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Abstract: Case Report

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Low risk Feature of Left Lateral Accessory Pathway during Electrophysiology Study Ablate or not to ablate?

A. Mushlih[1] ,G. Karwiky[1]
[1]Department of Cardiology and Vascular Medicine, Padjadjaran University, Bandung, Indonesia

Background: The prevalence of Wolff-Parkinson-White in general population is estimated to be 1-3 in 1000 individual. Symptomatic patients with Wolff-Parkinson-White often present with palpitation or syncope caused by atrioventricular reciprocation tachycardia or primary atrial tachycardia. Ventricular fibrillation and sudden cardiac death are rare manifestations of Wolff-Parkinson-White syndrome resulting from rapid conduction of atrial fibrillation over accessory pathway. On the other hand, 80% of patient with asymptomatic Wolff-Parkinson-White will not develop arrhythmia event. Catheter ablation eliminate risk of sudden cardiac death but can result in serious complication. This case will discuss risk stratification and catheter ablation consideration in asymptomatic patient with Wolff-Parkinson-White electrocardiographic pattern.

Case illustration: A-39-years old male presented for routine medical check-up. He was asymptomatic and never experienced palpitation or syncope. His electrocardiographic findings were suggestive of Wolff-Parkinson-White pattern. Echocardiography showed result of normal all chambers dimension, normal LV systolic function (LVEF 60% Biplane Simpson's), Normal LV diastolic function, normal valves, Low probability of PH, normal RV contractility. He was then planned to undergo electrophysiology study and ablation procedure. Electrophysiology study showed low risk left lateral accessory pathway with effective refractory period of 640ms and non-inducible tachycardia. Ablation was not done because of low-risk accessory pathway profile.

Conclusions:
In this case, electrophysiology study was chosen to risk stratify patient with asymptomatic Wolf-Parkinson-White electrocardiographic pattern. Inducibility of atrioventricular reciprocating tachycardia, multiple accessory pathways, and accessory pathway refractory period <250ms as features of increased risk of sudden cardiac death was not found during study. Identification of an abrupt and complete loss of preexcitation during exercise testing has been proposed as a surrogate method of assessing the accessory pathway refractory period. However, 20% patient with intermittent preexcitation have accessory pathway refractory period <250ms making this imperfect marker of low-risk accessory pathway. Catheter ablation is recommended in patient with high-risk accessory pathway features from electrophysiology study. When performed by experienced operator catheter ablation is associated with high success rate and low risk of major complication. Decision to perform catheter ablation in low-risk accessory pathway depend on centre experience and patient's preferences.

KEYWORD: *Wolf-Parkinson-White, Risk Stratification.*

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Phenytoin ''the old fashioned drug'' as an alternative therapy for Long QT Syndrome Type 3 Patient

A. P. Febrianto[1] ,P. V. Mustafiza[1]
[1]Department Cardiology and Vascular Medicine, Dr. Moewardi Hospital

Background: Long QT syndrome (LQTS) type 3, is one of inherited channelopathy presenting with QT prolongation, with high risk for ventricular arrhythmias. Sodium channel blockers such as mexiletine is indicated in LQTS3 patients with a prolonged QT interval. Implantation of ICD has class 1 recommendation for survivor cardiac arrest patient. Unfortunately, those treatments were not covered by our national health insurance. We proposed use of phenytoin, so far known as an anticonvulsant drug which also has sodium channel blocker property to reduced ventricular arrhythmias in LQTS3 patients.

Case illustration: A 30 y.o woman was consulted to cardiac department with polymorphic VT post defibrillation. She was brought to ER because of repeated seizures. She had history epilepsy since 15 y.o with routine phenytoin therapy and never had seizures since then. However, after giving birth 2 months ago, she discontinued phenytoin and had frequent seizures usually occurring while she was sleeping or resting. ECG post defibrillation showed sinus rhythm with prolonged QTc 600ms. Physical examination, transthoracic echocardiography, and electrolytes within normal limits and there was no history of taking drugs which could trigger QTc prolongation. Neurological examination and EEG also normal and show no evidence of epileptic activity. Recurrent polymorphic VT were documented when patient transferred to CVCU though she received MgSO4 intravenous therapy. Unfortunately, ICD implantation and sodium channel inhibitors could not be afforded for this patient due to health insurance problem. Based on patient's medical history, we administered phenytoin intravenously, which had class IB anti-arrhythmia property, combined with oral propranolol. Since that patient never had seizures and PVT episodes. Patient then discharge with therapy phenytoin 200mg b.i.d and propanolol 40mg t.i.d. Latest ECG showed sinus rhythm with QTc 580ms. One month 24-hours ambulatory ECG revealed frequent PVCs, prolonged QTc with average 561 ms and recurrent TdP. At that time patient didn't consume the drugs for 1 week. We educated patient to take the drugs routinely. 3 months later, 24-hours ambulatory showed no episode of TdP and QTc interval was shortened with average 480ms. Patient also never had seizure or fainted.

Conclusions:
Phenytoin could be used as alternative therapy to reduce ventricular arrhythmias in LQTS3 patient because of its sodium channel blocker properties.

KEYWORD: *phenytoin, LQTS3 , TdP.*

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Atrial Flutter 2:1 in 36 Years Old Male with Severe Mitral Stenosis and Congestive Heart Failure: A Case Report

A. Ciptasari[1] ,A. Novitasari[1] ,R. I. Rismawanti[1] ,R. Ayuningtyas[1] ,L. Pribadi[2] ,M. G. Suwandi[2]
[1]General Practitioner, RSPAU dr. Suhardi Hardjolukito, Yogyakarta, Indonesia
[2]Cardiologist, RSPAU dr. Suhardi Hardjolukito, Yogyakarta, Indonesia

Background: Patients with atrial flutter will eventually develop clinical AF, as atrial flutter frequently coexists with AF. Conditions such as mitral or tricuspid valve dysfunction, enlargement of the atria, especially the right atrium are linked to atrial flutter. The occurrence of AF in MS patients is correlated with the degree of valve obstruction and patient age, and it is the most frequent consequence of MS. When the ventricular rate is not adequately controlled, symptoms brought on by the loss of the atrial contribution to filling and by a brief diastolic filling period may be precipitated by or made worse by AF.
Case illustration: A 36-year-old male admitted to the ER with chief complain shortness of breath when walking a short distance in the last 4 days. Other symptoms were often waking up at night, swollen feet, and coughing with pinkish discharge with history of congestive heart failure. Patient's physical examination normal blood pressure and tachycardia with regular heartbeat. Rales were present in both lungs and murmur in mid-diastolic 3/6 mitral. The initial electrocardiogram showed atrial fibrillation 2:1, right axis deviation. Echocardiography showed dilatation of left atrium, right atrium, and right ventricle, mitral stenosis (MS) moderate-severe, tricuspid regurgitation (TR) severe with ejection fraction 55%. Laboratory findings were increased blood urea level, increased uric acid level and hypokalemia. The patient diagnosed with CHF CF III et causa severe MS, hypokalemia, and renal insufficiency. Treatments given for patient were furosemide 40mg injection/12 hours, spironolactone 1x25mg, digoxin 1x0,25mg, lansoprazole 30mg injection/12 hours, KSR 1x60mg, allopurinol 1x100mg, warfarin 1x2mg, bisoprolol 1x1,25mg. During treatment, patient's ecg changed from atrial flutter 2:1 to atrial fibrillation (AF) with rapid ventricular response then changed to atrial flutter 4:1. Patient was treated in IMCC for 4 days, later discharged and planned to get referred in tertiary hospital.

Conclusion:
Atrial flutter in this patient might be happened because remodeling of the cardiac structure. The remodeling is the result from high pressure on left atrium because mitral stenosis. The patient was treated with digoxin, bisoprolol, furosemide, and warfarin for his rate control, heart failure, and anticoagulant. Further treatment needed to consider ablation.

KEYWORD: *Atrial Flutter, Atrial Flutter 2:1, Mitral Stenosis, Heart Failure, Congestive Heart Failure.*

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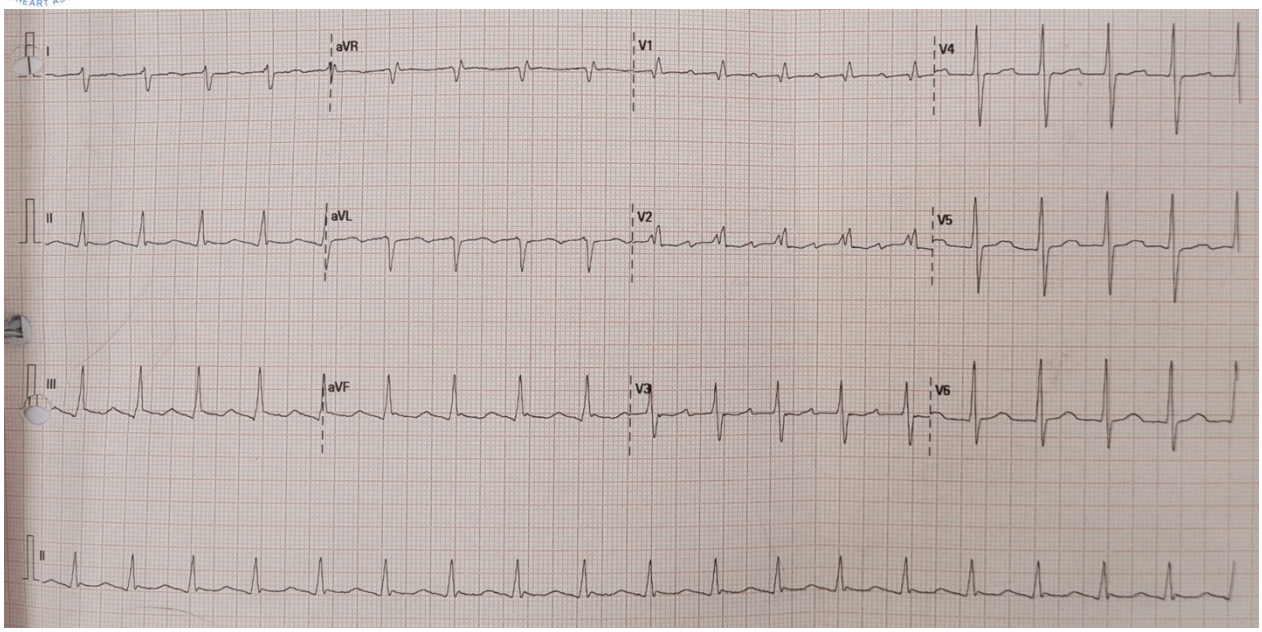

Figure 1 Patient's EGG

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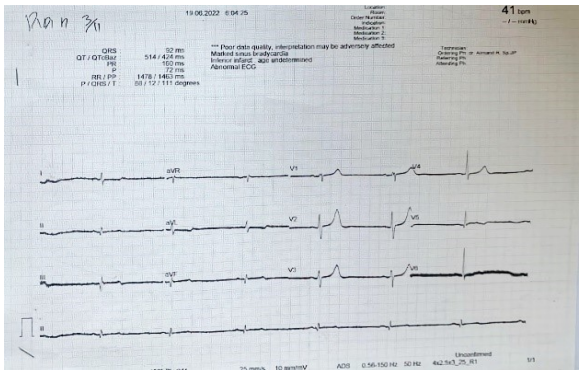
BRADYCARDIA INDUCED BY HYPERKALEMIA ON A RENAL IMPAIRMENT PATIENT

A. H. W. Ruhukail[1] ,T. Sugihharti[1] ,A. R. Ruhukail[2]
[1]General Practitioner RSUD 45, Kuningan, Jawa Barat
[2]Cardiologist RSUD 45, Kuningan, Jawa Barat

Background : Symptomatic bradycardia describes both a heart rhythm disturbance and the response to the disturbance. Hyperkalemia is known to cause cardiac irritability by increased resting membrane potential in the cardiac myocytes, impaired depolarization, and accelerated repolarization, resulting in a wide variety of dysrhythmias, including bradydysrhythmia, and ultimately resulting in cardiac arrest. The abnormal serum potassium levels (either high or low) often lead to myocardial instability and fatal arrhythmias. In this report we present a patient with bradycardia induced by hyperkalemia
Case illustration: A 52 year old male was admitted to our emergency departement with discomfort in the stomach, faint, and shortness of breath. Complaints lodge if engaged. He had a history of Coronary Heart Disease and NIDDM and an active smoker.. Blood pressure was 120/80 mmHg, heart rate was 41 bpm. The laboratory result showed ureum 141 mg/dL, creatinin 6.48 mg/dL, eGFR 8.5 mL, potassium 7.8 mmol/L, and NT-Pro BNP 7338 pg/mL. Electrocardiography (ECG) shown sinus bradycardia with Hyperacute T waves.. Patients were given Atropine sulfate (iv) 2x0,5mg but shown little increase in heart rate. After the laboratory result was shown, patient was given calcium gluconate and prepared for hemodialysis. The patient was dicharged from the hospital 6 days later without any problems.

Conclusions:
Patient with symptomatic bradicardya should be suspected of hyperkalemia. Extreme bradycardia ACLS algorhytm would give little benefit if there were no correction in potassium level. Correction in the potassium level into normal range convert ECG into sinus rhytm.

KEYWORD : *Bradycardia*, *hyperkalemia.*



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Sjögren's Syndrome Mimicking Ischemic Stroke After Catheter Ablation of Premature Ventricular Contraction Originating from Left Coronary Cusp

A. V. A. Ruhukail[1] ,D. Y. Hermanto[2] ,S. B. Raharjo[2] ,D. A. Hanafy[2] ,Y. Yuniadi[2]
[1]Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

[2]Staff of Arrhythmia Division,

National Cardiovascular Center Harapan Kita, Jakarta, Indonesia

Background: Sjögren's syndrome (SS) is a chronic progressive inflammatory autoimunne disease which characterized by the infiltration of lymphocyte of exocrine glands, with female predominance of 9:1. Peak incidence of SS at age of 50 years old. The coexistence of ptosis and diplopia in SS is rarely reported. To report a case of neurological involvement of Sjögren's Syndrome which mimicking ischemic stroke after catheter ablation of Premature Ventricular Contraction (PVC) originating from Left Coronary Cusp (LCC)

Case illustration: A 50-year-old woman with LCC origin PVC underwent catheter ablation at National Cardiovascular Center Harapan Kita. Patient experienced left sided ptosis and diplopia during first day admission after PVC ablation. The patient had Sjogren syndrome on her previous history and not taking corticosteroid for 1 months. Brain MSCT showed lenticular ischemic intracerebri and intracerebelli which had no relation with neurologic manifestation on this patient. The patient was referred to internist and neurologist in our hospital. Ptosis and diplopia was recovered after administration of intravenous corticosteroid. She refused to continue further laboratory examination. Therefore, we considered to refer the patient to divison of Allergy, Immunology and Rheumatology for further comprehensive management.

Conclusions:

The coexistence of neurological involvement of SS could be mimicking ischemic stroke in patient who underwent PVC's catheter ablation. Early diagnostic and further laboratory examination should be done for a more comprehensive management.

KEYWORD: *Sjögren's syndrome, LCC origin PVC, Catheter ablation, Ptosis, Diplopia.*

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Electrical Catastrophic in Mitral Annular Dysjunction

A. Z. Abidin[1] ,A. N. Sulma[1] ,A. R. Andini[1] ,P. Ardhianto[2]
[1]Faculty of Medicine, Diponegoro University, Semarang, Indonesia
[2]Department Cardiology and Vascular Medicine, Kariadi Hospital, Semarang, Indonesia

Background: Mitral Annular Dysjunction (MAD) describes as a structural abnormality where there a distinct separation of the mitral valve annulus and left atrial wall continuum and the basal portion of posterolateral ventricular myocardium. There was an increased frequency of Premature Ventricular Complex (PVC) and Non Sustained Ventricular Tachycardia (NSVT) in patients with MAD, and it has been associated with Sudden Cardiac Death (SCD). A prompt diagnosis, medication and device implantation can lower patient risk to SCD

Case illustration: A 28 years-old woman came to hospital with a history of syncope. When syncope occurred, it was preceded by a tachycardia. Physical examination in within normal limit. ECG showed sinus rhytm and a PVC with RBBB morphology and superior axis. Echocardiography showed good LV function,mild MR and MAD with a distance about 0.85 mm. An Electrophysiology Study were performed, when we do the burst RV pacing a non sustained polymorphic VT was being induce, and we are able to reset the rhytm back to sinus rhytm. Patient was diagnosed as Mitral Annular Dysjunction Arrhytmic Sindrome. Then the patient we give an oral bisoprolol 2.5mg/24H and educate the patient for the probability of implantation of Implantable Cardioverter Defibrillator (ICD).

Conclusions:
MAD is a structural abnormality that have an arrhytmogenic entity ranging from PVC to SCD. The symptoms that associated with MAD are palpitations, presyncope, syncope and ventricular arrhytmia. MAD can be easily detected on echocardiography and other imaging modality. If a patient had no other apparent cause of PVC we should do an echocardiography study to exclude the possibility of MAD. There are some treatment of choice for the ventricular arrhytmia ranging from usage of betablocker, ICD implantation, ablation at the ectopic foci and surgical MV repair of replacement. We should do proper management in patient with MAD to prevent SCD.

KEYWORD: *MAD, Polymorphic VT, SCD.*

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Asymptomatic Pacemaker Malfunction due to Twiddler Syndrome in Transient Total AV-Block Patients: Silent, Rare, but Serious Case

A. Yutha[1] ,R. Julario[2] ,B. Baktijasa[2] ,R. Amadis[2]
[1]*Faculty of Medicine Universitas Airlangga, Surabaya, Indonesia*
[2]*Department Cardiology and Vascular Medicine, Dr. Soetomo General Hospital, Surabaya, Indonesia*

Background: Twiddler's syndrome is an infrequent cause of permanent pacemaker malfunction due to the patient's manipulation of the pulse generator, in which twisting or rotating of the pacemaker generator device in its pocket. Leading to traction and lead dislodgement resulting in failure of ventricular pacing. It can be a lethal complication in pacemaker dependent patients, thus an urgent repositioned is warranted. In this report, we present a patient with Twiddler's syndrome.

Case illustration: An 65 year old lady who underwent a single chamber ventricular pacemaker (VVIR) implantation with the indication of transient complete heart block and syncopal hystory, 2 months later she come to our clinic for scheduled pacemaker interogation and programming with no chief complaint. Patient heart rate was 76 bpm, blood pressure was 120/70 mmHg. Progamming pacemaker showed the impedance was very high (3000 ohm). Immediate X-Ray was taken showing retraction of ventricular lead from subclavian vein and wrapped repeatedly arround the pulse generator, with its tip now positioned over the major pectoral muscle (Figure 1). Patient denied in pivotting her right arm and controlling the pacemaker after embedded pacemaker. Patient can follow order and no history of psychiatric disease, but one of the family member told us that the patient occassionally had body massage. We performed to extract the pulse generator and its lead. The ventricular lead was repositioned and placed at right ventricular apex. The pocket was made within the subpectoral muscle for adequate fixation.

Conclusions:

Twiddler's syndrome potentially lethal complication in pacemaker dependent patients. X-ray is a easily-available investigation that will rapidly clinch the diagnosis. Readmission and lead repositioning are always required. Management options for preventing or treating twiddling may include fixating the device at pectoral fascia, creating a smaller pacemaker pocket, anchoring the device into tightly fitting subpectoral pocket, and proper education for patient and family.

KEYWORD: *Twiddler's syndrome, permanent pacemaker, dislodge.*

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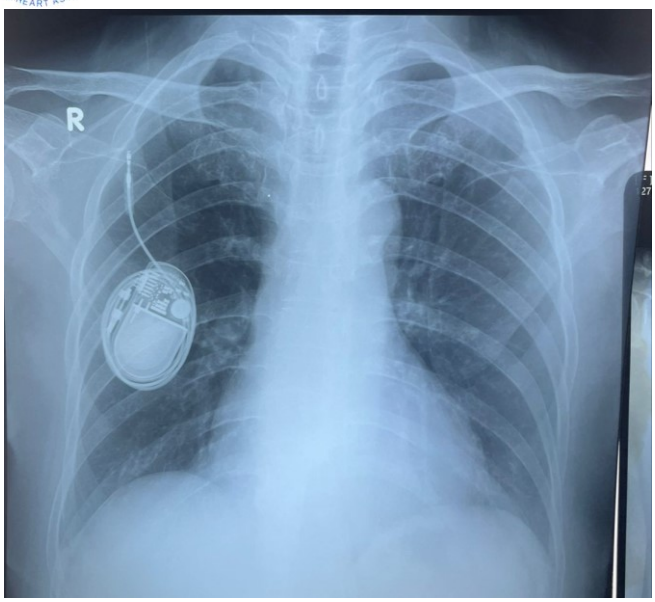

Figure 2. Posteroanterior chest X-Ray showing retraction of the lead and wrapped repeteadly around generator

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WPW Syndrome Presented with Ventricular Tachycardia as a Predictor of SCD : A Case Report

A. Putri[1] ,P. Utomo[2]
[1] General Practitioner, Cibabat General Hospital, West Java, Indonesia
[2] Cardiologist, Cibabat General Hospital, West Java, Indonesia

Background: Ventricular tachycardia (VT) is life-threatening that can occur for many reasons. This was a fatal case of 55-year-old male who presented with VT without any known illness which can lead to sudden cardiac death (SCD). After medical cardioversion was started his ECG converted to WPW syndrome. A Specific treatment procedures such as electrophysiological study (EPS) may be considered for differential diagnosis and curative treatment. The incidence of SCD in patients with WPW syndrome has been estimated to range from 0,15% to 0,39% over 3 to 10-year follow-up.[1] In some patients the first manifestation of WPW syndrome is SCD presumably due to Ventricular tachycardia (VT) or Ventricular fibrillation (VF).

Case illustration: A 55-year-old male was admitted to the emergency department with sudden onset of palpitations and syncope. He didn't give any medical history or family history of SCD. His HR was 206 bpm and BP 100/70 mmHg. ECG showed VT, but hemodynamically stable. We diagnosed the patient with VT hemodynamically stable. Medical cardioversion was started with 300 mg amiodarone to the patient based on Adult Cardiac Life Support (ACLS) algorithm. Within 1 hour ECG was converted to narrow QRS complexes followed by P waves and a longer PR interval, delta wave was positive. ECG findings are suggestive of WPW syndrome. After 3 days of hospitalization, his signs and symptoms decreased. Then the patient was transferred to EPS Center for further treatment. Many conditions can cause VT including WPW syndrome. WPW syndrome refers to the presence of an overt manifest accessory pathways. The ECG shows a short PR interval (≤120 ms), slurred upstroke (or downstroke) of the QRS complex (delta wave), and a wide QRS complex (>120 ms).[4] Studies of WPW syndrome patients who have experienced a cardiac arrest have retrospectively identified a number of markers that identify patients at increased risk. These include a shortest pre-excited R-R interval less than 250 ms during spontaneous or induced AF, a history of symptomatic tachycardia, multiple accessory pathways, and ebstein's anomaly.[2] The detection of intermittent pre-excitation,which is characterized by an abrupt loss of the delta wave and normalization of the QRS complex, is evidence that an accessory pathway has a relatively long refractory period and is unlikely to precipitate VF.[2] Life-saving interventions are a priority in acute treatment. In this case, an IV bolus of amiodarone could selectively decrease conduction through the bypass tract relative to the atrioventricular node, resulting in a break in the rhythm.[3] Indications for referral to a cardiac arrhythmia specialist include the presence of wide complex tachycardias, drug resistance, or intolerance. Because of the potential for lethal arrhythmias all patients with WPW syndrome should be referred for further evaluation.

Conclusions:
WPW syndrome can present as a life-threatening rhythm such as VT. It requires immediate intervention. Several non invasive and invasive tests have been proposed as useful in risk stratifying patients for SCD risk.

KEYWORD: *WPW Syndrome*, ***Ventricular Tachycardia (VT).***

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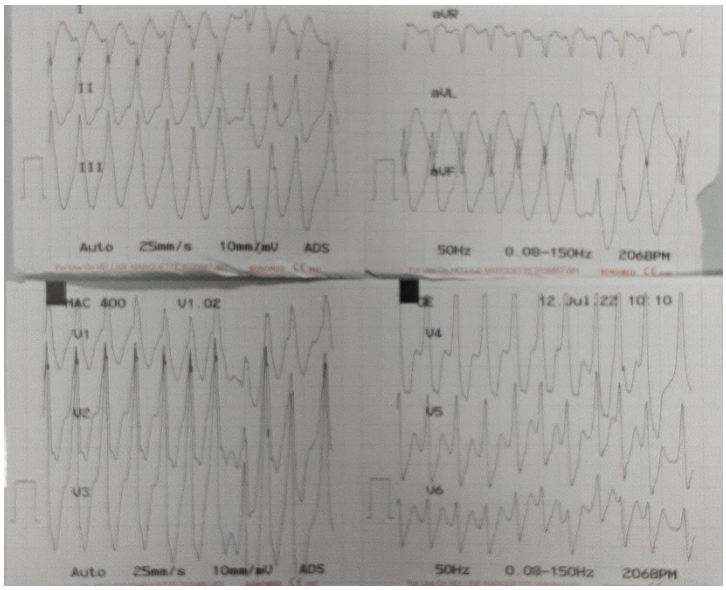

FIGURE 1 A 12-lead ECG showing Ventricular Tachycardia

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Brugada Type 1 Electrocardiographic Pattern in Dengue Fever Patient: A Case Report

A. Kristian[1]
[1]*General Practitioner, Cempaka Putih General Regional Hospital, Jakarta*

Background: Brugada syndrome (BS) is a channelopathy with a characteristic electrocardiogram (ECG) (ST-segment elevation of ≥2 mm with a coved-type morphology in ≥1 right precordial lead) and an increased risk of sudden cardiac death (SCD), in the absence of gross structural heart disease. The Brugada-type ECG pattern (BTEP) can be augmented by some things or events like drugs, ischemia, and fever. Brugada syndrome triggered by fever is known to predispose to malignant ventricular arrhythmias.

Case illustration: 59-year-old man patient presented to emergency department with fever one day before admission associated with myalgia, arthralgia, dizziness and vomiting. Patient also had a syncopal episode at home but denied any significant cardiovascular disease including family history of sudden death. Patient also said that he had twice syncope events when he was 40 tears old. Physical examination including vital signs was unremarkable except for lethargy and dehydration with temperature of 39^{O}C. Full blood count (FBC) showed thrombocytopenia of platelet count 88.000/mm3, haematocrit 40%, haemoglobin 13,9 g/dL and total white blood count (WBC) 11.100/mm3. Liver function test showed transaminitis with alanine aminotransferase (ALT) of 65 unit/L and aspartate aminotransferase (AST) of 64 unit/L.

ECG showed a “coved type” ST elevation and T wave inversions in leads V1 and V2 characteristic for BTEP Type I (Fig.1). Patient was treated as dengue fever in febrile phase with transaminitis and BTEP Type 1. Injection of Paracetamol 1 gram three times daily was administered for temperature control and 2000 ml of normal saline (NS) daily was given as per local dengue protocol. After six days of hospitalization, patient showed clinical improvement with no recurrence of syncopal episodes or documented arrhythmias. He was discharged with repeated ECG during defeverscence phase showed evolution to Type 2 pattern with saddle-shaped ST elevation appearance at V2 with positive T wave (Fig. 1). Brugada syndrome was initially described in 1992 with the first definition for several years known as the syndrome of right bundle branch block, persistent ST segment elevation and sudden death[1]. In 1996, several reports, mainly coming from Japan, started calling the syndrome Brugada syndrome[2]. Brugada syndrome (BS) is as a channelopathy with a characteristic electrocardiogram (ECG) (ST-segment elevation of ≥2 mm with a coved-type morphology in ≥1 right precordial lead) and an increased risk of sudden cardiac death (SCD), in the absence of gross structural heart disease[3]. BTEP induced by fever has been associated with factors such as younger age, male gender, and the effect of temperature on mutant type sodium channels which lead to sodium current reduction hence delaying conduction[4]. Thus, with higher degree of fever, the greater the imbalance of ionic current occurs, leading to deeper notch in action potential which further triggers ventricular arrhythmias. BTEP in fever increases incidence of cardiac arrest with higher prevalence among Brugada pattern Type 1 compared to Type 2[5]. Fever-triggered cardiac arrest can be prevented by early control of hyperthermia[5]. BTEP and BrS in dengue fever or dengue haemorrhagic fever are under reported as ECG is not routinely done for febrile patients. Diagnosis of dengue was confirmed by positive finding in dengue serology test where unfortunately had not been done in this case, and we did no genetic testing as well. Our management concentrated on temperature control and hydrating the patient. With acetaminophen and hydration as per dengue protocol, the temperature was controlled with subsequent evolution of BTEP Type 1 to Type 2.

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Conclusions:
ECG changes in BrS and can be augmented by dengue fever. ECG should be done to all febrile patients who has previously syncopal episodes or history of relatives' SCD for early detection of BrS. Patients with BTEP Type 1 induced by fever are at risk of developing BrS. The possibility of ventricular tachyarrhythmias and long-term clinical outcome remain unknown in this patient and requires a well-designed cohort studies to be elucidated. The goal of treatment is prevention of malignant arrhythmias, hence quick and effective control of temperature in fever induced BTEP is essential.

KEYWORD: *Brugada syndrome, Dengue fever, Brugada-type ECG pattern, Sudden Cardiac Death.*

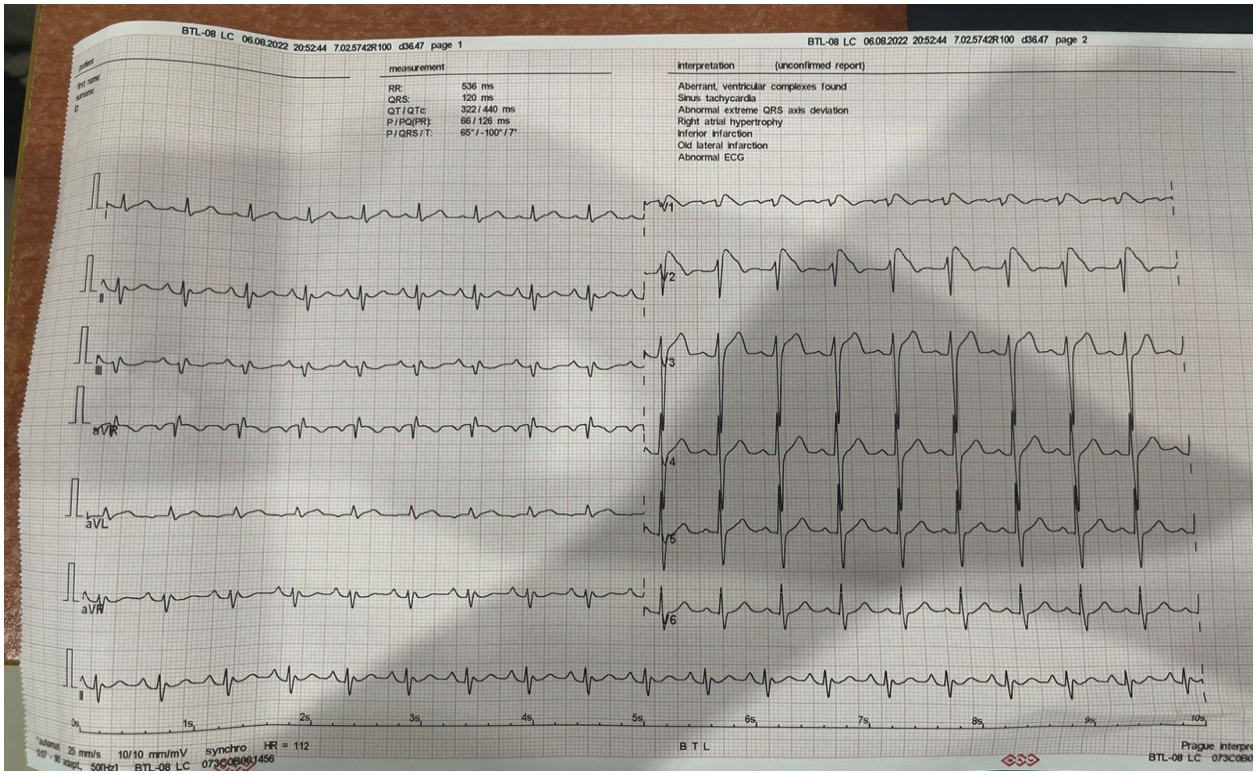

Fig. 1. (Above) First ECG on admission showing BTEP type I, coved ST segment elevation >2 mm in V1 & V2 with negative T wave. Temp: 39^{O}C. (Below) ECG after 6 days of admission showing BTEP type 2, saddleback shaped ST elevation at V2.

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Succesfully terminated AVNRT after oral administration of bisoprolol and digoxin at rural hospital: a case report

C. Nency[1] ,A. Kurnia[2]

[1]*General Practioner, Puskesmas Pulau Merbau, Riau, Indonesia*

[2]*General Practioner, RSUD Kabupaten Kepulauan Meranti, Riau, Indonesia*

Background: Atrio-ventricular nodal re-entrant tachycardia (AVNRT) is a type of supraventricular tachycardia (SVT) due to presence of a re-entry circuit at the AV node. Beta blocker as monotherapy is used to recommended for acute treatment of AVNRT. However, evidence for using combination of beta blocker especially bisoprolol and digoxin are still limited.

Case illustration: 66-years-old female presented to ER with sudden onset of palpitation lasting 1 hour. It wasn't associated with chest pain, shortness of breath, orthopnea, or syncope. She had history of hypertension and dyslipidemia. Vital signs were BP 123/65, pulse 166, RR 22, temp 36.7, SpO2 99% with normal in general physical examination. 12-leads ECG showed regular narrow complex tachycardia with pseudo R' in lead V1 that suggest an AVNRT (Figure 1). Beta blocker and digoxin are well known as medical treatment for SVT. However, the use of this combination is still rare. In this patient, we observe that combination of these drug had successfully terminated the SVT. This is possibly due to both of drugs had similar effect. Those can slowing electrical conduction by increasing the refractory periode of AV Node.

Conclusions:

Both of Bisoprolol and Digoxin can be considered as an alternative medical treatment to terminated AVNRT.

