Case Report

Myocardial rupture due to viral myocarditis — a case report

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Cardiac tamponade resulting from cardiac rupture causes sudden death. Cardiac rupture occurring secondary to myocarditis is a rare event. Myocarditis is an inflammatory disease of the myocardium; the diagnosis is often made at autopsy. We report a case of sudden death due to cardiac tamponade from rupture of heart due to fulminant myocarditis. Histological findings at the rupture site revealed myocyte necrosis and lymphocyte infiltrate, suggesting viral myocarditis. Present report highlights the role of histopathology during autopsy.

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Key words **: Sudden death, Cardiac tamponade, Viral myocarditis, Histology, Cardiac rupture.**

Sudden death is a death occurring in a person, not known to have been suffering from any dangerous disease, injury or poisoning, within 24 hours of the onset of terminal illness. Cardiovascular diseases are the commonest causes of sudden death followed by respiratory system diseases and central nervous system diseases.

Myocarditis, one of the causes of sudden death is defined as "a process characterized by an inflammatory infiltrate of the myocardium with necrosis and/or degeneration of adjacent myocytes, not typical of ischemic damage associated with coronary artery disease"[1]. It presents with a wide range of clinical features. In asymptomatic cases, the diagnosis is often made at postmortem. The necrosed muscle can give way for spontaneous cardiac rupture, leading to haemopericardium and cardiac tamponade.

Case Report

A 55 year old male social worker collapsed at a public forum while delivering a speech. He was brought to the hospital within half an hour of the incident and was declared brought dead. The body was sent for postmortem examination. On enquiry from the relatives of the deceased, it was found that he did not have previous history of any kind of illness and was not on any medications.

The deceased was a moderately built and nourished male, measuring 166 cm in length and weighing 65 kg. No visible external injuries were found over the body. On opening the pericardial cavity, 280 ml of blood and blood clots were seen (Fig 1). There was a rupture of the myocardium, measuring about 1.5x0.5xcavity depth over posterior surface of left ventricle (Fig 2). There was no gross evidence suggestive of old healed or recent myocardial infarction. Both the coronaries showed mild atherosclerotic changes with no significant blockade. The valves and heart chambers were normal. Other organs were unremarkable on gross examination.

Microscopic examination of heart revealed extensive lytic necrosis of myocardial fibres at the rupture site (Fig 3). Lipofuschin laden macrophages, with epicardial fat necrosis and subendocardial histiocytic infiltration with pericapillary lymphocytes were seen (Fig 4). In sections from posterior and left ventricular wall, occasional lymphohistiocytic infiltration was noted.

Chemical analysis report was negative for any toxic substances.

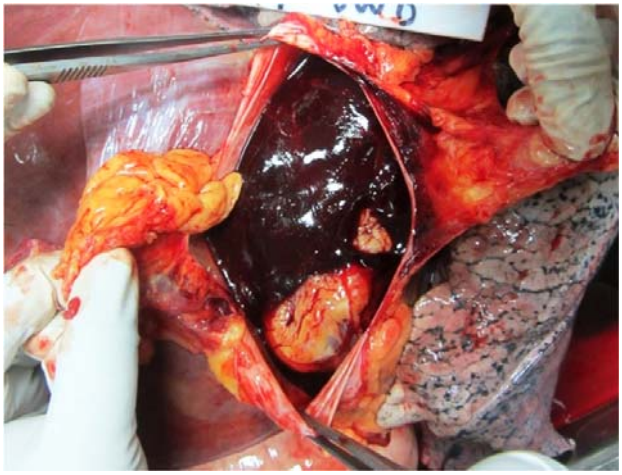

Fig 1 — 280 ml of blood mixed with clots present in pericardial cavity

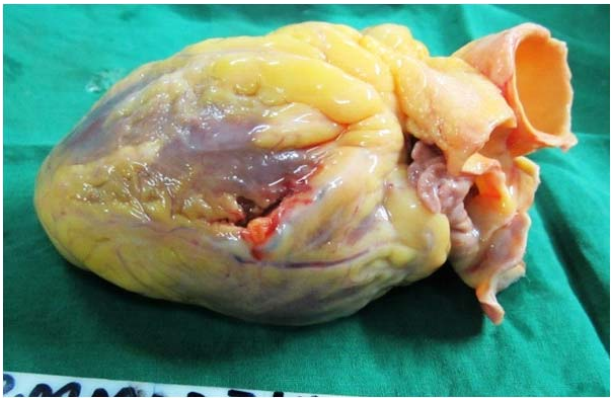

Fig 2 — Myocardial rupture over posterior surface of left ventricle 1.5X0.5cm X Cavity depth

