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Laporan Kasus

A young man with typical STEMI presentation: A case of myocarditis A Cardiac MRI Diagnosis

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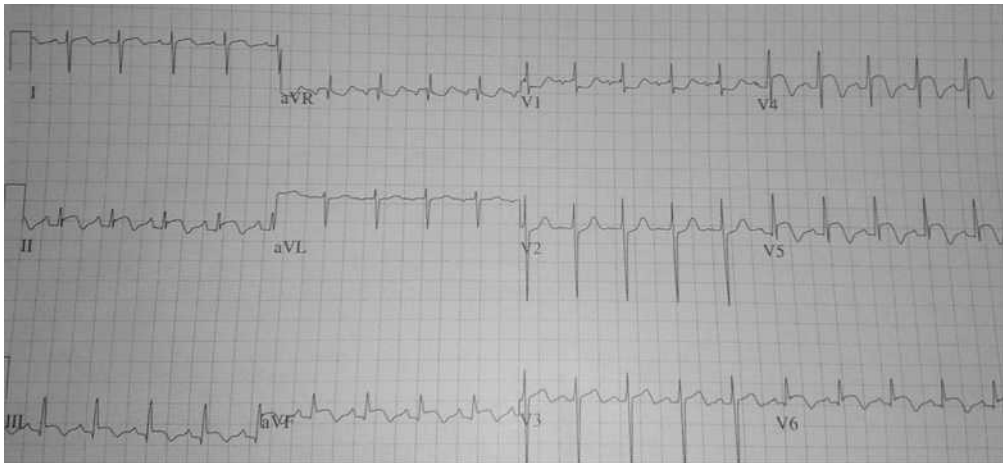
Case report

A 19 years old man, originated from Kongo, presented to a hospital with acute chest pain since several hours before admission. The pain was sharp in quality, distributed in the middle of left chest, not exercise related and not radiating. Although there were times in which the patient felt the pain was downgrading, it did not totally fade away. The patient was non-smoker with no risk factors of diabetic, hypertension and dyslipidaemia. He was oriented with temperature of 38.5 ° C, pulse was 110 tpm, blood pressure was 128/85 mmHg with fast and shallow respiration at the rate of 32 tpm. S1/S2 normal, no additional sound and significant murmur detected. Apart from minimal harsh respiratory sound at the basis of left lung, the examinations of JVP, lung and abdomen were unremarkable. Since 7 days before admission, the patient suffered cough, runny nose and throat pain with feverish but did not seek treatment for it. ECG showed sinus tachycardia with ST-elevation in inferior and anterolateral leads. Elevated cardiac enzymes CKMB 32 and Trop T 1,86.



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Bedside trans-thoracic echocardiography demonstrated inferolateral akinesis, with borderline LV function. Small circumferential pericardial effusion was noted. Given the need to proceed to immediate treatment should this be coronary events, the patient was immediately referred for angiogram. The angiogram, however, revealed the absences of thrombus and significant stenosis and ruled out coronary artery disease. The patient was then referred to CMR for further investigation.

Cardiac MRI with SPAR T2 weighted images showed high signal intensity in the anteroseptal segments, indicating the likelihood of edema/ inflammation. Delayed hyper-enhancement techniques with gadolinium also showed a patchy hyper-enhancement in the anterior and anteroseptal (mild-wall and epicardium), which was partly corresponding to the areas of high signal on the T2 sequences. MRI also recorded a reduced ejection fraction of both right and left ventricles (53% and 39% respectively), increased total cardiac mass and mass index (184 g and 112 g/m2 respectively), with significant reduction of SV of right and left ventricle (59 ml and 53 ml respectively). Circular pericardial effusion, left- and right- sided pleural effusion. This MRI findings were highly suggestive of acute myocarditis.

Discussion

Myocarditis is defined as the inflammation of myocardium with associated myocellular necrosis.

