coronary artery has been implicated as a potential mechanism involved in acute myocardial infarction since as early as 1910, and became generally accepted after the landmark report by De Wood and colleagues in the early 1980s. Large clinical trials confirmed the hypothesis that timely restoration of coronary patency had a notable impact on survival after ST elevation myocardial infarction: 20–30 lives saved per 1000 patients treated. But in the new era thrombolytics only is not clear enough.

Case Illustration: Patient came in with complaints of chest pain that had been felt for 6 hours before arrived at the Emergency Department. Chest pain like being hit by a heavy object on the left and radiating to the back, pain with a duration of > 20 minutes. Physical examination revealed that the patient was compos mentis with moderate general condition. Blood pressure: 140/72 mmHg, heart rate: 86 beats per minute, respiration: 30 times per minute, oxygen saturation: 88 % and temperature was not feverish. A chest examination found rales in 2/3 bilateral lung fields, Gallop (+). Electrocardiography (ECG) showed EKG : Sinus rhythm, qrs rate 90 bpm, ST elevation in V2-V6, I and AVL and ST depression in III, aVf. Laboratory examination found, Hb: 12,4 g/dL, WBC: $16,2 \times 10^3 / \text{mm}^3$, RGB: 199 gr/dL, Urea: 25 mg/dL, Creatinine: 0.5 mg/dL, Troponin T: 1.33. Patient was diagnosed with Akut STEMI anterior extensive onset 6 hours KILIP III and Acute pulmonary edema in ACS and treated with fibrinolytic streptokinase 1,5 millions Unit over 1 hour, with successful fibrinolytic . After 6 hours after Fibrinolytic patient felt heavy chest pain, cold sweat and dizziness (*Figure 1*) treated with Drip Dobutamine 5 mcg/I and Drip Norepinefrin 0.1 mcg/kgbb/I as Shock Cardiogenic, after that patient lost of consciousness and get Cardiopulmonary resuscitation.

Discussion: Regardless of the experience with fibrinolytic agents, the risk of reocclusion is very important. The concern is very high in assessing what fibrinolytic agent is used and the future routine PCI strategy to avoid reocclusion.

KEYWORD: *Fibrinolytic, Reocclusion, routine PCI*

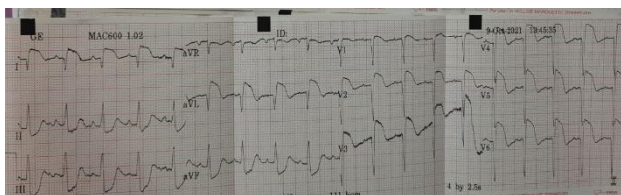


Figure 1. ECG 6 Hours after successful fibrinolytic



CASE REPORT

Life-threatening Severe Hyperkalemia Causing Atrial Fibrillation with Slow Ventricular Response Leading to Acute Pulmonary Oedema

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Background: Hyperkalemia is caused by excessive potassium intake, abnormal potassium excretion, or transcellular shifts which is a potentially life-threatening electrolyte abnormality that can result in fatal cardiac arrhythmias which are conditioned by the presence of signs such as hypotension, shock, acute pulmonary oedema or cardiac arrest.

Case presentation: In this case, a 63-year-old woman with a history of coronary artery bypass grafting (CABG) presented to the emergency department with the complaint of severe shortness of breath for 8 hours before admission. She routinely consumed clopidogrel, atorvastatin, candesartan, spironolactone and bisoprolol. Her blood pressure was 135/80 mmHg, her heart rate was 40 beats per minute, her respiratory rate was 36 times per minute and her oxygen saturation was 88%. There were rales in all of both lungs. The ECG showed atrial fibrillation with ventricular rate 40 beats per minute, intraventricular conduction block and QS pattern in V1-V6. Laboratory tests showed potassium level was 9.5 mmol/L and creatinine was 2.1 mg/dL. We assessed the patient with acute pulmonary oedema, atrial fibrillation with slow ventricular response, and hyperkalemia. Then she was managed with intravenous drips Furosemide 20 mg/hour and intravenous drip Dopamine 5 mcg/KgBW/minute, then hyperkalemia was corrected 3 times using intravenous bolus Insulin 8 iu in 50 ml Dextrose 40% and intravenous Calcium Gluconate 1 gram until potassium level was 4.9 mmol/L which improved patient's complaint and ECG finding showed sinus rhythm with QRS rate 74 beats per minute with poor R waves progression in V1-V6. In severe hyperkalemia, life-threatening cardiac arrhythmias may occur such as bradyarrhythmia due to sinus arrest, total AV block or atrial fibrillation with slow ventricular response. Successful treatment of acute hyperkalemia involves protecting the heart from fatal arrhythmias with the administration of calcium, shifting potassium into the cells with intravenous short-acting insulin, and enhancing the elimination of potassium from the body.

Conclusion: Hyperkalemia is a frequently encountered medical emergency that can lead to fatal cardiac arrhythmias. The prompt treatment of the underlying problem that was hyperkalemia correction is required to prevent the life-threatening event.

KEYWORDS: Hyperkalemia, Atrial Fibrillation, Acute Pulmonary Oedema

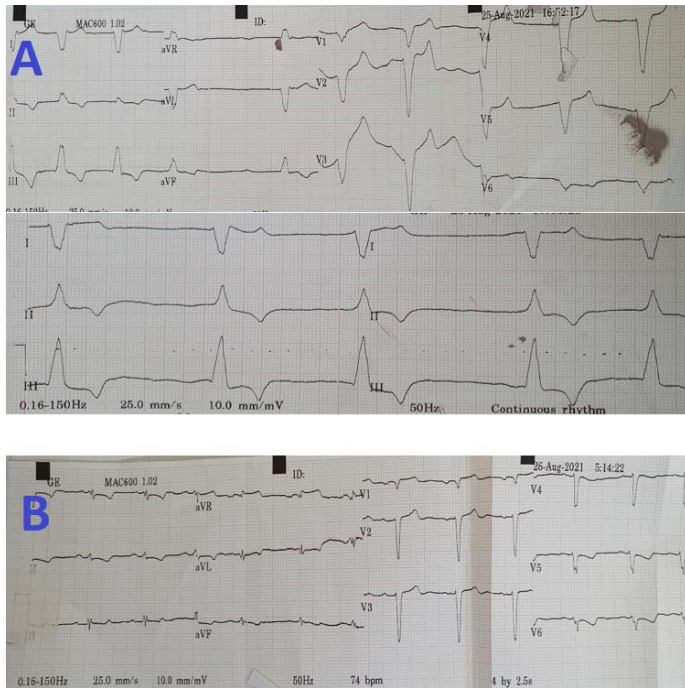


Figure 1. ECG showed atrial fibrillation with slow ventricular response, intraventricular conduction block and QS pattern in V1-V6. (B) ECG after hyperkalemia correction showed sinus rhythm with heart rate 74 beats per minute with poor R waves progression in V1-V6.

CASE REPORT

Persistent AV Node Dysfunction After Primary PCI In Inferior STEMI Patient : Case Report

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Background: Sinoatrial node atrioventricular (AV) conduction disorders often complicated inferior myocardial infarction immediately to a few days following the initial ischemic event, High Degree Atrioventricular Block (HDAVB) after an inferior myocardial infarction was associated with increased short term mortality. But until now, Clinicians often face a dilemma with regards to the optimal timing of permanent pacemaker (PPM) implantation for HDAVB following an inferior myocardial infarction

Case Summary: A 59-year-old male patient presented with a complaint of chest discomfort that had been felt for 3 days before admission to the hospital. The patient had a history of hypertension and diabetes mellitus since 3 years ago and uncontrolled. ECG showed High degree AV Block with QRS rate 29 BPM, Q Path II I, AVF and ST elevation II, III, AVF (Figure 1 Panel A). Coroangiography showed RCA with total occlusion then installed 2 des, LAD: Critical Stenosis 99% proximal, LCx: Stenosis 70% proximal (Figure 1 Panel B), Temporary Pacemaker (TPM) was inserted with HR 70 BPM. After 5 days, on the ECG it was found that it was still HDAVB and it was planned to be an earlier PPM Implantation, but the patient later had pulseless VT and died. Prior reports demonstrate it may take up to 2 weeks for HDAVB to recover, The patients who had successful PCI entirely returned to the sinus rhythm within 34 hours after PCI, while in the patients with failed PCI, this time was 180 hours. patients with HDAVB, the median interval from admission to permanent pacemaker implantation was 3 days (Interquartile ratio: 2-5 days), more than one-third (34.9%) of permanent pacemakers were implanted within 72 hours after admission. HDAVB is also strongly associated with cardiogenic shock, either as a cause or consequence, and the association with higher mortality during the acute or subacute phase of inferior myocardial infarction has been well-documented.

Conclusion: Earlier PPM implantation should be considered in non-recovered Sinus Rhythm after PCI in inferior STEMI, the exact timing for this warrant an CRT.

KEYWORD: High Degree Atrioventricular Block, ST Elevation, Myocardial Infarction Inferior, Percutaneous Coronary Intervention.

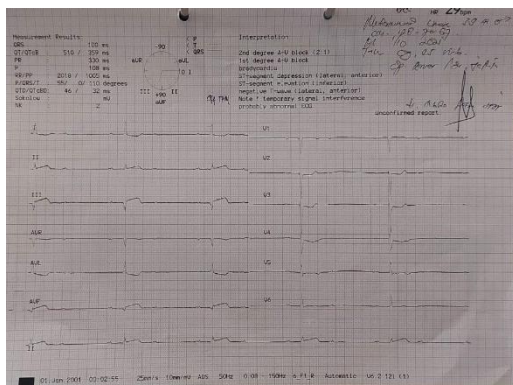


Figure 1. ECG day 2 hospitalization (TPM off), after Primary PCI, showed High degree AV Block with QRS rate 29 BPM, Q Path II I, AVF and ST elevation II, III, AVF.



CASE REPORT

Multiple Factors Inducing Recurrent Torsades de Pointes in Heart Failure Patient: a Case Report

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Background: Torsades de pointes (TdP) is one of hazardous arrhythmias. TdP is correspondent with prolonged QT interval. One of the common causes of TdP is electrolyte imbalance. Here we present a case of recurrent hypokalemia refractor caused by diuretic inducing recurrent TdP.

Case illustration: A 44-year-old woman presented to the emergency room of Kediri General Hospital with dyspnea, and palpitation for two days. Previously she experienced atypical chest pain. She had normal blood pressure, and positive signs of peripheral edema. Electrocardiogram showed sinus tachycardia, left atrial enlargement, Q wave in anterior lead and poor R wave progression, prolonged QTc interval (550 msec). Laboratorium results were leukocytosis, elevated liver enzyme, hipoalbumin, hyperglycemia and bacteriuria, normal electrolyte level. Chest X-Ray was cardiomegaly and pulmonary congestion. She was diagnosed with acute decompensated heart failure, urosepsis, suspect of congestive liver, coronary artery disease and type 2 diabetes mellitus. Echocardiography showed eccentric hypertrophy with dilated chamber and global hypokinetic. Patients were treated with beta-lactam antibiotic meropenem, diuretic, ACE inhibitor, mineralocorticoid receptor antagonist, antiplatelet, anticoagulant, statin, vasodilator, insulin, and she was hospitalized in ICVCU. After 2 days of treatment she had an episode of R on T phenomenon and TdP. Evaluation of electrolyte serum showed hypokalemia (from 4.0 mmol/L to 2.6 mmol/L), hipocalcemia (5.6 mg/dL) and hyponatremia (126 mmol/L). She was treated with electrical cardioversion when hemodynamically unstable, potassium infusion, calcium gluconas, amiodaron, and magnesium. During treatment she experiences recurrent torsades de pointes with the longest duration being approximately seven seconds. Laboratory result was a hypokalemia refractor. Lidocaine was added to the treatment. After 14 days, the clinical condition improved, and ECG resolved to normal. Acquired prolonged QT interval in this patient may be induced by electrolyte abnormality (hypokalemia, hypocalcemia, hyponatremia), use of diuretics, use of antibiotic, female gender, and ischemic heart disease.

Conclusion: healthcare practitioners should be aware and identify risk factors associated with prolonged QTc interval, since prolonged QTc interval can be a lethal arrhythmia such as TdP.

KEYWORDS: *Torsades de pointes, heart failure, hypokalemia, arrhythmia*

CASE REPORT

Diagnostic Delay of Acute Pulmonary Embolism in COVID-19 Era

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Background: Dyspnea, fever and cough may occur in both pneumonia and acute pulmonary embolism (APE). These similarities may lead to a delay in the diagnosis of APE in COVID-19 era. We report an APE case which is initially diagnosed as suspected pneumonia COVID-19.