KEYWORD: *AVNRT, Beta blocker and Digoxin, SVT.*

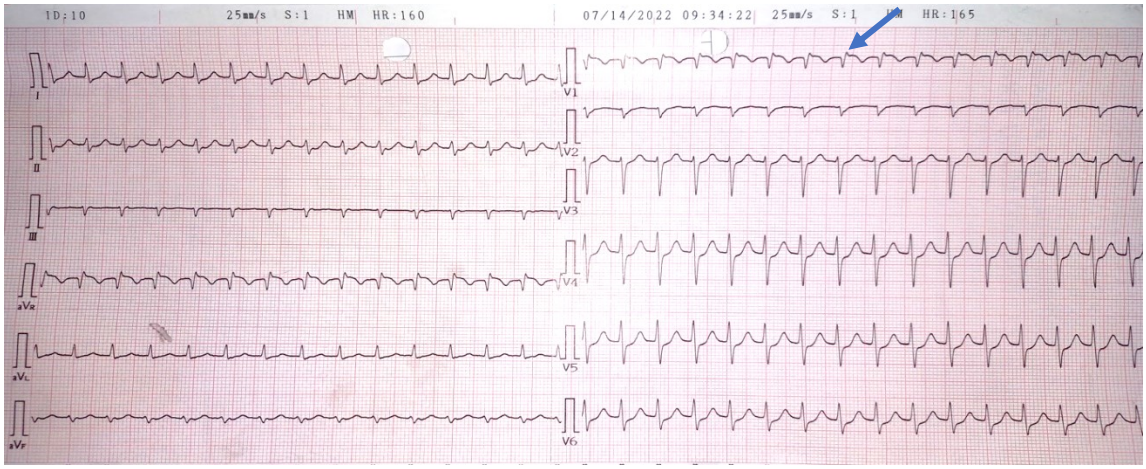

Figure 1 SVT with Psuedo R' in lead V1 (AVNRT)

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Case Report: Ramsay Hunt Syndrome and Bradycardia, Is It Related?

A. Gunawan[1] ,M. L. Peta[2] ,O. Litanto[3]

[1]General Practitioner, Subang General Hospital, Subang, Indonesia

[2]General Cardiologist, Subang General Hospital, Subang, Indonesia

Background: Ramsay Hunt Syndrome (RHS) is caused by varicella-zoster virus (VZV), the same virus that causes varicella in children and herpes zoster in adults, and consists of a severe facial palsy associated with a vesicular eruption in the external auditory canal, pharynx and other parts of the cranial integument. RHS may involve other cranial nerves but rarely occur. Paralysis of the vagus nerve is associated with swallowing difficulties or vasovagal reactions, such as cardiac arrhythmia. We report a patient of RHS with symptomatic bradycardia.

Case illustration: A 62 years old male came to the Emergency Department with a history of syncope. The patient reported facial paralysis, and a painful vesicular rash on his left external ear about 1 week before admission, and has been treated with acyclovir 4x800mg. The patient was fully aware, with no signs of shock, but hypotensive and bradycardic. Left peripheral facial nerve palsy and a few crusts in the left external auditory ear canal were seen. Hematology studies within normal limits. ECG showed sinus bradycardia (44 beats/minute). Echocardiogram showed normal LV function without hypokinesis. The patient was diagnosed with “Symptomatic Bradycardia and RHS”, and was treated with 1mg intravenous atropine sulfate, oral prednisone 2x10mg, acyclovir 4x800mg, and carbamazepine 2x200mg. Sinus rhythm at 75 beats/minute was achieved after given atropine sulfate. The patient showed no bradycardic episode during hospitalization and was discharged after 3 days of observation.

Conclusions:

Several case reports had reported bradycardia events because of the infection of VZV. In this case, bradycardia is caused by two different possibilities, specifically the virus, and the acyclovir itself. Although, the pathogenesis of this condition remains unclear, and the treatment itself (acyclovir) has been reported to its unusual side effect: bradycardia episode, we suggest keeping acyclovir as a possible precipitating factor for the evaluation of bradycardia and suspect VZV to be the autonomic disturbances.

KEYWORD: *Ramsay Hunt Syndrome, varicella-zoster virus, acyclovir, bradycardia.*

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Case Report: Complete Heart Block in Young Adult Caused by Systemic Lupus Erythematosus

I. D. G. S. M. Badung[1] ,I. M. P. S. Antara[2] ,A. Setiawan[3]
[1,3] Cardiology resident Sanglah General Hospital Bali
[2] Cardiologist Sanglah General Hospital Bali

Background: Rhythm and conduction disorders are important manifestations of cardiac involvement in autoimmune diseases. Autoimmune diseases have been associated with advanced AV conduction abnormalities. In most conditions, AV block occurrence is preceded by many signs or symptoms, but, in some circumstances, complete heart block may be the first manifestation of the disease. AV block may also be a consequence of systemic lupus erythematosus (SLE), and few cases of complete AV block as the first manifestation of adult lupus have been previously described. The small vessel vasculitis and the infiltration by fibrous tissue are significant causes of the dysfunction of AV nodes in SLE. We present a case 38 years old man with a complete heart block caused by SLE.
Case illustration: A 38 years old man came to the emergency department with a history of syncope. He complained of chest pain before a loss of consciousness. It is accompanied by pain in both knee joints for one month. He also had a fever for one week. There was no dyspnea or palpitation. There is no history of past illness or family history. BP: 131/71 mmHg, HR: 48x/min, RR: 20x/min, T: 37,5°C. Physical examination showed a malar rash with no cardiac abnormality. Chest X-ray showed cardiomegaly but normal Echocardiography. The ECG showed that it was in complete heart block. We tested him for cardiac enzyme and ANA. The ANA test was positive, and the troponin was negative. So, we gave him SLE therapy and inserted a temporary pacemaker (TPM) for his complete heart block. After ten days of therapy, we turned off the TPM, and the rhythm showed First-degree AV Block. The pathophysiology of arrhythmias in SLE includes initial inflammatory cell infiltration, myocardial necrosis, and fibrotic replacement. It can affect atrial and ventricular conduction, leading to cardiac rhythm disorders.

Conclusions:
One possible etiology for complete heart block in young adults to remember is autoimmune diseases.

KEYWORD: *SLE, autoimmune diseases, AV block.*

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Persistent Narrow Complex Ventricular Tachycardia: How to Approach?

A. Y. Putranto[1] ,H. A. Panjaitan[1] ,W. A. Widodo[2]
[1]General Practicioner, Jakarta Heart Center, Jakarta, Indonesia
[2]Cardiologist, Jakarta Heart Center, Jakarta, Indonesia

Blackground: Ventricular tachycardia (VT) is classically characterized by a rapid heart rate with a wide QRS complex (QRSd) duration of more than 140 ms. In some cases of VT, the QRSd is relatively narrow (120-140 ms) which is often misinterpreted.[1] Various algorithms have been proposed to help differentiate VT from SVT with deviations in the form of wide-complex tachycardia, while narrow-complex tachycardia is almost always considered an SVT. [2]

Case illustration: A 46-year-old male presented to the ER with palpitations and shortness of breath since 2 days ago. He looked fully alert with BP 82/61, HR 150 bpm, RR 16/min, with normal PE. A 12-lead ECG showed tachycardia 152 bpm, regular with relatively narrow complexes (QRSd 118 ms), LAD and dissociated P waves in Lead II. He was given 6 mg and 12 mg doses of adenosine I.V and later amiodarone I.V 150 mg/10 minutes, without any success. The laboratory examination showed increased Troponin I (0,233 ng/mL) and mild hypokalemia (3,27 mEq/L). The patient was admitted to HCU with continuous amiodarone I.V. Corangiography showed normal coronaries. Tachycardia persists for another day and electrical cardioversion was performed twice, with 100 joule and 150 joule synchronised, but also unsuccessful. On the third day 1mg/kg lidocaine I.V was given, HR was converted to sinus 12 hours later. TTE revealed a normal LVEF of 55% with no significant valvular abnormalities. Tachyarrhytmia is divided based QRSd into narrow-complex (QRSd< 120 ms) and wide-complex (QRSd>120 ms). The Brugada Criteria are commonly used to determine whether a wide complex tachycardia is from VT or SVT with aberrancy. Criteria include in this case are RS interval > 100 ms, the presence of AV dissociation and RBBB morphology criteria showed the diagnosis leads to VT. In this case the patient surface ECG didn't show typical form of VT because it has relatively narrow complex QRS so its often misdiagnosis as a SVT. (*Figure 1*)

Conclusions:
The diagnosis of narrow-complexes VT is difficult due to the relatively narrow complexes (QRSd 110-140 ms). Evidence of AV dissociation is one useful characteristic to diagnosis these kind of tachyarrhytmia.

KEYWORD: *Ventricular Tachycardia, Narrow-Complex Ventricular Tachycardia*.

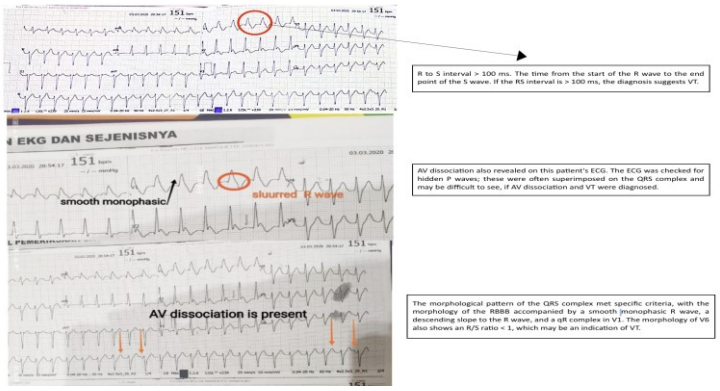

Figure 1. ECG showed RS interval > 100 ms, the presence of AV dissociation and RBBB morphology criteria showed the diagnosis leads to VT

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Hypersensitivity Caused by Conductor Material Mimicking Cardiovascular Implantable Electronic Device (CIED) Infection: A Rare, but True Phenomenon

B. A. Pratama[1] ,E. Maharani[1] ,F. Hidayati[1] ,D. P. Mulya[2] ,D. Priambodo[2] ,F. Waskito[3]
[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Gadjah Mada, Yogyakarta, Indonesia
[2]Department of Internal Medicine, Faculty of Medicine, Universitas Gadjah Mada, Yogyakarta, Indonesia
[3]Department of Dermatovenereology, Faculty of Medicine, Universitas Gadjah Mada, Yogyakarta, Indonesia

Background: Allergic contact dermatitis caused by CIED implantation was a rare condition. This condition is being not detected and being misdiagnosed such as in CIED Infection.
Case illustration: A 70 years old male with heart failure reduced ejection fraction (HFrEF) New York Heart Association (NYHA) III due to dilated cardiomyopathy with no coronary artery disease. Electrocardiography showed sinus rhythm with left bundle branch block and QRS duration was 200 ms. Echocardiography showed LV dilatation with EF 26%. Patient still symptomatic despite of guideline directed medical treatment (GDMT). During follow up sinus node dysfunction was found. Implantation of cardiac resynchronization therapy-pacemaker (CRT-P) was performed. Five months after the procedure, erythema and itching around pacemaker wound were complained. Five days later wound get worsen with purulent pus with the suspicious of pacemaker infection. Blood, wound, and pus culture were taken preceded antibiotic therapy and it showed no microbial colonization. The holistic management was conducted through collaboration care among tropical medicine, allergy immunology and dermatovenereology department. The patch test revealed Nickel (II) Sulphate Hexahydrate and Potassium Dichromate which was conductor material in device lead as allergen. The CRT-P system was explanted, and dual chamber pacemaker was implanted. Evaluation showed improvement of symptom. We reported an allergic contact dermatitis due to metallic component in CIED lead which causing desensitization hypersensitivity process.

Conclusions:
Allergic contact dermatitis was delayed type of hypersensitivity caused by generator or metallic and synthetic material in lead component. Removal of the device and replacement with another model that not containing documented allergens is the best treatment.

KEYWORD: *Allergic Contact Dermatitis, CIED, Nickel (II) Sulphate Hexahydrate, Potassium Dichromate.*

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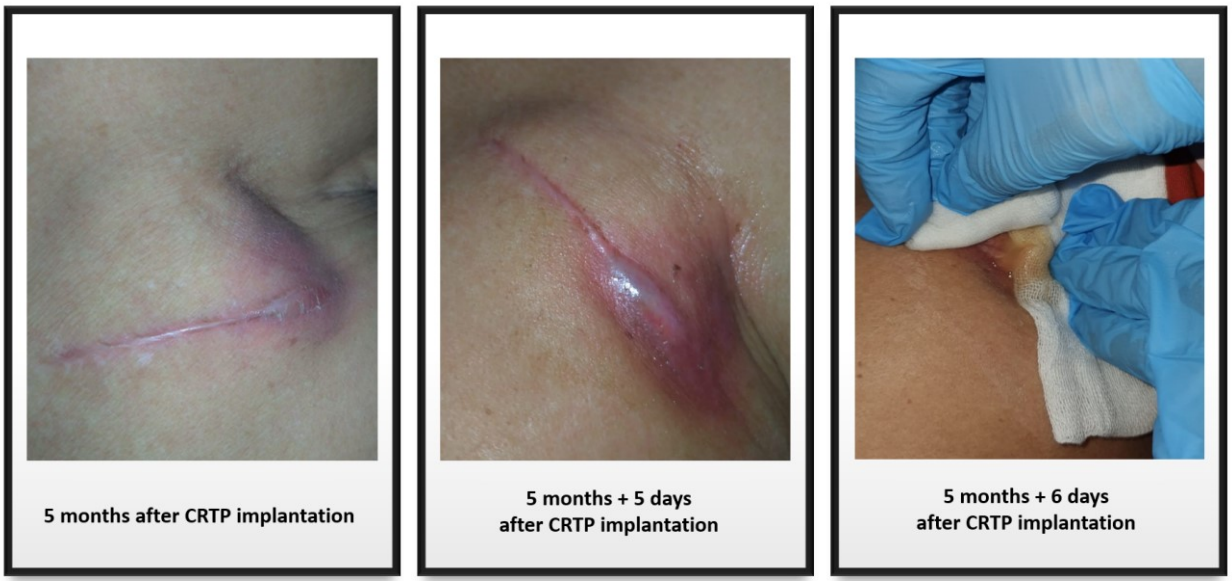

Figure 1. Left: skin feature showed linier hypertrophic scar with mild erythema in surrounded wound at 5 months after CRT-P implantation, Middle: 5 days later it showed bullae at the edge of wound, Right: 6 days after the bullae disrupted and purulent discharge was productive.

Table 1. Patch Test Using CIED Materials Showed Nickel (II) Sulphate Hexahydrate and Potassium Dichromate as Allergen

Alergen	48 Hours	72 Hours	96 Hours	120 Hours	144 Hours	168 Hours	218 Hours	Reults
Generator Pacemaker (X)	-	-	-	-	-	-	-	Negative
Generator Pacemaker (Y)	-	-	-	-	-	-	-	Negative
Potassium dichromate	+/-	+	+	+	+	+	+	A (Past)
Cobalt (II) chloride hexahydrate)	+/-	+/-	+/-	+/-	+/-	+/-	+/-	Negative
Colophonium	-	-	-	-	-	-	-	Negative
Nickel (II) Sulfate hexahydrate	+	+	++	++	++	+	+	A (Past)
Epoxy resin	-	-	-	-	-	-	-	Negative
Toluenesulfonamide formaldehyde resin	-	+/-	-	-	-	-	-	Negative
Mercaptobencothiazole	-	-	-	-	-	-	-	Negative
Thiuram Mix	-	-	-	-	-	-	-	Negative

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Successful Management of Refractory Torsades de Pointes: Limited Data, Limited Time

B. W. Jhundy[1] ,R. M. Prihatiningrum[2] ,S. A. Sugiman[3] ,A. Sain[4]

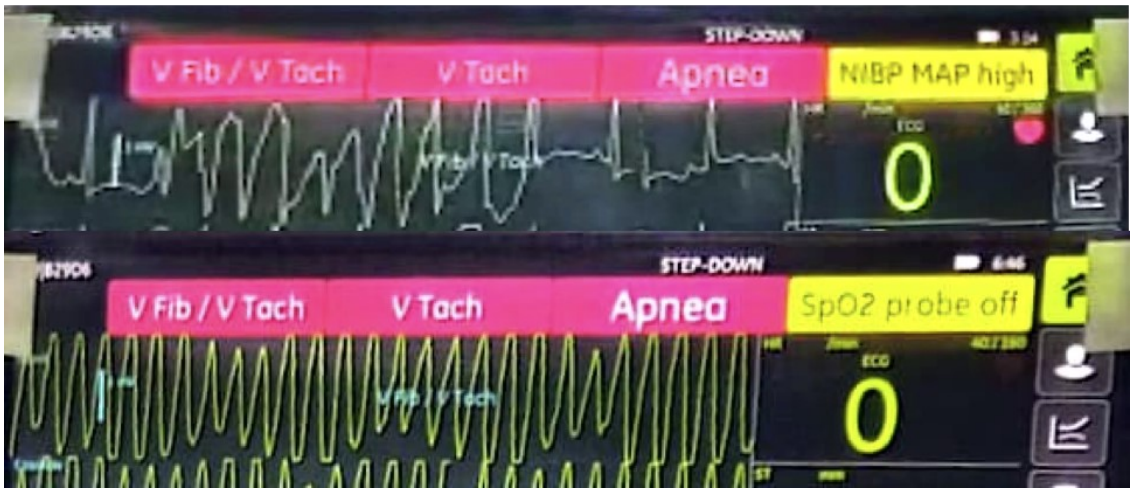
[1,2,3]General Practitioner, Mitra Plumbon Indramayu Hospital, West Java, Indonesia; [4]Cardiologist, Mitra Plumbon Indramayu Hospital, West Java, Indonesia

Background: Torsades de pointes (TdP) is a polymorphic ventricular tachycardia associated with prolonged QT interval, which can be acquired or congenital. TdP can progress to ventricular fibrillation and cardiac arrest, thus aggressive clinical management is needed.
Case illustration: We present a case of a 50 year old female patient who came to the emergency department with main symptoms of multiple syncope and a history of vomiting prior to syncope, she also had a history of congestive heart failure without signs of dyspnea or chest pain during admission. Admission electrocardiogram (ECG) showed bigeminy premature ventricular complex. Laboratory examination showed moderate hypokalemia (K: 2,7 mmol/L) and mild hyponatremia (Na: 133), other electrolytes were not available. She received KCl for potassium correction and was admitted to the intensive care unit (ICU) for continuous monitoring. In ICU, she had repeated pulseless TdP that rapidly resolved without electrical cardioversion and strip ECG showed prolonged QT interval of 563 msec (corrected QT interval with Fridericia formula) and prominent U wave. We administered 1 gram of intravenous magnesium sulphate and ca gluconas despite unavailable data. Evaluation ECG showed reduced QTc to 531 msec, corrected potassium increased to 3,9 mmol/L, she had no further TdP or syncopal episode and was discharged within two days.

Conclusions:

In our case, it is important to remember that magnesium and calcium examination may not always be available, and correction may be necessary. Administering magnesium and calcium start with low dose and continuous observation for the signs of hypermagnesemia and hypercalcemia can be life-saving.

KEYWORD: *Torsades de Pointes; prolonged QT; magnesium; calcium; kalium.*



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An atypical case of preexcitation: Nodofascicular accessory pathway with atrial fibrillation and early repolarisation

C. J. Hardi[1] ,D. A. Hanafy[1,2]

[1]National Cardiovascular Center Harapan Kita, Jakarta, Indonesia

[2] Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

Background: Nodofascicular (NF) and nodoventricular (NV) are uncommon accessory pathways (AP) that originate from the atrioventricular node (AVN) and insert to the right bundle branch or ventricular myocardium.[1] These NF/NV APs can participate as the retrograde limb of orthodromic Atrioventricular Reentrant Tachycardia.[2] Atrial Fibrillation (AF) is prevalent in 30% of patients with Wolff-Parkinson-White (WPW) syndrome.[3,4] Early Repolarisation (ER) syndrome has also been previously reported in WPW syndrome.[5]

Case illustration: A 34-year-old male was referred to the arrhythmia clinic for incidentally discovered delta waves during a treadmill test. 12-lead ECG showed sinus rhythm with delta waves suggesting right posteroseptal origin AP and slight J-point elevation in V3-4. Wide QRS tachycardias and multiple episodes of AF were documented in Holter monitoring, with various degrees of preexcitation. Echocardiography showed no structural heart disease. Electrophysiological study (EPS) was performed with two quadripolar catheters placed at HRA/HIS and RV respectively, and one decapolar catheter was placed into the CS via the right femoral vein. Baseline A-H interval was 69 ms, and the H-V interval was 5 ms with preexcitation. Ventricular Pacing showed Retrograde Block at S1 500 ms. Programmed atrial stimulation was unable to induce SVT with and without Isoproterenol infusion. Sustained AF occurred and terminated spontaneously after the effects of Isoproterenol subsided. SNRT showed SA node dysfunction, following AF termination SNRT was 2200ms. Continuous pacing at 400ms showed maximum preexcitation which changed to AVN conduction after reaching AP refractory. His Potential was recorded at the Right Posteroseptal area. Programmed ventricular stimulation (500S4 from VA and reproducible with 400S3 at RVOT) was able to induce hemodynamically unstable polymorphic VT which degenerated into VF and was terminated with 200J DC Shock. Due to the decremental nature of the AP and presence of reproducible lethal ventricular arrhythmias, EPS was not continued with ablation of the AP.

Conclusion:

Nodofasicular/Nodoventricular APs are uncommon yet important mediators during pre-excited AF. ER Syndrome may also occur in WPW syndrome, however masked by ST-segment depression secondary to the pre-excited QRS complex. Lethal ventricular arrythmias can occur in WPW patients not just by preexcited AF, but ER syndrome.

KEYWORDS: *Nodofascicular Pathway; Preexcitation syndrome; Ventricular Arrhythmias; Atrial Fibrillation.*

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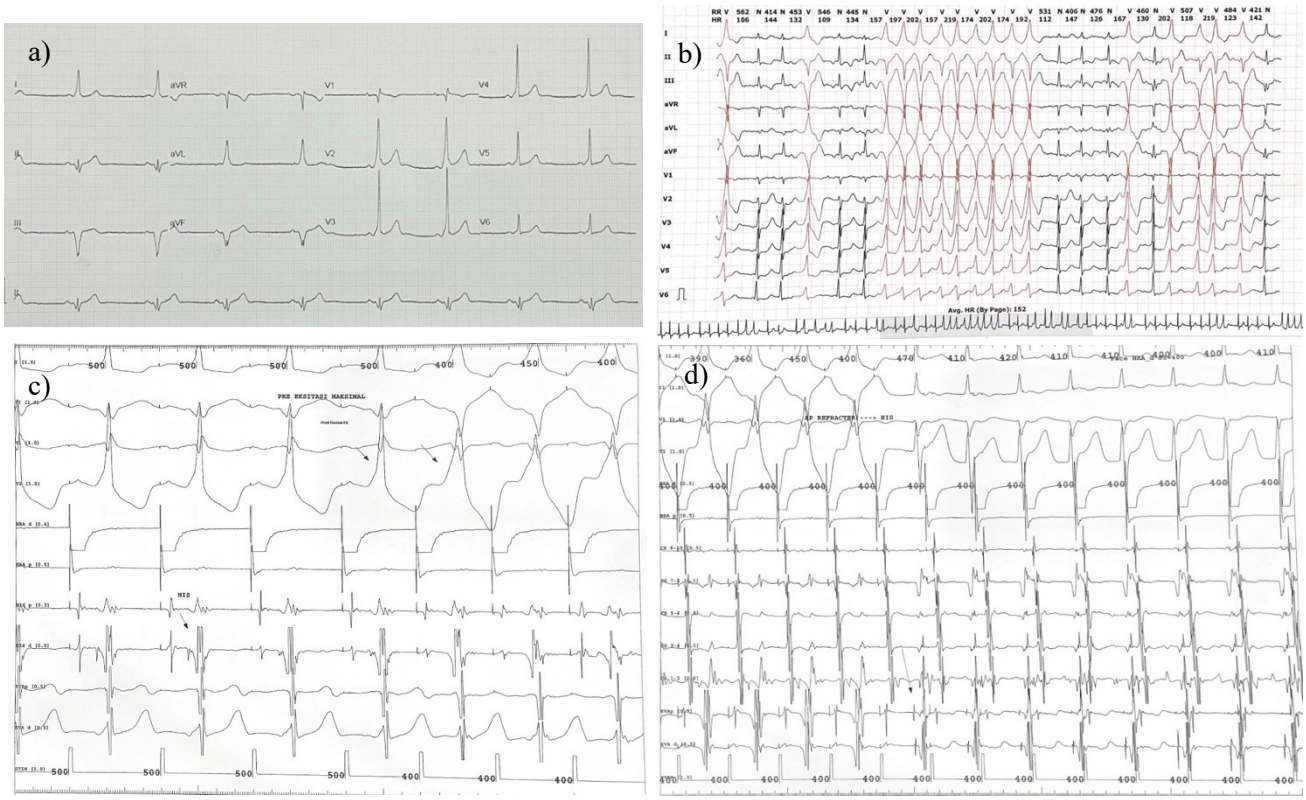

Figure 1 – 1a) Baseline 12-lead ECG showing preexcitation with notable delta waves and slight J-point notching at V3-4; 1b) Holter monitoring showing multiple wide complex tachycardias during atrial fibrillation rhythm; 1c) Maximum preexcitation at continuous atrial pacing S1 500 ms, His potential recorded at right posteroseptal region; 1d) Continuous pacing at 400 ms showed maximum preexcitation which then changed to AVN conduction after reaching AP refractory.

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His Bundle Pacing as Alternative Strategy to Cardiac Resynchronization Therapy in Chronic Heart Failure

C. A. A. Rosmaningtyas[1] ,B. M. Setiadi[1] ,J. A. Pangemanan[1] ,A. L. Panda[1] ,G. Karwiky[2]
[1]Faculty of Medicine, Universitas Sam Ratulangi, Manado, Indonesia
[2]Faculty of Medicine, Universitas Padjajaran, Bandung, Indonesia

Background: Cardiac resynchronization therapy (CRT) is recommended in chronic reduced ejection fraction heart failure patients with sinus rhythm and left bundle branch block (LBBB) with QRS duration ≥150 msec who remained symptomatic despite optimal medical therapy to reduce morbidity and mortality as well as improve systolic left ventricular function and quality of life. HBP could be considered as rescue strategy for failed biventricular pacing or primary alternative to biventricular pacing.

Case illustration: A 73-year-old man with chronic heart failure reduced ejection fraction, hypertension, and type 2 diabetes mellitus came with New York Heart Association (NYHA) class III despite optimal medical therapy. Pre-operative electrocardiogram (ECG) showed 1st degree AV block with complete LBBB. Left ventricular ejection fraction (LVEF) was 26% with akinesia of the basal to mid inferoseptal, anteroseptal, anterior, apikoseptal, apikoanterior, and moderate mitral regurgitation. CRT with His bundle pacing was attempted as a primary strategy. Using right femoral approach, temporary pacing wire was placed at the right ventricular apex as per protocol. Bipolar lead was inserted into His bundle via left axillary vein and pacing was done after His signal was obtained on electrogram. QRS complex duration decreased significantly from 162 msec at baseline to 128 msec with pseudo delta wave (non-selective). His bundle capture threshold was 2.5 V at 1 msec at implant and increased slightly to 3.5 V at 1 msec at six months follow-up. There was improvement in NYHA functional class by one class and no heart failure hospitalisation six months after implantation. LVEF improved significantly from 26% to 40% six months after HBP implantation. There was also absolute reduction in LV end-systolic volume index from 62.275 ml/m^2 to 49.76 ml/m^2.

Conclusions:
HBP provides a physiological ventricular activation pattern through the His-Purkinje system maintaining contractility function, optimizing atrioventricular synchrony, and reducing right ventricular pacing complications. HBP was safe, feasible and reasonable primary alternative to biventricular pacing for CRT in patients with cardiomyopathy, AV block and LBBB. Permanent HBP could lead to significant improvement in QRS duration, clinical and echocardiographic response.

KEYWORD: *Heart failure reduced ejection fraction, left bundle branch block, his bundle pacing, cardiac resynchronization therapy.*

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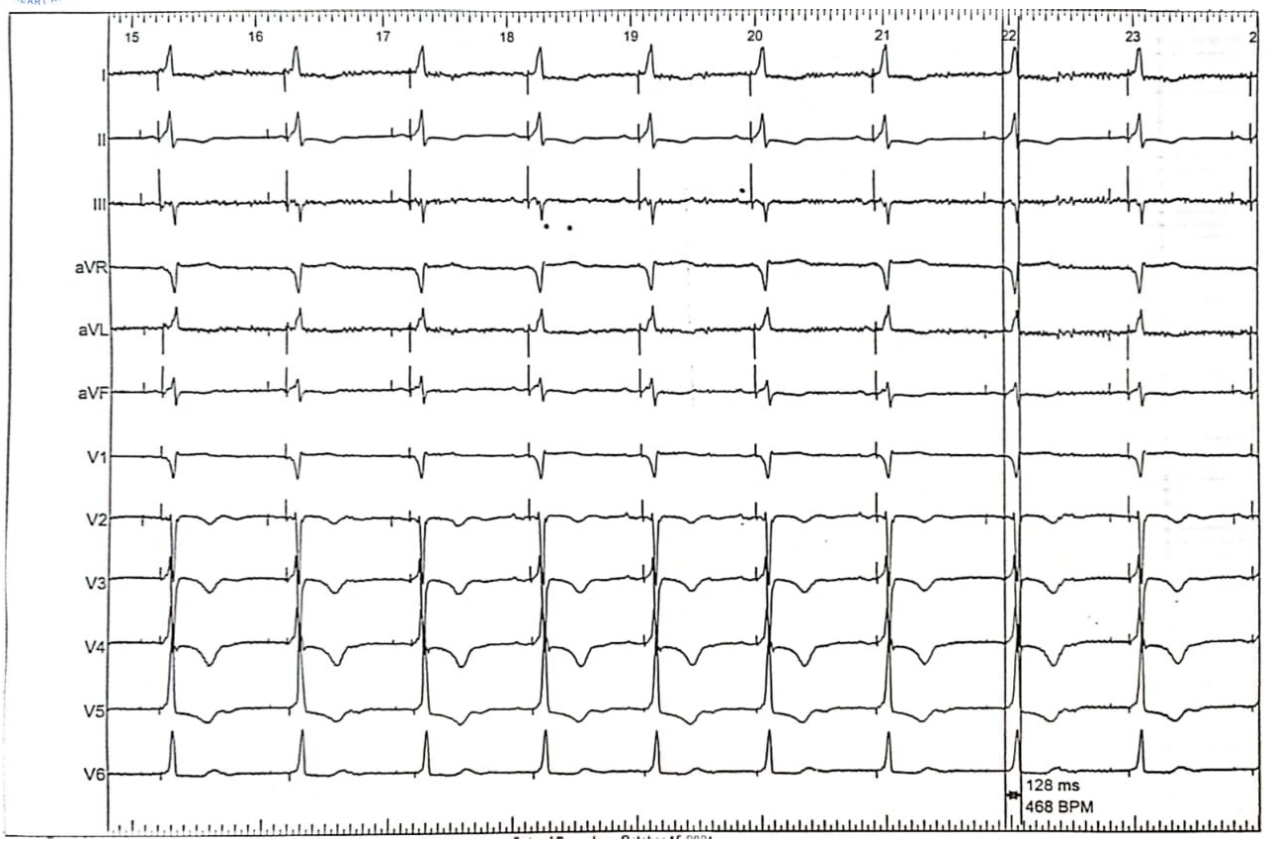

Figure 1 Electrogram after Successful HBP Implantation

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STANDING AT THE CROSSROAD : EFFICACY OF AMIODARONE IN PRE-EXCITED ATRIAL FIBRILLATION WITH A PRECARIOUS CONDUCTION PROPERTY OF ACCESORY PATHWAY

D. Ilmasari[1] ,F. Hidayati[2] ,E. Maharani[2] ,H. Hariawan[2]

[1]Resident of Department of Cardiology and Vascular Medicine, Dr Sardjito General Hospital/Gadjah Mada University, Yogyakarta, Indonesia,

[2]Staff of Department of Cardiology and Vascular Medicine, Dr Sardjito General Hospital/Gadjah Mada University, Yogyakarta, Indonesia

Background: The use of amiodarone for pre-excited atrial fibrillation (AF) has been reported to lead spontaneous ventricular fibrillation. We reported a case in which amiodarone was effective for this arrhythmia.

Case illustration: A 67 year old male complained of palpitation and shortness of breath. He had been previously diagnosed as AF-preexcitation with comorbid hypertension, obesity, heart failure, and history of haemoragic stroke. Hemoynamic was stable. The ECG showed AF-preexcitation. Electrical cardioversion 50 joule was delivered and the rhythm converted to sinus 60 bpm. During hospitalization, he had recurrent episode stable antidromic atrioventricular reentrant tachycardia and sustained AF-preexcitation Multiple electrical cardioversion was performed with energy up to 120 joule, but he had no response. Considering the shortest pre-excitation R-R interval (SPERRI) >250 ms (280 ms), then we gave this patient with amiodarone intravenous. Urgent radiofrequency ablation was conducted under general anesthesia. Electrical cardioversion 150 joule was delivered and the rhythm converted to sinus. Left anterolateral accessory pathway was ablated successfully.

Conclusions:

Management patient AF-preexcitation was challenging. The limited choice of antiarrhythmic drugs leading to the selection of drugs that aren't recommended by the guidelines. Amiodarone might be beneficial and effective in close hemodynamic monitoring.

KEYWORD: *Atrial fibrillation with pre-excitation, Amiodarone.*

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Detection of An Overlooked Microvolt T-Wave Alternans in Acquired Long QT Syndrome by A Novel Enhanced Adaptive Matched Filter (EAMF) Method: A Case Report

A. E. Tondas[1] ,D. Ulfiarakhma[1] ,M. Hadi[1] ,N. Y. Sari[1] ,I. Marcantoni[2] ,L. Burattini[2]
[1]Department of Cardiology and Vascular Medicine, Dr. Mohammad Hoesin General Hospital, Palembang, Indonesia. [2]Department of Information Engineering, Università Politecnica delle Marche, Ancona, Italy

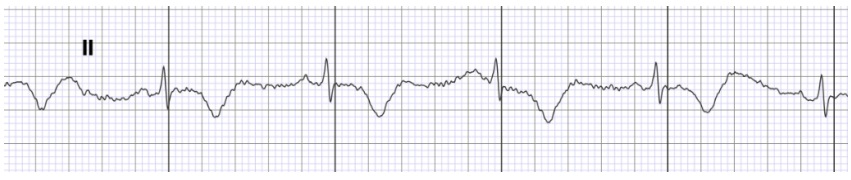
Background: T-wave alternans (TWA) in long QT syndrome (LQTS) serves as a marker of risk for cardiac events. Microvolt TWA (MTWA) is far more prevalent in LQTS patients than previously recognized. However, the use of conventional ECG may lead to an underestimation of MTWA detection.

Case illustration: A 21-year-old male was referred to our tertiary center with a chief complaint of palpitation and pre-syncope several hours after taking pseudoephedrine. His ECG on admission demonstrated sinus rhythm with prolonged corrected QT interval of 490 msec and diffuse T wave inversion. His physical examination, chest x-ray, laboratory, and echocardiography values were unremarkable. We performed 2-minute ECG recording by using KardiaMobile 6L™ (Alivecor®), a portable ECG device. By applying EAMF method, it revealed high MTWA amplitude and prolonged QTc in all leads (table 1). The EAMF-based procedure automatically identified ECG alternans through pre-processing step and alternans identification and measurement step. EAMF method can robust against interferences and noises in most frequency bands by filtering out the ECG signal components around alternans frequency, allowing a more accurate evaluation of MTWA. Several weeks after drug cessation, the follow-up ECG showed normal QTc and T wave morphology. The interesting finding is the reduction of MTWA amplitude as the QTc interval dropped. On average, MTWA amplitudes, QT interval, and QTc interval values dropped by 53%, 15%, and 11%, respectively compared to ECG on admission. To support our findings, a study showed the occurrence of TWA was directly proportional to QTc interval length. It demonstrated a significant association with an odds ratio of 1.23 per 0.01-$s^{1/2}$ unit increase in QTc length (95% CI 1.18 – 1.29, p <0.0001).

