Sudden Cardiac Death in Tuberculous Myocarditis, a Review

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Introduction: Tuberculosis (TB) is still a big burden for Indonesia and the rest of the world. The increase of TB prevalence urges the consideration of potentially fatal tuberculous myocarditis. Myocarditis is one of rare cardiac involvement in TB infection. With its vast variability of clinical manifestations from asymptomatic to sudden cardiac death (SCD), TB myocarditis is very difficult to study. Histopathological examination is needed to confirm the diagnosis, which is usually being made at autopsy.

Methods: Three recognised histopathological types of myocardial involvement in TB are nodular tubercles with central caseation (tuberculoma), miliary tubercles as a complication of systemic miliary dissemination which spread hematogenously, and diffuse infiltration by granulomas containing Langhans giant cells, epitheloids and lymphocytes. The exact pathological mechanism of TB myocarditis remains unclear. Infiltration to myocardium may occur by hematogenous seeding, direct invasion from pericardium, or retrograde lymphatic spread via mediastinal lymph nodes. Clinical manifestations of TB myocarditis varies from clinically asymptomatic with post-mortem examination as the base of diagnosis; conduction failure such as long QT syndrome or atrioventricular block due to extensive disease surrounding conducting tissue; intractable arrhythmias including atrial fibrillation, paroxysmal ventricular tachycardia, ventricular fibrillation; valve dysfunction, obstruction of the superior vena cava, right ventricular outflow tract, or pulmonary veins due to obstructive lesions caused by large nodular tubercles; congestive heart failure; even SCD. Definitive diagnosis can only be made by endomyocardial biopsy with proof of TB infection in myocardium such as by PCR, culture, or staining of acid-fast bacilli of myocardial tissue sample supported by suggestive radiological imaging such as serial late gadolinium enhanced MRI.

Result: Due to the limitation of ante-mortem case findings, the mechanism of SCD is very difficult to be determined. Hypothesised mechanisms include ventricular arrhythmia due to granulomatous proliferation in the interventricular septum structures, impaired myocardial contractility, cardiac rupture, coronary occlusion, obstruction to pulmonary blood flow leading to fatal haemorrhage, and cardiopulmonary collapse leading to bradycardia. Risk stratification and interventions with implantable cardiac defibrillator are extremely difficult as risk factors such as family history are absent and patients are usually asymptomatic in TB myocarditis. Anti-tuberculosis chemotherapy is effective in improving clinical conditions and shorten hospital stay for patient with TB myocarditis, but it has no benefit in the prevention of SCD.

Conclusion: Therefore, greater awareness concerning TB myocarditis with early and prompt diagnosis should be raised to improve the outcomes and prevent SCD for this rare cardiac involvement in TB infection.