Case Illustration: A-76-year-old male came to the emergency room with dyspnea, fever and cough. His vital sign was BP: 76/60 mmHg, HR: 76 bpm, RR: 28 rpm, T: 38.1⁰C, SpO₂: 100% on NRM 15 lpm. He was given fluid bolus followed by norepinephrine maintenance. Initial laboratory reports showed reactive rapid antigen for SARS-Cov-2, leukocytosis at 18000/mm³ with NLR 15. Chest x-ray showed cardiomegaly. We suspected COVID-19 pulmonary infection in chronic heart failure patients considering the clinical presentation and the actual pandemic condition. When he was transferred to an isolation ward, dyspnea got worse. We found basal bilateral crackles and recurrence of hypotensive. He was diagnosed with acute pulmonary edema and treated with furosemide continuously along with norepinephrine. There was reduction of dyspnea and stable vital signs until the 2nd day. Symptoms worsening on the 3rd day with acute respiratory distress, right lower chest pain, hemoptysis, and bilateral lower extremity pitting edema. Reverse transcription polymerase chain reactions for SARS-Cov-2 were negative. Follow-up chest x-ray showed Hampton hump, D-Dimer: 1108.4 ng/mL, and Well's criteria for APE: 7 (high risk group). Patient transferred to cardiac intensive care unit for noninvasive ventilation and bedside transthoracic echocardiography (TTE). Transthoracic echocardiography revealed a thrombus (21.4mmx23.8mm) in the main pulmonary artery. Patient diagnosed with APE, PESI class V. Rescue fibrinolytic was performed with heparin. We were unable to perform invasive airway management due to not resuscitating order and the patient died on day 4.

Conclusions: Delayed diagnosis of APE may lead to worse prognosis. We must be aware of the possibility of APE in COVID-19 era, especially when there are uncertain etiology of hemodynamic instability and dyspnea that worsen even with optimal treatment.

KEYWORDS: *Diagnostic delay, pulmonary embolism, COVID-19.*

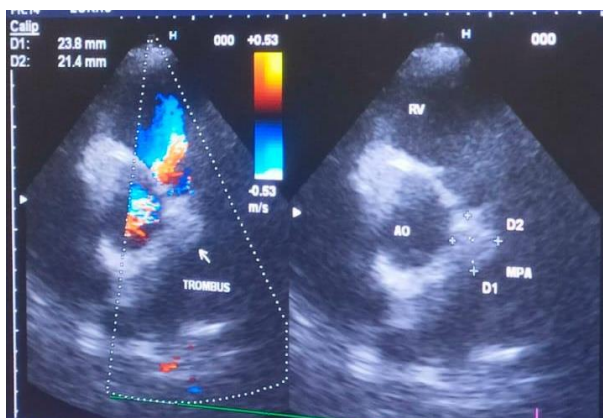


Figure 1. Transthoracic Echocardiography Result

CASE REPORT

Conservative Strategy In Converting Total Atrioventricular Block In Rural Area Hospital: A Case Report

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Background: Pacemaker implantation is mainly indicated for all symptomatic patients with total atrioventricular block. The management of this case remains challenging in the rural hospital that does not provide pacemaker devices.

Case Summary: A 66-year-old man was admitted to the emergency department after presenting with epigastric pain, accompanied by diaphoresis worsened from 1 day before admission. He had a history of smoking, hypertension and dyslipidemia with no routine medication. His vital signs showed a heart rate of 48 beats per second. Electrocardiogram (ECG) demonstrated total atrioventricular block with junctional escape rhythm, with Q and inverted T wave in inferior leads. Physical examination, chest x-ray and serum electrolytes were normal findings. Medications given during hospitalization were Sulfas Atropine, Salbutamol, Aspirin, Clopidogrel, Trimetazidine, and Simvastatin. On the fourth day of admission, the rhythm was converted into sinus rhythm. Some possible causes of total atrioventricular block finding are electrolyte imbalance, infection, coronary artery disease, congenital heart disease, neoplasm and autoimmune disorder.

Conclusion: In certain cases, the total atrioventricular block can be converted following successful treatment of its etiology. From the chief complaint, past medical history, ECG findings, and rhythm conversion after antiplatelet medication, we consider that coronary artery disease as the most possible cause of atrioventricular block. However, further investigations are still needed to confirm the specific etiology of this case.

KEYWORD: *total atrioventricular block, rural area, management, electrocardiography*

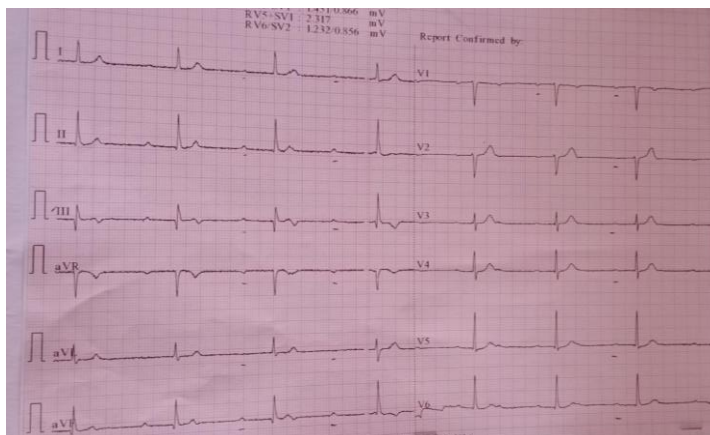


Figure 1. ECG on admission



CASE REPORT

Clinical and Angiographic Profile of Asymptomatic COVID-19 in Young Adult Male Presenting with Acute ST - Elevation Myocardial Infarction: A Case Report.

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Background: In patients with STEMI and concomitant COVID-19 infection, there is strong evidence of more severe clinical course of cardiac manifestations and subsequent myocardial remodeling, despite of young age and absence of co-morbidities. STEMI patients with even asymptomatic COVID-19 infection may be presented with significantly higher rates of acute heart failure.

Case Illustration. We report a 24-year-old male presented antero extensive myocardial infarction (MI). He complained of severe chest pain and cold sweating. He was a smoker without any other traditional risk factor. He has no clear family history with cardiovascular disease. We performed coronary angiography, found a total occlusion in the left Main (LM) artery, and placed a stent in LM and LAD. Echocardiogram showed a decrease of left ventricular function with 38% ejection fraction. Lipid profile was normal. After the angioplasty, PCR performed on a nasopharyngeal swab was performed and showed a positive COVID-19 result. This fact provides further evidence that multiple heterogeneous mechanisms may be associated with a more severe course of STEMI, depending on thrombus grade, inflammatory activity, respiratory disorders, and secondary myocardial injury even in asymptomatic persons. Currently we are also testing markers of coagulation status in this patient and still waiting for the results.

Conclusion: STEMI patients with COVID-19 infection may be presented with significantly higher rates of acute heart failure despite of young age and absence of clinical symptoms of infection and comorbidities. In critical cardiovascular states of STEMI and COVID-19 resulting in acute heart failure and haemodynamic disturbance, the urgent primary percutaneous coronary intervention with further complex treatment is an effective strategy.

KEYWORDS : *ST-elevation myocardial infarction; Acute heart failure; Young patients; Asymptomatic COVID-19*



CASE REPORT

Conservative Approach to Maintain Hemodynamic Stability in Patient With Acute Myocardial Infarction Complicated by Ventricular Septal Rupture: A Case Report

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Background: Ventricular Septal Rupture (VSR) complicating acute myocardial infarction is uncommon with a reported incidence in the pre thrombolytic era of 1% to 2%, and less than 1% in this modern era. Most cases show unstable hemodynamics dan high mortality rate.

Case Illustration: A 69-year-old man complained of chest pain radiating to the back and shortness of breath two days before admission. Physical examination found rhonchi in 2/3 bilateral lung areas accompanied with a decrease of oxygen saturation. ECG showed ST elevation at the extensive anterior leads. Laboratory examination found an increased troponin, blood glucose, and HbA1c. The patient was diagnosed with recent extensive anterior ST-Elevation Myocardial Infarction (STEMI) accompanied with Acute Lung Oedema and Uncontrolled DM Type II. On the 3rd day of treatment, the patient's congestive condition improved but the hemodynamic became unstable and found a new onset of murmur on the 3rd space of the left-parasternal intercostal. Echocardiography examination found a rupture in the apical interventricular septal with a left-to-right shunt that indicated a Ventricular Septal Rupture as a mechanical complication of myocardial infarction. The patient received conservative therapy and close monitoring in the ICU. This is a challenging case because the hospital facilities do not yet have the capacity for cardiac surgical intervention. For this case, we focused on conservative management to maintain the stability of hemodynamic and patient survival. Three weeks of treatment the patient showed improvement with stable hemodynamics but limited mobilization with only light activities. The patient was discharged after one month of treatment and was planned to be referred for surgical intervention.

Conclusion: Ventricular septal rupture is a rare mechanical complication following myocardial infarction. Every clinician must be alert of this complication due to its high mortality, especially in those who do not receive reperfusion therapy. Conservative management to stabilize the patient hemodynamic followed by surgical repair of VSR may give a better outcome.

KEYWORDS: *Acute Myocardial Infarction, Ventricular Septal Rupture, Hemodynamic Stability, Conservative Management.*



CASE REPORT

Non-cardiac and Cardiac Complications of Symptomatic Severe Aortic Stenosis: A- Study form Case Series in Central Borneo, Indonesia

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Background: The AS had a broad spectrum from subclinical to severe clinical symptoms. Unfortunately, its complications might become a disaster. However, there was little information about complications from systematic severe aortic stenosis.

Case Presentation: This case series consisted of two complications of severe aortic stenosis. A 56-year-old man who had a history of symptomatic severe aortic stenosis presented with chest pain and new onset of abrupt left hemiparesis. The brain ct-scan showed an ischemic area. Another case, a 58-year-old man with chest pain and a pounding sensation. After full examination, we found that the patient suffered total atrioventricular block and severe aortic stenosis. One of the non-cardiac adverse outcomes from severe aortic stenosis was stroke. The aortic stenosis raises risk of stroke presumably via low stroke volume and thrombus formation in the left atrial appendages region. Meanwhile, cardiac complication from the aortic stenosis was conduction system disorder due to alteration of volume-pressure in cardiac chamber and proximity location between aortic valve and His bundle.

Conclusion: Severe conditions of AS could complicate any other organs. Early diagnosis and recognition of its adverse effect were the key for suppress its morbidity and mortality.

KEYWORDS: *aortic stenosis, complication, valve heart disease, stroke, arrhythmias*



CASE REPORT

Autolysis in Anterior Extensive ST-Elevation Myocardial Infarction Patient: A Case Report

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Background: Anterior Extensive ST-Elevation Myocardial Infarction (STEMI) occurred when an anterior myocardial infarction extends to the septal and lateral regions. It usually results from occlusion of the proximal left anterior descending artery (LAD) or even in the left main coronary artery.

Case presentation: A 64-year-old male presented to the emergency department with pain in the left of the chest that arises and subside for 2 days. He also complained of shortness of breath for 2 days. He has a history of hypertension. There were no abnormal findings from the physical examination. From vital sign findings, blood pressure was 150/90 mmHg, heart rate was 80 times a minute, respiratory rate was 22 bpm, temperature was 36.7°C, and respiratory rate was 98% room air. Electrocardiography (ECG) demonstrated massive ST-segment elevation with tombstone morphology in precordial leads (V1-V6) that indicated anterior extensive STEMI. The patient was given loading dual antiplatelet therapy (DAPT), nitrate, and statin. Afterward, he was planning to receive fibrinolytic in the intensive cardiac care unit (ICCU). However, before performing fibrinolytic, the patient's ECG demonstrated autolysis. The autolysis was presumed due to administration of DAPT, nitrate, and statin before performing thrombolytic.

Conclusion: Dual antiplatelet therapy, nitrate, and statin were the standard treatments for STEMI management as both ESC and ACC/AHA guideline recommendations. Their role has an important impact on clinical outcomes of coronary disease, particularly in the emergency setting before thrombolytic or primary percutaneous coronary intervention (PCI). It was relevant to be implemented. Even, in our case, their role results in autolysis before we perform thrombolytic.

KEYWORDS: *Anterior Extensive STEMI, Autolysis*



CASE REPORT

Unstable Hemodynamic Supraventricular Tachycardia Induced by Hypothyroidism on Newly Diagnosed of Peripartum Cardiomyopathy in Young Woman: A Rare Case

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Background: The incidence of cardiac arrhythmias is higher in pregnancy with structural heart disease. Supraventricular Tachycardia (SVT) has been associated with Peripartum Cardiomyopathy (PPCM) but induced by hypothyroidism is a rare case, its incidence and outcomes have not been well documented. life-threatening arrhythmias contribute significantly to sudden cardiac death in this population. Electrical cardioversion is a reasonable option at all stages of pregnancy when arrhythmias are associated with hemodynamic instability.