Conclusions:

MTWA can be determined by EAMF method along with the utilization of portable ECG device. In our LQTS patient, the occurrence of MTWA is associated with QTc interval length, and its finding might become a marker of major electrical instability and identify high risk patient. Therefore, MTWA detection could potentially be useful to prompt the discontinuation of culprit medications to reduce the risk of malignant arrhythmia.

KEYWORD: *Microvolt T-Wave Alternans, Long QT Syndrome, Enhanced Adaptive Matched Filter.*



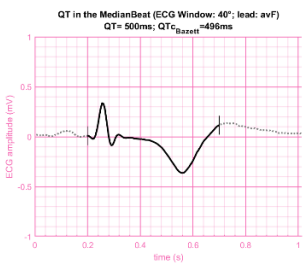


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Figure 1. (A) ECG recorded with portable device KardiaMobile 6L™; (B) analysis of TWA and QT interval by using EAMF method

Performing Safe and Successful Radiofrequency Ablation of The Rare Left Coronary Cusp Origin Premature Ventricular Conduction

D. N. Asmarani[1] ,R. Julario[1,2] ,N. D. Larasati[1] ,M. Aditya[1]

[1]Faculty of Medicine, Airlangga University, Surabaya, Indonesia

[2]Department of Cardiology and Vascular Medicine, Dr. Soetomo Hospital, Surabaya, Indonesia

Background: Idiopathic premature ventricular contraction (PVC) are part of ventricular arrhytmias whose mechanisms are not associated with structural heart disease, and usually originate from the specific anatomical structures. Clasically, idiopathic PVC originate from the right ventricular (RV) site, in which right ventricular outflow tract (RVOT) is the most common site of origin. Otherwise, there are only 10% incidence of PVCs originate from left outflow tract (LVOT), with the most common site of origin is aortic cusps. PVCs are basically benign, but they require medical treatment or radiofrequency ablation (RFA) if they remain symptomatic, incessant, or produce left ventricular dysfunction. We present a case report of a successful RFA of idiopathic PVC originate from left aortic cusp (LCC). With the high burden of greater procedural risk due to nearby anatomical structures such as coronary arteries or aortic valve cusps, the procedure was done safely with the guidance of fluoroscopic coronary artery angiography in order to secure left main coronary artery (LMCA).

Case illustration: A 20 years old man, with symptomatic and high burden of PVCs (27.3%), had a LCC origin PVC based on the 12-lead surface electrocardiogram. The 3D activation pace mapping showed 50 ms early activation and unipolar QS pattern in LCC site, with the QRS morphology similarity was 99% to spontaneous PVC morphology. Fluoroscopic angiography of the LMCA through right radial access was done prior the RFA and found that the ablation site was away from the ostium of LMCA. RFA was performed carefully with an angiographic catheter positioned within the LMCA ostium and continuously hand injected contrast to evaluate coronary artery and aortic sinus cusps. Repeated transthoracic echocardiography (TTE) was performed after each RFA. The patient was observed for 24 hours after the procedure, and evaluate by holter monitoring after one month. There were no PVCs and complications found.

Conclusions:
A successful catheter ablation within LCC site of origin was conducted safely with the guidance of LMCA angiography during the RFA procedure.

KEYWORD: *Premature Ventricular Contraction, Left Coronary Cusp Ventricular Arrhythmia, Radiofrequency Ablation.*

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When the Husband Touch Your Heart and Induced Ventricular Tachycardia

D. A. G. Nusantari[1] ,E. Maharani[2] ,F. Hidayati [3]

[1]Resident of Cardiology and Vascular Medicine Departement Faculty of Medicine, Gadjah Mada University, Yogyakarta, Indonesia

[2] Staff of Cardiology and Vascular Medicine Department of Cardiology and Vascular Medicine, Faculty of Medicine, Gadjah Mada University, Yogyakarta, Indonesia

Background: Sudden cardiac death has long been linked with strong emotion. Emotion can increase sympathetic nervous system activation. Arrhythmia could provide the physiological link between these stressors, catecholamines, and sudden death. Whether emotional or physical stress can directly trigger potentially fatal arrhythmias and how frequently this occurs had not been systematically investigated previously.
Case illustration: A 33 years old female with a history of shortness of breath and frequent episodes of palpitation when drinking coffee or having a problem with her husband. The ECG showed sustained ventricular tachycardia with right bundle branch block morphology, inferior axis, and late transitional zone at precordial lead despite the treatment using beta-blocker combined with calcium channel blocker. The echocardiography showed dilated right ventricle (RV), normal Ejection Fraction (EF 65%), TAPSE 18, and didn't meet the criteria for major or minor ARVD. The cardiac magnetic resonance imaging didn't support the criteria for ARVD or MVAD. The First electrophysiology procedure was performed, no inducible tachyarrhytmia despite the administration of sulphas atropine (SA) and adrenaline drip. In the next episode, she came with stable sustained VT and similar morphology. The second electrophysiology study was conducted, but no inducible VT was noted despite administration of SA and adrenaline, or even with two cups of caffeine. Then we asked her husband to provoke her emotion inside the EP lab, and the sustained VT with the same morphology was induced which sustained and stable hemodynamic. RFA was performed and showed the earliest activation at posteroseptal RVOT with 12/12 pacemapping similarity. No inducible VT afterward despite arrhythmia drug nor emotional provocation. One month after ablation patient is still symptom-free from ventricle tachycardia.

Conclusions:
Stress can exert adverse effect on cardiovascular health. Psychosocial stress adversely affects the autonomic homeostasis. Changes in the autonomic homeostasis can be a major trigger for ventricular tachyarrhythmias. Increased sympathetic nervous activity can cause increased proarrhythmic repolarization instability leading to spontaneous ventricular arrhythmias.

KEYWORD: *Nonsustained Ventricular Tachycardia, Arrhythmia, Psycosocial stress.*

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Shock-resistant ventricular fibrillation in an in-hospital witnessed cardiac arrest: a case report

D. Larasati[1] ,P. S. Phoa[1] ,A. D. Kemalasari[1]

[1]Radjak Hospital Salemba, Jakarta, Indonesia

Abstract

Refractory ventricular tachycardia or ventricular fibrillation (VT/VF) is defined as failure to achieve sustained return of spontaneous circulation (ROSC) after treatment with three defibrillation attempts and administration of 300 mg of amiodarone. We report a case of sixty-nine years old male who was admitted to the hospital with acute gastroenteritis and electrolyte imbalance. A few hours before leaving the hospital, he was seen chatting with his wife, and shortly after showed sign of cardiac incident. He suddenly stopped talking, his arms were extended upward, and was described by his wife as having seizure-like activity for three seconds before having no pulse. Chest compression was initiated and advanced cardiac life support was delivered. The monitor showed ventricular fibrillation (VF) and the first direct current (DC) shock of 360 joule was delivered and patient was intubated. After subsequent shock and chest compression, monitor still showed VF. The second shock was then delivered and 1 mg of intravenous epinephrine was administered. The monitor still showed VF and the third shock was delivered with 300 mg of amiodarone given intravenously. The monitor still showed refractory VF, and the fourth shock was delivered with the same amount of energy. Cardiology consult was then made, shock-resistant VF was suspected and lidocaine was prepared for refractory VF. After the chest compression, monitor still showed VF and the fifth shock was delivered. The second dose of 150 mg amiodarone was injected intravenously. We continued chest compression while preparing the next shock. Lidocaine intravenous bolus of 1 mg/kg body weight was then administered as bolus and 4 mg/min was continued as infusion. Subsequently the sixth shock was delivered after monitor still showed VF. After a few seconds, the monitor showed pulseless electrical activity. Chest compression was then continued but still he did not convert. After 45 minutes of chest compression and no sign of return of spontaneous circulation, the resuscitation was stopped. Patient was examined and pronounced death at scene. The cause of shock-resistant VF in our patient is still unknown and amiodarone and lidocaine is seen as ineffective for this particular patient.

Keyword: *shock-resistant ventricular fibrillation, cardiac arrest, amiodarone, lidocaine.*

Supplementary File: 1

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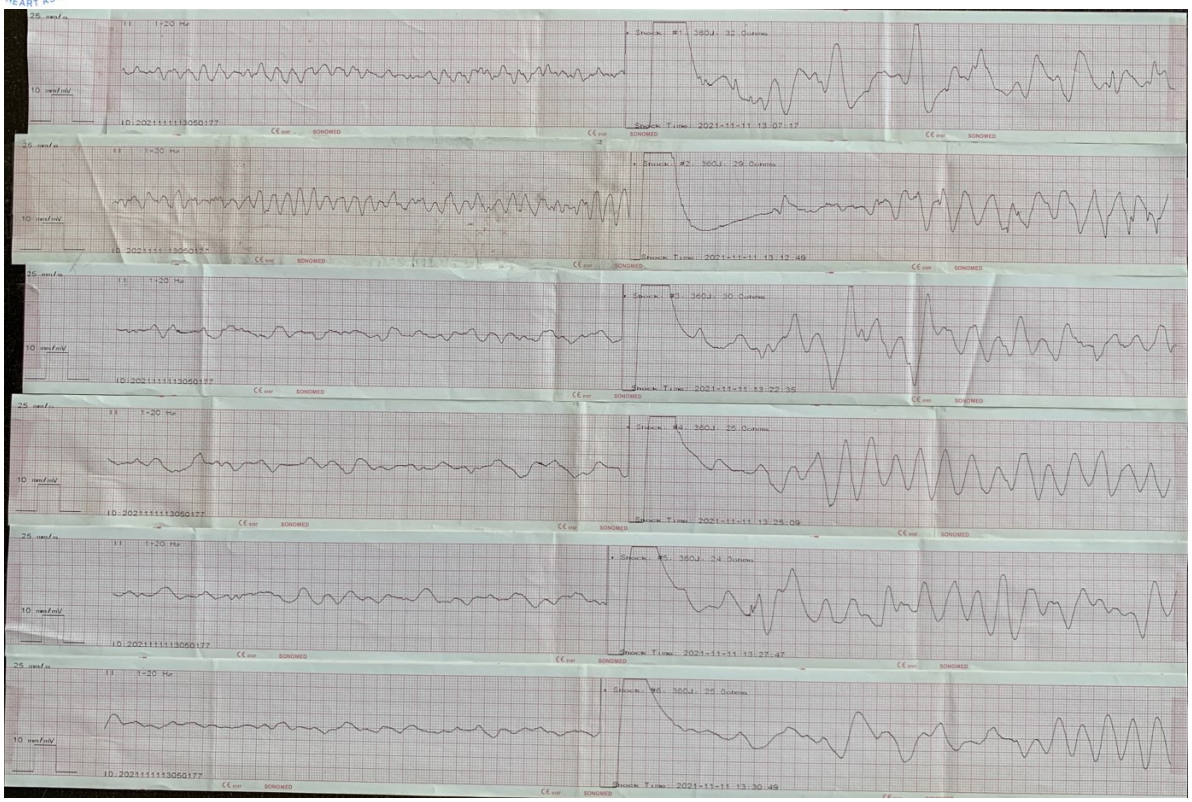

Figure 1. Electrocardiogram recorded by the external defibrillator device prior and after first-sixth shocks delivered

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The Discovery of Sinus Node Dysfunction on Preoperative Evaluation

A. A. G. I. Putra[1] ,K. E. Bagiari[2] ,N. L. E. S. Wulandari[2]
[1]General Practitioner, Sanjiwani General Hospital, Gianyar, Indonesia

[2]Cardiologist, Sanjiwani General Hospital, Gianyar, Indonesia

Background: Sinus node dysfunction (SND) refers to a broad range of abnormalities involving sinus node and atrial impulse generation and propagation. SND is commonly diagnosed in geriatric population. Only small amount of case was seen in young population.[1,2,3]

Case illustration: A 35-year-old male with no significant medical history was referred for preoperative evaluation because of asymptomatic junctional bradycardia. The ECG finding includes incomplete RBBB and inverted P wave in II, III, avF. The patient denied any symptoms such as presyncope or syncope. However, lately the patient experiences exercise tolerance and dizziness especially when he performs sports activity. Results from initial medical evaluation, including CBC, ESR, electrolyte serum were normal. A TTE showed an LV EF of 59.1%. The patient referred to an electrophysiologist for further evaluation. One month later, holter monitoring test results uncovered baseline junctional rhythm with no symptoms during recording. Exercise stress tests revealed average functional capacity (10.17 METs) and baseline bradycardia with only 65% maximum predicted heart rate was achieved. SND results from various conditions which have the capability to depress automaticity and electrical conduction from the sinus node. Most SND cases in young patients are related to congenital heart disease.[3,4] Holter monitor can exclude SND as the cause of symptoms if normal sinus rhythm is documented during presyncope, or syncope.[3] Exercise stress test is useful in differentiating patients with chronotropic incompetence from those with resting bradycardia.[3] Long-term prognosis of asymptomatic bradycardia patient is generally benign. However, conflicting evidence seen from a guideline perspective which suggest permanent pacing with rate-responsive programming can improve symptoms.[5,6]

Conclusions:

Diagnostic approach for SND includes baseline ECG data followed by 24-h holter monitoring as well as treadmill test to confirm chronotropic incompetence in this patient. Further monitoring is still necessary to monitor the patient's symptoms and possible options for permanent pacing.

KEYWORD: *Sinus node dysfunction, holter monitor, exercise stress test.*

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A Case Report: Ventricular Tachycardia in Geriatric Patient with Thyroid Heart Disease: Thyroid Storm or Ventricular Tachycardia Storm in Acute Coronary Syndrome?

P. N. Permatasari[1] ,R. Rozqie[2]
[1]General Practitioner, RSA Gajah Mada University, Yogyakarta, Indonesia
[2]Cardiologist, RSA Gajah Mada University, Yogyakarta, Indonesia

Background: VT storm is defined as three or more episodes of sustained VT within 24 hours. It's caused by structural or non-structural heart disease and needs quick management to prevent life-threatening conditions.
Case Illustration: A 69 -year-old female patient came to the ER with epigastric pain, nausea, vomitus, diaphoresis, and palpitation. She had THD and AF controlled by warfarin 1x2 mg, bisoprolol 1x5mg, and candesartan 1x8mg. Physical Examination shows tachycardia 190 times/minute, S1-S2 regular, cardiomegaly, and epigastric pain. Burch-Warsofsky score 45. A 12-lead electrocardiogram showed Ventricular Tachycardia. Immediately, the patient was administrated with amiodarone 150 mg bolus within 15-30 minutes, drip amiodarone 1 mg/hour within 6 hours then 0,5 mg/hour within 18 hours. Post bolus, 12-lead ECG shows NSTEMI in lead II, III, aVF, V2-6. HS-Troponin 16. Because of hypotension, the patient gets Dual Antiplatelet Therapy and Dobutamine 5 mcq/kgbw/hour. In ICU, 12-lead ECG showed Sinus, 75 bpm, normo axis, PVC bigeminy on maintenance amiodarone drip 0,5 mg/hour within 18 hours also on support Dobutamine and Norepinephrine Patient-reported bradycardia and asystole we start cardio pulmonary resuscitation. The Patient ROSC 2 times on intubation. ECG monitor showed VT sustained, we bolus lidocaine 40 mg, drip maintenance 2 mg/minutes and cardioversion non-synchronized. 12-lead ECG evaluation shows dynamic in V3-V5. Then the patient was referred to a type A Hospital to get Immediate PCI.

Conclusions:
VT Storm can be triggered by Thyroid Storm or Acute Coronary Syndrome. In this case, Acute Coronary Syndrome; NSTEMI caused VT Storm although Burch-Warsofsky score 45. 12-lead ECG showed sinus after administrated with amiodaron and DAPT. The patient had sustained VT-Storm with maximal pharmacological therapy so the patient must get Immediate PCI.

KEYWORD: *Ventricular Tachycardia Storm, Acute Coronary Syndrome, Thyroid Heart Disease, Amiodarone, DAPT.*

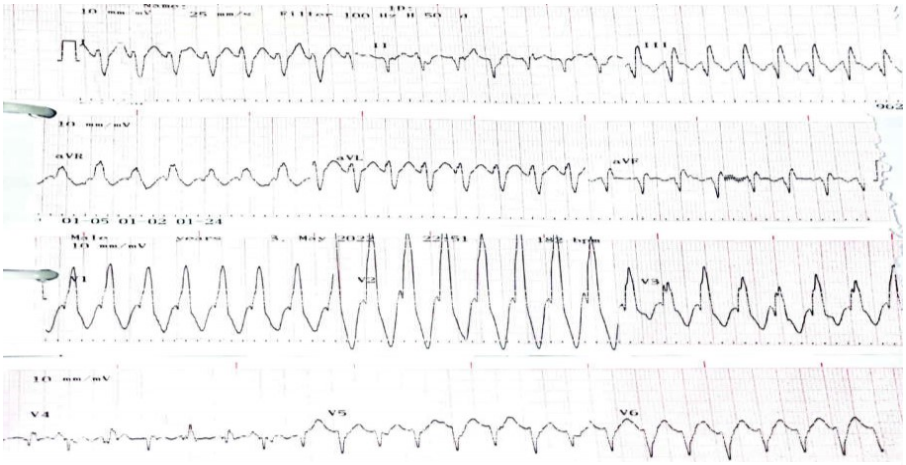

Figure 3. Patient's 12-lead electrocardiogram in ER

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Challenge in The Management of Supraventricular Tachycardia with Wolff-Parkinson-White Syndrome in Third Trimester of Pregnancy in Rural Hospital

R. T. Hendrayani[1] ,I. Rosmanadewi[1]
[1]*PKU Muhammadiyah Gombong Hospital, Jawa Tengah, Indonesia*

Background: Wolff-Parkinson-White (WPW) syndrome is characterized by an accessory pathway that predisposes patients to tachyarrhythmias and sudden cardiac death. Early recognition is important and should be evaluated by a multidisciplinary team for adequate management and treatment. We present a pregnant woman that presented to the emergency department and discuss her treatment in rural hospital.

Case Illustration: A 23-year-old woman at 34 weeks of gestation (G3P2A0) presented to the emergency room PKU Gombong Hospital with chief complaint of palpitations two hours before admission. She was conscious with heart rate of 201 beats per minute and stable hemodynamic. She had no vaginal bleeding. Her first and second trimester was uneventful; the patient has a history of WPW patern since her first trimester managed with oral bisoprolol during pregnancy. She had no other relevant medical history or family history of cardiac history. Blood count, electrolytes, liver function, renal function, TSH, FT4, coagulation test were performed which were normal. An electrocardiogram (ECG) was performed during the episode which showed narrow complex tachycardia findings of supraventricular tachycardia. Vagal maneuvers such as carotid massage and valsalva maneuvers failed to correct the pulse. Due to the lack of adenosine and verapamil in our hospital, 5 mg bisoprolol was given twice, failed to correct the pulse. Then, 0,5 mg of intravenous digoxin was given twice in bolus. The patient regained normal heart rate after treatment and the ECG showed findings of Wolff-Parkinson-White (WPW) syndrome. The cardiotocography was reactive with good variability and no uterine contractions. Then we refer the patient to the Sardjito Hospital.

Conclusions:
Wolff-Parkinson-White syndrome is a rare condition and diagnosis might be missed. Identification of this disease is important to prevent further arrhythmias and sudden cardiac death. Arrhythmias can be life-threatening to both the fetus and the mother, so close monitoring should be done. Management of this condition should be done by a multidisciplinary team.

KEYWORD: *Pregnancy, Supraventricular Tachycardia, Wolff-Parkinson-White Syndrome.*

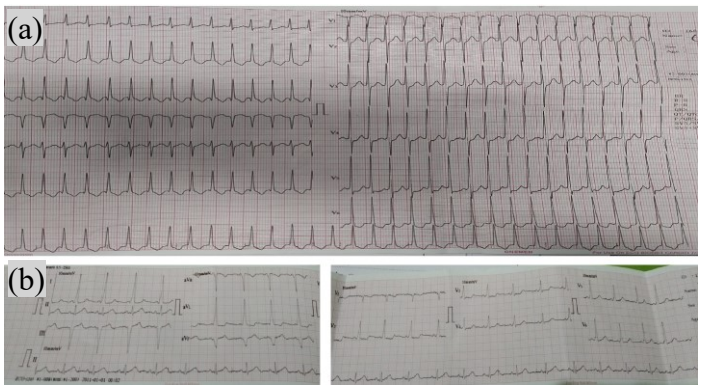

Figure 1. Electrocardiogram (ECG) on admission showing supraventricular tachycardia (a); ECG following treatment showing sinus rhythm with short PR interval and delta wave (b)

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Electrical Storm in Acute Heart Failure Patients with the History of COVID-19 : How To Juggle the Lightning in Rural Area

R. Mulawarman[1] ,D. Yuliani[1] ,K. Janitra[1] ,R. A. Halim[2] ,M. Trifitriana[2]
[1]Prabumulih General Hospital, Prabumulih, South Sumatra
[2]Faculty of Medicine, Sriwijaya University, Palembang, South Sumatra, Indonesia

Background: Electrical storm is a dramatic clinical life-threatening condition of 3 or more episodes of ventricular arrhytmias (VAs) such as ventricular tachycardia (VT) or ventricular fibrillation (VF) within a 24 hour period. Hereby we report a case of acute heart failure (AHF) patient presenting with electrical storm in a rural area. Referrals to tertiary health services take time due to the COVID-19 pandemic.

Case illustration: A 55-year old male was admitted to Prabumulih General Hospital with shortness of breath after gardening since 2 hours before admission. He also felt palpitation and swelling in both legs. He also has history of hypertension and cardiogenic shock along with COVID-19 10 months ago. ECG shows monomorphic VT with rate of 261 beats/minutes and blood pressure of 71/56 mmHg. Physical examination is significant for elevated jugular veins, rales on both of lungs, and bilateral pretibial edema. Synchronized cardioversion with 100 joule was shocked to the patients, then the rhythm convert to atrial fibrillation with rapid ventricular response (AF RVR). Then, he was treated in intensive care unit (ICU) and got intravenous amiodarone therapy. The rhythm returns to monomorphic VT for totally four episodes which is shocked and sucessfully converted in ICU. Laboratory findings revealed normal, but cardiac enzyme level and echocardiography exam is not available in the hospital. The patient was referred to Mohammad Hoesin General Hospital Palembang. Coronary angiography demonstrated non-significant coronary disease with 40% occlusion in LAD and 43% ejection fraction (EF). ECG demonstrated normal sinus rhythm. Patients is started on aspilet, clopidogrel, nitrat, candesartan, furosemide and bisoprolol for ambulatory medication. In patients without an ICD, the presentation of patients with electrical storm be affected by the hemodynamic significance of the VA.[1,2] In most cases, VT is due to electrical reentry around a fixed anatomic barrier, most commonly scar tissue after myocardial infarction.[3] However, in our case report the patient has COVID-19 history. Previously, a study reported that cardiac arrhytmias were significantly associated with an increased risk of poor outcome in COVID-19 due to cardiac injury as complication.[4] In peripheral hospital advanced cardiac life support (ACLS) algorithm is very important. Because of the shortness of breath leading to acute heart failure accompanied by hypotension, we decided to perform a DC shock starting at 100 joules and increased gradually to 200 joules according to the algorithm. We don't use intravenous amiodarone due to the hypotension side effect. In the acute setting, amiodarone can cause hypotension due to vasodilation and depression of myocardial contractility.[5] Initial treatment based on the ACLS algorithm for general practitioners is very important for lifesaving patients before being referred to a central hospital.

Conclusions:
In rural settings where cardiac laboratory, echocardiography and other cardiac facilitation are not available, as general practitioner we can use the minimal equipment and facilities improvised effectively according to ACLS guidelines. Cardioversion is the best choice then intravenous antiarrhythmic drugs such as amiodarone due to the patient unstable condition.

KEYWORD: *Electrical storm, cardioversion, ventricular tachycardia.*

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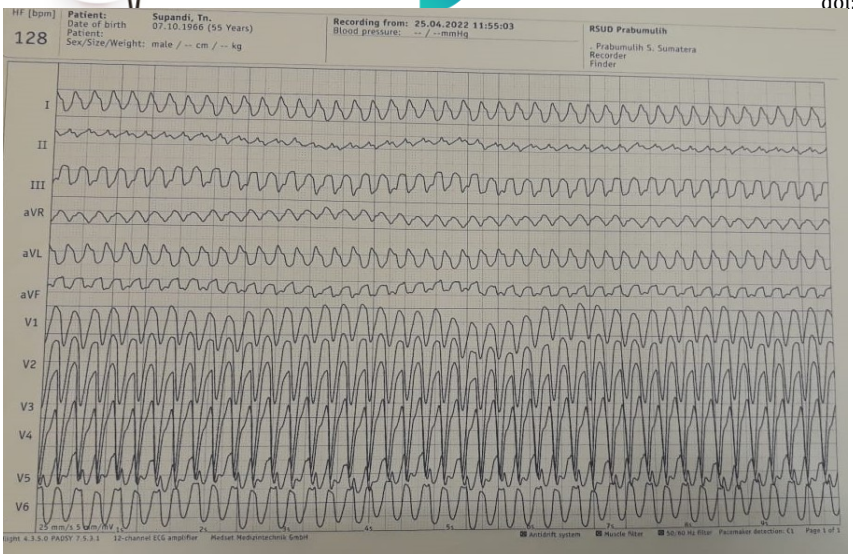

Figure 1 ECG in admission ventricular tachycardia

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Challenges in the Management of Thyrotoxicosis Associated with Repetitive Ventricular Tachycardia

D. P. Mentari[1] ,R. Setiarsa[2]

[1,2]Krakatau Medika Hospital, Cilegon, Indonesia

Background: Transient ventricular tachycardia (VT) is a unique manifestation of hyperthyroidism. We present the case of a 52-year-old female with a history of hyperthyroidism presenting with palpitations secondary to episodes of transient VT. Cardiac arrhythmias due to thyrotoxicosis are perpetually supraventricular in origin. Monomorphic VT in the setting of thyrotoxicosis in the absence of structural heart disease is exceedingly rare. After starting amiodarone and increasing the dose of thiamazole, the patient had no further episodes of VT. It is important to recognize repetitive monomorphic VT as an understated but important manifestation of thyrotoxicosis. Amiodarone is a drug of choice for ventricular tachycardia but Amiodarone can also have a toxic effect on the thyroid gland that can induced thyrotoxicosis.

Case illustration: A woman, 52 years old, came to the emergency room with palpitations and muscle weakness. Her past medical history was hyperthyroidism and recurrent ventricular tachycardia. She was taking thiamazole and propranolol regularly as medication. There is no sign of heart failure and coronary artery disease. ECG displayed Transient Ventricular Tachycardia. Laboratory tests showed elevated of thyroid function, low serum TSH concentration and mild hypokalemia. Amiodarone was given as an antiarrhythmic agent and the patient had no episodes of Ventricular Tachycardia.

Conclusions:
It is important to recognize repetitive monomorphic VT as an understated but important manifestation of thyrotoxicosis. Propranolol is associated with an excellent response in these patients and anti-thyroid medications such as thiamazole effectively reverse thyrotoxicity but still not cure enough for repetitive monomorphic VT. Administration of amiodarone can be effective in treating acute ventricular tachycardia but in long-term use it can cause thyrotoxicosis so that it can be a challenge in the management of hyperthyroidism with recurrent ventricular tachycardia manifestations.

KEYWORD: *Ventricular Tachycardia; Thyrotoxicosis; Amiodarone; Hyperthyroidism.*

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Sudden Onset of Arrhythmia in Patient Receiving Hormonal Therapy: Is there any relation between them?

E. K. Rofiah[1] ,M. U. A. Jomansyah[1]

[1] JIH Yogyakarta Hospital, Yogyakarta, Indonesia

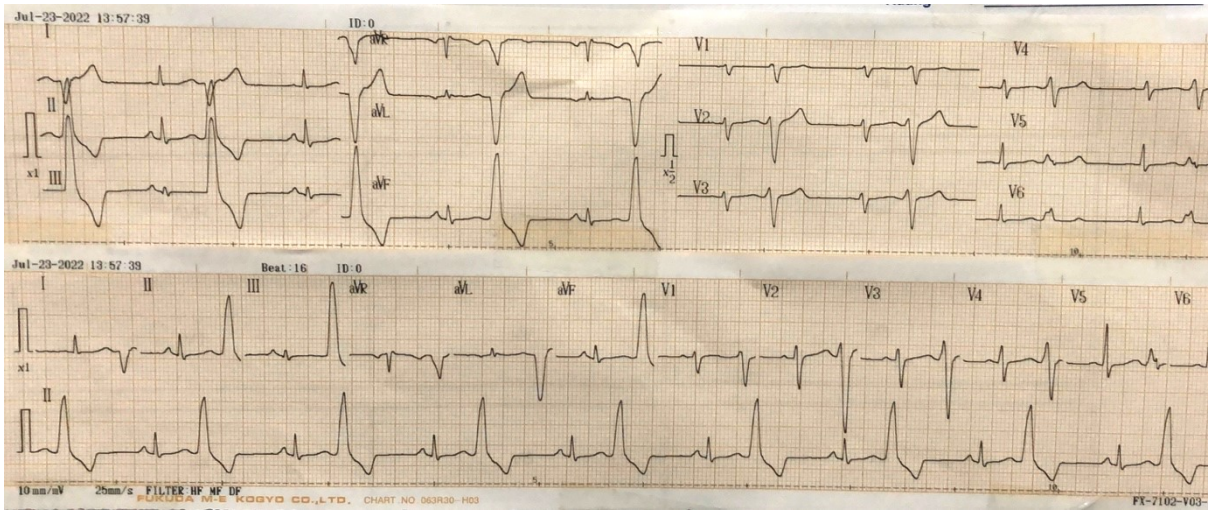
Background: In-vitro fertilisation (IVF) procedure is one of the treatment of choice for infertile couple. Hormonal therapy frequently administered to the female patient before the procedure. Despite the limited data, cardiovascular disease including cardiac arrythmia has been shown to occur following hormonal preparation in IVF procedure.

Case Illustration: A 39-year-old woman came to the hospital to have an electrocardiogram (ECG) examination before undergoing IVF procedure. She complained of mild palpitation that she has never had before. Meanwhile, chest pain, shortness of breath, and syncope/pre-syncope were all ignored. She was known receiving Choriogonadotropin Alfa injection, a hormonal therapy to stimulate ovulation, in the past 5 days. There was not history of patient's previous cardiovascular disease. Upon arrival in hospital, the patient was fully alert with BP: 128/66 mmHg, HR: 98 x/m palpable-irregular, RR: 18 x/m, SpO2: 98% RA. Physical examination was found normal. First ECG examination revealed normal sinus rhythm with infrequent PVC. Therefore, serial ECG was done at 20 minutes interval, the second result showed trigeminy PVC, and the third one was bigeminy PVC. The patient was then observed in emergency unit.

Conclusions:

Injection of Choriogonadotropin Alfa for several days in preparing IVF procedure may precipitate the development of sudden onset of arrhythmia in this patient. Cardiovascular disease monitoring before and after hormonal administration especially in IVF procedure should be done to minimize unwanted cardiac event.

KEYWORD: *Cardiac arrhythmia, hormonal therapy, in-vitro fertilisation.*



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Type-1 Brugada ECG-pattern Unmasked by COVID-19 in Young Male Patient Without Cardiovascular Risk Factors: Should Routine ECG Be Considered as A Mandatory Screening?

E. F. Jambormias[1] ,K. Kurniawan[2] ,R. G. Aurora[3]
[1]General Practitioner, Jailolo General Hospital, West Halmahera, North Maluku, Indonesia
[2]Internship Doctor, Jailolo General Hospital, West Halmahera, North Maluku, Indonesia
[3]Cardiologist, National Cardiovascular Center Harapan Kita Jakarta, Indonesia

Background: COVID-19 is primarily a respiratory illness, usually presents with fever, however cardiovascular symptoms can be involved. Fever was found to be an inflammatory response of infection that may unmask a Brugada ECG-pattern in asymptomatic patients, who are at risk of arrythmia and sudden cardiac arrest.
Case illustration: A 37-year-old male without known comorbidities presented to ER following 2-day history of fever, cough, sore throat, and nausea. He was not taking any medications. He denied any syncopal event, palpitation, family history of sudden cardiac death and cardiac diseases. On physical examination, he was febrile (39,9°C). An ECG showed an over-shift of the ST-segment in V1-V2 evoking a Type-1 Brugada (Fig 1). The chest radiograph showed bilateral infiltrations and positive result of RT-PCR confirming the diagnosis of COVID-19. Hydration and antipyretic agent were administered. Type-1 Brugada ECG-pattern resolved after fever subsided.

Conclusions:
Brugada syndrome (BrS) defined by type-1 Brugada ECG-pattern and pertinent clinical features such as personal history of syncope, ventricular arrhythmia, or family history of sudden cardiac death. Type-1 Brugada ECG-pattern, describe as coved ST-segment elevations in V1–V3. The shortening of action potential duration occurs because fever might facilitate re-entrant ventricular tachycardia. Performing early assessment of ECG might be a breakthrough to screen BrS. Additionally, to standard isolation procedure of COVID-19, those with known Brugada ECG-pattern may warrant more aggressive antipyretic therapy and serial ECGs screening.

KEYWORD: *Brugada Syndrome (BrS), Type-1 Brugada, ECG, Covid-19, Young male.*

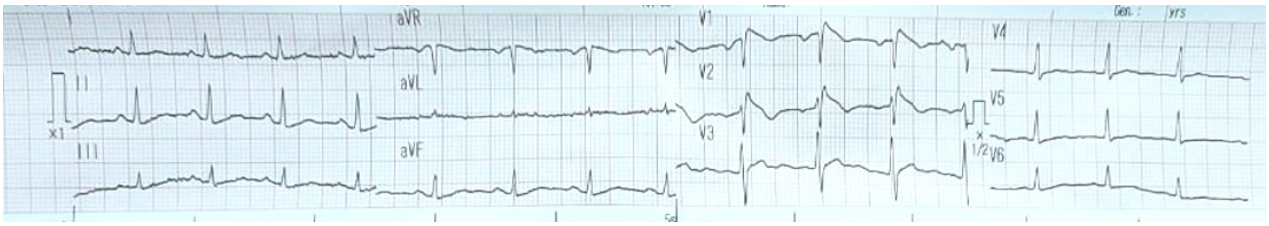

Figure 1 The 12-lead electrocardiogram showing Brugada type-1 pattern (ECG > 2 mm elevation of the J-point with a downward sloping of the ST segment in leads V1-V2, with associated inverted T-waves).

