



## Acute lymphocytic myocarditis

Maedeh Ganji<sup>1</sup>, Jose Ruiz-Morales<sup>2,\*</sup>, Saif Ibrahim<sup>3</sup>

<sup>1</sup>Cardiology Department, University of Florida, Jacksonville, FL, USA

<sup>2</sup>Internal Medicine Department, University of Florida, Jacksonville, FL, USA

<sup>3</sup>Interventional Cardiology Department, University of Alabama-Birmingham, Birmingham, AL, USA

*J Geriatr Cardiol* 2018; 15: 517–518. doi:10.11909/j.issn.1671-5411.2018.07.009

**Keywords:** Cardiomyopathy; Myocarditis; Heart attack; STEMI; Sudden cardiac death

Myocarditis is an inflammatory disease of the myocardium. The clinical presentation of myocarditis may range from subclinical to sudden death. The incidence of fatal myocarditis, which often presents with sudden or rapid death, has been estimated at 0.15/100.000 in the general population and is highest in infants and young adults (but may affect any age group). However, diffuse myocarditis in autopsies of sudden death is < 2% in adult. Myocarditis usually presents with heart failure symptoms over a few days to weeks. The classic presentation of viral myocarditis includes a viral prodrome with fever, myalgia, and upper respiratory symptoms. Patients present with dyspnea, chest pain, and arrhythmias. ECG abnormalities are often present, along with evidence of myocardial damage with elevated troponin levels. We present the case of a 78-year old white male who died from acute cardiac decompensation from diffuse acute lymphocytic myocarditis.

A 78-year old white male with a medical history of hypertension and asbestosis who presented to the emergency department with agitation and wide complex tachycardia after three nights of difficulty sleeping, shortness of breath and diaphoresis. Patient required emergent intubation for hypoxic respiratory failure. An ECG performed at the time of admission was read as possible ST-segment elevation myocardial infarction (STEMI) and the patient was taken to the cardiac catheterization lab. The procedure found open and patent coronary vasculature within the exception of occlusion of a branch of the second obtuse marginal artery branching from the left circumflex artery which was treated with a Plain Old Balloon Angioplasty (POBA). During the same day, patient went into cardiac arrest and deceased.

An autopsy, the major pathological findings that explain the patient's terminal course were in the heart which was affected by extensive diffuse myocarditis, predominantly

lymphocytic with associated myocyte necrosis and dilated cardiomyopathy. The inflammation is more marked in the left than the right ventricle and is also involving the papillary muscles, endocardium and focally the epicardium. Scarring with dystrophic calcification in addition to the inflammation is present at the atrioventricular-septal junction (Atrioventricular node site). Inflammation is also focally present at the junction of right atrium and superior vena cava (Sinoatrial node site). Myocardial scar tissue from remote infarction or chronic ischemia is present in the inner lateral wall of the left ventricle.

Other findings in the cardiovascular system include moderate calcific arteriosclerosis of aorta and aortic valve with mild coronary arteriosclerosis. The coronary arteries thrombosis reported in the provisional gross anatomic evaluation was actually blood clots dissolved during processing for histological examination. Irrelevant to the patient's demise, include dense hyaline plaques in the pleura, diaphragm, ribcage with focal adherence to the lung and subpleural fibrosis consistent with the patient's clinical history asbestosis. No evidence of mesothelioma is present. Congested viscera, diverticulosis and small cortical infarct (1cm) in right kidney are present.

Myocarditis may be idiopathic or caused by viral, bacterial or fungal infections, immune disturbances (autoimmunity or hypersensitivity) or a combination of infections trigger by secondary autoimmunity. The identification of underlying organism is not usually possible or effective because organisms, particularly the virus, is often cleared by the immune system before significant inflammation occurs.

Clinical subsets of myocarditis include: (1) fulminant myocarditis associated with fever; (2) acute myocarditis without fever, self-limited or associated with acute onset heart failure; (3) chronic active myocarditis characterized by development of fibrosis and giant cells; (4) chronic persistent myocarditis characterized by persistent myocyte necrosis.

\*Correspondence to: joseruiz\_21@hotmail.com

The differential diagnosis of the underlying cause of myocarditis can be categorized according to the predominant pattern in the inflammation as follows: (1) lymphocytic myocarditis (as in this case) is the most common form of myocarditis and is associated with viral/postviral infection, autoimmune/connective tissue disease or idiopathic; (2) neutrophils rich and microabscesses are mostly bacterial or fungal myocarditis; (3) eosinophils predominant myocarditis is more consistent with underlying allergy/hypersensitivity to drugs, parasitic, hypereosinophilic syndrome or sometimes idiopathic; (4) giant cells and granulomatous myocarditis are most likely from auto-immunity, mycobacterial or fungal infection or sarcoidosis; (5) paucity of inflammatory cells occurs in toxic myocarditis/catecholamine induced myocardial injury.

In this case, the predominance of lymphocytic infiltrate in myocarditis without neutrophil microabscesses, prominent eosinophils, giant cells or granuloma is most consistent with the differential etiology of viral/postviral infection, autoimmune/connective tissue disease or idiopathic.

## References

- 1 Fabre A, Sheppard MN. Sudden adult death syndrome and other non-ischaemic causes of sudden cardiac death. *Heart* 2006; 92: 316–320.
- 2 Friedrich MG, Strohm O, Schulz-Menger J, *et al.* Contrast media-enhanced magnetic resonance imaging visualizes myocardial changes in the course of viral myocarditis. *Circulation* 1998; 97: 1802–1809.
- 3 Ginsberg F, Parillo JE. Fulminant myocarditis. *Crit Care Clin* 2013; 29; 465–483.
- 4 Hunt SA, Abraham WT, Chin MH, *et al.* ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure). *Circulation* 2005; 112: 1825–1852.
- 5 Kuhl U, Seeberg B, Schultheiss HP, *et al.* Immunohistological characterization of infiltrating lymphocytes in biopsies of patients with clinically suspected dilated cardiomyopathy. *Eur Heart J* 1994; 15: 62–67.