Case Illustration: A 27-year-old woman presented with dyspnea, palpitations, and weakness one day previously. She also complained of swollen extremities and abdominal distension after primigravida postpartum two months ago. There was no comorbid of preeclampsia, primary hypertension, obesity, high risk gravidity, and cardiac disease during pregnancy and maternity. A physical examination found a decrease of systolic blood pressure of 70 over palpation with heart rate 170 bpm regular rhythm, respiration 34 bpm and anasarca edema. Electrocardiography (ECG) showed SVT with aberrancy. Cardioversion of 100-150 joule was performed and converted to sinus rhythm. Echocardiography showed left ventricular (LV) dysfunction with decreased ejection fraction (EF) 27% and LV enlargement. This patient was diagnosed with PPCM accompanied with SVT and Cardiogenic Shock. Then the patient received optimal treatment of PPCM and close monitoring in the ICU while we evaluated the risk factors for PPCM. On the third day of treatment, hypothyroidism was diagnosed with increased of TSH level 3 times than normal (12,78 uIU/mL) and decreased of FT4 level 2 times than normal (1,07 mmol/L), Levothyroxine was started at 50mcg/day. After two weeks of treatment, the patient's condition improved; there was no further recurrence of SVT, manifestation of congestion was resolved, thyroid marker level already within normal limits and patient was discharged. After a 5 months follow-up of optimal therapy, the patient showed improvement in heart condition with improvement of LV function to normal.

Conclusion: Peripartum cardiomyopathy may have unpredictable and life-threatening clinical courses. The management of PPCM should focus on controlling symptoms and preventing complications during pregnancy and relies on a multidisciplinary approach focused on hemodynamic stability of mother and fetus.

KEYWORD: *Peripartum Cardiomyopathy, Supraventricular Tachycardia, Hypothyroidism*



CASE REPORT

Multidisciplinary Approach for Management of Pulmonary Hypertension in Young Woman with Failure of Ventilator Weaning Complicated by Postpartum Hemorrhage and Septic Condition: A Difficult Case Report

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Background: Pulmonary hypertension is common in critical care settings and in presence of right ventricular failure is challenging to manage. Pulmonary hypertension in pregnant patients carries a high mortality rate between 30–56%. A multidisciplinary team approach is crucial to achieve successful outcomes in these difficult cases.

Case Illustration: A 39-year-old pregnant woman with postpartum hemorrhage (PPH) and septic condition has been consulted to Cardiology Department. Her condition deteriorated; after 3 days of treatment, oxygen saturation remains below 80%, and the patient cannot be weaned off the ventilator. Chest radiography revealed bronchitis and cardiomegaly, leukocyte count $47 \times 10^3/\mu\text{l}$. Echocardiogram revealed right atrial and ventricle dilatation with moderate to severe tricuspid valve regurgitation. We diagnosed this patient with PPH with septic conditions accompanied with newly diagnosed moderate pulmonary hypertension. Administration of cardiac inotropes and diuretics gave an improvement for RV failure and fluid retention also broad-spectrum antibiotics to treat sepsis, but the oxygen saturation still did not give great improvement. Increases of oxygen saturation occurred after the phosphodiesterase type 5 inhibitors after 5 days of administration for maximal dose. Pregnancy and delivery are associated by increase of cardiac output (CO), oxygen consumption, venous return and activating vasovagal reflex while systemic vascular resistance (SVR) is decreased. This condition will trigger hypoxia, RV failure and fluid overload, ending up in respiratory failure. Treatment of this patient was managed and closely monitored by a multidisciplinary team in the Intensive Care Unit (ICU). After two weeks of treatment and monitoring, the patient clinically improved, SpO₂ >94%, enabling the patient to be extubated and later discharged.

Conclusion: PH in pregnancy is associated with high risk of mortality and morbidity for maternal and fetal death. Multidisciplinary team approach is mandatory to monitor patient's condition and improve outcome.

KEYWORD: *Pulmonary Hypertension, Pregnancy, Postpartum Hemorrhage, Sepsis*



CASE REPORT

Out of the Blue New Onset Peripartum Cardiomyopathy in Young Female Patient : Case Report

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Background: Peripartum cardiomyopathy (PPCM) is defined as dilated cardiomyopathy that manifests as systolic cardiac heart failure in the last month of pregnancy or within five months following delivery.

Case Illustration: A 20-year-old young female presented to our emergency room with three days of dyspnea and chest pain radiating into her right shoulder. She had given birth to her second child by cesarean surgery two months ago and was in relatively good health until her last month of pregnancy, no hypertension was reported. She had no recent travel or contact with covid-19 patients. On physical examination, she was found to be dyspneic with saturation on room air was 98%, with blood pressure of 110/70 mm Hg, heart rate of 110 beats per minute. Auscultation of heart revealed rapid and regular heart rate with normal first and second heart sounds having no murmurs. A transthoracic echocardiogram done at admission showed an LV ejection fraction of 30%, dilated all chambers, and LV thrombus. The patient was being admitted for new-onset PPCM, then given furosemide intravenously and heparin subcutaneously. The dyspnea significantly decreased with diuresis and the patient was discharged from the hospital six days later, with instructions to take lisinopril, furosemid, bisoprolol, warfarin, and spironolactone.

Conclusion: We presented a case report of a patient with PPCM and congestive heart failure. It is necessary for physicians to be concerned with PPCM and to consider it when assessing dyspneic in female patients with a recent pregnancy history.

KEYWORDS : *PPCM, Heart Failure, Young Woman*



CASE REPORT

Scavenger Hunt in Determining Pediatric Dilated Cardiomyopathy with Heart Failure beyond Pandemic : Case Report

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Background: Dilated cardiomyopathy is responsible for one of every three instances of congestive heart failure. And now diagnosing heart failure patients facing a big challenge in high surge covid19 cases during pandemic due to it's similar symptoms.

Case Illustration: A 16-year-old young girl brought to the emergency room due to one week progressive shortness of breath and aggravated for two days, with fatigue and dry cough. There was nothing in her medical history nor her family history, and no contact with covid-19 patients. The oxygen saturation was 70% room air with a respiratory rate of 30 breaths per minute. The auscultation revealed normal heart sounds with rales heard bilaterally in the basal lung field. Chest X Ray showed cardiomegaly and bilateral pneumonia differential diagnosed with acute lung oedema. A RT-PCR swab was tested negative. Transthoracic echocardiogram demonstrated dilated cardiomyopathy in all chambers with low ejection fraction (37%) and global hypokinetic wall motion. The patient was treated effectively given intravenous nitroglycerin and furosemide also successfully being weaned from non-invasive ventilation in ICCU for four days then being discharged two days later without any symptoms remaining.

Conclusion: The COVID-19 pandemic has been challenging as patients admitted with progressive symptoms, either caused by COVID-19 or other underlying disease. The principal of dilated cardiomyopathy diagnosis and treatment in children is identifying and eliminating the precipitation factor, etiology, and managing systemic or pulmonary congestion. The prognosis varied based on etiology, severity, and reversibility of heart failure.

KEYWORDS : *Dilated Cardiomyopathy, Heart Failure, Children*



CASE REPORT

Early Continuous Renal Replacement Therapy In Cardiogenic Shock

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Background: Acute myocardial infarction with cardiogenic shock (AMI-CS) is associated with high mortality and morbidity despite advancements in cardiovascular care. AMI-CS is associated with multiorgan failure of non-cardiac organ systems. Acute kidney injury (AKI) is frequently seen in patients with AMI-CS and is associated with worse mortality and outcomes. Over the years, renal replacement therapy has emerged as the mainstay of the treatment for AKI.

Case Illustration: A 46 year old male complained of typical chest pain, shortness of breath and he came to the district hospital. He was diagnosed with anterior STEMI, coroangiography was performed and revealed coronary artery disease three vessel disease (CAD3VD) POBA in LAD. During hospitalization in the intensive care unit, he had hypotension and additional support vasopressor and inotropes were given. Urine evaluation showed urine output was decreased and creatinine value was increased (3.9 mg/dl). During monitoring, his conditions worsened and was diagnosed with cardiogenic shock, anterior STEMI, acute kidney injury, diabetes mellitus with problem oliguria then he was referred to Sardjito Hospital for further treatment. He was hospitalized in an intensive cardiac care unit (ICCU) and due to worsening of kidney function, early continuous renal replacement therapy (CRRT) mode CCVHDF was performed within 24 hour. Two days after, his kidney function was improved, marked by increasing urine output and creatinine value decreased. Four days later his condition was improved and he was transferred to the cardiology ward.

Conclusion: In patients with acute kidney injury and cardiogenic shock, early initiation of continuous renal replacement therapeutic strategy may beneficial and improve patients' outcomes

KEYWORDS: *Cardiogenic shock, acute kidney injury, continuous renal replacement therapy*



CASE REPORT

Perioperative Myocardial Infarction (PMI) in Non Cardiac Surgery: Bleeding vs Ischaemic Risk, How to Deal with it?

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Backgrounds: More than 230 million major surgeries are performed annually worldwide. The 30-day mortality associated with moderate- to high-risk noncardiac surgery in recent large cohorts and population-based studies exceeds 2% and surpasses 5% in patients at high cardiac risk. Patients without coronary artery disease (CAD) had a PMI rate of 0.8% and mortality rate of 1%. Cardiac complications constitute the most common cause of postoperative morbidity and mortality, having considerable impact on the length and cost of hospitalization.

Case Illustration: a-46 years old man referred to our Emergency Department (ED) complained chest pain with diaphoresis immediately after pyelolithotomy of his right nephrolithiasis. He denied any cardiac complaints before. His CAD risk factor was uncontrolled diabetes mellitus. On ED his pressure was 96/67, pulse 81 bpm, respiratory rate 18, oxygen saturation 100% with 3 lpm cannula, gross hematuria and attached renal drain volume 150 cc. There were normal cardiac and pulmonary examinations, ECG showed ST elevation in V2-5 and I, aVL. Chest X Ray revealed Cardiomegaly LV and cephalization. The laboratory test revealed anemia (11.2 mg/dL) elevating blood glucose (253 mg/dL) and cardiac enzyme level (troponin >50 ug/L, CKMB 216 U/L). Due to crusade score was 50 and active bleeding, he was only given a maintenance dose of unfractionated heparin. double anti platelet with acetylsalicylic acid, clopidogrel, and stabilized in ICCU for further evaluation.

Conclusion : PMI is often silent and its ECG changes are frequently transient. Coronary intervention is rarely indicated as the first line of treatment, and antithrombotic therapy may exacerbate bleeding. Bleeding in ACS has consequences, including cessation and reversal of anti-thrombotic therapy, hypotension, and blood transfusion, benefit and risk of bleeding using antithrombotic therapy in PMI should be carefully considered.

KEYWORDS : *Perioperative Myocardial Infarction, STEMI, Bleeding*



Successful Thrombolysis of Acute Mechanical Valve Thrombosis

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Background : Acute valve thrombosis of mechanical prosthetic heart valves is one of the major complications of valve replacement. The diagnosis remains challenging mainly due to a general lack of awareness of its existence. We report a case of successful thrombolysis in acute mitral mechanical valve thrombosis.

Case illustration : A 53-year-old man came to the emergency room with dyspnea in the last 4 hours before admission. Vital signs presenting hypotension, tachycardia, and tachypnea. Physical examination shows dimmed metallic sound and elevated jugular venous pressure. He had a history of definite infective endocarditis and chronic heart failure with underlying moderate mitral regurgitation and received a mechanical valve with a 27 mm Medtronic Mechanical Heart Valve and evacuation of myxoma and vegetation of the left heart in May 2020. After the surgery, the patient also had an incident of non-hemorrhagic stroke. The patient routinely consumes warfarin, and has a serial check of INR (international normalized ratio) with results always under 3. The patient was observed in intermediate cardiac care, and had a worsening condition. The blood pressure dropped and my heart rate increased. Urine output is about 0.2cc/kg/hours. Norepinephrine and dobutamine were used to increase the blood pressure. Echocardiography was performed with the result mechanical mitral valve was significantly stenotic with a mitral valve velocity time integral (MV VTI) of 106 cm, peak velocity of the E/E wave peak velocity 2.90 m/s, mean mitral valve pressure gradient 33 mmHg, dimensionless valve index (DVI) 6.6, mitral valve effective orifice area (MV EOA) 0.3 cm². The left ventricle (LV) was collapsing, the systolic function decreased. The patient was transferred to the intensive care unit and thrombolysis was performed with alteplase 10 mg slow bolus followed by 90 mg for 90 minutes. The echocardiography evaluation showed a gradual and significant decrease in the mean mitral valve gradient, increase of MV EOA, decrease of DVI, and increase of LV and RV function.