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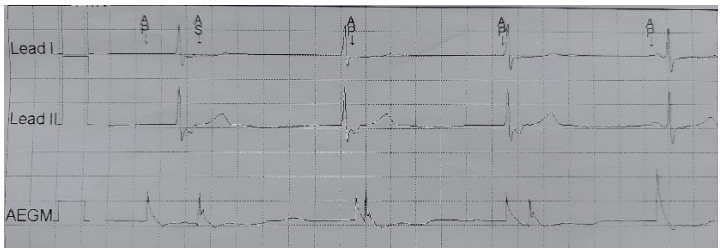
Rate-dependent Change in The Atrial Capture during Cardiac Pacemaker Implantation in Patients with Sick Sinus Syndrome

E. J. Gunawan[1] ,Johan[1] ,Robert[2] ,I. Citra[2] ,D. L. Munawar[1] ,B. Hartono[1] ,M. Munawar[1]
[1]Cardiology and Vascular Department of Binawaluya Cardiac Center
[2]Cardiology and Vascular Department of Sam Ratulangi University

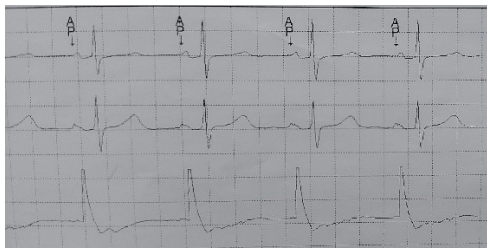
Background: Pacemaker is the treatment of choice for patient with symptomatic severe bradycardia. Various factors can influence the threshold of myocardial tissue during pacing. In this case report, we present a case of rate-dependent change in atrial capture during pacemaker implantation.
Case illustration: A 74 years old woman with severe bradycardia with documented 9.7 sec pause. She received dual chamber permanent pacemaker (PPM) with right atrial (RA) passive lead in right atrial appendage (RAA) and right ventricular (RV) passive lead in high septum RV. Two days after implantation, patient monitoring showed atrial pacing spikes without P waves. Pacemaker interrogation revealed a marked increase in the atrial capture threshold to 6 V at 0.4 ms with no significant change in impedance, and RV lead had no issue. No apparent RA lead dislodgement in chest X-ray compare to initial implantation. Reposition of RA lead was performed. After repositioning, the interrogation results were atrial threshold of 0.5V at 0.4 ms with 90 bpm and impedance of 584 ohm. Pacing at RA with amplitude of 1.5 V at 0.4 ms with 70 bpm, and the pacing was captured. However, with the same amplitude of 1.5 V at 0.4 ms with 50 bpm, the pacing was not captured. Therefore, the lower rate rose to 70 bpm to avoid pacing failure. The mechanism of rate-dependent change in atrial capture especially in lower rate is unclear, it is possible that pacemaker lead-induced inflammation, and fibrosis of the atrial myocardium may cause spontaneous phase 4 depolarization. Another possible mechanism is because higher pacing rates reduce atrial end-diastolic diameter, and improves electrode contact with myocardial surface.

Conclusions:
Rate-dependent change in the atrial capture can be observed during pacemaker implantation. This phenomenon can be observed by changing the pacing rate by increasing the pacing rate and can improve the myocardial capture. It is important to program with higher pacing rate to avoid pacing failure.

KEYWORD**:** *Pacemaker, sick sinus syndrome, rate-dependent, atrial capture.*



Pacing at 1.5 V at 0.5 ms with 50bpm, loss capture



Pacing at 1.5 V at 0.5 ms with 70bpm, capture

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Complete Heart Block in Postpartum Hypocalcaemia in Rural Hospital. Can It Be Resolved Without Implanting Pacemaker?

F. K. Wijayanti[1] ,I. W. Rosmanadewi[2] ,B. E. Putra[3]

[1]General Practitioner, RS PKU Muhammadiyah Gombong, Kebumen, Indonesia

[2]Cardiologist, RS PKU Muhammadiyah Gombong, Kebumen, Indonesia

[3]Cardiology Division, RSUD Berkah Pandeglang, Banten, Indonesia

Background: A third-degree atrioventricular (AV) block (complete heart block) indicates a total breakdown of the ventricles' ability to communicate with the atria. Without proper conduction through the AV node, the SA node cannot control the heart rhythm, and cardiac output may decrease as a result of a loss of coordination between the atria and the ventricles. New-onset third-degree AV block is a medical emergency. If not treated promptly, the condition may be fatal. The definitive treatment of high-degree AV block is implanting a pacemaker or administering Dopamine or Epinephrine. Hypocalcaemia in pregnancy and lactating period is not frequently reported in the literature but it can happen to mothers with severe dietary deficiencies and hypoparathyroidism. Hypocalcaemia may lead to heart block. Here we present an interesting case about treating third-degree AV block without implanting a pacemaker.

Case illustration: A 22 years-old postpartum woman came to emergency room with sudden chest discomfort, heart pounding, and slight shortness of breath. She delivered her baby five months ago. She had no history of cardiac arrhythmia, pre-eclampsia, or hypertension before pregnancy. Laboratory indicated severe hypocalcaemia (4,29 mg/dl) without abnormality of potassium serum. She was diagnosed with peripartum cardiomyopathy as her echocardiography showed reduced systolic function (LVEF 38%). ECG showed first-degree AV block and transformed into complete AV block with rate of 46 bpm after two days. Calcium Gluconate IV was administered, and the complete heart block was successfully converted to normal ECG after three days.

Conclusions:

Patients with complete heart block should be managed thoroughly, from history taking to proper diagnostic testing. We highlight the importance of correctly diagnosing hypocalcaemia in complete heart block and doing serum correction before deciding to implant a pacemaker.

KEYWORD: *Third-degree AV block, complete heart block, hypocalcaemia.*

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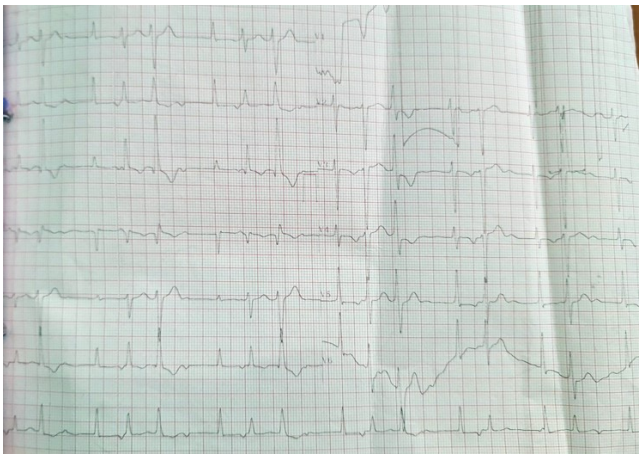

Figure 1 ECG showed third-degree AV block with PVC

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Brugada Syndrome Unmasked by Electrolyte Imbalance

F. H. Ahmad[1] ,D. D. Utomo[1] ,A. Putri[1] ,P. Utomo[1]
[1]Department of Cardiology and Vascular Medicine, Cibabat General Hospital, Cimahi, Indonesia

Background: Brugada Syndrome is an autosomal dominant arrhythmogenic disease characterized by the typical ECG pattern of ST-Segment elevation in the right precordial leads, incomplete right bundle branch block, and associated with high risk of sudden cardiac death in structurally normal heart. Brugada syndrome phenotype is associated with decrease in the sodium current owing to reduction in sodium channels and/or reduced function of the sodium channels.
Case illustration: A 23-year-old man without significant medical history presented with chest pain radiating to the back for 3 days before and worsened since 3 hours before admission, accompanied by episodes of palpitation, fever, epigastric pain, and nausea. He had no history of faintness or syncope, shortness of breath, spontaneous type I Brugada ECG Pattern, or familial history of sudden cardiac death. Physical examination were all within normal range despite complaint of fever during hospital stay. The 12-lead ECG exhibited a type 1 Brugada ECG in the right sided precordial leads. The laboratory data showed significant hyponatremia (125mmol/L) and slight hypokalemia (3.4 mmol/L) with others within normal limit.

Conclusions:
In conclusion, we described patient with Brugada Syndrome that unmasked by electrolyte imbalance. It is important to assess patient thoroughly and the need of electrophysiology study in Brugada Syndrome as its associated with high risk of sudden cardiac death.

KEYWORD: *Brugada Syndrome, hyponatremia, electrolyte imbalance.*

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Torsades de Pointes Associated with Long QT Syndrome Predisposed by Combination of Electrolyte Abnormalities and Medication: A Case Report

F. A. Nurullah[1] ,R.A. Fanani[2]

[1.]General Practitioner, Hermina Podomoro Hospital, Jakarta, Indonesia

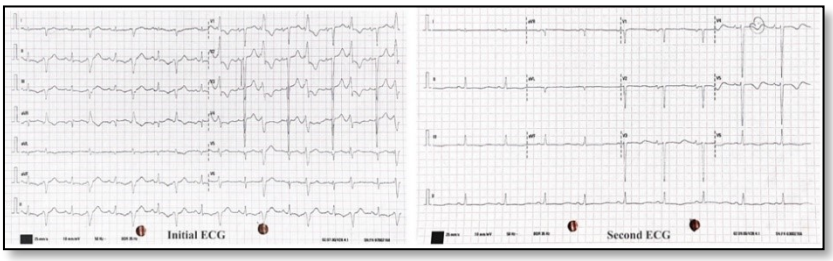
[2.] Cardiologist, Hermina Podomoro Hospital, Jakarta, Indonesia

Background: Long QT syndrome (LQTS) is a disorder of cardiac repolarization leading to prolonged QT interval and T-wave abnormalities on the ECG, thus deteriorates to Torsades de Pointes (TdP) and sudden cardiac death (SCD). Electrolyte abnormalities and medications can induce TdP in the setting of latent LQTS.

Case Illustration: A 49-year-old woman came to outpatient department with palpitations without chest pain and history of hypertension, chronic heart failure and syncope episodes in couple months, no history of prior medication, diarrhea and vomiting and no known familial history of SCD. On physical examination she was alert and fully oriented. Initial ECG showed sinus rhythm with ventricular extrasystole bigeminy, QT was 280 ms and QTc was 361 ms. Her initial laboratory result showed serum sodium 137mEq/L, potassium 3.8mEq/L, magnesium 2.20mEq/L, troponin T was undetectable, other results were unremarkable. She was admitted to ICU received a 150 mg intravenous amiodarone followed by maintenance dose of 900 mg/24 hrs. Echocardiography showed dilated LV, hypertrophic LV, LVEF 39%, akinetic anterior, anteroseptal and anterolateral segments, hypokinetic inferoseptal segment, RV systolic function was reduced, mild tricuspid regurgitation, other valves were normal. While being monitored in ICU, she developed multiple TdP, some episodes terminated spontaneously but dozens required DC shock. Her labs showed potassium level dropped to 3.17mEq/L and magnesium 2.00mEq/L. Post resuscitation ECG showed sinus bradycardia, prolonged QTc as QT 520 ms, QTc interval 511 ms, biphasic T wave and U wave in all leads. She was diagnosed with TdP, acquired LQTS. Forceful management aimed to stop TdP recurrency and stabilize her vital signs. Amiodarone and other medications which may cause QT prolongation were stopped. We started intravenous magnesium and electrolytes correction continued to maintain potassium above 4.0mEq/L and magnesium above 2.00mEq/L.

Conclusions:
Our patient had TdP due to LQTS unmasked by combination of electrolyte abnormalities and medication. Early detection by QTc interval analysis is useful to diagnose LQTS. When congenital LQTS is suspected, genetic testing is the preferred diagnostic test. It is also important to maintain electrolyte level and cautiously avoid to use several drugs that prolong QT interval in this patient.

KEYWORD: *Torsades de Pointes, Long QT Syndrome, Electrolytes Abnormalities, Hypokalaemia, Amiodarone.*



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Non-Sustained VT, Antiarrhythmics: Are they necessary?

I. J. Aziz[1,2] ,S. Widito [1,2]

[1]Department of Cardiology and Vascular Medicine Faculty of Medicine Universitas Brawijaya, [2]Dr. Saiful Anwar General Hospital

Background: The most common cause of sudden cardiac death (SCD) is ventricular tachyarrhythmias (VTs). Hypokalemia is a well-recognized risk factor for VT. In patients presenting with ventricular tachyarrhythmias, normokalemia was associated with the highest 30-day and 3-year survival, while hyperkalemia and hypokalemia were associated with an increase in mortality. Recent experimental studies suggest that hypokalemia-induced arrhythmias are triggered by the decreased activity of the Na+/K+-ATPase (NKA), which then leads to Ca2+ overload, Ca2+/Calmodulin-dependent kinase II (CaMKII) activation, and the development of afterdepolarizations. We present a female patient with complaints of palpitations and paraparesis of the lower extremities. PVC bigeminy was found in the patient's ecg

Case illustration: A 32-year-old woman came with complaints of palpitations since 2 days ago. The patient also complained of weakness in both legs since 2 weeks ago. Complaints of weakness in the legs followed by a parasthesia in lower extremity. In the ED, she appeared cachetic and physical examination was found to be weak with an irregularly regular pulse, with blood pressure 113/65 mmHg (MAP 81), HR 100 bpm, wita a temperature 36.5, and a blood oxygen saturation level of 96% room air. Laboratory tests revealed with potassium level 1.75 mmol/L, sodium level 139 mmol/L, Chloride level 119 mmol/L, and slightly increased of creatine 1.38 mg/dL. At blood gas analysis revealed patient with severe uncompensated metabolic acidosis with pH 7.09, pCO2 44.6, decreased HCO3 13.6 mmol/L with Base Excess -16.5 mmol/L. An electrocardiogram showed non-sustained VT and on echocardiography, no structural abnormalities of the heart were found. We immediately plan a CVC to quickly correct potassium and metabolic acidosis for the patient. After correcting potassium and metabolic acidosis, the ECG returned to sinus rhythm. After evaluating the patient, we discovered that polyuria was accompanied by potassium loss from renal. The patient is currently presenting with a diagnosis of diabetes insipidus with a differential diagnosis of renal tubular acidosis.

Conclusions:

Non-Sustained is one of the arrhythmias that requires attention, as it might trigger lethal VT and torsade de pointes. However, not all non-sustained VT require immediate treatment with anti-arrhythmics. The physician must first rule out reversible causes. Adult and congenital structural heart disease, acquired and inherited channelopathies, infiltrative cardiomyopathy, electrolyte imbalances (hypokalemia, hypocalcemia, hypomagnesemia), illicit drugs such as cocaine or methamphetamine, and digitalis toxicity are additional causes of wide complex ventricular tachycardia. A common cause is hypomagnesemia followed by hypokalemia. This patient was suffering from a severe low potassium level and severe uncompensated metabolic acidosis.

KEYWORD: *Non-sustained VT; Low potassium level; Lethal VT.*

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Long-Standing Complete Heart Block complicating Atrial Septal Defect in Paediatric : a Case Report

I. P. Farissa[1] ,A. Alamsyaputra[1] ,S. Y. Pradana[1] ,A. Soetadji[2]

[1] Department of Cardiology and Vascular Medicine, Medical Faculty Diponegoro University, Kariadi General Hospital, Semarang, Indonesia

[2] Department of Pediatric cardiology, Medical Faculty Diponegoro University, Kariadi General Hospital, Semarang, Indonesia

Background: Congenital AV block commonly occurs due to passively transferred auto antibodies in maternal connective tissue disease or in association with complex congenital heart disease (CHD) such as congenitally corrected transposition of the great arteries (CC-TGA). However, the aetiology in childhood variant could be in association with complex CHD, acquired as in post atrial septal defect (ASD) closure, genetics and rarely inherited cardiac conduction disorder.

Case illustration: A-9 years old boy with exertional dyspneu a year prior to admission. He was known to have Atrial Septal Defect when he was 6 years old. His mother denied any maternal disease. Physical examination showed well-nourished and good stature, heart rate of 48 beats per minute. ECG revealed total atrioventricular block with junctional escape rhythm, PVCs, and right axis deviation. Echo showed large non deviceable ASD left to right shunt with mild TR confirmed with right heart catheterization of high flow low resistance and Pulmonary Artery Pressure 36 mmHg. Later he underwent surgical ASD closure with VVIR Permanent Pacemaker Implantation.

Conclusions:

The chronic left-to-right shunt associated with ASDs leads to increased hemodynamic load and geometric remodelling. This chronic volume stress leads, in turn, to electrical remodelling that may precipitate development of arrhythmias. Both sinus node and AV nodal conduction abnormalities have been reported in patients with ASDs. Risk of developing sinus node dysfunction is correlated with larger shunt size and older age at closure In the AHA 2012 guidelines, permanent pacemaker was suggested for symptomatic complete congenital heart block, irreversible AV nodal disease, and asymptomatic with a ventricular rate less than 55 bpm or less than 70 bpm in complete congenital heart block with major cardiac structural defects

KEYWORD: *ASD, Complete Heart Block.*

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Complete Congenital Heart Block without Complex Congenital Heart Defect : Predict the Unpredictable

I. P. Farissa ,A. Alamsyahputra ,S. Y. Pradana[1] ,A. Prayitno[2]
[1] Department of Cardiology and Vascular Medicine, Medical Faculty Diponegoro University, Kariadi General Hospital, Semarang, Indonesia
[2] Department of Pediatric cardiology, Medical Faculty Diponegoro University, Kariadi General Hospital, Semarang, Indonesia

Background: Congenital heart block (CHB) is rare disorder that has a higher mortality when associated with structural congenital heart defects. Isolated CHB frequently results from neonatal lupus erythematosus, a disease associated with transplacental passage of maternal anti-Ro/SSA and/or anti-La/SSB antibodies to the fetus. It requires a high index of suspicion when there is a finding of fetal bradycardia for early diagnosis and appropriate planning of perinatal management in centers with facility for pacemaker treatment but this not often possible in low resource settings
Case illustration: A newborn was consulted in our department for further evaluation of bradycardia. Physical examination showed well developed neonate with heart rate 59 bpm with osygen saturation 95% room air. There was no heart failure. The ECG revealed total atrioventricular block with P rate 160 bpm and QRS rate 60 bpm. The Trans Thoracal Echocardiography revealed persistent foramen ovale and patent duct arteriosum.

Conclusions:
CHB is an uncommon disorder, but may be associated with a high morbidity and mortality. Diagnosing and managing of CHB in low resource settings are still challenging. A proper pregnancy follow up in antenatal diagnosis and well planned perinatal management are needed to optimize the outcome. This emphasizes the clinical value of high quality antenatal care and proper screening.

KEYWORD: *Congenital Heart Block, rare disease.*

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Orthodromic Atrioventricular Reentrant Tachycardia associated with Wolff-Parkinson-White Syndrome which Unresponsive to Cardioversion: A Case Report

I. K. Dewi[1] ,N. A. A. Pratama[1] ,F. Y. Mufaza[1] ,M. A. Sjughiarto[2]
[1]General Practitioner, SLG General Hospital, Kediri, Indonesia
[2]Department of Cardiology and Vascular Medicine, SLG General Hospital, Kediri, Indonesia

Background : Wolff-Parkinson-White (WPW) syndrome is one of the most common pre-excitation disorders. It is characterized by the presence of accessory pathways between the atria and ventricles that allow alternative pathways for ventricular depolarization. WPW syndrome is usually asymptomatic, but can also lead to life-threatening Supraventricular Tachycardia (SVT). Orthodromic Atrioventricular Reentrant Tachycardia (AVRT) is the most common SVT found in WPW Syndrome. Patients with Orthodromic AVRT may present with hemodynamic instability in which needs specific treatment such as cardioversion. We will report a case of WPW Syndrome with Orthodromic AVRT which unresponsive to cardioversion. The rhythm returned to sinus after administration of Amiodarone.

Case illustration : An 18-years-old male came to the ED with the chief complaint of heart palpitations since 4 hours earlier. He also experienced chest pain, shortness of breath, nausea, and vomiting. He was fully awake, GCS E4V5M6, BP 94/55 mmHg, pulse 191 x/minute regular, RR 26 x/minute. Laboratory examination only showed leukocytosis. His electrocardiogram showed SVT with regular and narrow QRS complex, and a heart rate of 189 beats/min. He received fluid loading, digoxin, and beta blocker as initial therapy. Then, three times synchronized cardioversion of 50J, 50J and 100J was performed. Unfortunately, the electrocardiogram still showed SVT with a heart rate of 171 beats/minute. He then fell into cardiogenic shock. 100J Synchronized cardioversion was again performed followed by administration of antiarrhythmic agent Amiodarone and inotropic agent Dobutamine. Rhythm changed after continuous administration of Amiodarone. His electrocardiogram showed a sinus rhythm with a heart rate of 88 beats/minute with a shortened PR interval and delta waves which were suggestive of WPW syndrome.

Conclusions:
Clinicians should consider to give combined therapy between electrical and pharmacological cardioversion simultaneously in patients with hemodynamically unstable SVT which unresponsive to cardioversion alone.

KEYWORD: *Orthodromic Atrioventricular Reentrant Tachycardia (AVRT), Wolff-Parkinson-White (WPW) Syndrome, Cardioversion, Amiodarone.*

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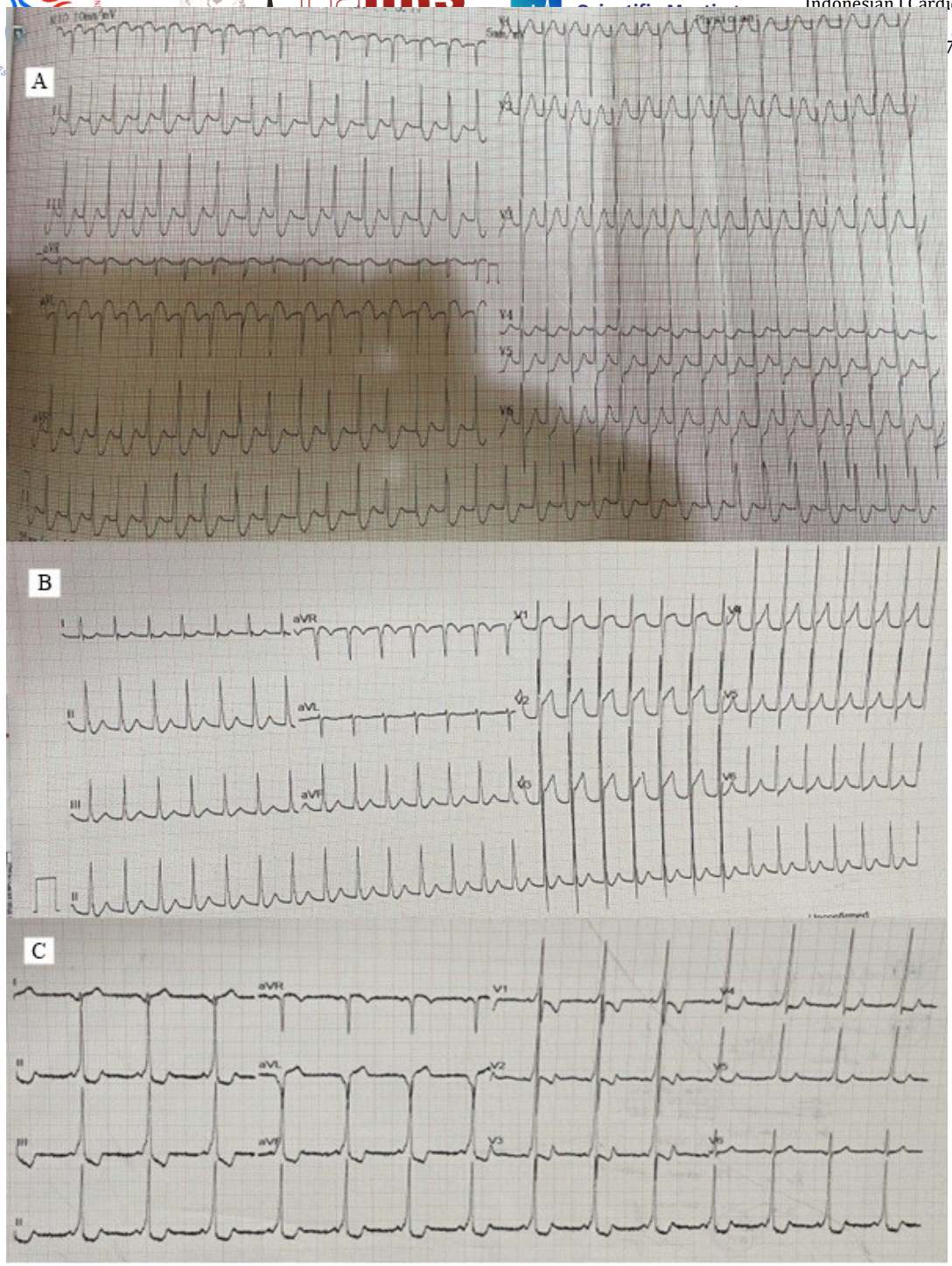

Figure 1. (A) ECG upon arrival shows SVT with regular and narrow QRS complex at a rate of 189 beats/minute. (B) ECG after 3rd cardioversion still shows SVT with regular and narrow QRS complex at a rate of 171 beats/minute. (C) ECG after 4th cardioversion followed by continuous administration of Amiodarone shows sinus rhythm with a shortened PR interval and delta waves at a rate of 88 beats/minute which are suggestive of WPW syndrome.

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Intraaortic Balloon Pump as A Bridging Management of Intractable Arrhythmia in Cardiogenic Shock Complicating Acute Myocardial Infarction: A Case Report

I. M. Sihotang[1] ,L. H. Hutapea[2] ,K. K. Ilyas[3] ,A. C. Lubis[4] ,T.B. Haykal[5] ,C. A. Andra[6]
[1,2]Cardiology Resident at Cardiac Center H. Adam Malik Hospital, Faculty of Medicine Universitas Sumatera Utara, Medan, Indonesia
[3,4,5,6]Cardiologist at Cardiac Center H. Adam Malik Hospital, Faculty of Medicine Universitas Sumatera Utara, Medan, Indonesia

Backgroud: In patient with cardiogenic shock after acute myocardial infarction (AMICS) even after successful revascularization, severe left ventricular dysfunction, recurrent ventricular arrhythmia (VA) non-responsive to antiarrhythmic therapies may cause worsening of cardiac function and haemodynamic deterioration. The use of Intraaortic Balloon Pump (IABP) in setting of haemodyanmically unstable VT may allow rhythm stabilization and can be effective as a bridge providing haemodynamic stability and optimize management post revascularization therapy.
Case illustration: We report a 40-year-old male presented cardiogenic shock and Ventricular Tachycardia (VT) in the early post-anteroextensive MI period. He complained severe chest pain and cold sweating. He was a smoker without any other risk factor. He has no clear family history with cardiovascular disease. Echocardiogram showed a severe left ventricular dysfunction (38% ejection fraction). We performed coronary angiography, found a total occlusion in Left Anterior Descending Artery (LAD) artery, and placed a stent in Proximal – Mid LAD. After revascularization, the patient still have unstable haemodynamic, recurrent attack of VT, which become more frequent and non-responsive to medical treatment, and decision was made to place the Intraaortic Balloon Pump (IABP). Haemodynamic gradually improved, VT attacks dissapeard, no other arrythmia was found and the patient was weaned from IABP after 5 days.

Conclusions:
Routine use of IABP in patient with AMICS is not recommended, but many patients with CS cause may benefit from IABP at least for haemodynamic improvement. In this case, Placing the IABP not only improved the haemodynamic but also allowing rhythm stabilization and bridging decompensated patient to optimalization therapy.

KEYWORD: *Intraaortic Balloon Pump, Cardiogenic Shock, Ventricular Arrhythmia, Acute Myocardial Infarction.*

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Mechanical Circulatory Support As a Bridge to Optimize Therapy of Refractory Ventricular Arrhythmia in Acute Myocardial Infarction Complicated by Cardiogenic Shock: A Case Report

I. M. Sihotang[1] ,L. H. Hutapea[2] ,K. K. Ilyas[3] ,A. C. Lubis[4] ,T. B. Haykal[5] ,C. A. Andra[6]
[1,2]Cardiology Resident at Cardiac Center H. Adam Malik Hospital, Faculty of Medicine Universitas Sumatera Utara, Medan, Indonesia
[3,4,5,6]Cardiologist at Cardiac Center H. Adam Malik Hospital, Faculty of Medicine Universitas Sumatera Utara, Medan, Indonesia

Backgroud: In patient with cardiogenic shock after acute myocardial infarction (AMICS) even after successful revascularization, severe left ventricular dysfunction, recurrent ventricular arrhythmia (VA) non-responsive to antiarrhythmic therapies may cause worsening of cardiac function and haemodynamic deterioration. The use of Intraaortic Balloon Pump (IABP) in setting of haemodyanmically unstable VT may allow rhythm stabilization and can be effective as a bridge providing haemodynamic stability and optimize management post revascularization therapy.
Case illustration: We report a 40-year-old male presented cardiogenic shock and Ventricular Tachycardia (VT) in the early post-anteroextensive MI period. He complained severe chest pain and cold sweating. He was a smoker without any other risk factor. He has no clear family history with cardiovascular disease. Echocardiogram showed a severe left ventricular dysfunction (38% ejection fraction). We performed coronary angiography, found a total occlusion in Left Anterior Descending Artery (LAD) artery, and placed a stent in Proximal – Mid LAD. After revascularization, the patient still have unstable haemodynamic, recurrent attack of VT, which become more frequent and non-responsive to medical treatment, and decision was made to place the Intraaortic Balloon Pump (IABP). Haemodynamic gradually improved, VT attacks dissapeard, no other arrythmia was found and the patient was weaned from IABP after 5 days.

Conclusions:
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KEYWORD: *Intraaortic Balloon Pump, Cardiogenic Shock, Ventricular Arrhythmia, Acute Myocardial Infarction.*

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HD-grid for Mapping Improvement of Right Posterolateral Accessory Pathway

Irnizarifka[1,3] ,S. B. Raharjo[1,2] ,D. A. Hanafy[1,2] ,D. Y. Hermanto[1,2] ,A. I. Supit[1] ,Y. Yuniadi[1,2]

[1]National Cardiovascular Centre Harapan Kita, Jakarta, Indonesia

[2]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

[3]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Indonesia

Background: The right posterolateral area is a thought-provoking anatomic zone since its stabilization of mapping catheter became a challenge. Accessory pathways (AP) located in this area pose a relatively difficult task for electrophysiologists, especially in patient with large right heart and significant tricuspid regurgitation (TR). Despite maneuver with long sheath was already well established, the use of HD-grid thought to add better precision of intracardiac AP localization.

Case illustration: A 39-years old man with manifest right-sided WPW and recurrent palpitation of documented aberrant AVRT was scheduled for AP conventional ablation. He had history of mitral valve repair in 1990, and currently has severe TR with decrease RV function (TAPSE 10 mm). Conventional non-irrigated radio-frequency ablation (RFA) catheter mapping with SL-0 long sheath support was challengingly not be able to get accurate site of the AP. This was not unexpected since severe TR tends to undulate the catheter. Hence, HD-grid catheter was used in order to yield meticulous mapping at right posterolateral area. Despite there was no antegrade fused AV EGM, right ventricular apical (RVA) pacing evinced retrograde fused VA at right posterolateral (HD-grid at B1-C1). Delta wave disappeared and VA separated after 5 seconds of multiple RFAs (45° Celsius, 30-40 Watt, 120 seconds) delivered to this area during RVA pacing. After 20 minutes of evaluation, neither delta wave nor inducible tachyarrhythmia were evidenced.

Conclusions:

HD-grid catheter could potentially be a secret armament to aid stabilization and thus get meticulous conventional mapping of right posterolateral AP through its stiff shaft and soft-deflectable grid. To the best of our knowledge, this HD-grid guided conventional mapping technique is the first to be reported.

KEYWORD: *Right posterolateral accessory pathway, HD-grid catheter, unstable conventional catheter mapping.*

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TACKLING VENTRICULAR ARRHYTMIAS IN PREGNACY WITH PROPRANOLOL : AN UNDERAPPRECIATED DRUG

I. Darwis[1] ,F. Hidayati[1] ,E. Maharani[1] ,L. K. Dinarti[1] ,D. W. Anggrahini[1]
[1]Department of Cardiology and Vascular Medicine
Faculty of Medicine, Public Health and Nursing
Universitas Gadjah Mada

Background: Arrhythmias can increase maternal and fetal mortality. Pregnancy conditions can also trigger an exacerbation of pre-existing arrhythmias. Electrophysiological studies and ablation therapy in pregnant women have a high enough risk. Therefore, conservative therapy with antiarrhythmic drugs can be a therapeutic modality by taking into account the effectiveness and considering the side effects on the mother and fetus. Propranolol has a fairly potent effect for arrhythmias in pregnant women with minimal side effects on the fetus.

Case illustration: Mrs. TS, 24 years old, G1P0A0, 26 weeks pregnancy with high burden PVC RVOT origin modified WHO class risk II. Echocardiographic showed no ARVD with LVEF 67%. Propranolol 10 mg tid was given orally. The patient gave birth vaginally by vacuum extraction at term, birth weight of 3100 mg, APGAR score 8/9. No congenital abnormalities were found in the infant. Mrs. DEPS, 31 years old, G2P0A1 24 weeks pregnancy, non-sustained RVOT ventricular tachycardia modified WHO class risk III. No ARVD features with LVEF 66% were found by echocardiography. Propranolol 10 mg tid was administered orally. The patient gave birth by sectio caesarean at term due to patient preference with a birth weight of 3200 mg. Apgar score 8/9. No congenital abnormalities were found in the infant. Mrs. ES, 27 years old, G2P0A1 36 weeks pregnancy, PVC bigeminy RVOT origin modified WHO class risk II. On echocardiography examination, there was no ARVD with LVEF 70%. Propranolol 10 mg tid was given orally. The patient gave birth by sectio caesarean at term due to cephalopelvic disproportion with a birth weight of 2950mg. Apgar score 9/10. No congenital abnormalities were found in the infant.

Conclusions:
The use of propranolol in pregnant women with arrhythmias is quite effective for the control of arrhythmias with low risk to the fetus.

KEYWORD: *Propranolol, pregnancy, ventricular arrhythmia*.

Criteria	Case I	Case II	Case III
Pregnancy	G1P0A0	G2P0A1	G2P1A0
Ventricular arrhytmia	VPC RVOT frequence	Non Sustained RVOT Ventricular tachycardia and RBBB type	Ventricular extrasystole bigeminy
Modified WHO	Class Risk III	Class Risk III	Class Risk II
Echocardiography	Ejection fraction 67%, ARVD (-)	Ejection fraction 66%, ARVD (-)	Ejection fraction 70%, ARVD (-)
Antiarrythmia therapy	Propranolol 10mg/8 hours tablet orally	Propranolol 10mg/8jam tablet orally	Propranolol 10mg/8jam tablet orally
Antriarrytmia therapy during delivery	Lidocain	Lidocain	Lidocain
Time to delivery	Aterm	Aterm	Aterm
Mode of delivery	Vaccum extraction	Sectio caesarean	Sectio caesarean
Weight of birth	3100 mg	3200 mg	2950 mg
APGAR Score	8/9	8/9	9/10
Congenital defect	No	No	No

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Pacemaker-induced Cardiomyopathy Upgrade to Left Bundle Pacing

I. J. T. Allo[1] ,G. Karwiky[1]

[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Padjadjaran, Dr. Hasan Sadikin General Hospital, Bandung, Indonesia

Background: Pacemaker-induced cardiomyopathy (PICM) is reported in 10.7-13.7% of patients receiving right ventricular pacing (DDDPM). Therefore, PICM can be evaluated to deploy a more sophisticated technique, including the left-sided His Bundle Pacing (HBP). We present a patient with dyspnea following DDDPM implantation with coronary artery disease (CAD) who received an upgrade to HBP.