Figure 1. T2 weighted images

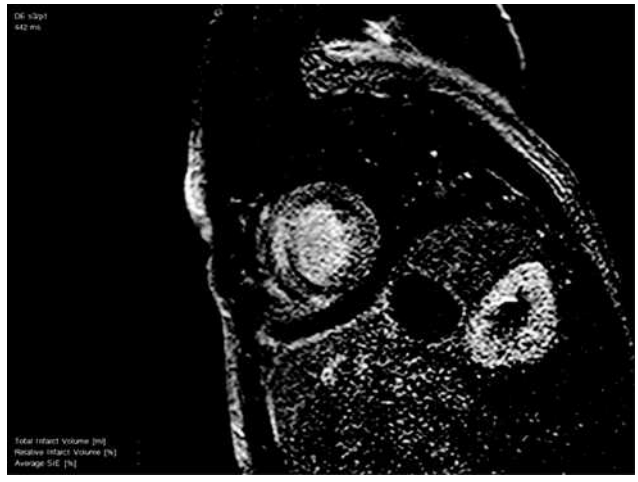

Figure 2. Delayed hyperenhancement on mid SA

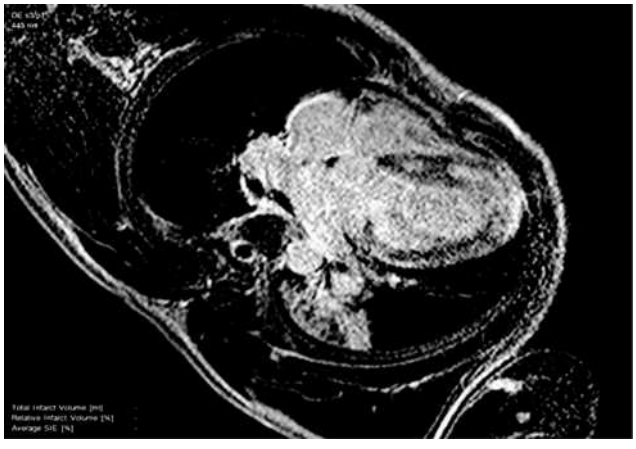

Figure 3. Delayed hyperenhancement on HLA

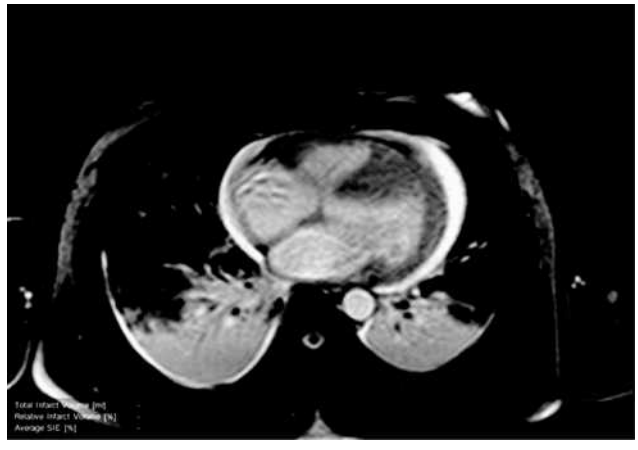

Figure 4. White blood axial

The causative agents may include infections, systemic diseases, drugs and toxin.[1] Myocarditis patients may present with various clinical symptoms, ranging from asymptomatic or non-specific symptoms (with subtle findings of ECG and echocardiography) to acute heart failure or death. They may also present with clinical signs and symptoms mimicking acute myocardial infarction.[2] In this subset, the presence of specific chest pain, completed with ECG and biomarkers findings suggestive of myocardial infarction may mislead clinician to the diagnosis of acute myocardial infarction. Although the incidence rate of this subset is unknown, the prevalence of normal angiography in the patients presenting with clinical signs and symptoms signifying acute myocardial infarction was 1-12%, depending on the definition of normal stenosis applied.[3] This prevalence increased significantly to be roughly 20% when the population of interest was the people of less than 35 years old.[4] Although some conditions, such as coronary vasospasm, thrombosis/embolization with spontaneous lysis, cocaine abuse, aortic dissection and hypercoagulable states, might be underlying such cases, many believe that myocarditis is the most prominent cause. Angelini et al reported the cases of 12 patients who were admitted to coronary unit due to severe chest pain, ST elevation on ECG, increased creatinin kinase and MB isoenzym with wall motion abnormality on echocardiography who were subsequently found to have normal angiography. Of these patients, 11 had evidence of myocarditis in endomycardial biopsy.[2]

Using Dallas criteria, endomyocardial biopsy has remained the gold standard for the diagnosis of myocarditis. Under this examination, myocarditis is defined as the findings of inflammatory cellular infiltrate with evidence of myocyte necrosis; while borderline myocarditis is defined as inflammatory cellular without myocite necrosis.[5] In addition to histopathological examination, the assessment of viral genome through polymerase chain reaction, serologic examinations of creatinin kinase, troponin I, troponin T, TNF alfa and the use of various non-invasive means such as atinmyosin scintigraphy and echocardiography digital image processing have been supportive for the diagnosis.[6]

Cardiac MRI has become the most promising technique and primary tool for non-invasive assessment of myocardial inflammation in patients with suspected myocarditis. Contrast-enhanced T1-weighted subtracted imaging was reported to have 100% of both sensitivity and specificity in detecting myocarditis, and an additional post-contrast T1 would be able to distinguish early phase (nodular enhancement) and later phase (diffuse enhancement) of the disease.[7] MRI can elucidate anatomic and morphological information of the heart and at the same time can provide accurate tissue characterization through the measurements of T1 and T2 relaxation times and spin densities. As active myocarditis is associated with edema and cellular swelling due to myocyte injury, the assessment of relaxation time provides measure for its elucidation.[8] Under MRI, myocarditis is identified as a typical pattern of contrast enhancement, which originates from epicardium while still sparing subendocardial layer. In contrast, myocardial infarctions show subendocardial enhancement.[9] Some studies have reported the typical findings of myocarditis in MRI. First, early in the presentation of myocarditis, there is focal myocardial enhancement that is turning to be global enhancement during later imaging times; this will return to baseline in 90 days.[10] Second, using T1 spin-echo cine MR angiography and gadolinium-enhanced spin-echo imaging it was revealed the association between global wall motion abnormalities (hypokinesis, akinesis and dyskinesis) and focal myocardial enhancement in myocarditis cases and the presence of these two states strongly support the diagnosis of myocarditis.[11] In addition to this, MRI can be used as a tool to confirm predictive value of 'the gold standard' biopsy in detecting myocarditis. Using a new technique, Mahrholt et al performed left ventricular biopsy in the region showing marked contract enhancement and revealed that this specific site biopsy resulted in positive and negative predictive values of detecting myocarditis were 71 % and 100 % respectively.[12]

Recently, The International Consensus Group on CMR Diagnosis of Myocarditis has introduced the recommendation on the current state of the art use of CMR for myocarditis. The recommendation includes some pivotal aspects on the use of MRI in myocarditis such as indications, protocol standards and diagnostic criteria (Lake Louise Criteria).[13] This recommendation also promotes the MRI as the most reliable non-invasive tool of diagnosing myocarditis, as an alternative to invasive mean of endomyocardial biopsy.

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