Conclusions : Acute mechanical valve thrombosis is a life-threatening complication of valve replacement surgery. Modalities such as physical examination and echocardiography should be performed to assess the valve function. Thrombolysis could be an option of treatment.

KEYWORD: thrombolysis, mechanical valve, thrombosis, echocardiography

CASE REPORT

A Rare Brady-Tachy Syndrome Sinus Node Dysfunction Associated with Left Circumflex Artery Occlusion Successfully Reversible with Early Revascularization Strategy

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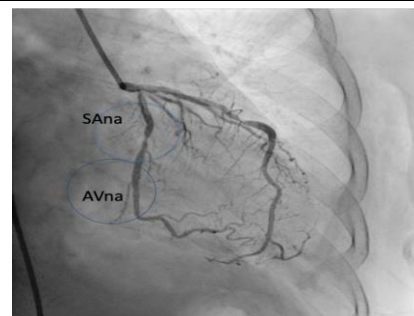
Background: Brady-tachy syndrome, identified by bradycardia alternating with paroxysmal tachyarrhythmias resulting from abnormal conduction within the atrial issue, is a common manifestation of patients with sinus node dysfunction (SND). One of the reversible causes of SND includes acute coronary syndrome (ACS) with reported incidence of 0,3% to 18%. It is most commonly of right coronary artery (RCA) origin, yet a small percentage (7%) are associated with left circumflex artery (LCx) lesions. We present a case of brady-tachy syndrome in a non-ST elevation ACS with significant LCx occlusion successfully reversed with prompt revascularization strategy.

Case illustration: A 71-year-old female was referred to our unit with episodic syncope preceded by atypical chest discomfort associated by sharp epigastric pain and tenderness. Her electrocardiogram at first medical contact demonstrated a total atrioventricular block and atropine sulfate administration was commenced during hospital transfer. At the presentation, patients experienced sudden chest pain with ECG presentation of sinus tachycardia and diffuse ST-depression while troponin levels showed no increase. Diagnostic coronary angiography revealed left dominance, double vessel coronary artery disease with 99% critical stenosis at ostial LCx, 80% significant stenosis at proximal LCx, and long lesion at proximal-mid left descending artery (LAD) with maximum 90% stenosis, and otherwise a normal RCA. The Sinoatrial Nodal artery (SANA) and Atrioventricular Nodal artery (AVNa) come as a branch of the LCx, distal to the occlusion. Percutaneous coronary interventions were performed at proximal to distal LCx and mid LAD as well as balloon angioplasty at ostial LCx, resulting in complete revascularization with TIMI flow III. Electrocardiogram converted into 1st degree AV block with otherwise normal clinical presentations. Recovery was uneventful and the patient was discharged 4 days later in stable condition.

Conclusion: Coronary involvements of reversible SND must be aptly investigated in brady-tachy syndromes and is not exclusively associated with RCA occlusion as sinoatrial nodal artery may arise from LCx in 40% of the population. In such cases, early invasive strategies may provide better outcomes.

KEYWORDS: *sinus node dysfunction, brady-tachy syndrome, coronary artery disease, syncope, arrhythmia*

Figure 1. SANA and AVNa branches from the LCx.





CASE REPORT

Successful Management of Ventricular Septal Rupture after Myocardial Infarction in Elderly : Determine the optimal timing for surgical intervention

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Background : Ventricular septal rupture (VSR) is an uncommon but life-threatening complication after myocardial infarction (MI). Surgical VSR closure associated with high mortality varied significantly depending on timing of surgery, so determining surgical time of VSR is essential.

Case Presentation : A 70-year-old female referred from local hospital with chief complaint was shortness of breath (SoB). 17 days before admission to our hospital, she complained of crushed chest pain, accompanied with cold sweat and nausea then she was hospitalized at a local hospital for 7 days in the intensive cardiac care unit. An ECG showed ST elevation and Q wave at the anterior segment indicating anterior MI. During admission the patient developed worsening SoB and hemodynamic instability. She was referred to dr. Kariadi Hospital for further management.

Echocardiography examination showed interventricular septal defect left to right shunt indicating VSR at apical, ejection fraction was 40%. Coroangiography showed total occlusion at mid LAD. Inotropic, afterload reduction, and mechanical circulatory support with IABP was performed for stabilized hemodynamic and as bridging for surgery. VSR surgical closure and coronary artery bypass grafting (CABG) was successfully performed at days 22 after the onset of MI. The patient was discharged 10 days after surgery with a good condition and improved cardiac performance.

Conclusion : The mortality of VSR varied significantly depending on the timing of surgery. Patients who underwent surgery within 7 days of presentation had a 54.1% mortality compared with 10% mortality if it was delayed until after 21 days. Delayed might facilitate successful repair by allowing friable tissue to organize, strengthen, and become well-differentiated from surrounding healthy tissue and have a good outcome in VSR patients as well.

KEYWORDS : *Ventricular septal rupture; myocardial infarction; timing surgery*



CASE REPORT

Acute Stanford A DeBakey Type I Aortic Dissection in the Third Trimester of Pregnant Woman: A Challenging Case

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Introduction: Acute aortic dissection during pregnancy is a rare condition and life threatening for both mother and the baby. This condition is commonly associated with connective tissue disorder, but may also occur in the absence of these risk factors. Hormonal and hyperdynamic states in pregnancy increase the risk of aortic dissection. We describe a previously healthy woman with acute aortic dissection during pregnancy .

Case Illustration: A 38-year-old with 38th week of pregnancy woman presented with sudden onset of severe ripping back pain in 2 days prior to hospital. She had only mild hypertension during this pregnancy. There was no family history of aortic dissection, connective tissue disease, or Marfan Syndrome. Her blood pressure was 113/50 in right arm and 146/49 in left arm. On physical examination, we found early diastolic murmur in Erb's area. Laboratory test showed anaemia with Haemoglobin 10 g/dL and elevated D-Dimer 3880 ug/L. Echocardiography revealed intimal flap in aortic root, aortic root dilatation with moderate aortic regurgitation, and good left ventricle ejection fraction. CT angiography showed Stanford A De Bakey type I aortic dissection, started from aortic root until descending aorta 5 cm above bifurcation. Patient underwent an emergent caesarean section and aggressive medical therapy to obtain strict control of blood pressure and heart rate with intravenous calcium channel blocker dihydropyridine and oral beta blocker. ACE-Inhibitor was started after delivery. Patient delivered a healthy female baby and was discharged in stable condition. There was no recurrent symptom in 2 month follow up at the outward patient clinic.

Conclusion: Treatment strategy for aortic dissection during pregnancy should be considered based on an individual's comprehensive clinical condition. Appropriate medical management of acute aortic dissection in pregnancy may offer a life-saving procedure for the mother and baby.

KEYWORD : *acute aortic dissection, De Bakey, pregnancy, Stanford*

CASE REPORT

Electrocardiogram manifestation in profound hypokalemia masquerading myocardial ischemia: an emergency general practitioner's perspective

D. Ulfiarakhma¹

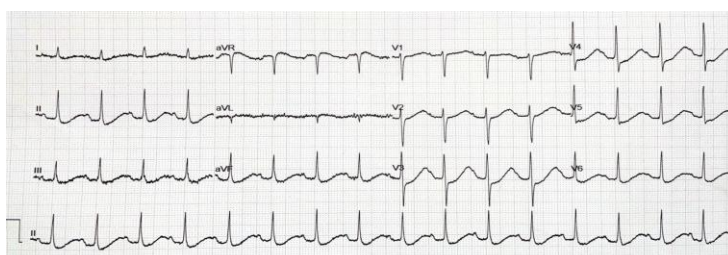
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Background: Hypokalemia is one of the commonly encountered electrolyte disturbances. The physician in the emergency room should be aware of ECG changes caused by hypokalemia, as it might lead to arrhythmia if left untreated.

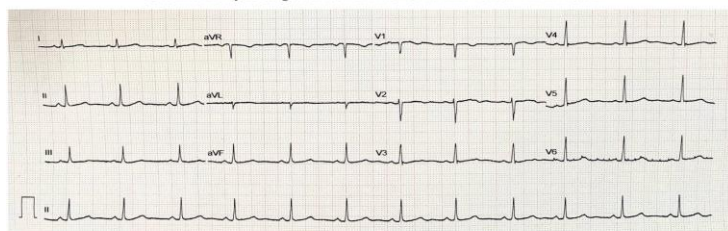
Case description: A 27-year-old non-pregnant woman presented with nausea and severe vomiting. She also complained of chest discomfort spreading from chest to the neck. The pulmonary and cardiac examinations were unremarkable. ECG showed upsloping ST-depression in leads II, III, aVF, V3-V6; and corrected QT interval prolongation at 582 ms. Patient was initially suspected with myocardial ischemia. A detailed history taking revealed that the patient had been suffering from vomiting for two weeks and functional dyspepsia for two years. On neurologic examination, she demonstrated a slow gait. Laboratory values revealed potassium of 1.2 mmol/L and CKMB levels were within normal limits. So the diagnosis of severe hypokalemia was made. Intravenous potassium chloride (KCl) 50 mEq over 5 hours were administered with continuous cardiac monitoring. Patient was discharged 7 days after admission with normal serum electrolyte levels and ECG.

Conclusion: Hypokalemia results in slowed conduction, delayed ventricular repolarization, shortened refractory period, and increased automaticity which can lead to ST-T segment changes and QT prolongation on ECG. Hypokalemia induced ST depression might mimic those of myocardial ischemia, and its interpretation might be difficult when accompanied with chest discomfort. In our patient, normal CKMB level and normalization of ECG findings after potassium correction suggests that ECG abnormalities on admission were secondary to severe hypokalemia.

KEYWORDS: *Hypokalemia, electrocardiogram, ST depression*



a. ECG on admission showed upsloping ST depressions in leads II, III, aVF, and V3-V6 with QTc interval prolongation at 582 ms. Serum K⁺ level: 1.2 mmol/L.



b. ECG on day 7 after correction of hypokalemia showed normal sinus rhythm with improvement in ST-segment depression and decreased QT prolongation. Serum K⁺ level: 3.4 mmol/L.



CASE REPORT

Various Factors for the Occurrence of Revascularization in Patients with Acute Myocardial Infarction : a Case Report

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Background: Chest pain is the main symptom and is most commonly seen in acute myocardial infarction followed by abnormal ECG and elevated cardiac enzymes. Timeliness in reperfusion is one of the keys to a good prognosis. In this case, we will describe the course of the disease in patients with acute myocardial infarction.

Case illustration: A 43-year-old male patient comes to the emergency room at the Kediri General Hospital with chest pain. chest pain can't be pinpointed, radiates to the hand for 4 hours, still hurts at rest, other symptoms are dyspnea and vomiting. The ECG did not show significant abnormality in all leads. however, before the patient was admitted to the emergency unit at the Kediri General Hospital, the patient had been admitted to another hospital and was given dual antiplatelets therapy (DAPT) as initial therapy (aspirin 300 mg and clopidogrel 300 mg) because at that time the ECG showed ST elevation in leads V1-V4. Cardiac enzyme examinations were not carried out there because of inadequate facilities. At the Kediri General Hospital the results of vital signs such as normal blood pressure, normal chest X-ray results, then laboratory results obtained leukocytosis (14,400/ml), hyponatremia (129 mmol/L), elevated troponin enzymes (0.58ng/ml) and normal results on CK-MB (2.15 mg/ml) and procalcitonin (0.15 ng/ml). This patient was treated with DAPT (aspirin and clopidogrel), statins and subcutaneous anticoagulants given for 5 days. During 5 days of treatment, the patient's condition was getting better and his daily ECG evaluation did not show any abnormalities such as ST elevation and others. Revascularization in this patient may occur due to several factors: the duration of symptoms (<4 hours), the low score (TIMI: 2, Killip: 1, Grace: 63) and the time in giving DAPT as initial treatment.

Conclusion : In this case, we conclude that several possible revascularizations that occur in this patient are due to these three factors, namely: the duration of symptoms (<4 hours), the low score (TIMI: 2, Killip: 1, Grace: 63) and the time in giving DAPT as initial treatment, but there are many factors that can lead to spontaneous revascularization in patients with acute myocardial infarction and should be explored further because they will help in the management of this disease in the future.

KEYWORDS: *Chest pain, acute myocardial infarction, Revascularization*



CASE REPORT

Takotsubo Syndrome In A Patient With Burn Injury: A Case Report

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Background: Takotsubo syndrome is an uncommon disease related with physical and emotional stress and its clinical manifestation often mimics acute myocardial infarction. Takotsubo syndrome, also known as heartbroken syndrome, is a reversible cardiomyopathy and typically occurs in older women. Patients with burn injury are having both physical and emotional stress due to inflammatory response, severe pain, limited mobility and concern about cosmetics after burn injury. This patient population is at high risk of takotsubo syndrome.