Case illustration: A 78-year-old male visited our cardiovascular clinic complaining of progressive dyspnoea for three years; four years following DDDPM implantation due to atrioventricular block. The complaint has worsened, and he has experienced orthopnoea for a while. His electrocardiography (ECG) showed pacing beats with left bundle branch block morphology (QRS duration: 200ms). The echocardiography showed dilated all chambers and eccentric left ventricular (LV) hypertrophy with severely reduced LV ejection fraction (LVEF) of 16%. He underwent an angiography which indicated a three vessels disease with a bifurcation lesion. Myocardial perfusion scintigraphy suggested myocardial ischemia at apex, apicoseptal, apicolateral, inferolateral, and inferior wall segments, yet all segments were viable. He declined a bypass, and thus, we conducted consecutive percutaneous coronary interventions. Three drug-eluted stents (DES) were placed at the proximal right coronary artery to the right posterolateral artery. Later, we placed two DES at the left main artery to the left anterior descending artery and inflated a drug-coated balloon at his left main to the left circumflex artery. However, despite the total revascularization, his LVEF remained low. Therefore, his DDDPM was upgraded to a left-sided HBP. On evaluation, his ECG showed a pacing QRS duration of 115 ms. His LVEF was increased six months later (LVEF: 28%), accompanied by improved symptoms proven by an improvement in his functional class (III to II) and Minnesota Living Heart Failure Questionnaire score (66 to 27).

Conclusions:

In managing PICM, several factors should be considered, including CAD and an upgrade to HBP. This case was a patient with CAD and PICM whose cardiac condition and symptoms improved following revascularization and HBP. His post-HBP ECG indicated an intact infrahisian conduction system. Further studies should be conducted to understand a better technique for the benefit of HBP in a patient with PICM or bradycardia.

KEYWORD: *His Bundle Pacing, Pacemaker, Pacemaker-induced Cardiomyopathy.*

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Serial electrocardiographic changes during provocative testing with low dose oral Flecainide in patient with suspected Brugada syndrome

I. Rahma[1,2] ,M. Amir[1,2]
[1]Faculty of Medicine, University of Hasanuddin, Makassar, Indonesia
[2] Cardiac Centre, Wahidin Sudirohusodo Hospital, Makassar, Indonesia

Background: Brugada syndrome (BrS) is a genetic disease characterized by coved ST-segment elevation (STE) in right precordial leads (V1 to V3) that predisposes to life-threatening ventricular tachyarrhythmia and sudden cardiac death (SCD). The electrocardiographic signature is dynamic and often concealed but can be unmasked by potent sodium channel blockers such as Flecainide.
Case illustration: A 21-year-old asymptomatic man was referred to outpatient clinic for further evaluation ohis electrocardiography (ECG) as a part of army education recruitment. None of his relatives died of heart problems. The physical examination was unremarkable, with body weight of 57 kg. Resting 12-lead ECG showed saddle-back STE in V2 suggestive of Brugada pattern. Provocative testing was performed using low dose oral Flecainide (300 mg) to clarify the diagnosis. The test was closely monitored with continuous ECG monitoring for 24 hours. Serial ECGs with standard lead and upper right lead (one ICS above standard position) were performed at 15, 30, 60, 90 min, hourly (first 6 hours), then every 2 hours. The result was positive with coved STE developed in leads V1-V3 30 minutes after drug administration and reached maximal positivity at 3.5 hours with STE 11 mm in V1 (Figure 1). No episode of ventricular arrhythmia was observed during the procedure.
Potent sodium channel blocker facilitates loss of the right ventricular epicardial action dome (plateau phase) and considerable reduction of the epicardial action potential duration that creates a transmural voltage gradient and manifests as an STE in the right precordial leads. Time to positivity was fairly rapid, and the time to maximal STE seemed close to peak Flecainide plasma level (between 1 to 6 hours).

Conclusions:
Provocative testing with low dose oral flecainide is an effective alternative method to unmask type-1 Brugada ECG.

KEYWORD: *Brugada syndrome, Flecainide challenge test, electrocardiogram.*

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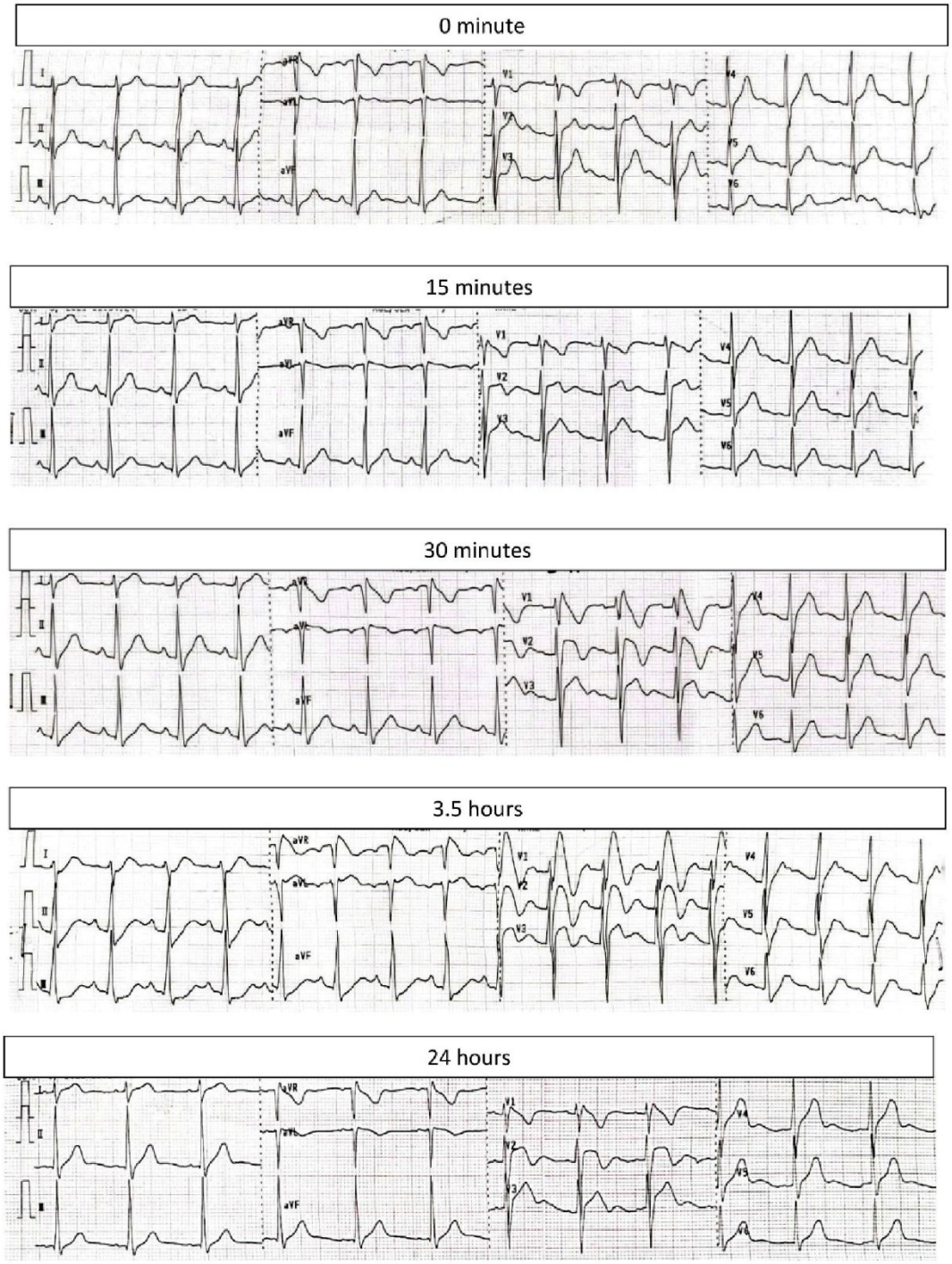

Figure 1 Serial electrocardiography (standard lead) during provocative testing with low dose oral Flecainide

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MOTHER'S HEART IN BURDEN:

LATE DIAGNOSIS OF EBSTEIN ANOMALY DURING PREGNANCY:

A RARE CASE REPORT

J. Lesmana[1] ,M. Ramadhan[1]

[1]General Practitioner at Jantung Diagram Hospital

Background: Congenital heart disease is a concerning disease among those who are affected since it affects the patient's life quality. One of which is Ebstein Anomaly (EA) with prevalence of 1 in 200.000 birth with clinical characteristic of displacement of tricuspid valve that causes atrialization of the right-side heart and may accompanied with other findings, such as atrial septal defect or an accessory pathway. Special population, especially in women of childbearing age is at risk because they are asymptomatic despite having EA. Thus, they are compromise to hemodynamic consequence and risk of maternal complication during periods of pregnancy until delivery.

Case illustration: A 25-year-old woman came to the emergency department with worsening shortness of breath in the last week during rest and was accompanied by decreased physical activity tolerance. Symptoms are sometimes accompanied by edema on both ankles and bluish colour of the lips and nail bed. On admission, the patient was difficulty breathing, pale and weak. On examination obtained as follows: BP: 160/121 mmHg, HR: 105 bpm, RR: 24 rpm, SpO_2: 82% without oxygen supplement. JVP was elevated, bilateral crackles at both lung bases, 5/6 holosystolic murmur at LLSB with S3 gallop, bilateral pretibial edema. The blood test was within normal limit, and routine MSCT Thorax showed mild cardiomegaly with bilateral lung congestion. Her previous clinical evaluation in detecting EA was with echocardiography, and cardiac multi-slice computed tomography before and after delivery of her first child, respectively. She was well controlled with loop diuretic, phosphodiesterase type 5 inhibitor and beta-blocker but unable to care for her child due to her heart condition. The patient was discharged after symptoms were relieved and were planned for further interventional evaluation.

Conclusions:

Although EA is rare, it has high morbidity and mortality, especially in women of childbearing age since it can be asymptomatic during childhood and be missed diagnose in early age. Echocardiography is a non-invasive imaging modality and also can be used for early detection of EA in women who are planning pregnancy to avoid unwanted maternal complication and minimize mortality in mother and child incidence.

KEYWORD: *Ebstein Anomaly, Echocardiography, Pregnancy.*

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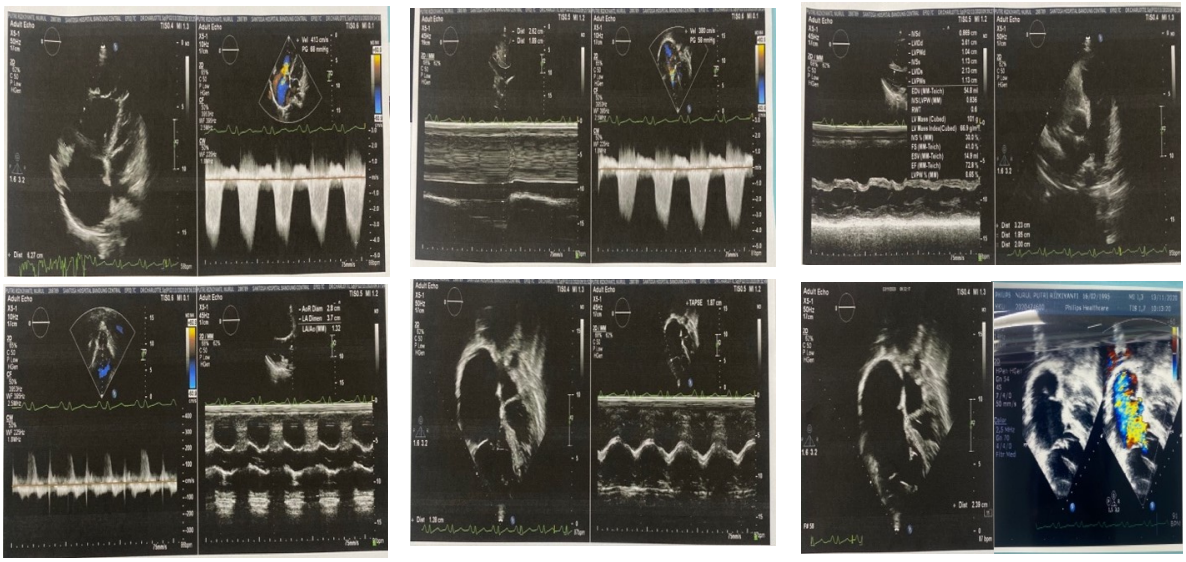

Figure 4. TTE image showing large right atrial chamber with displacement of tricuspid valve, normal RV function and high probability of pulmonary hypertension. The doppler showed a severe tricuspid regurgitation.

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Inadvertent Malposition of Temporary Pacemaker Lead – Stab to The Heart

J. Sihura[1] ,R. K. Marsam[2] ,F. Hidayati[2] ,E. Maharani[2]
[1]Resident of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing Gadjah Mada University, Yogyakarta, Indonesia
[2]Staf of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing Gadjah Mada University, Yogyakarta, Indonesia

Background: Displacement of temporary pacemaker (TPM) lead is a rare but life-threatening complication with an incidence of 0.1–6%. After TPM implantation, perforations may occur acutely, at lead insertion, or alternatively be delayed for several times. Although most patients that develop this complication are asymptomatic, some experience stabbing chest pain, shortness of breath, pacemaker malfunction, and cardiac tamponade due to cardiac rupture.
Case illustration: A 79-years-old male came to emergency department with history of syncope 6-hours before admitted. Electrocardiography (ECG) showed complete heart block with junctional escape rhythm 45 bpm. Marked leucocytosis with normal electrolyte level were noted. Transvenous TPM procedure guided fluoroscopy was performed. ECG evaluation showed ventricular pacing with right bundle branch block (RBBB) morphology. Echocardiography revealed the tip of lead migrated to apical left ventricular. Cardiac computed tomography confirmed that the TPM lead passed through the right atrium - apical right ventricle – intraventricular septum - apical left ventricle - pericard which the lead tip advanced to pericardial space near diaphragm. The hemodynamic was stable and no sign of tamponade. Permanent pacemaker implantation was planned with back up surgery. However, patient positive Covid-19 2 days later and the ECG showed normal sinus rhythm after recover from Covid-19. Removal TPM lead by transvenous without complication and PPM implantation aborted because AV-block resolved.

Conclusions:
Early sign of TPM lead displacement may be assessed by symptom, ECG, echocardiography, and cardiac CT. The RBBB pattern in patient with transvenous TPM should be suspicious of displacement lead to LV. Echocardiography and cardiac CT has an excellent diagnostic capability in the setting of iatrogenic sub-acute and chronic cardiac perforation. Management of lead perforation includes two approaches includes transvenous removal and conventional median full sternotomy.

KEYWORD: *Complete heart block, TPM lead displacement, cardiac perforation*.

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Lidocaine Induced Systemic Toxicity : from Local to Systemic Life-Threatening Event – A Case Series

L. D. Pradipta[1] ,A. Alamsyaputra[1] ,I. P. Farissa[1] ,A. Yudanto[1] ,P. Ardhianto[1]
[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Diponegoro University – Dr. Kariadi Central General Hospital Semarang, Indonesia

Background: Local anesthetics such as lidocaine have been widely used in many medical practices. It is not only limited to anesthesia and surgery, but also frequently used in internal medicine and primary care setting for bedside procedures. In spite of its frequent use, several physicians are unacquainted with local anesthetic systemic toxicity (LAST) and the treatment as well. Systemic toxicity from local anesthetics is rarely seen but can be potentially lethal by causing seizures, arrhythmias and even cardiovascular arrest. The site of administration and the dose of the local anesthetic delivered are independent risk factors for systemic toxicity. We demonstrate case series in patient who underwent permanent pacemaker implantation procedure. Our aim is to raise awareness among physician to identify and understand the warning signs of local anesthetic toxicity, its pathophysiology, and the use of intravenous lipid infusion as the treatment of choice to reverse the symptoms.
Case illustration: We delivered cases of a 66-years-old man with BMI 19.9; 54-years-old man with BMI 18.4; and 58-years-old man with BMI 19.5; underwent pacemaker implantation procedure under local anesthesia with lidocaine 2%. First two cases underwent permanent pacemaker with stable hemodynamic. The last patient underwent temporary pacemaker due to complete heart block. The patients denied having any allergies, alcohol, or illicit drug use. Physical examinations were unremarkable. We gave 360 mg, 320 mg, and 400 mg total dose of lidocaine, respectively. Shortly after receiving the lidocaine, first and second patient developed into blurry vision, seizure, and unconscious, moreover last patient had cardiac arrest and rescucitated. LAST was suspected, and the patient was given a 100 ml bolus of 20% intravenous lipid emulsion (intralipids) with restoration of alertness and vital sign within minutes after intralipids administration.

Conclusions:
Local anesthetic systemic toxicity (LAST) is a life-threatening complication. Early identification of its sign and symptoms is a key to prevent mortality. Once LAST developed, administration of intravenous lipid infusion is mandatory to reverse the toxicity and prevent mortality.

KEYWORD: *Local anesthetic systemic toxicity, lidocaine, lipid emulsion.*

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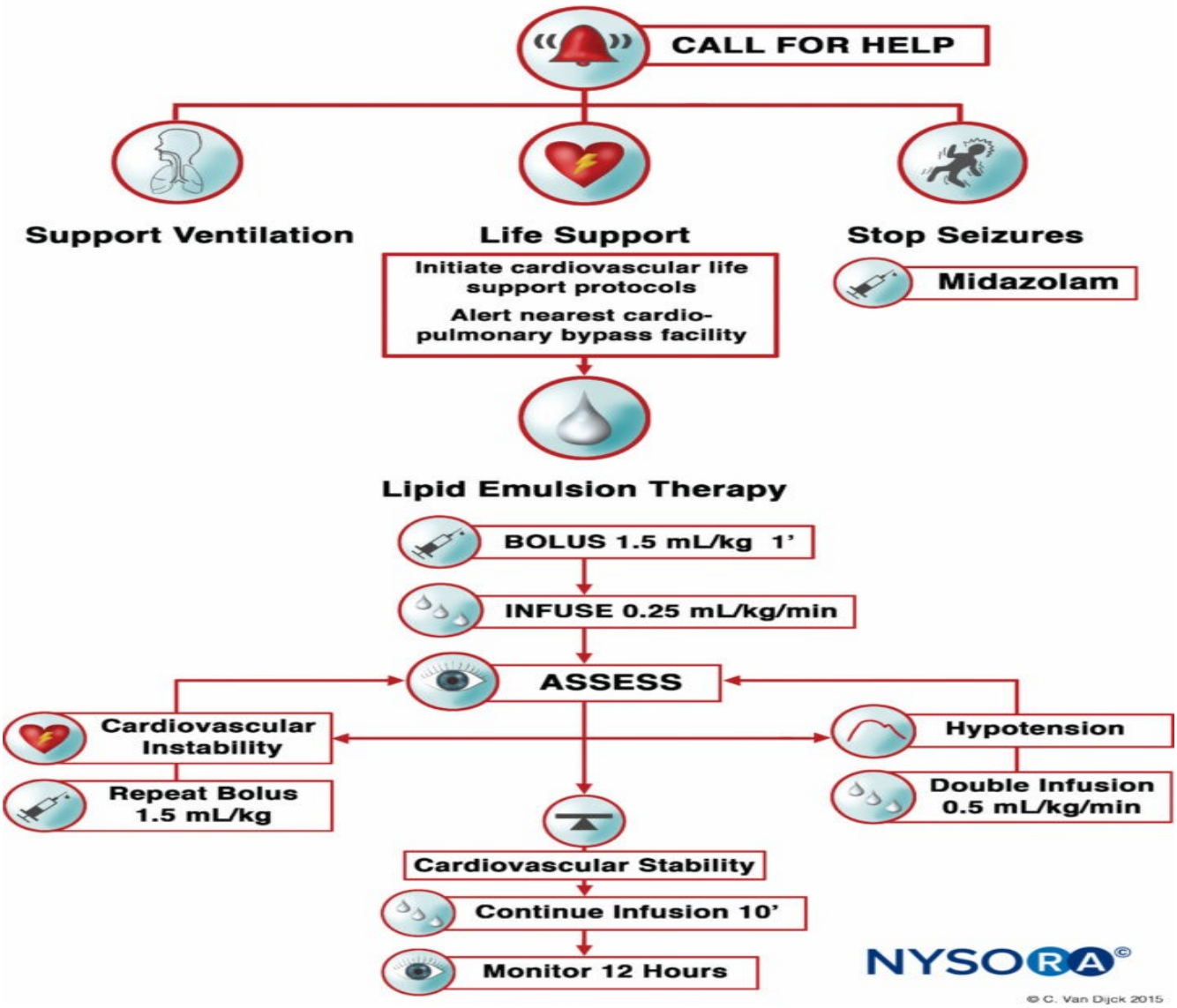
Local Anesthetic Systemic Toxicity



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Fever-Induced Brugada Syndrome: The Forgotten ST-Segment Elevation, Insight From Rural Area

M. F. Adam[1,2] ,S. Erriyanti[3] ,D. Y. Hermanto[4] ,D. A. Hanafy[4] ,S. B. Raharjo[4] ,Y. Yuniadi[4]
[1]Pupuk Kaltim Hospital, Bontang, East Kalimantan, Indonesia
[2]Department of Cardiology and Vascular Medicine, Faculty of Medicine Universitas Indonesia, National Cardiovascular Center Harapan Kita, Jakarta, Indonesia
[3]Hermina Hospital, Bandar Lampung, Sumatra, Indonesia
[4]Division of Arrhytmia,, Department of Cardiology and Vascular Medicine, Faculty of Medicine Universitas Indonesia, National Cardiovascular Center Harapan Kita, Jakarta, Indonesia

Background: Brugada syndrome is an abnormal ECG pattern characterized by coved type ST-segment elevation pattern (Type 1) in right precordial leads associated with malignant arrhythmia that can lead to sudden cardiac death (SCD). Brugada pattern might appear more prominent in certain situation such as fever. We aim to highlight the importance of identifying fever-induced-Brugada in emergency settings especially in rural area in Indonesia.

Case illustration: We present 2 cases of a 59-year-old man and a 35-year-old male with fever > 39^0C. Our first patient in Bontang, East Kalimantan had chronic cough accompanied by night sweating for the past 1 month while our second patient in Lampung, Sumatra had right lower abdominal pain for the past 3 days. Both patients' initial ECG showed an rSR' pattern in V1 and V2, with coved ST segment elevation > 2 mm in V1 followed by an inverted T wave consistent with a type 1 Brugada pattern. Laboratory findings of both patients showed leukocytosis of 22.000 and 15.000, respectively. Our first patient was later diagnosed with active tuberculosis infection then subsequently treated with broad-spectrum antibiotics and anti-tuberculosis drugs while our second patient was diagnosed with acute appendicitis and underwent appendectomy. Fever resolved within 2 days with normal ECG findings when both patients were no longer feverish.

Conclusions:
It is necessary to identify the classical ST-segment elevation pattern of type 1 Brugada and point out the trigger especially in patients with signs and symptoms of infection so that appropriate treatment can be given.

KEYWORD: *Brugada syndrome, fever, sudden cardiac death.*

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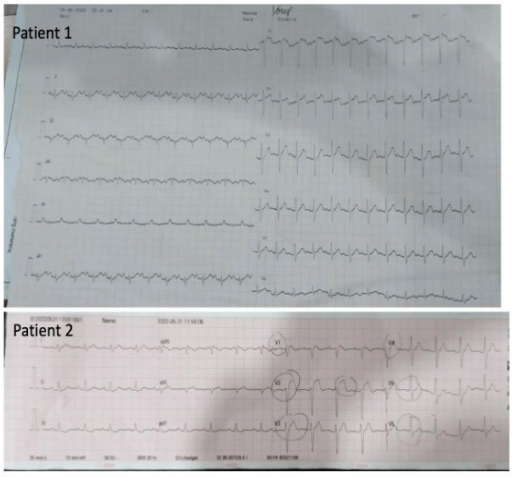

Fig1. Initial ECG on admission, brugada pattern appeared during fever on both patient

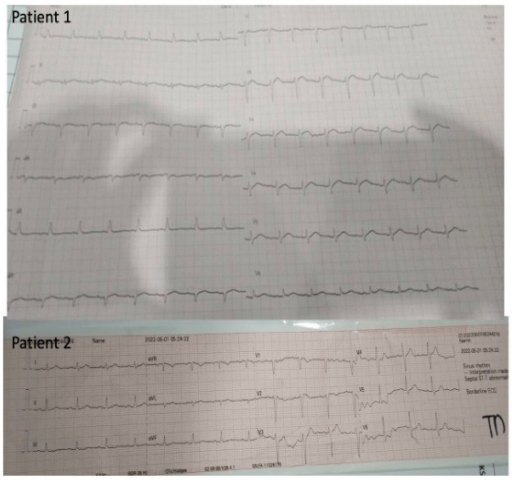

Fig2. ECG after fever resolved: no brugda pattern was identified

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Tombstone or Shark Fin Pattern, a Sign of a Poor Prognosis in Patients with ST-segment Elevation Myocardial Infarction

M.R. A. Shiddieqy[1] ,S. Inayasari[1] ,N. Sukmadi[1] ,L. Pribadi[2] ,M. G. Suwandi[2]
[1] General Practitioner, RSPAU dr. S. Hardjolukito, Yogyakarta, Indonesia
[2] Cardiologist, RSPAU Dr. Suhardi Hardjolukito, Yogyakarta, Indonesia

Background: The triangular QRS-ST-T waveform (TW) pattern, also called 'tombstone' or 'graveyard' and more recently 'shark fin' pattern, is a rare electrocardiography pattern that is associated with a poor prognosis in patients with ST-segment elevation myocardial infarction. This ECG pattern consists of blurring of the QRS complex due to its fusion with the ST-segment and the T-wave showing a triangular lambda pattern where a positive deflection in the leads suggests ischemia in corresponding areas. This ECG pattern may be misdiagnosed as wide complex tachycardia or the ECG changes of hyperkalemia. Thus, differentiating it from other conditions causing similar ECG changes and prompt management is highly important to save the patient from serious complications.

Case illustration: 44 Years old male came to our emergency room unconscious, two hours before the patient rode a bicycle, and then the patient complained of sudden typical chest pain, agitation, and diaphoresis, he was a smoker. The patient in emergency room was unconscious, pulse impalpable, and didnt breath. CPR was conducted for 45 minutes, the ECG showed ventricular tachycardia, and after intubated, defibrillated seven times, six times epinephrine intravenous, and 300 mg and 150 mg of Amiodarone intravenous, the patient ROSC. Patient blood pressure was 80/40 mmHg, heart rate was 110 bpm. The laboratory results showed CKMB elevated 163. The ECG showed ST elevation in V1-V6, lead 1, and aVL with giant R waves. Patient was diagnosed with Extensive STEMI Anterior Killip IV, Cardiogenic Shock SCAI C, ROSC Post Cardiac arrest mode VT/VF. PCI was established, coronary angiography showed proximal total occlusion of the left anterior descending coronary artery.

Conclusions:
The shark fin waveform is an uncommon but high-risk ECG pattern of STEMI which should be diagnosed early and differentiated from other conditions causing similar waveforms such as wide-complex tachycardia and hyperkalemia. The shark fin STEMI is associated with a high risk of both ventricular fibrillation (VF) and cardiogenic shock, accounting for increased in-hospital mortality.

KEYWORD: *ACS, STEMI, Shark Fin ECG, Acute Myocardial Infarction, Cardiogenic Syock.*

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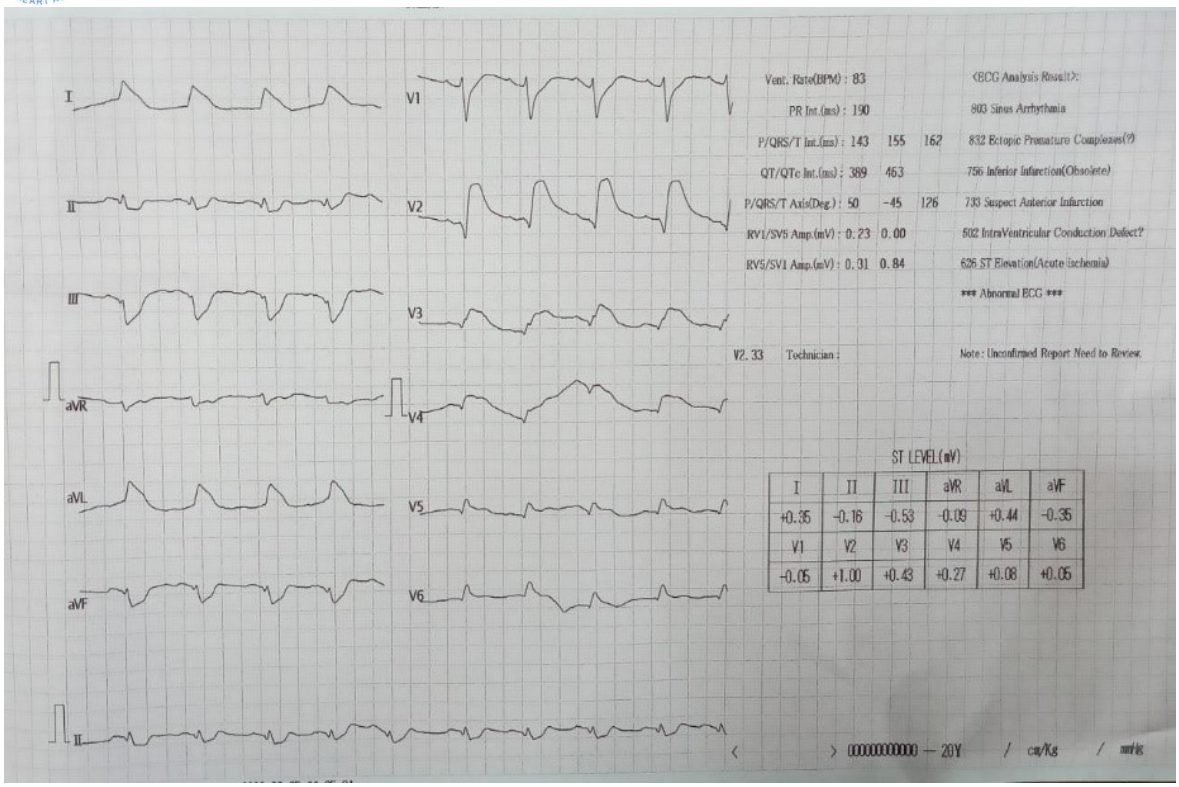

Figure 1 ECG at presentation showed a 1, aVL, V1-V6 ST-Elevation post ROSC

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Atrial Flutter 2:1 with Rapid Ventricular Response vs Sinus Tachycardia: Similar Yet Distinct Narrow QRS Complex Tachycardia

M.R. A. Shiddieqy[1] ,R. I. Rismawanti[2] ,N. Sukmadi[1] ,S. Inayasari[1] ,L. Pribadi[3] ,M.G. Suwandi[3]
[1]Internship, RS Pusat Angkatan Udara dr. S. Hardjolukito, Yogyakarta, Indonesia
[2]General Practitioner, RS Pusat Angkatan Udara dr. S. Hardjolukito, Yogyakarta, Indonesia
3Cardiologist, RS Pusat Angkatan Udara dr. S. Hardjolukito, Yogyakarta, Indonesia

Background: Atrial flutter is the most common arrhythmia and is characterized by an abnormal cardiac rhythm that can be fixed or be variable. Atrial flutter is easy to recognize when the ventricular rate is slow as 4:1 AV conduction. Approximately 10% of tachycardia thought to be SVT is due to atrial flutter. Atrial flutter should always be distinguished from SVT because the acute treatment of atrial flutter is different from that of SVT. This case report deals with an electrocardiogram showing atrial flutter with 2:1 AV conduction ratio associated with a regular alternation of narrow QRS complexes that are often missed in sinus tachycardia.
Case illustration: A man, 78 years old, came to ER with chief complaint worsening dyspneu for tbe last 9 hours before with dyspneu d' effort and orthopneu. His medical history was chronic heart failure (CHF) with 40% EF, the patient received spironolactone and furosemide therapy with poor compliance. At physical examination, was found BP 140/100, HR 140, SpO2 96% with NK 4lpm, cardiomegaly, rales in pulmo, pitting edema in inferior extremity and other normal values. The ECG showed atrial flutter 2:1 with rapid ventricular response (RVR), left atrial enlargement, left ventricular hypertrophy, old myocard infarc inferior. After being diagnosed with CHF CF III with EF 40% EC cardiomyopathy DE: IHD, IHD, atrial flutter 2:1 with RVR, he was administered 2 ampoules of furosemide and 1/2 ampoule of digoxin then admitted to the intensive cardiac room.

Conclusions:
Atrial flutter with 2:1 AV conduction is the most common presentation of atrial flutter in the acute setting. It is also the most difficult to detect and the most commonly missed tachycardia with a narrow QRS complex. The method used to differentiate atrial flutter 2:1 from supraventricular tachycardia (SVT) and STC is vagal maneuvers, such as carotid sinus pressure. In an emergency, however, Lewis lead may be considered. The difference in therapy is the foundation for the accuracy of the critical diagnosis in this case.

KEYWORD*: Atrial flutter, supraventricular tachycardia, sinus tachycardia, narrow QRS complex, arrhythmia.*

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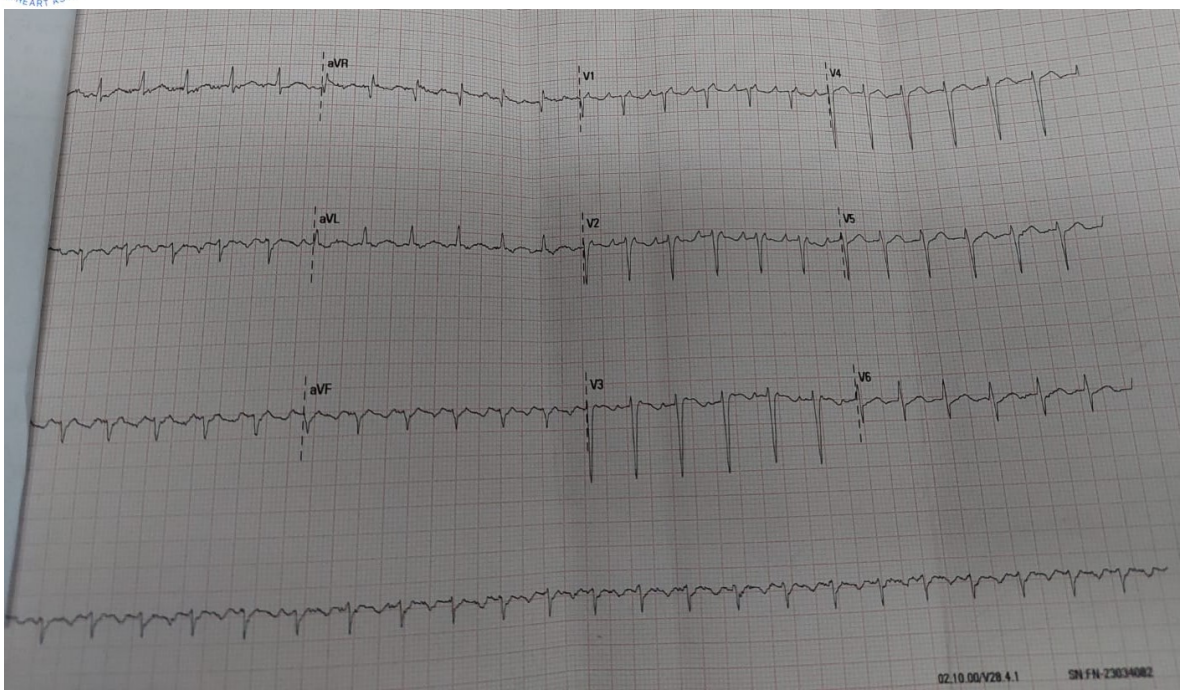

Figure 1 The ECG at ER showed Atrial Flutter 2:1 RVR, HR 140, LAE, LVH, OMI inferior

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Wellens's type B Syndrome: a Warning Sign of Critical Proximal LAD Artery Stenosis and Impending Anterior Myocardial Infarction

M. R. A. Shiddieqy[1] ,R. I. Rismawanti[1] ,N. Sukmadi[1] ,S. Inayasari[1] ,L. Pribadi[2] ,M. G. Suwandi[2]
[1]General Practitioner, RSPAU dr. S. Hardjolukito, Yogyakarta, Indonesia
[2]Cardiology and Vascular Medicine Department, RSPAU dr. S. Hardjolukito, Yogyakarta, Indonesia

Background: Wellens' syndrome is a subtle electrocardiographic (ECG) manifestation of critical left anterior descending (LAD) coronary artery stenosis. It includes biphasic or deeply inverted T waves in precordial leads. It is seen in a subset of patients with unstable angina during a chest pain-free periods. This is a pre-infarction condition of coronary artery disease with a high likelihood of developing acute myocardial infarction within a few days to weeks when unintervened. It is important for the emergency physicians, to recognize the typical ECG findings of Wellens' Syndrome, because these characteristic ECG findings are considered as a marker for critical LAD occlusions. In this report, we present a patient with high-risk NSTEMI Killip 1 (Wallen's type B), Hypertension stage II, and HHD.

Case illustration: A 64-year-old male presented to emergency department with chief complaint of crushing substernal chest pain since 1 hour ago; the pain was strong and irradiated to upper chest. The pain located in substernal chest, shoulder and neck; described like heart burn and diaphoresis. 3 days earlier the patient also admitted to having similar complaints. His past medical history revealed a diagnosis of uncontrolled hypertension without any medical treatment. he was a smoker. Patient blood pressure was 179/100 mmHg, heart rate was 83 bpm. The laboratory results showed CKMB elevated, leukocytosis, and elevated ureum creatinin. The ECG showed a deep T inverted at V2-V3 and T Bifasik at V4, I, AvL. Patient was administrated clopidogrel 4 x 75mg, aspillet 4 x 80mg, ISDN 1 x 5mg SL, Arixtra 1,5mg SC, and two times captopril 25mg SL. Then the patient transferred to ICU and planning for elective PCI. Coronary angiography showed proximal-mid LAD stenosis 75-85%.

Conclusions:
Electrocardiographic changes of type-2 Wellens syndrome are highly specific for the diagnosis of critical stenosis of the proximal left anterior descending artery (LAD), with a high risk for the development of anterior MI. Patients with clinical manifestations and tests suggestive of the syndrome should be promptly referred for angiographic study for diagnostic confirmation and intervention.

KEYWORD: *NSTEMI, Wallen's Syndrome, Coronary Artery Diseases.*

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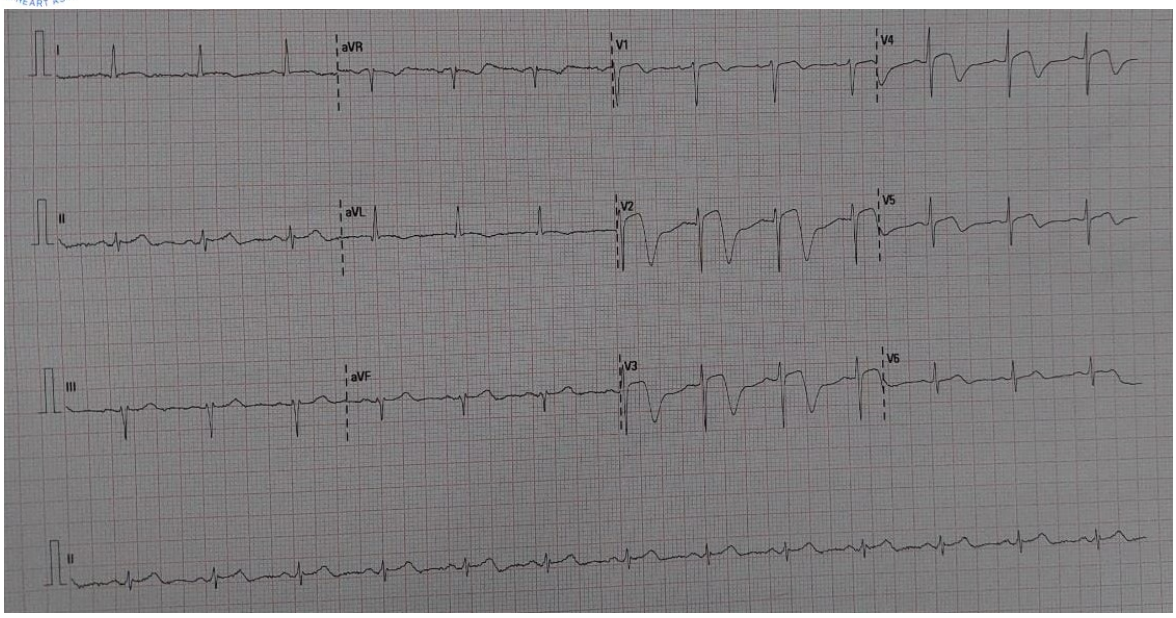

Figure 1 ECG at presentation showed a deep T inverted at V2-V3 and T Bifasik at V4, I, AvL

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A Rare Case Presentation
WPW Syndrome and Preexcitation-Induced Cardiomyopathy: Is It a Causal or Casual Relationship?

M. R. Felani[1] ,D. A. Hanafy[2] ,D. Y. Hermanto[2] ,S. B. Raharjo[2] ,Y. Yuniadi[2]
[1]Resident of Cardiology and Vascular Department, National Cardiovascular Center Harapan Kita (NCCHK), Jakarta, Indonesia
[2]Staff of Arrhythmia Division, Cardiology and Vascular Department, National Cardiovascular Center Harapan Kita (NCCHK), Jakarta, Indonesia

Background: Patients with Wolff-Parkinson-White (WPW) syndrome, presence of an accessory pathway (AP), results in palpitation symptom most commonly due to Atrioventricular Reentrant Tachycardia (AVRT). The mechanism for the development of left ventricular dysfunction in patients with pre-excitation syndrome has not yet been fully elucidated, and its prevalency in Indonesia is not well known. The eccentric ventricular activation via AP, may arise in right-sided AP, could result in an asynchronous spread of ventricular depolarization, then leads to LV dyssynchrony and worsening LV dysfunction, defines as Preexcitation-Induced Cardiomyopathy (PIC). To review the magnitude of impact of Right AP of Pre-excitation Syndrome resulting in PIC and to discuss the result of successful Right AP ablation for LV systolic function improvement of our patient in NCCHK.
Case illustration: A 12-year-old female patient with WPW Syndrome and congestive heart failure due to Non-Ischaemic Cardiomyopathy with 18% of LV ejection fraction (EF). During the radiofrequency ablation (RFA), the AP was found in the right anterior location, in accordance with the 12-lead ECG analysis. The RFA for AP procedure was done successfully. Six month after successful AP ablation, her LVEF was increased significantly to 33%.

Conclusions:
The cardiomyopathy in this patient was presumably due to LV dyssynchrony from marked ventricular preexcitation. The right anterior AP of the patient had successfully ablated, and no more complaints of palpitations were recognized by the patient. Long-term follow-up, especially on clinical and echocardiographic results, still needs to be done. By far, her LVEF's improvement has been excellent without remaining symptoms.

KEYWORD: *WPW Syndrome, Cardiomyopathy, LV Dyssynchrony.*

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Anterior Extensive STEMI with Irregular Wide Complex Tachycardia: Is It Atrial Fibrillation or Ventricular Tachycardia?

M. R. Felani[1], Hendrianus[1] ,E. A. Pradipta[1] ,D. Y. Hermanto[2] ,D. Zamroni[3]
[1]Resident of Cardiology and Vascular Department, National Cardiovascular Center Harapan Kita (NCCHK), Jakarta, Indonesia
[2]Staff of Arrhythmia Division, Cardiology and Vascular Department, National Cardiovascular Center Harapan Kita (NCCHK), Jakarta, Indonesia
[3]Staff of Cardiovascular Intensive and Acute Care Division, Cardiology and Vascular Department, National Cardiovascular Center Harapan Kita (NCCHK), Jakarta, Indonesia

Background: The differential diagnosis of wide complex tachycardia includes ventricular tachycardia (VT), supraventricular tachycardia (SVT) with aberrant His-Purkinje conduction, and SVT with ventricular activation using an accessory pathway. With its irregularly irregular rhythm, ECG is often suggested as an atrial tachyarrhythmia (fibrillation or flutter), with aberrant His-Purkinje conduction resulting in wide QRS complexes. Slow incessant VT with an irregular rhythm, especially if haemodynamically tolerated, can be mistaken for atrial tachyarrhythmia.

Case illustration: A 72-year-old man with a past medical history of coronary artery disease presented to the emergency department with acute chest pain 3 hours before admission. He was haemodynamically stable and looked mildly distressed. The electrocardiogram (ECG) showed an irregularly irregular wide complex tachycardia at a rate of 150 beats/min with right bundle branch block (RBBB)-like morphology. A transthoracic echocardiogram showed a severely reduced left ventricular function. A diagnosis of atrial fibrillation (AFib) with a rapid ventricular response was made. Intravenous digoxin was administered, however, his heart rate remained uncontrolled at between 110 and 180 beats/min. A closer inspection of the ECG monitor showed different QRS morphology captured during tachycardia. A repeat ECG was obtained and showed captured beats indicative of ventricular tachycardia. Therapy with amiodarone was commenced, and the rhythm was converted to sinus rhythm with RBBB and anterior extensive ST-segment elevation. After terminating the tachycardia, his chest pain was resolved. He was treated as acute coronary syndrome and sent to the catheterization laboratory.

Conclusions:
The patient's initial presenting ECG was misdiagnosed as Rapid AFib because of the irregularly irregular rhythm. In VT, the rhythm is usually regular or fairly regular, but beat-to-beat variations can occur. Irregular VT and AFib with aberrancy may produce similar ECG. However, if the QRS morphology changes during tachycardia (compared to the prior ECG) or if there are captured beats observed, then VT is likely. The mechanism of this VT is postulated to be either due to abnormal automaticity or electrical re-entry resulted from myocardial ischemia.

KEYWORD: *Ventricular Tachycardia, Atrial Fibrillation, Acute Coronary Syndrome.*

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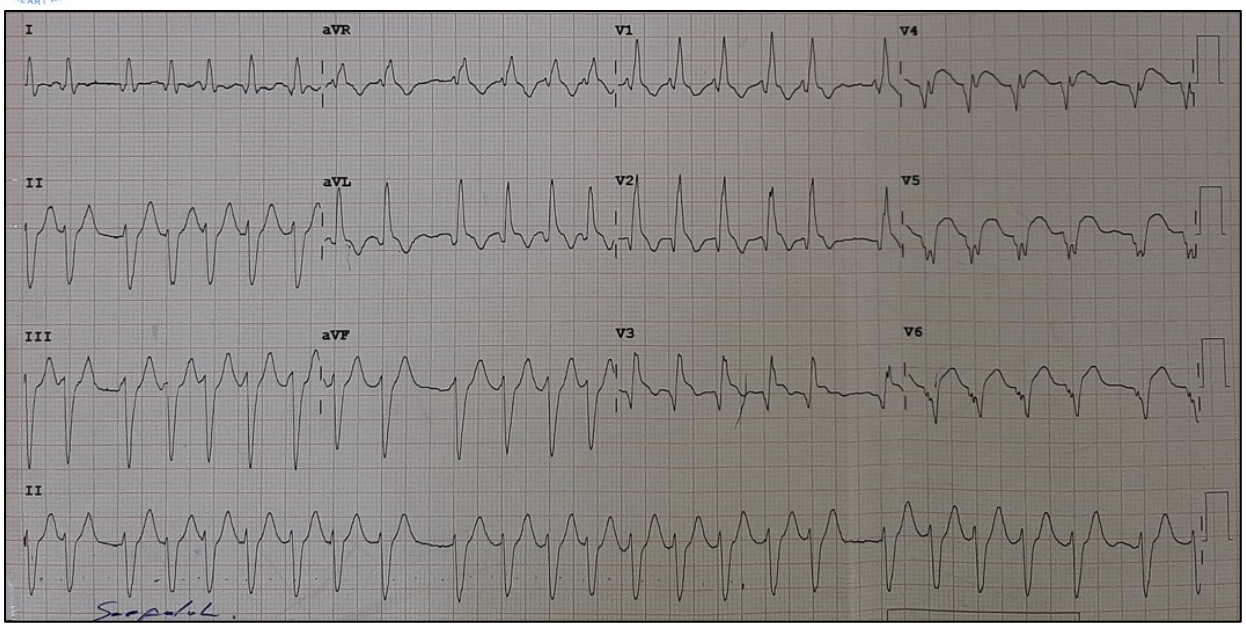

Figure 1 ECG taken on presentation showing the patient's irregularly irregular broad complextachycardia with RBBB pattern, mimicking Rapid AFib with aberrant conduction

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Contemporary Approach in Brady-Tachy Arrhythmia Related Myocarditis

N. L. Adani[1] ,F. Hidayati[1] ,L. Krisdinarti[1] ,E. Maharani[1]
[1] Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, Gadjah Mada University, Yogyakarta, Indonesia

Background: A wide spectrum of arrhythmias has described in patients with myocarditis, including sinus node and atrioventricular node dysfunction (SND-AVND), ventricular arrhythmia (VA) and others. Various arrhythmias occur at once in myocarditis patient are rarely found.
Case illustration: A 43-year-old man referred to emergency department with 6 days history of lightheadedness and palpitation. Electrocardiograms revealed complete heart block, alternating high grade atrioventrivular block and pulsed monomorphic ventricular tachycardia (VT). HsTroponinT value was 160ng/L (normal range <14ng/L) as the angiogram showed normal coronary arteries with right coronary artery fistule. Hence, patient implanted by temporary pacemaker. In the intensive ward, there were recurrent episodes of pulsed VT and intermitten accelerated junctional rhythm. Echocardiogram showed global hypokinesia with left ventricle ejection fraction 43%. Cardiac magnetic resonance revealed chronic myocarditis features as no signs of edema, and subepicardial fibrosis at basal mid septal, basal anterior and basal inferoseptal (11%) on late gadolinium enhancement which are consistent with atrioventricular node area. A dual chamber pacemaker with rate responsive was implanted subsequently, after considering various arrhythmias involving SND-AVND which remaining at 14 days after onset, and limited hospital resources to provide high power device. Two weeks after discharge, patient readmitted to hospital due to experiencing pulsed VT that has similar morphology as previous VT. After optimizing antiarrhythmic drugs (AADs), no VA was seen. When promptly managed, acute-phase arrhythmias tend to be self-limiting, european guidelines suggest to consider AADs such as amiodarone and/or beta-blockers during the acute phase.

Conclusions:
Various arrythmias related myocarditis that occurs at a time are rarely reported. Guidelines suggest the management based on clinical manifestation and phase of myocarditis. Treatment of these arrythmias related myocarditis alongside limited device resource are particularly challenging.

KEYWORD: *Ventricular arrhythmia, myocarditis, arrhythmia related myocarditis.*

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Total Atrioventricular Block (TAVB) Accompanied by Dyspepsia Syndrome in Adult with Type 2 Diabetes Mellitus, is it Asymptomatic or Symptomatic?

N. Sukmadi[1] ,S. Inayasari[1] ,M.R.A. Shiddieqy[1] ,L. Pribadi[2] ,M.G. Suwandi[2]
[1]*Internship Doctor, RS Pusat Angkatan Udara dr. S. Hardjolukito, Yogyakarta, Indonesia*
[2]*Cardiologist, RS Pusat Angkatan Udara dr. S. Hardjolukito, Yogyakarta, Indonesia*

Background: Asymptomatic TAVB in adults is extremely rare, and its precise epidemiology is unknown. Patients with TAVB have potential for wide range of symptoms such as lightheadedness, dizziness, syncope, or angina; but not dyspepsia, and some of which are life threathening (ventricular arrhytmia, and sudden death). In this report, we present a TAVB patient with dyspepsia syndrome in the emergency room (ER).
Case illustration: A 46 years old man came to our ER with nausea and 3 times vomiting since 1 day before admitted to the ER. Patient usually have this symptoms throughout the year, and this time is the worst. The patient's past history was remarkable for uncontrolled T2DM, and he denied any lightheadedness, syncope, dizziness, and chest pain before. He actively playing football amateurly before. On physical examination, his blood pressure was 114/70, with heart rate of 40 bpm, with no significant orthostatic changes. The cardiac examination is normal with epigastric tenderness in accordance with the chief complain. 12 lead ecg was conducted and revealed a TAVB with escape junctional rhythm, and right bundle brach block (RBBB) with RAD. Remarkable laboratory testing shows high random blood glucose (398 mg/dL), with renal insufficiency (ureum 51 mg/dL, and creatinine 1.95 mg/dL). Later patient was reffered to another hospital for further interventions.

Conclusions:
Dyspepsia syndome is the most common thing ER chief complaint. ER doctor should always checked the vital signs, and patients with bradycardia should always conduct a 12 lead ECG. Patients with bradyarritmia can be asymptomatic, and the presence of dyspepsia in bradyarritmia with T2DM is not well known. Further study should be conducted whether dyspepsia is one of the sign patients with T2DM of low cardiac output in TAVB.

KEYWORD: *Total atrioventricular block, asymptomatic, dyspepsia, T2DM*.

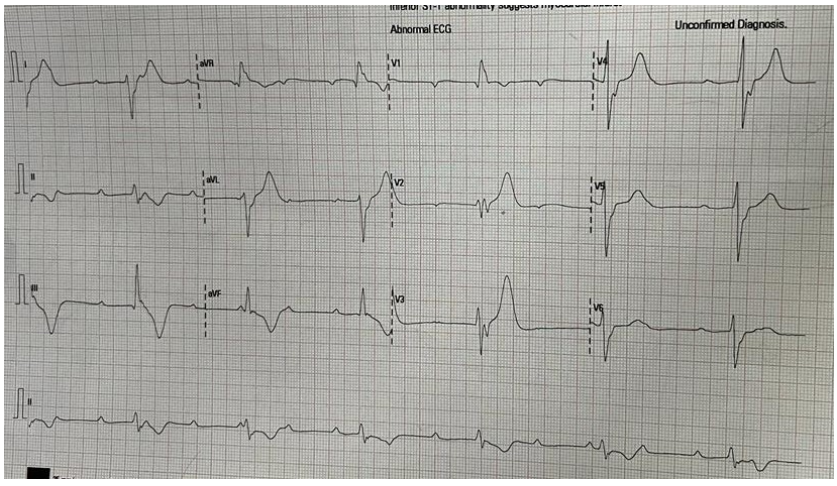

Figure 1. ECG showed a TAVB with Junctional Escape Rhythm and RBBB

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Idiopathic posterior fascicle left ventricular tachycardia: case report

P. Gandi[1] ,B. B. Dharmadjati[1] ,R. Julario[1] ,M. R. Amadis[1] ,T. R. Mahmudah[1]

[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Airlangga – Dr. Soetomo General Academic Hospital, Surabaya, Indonesia

Background: Idiopathic Left Ventricular Tachycardia (ILVT) is one of the idiopathic Ventricular Tachycardia (VT) manifestations, with right bundle branch block and left axis deviation electrocardiogram morphology. The mechanism of ILVT is thought to be due to the localized closed reentry in the posterior fascicle.

Case illustration: We present the case of a 25-year-old man who was admitted to the emergency department with complaints of palpitations while running. The ECG showed a feature suggestive of ILVT. Vagal manoeuvres, adenosine and intravenous metoprolol, were ineffective in terminating the arrhythmia. Conversion of heart rhythm to sinus was achieved after intravenous administration of verapamil. Electrophysiological studies with entrainment demonstrated a macro-reentry mechanism in the posterior fasciculus when a multipolar ablation catheter was placed at the apex of the LV septum, with the orthodromic appearance of Purkinje potential P1 and retrograde diastolic potential P2. Radiofrequency ablation was targeted at the position where PP1 appeared earliest. In addition, there was also a 52 ms Atrial-His bundle (AH) jump, which indicated the presence of Dual AV Node Physiology. Ablation was performed on the apical third of the septum, shifting toward the basal site until successful ablation was achieved to avoid accidental injury to the left bundle branch or His bundles. Successful ablation was indicated by the inability to induce VT.

Conclusions:

The first-line treatment for ILVT is the radiofrequency ablation that targets the P1 or early P2 potential to eliminate this type of VT. Understanding the anatomy of the Purkinje system and the mechanism of ILVT helps cardiologists devise a comprehensive approach to successful ILVT radiofrequency ablation.

KEYWORD: *Ventricular tachycardia, left posterior fascicle, ablation, verapamil.*

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Lesson Learn from Sudden Cardiac Death (SCD) Victim with Masked and Neglected Risk Factor: A Case Report and Literature Review

P. A. Arimukti [1]
[1]Company Doctor PT Medika Plaza

Background: SCD in general population are most caused by coronary artery disease (CAD), arrhytmogenic cardiomyopathy and chanelopathies. Some of the cause are hereditary disease. Lack of investigations on SCD case leave a lot of etiologically unexplained. ECG can be a modality to screen the risk of SCD, although some of benign arrhythmia can be masked the others malignant.
Case illustration: A 36 years old man was known had CRBBB (complete right bundle branch block) since 2018 from ECG result. Based on his last ECG result in 2020 the rhythm was sinus, heart rate 71 bpm, PR interval 120 ms, QRS duration 140 ms with CRBB pattern, and Bazzet QTc 392 ms. His father dead at 45 years old due to unknown cause. His last medical checkup in 2020 showed asymptomatic, normal physical examination, normal chest x-ray, normal laboratory result and no ischemic response on treadmill test. Echocardiogram result in 2019 was within normal limit. Based on our clinic medical record there was no health complain within last 3 months. On early 2021 he had sudden cardiac death at his night shift work. In this case, several facts were found that increase suspicion of SCD caused by Brugada Syndrome (Brs), including family history, SCD at night, age, gender and low risk of CAD. Even though the prior ECG did not found BrS pattern, CRBBB morphology was reported can completely mask the typical ECG in BrS, it probably causes the BrS undiagnosed and unmanaged. The last guideline state SCD victim with family history of SCD are recommended for autopsy and toxicology test, whereas postmortem examination and autopsy are uncommon in Indonesia. Beside that SCD victim's first degree relative are recommended for workup study include medical history, physical examination, standard and high precordial ECG, echocardiography and exercise testing. Further pharmacological testing with sodium channel blocker for victim relative should be considered when BrS are suspicious cause.

Conclusions:
SCD case is not the end of medical service can be provide by doctor. Postmortem exam and first degree relative screening should be offer to SDC victim's family. The result could be beneficial as base on treatment choice for preventable SCD.

KEYWORD: *Sudden Cardiac Death, Brugada Syndrome, Right Bundle Branch Block.*

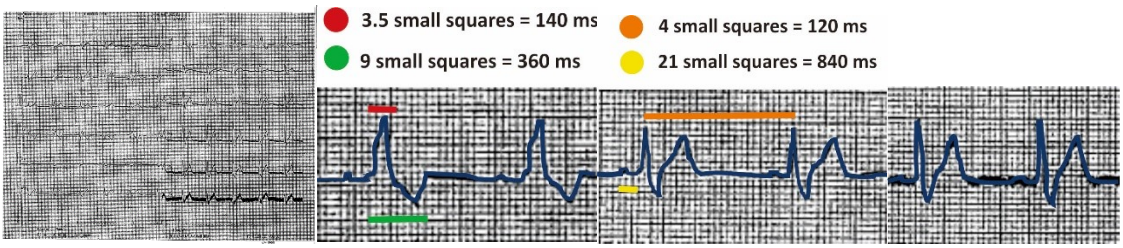

Figure 1. Complete ECG, Magnified Lead II, V1, V5 ECG Result

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Dual Reversal of Tachycardia-induced Cardiomyopathy and Sinus Node Dysfunction by Rhythm Control Approach

R. A. Anzali[1] ,S. B. Raharjo[2] ,D. Y. Hermanto[2] ,D. A. Hanafy[2] ,Y. Yuniadi[2]

[1]Department of Cardiology and Vascular Medicine , Faculty of Medicine, Universitas Indonesia, Jakarta

[2]National Cardiovascular Center Harapan Kita, Jakarta

Background: Tachycardia-induced cardiomyopathy (TICM) is a subform of reversible dilated cardiomyopathy. Early recognition and prompt treatment of the arrhythmia results in symptom resolution and recovery of ventricular function.

Case illustration: A 13-year-old girl was referred to NCC Harapan Kita with complained of shortness of breath during activity, palpitations, and a history of fainting. On admission, BP 95/59 mmHg, HR 170 bpm with NYHA III functional class. ECG showed atrial tachycardia. Echocardiography showed dilated all chambers with LVEF of 32%, and moderate functional mitral regurgitation. The patient was initially diagnosed as dilated cardiomyopathy due to a myocarditis. Further investigation revealed that the cardiac MRI showed no evidence of oedema and fibrosis in the myocardium. In addition to the presence of AT, the holter monitoring revealed 590 episodes of sinus arrest with the longest duration of >5.6 seconds. A sinus node dysfunction (SND) was diagnosed treated with AAI permanent pacemaker (PPM) implantation. After PPM, the patient still had complaints of palpitation and readmission 4 times in a year despite optimal antiarrhythmic and heart failure medication. Therefore, AT ablation was performed as the definitive therapy. After ablation, the LV ejection fraction improved to 63% soon after ablation, then increased to 75% at the last follow-up. The sinus node function was also improved as indicated by the decreased in pacing percentage from 50% to 15%. Medical therapy was rationalized after obtaining good reverse remodelling of the ejection fraction and sinus node function after ablation.

Conclusions:

We describe a case of a girl with TICM who was initially diagnosed as DCM. The definite diagnosis was AT-induced cardiomyopathy and SND. The patient was treated with AAI PPM and AT ablation. After ablation, there was good reverse remodelling of the ejection fraction and sinus node function.

KEYWORD: *Dilated cardiomyopathy, Tachycardia-induced cardiomyopathy in children, Atrial Tachycardia, Reverse remodelling.*

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WOLF-PARKINSON WHITE (WPW) SYNDROME WITH MULTIPLE AND LONG ACCESSORY PATHWAYS: LEFT SEPTAL TO LEFT LATERAL ACCESSORY PATHWAY

R. N. Rosyadi[1] ,G. Lilihata[1] ,D. A. Hanafy[2]

[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

[2]National Cardiovascular Center Harapan Kita, Jakarta, Indonesia

Background: Wolf-Parkinson White (WPW) syndrome is a cardiac conduction disorder that may present with palpitation and lead to sudden cardiac death. WPW syndrome's accessory pathway (AP) could be detected and predicted by the electrocardiogram. The incidence of multiple APs was rare but may be associated with higher risk of atrial fibrillation (AF) and ventricular fibrillation (VF).

Case illustration: We present the case of a 52-year-old male patient, who came to arrhythmia outpatient clinic for recurrent palpitation after ablation of accessory pathway two years before. Basic ECG showed sinus rhythm with negative delta wave at V1, II, III, aVF suggesting right posteroseptal AP. Electrophysiology study showed most atrio-ventricular (AV) fusion at left posterolateral area (CS 3,4). Ablation of left posterolateral AP was performed and AV fusion was separated. Delta wave reappeared and showed most AV fusion at distal CS. Ablation of left lateral AP was performed and succeed. Ventricular pacing showed eccentric atrial activation with most ventriculo-atrial (VA) fusion at left posterior area (CS 7,8). Ablation at left posterior area was performed and ventricular pacing still showed eccentric atrial activation with VA fusion at proximal CS. Ablation at posteroseptal area was performed and ventricular pacing showed retrograde block.

Conclusions:
Previous studies support the view that patients with multiple APs are more likely to present with both AF and VF. The presence of multiple APs provide the potential for complex re-entrant circuits, which may result in multiple atrial wavefronts during reciprocating tachycardia and thus a greater potential for atrial fibrillation. Patients with multiple APs can be treated by radiofrequency ablation in only one session with a high success rate. Precise electrophysiological study and careful ablation are needed to avoid recurrent procedure.

KEYWORD: *Wolf-Parkinson White (WPW) Syndrome, Multiple Accessory Pathway, Radiofrequency Ablation*.

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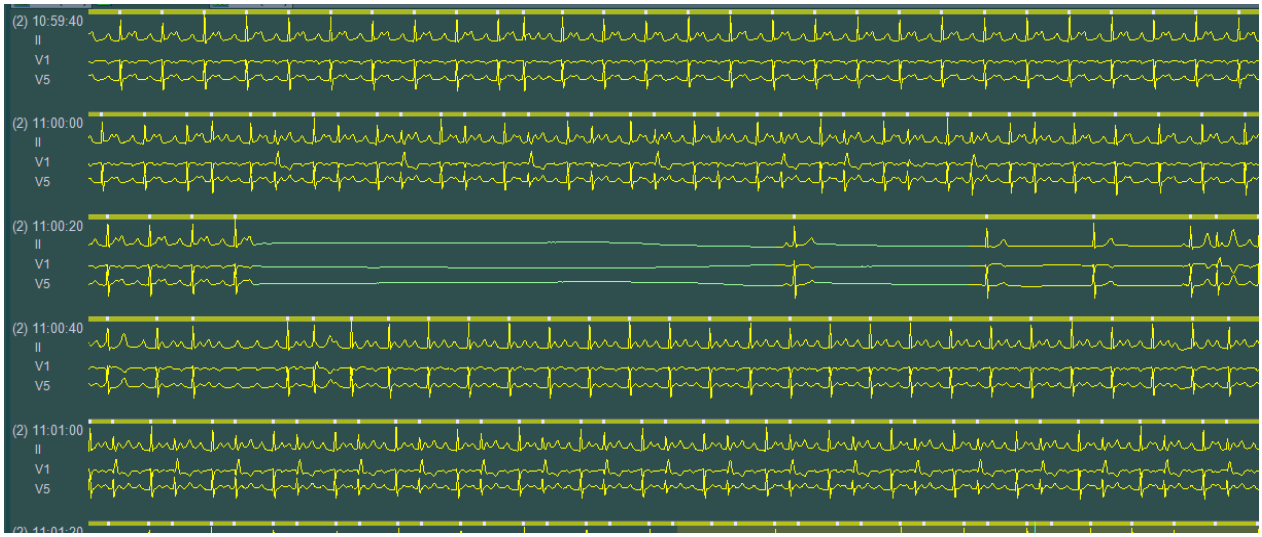
To Pace or Ablate: Clinical Conundrum for Tachycardia-Bradycardia Syndrome

R. Muliawan[1] ,F. Hidayati[2] ,E. Maharani[2]
[1]Resident of Cardiology and Vascular Medicine, Sardjito General Hospital, Yogyakarta
[2]Staff of Department of Cardiology and Vascular Medicine, Sardjito General Hospital Yogyakarta

Background: In patients with tachycardia-bradycardia syndrome, incessant atrial tachycardia (AT) often results in sinus pause after the termination of tachycardia. Tachycardia-bradycardia syndrome is difficult to treat with antiarrhythmic agents alone because controlling tachycardia may lead to aggravation of bradycardia. Implantation of a permanent pacemaker with antiarrhythmic agents has been the traditional treatment for tachycardia-bradycardia syndrome. On the other hand, radiofrequency catheter ablation has emerged as an alternative treatment strategy for this case.
Case illustration: A 47-year-old woman came to the emergency room with an episode of palpitation, weakness, and dizziness. History of pre-syncope and palpitations for two years with anti-arrhythmic drugs (beta-blocker or calcium channel blocker). From 48 hours holter monitoring results obtained atrial tachycardia with frequent episodes of sinus pause, with the longest being 12.912 milliseconds(ms). Since AT was incessant with palpitation as predominant complain electrophysiology study was procceded it yield the tachyarrhythmia as focal atrial tachycardia with high crista terminalis origin. The AT was terminated during conventional radiofrequency ablations (RFA) and remained an inducible with sinus node recovery time (SNRT) post-RFA showed within the normal limit (1250ms). After the ablation, the AT is still recurrent with episode frequent sinus pauses. Due to our limited resources, we decided to make the contemporary approach with AAIR permanent pacemaker implantation. Gave optimization of anti-arrhythmic drugs, and the patient was planned for three-dimension ablation for the AT.

Conclusions:
In limited resources hospitals, implantation of a permanent pacemaker in tachycardia-bradycardia syndrome patients with AT and sinus pause is more necessary. It takes precedence over conventional ablation because of the possibility of other sources of AT that have not been removed in the patient.

KEYWORD: *Tachycardia-bradycardia syndrome, atrial tachycardia, sinus pause.*



Picture 1. Atrial tachycardia with very long sinus pause.

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Lidocaine Challenge in Wide-Complex Tachycardia of Uncertain Origin in Primary Health Care in Rural Area

R. Ramadhani[1] ,F. Rusnanta[1] ,S. P. Noviantoro[1]
[1]Cardiology Department, General Hospital Kanjuruhan, Kepanjen, Malang

Background: Wide-complex tachycardia (WCT) refers to rhythm with rate > 100 beats/minute and QRS complex duration >120 milliseconds. In several circumstances, this rhythm may cause hemodynamic instability which prompts adequate treatment to improve patient outcomes. However, in primary health care with minimal resources, it may be hard to provide sufficient therapy for this kind of case. The use of lidocaine challenge in unstable hemodynamic wide complex tachycardia of uncertain origin along with adequate monitoring may have beneficial effect on the patient.
Case illustration: 67 year-old-man came to emergency room of primary health care with chief complaint shortness of breath with palpitation and history of syncope. Physical examination revealed low blood pressure with regular rapid heart rate and poor peripheral perfusion. Electrocardiogram (ECG) showed a wide-complex tachycardia with rate 214 beats/minute. Stabilization of airway and breathing also fluid management was done then patient was planned to get referred to secondary hospital. Due to uncertainty of origin of wide-complex tachycardia, intravenous lidocaine was administered before patient being referred. In secondary hospital, re-evaluation of the patient revealed improvement in blood pressure and peripheral perfusion. ECG showed sinus tachycardia rate 115 bpm with right bundle branch block (RBBB) morphology. We did adequate hemodynamic monitoring and continued arrhythmia management by using non-dihydropyridine calcium channel blocker. Patient was fully stabilized on 2nd day and got discharged after 6 days of hospitalization. Several small studies in the past showed that lidocaine may be used to treat wide-complex tachycardia of uncertain origin and convert them into sinus rhythm. Beneficial effect was said to be more effectively achieved in the ventricular origin wide-complex tachycardia. Further studies may be needed to explain lidocaine's effect on wide-complex tachycardia.