Case Presentation: A 84-year-old lone woman was brought to the emergency room with mid dermal and deep dermal burn injury due to scald. Electrocardiography (ECG) showed ST-segment elevation in leads V1 through V5 and ECG evaluation showed deep T wave inversion in leads V2 through V6, and in leads I and AVL. Cardiac enzyme levels were elevated. Cardiac catheterization showed no significant coronary artery stenosis and left ventriculography showed Left ventricular apical ballooning. Echocardiography showed an ejection fraction of 45 percent, with severe hypokinetic at apical segment of left ventricle and ballooning of the apical left ventricle. Patient was admitted to Intensive Cardiovascular Care Unit to stabilize the condition before moving to Burn Unit for advanced wound care and underwent a surgical debridement. Patient was hospitalized for 31 days due to sepsis complications. Four weeks later echocardiography evaluation showed normal ejection fraction (51 percent) with mild hypokinetic at apical left ventricle and normal shape of the left ventricle. Burn injury was suggested as the underlying trigger of this condition. Management in this patient was not only improving left ventricular function but also treating the underlying trigger to prevent complications that would worsen the outcome.

Conclusions: Takotsubo cardiomyopathy is a reversible cardiomyopathy, thus prospecting underlying triggers is important for determining comprehensive treatment and improves outcome of the patient.

KEYWORD: *takotsubo, cardiomyopathy, burn injury, heart broken syndrome,*



CASE REPORT

Role of Point of Care Ultrasonography in the Management of Acute Pulmonary Edema in Patient with Diabetic Ketoacidosis and Heart Failure

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Background: Heart failure and diabetes mellitus are two conditions that frequently coexist. Diabetic ketoacidosis is a complication of diabetes mellitus which requires aggressive intravenous fluid replacement. On the other hand, diuretic use and fluid restriction are required for the management of heart failure. Tight monitoring and fluid balance are needed in the management of diabetic ketoacidosis in patients with heart failure. Point of care ultrasonography (PoCUS) plays an important role in diagnosing and monitoring response to fluid therapy.

Objective: To present a case of diabetic ketoacidosis in a patient with acute heart failure, how to recognize the underlying etiology of the developing pulmonary edema in this patient based on history, symptoms, physical and supporting examination, and how to administer appropriate therapy in this patient.

Case illustration: A 71-year-old woman came to the emergency department with a chief complaint of breathlessness. From history taking, it was noted that she had a history of coronary artery disease, heart failure and diabetes mellitus. From physical and supporting examination, she was then diagnosed as having Non-ST elevation myocardial infarction (NSTEMI), diabetic ketoacidosis and acute pulmonary edema. She was then given intravenous fluid replacement, insulin therapy and infusion drip of nitroglycerin.

Conclusion: Underlying etiology of pulmonary edema in diabetic ketoacidosis might be due to increased hydrostatic pressure or increased pulmonary permeability. Identifying underlying etiology of pulmonary edema can be done from history taking and physical and supporting examination since early presentation in the emergency department.

Keywords: *Heart Failure, Diabetic Ketoacidosis, Pulmonary Edema, Point of Care Ultrasonography*

CASE REPORT

Acute Decompensated Heart Failure And Severe Pulmonary Hypertension In Pneumonia And C opd: How To Manage?

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Background: Acute Decompensated Heart Failure (ADHF) patients frequently receive coinciding therapies for pneumonia, acute asthma, or exacerbated chronic obstructive pulmonary disease (COPD). ADHF has higher risk of pneumonia than age and sex-matched in the population and survival from pneumonia is lower in patients with ADHF. Pulmonary hypertension (PH) is a common complication of COPD. Survival and risk of exacerbations were affected by PH in COPD.

Case Illustration: A 55-year-old male came to ER with complaints of shortness of breath and wet cough for one month. The patient had no history of previous disease. Bilateral rales and 15 kg of weight loss (BMI = 16.5 kg/m²) in 5-months were found in physical examination. SARS-CoV-2 rapid test PCR was negative. A blood test revealed leukocytosis. Chest X-Ray indicated the presence of pneumonia with CTR 55%. On the 3rd day, the shortness of breath was worsening. Increased systolic blood pressure from 110 mmHg to 170 mmHg, 3/6 pansystolic murmur in lower left sternal border and cold extremities were found in physical examination. Initial doses of furosemide and NTG titration were given due to the suspicion of ADHF. The patient was admitted to ICCU. On the next day, echocardiography was performed. Moderate tricuspid regurgitation and LV diastolic dysfunction in grade III that indicated PH were found. Berapost sodium, aminophylline, ampicillin-sulbactam, dexamethasone and adequate intake were given. The patient showed clinical improvement and gained 5 kg of weight (BMI = 18.4 kg/m²). 3-days later, the patient became more stable and continued treatment in the regular ward.

Conclusion: Coinciding therapies in patients with two or more acute cardiopulmonary syndromes often had adverse effects from the treatment themselves. There is a theoretical concern about adverse drug effects as all studied drug classes including corticosteroids, antibiotics and loop diuretics can cause direct harms and may work at cross-purposes. In this patient, clinical improvement was achieved with coinciding therapies that were given.

KEYWORDS: *Acute Decompensated Heart Failure, Chronic Obstructive Pulmonary Disease, Pneumonia, Pulmonary Hypertension*

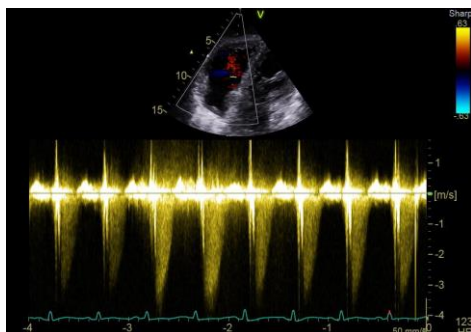


Figure 1. Echocardiography showed TVG 57.65 mmHg and RVP 67.65 that indicated pulmonary hypertension (PH)



CASE REPORT

Ventricular Septal Rupture Complicating Anterior Acute Myocardial Infarction: A Case Series

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Background: Ventricular septal rupture (VSR) is a rare and lethal complication of acute myocardial infarction (AMI). Although progressive breakthrough in cardiovascular care, VSR is still rare and high in mortality.

Case Illustration: We presented 2 documented post-infarction VSR between 1 January to 17 January 2022. The cases consisted of 2 males with age 54 and 73 years old respectively. One patient had hypertension and one had type-2 Diabetes and both are smokers. Both cases experienced the same typical angina and later was diagnosed as STEMI based on electrocardiography and positive troponin results. Both patients showed pansystolic murmur grade 3/6 at the Lower left sternal border. Both patients reveal ST-elevation on the Anterior region. From echocardiography, both patients showed VSR with 5-8mm and 20 mm in diameter with obvious L-R shut and Low BP Simpson EF with also diastolic impairment. Between treatments, the second patient rejected the coronary angiography advice from the hospital while the first patient was scheduled for the coronary angiography. Both of the patients developed the VSR within the 10 days since diagnosis of Acute Coronary Syndrome. Both patients were then admitted to the ICU and medical treatments were given: UFH IV, Aspirin, Ticagrelor, Bisoprolol, Atorvastatin. The second patient was scheduled for an AMVO procedure.

Conclusion: Myocardial infarction is the leading cause of death worldwide. One of its late mechanical complications is VSR. Neglected non-perfused transmural infarction leads to excessive inflammation leaving a yielded VSR as mechanical complication. Some of the established clinical predictors for VSR occurrence were advanced age, anterior location of infarct, no reperfusion treatment, history of Hypertension, Diabetes, and smoking status. It is possible to obtain benefit from septal occluder placement.

KEYWORD: *Ventricular Septal Rupture (VSR), Acute Myocardial Infarction (AMI), Septal occluder*



CASE REPORT

Myocarditis Associated with Covid-19 Infection and Arrhythmia A Case Report

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Background: Diagnosis viral myocarditis is multifactorial and involves detection of elevated cardiac biomarkers and echocardiographic evidence of cardiomyopathy. We describe patients with myocarditis associated with covid-19 and arrhythmia.

Case Description: A 30 years old woman came with a chief complaint of shortness of breath that has been persistent for 4 hour. There was no history of any other disease. She looked ill with GCS E4V5M6, his blood pressure was 80/40, pulse rate 48 beats/min, oxygen saturation 94% with high flow nasal canul (HFNC) Flow 50 Fio2 100%. Temperature was 38°C. Physical examination showed rales bilateral in the lower part of the lungs. Laboratory test showed leukocyte $21,55 \times 10^9/L$, creatinine (cr) 5,8 mg/dl ureum (ur) 193 mg/dl, high sensitive cardiac troponin T (hs-CT) >10000 ng/L, sodium 130 mEq/L, potassium 5,7 mEq/L. PCR swab test showed Positive (+) infection of Covid-19. Electrocardiography (ECG) showed Total AV block (Junctional escape rhythm) with diffuse T wave inversion. Chest x ray showed pneumonia with bilateral lung edema. Echocardiography showed Ejection fraction (EF) 45% with Global hypokinetic. A diagnosis of myocarditis associated with covid-19 infection and total AV block was made. Patient was treated with supplemental oxygen, Covid-19 therapy regimen, ceftazidime 3x1gr/day, dobutamine 5mcg/kg/min, Norepinefrin 0,05 mcg/kg/min. After the hemodynamically stable patient underwent ultrafiltration therapy. After ultrafiltration the patient showed modest improvement. Laboratory evaluation after treatment showed Cr:1,3 Ur:81 leucocyte $8,1 \times 10^9/L$ and ECG showed Sinus Tachycardia with incomplete right bundle branch block.. After that patient was discharged and would be monitored from the outpatient clinic. Acute viral myocarditis is the inflammation of myocardium secondary to immune-mediated lymphocytic infiltration and/or pathogen-directed cytotoxicity resulting in myocyte degeneration and necrosis of nonischemic origin. viral myocarditis is an important cause of myocardial injury. It manifests with a pseudo-infarct presentation consisting of elevated cardiac biomarkers, echocardiographic evidence of cardiomyopathy, in the absence of diseased coronary arteries. The exact mechanisms of cardiac injury in covid-19 are not well established. Cardiac manifestations are partially due to the reduction of ACE2, increase of angiotensin II relative to angiotensin 1–7, hypoxia, and disruptions in the coagulation pathway. The inflammatory response plays a large role in myocarditis resulting from SARS-CoV-2 infection.

Conclusion: Myocarditis associated with COVID-19 has widely variable presentations, which range asymptomatic to life-threatening arrhythmias and hemodynamic compromise. Further research is needed to enhance our understanding of this disease process thus improving risk stratification in this high-risk group, especially in the acute setting.



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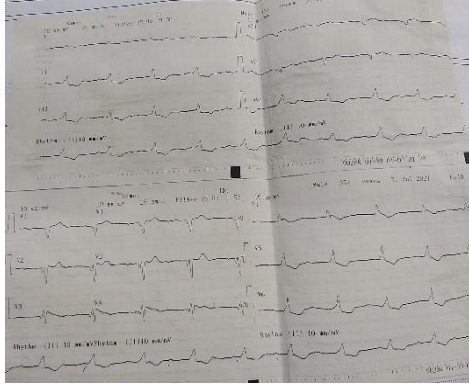


Fig 1. ECG showed total AV block with junctional escape rhythm and diffuse T wave inversion.



CASE REPORT

Recovery of Total Atrioventricular Block in Inferior STEMI with Concomitant Right Ventricular Infarction in 90 Minutes following Fibrinolysis in Incapable Cath Lab Hospital: A Case Report

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Background: Inferior STEMI accounts for 40-50% of all STEMI. It generally has a more favorable prognosis, with only 2-9% in-hospital mortality. However, certain conditions, such as RV infarction, AV block, and posterior infarction, indicate a worse outcome.

Case Presentation: A 45-years-old man presented with typical chest pain 2 hours prior to admission. The ECG showed ST-segment elevation in the lead II, III, aVF, and V2R-V6R with a total atrioventricular block (TAVB) accompanied by hemodynamic stability. Troponin I increased (125.7 ng/L). The patient was treated with Streptokinase, dual antiplatelet, and atorvastatin 40 mg. Door-to-needle time \leq 30 minutes was performed in this patient. Ninety minutes after fibrinolytic chest pain was gone, the elevation of the ST-T segment was reduced $>$ 50 %, and Troponin I increased significantly (1436.6 ng/L). Six hours after fibrinolytic administration, heparin 700 units/h was given. ECG showed improvement and was back to normal on the fifth day. The patient was discharged on the fifth day of treatment.

Conclusion: Immediate diagnosis and management for TAVB in inferior STEMI with concomitant RV infarction are critical to achieving optimal patient outcomes.