Conclusions:
Wide-complex tachycardia may cause hemodynamic instability which prompts sufficient treatment. Lidocaine administration may cause beneficial effect in wide complex tachycardias, especially in those of ventricular origin. Further studies are still needed to explain the use of lidocaine on wide-complex tachycardia.

KEYWORD: *Wide complex tachycardia*, *Lidocaine, Origin*.

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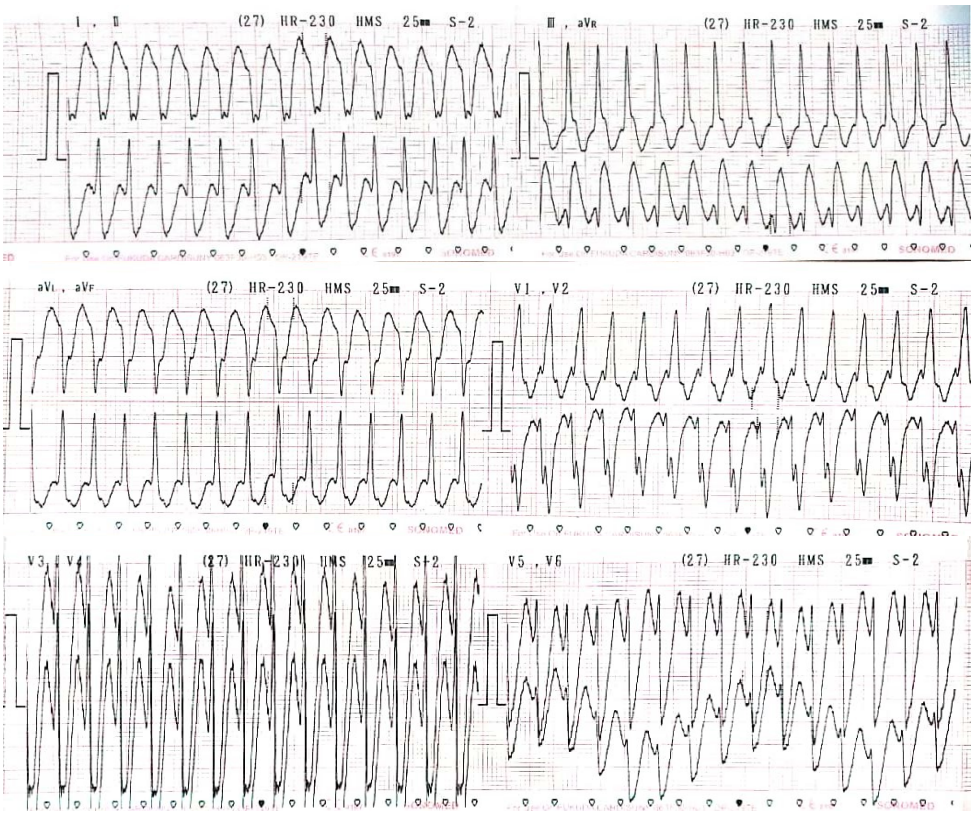

Figure 1. ECG in Primary Health Care showed Wide Complex Tachycardia 214 bpm

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Sinus node dysfunction complicating a resuscitated patient with terminal condition: A case report

R. Ayuningtyas[1] ,R. I. Rismawanti[1] ,A. Novitasari[1] ,A. Ciptasari[1] ,M. G. Suwandi[2] ,L. Pribadi[2]
[1]General Practitioner, Rumah Sakit Pusat Angkatan Udara Suhardi Hardjolukito, Bantul, Indonesia
[2]Cardiologist, Rumah Sakit Pusat Angkatan Udara Suhardi Hardjolukito, Bantul, Indonesia

Background: Sinus arrest refers to one of Sinus Node Dysfunction (SND) subtype in which there's prolonged asystole >3 seconds with/without escape rhythm. SND can be due to intrinsic or extrinsic cause (neurocardiogenic reflexes, enhanced vagal tone, hypoxia and hypercapnia, increased intracranial pressure, hypothyroidism, hyperkalemia or drugs). Early recognition of such arrhythmia and elimination of any reversible causes may reduce morbidity and improve survival.
Case illustration: A 79-year-old woman presented to emergency department with high fever and difficulty in breathing since a day before. She was somnolent (E3V2M5), 40.3℃ in temperature, and tachypnea with oxygen saturation 98% using NRM 10 lpm. Her blood pressure and heart rate were normal. Crackles were evident at both of her lungs, and her xray depicted bilateral pneumonia with left calcification. Her laboratory findings showed increased blood glucose and kidney function markers; and a decrease in blood gas' P/F ratio. The initial electrocardiogram (ECG) demonstrated normal sinus rhythm. The patient was assessed with moderate respiratory failure, sepsis, bilateral pneumonia, diabetes mellitus type II, acute kidney injury. She was admitted to intensive care, but after 4 hours monitoring, the patient had cardiac arrest and was resuscitated and intubated. Post intubation, her ECG occasionally demonstrated sinus arrests with varied duration, each of which always went back to sinus tachycardia of 130-140 bpm. She had no history of medications that could trigger sinus arrest. The patient requires pacemaker placement but her condition is not stable enough to be referred to get further management.

Conclusions:
Sinus arrest is diagnosed by symptoms and ECG findings. In this case, neurocardiogenic reflexes or enhanced vagal tone such as getting intubated seem to be the more plausible causes for the occurence of sinus arrest. The points of SND treatment are to eliminate reversible causes and to consider pacemaker in symptomatic and haemodynamically-unstable patients.

KEYWORD: *Sinus arrest, Sinus node dysfunction, Diagnosis, Electrocardiogram.*

Figure 1. The ECG at ICU with occurence of sinus arrest after the patient was being intubated

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Transesophageal echocardiography assisted ablation of papillary muscle origin premature ventricular contraction: A challenging structure in perpetual motion

R. Wijaya[1] ,S. B. Raharjo[1] ,A. S. Kuncoro[1]
[1]Heartology Cardiovascular Centre, Jakarta, Indonesia

Background: Catheter ablation is the most recommended interventional therapy for premature ventricular contraction. Interestingly, papillary muscle origin premature ventricular contractions are especially difficult to treat due to its perpetual motion during heart contractions, resulting in higher recurrence rate after ablation procedure. Hence, many recommendations encourage using echocardiography to better assist the catheter trajectory. In this study, we demonstrate that transesophageal echocardiography was comparable to intracardiac echocardiography in assisting 3D-ablation of papillary muscle origin premature ventricular contraction with desirable outcome.

Case illustration: A 41-years old female with worsening lethargy and recurring left chest discomfort. 12-lead electrocardiogram showed infrequent premature ventricular contraction of right bundle branch block morphology with 24 hours Holter showing frequent multifocal premature ventricular contraction complex (20.85%), right bundle branch block morphology, superior axis, RS-transition at precordial leads V3-V4, QRS duration 111-122 msec, predominantly posterior papillary muscle origin and infrequent premature atrial contraction (0.21%). Activation and pace mapping of the 2 dominant premature ventricular contractions were obtained assisted by transesophageal echocardiography, locating the premature ventricular contractions origin at the left ventricular posterior papillary muscle. Papillary muscle potentials were identified as a pair of small electrical signals captured prior and in close proximity to the premature ventricular contraction potential. As widely known, premature ventricular contraction recurrence is highly dependent on the accuracy of catheter tip in ablating the premature ventricular contraction origin. Hence, additional visualization modality using transesophageal echocardiography was employed to ensure good catheter contact.

Conclusion: Transesophageal echocardiography poses as a promising modality in ensuring good contact of catheter tip with challenging heart structures that are in perpetual motion. Based on our experience, a 3D-electroanatomic mapping combined with real-time transesophageal echocardiography is able to clearly navigate catheter position relative to the papillary muscle which is crucial to suppress likelihood of recurrence, but with lesser cost and wider availability compared to intracardiac echocardiography.

KEYWORD: *Papillary muscle; premature ventricular contraction; ablation; transesophageal echocardiography.*

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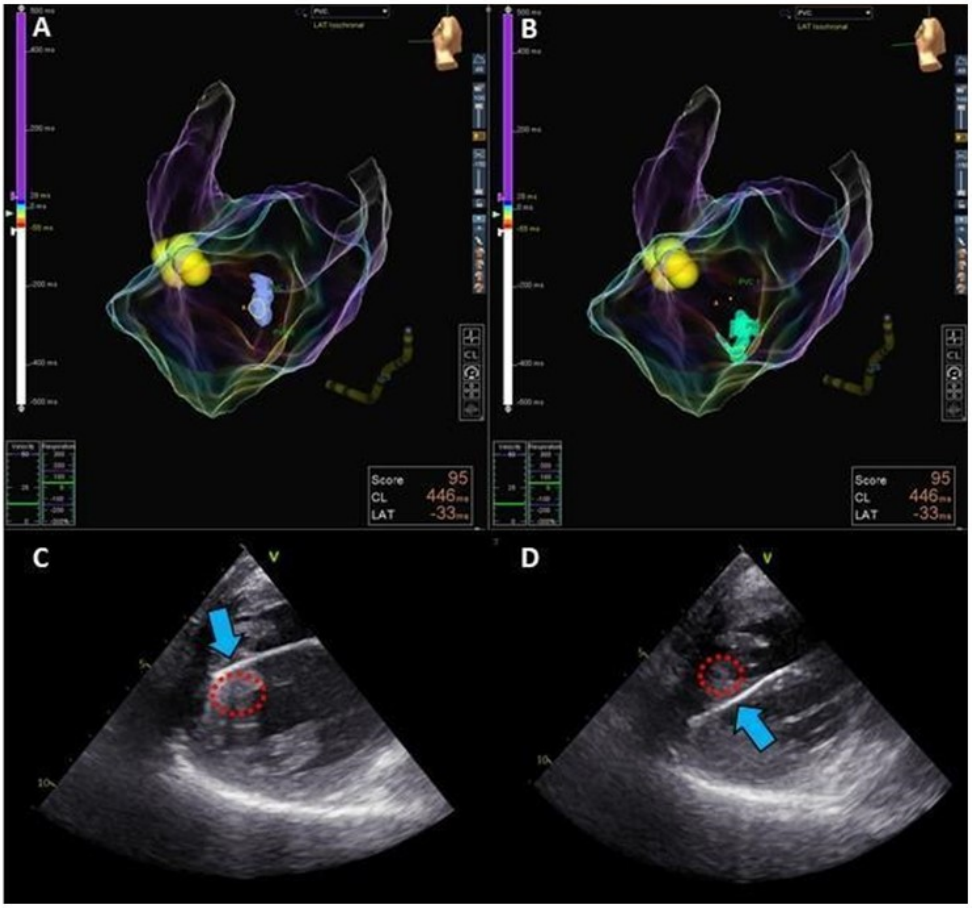

Figure 1 3D-ablation procedure visualized by transesophageal echocardiography.
Top panels showing the ablation target site for PVC-1 (blue circles) and PVC-6 (green circles). Real-time transesophageal echocardiography showed ablation catheter (blue arrow) in good contact positioned superior (C) and inferior (D) to the posterior papillary muscle (red-dotted circle).

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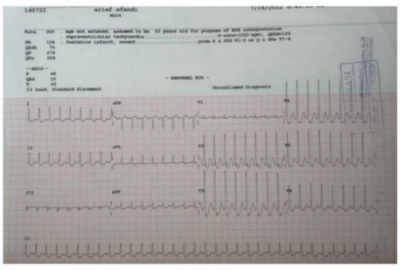
A Rare Case : Supraventricular Tachycardia in WPW (Wolff-Parkinson-White) Syndrome in Young Adult Male

Restu[1] ,H. Zaputra[2]
[1]General Practitioner in Prof.DR.M.A Hanafiah SM General Hospital Batusangkar
[2]Cardiologist in Prof.DR.M.A Hanafiah SM General Hospital Batusangkar

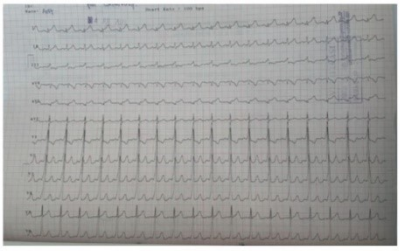
Background: SVT is defined as tachycardia (atrial rate >100 bpm at rest), the mechanism of which involves tissue from His bundle or above.
Patients with Wolff-Parkinson-White syndrome, atrial impulse can pass in an anterograde direction to the ventricles through both the AV node and the accessory pathway.
Case Illustration: Mr. AE, 36-year-old came to the Emergency room with chief complaint of palpitations since 4 hours ago. Alterred mental status (-), dyspneu (-), chest pain (-), dizziness (-). History of the same complaint 4 years ago. From vital sign, patient CM, BP 100/70 mmHg, HR 200 bpm, RR was 20, SpO2 99%. Physical examination, heart sound I-II regular and fast. Electrocardiography showed a narrow-QRS tachycardia or SVT (rate 200 bpm). Laboratory showed, routine blood and cardiac enzyme were within normal limits, electrolyte with hypokalemia (3,3 mmol/l). Patient was in stable condition. Then vagal maneuver was perfomed, there was no response. After 10 minutes, patient complained of dizziness, diaphoresis. BP 70/50 mmHg, HR 190-200 bpm (weak), cold extremity. Patient was in an unstable condition. Performed syncronized cardioversion (50 joule) with sedation. After cardioversion, heart rhytm converted to sinus (rate 100 bpm). Patients condition was stable, BP 100/70 mmHg, HR 100 bpm (strong), warm extremity. Electrocardiography showed characteristics of WPW syndrome (shortened PR interval, delta waves, and wide QRS complex).

Conclusions:
Patients with WPW syndrome, atrial impulse can pass in an anterograde direction to the ventricles through both the AV node and the accessory pathway. During sinus rhytm, activation of the ventricle from the accessory pathway causes a characteristic ECG appearance:
(1) the PR interval is short
(2) delta wave
(3) the QRS complex is widened
Patient with WPW syndrome are predisposed to PSVTs because the accessory pathway provide a potential limb of a reentrant loop.
However, approximately 1 in 1500 people has the WPW syndrome and is born with an additional connection between an atrium and ventricle.

KEYWORD: *SVT, WPW syndrome.*



Electrocardiography before cardioversion (SVT)



Electrocardiography post cardioversion

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SUPRAVENTRICULAR TACHYCARDIA AND PREGNANCY: A DILEMMATIC ANTIARHYTHMIA DRUG OF CHOICE IN RURAL AREA

R. S. Yusuf[1] ,Y. Pintaningrum[2,3] ,M. R. Jumsa[4,5]

[1]Medical Professional Student, Faculty of Medicine, Mataram University, Mataram, Indonesia
[2]Cardiovascular Department, Faculty of Medicine, Mataram University, Mataram, Indonesia

[3]Cardiovascular Department, West Nusa Tenggara General Provincial Hospital, Mataram, Indonesia
[4]Obstetrics and Gynecology Department, Faculty of Medicine, Mataram University, Mataram, Indonesia
[5]Obstetrics and Gynecology Department, West Nusa Tenggara General Provincial Hospital, Mataram, Indonesia

Background: Supraventricular tachycardia (SVT) is a heterogeneous group of cardiac tachyarrhythmia. Pregnancy accompanied by SVT is rare, with about 22-33 cases per 100,000 pregnancies reported. It is an emergency case that may lead to maternal and fetal death. In rural areas, some antiarrhythmic drugs are unavailable; on the other hand, the side effect of drugs may lead to abnormalities in the fetus.

Case illustration: A pregnant woman, thirty-five years old, came to the hospital with shortness of breath as the main complaint. ECG examination shows that there was supraventricular tachycardia. After stabilization was conducted and an anti-arrhythmia drug was given, she converted to a normal condition. An echocardiography examination showed typical structure. During pregnancy, blood volume increases up to 50%, contributing to cardiac output stretching the myocardial tissue. Hemodynamic changes result in an increased heart rate of about 20% during the third trimester due to a fall in systemic vascular resistance.

Conclusions:

SVT in pregnancy is a rare and emergency case that may lead to maternal and fetal death. Diltiazem may be chosen as the first-line antiarrhythmic drug if there is no other drug of choice.

KEYWORD: *Pregnancy; Supraventricular Tachycardia; Diltiazem; Antiarrhythmic drug.*

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Ambiguous ECG in an Octogenarian: Is It an Atrial rhythm (AR) or Just a Simple Left Atrial Enlargement (LAE)?

R. A. P. Fernandes[1,2] ,A. A. Lukito[1,2] ,S. Chandra[1,2] ,H. Hutabarat[1,2]

[1]Faculty of Medicine, Pelita Harapan University, Tangerang, Indonesia

[2]Siloam Hospital Lippo Village, Tangerang, Indonesia

Background: Ageing is proportionally associated with changes in the cardiac conduction activity. Variety of ECG features could be found in an octogenarian population. Considering as a population with multiple co-morbidities, ECG features such as an AR and LAE could overlap each other which baffling for clinician.

Case illustration: 82 years-old-man, hypertensive, multi-comorbidities, and strong familial history (FH) of CVD presented with a history of seizure. Physical examinations revealed BP 100/60 mmHg and HR 53 bpm. ECG presented was ambiguous whether it was an AR or SR with LAE. Laboratory result showed anaemia with reduced kidney function. Previous echocardiography showed dilated LA, concentric LVH with normal LVEF. Hypertensive heart disease in CKD with a history of seizure and a strong FH of CVD was confirmed. Regular hypertensive drugs and lifestyle modification was recommended. LAE itself can independently predict the development of variety cardiovascular disease and heart failure. In the other hand, AR might be cause by several possible causal but usually isn't dangerous. ECG criterion of LAE are bifid P wave >40 ms between the two peaks with the total P wave duration >110 ms in lead II, biphasic P wave with terminal negative portion >40 ms duration, and >1 mm deep in lead V1. AR might resemble a normal SR but origins from another atria focus other than the SA node. Upright/isoelectric P waves in lead I and inverted P waves in lead V6 is the most frequent ECG pattern of AR. However, it mostly appeared as an inverted P waves in inferior leads. Even though standard criterion for differentiation has been established, these ECG features could confuse clinicians in their daily practice. Due to their clinical significancy for further complications, distinguishing them is really a key factor. Therefore, correlation with clinical findings, age, co-morbidities, and supporting data such as an echocardiography is needed.

Conclusions:

ECG solely couldn't be use as a diagnostic criterion without further supporting examination to complete a diagnosis of the patient. Holistic examination is needed in preventing a misdiagnosis, especially in a newly cardio patient.

KEYWORD: *Atrial rhythm, LAE, Octogenarian.*

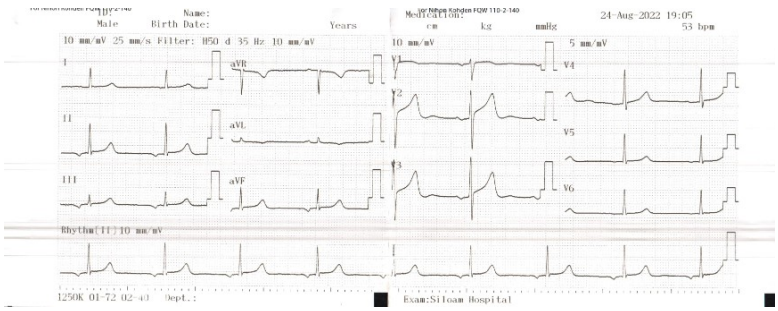

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Figure 1 ECG on admission

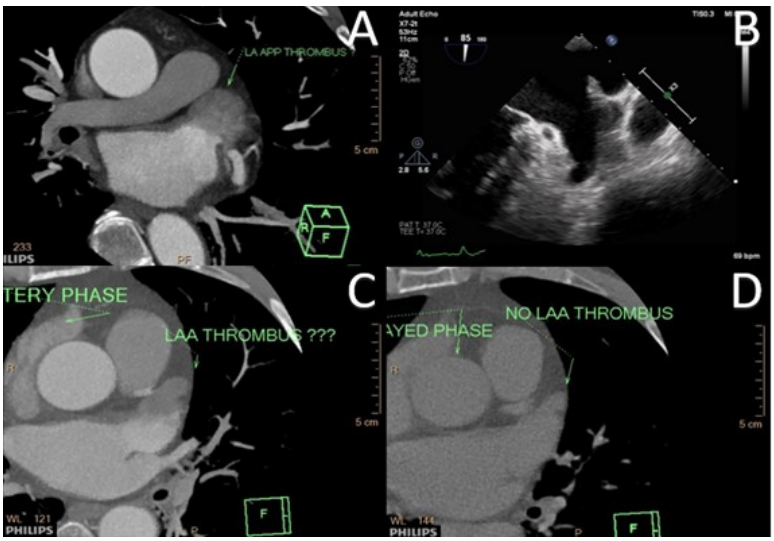
Atrial Fibrillation with Filling Defect Grade II in Left Atrial Appendage. Proceed or Defer Ablation?

Robert[2] ,E. J. Gunawan[1] ,Johan[1] ,I. Citra[2] ,B. Hartono[1] ,M. Munawar[1] ,D. L. Munawar[1] [1]Cardiology and Vascular Department of Binawaluya Cardiac Center
[2]Cardiology and Vascular Department of Sam Ratulangi University

Background: The use of contrast computed tomography (CCT) in evaluating left atrial appendage (LAA) thrombus has been reported to be useful besides transesophageal echocardiography (TEE). Filling defect (FD) in LAA is found in around 10% of patients who underwent atrial fibrillation (AF) ablation.
Case illustration: A 50-year-old man with AF who was scheduled for ablation had FD grade II in LAA from the CCT and was suspected of LAA thrombus. The patient was examined with TEE and there was no LAA thrombus found. With CCT modified technique when injecting contrast, delayed images for 25-60 seconds the FD in LAA disappeared and concluded there was no LAA thrombus. If FD is still persistent 1 minute after contrast injection, it is more likely to represent thrombus. Patient underwent AF ablation and there was no periprocedural complication including stroke or neurological deficit. This technique is according to multiple previous studies that suggest delaying images while injecting contrast would make FD disappear and could be ablated. The diagnostic accuracy was 94%, sensitivity and specificity improved significantly (100% and 99%) compared to non-delayed images. Nevertheless, patient with FD in LAA that underwent AF ablation has worse outcome, compared to non-FD, in 5 years cumulative incidence of recurrent atrial arrhythmia, heart failure hospitalization, stroke, and death.

Conclusion:
This case report showed that CCT with delayed imaging method is a noninvasive, fast, and sensitive modality for the detection of thrombus in the LAA. It is a reliable alternative to TEE.

KEYWORD: *Filling defect, computed tomography, atrial fibrillation, ablation.*



A. CCT non delayed / arterial phase image technique with FD grade II in LAA; B.TEE with no LAA thrombus; C. CCT after a month with delayed image technique (arterial phase), still had FD in LAA. D. CCT delayed image for 60 seconds (delayed phase), FD in LAA was disappear.

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Typical Atrioventricular Nodal Reentrant Tachycardia with Right Bundle Branch Block vs Ventricular Tachycardia: Old Battle between Supraventricular Tachycardia with Abberancy and Ventricular Tachycardia?

R. I. Rismawanti[1] ,A. Novitasari[1] ,J. S. Alexangela[1] ,A. Ciptasari[1] ,R. Ayuningtyas[1] ,W. Pamungkas[2] ,L. Pribadi[3] ,M. G. Suwandi[3]
[1]General Practitioner, Rumah Sakit Pusat Angkatan Udara Suhardi Hardjolukito, Bantul, Indonesia
[2]General Practitioner, Adisuciptjo Air Force Base, Bantul, Indonesia
[3]Cardiologist, Rumah Sakit Pusat Angkatan Udara Suhardi Hardjolukito, Bantul, Indonesia

Background: Differential diagnosis of wide QRS tachycardia (WQRST) on the ECG in emergency situations is still a challenging. The correct diagnosis is important for recommending the right therapy and figuring out the prognosis. Differentiating between supraventricular tachycardia (SVT) with aberrant conduction and ventricular tachycardia (VT) is key in the differential diagnosis of broad QRS tachycardia. Typical atrioventricular nodal reentrant tachycardia (AVNRT) is the most common AVNRT from SVT classification.
Case illustration: A man, 48 years old, presented to the ER with dyspnea three hours before admission after finishing his hemodialysis (HD). He denied having cold sweat, angina symptoms, headache, and syncope. Previously, he had hypertension and chronic kidney disease (CKD) stage V on routine HD with AV shunt access at right antebrachia for 4 years, on therapy with bisoprolol, amlodipine, candesartan, and nitroglycerin. On physical examination, we found tachycardia 110bpm and other findings within normal limits. Few minutes later, he felt palpitation, and ECG was performed and showed wide QRS tachycardia, ventricular rate was 150bpm, right axis deviation. A routine blood test showed increased SGOT 88U/L, SGPT 91U/L, CKMB 36U/I, ureum 94mg/dL, creatinine 10.70mg/dL, and rapid antigen SARS-COV2 negative. He was diagnosed with SVT typical AVNRT type with pre-existing right bundle branch block (RBBB), CKD stage V on routine HD, and hypertension, then received oxygen and amiodarone bolus dosages 2 times continuing with maintenance dosage. Then the ECG converted into RBBB with wandering atrial pacemaker and admitted to ICU.

Conclusions: Causes of WQRST are SVT with functional or persistent bundle branch block, VT, SVT with preexcitation, or ventricular paced rhythm. Management of WQRST includes vagal maneuvers or adenosine IV or procainamide IV or amiodarone IV or synchronized cardioversion. Diagnosing this condition requires speed and accuracy. If there are doubts, scoring calculations can be carried out such as the Brugada algorithm, Vereckei algorithm, Griffith criteria, Wellens criteria, lead II R wave peak time, RS/QRS ratio, and limb lead algorithm. Artifacts can be mistaken for WQRST, particularly if obtained as a rhythm strip in a telemetry or intensive care unit and may lead to additional but unnecessary tests or treatment.

KEYWORD: *Wide QRS tachycardia, ventricular tachycardia, supraventricular tachycardia with abberancy, right bundle branch block.*

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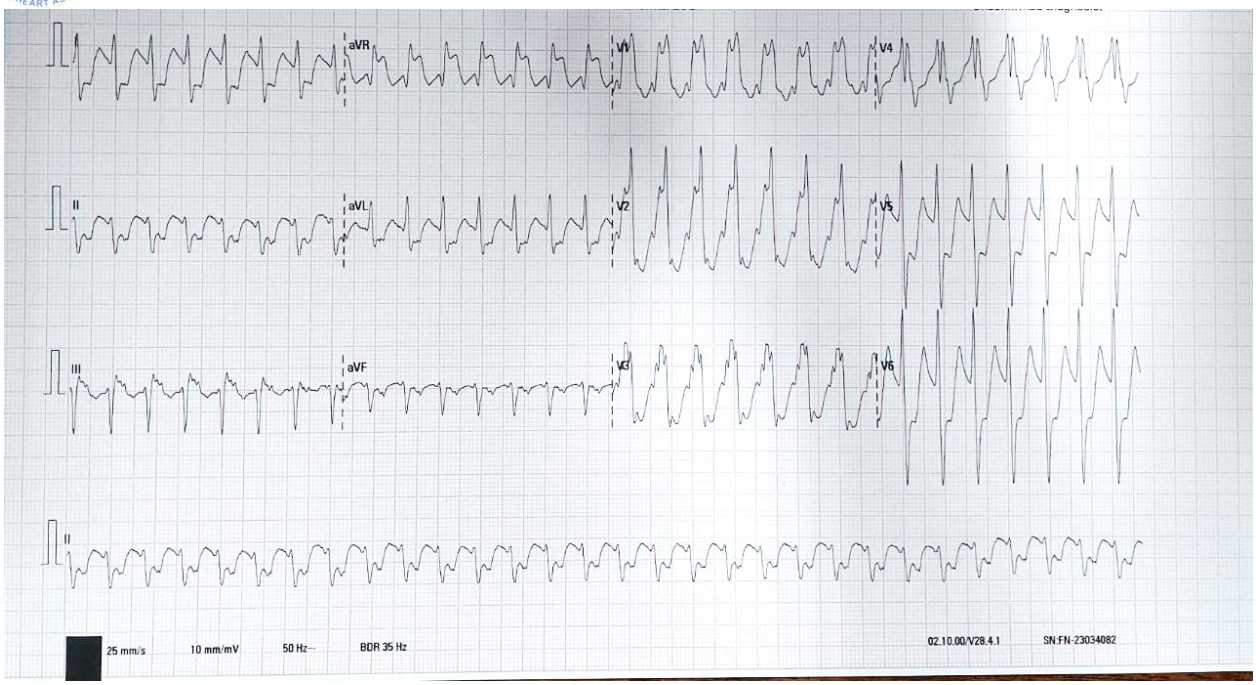

Figure 1 The ECG at ER with SVT typical AVNRT type with pre-existing right bundle branch block (RBBB)

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Sinus Arrhythmia vs Sinoatrial Exit Block 2nd Degree type 1: How to Differentiate?

R. I. Rismawanti[1] ,A. Novitasari[1] ,A. Ciptasari[1] ,R. Ayuningtyas[1] ,W. Pamungkas[2] ,L. Pribadi[2] ,M. G. Suwandi[2]

[1]General Practitioner, Rumah Sakit Pusat Angkatan Udara Suhardi Hardjolukito, Bantul, Indonesia
[2]General Practitioner, Adisuciptjo Air Force Base, Bantul, Indonesia
[3]Cardiologist, Rumah Sakit Pusat Angkatan Udara Suhardi Hardjolukito, Bantul, Indonesia

Background: In sinoatrial (SA) exit block, the sinus node continues to discharge at regular intervals, but some impulses are blocked and are unable to reach the surrounding atria. Second-degree type I SA block or SA Wenckebach should always be suspected when there is group beating. There is gradual delay in conduction between the sinus node and the atrium. Another common error that can be confused with sinus node dysfunction is sinus arrhythmia. In sinus arrhythmia, the sinus rate is irregular. Although sinus arrhythmia is a normal finding, the long P-P (or R-R) intervals can be easily mistaken for sinus pauses. In sinus arrhythmia, the difference between the longest and shortest P-P interval should be >10% or >0.12 seconds. The P waves are generally uniform in configuration and the P-R interval is usually the same. The shortening and lengthening of the P-P intervals are usually cyclic.
Case illustration: A woman 79-year-old patient presented to ER with loss of consciousness two hours before admission after fell when praying. She refused any angina symptoms. On examination, vital signs within normal limit and hypereflexia at upper and lower extremities. The ECG showed sinoatrial exit block 2nd degree type 2 but diagnosed as sinus arrhythmia by the general practitioner and atrial fibrillation normal ventricular response (AFNVR) by the neurologist. A routine blood test showed no abnormality and rapid antigen SARS-COV2 negative. The patient was diagnosed with motoric aphasia acute onset ec stroke non hemorrhagic OH1, AFNVR, DM type 2 on therapy, and HT uncontrolled. Then, given oxygen, piracetam, mecobalamin, cilostazol, lansoprazole, folic acid, amlodipine, candesartan, metformin, bisoprolol, and serial ECG next morning. ECG post therapy converted into sinus rhythm after 26 hours.

Conclusions: Establishing the diagnosis of sinus arrhythmia with sinus exit block grade 2 type 1 is prone to in the emergency department. So that an understanding of the diagnostic criteria really needs to be mastered by general practitioners.

KEYWORD: Sinoatrial exit block, sinus node dysfunction, sinus arrhythmia, stroke non hemorhagic.

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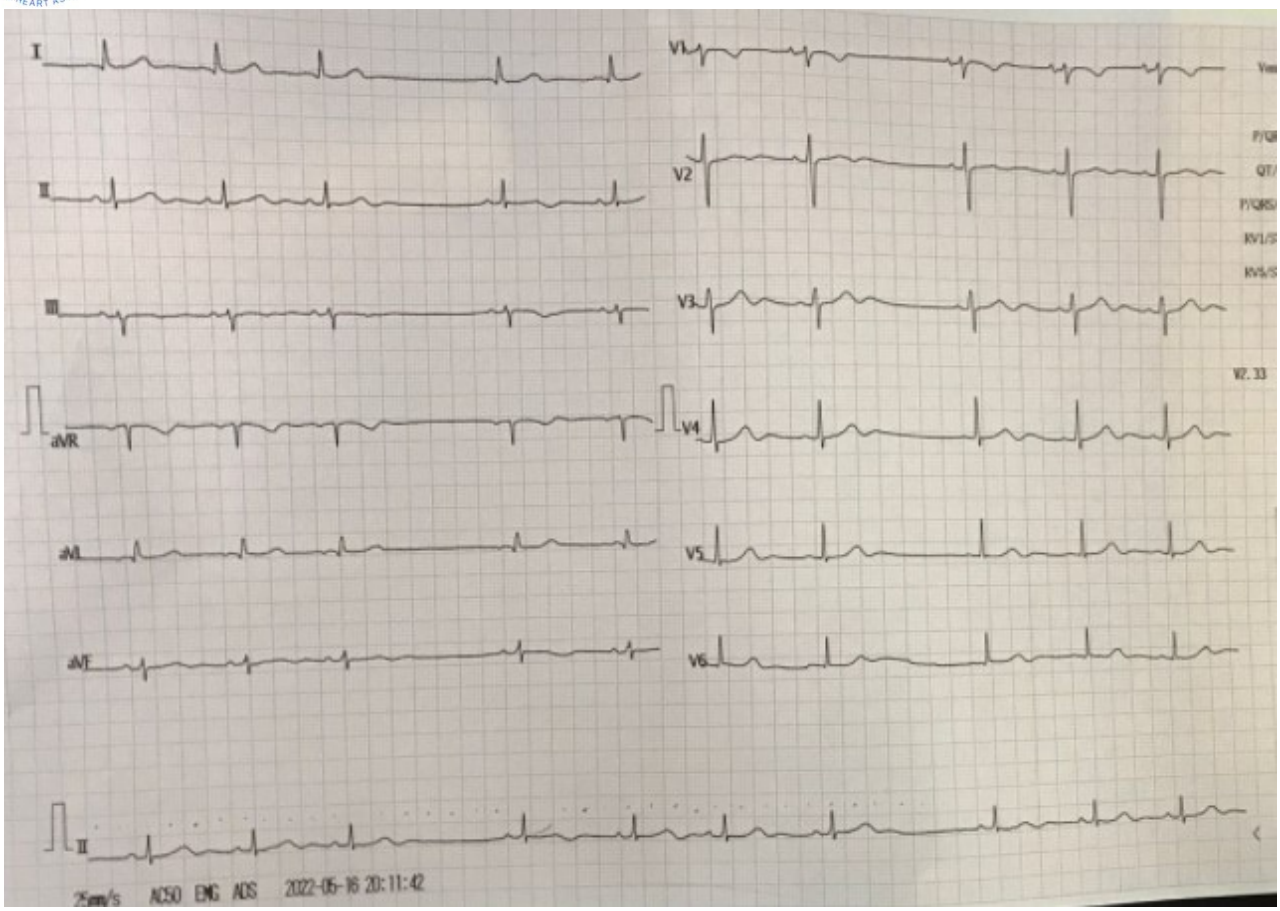

Figure 1 The ECG at ER with sinoatrial exit block 2nd degree type 2

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The Tale of ST Segment Elevation : Is it Acute Myocardial Infarction or Chronic Myocardial Infarction or both ? Could Electrocardiogram Distinguish them ?

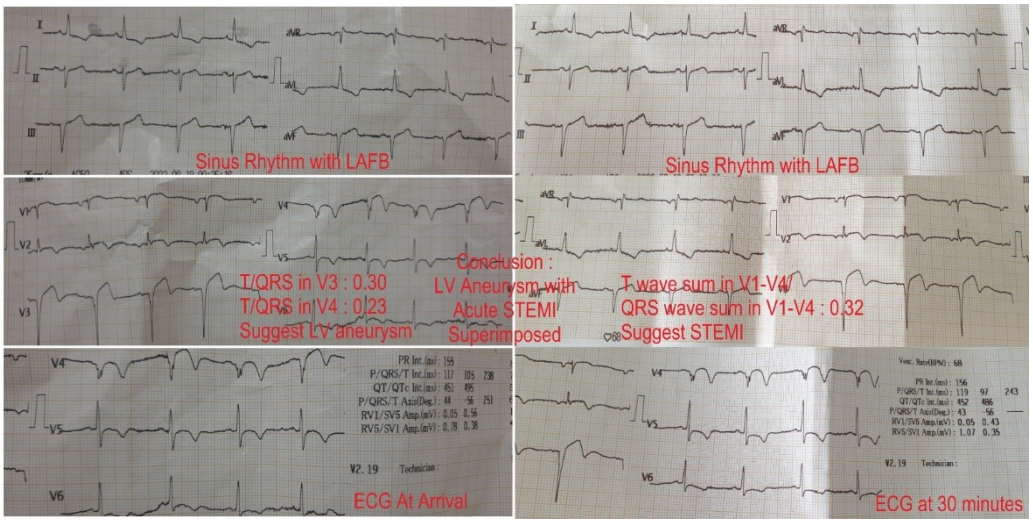
S. D. Sitanggang[1] ,S. Sembiring[2] ,B. Stefanus[2] ,A. A. Kesumaningputri[3]
[1]General Practitioner, Batu Ceper Primary Health Care, Tangerang, Indonesia
[2]General Practitioner, DR. Sitanala Central General Hospital, Tangerang, Indonesia
[3]Cardiologist, DR. Sitanala General Hospital, Tangerang, Indonesia

Background : ST segment elevation is a part of ACS and oftenly regarded as a sign of cardiac emergency reflecting acute myocardial infarction (AMI). However, not all ST segment elevation in ECG reflects acute process of MI. Dynamic ECG evolution and other supporting findings needs to be exist in order confirm whether the process is acute (STEMI) or chronic (LV aneurysm).This case report aims to present what ECG findings and formula could be used to differentiate acute or chronic process of myocardial infarction.

Case illustration : A 63 year old male patient routinely came to our outpatient clinic with a chief complain of prolonged dyspnea when he tried to walk a short distance since 3 months ago. No chest pain was reported. No history of diabetes. Afterwards, he was admitted to our emergency unit for initial treatment. His vital sign in ER, BP 182/97, HR 78 with regular pulse, RR 30, SpO2 97 % room air, glassgow coma scale of 15. Gallop and lung rales were found in his physical examination. First ECG recorded a sinus rhythm with high ST elevation and deep QS wave in V3–V4 leads. T wave inversions was found in V2, V5–V6 leads and biphasic T wave V3-V4 leads. Second ECG was implented in next 30 minutes. Second ECG shows no evolution of ECG. A study reported two formula developed to differentiate acute MI or LV aneurysm. First, T/QRS ratio in any V1-V4 more than 0.36 suggest STEMI and second, the sum of T wave in V1-V4 divided by sum of QRS in V1-V4 more than 0.22 suggest STEMI. In our case, first formula results in likely a LV aneurysm while second formula results in likely an acute STEMI. Hence, in our case, LV aneurysm might be superimposed with acute STEMI.

Conclusions:
Acute or chronic process of myocardial infarction may be distinguished by ECG findings such as QS and R wave presence, height of T wave and ST segment, T wave inversion, dynamic evolution along with reliable ECG formulas. However, these ECG findings and formula alone cannot determine the infarct process. Consequently, other supporting examination and clinical symptom need to be considered to confirm the diagnosis.

KEYWORD: *ST Segment Elevation ; Chronic MI ; Acute MI ; ECG.*



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Expecting the unexpected: acute perimyocarditis during dengue fever infection in limited settings – a case report

S. E. T. Tampubolon[1] ,S. A. Laksono[2]

[1]General Practitioner, RS Panti Secanti, Lampung, Indonesia

[2]Internist, RS Panti Secanti, Lampung, Indonesia

Background: Although uncommon, myocardial and pericardial involvement in dengue fever cases have been reported in several occasions. Since formal cardiac investigation is not part of the usual assessment of dengue fever cases especially in endemic area, the incidence of dengue myocarditis nor pericarditis remains unknown.

Case illustrations: A 28-year-old woman was admitted to the emergency room presented with five days of fever, nausea, abdominal pain, and headache. She has no prior medical or cardiac history. Her initial examination showed normal blood pressure of 102/60 mmHg, heart rate of 87 beats per minute, respiratory rate of 20 breaths per minute, temperature of 38.7^{o}C. Admission cardiac exam showed normal heart sound and normal electrocardiogram (ECG), with laboratory result of thrombocytopenia, positive Dengue IgM and IgG serology. Two days later, patient was slightly lethargic and experienced chills, shortness of breath and occasional chest pain. Physical examination then showed lower blood pressure (90/54 mmHg), heart rate of 63x/min, rapid breathing (28x/min), normal temperature and oxygen saturation. The heart sounds were normal with slightly audible pericardial friction rub, and dropped thrombocyte count as well as widespread ST elevations in ECG were noted. Chest radiograph was uneventful. Patient's symptoms together with laboratory and ECG findings suggested acute perimyocarditis. Exact diagnosis was difficult to be made with the unavailability of cardiac markers and limited access to cardiac imaging modalities. Refusal to be referred was noted by patient's family due to lack of health insurance. With fluid therapy and corticosteroids, the patient made to recovery in the next three days. Patient was discharged with resolution of the symptoms, normal laboratory and ECG exams. Dengue perimyocarditis is infrequent but may be life-threatening. Prompt recognition of atypical cardiac involvement of seemingly straightforward, judicious fluid resuscitation with continuous monitoring are essential in dengue perimyocarditis. Lack of resources especially in rural areas still remains a barrier.

Conclusions: A young female patient diagnosed with dengue fever complained of new onset chest pain. Physical examination and changes in ECG suggesting presence of acute perimyocarditis. In this case we report atypical cardiac manifestation of dengue fever.

KEYWORD: *Pericarditis, myocarditis, dengue fever.*

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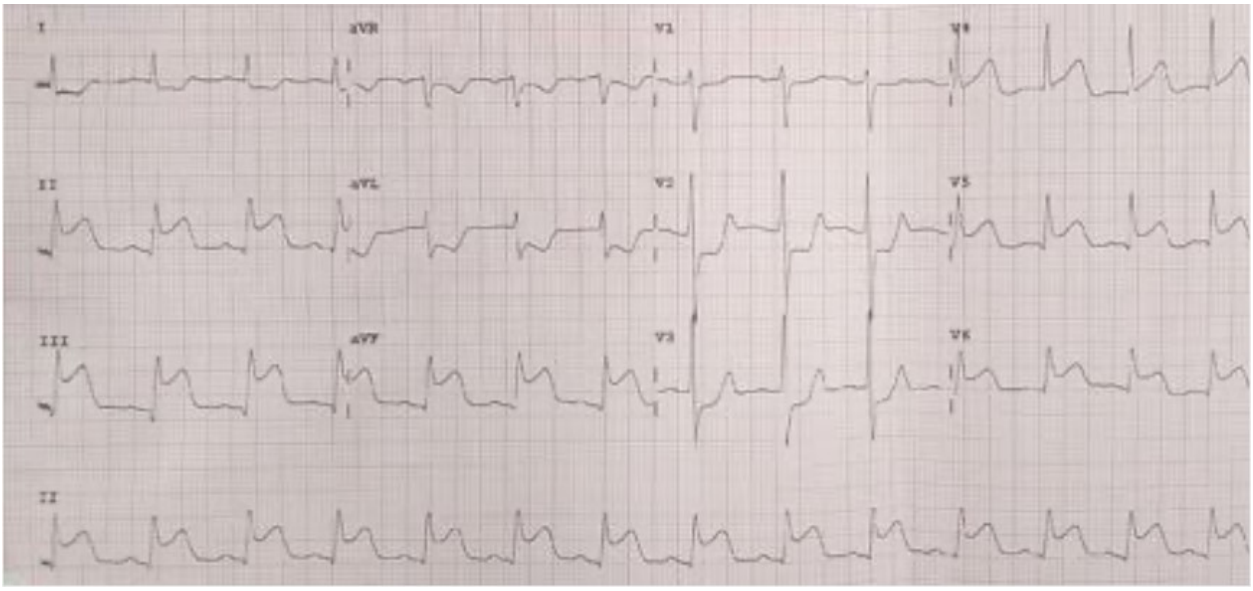

Figure 2. ECG Day 3 (chest pain complaint) showed ST elevation on lead II, III, aVF, V4-V6, and ST depression on I, aVL, V1-V3

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SND and AVND in a Young Pilot

S. Chandra[1] ,A .A. Lukito[1] ,R. A. P. Fernandes[1] ,H. Hutabarat[1]
[1]Department Cardiology and Vascular Medicine, Siloam Hospital Lippo Village, Tangerang, Indonesia.

Background: Epidemiologic information about SND is limited and difficult due to the variable disease of manifestation and ECG findings. To define sinus node dysfunction both the symptoms and the ECG findings has to be present. Age showed the greatest significance as a risk factor for having SND. The common cause SND was formation of scar tissue in the sinus node (for example was rheumatic fever, myocarditis).
Case illustration: A 39 year-old man Caucasian presented with chest discomfort and near syncope. His pulse was 42 bpm before admission. No previous medical history noted. On physical examination, blood pressure 115/72 mmHg, pulse 54 bpm regular. ECG on admission was sinus bradycardia with normal PR interval. Laboratory results were normal cardiac enzyme, normal NT-proBNP, and normal thyroid hormone. The past history of living in Papua, malaria test was done and the result negative. First imaging, MSCT coronary result was no significant stenosis. Second imaging, cardiac MRI result was post myocarditis, diffuse patchy myocardial fibrosis at LV and RV, but not suit for Chagas disease or Sarcoidosis, with preserved LVEF and RVEF, no rest perfusion defect. Spyder ECG record no AF was detected. Next step was electrophysiology study. When the EP study was ongoing there was an episode of atrial fibrillation. The final result was SND and AVND. Recommendation therapy for this patient was dual chamber pacemaker.

Conclusions:
In this case illustrates a young age man Caucasian and without contributory medical history had presentation of SND. Most likely the etiology of SND and AVND was diffuse myocardial fibrosis at LV and RV (post myocarditis) but not suitable for Chagas disease or sarcoidosis. Recommendation for this patient was dual chamber pacemaker because he still in young age and had high-risk job as a pilot.

KEYWORD: *SND, AVND, Young-age, myocarditis.*

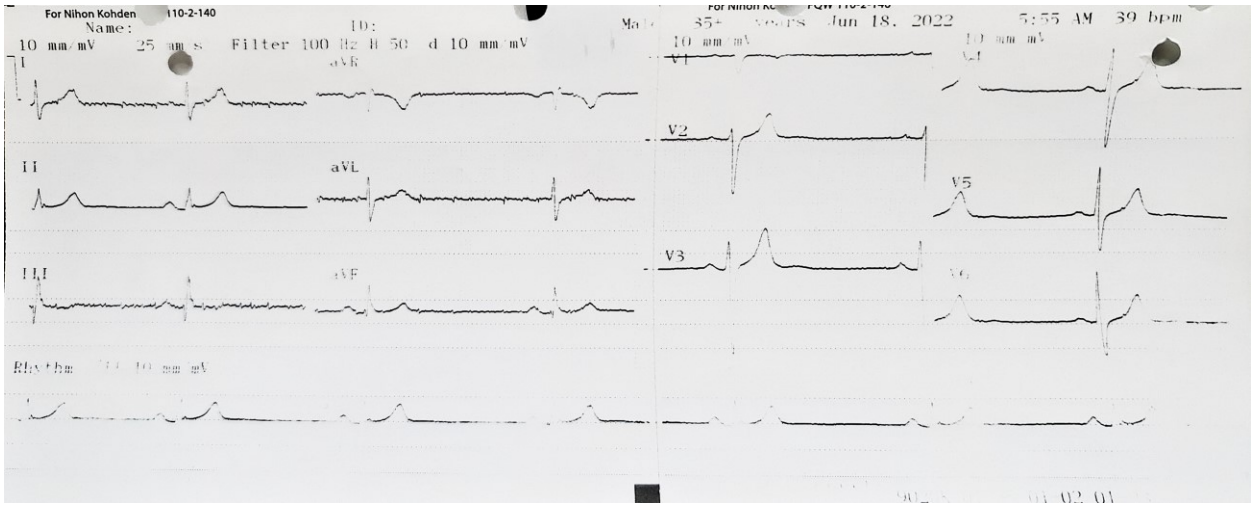

Figure 1 ECG at admission

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Axillary Venous Spasm during Pacemaker Implantation: A Rare but Serious Phenomenon

T. Oktavio[1] ,S. Adiwinata[1] ,B. Setiadi[1]
[1]Department of Cardiology and Vascular Medicine, University of Sam Ratulangi, Prof. Dr. R. D. Kandou General Hospital, Manado, Indonesia

Background: Venous spasm during pacemaker implantation is a rare phenomenon. Larger proportion of muscle fibres in the tunica media and elastic fibers in the tunica intima of veins predisposes to contraction.
Case illustration: A 55-years-old female presented with presyncope, recurrent palpitation and chest pain. She had history of hypertension. Physical examination showed heart rate 55 bpm, blood pressure 124/72 mmHg, with a normal heart sounds and no murmur. Electrocardiogram showed Mobitz I Second-degree atrioventricular block. Electrocardiogram and echocardiogram did not show any evidence of ischaemia. A Holter monitoring showed several bradycardia episodes with the lowest rate around 40 bpm. Patient was diagnosed with Sick Sinus Syndrome and dual chamber pacemaker implantation (DDD) was planned. The left axillary vein was selected for venous access. A venogram was performed and showed a good vein access. The axillary vein could not be cannulated despite multiple attempts. The second venography showed a spasm in both axillary and subclavian vein. A bolus of intravenous 10 mg isosorbide dinitrate was given. After 15 minutes, third venogram showed the spasm had been relieved partially. Single chamber pacemaker was implanted because of the spasm.

Conclusions: The mechanism of venous spasm is not clearly understood, it may be related to direct mechanical vascular injury, compression by surrounding tissues, temperature and chemical factors. Intravenous isosorbide dinitrate, nitroglycerine, calcium channel blocker, and sedatives might have role in shortening the spasm duration. Monitoring and precaution should be performed to prevent venous spasm during pacemaker implantation.

KEYWORD: *Venous spasm, single chamber pacemaker, axillary vein spasm.*

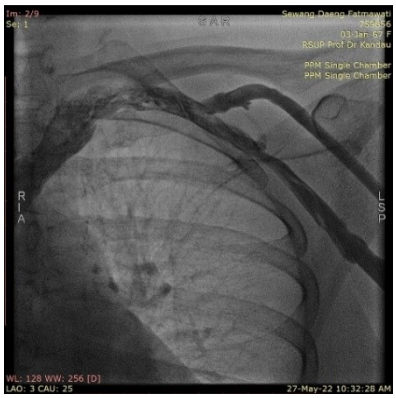

Figure 1 Venogram of Left Axillary Vein Subclavian Vein

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The Story of Arrhythmia in Hypertrophic Cardiomyopathy : a Series of Unfortunate Events

V. C. Dewanto[1] ,F. Hidayati[1] ,E. Maharani[1]
[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada, Yogyakarta, Indonesia

Background: People with hypertrophic cardiomyopathy are at risk for having irregular heart beats. The disease is characterized by left ventricular hypertrophy not attributable to another cardiac, systemic, or metabolic disease. It presents significant clinical heterogeneity consequences of possible symptoms, from heart failure to sudden cardiac death. Here, we present varies of arrhythmia cases to report the damage outcome of hypertrophic cardiomyopathy.
Case illustration: These cases involved 5 patients with hypertrophic cardiomyopathy confirmed by trans-thoracal echocardiography and cardiac resonance imaging. The first patient was a male, 30 y.o., survivor of sudden cardiac arrest, also had an atrial fibrillation with rapid ventricular response, the imaging examination showed a significant asymmetrical hypertrophy with a decreased left ventricular function. He was proceeded to an implantable cardioverter defibrillator implantation. Meanwhile, the second patient was a male, 51 y.o., experienced a dyspneu of congestive heart failure class functional IV showed a junctional rhythm with episode of ventricular tachycardia. He was managed to permanent pacemaker implantation but then showed a worsening of cardiac function and not survived. The third patient was a male, 24 y.o., with frequent palpitation and presyncope symptoms, had an atrial but not ventricular tachycardia. He was managed with certain medication and also considered to have cardioverter defibrillator. The fourth patient was a female, 62 y.o., who had a sudden loss of consciousness suspected neurologic symptom, but the monitor electrocardiography showed frequent non sustained ventricular tachycardia followed by torsade de pointes. The following echocardiography highlighting an asymmetrical hypertrophy of left ventricle as the main cause of her condition. The last patient, was a female, 57 y.o., experiencing a frequent angina symptom, she had a frequent non sustained ventricular tachycardia, was reported having a significant concentric hypertrophy of left ventricle.

Conclusions:
A strategy of more frequent or prolonged monitoring would lead to earlier arrhythmia recognition and the potential for appropriate treatment. Considerable effort focused on improving the precision of such a malignant arrhythmias risk prediction in hypertrophy cardiomyopathy will improve the outcome and help in reducing morbidity and mortality of patients.

KEYWORD: *Life threatening arrhythmia, hypertrophy cardiomyopathy, ventricular tachycardia, atrial tachycardia, sudden cardiac death.*

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Paroxysmal atrial fibrillation in a case of adult ventricular septal defect and pulmonary hypertension complicated by hypokalemia

V. A. N. Fatimah[1]
[1]Raffa Hospital, Cilacap, Indonesia

Background: Structural remodeling in congenital heart diseases (CHD), particularly those which involve the atrium, can trigger the development of atrial fibrillation (AF). The remaining atrioventricular synchrony may predict the fast ventricular response. Meanwhile, hypokalemia which commonly follows the long-term use of furosemide in CHD puts patient at risk of experiencing AF. Adequate rate and rhythm control is sought to prevent the negative hemodynamic effect.
Case illustration: A 23 years old female presented to the emergency department with palpitation that started 3 hours before admission. Her heart rate was irregular at around 154 beats/minute. Compared to her previous electrocardiogram, she developed an episode of paroxysmal AF. She had a history of readmission due to heart failure and arrhythmia from subaortic ventricular septal defect (VSD) and pulmonary hypertension (PH). She was on 20mcg daily Dorner®, 20mg daily furosemide, 12.5mg daily spironolactone, 2.5mg daily ramipril, 2.5mg daily bisoprolol, and 20 mg bid sildenafil. The dose of Dorner® was adjusted and bisoprolol and ramipril were added after the last time she had an episode of multifocal premature ventricular contractions (PVC). She was hypokalemic (2.6mmol/L). Bolus of 150mg amiodarone for 15 minutes and correction with 25mEq kalium chloride resulted in conversion to sinus rhythm. During hospitalization, Dorner® and ramipril was stopped while spironolactone dose was escalated to 25mg daily. The sinus rhythm was maintained until she was discharged on the 4th day.
Conclusions:
This case depicted the paroxysmal AF in an adult VSD complicated by PH and hypokalemia. Kalium correction, rhythm control with amiodarone, and rate control with beta-blocker were beneficial in converting the rhythm into sinus. Starting the anticoagulant is the next approach to be considered if the AF recurs and/or becomes permanent.
KEYWORD: *Atrial fibrillation, adult ventricular septal defect, pulmonary hypertension, hypokalemia.*

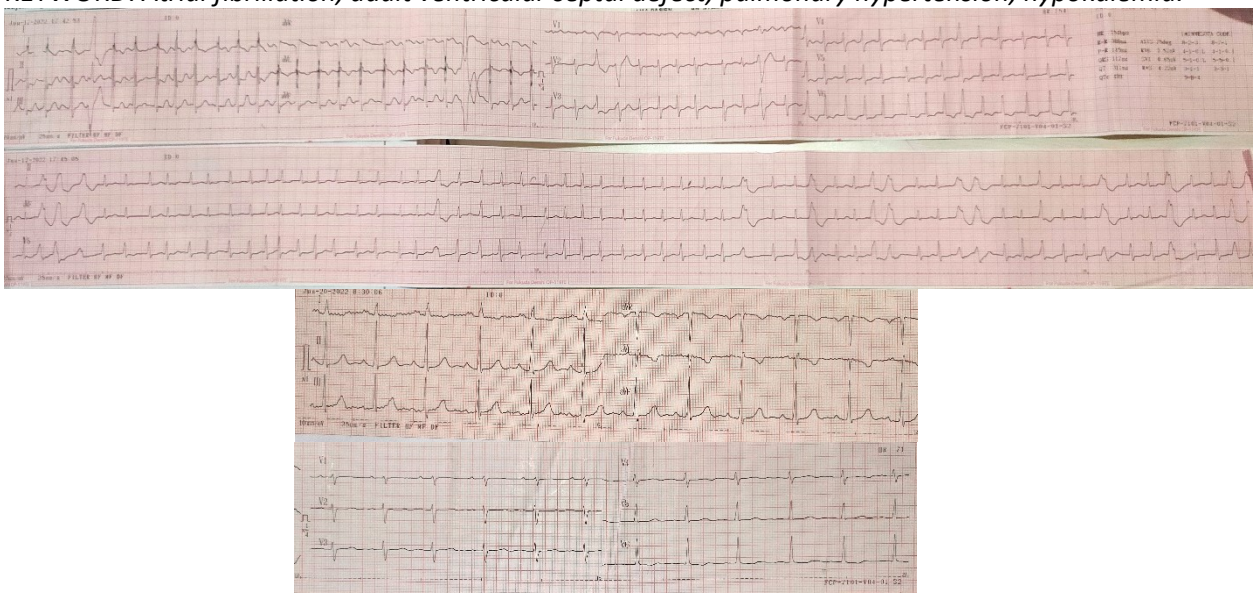

Figure showing the ECG of AF with multifocal PVCs (top) and conversion to sinus rhythm after kalium correction and rate-rhythm control (bottom)

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His Bundle Pacing corrected Intrahissian block in Pacemaker Induced Cardiomyopathy

V. K. Fadhilah[1] ,G. Karwiky[1]
[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Padjadjaran, Dr. Hasan Sadikin General Hospital, Bandung, Indonesia

Background: Chronic right ventricular pacing (RVP) can cause deleterious effects on cardiac function causing pacing-induced cardiomyopathy (PICM). A new technique has been introduced to prevent this complication. His bundle pacing (HBP) is a physiologic alternative to RVP, it reduces the risk of heart failure (HF) hospitalization, death, and/or upgrade to biventricular pacing compared to RVP. Patients with decreased LVEF to 40% may represent only the tip of the iceberg of PICM. Patients with symptoms of HF even before the decrease in LVEF can have potential manifestations of PICM. The prevention of PICM is a better strategy than treating it. Approach to prevent and treat PICM may include CRT and CSP (Conduction System Pacing). The present case describes a patient with symptoms of HF with preserved EF who got upgrading from single RVP to HBP.
Case illustration: A 63-year-old male has been implanted permanent pacemaker in 2014 because of total AV block. He came back to the hospital in 2022 for battery change. He has history of dyspnoea on effort. His ECG showed ventricular pacing rhythm with QRS duration of 160 ms. His echocardiography test result was concentric LVH, preserved EF, and diastolic dysfunction grade I. The patient got upgrade pacemaker because of experiencing symptoms of HF from single RV pacing to DDDR with the HBP technique. His ECG test 1 week and 1 month post-procedure showed pacing rhythm with narrow QRS 130 ms. His quality of life compared before and after upgrading pacemaker by Aquarel and SF-36 questionnaires showed improvement.

Conclusions:
HF symptoms may be caused by atrioventricular dyssynchrony, intraventricular dyssynchrony, and interventricular dyssynchrony. Upgrade to HBP could reduce the symptoms of HF evidenced by the increase in the quality of life and more physiologic conduction (narrower QRS). HBP may be an excellent option to prevent and treat PICM in the first place.

KEYWORD: His bundle pacing, Intrahissian block, Pacemaker-induced cardiomyopathy, Single right ventricular pacing, Upgrade pacemaker.

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Brugada Pattern in NSTEACS: Is it Brugada Phenocopy or Brugada Syndrome in Disguise?

W. Pamungkas[1] ,R. I. Rismawanti[2] ,M. R. A. Shiddieqy[3] ,A. Novitasari[2] ,A. Ciptasari[2] ,L. Pribadi[4] ,M. G. Suwandi[4]

[1]General Practitioner, Adisuciptjo Air Force Base, Bantul, Indonesia
[2]General Practitioner, dr. Suhardi Hardjolukito Air Force Central Hospital, Bantul, Indonesia
[3]Internship, dr. Suhardi Hardjolukito Air Force Central Hospital, Bantul, Indonesia
[4]Cardiologist, dr. Suhardi Hardjolukito Air Force Central Hospital, Bantul, Indonesia

Background: Brugada phenocopy (BrP) is different phenomenon from congenital Brugada syndrome (BrS). BrP is elicited by various underlying conditions such as myocardial ischemia, pulmonary embolism, electrolyte imbalance and poor ECG filters, but improvement of the underlying condition in line with the ECG. BrS is congenitally inherited cardiac channelopathy with type 1 and 2 ECG patterns that predispose to malignant ventricular arrhythmia and sudden cardiac death. Differentiating between BrS and BrP is the important key to considering further management.
Case illustration: A man, 49 years old, came to ER with chief complaint worsening epigastric pain with VAS 5 for the last 5 hours before without radiated pain and sore all over. There was no nausea, diaphoresis, headache, or syncope. Past medical history was denied but two sudden deaths with unknown cause in family history at >45 years old. During the physical examination, we discovered BP 188/115, HR 110, RR 28, afebrile, SpO2 98% room air, and other normal values. The ECG showed sinus tachycardia, left axis deviation, and coved ST segment lead V1-2, but at ECG serial obtained ST-T changes in leads V5-6. A routine blood test showed leukocytosis at 12.820/mm3, increased neutrophil lymphocyte ratio at 8.42, mild hypokalemia at 3.41mEq/L, and normal CKMB 21U/L. He was diagnosed with hypertensive emergency with cardiac target organ damage, non-ST segment elevation acute coronary syndrome (NSTEACS), hypertensive heart disease, and brugada pattern type I, then given heparinization in cardiac care. The serial ECG showed saddle-back shaped ST segment leads V1-2. Coronary angiography was established, the result was non-significant stenosis in LAD with TIMI 2 Flow.

Conclusions: Using history, identifiable underlying condition, and sodium channel blocker test, BrP can be distinguished from BrS. Determining whether it's BrP or BrS must be done precisely. The Brugada pattern in BrP will normalize as the underlying condition is resolved. Indeed, the defining feature of BrP is the absence of true congenital BrS.

KEYWORD: *Brugada pattern, brugada phenocopy, brugada syndrome.*

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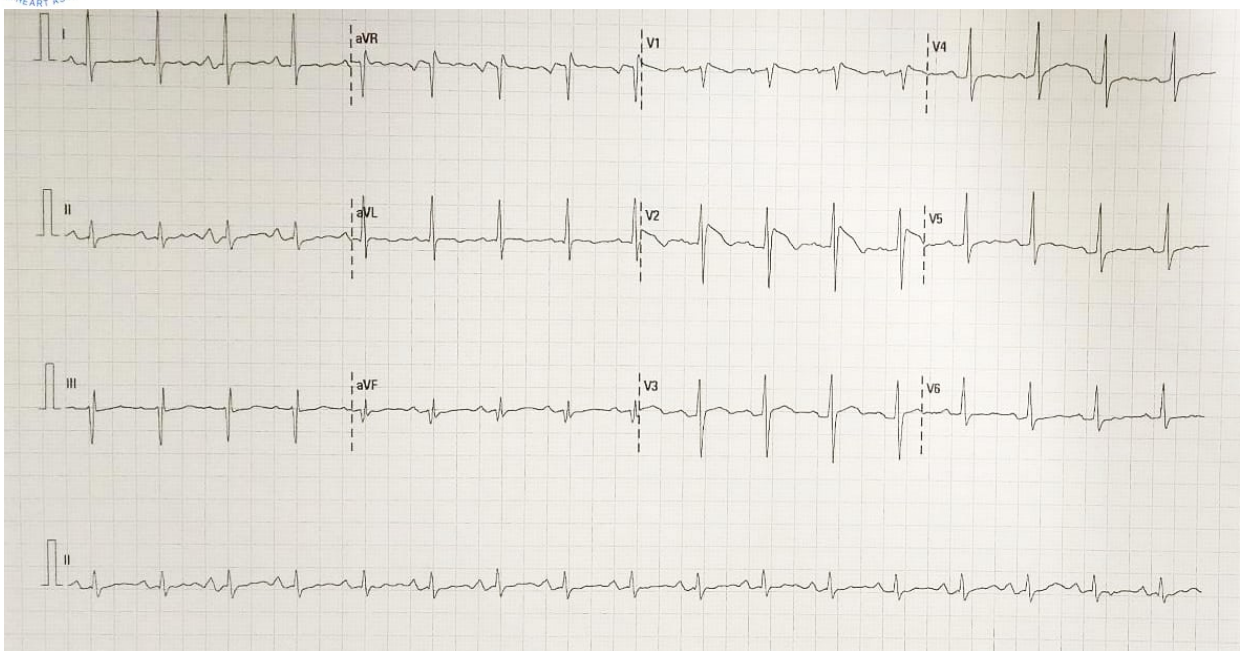

Figure 1 First ECG at ER showed a LAE, Brugada pattern type 1 with incomplete right bundle branch block.

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WPW SYNDROME CONCOMITANT WITH ATRIAL FIBRILLATION: WHICH ONE SHOULD BE TREATED FIRST?

W. Safitri[1] ,Y. Yuniadi[1]
[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

Background: WPW syndrome is a clinical entity characterized by pre-excitation of the ventricles with symptoms of paroxysmal tachycardia due to accessory pathway between atria and ventricle. Atrial fibrillation in patients with Wolff-Parkinson-White (WPW) syndrome can occur concomitantly and may lead to syncope, ventricular fibrillation, and sudden cardiac death. Radiofrequency ablation (RFA) is one of the treatment options for this disorder, but it is still a debate whether WPW should be ablated first or should be ablation done for both disorders. To present two cases of WPW with concomitant atrial fibrillation, the relationship between these two conditions and their appropriate management.
Case illustration: A 75 years old man come to Arrhythmia outpatient clinic with chief complain recurrent syncope. ECG and holter examination confirmed the diagnosis WPW syndrome and atrial fibrillation. The patient then underwent an electrophysiological examination and Radiofrequency ablation. The RFA of Accessory pathway was successful, WPW and AF were resolved. A 19 years old woman in outpatient clinic came with chief complain recurrent palpitation since one year before visitation. ECG confirmed WPW pattern. This patient then underwent EPSL and Radiofrequency ablation. During atrial stimulation in EPSL, atrial fibrillation with rapid ventricular response was triggered. RFA of the Accessory Pathway was done successfully.

Conclusions:
Wollf Parkinson White syndrome coexist with atrial fibrillation can lead to worsen outcome such as sudden cardiac death. Appropriate treatment for WPW syndrome can overcome the occurrence of atrial fibrillation and sudden cardiac death.

KEYWORD: *Wpw syndrome, atrial fibrilltation, treatment, radiofrequency ablation.*

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After Cardiac Arrest in hypertrophic cardiomyopathy combined with Trifascicular Block : Dilemma Between Implantable Cardioverter Defibrillator (ICD) and Dual Chamber Permanent Pacing

M. R. Fadlan[1] ,A. Rizal[1]
[1]Department of Cardiology and Vascular Medicine, Faculty of Medicine, Universitas Brawijaya, dr.Saiful Anwar General Hospital, Malang East Java, Indonesia

Background: Hypertrophic cardiomyopathy (HCM) is a common disorder of cardiac muscle associated with sudden cardiac death (SCD). Bradyarrhythmia such as atrioventricular conduction disturbance, a relatively rare complication associated with hypertrophic cardiomyopathy, may also cause syncope and sudden death.
Case illustration: A 26 years old woman came with the chief complaint worsening light headedness, dizziness associated with a hypotension and bradycardia since 6 hour before hospital admission. She had history of syncope 6 month ago. On presentation, patient's blood pressure was 86/62 mmHg with HR 42x/minutes. The bradycardia progressing to asystole. Cardiopulmonary resuscitation (CPR) was started immediately. Return of spontaneous circulation (ROSC) was achieved in 10 minutes with total adrenaline 3 mg. The electrocardiogram (ECG) was suggestive of trifascicular block showed in Left Axis Defiation, Right Bundle Branch Block (RBBB), and 2:1 AV block with ventricular rate 38x/minute. The complete blood count, comprehensive metabolic panel, troponin I and electrolyte serum were unremarkable. Echocardiogram revealed moderate asymmetric septal left ventricular hypertrophy with ejection fraction of 68%, Left Atrial (LA) size was 56.8 mm,peak and mean left ventricular outflow tract (LVOT) gradients less than 7 and 6 mmHg respectively with no increase in gradient with provocative maneuvers such as Valsalva. According this data risk of SCD at 5 years was 8.42% and ICD should be considered Subsequently, an Implantable cardioverter defibrillator (ICD) with Pacemaker function was implanted to the patient.

Conclusions:
This case describes cardiac arrest caused by severe bradyarrhythmias related to trifascicular block block in a patient with HCM who was managed successfully with a ICD implantation. Although a rare complication, we should keep in mind the probability of atrioventricular (AV) block as a cause of sudden cardiac arrest in a patient with HCM

KEYWORD: *HCM , Cardiac Arrest, Trifascicular block.*