KEYWORDS: TAVB, STEMI, fibrinolytic



CASE REPORT

A Rare Case: Concealed Wolff-Parkinson-White Syndrome Revealed After Successful Fibrinolytic

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Background: WPW syndrome is a reentry tachycardia caused by the presence of an accessory pathway that can conduct electricity from the atria to the ventricles without going through the av node. This accessory pathway can be identified by the ecg by the presence of shortened pr intervals and delta waves. However, not all WPW patterns can be found in every patient. This is called concealed WPW syndrome. The presence of new atrial fibrillation caused by acute myocardial infarction can be fatal if followed by WPW syndrome.

Case Illustration: A 55-year-old man comes to the emergency room with complaints of chest tightness that was felt 2 hours earlier. Complaints accompanied by cold sweats. The patient denied having a history of heart disease, hypertension or diabetes. Physical examination was unremarkable. On initial ecg examination found sinus rhythm at a rate of 61 beats per minute, normal axis, normal pr interval, st elevation in leads II III aVF, st depression in leads I aVL V2 to V6. The patient was then reperfused using fibrinolytic with satisfactory results. After fibrinolytic the patient had no further complaints, the st elevation fell by more than 50% and there were episodes of idioventricular rhythm at a rate of 70 beats per minute. However, on ecg examination after fibrinolytic sinus rhythm was found at a rate of 72 beats per minute, shortened pr interval followed by initial formation of slurred qrs complex matching the delta waves in WPW syndrome. This may occur because myocardial ischemia results either shortening the refractory period in the accessory pathway or prolonging the refractory period in the sinus node or av node. During hospitalization there was no tachycardia event, the patient was then discharged.

Conclusion: WPW syndrome can manifest with stable tachycardia and even life-threatening tachycardia. Delta waves in WPW syndrome can aid in the diagnosis. But in asymptomatic patients and concealed WPW the diagnosis will be difficult to establish. It is very rare for concealed WPW to be revealed after an acute myocardial infarction. This case will have implications for standard treatment of acute myocardial infarction such as beta blockers which may be contraindicated.

KEYWORD: *Concealed WPW Syndrome, Tachycardia, Acute Myocardial Infarction, Fibrinolytic*

CASE REPORT

Wellens' Syndrome, a case report of an electrocardiographic warning sign

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Introduction: Wellens' Syndrome is a clinical condition characterized by biphasic or symmetrical electrocardiographic T-wave inversions in precordial leads seen in a subset of patients with unstable angina. This condition is associated with anterior wall myocardial infarction at a later time.

Case Illustration: A 59 years old female patient presented to the emergency department with the complaint of pressure like chest pain lasting for 4 hours and expressed gradually decreasing of this chest pain. She had no history of risk factor for CAD. The patient's initial ECG showed normal sinus rhythm with a biphasic T wave on V2-V3 precordial leads. ECG was performed one hour later, in the painless period, normal sinus rhythm with T wave changes; inversion in V2-V6 precordial leads was found and her troponin level was slightly elevated. In the next day, ECG results showed deep symmetrical T wave inversion in V2-V6 precordial leads. Loading doses of CPG 300 mg and Aspilet 320 mg were given, and also NTG, Atorvastatin 40 mg, Ramipril 5mg, concor 1,25mg and Lovenox 0.6cc SC. She was discharged after 6 days observation without chest pain.

Conclusion: The patient's ECG showed T wave changes can evolve time from Wellens' Syndrome Type A (biphasic T wave on V2-V3 precordial leads) to Type B (deep symmetrical T wave inversion in V2-V6 precordial leads), even in painless period.

This case report underlines the importance of recognizing the characteristic ECG patterns in Wellens' Syndrome which may be a warning signal for a critical LAD lesion that can result in acute MI and sudden death in a week.

KEYWORDS : *Wellens' Syndrome, anterior myocardial infarction*

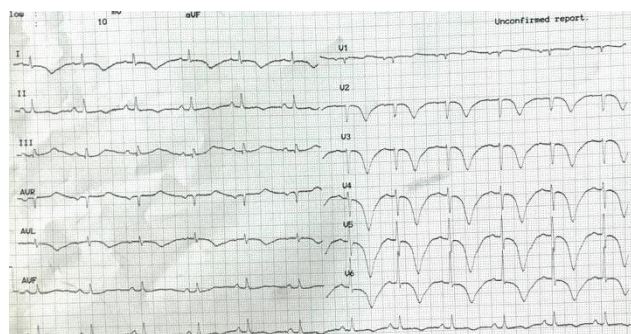


Figure 1. Very deep T inversion, a pathognomonic sign of Wellen Syndrome



CASE REPORT

Echocardiography Guided Pericardiocentesis: Saving lives, one day at a time, just like always

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Background: Pericardial effusion and cardiac tamponade represent a spectrum of disease with wide variation in clinical presentation. Cardiac tamponade with hemodynamic collapse is an absolute indication for emergent pericardial drainage via pericardiocentesis or surgical pericardiotomy. Echocardiography guided pericardiocentesis is currently considered the standard of care for patient life saving.

Case Presentation: We report a 51-year-old woman with main symptoms of progressively worsening dyspnea and shortness of breath 2 days before hospitalization. She has a history of breast tumors that was discovered about 2 months ago. From physical examination, she was found to have dyspnea at rest, hypotension, and jugular venous distension. Bedside transthoracic echocardiography (TTE) was done. It showed massive pericardial effusion in the anterior (2.2 cm), left lateral (3.4 cm), and right lateral (4.1cm); moderate pericardial effusion in the inferior (1.6) with right atrium and ventricle collapse assessed qualitatively. Working up in the emergency room, we also suspect pneumonia due to COVID-19. Because the limitation of cath lab for COVID-19 patients, thus pericardiocentesis with subxiphoid approach was done with echocardiography guidance. Pericardial fluid was obtained with a serous hemorrhagic color, and it was sent to the laboratory for pericardial fluid analysis. The symptoms of the patient were improved after the procedure.

Conclusion: Acute pericardial effusion is one of common manifestations in malignancy. Patients with massive pericardial effusion accompanied with increased jugular venous pressure and the impression of a distant heart sound are indicated for pericardiocentesis. Echocardiography guided pericardiocentesis is a simple procedure that has been shown to be both safe and effective therapy for emergent rescue in the setting of tamponade. Novice echosonographers can both identify and successfully conduct bedside pericardiocentesis safely with an in-plane technique which allows for needle visualization.

KEYWORDS: *Pericardial effusion, cardiac tamponade, echocardiography guided pericardiocentesis*



CASE REPORT

Acute Myocardial Infarction in a Patient with End-Stage Colon Carcinoma: A Case Report

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Background: The incidence of acute myocardial infarction in cancer patients is common. Notably, myocardial infarction may develop before or after diagnosis of cancer; sometimes, the diagnosis is made during hospitalization for myocardial infarction and, conversely, in some cases, acute myocardial infarction may occur during hospitalization for active cancer. This article reports an 83-year-old female with end-stage colon carcinoma experienced acute myocardial infarction during hospitalization.

Case Illustration: An 83-year-old female was admitted to hospital with end-stage colon carcinoma metastatic to liver and lung with a chief complaint of general weakness and abdominal pain. The patient was planned to undergo laparoscopic left hemicolectomy after general condition had improved. On the 6th day of hospitalization, the patient experienced decreased consciousness, shortness of breath, with no diaphoresis. The physical examination showed stable hemodynamic. There was gallop present at cardiac auscultation and crackles with decreased vesicular lung sound at the right side of the lung. ECG showed inverted T at lead V1- V4. There was an elevation in cardiac troponin and significant elevation in NT pro-BNP. The patient was diagnosed with lateral non-ST elevation myocardial infarction KILLIP II with massive right pleural effusion. The patient was transferred to the intensive care unit and intubated afterward. The patient got initial management for acute myocardial infarction. Anticoagulant was put on hold as it could increase the risk of gastrointestinal bleeding for the patient.

Conclusion: Acute myocardial infarction may occur during hospitalization for active cancer. In this case, it was thought that further decrease in cardiac function had developed after diagnosis of cancer as the patient had no history of cardiac diseases and hypertension nor diabetes mellitus. It is necessary to closely monitor the risk of myocardial infarction in patients with cancer during hospitalization. Therapy given in patients with active cancer is limited considering the frailty of the patients.

KEYWORDS: *Myocardial infarction, cancer, hospitalization, colon carcinoma*



CASE REPORT

The Importance of Comprehensive Examination for Early Diagnosis of Persistent Pulmonary Hypertension of the Newborn in Limited NICU setting: A Case Report

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Background: Persistent Pulmonary Hypertension of the Newborn (PPHN) is one of the leading causes of infant deaths in the Neonatal Intensive Care Unit (NICU), with a mortality rate ranging between 4% and 33%. PPHN diagnosis and therapy rely heavily on modern therapies such as inhaled nitric oxide (iNO), high-frequency oscillatory ventilation (HFOH), and advanced pulmonary vasodilator agents with echocardiography remain the gold standard for diagnosis. In a resources-limited setting, these therapies might not be available. Herein, we describe a case of timely diagnosis of PPHN, using a simple diagnostic measure, careful risk factors evaluation with practical interventions using readily available resources in a limited NICU setting.

Case Illustration: A male infant was born at 38 weeks of gestation with a birth weight of 3740 g from an obese mother. Soon after, he experienced central cyanotic and tachypnea. There is a significant difference between pre- and postductal oxygen saturation. Echocardiography revealed left ventricle dominant with an ejection fraction of 54.14%, severe mitral and tricuspid regurgitation, and high tricuspid valve pressure gradient; hence we suspected severe pulmonary hypertension. Blood gas analysis (BGA) showed partially compensated metabolic acidosis. The patient was sedated and intubated, and then we administered iloprost inhalation at a dosage of 4 microgram/dose Q4H soon after he was diagnosed. Dopamine was given to dilate pulmonary arteries. On the twelfth day of hospitalization, the patient was discharged.

Conclusions: Diagnosis of PPHN in a limited resource setting relied heavily on clinician high-index of suspicion. Identifying major risk factors for PPHN and using simple examination (e.g., pre and postductal O₂ saturation, hyperoxia test) to eliminate other differential diagnoses; may effectively assist the diagnosis. In the setting of limited facilities and manpower, maximizing oxygen supply, decreasing oxygen demand, correcting and preventing metabolic disturbance with adequate ventilatory support can help significantly improve patient condition before referral to the next level of care.

KEYWORDS: *persistent PH, newborn*

CASE REPORT

Hypokalemia-Induced Bidirectional Ventricular Tachycardia in Hypokalemic Periodic Paralysis: A Rare Case Report from Rural Hospital

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Background: Hypokalemia-induced bidirectional ventricular tachycardia (BVT) is a rare case but potentially fatal without prompt treatment. There are several causes of BVT previously reported, one of them is hypokalemic periodic paralysis.

Case description: A 28-year-old female presented to the emergency department with weakness in the extremities one day prior admission. She had been suffering two similar episodes within one year. Patient was alert with a blood pressure of 129/89 and heart rate of 105 beats/min. Her motor strength was 1/5 in all extremities. First ECG showed sinus rhythm with ventricular bigeminy with R on T phenomenon. Laboratory values revealed potassium of 1.9 mEq/L and immediately her ECG changed into BVT on monitor. A diagnosis BVT and hypokalemic periodic paralysis was made. Patient suddenly went into cardiac arrest and then ROSC after one minute cardiopulmonary resuscitation (CPR). Immediate intravenous potassium chloride (KCl) 40 mEq over 4 hours and oral potassium (KSR) tablet 3 times a day were administered. Patient paralysis was resolved and ECG converted to sinus rhythm with occasional premature ventricular complex in the next few hours. Her potassium serum level increased to 2.4 mEq/L. She was discharged 7 days after admission with normal potassium serum level (3.5 mEq/L) and ECG. Hypokalemia results in slowed conduction, delayed ventricular repolarization, shortened refractory period, and increased automaticity. In our patient, low extracellular potassium level reduced the permeability of myocardial cell membrane to potassium. This will inhibit outward potassium currents, thus prolong repolarization and afterdepolarization which promotes triggered BVT.

Conclusion: Acute management of hypokalemia-induced BVT consists of the prompt administration of intravenous potassium cautiously. After potassium correction, our patient showed no episodes of arrhythmia and paralysis. These findings strongly suggest hypokalemia-induced BVT.

KEYWORDS: *Bidirectional ventricular tachycardia, periodic paralysis hypokalemia*

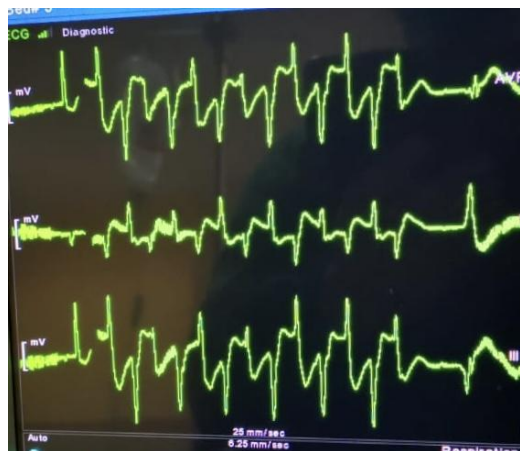


Figure 1. Episode of biventricular VT recorded on monitor

CASE REPORT

Successful Management in Rare Case of Bacterial Sepsis Induced Acute Myocarditis Complicated with Total Atrioventricular Block: Focus on The Role of Steroid

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Background: Myocarditis refers to inflammation of the heart muscle which occurs as a consequence of infections, exposure to toxic substances, or immune system activation. Myocarditis has a wide range of life-threatening clinical presentations including acute heart failure, cardiogenic shock, and life-threatening arrhythmias. Systemic steroids might be considered to overcome myocarditis and provide better cardiac recovery.

Case Illustration: A-22-year-old-woman came to the emergency room with a chief complaint of general weakness then got a seizure several times. She had a history of fever, chest pain, and unexplained syncope since a week ago. Physical examination revealed low blood pressure, low heart rate, and poor peripheral perfusion. Laboratory results revealed increased leukocyte, liver enzyme, and cardiac troponin. Bacteriuria was also found. ECG showed total atrioventricular block (TAVB) with ventricular escape rhythm 30 bpm. She was diagnosed with sepsis induced acute myocarditis. We aggressively treated her with high-dose systemic steroids to suppress myocardial inflammation, double antibiotics to treat the infection, and a dopamine and aminophylline combination to treat TAVB. Those medications completely improved her clinical status. She was stabilized on the second day of hospitalization and discharged after being treated for 7 days. In our case, myocarditis was caused by a hyper-inflammatory response induced by bacterial sepsis. Current experts' recommendation suggests empirical use of high dose systemic steroids in acute myocarditis complicated with unstable conditions such as acute heart failure, cardiogenic shock, or advanced heart block. This immunosuppression-based therapy plays a role to suppress inflammatory mediators activity which promotes faster cardiac recovery in myocarditis.

Conclusion: Myocarditis may present with life-threatening conditions such as cardiogenic shock or advanced heart block which need appropriate treatment to prevent morbidity and mortality. Empirical use of high-dose systemic steroids plays a role to suppress hyper-inflammation response in myocarditis which promotes faster cardiac recovery.

Keywords: Acute Myocarditis, Bacterial Sepsis, Inflammation, Systemic Steroid

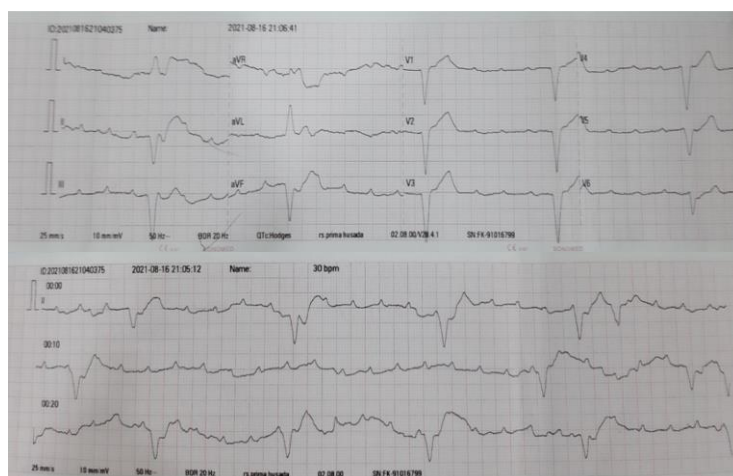


Figure 1. Patient's ECG shows TAVB



CASE REPORT

Refractory Ventricular Tachycardia Unconverted with Electrical Cardioversion: A Case Report

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Background: Unstable ventricular tachycardia (VT) is a life-threatening condition. Its cause varies from myocardial injury to electrolyte abnormality. The initial management of unstable VT relies on electrical cardioversion. Afterward, the underlying reversible cause [hypovolemia, hypoxia, hypothermia, hydrogen ion, hyper/hypokalemia, tension pneumothorax, cardiac tamponade, coronary thrombosis, pulmonary thrombosis, and toxin (5H & 5T)] should be identified. We report a case in which we managed a patient with refractory VT unconverted with electrical cardioversion.

Case Illustration: A 66-year-old woman presented to the emergency room (ER) with fatigue, chest pain, and palpitation that was occurring for 12 hours. The patient's heart rate was 184 bpm. The initial electrocardiogram showed monomorphic VT. Due to the unstable condition and VT, cardioversion was performed 4 times (100 J, 150 J, 200 J, 300 J) in the ER. However, the VT remains unconverted. The initial laboratory results showed Troponin T 80 ng/mL, potassium 6.2 mmol/L, serum ureum 219.1 mg/dL, serum creatinine 3.86 mg/dL, and eGFR 11.5 mL/min/1.73m². The patient was assessed as unstable unconverted VT, NSTEMI, CKD, and hyperkalemia. NSTEMI initial therapy was given and amiodarone infusion was initiated based on the advice of the attending cardiologist. Then, 2 flacons of D40%, 5 units of fast-acting insulin/4 hours and 1 gram of calcium gluconate/4 hours were administered to correct hyperkalemia as advised by the attending internist. The patient was then admitted to the ICU. The bedside echocardiography showed hypokinetic in the basal-mid inferior, and inferoseptal segment, mild tricuspid regurgitation, and LVEF 64%. Post-potassium correction and amiodarone administration, the serum potassium declined to 4.7 mmol/L and the VT was converted into multiple premature ventricular complexes, right bundle branch block, and inverted T wave in V1-V6.

Conclusion: Electrical cardioversion should be attempted as the initial management in unstable VT patients. The underlying reversible cause of VT (5H & 5T) should be identified and managed promptly. Amiodarone administration can be considered to be given in refractory VT. NSTEMI and hyperkalemia were suspected to be the underlying cause of refractory VT in our patient and these conditions should be managed by a multidisciplinary team.

KEYWORDS: *Cardioversion, Hyperkalemia, Ventricular tachycardia*

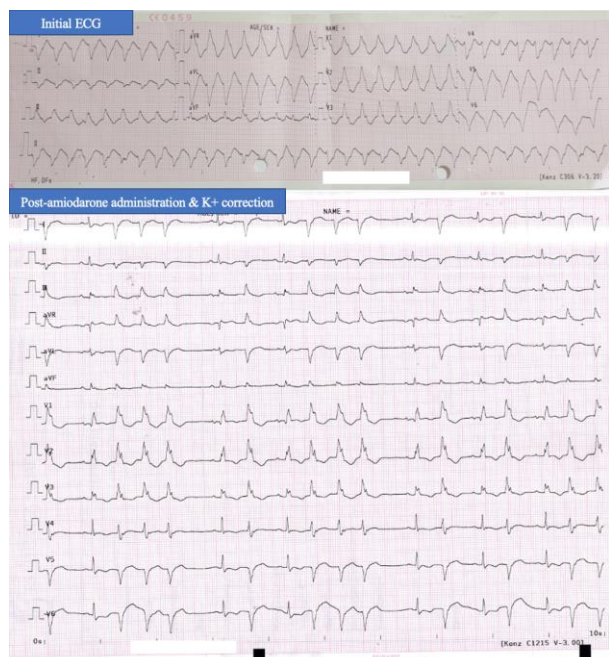


Figure 1. Electrocardiography examination

CASE REPORT

Acute Myocardial Infarction with High Thrombus Burden as a Complication of COVID-19 : a Case Series

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Background: COVID-19 and other coronavirus infections have been associated with cardiovascular complications as a result of the pro-inflammatory response and potential for vascular endothelial damage which can result in acute cardiac injury (myocarditis, acute myocardial infarction), heart failure and arrhythmia. There have been several publications showing an increased thrombus burden in STEMI patients infected with COVID-19 compared to uninfected STEMI patients.

Case Illustration: We reported 2 cases of STEMI events in patients with COVID-19. The first patient (39-year-old man) admitted to the emergency department with 2 hour onset inferior STEMI presentation and had confirmed COVID 19 by PCR test 5 days before STEMI onset, while the second patient (50-year-old man) admitted to the emergency department with severe ARDS due to COVID 19 and had acute inferior STEMI while in the CVCU isolation room with VT/VT onset at day 7th admission. Both patients were treated for PCI of the RCA lesion by performing procedures according to the high thrombus burden strategy, such as thrombectomy, balloon angioplasty, intracoronary vasodilators, use of glycoprotein IIb/IIIa inhibitors and deferred stenting strategy. Post procedure was maintained with drip infusion of glycoprotein IIb/IIIa inhibitors and continued with heparinization using LMWH. Both clinical outcomes were good, LVEF evaluation 70% and 42% respectively. Planning for re-invasive coronary angiography was scheduled during outpatient control at the polyclinic due to limited government insurance regulations. We hypothesized the STEMI was as thrombotic complication of COVID-19, because the STEMI onset occurred in the hyperinflammatory phase of COVID-19, characterized by high inflammatory parameters, coagulation, cardiac enzymes, and high thrombus burden from diagnostic angiography and PCI.

Conclusion: Early detection and management of cardiovascular events in COVID-19 patients is very important to reduce the risk of mortality and morbidity. Secondary prevention of cardiovascular disease is very beneficial to be carried out and strengthened in COVID-19 patients who have comorbid cardiovascular and metabolic diseases, in addition to providing optimal COVID-19 therapy.

KEYWORDS: COVID-19, Cytokine Storm, STEMI, High Thrombus Burden

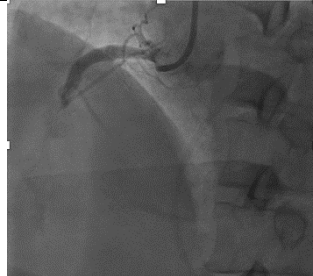

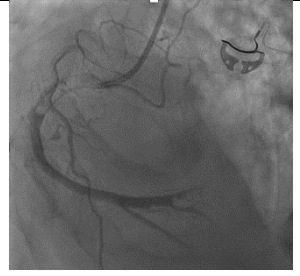

			
Patient 1. Total occlusion mid RCA. Thrombus grade V. TIMI flow 0	TIMI flow 2. Diseksi (-), residual thrombus grade IV (+)	Patient 2. Thrombus grade IV and V proximal and distal RCA. TIMI flow 1	TIMI flow 2. Diseksi (-), residual thrombus grade IV (+)

Figure 1. Angiography result before and after stent implantation



CASE REPORT

Postpartum Pulmonary Embolism With Hemodynamic Deterioration: What To Do Next?

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Background: Pulmonary embolism is one of the leading causes of non-obstetric mortality in pregnancy in developing countries. Almost 60% of the incidence of pulmonary embolism occurs after delivery. Delay in diagnosis and therapy are going to cause mortality from venous thromboembolism.

Case Illustration: A 36-years old female, with a history of cesarean delivery a month ago, was admitted to the ER with shortness of breath for 6 hours before admission. There was no abnormality on her physical examination but we found tachycardia with SBP > 100 mmHg. The revised Geneva score was 3 (tachycardia and history of surgery). D-dimer test was highly increased but the troponin level was normal. The ECG showed the S1Q3T3 pattern and echocardiography revealed the large thrombus in inferior vena cava with no sign of right ventricle pressure overload. CTPA cannot be performed. So, the patient was classified as pulmonary embolism with intermediate-low risk and treated with parenteral anticoagulant. Twelve hours after admission, the SBP was below 100 mmHg, tachycardia was increased with anuria despite adequate fluid input. The bedside TTE showed signs of RV dysfunction. So the patient was classified as high risk pulmonary embolism. So, we treated the patient with 100 mg rtPA agent over 2 hours. There was no complication during the procedure. After the procedure, the shortness of breath was decreased, the BP was 132/87 and heart rate 90 bpm with urine output 0.8 cc/kgBW/min. The bedside TTE and vascular Doppler showed that no thrombus was found for the next day. We planned to treat the patient with oral anticoagulants for 3 months.

Conclusion: The incidence of VTE postpartum (until 6 weeks) is 3–7 incidence per 10 000 deliveries. Pregnancy and the postpartum period are associated with a manifest shift of the coagulation and fibrinolytic systems towards hypercoagulability state, venous stasis, and vascular damage. The PE event increases on cesarean delivery patients. The diagnosis and treatment of the patient can be tricky and must conclude from the anamnesis, vital sign, laboratory test, and imaging test. For the patient with hemodynamic instability, thrombolytic therapy is the step we must choose.

KEYWORDS : *Postpartum, pulmonary embolism, thrombolytic*



CASE REPORT

Pulmonary Embolism Presenting as Paroxysmal Atrial Tachycardia: Case Report

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Background: Pulmonary embolism usually has multiple presentations. Atrial tachyarrhythmias occur in 4%–14% of patients presenting with pulmonary embolism. Sinus tachycardia being the most common, followed by atrial tachycardia, atrial fibrillation, and atrial flutter.

Case Illustration: A 42-year-old female patient with a history of oral contraceptive use was admitted to hospital because of sudden dyspnoea, palpitations, chest discomfort and weakness. On physical examination, an increased resting heart rate of about 150 per minute was found. ECG revealed atrial tachycardia with right bundle branch block. Since the clinical presentation was suggestive of some heart disease the patient underwent echocardiography which revealed indirect signs of acute pulmonary embolism. The diagnosis was confirmed with multislice computed tomography of the pulmonary arteries. Blood test results raised of troponin and D dimer. Based on imaging, blood test results and clinical presentation, intermediate – high risk pulmonary embolism was diagnosed in the patient. Since the patient was stable without signs or symptoms of haemodynamic compromise, unfractionated heparin was administered. The patient's condition did not improve significantly. There were recurrent dyspnoea and desaturation. Thrombolytic therapy using alteplase was performed. The patient's state improved significantly within four days and continued the monitoring at the ward.

Conclusion: Occurrence of paroxysmal atrial tachycardia in pulmonary embolism is rare. High index of suspicion is required and wherever appropriate, D dimer testing should be done when all other causes are eliminated. So that appropriate treatment and delay of diagnosis won't increase the chance of mortality due to pulmonary embolism.

KEYWORDS: *Pulmonary embolism, atrial tachycardia*



CASE REPORT

Value of Hemodynamic Monitoring in a Patient with Respiratory Failure Using Mechanical Ventilation: a Case Report

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Background. Positive pressure ventilation is an essential life support measure in the intensive care and extended care environments. The physiologic effects of positive pressure ventilation have complex interactions with the lungs and other organ systems. Some of these physiologic effects are beneficial, while others may cause complications. Mechanical ventilation creates airflow by generating a pressure gradient. In turn, the pressures in the airways, thoracic cage, and pulmonary blood vessels are altered. In a clinical setting, the cardiovascular functions should be evaluated and monitored to prevent the adverse effects of positive pressure ventilation on the heart and blood vessels.

Case illustration. We present a case of a 67-year-old woman with pre-existing hypertension, recurrent asthma bronchial and heart failure who entered the intensive care unit with signs and symptoms of respiratory failure and septic condition related to pneumonia. At the time of admission, the patient showed a poor overall condition, hemodynamic instability, tachycardia, hypotension, lung oedema and needed inotropic and vasoactive drugs. Initially the patient was treated with pressure control ventilation. However, hemodynamic complications led to further hypotension and shock. We performed daily monitoring to evaluate hemodynamic parameters using trans-thoracic echocardiography. Higher positive end-expiratory pressure was further needed to overcome the acute respiratory distress syndrome, but consequently, a higher dose of inotropic was needed to cope with the shock condition. Daily adjustment of ventilator setting was performed, and eventually in the 6th day, inotropic and vasoactive drugs were no longer needed. Patient was in stable haemodynamic condition with positive end-expiratory pressure of 6 cmH₂O.

Conclusion. Daily hemodynamic monitoring should be performed in patients with mechanical ventilation. Adjustment of ventilator setting, together with maintenance of intravascular fluid adequacy, according to hemodynamic status of the patient turned out to be very important to produce better outcomes.

KEYWORDS: *hemodynamic monitoring, mechanical ventilation, respiratory failure, shock*



CASE REPORT

Arterial Thrombosis Induced By Coagulopathy Due To Coronavirus Infection Concomitant With Heparin Resistance: A Case Report

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Background: A newly emerging pandemic of coronavirus disease 2019 caused by severe acute respiratory coronavirus 2 is responsible for significant morbidity and mortality worldwide. One of the effects is hematological changes related to the COVID-19 infection causing patients to tend to thrombosis rather than hemorrhagic. Current review of evidence and statements on management of coagulopathy and thrombotic complications related to this novel disease needs to be explored.

Case Illustration: We present male, 53 years old, who has had chief complaint pain and tingling in both of his feet since 11 days before admission with mild activity and was not relieved by rest. His complaint was getting worse accompanied by leg swelling, cold and pale. From physical examination, we found his blood pressure was 143/88mmHg, HR 112x/m regular, RR 20x/m with SaO₂ 98% on nasal cannula. Moreover, for vascular status, we found pain, pale, paresthesia, pulseless, poikilothermia and motoric disturbance at both of his legs. From Chest X ray there is cardiomegaly with pulmonary infiltration. From doppler ultrasonography examination we found thrombus >50% in the left superficial femoral artery and total occlusion in the left and right popliteal artery. Laboratorium result showed an increment in D-dimer from 2.15 mg/dL into 3.42 mg/dL, ferritin 756.6 ng/mL, and fibrinogen 489 mg/dL. Swab antigen result for COVID 19 was positif. This patient was assessed as Pneumonia COVID-19 severe with acute limb ischemia bilateral grade IIB. He then performed a surgical embolectomy and was given heparin drip after that. Target aPTT was difficult to achieve with optimal heparin dosage.

Conclusion: We present one of the cases about coagulopathy related to coronavirus infection with heparin resistance as a concomitant condition. In this case report we discuss the management to overcome heparin resistance.

KEYWORDS: *coronavirus infection, coagulopathy, acute limb ischemia, thromboembolism, heparin resistance*



CASE REPORT

Acute Intestinal Bleeding After Percutaneous Coronary Intervention: How to Manage

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Background: Globally, antiplatelet therapy is used in the PPCI setting for treating patients with AMI, and has been shown to reduce adverse cardiac events and prevent short- and long-term [stent thrombosis](#) after PCI. However, antiplatelet therapy also increases the risk of post-PCI bleeding complications.

Case Description: A 53 yo male was referred to the ER with STEMI Inferoposterior RV Infarct. Aspirin 320 mg and Clopidogrel 300 mg has been given in previous hospitals. Then, he underwent PPCI at our hospital and has been added ticagrelor 180 mg before. He was admitted to CVCU. At the 2nd day of care, there was a decrease of Hb from 13.7 to 10.1 but there is no bleeding manifestation. Then the DAPT (Aspirin and Ticagrelor) was continued with monitoring of bleeding signs. On the 4th day of care, the patient defecated with blackish stool. We decided to examine blood examination cito, NGT insertion, gastric lavage and fasting for the patient. We assess the hemodynamically stable patient with moderate bleeding with blood loss >3g/dL so we decided to switch to a less potent P2Y12 inhibitor with clopidogrel and shortening DAPT duration in the long-term plan and give PPI injection. The next day, melena was resolved and gastric lavage was clean. Patient was discharged on the 6th day of care and controlled in the outpatient clinic with good condition. Accurately predicting a patient's individual risk of bleeding post PCI can be a useful tool for a clinician. We can use DAPT and Precise DAPT score can be used to calculate predicting bleeding complications in patients undergoing stent implantation and subsequent DAPT. We should calculate these scores in every situation of the patient that is undergoing PCI.

Conclusion: Bleeding from the GI tract is a commonly encountered clinical problem after PCI. To appropriately manage GI bleeding in this setting, the clinician must strike a balance between arresting hemorrhage and preventing ischemic coronary complications. The decision to withhold or continue DAPT in this setting largely depends on ischaemic vs recurrent/prolonged bleeding risks.

KEYWORDS: *Dual Antiplatelets, Gastrointestinal Bleeding, Percutaneous Coronary Intervention*



CASE REPORT

**The Role of Emergent Percutaneous Right Ventricular Outflow Tract Intervention
in Adults Congenital Heart Disease with Severe Cyanotic Spells: A Case Report**

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Background. Cyanotic spell is an emergency in cyanotic congenital heart disease (CHD), especially in right ventricular outflow tract (RVOT) obstruction with shunt. It needs appropriate and timely management to prevent death. The goal of cyanotic spell management is pulmonary blood flow restoration achieved either by medical therapy or emergent surgery, such as Blalock Taussig (BT) shunt. However, if medical therapy fails and the risk of surgery is too high, percutaneous RVOT intervention is an alternative option to restore pulmonary blood flow. This case reports a successful percutaneous RVOT intervention as a live saving management in adults with severe cyanotic spell.

Case Illustration. A 19-years-old man with double outlet right ventricle (DORV), inlet ventricular septal defect (VSD), severe infundibular pulmonary stenosis (PS) came to our hospital for elective right heart catheterization. In the morning before the procedure, he experienced a severe cyanotic spell where his SpO₂ dropped from 63% to 30%, BP 100/60 mmHg, heart rate 124 bpm. Arterial blood gas analysis showed severe acidosis and hypercarbia. Fluid loading and intravenous morphine were given and the patient was intubated. Unfortunately, there were no intravenous beta blockers available and the risk of surgery was too high. So, we decided to do percutaneous RVOT intervention. Predilation was performed with Passeo 35 8.0 x 80 x 130 mm balloon along the RVOT and pulmonary artery (PA). The balloon was inflated several times at 7-8 atm for 10 seconds. The balloon was deflated and ejected. Dynamic 10 x 56 x 80 mm stent was placed and inflated at 12 atm for 10 seconds in RVOT. After intervention, his peripheral saturation raised to 89% and there was not any significant complication. Patient was transferred to an intensive cardiovascular unit. The patient was extubated the next day and was discharged on day 7.

Conclusion. Emergent percutaneous RVOT intervention is a safe and effective life saving management for patients with severe cyanotic spells when medical therapy fails and the risk of surgery is too high.

KEYWORDS: *Percutaneous right ventricular outflow tract intervention, cyanotic spell*

CASE REPORT

Uncommon Presentation of Takotsubo Cardiomyopathy: Does the Diagnosis Still Stand?

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Background: Prevalence-wise, Takotsubo cardiomyopathy has been reported to be 2% of all patients with clinical presentation of acute coronary syndrome. The challenge is to be aware of the disease regardless of the low prevalence. Moreover, an uncommon presentation of Takotsubo Cardiomyopathy makes it harder to recognize the entity let alone proving the diagnosis. Thus, the uncommon presentation of Takotsubo Cardiomyopathy is highlighted in this case report.

Case illustration: A 71 years old woman came to the emergency room with a chief complaint of dyspnea from one week ago and worsened in the last three days. The initial electrocardiogram showed slight ST-elevation, thorax Rontgen showed the congestion and elongation of the aorta. Increase in high-sensitive Cardiac Troponin T and The NT-Pro BNP levels were present along with apical ballooning of the LV and reduced RV function. Physiological stress was found to be the death of her husband one week ago. Although the left ventriculography of this patient was classically depicted as the octopus trap, we did find obstructive coronary artery disease in the left anterior descending artery. Presented in this case is a 71 years old woman with uncommon presentations of Takotsubo Cardiomyopathy. These uncommon presentations added challenges to prove the diagnosis of Takotsubo which further affected the treatment.

Conclusion: Regardless of the uncommon presentation such as symptoms of dyspnea with no chest pain, electrocardiography of only slight ST elevation, the presence of obstructive coronary artery disease in left anterior descending artery, and reduced right ventricular function proofed by the TAPSE measurement, it was proven that the Takotsubo Diagnosis still stand and thus the treatment of this patient was based on the diagnosis of Takotsubo Cardiomyopathy. The excellent result of improvement in left ventricular function has proven that the treatment was adequate and appropriate for this patient.

KEYWORDS: *Takotsubo Cardiomyopathy, Uncommon presentation, Coronary artery disease, apical ballooning*

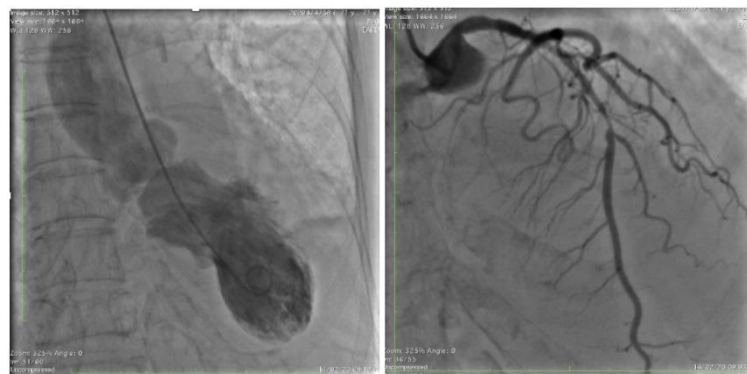


Figure 1. Left: Apical Ballooning of the Left Ventricle from the Left Ventriculography;
Right: LAD stenosis from ostial to mid