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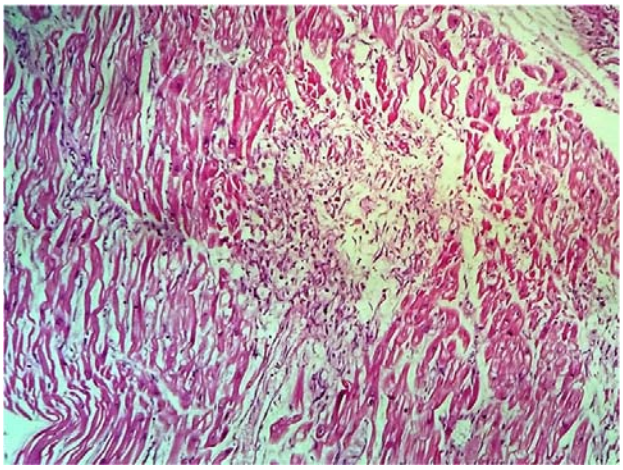

Fig 3 — Lytic necrosis of myocardial fibres at rupture site (H&E-5X)

On perusal of chemical analysis report, histopathological examination and postmortem findings, cause of death was opined as Cardiac tamponade following left ventricular rupture secondary to fulminant myocarditis.

Discussion

The accumulation of fluid in the pericardium in an amount sufficient to cause serious obstruction to the inflow of blood to the ventricles and therefore reduced cardiac output, results in cardiac tamponade. The quantity of the pericardial fluid may be as small as 200 ml when the fluid develops rapidly or more than 2000 ml in slowly developing effusions[2].

Cardiac tamponade due to cardiac rupture is a common complication, resulting from the mechanical weakening that occurs in necrotic and inflamed myocardium resulting in decrease in the cardiac output and circulatory collapse. Acute free wall ruptures are usually rapidly fatal[3]. It is commonly seen in myocardial infarction[4]. However, some cases have also reported cardiac rupture associated with myocarditis[5,6].

The prevalence of acute myocarditis is unknown because most cases are not recognized on account of non-specific or no symptoms (but sudden death may occur)[7]. The diagnosis is often made at postmortem, where no clinical evidence of myocardial failure has been present[8]. Studies report that frequency of myocarditis range from 0.11 to 5.55% in the general population[8,9]. Myocarditis encompasses a diverse group of clinical entities in which infectious agents and/or inflammatory processes primarily target the myocardium[3]. Among the infectious agents, viruses have been considered as important cause of myocarditis[1,10], with Coxsackieviruses A and B accounting for a majority of cases[3], followed by Adenovirus, Cytomegalovirus, Epstein-Barr virus & influenza virus. Other infectious causes are bacterial, parasitic and fungal which are rare. The non-infectious causes include autoimmune disorders and exposure to toxic agents.

Viral myocarditis manifests as a sequence of three phases. In the first phase, there is direct destruction of the cardiomyocytes viruses, causing degradation of the cell structures, which in turn facilitates entry of the virus into the cells leading to further myocyte injury and cardiac dilatation. This phase often goes unnoticed because the further damage is prevented by innate immune response. The second phase occurs due to immune dysregulation caused by the

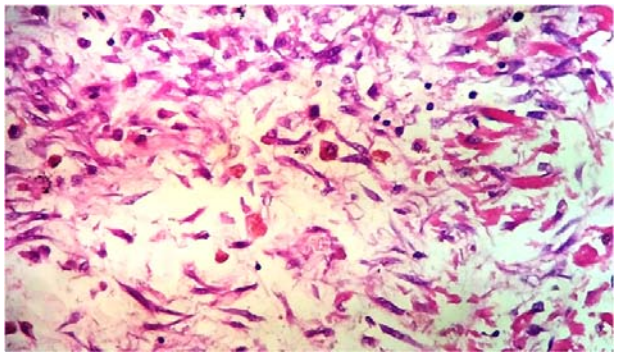

Fig 4 — Lipofuschin laden macrophages, histiocytes, lymphocytes with epicardial fat necrosis (H&E-20X)

epitopes shared between the cardiac and the viral antigens. In the third phase, chronic myocarditis or dilated cardiomyopathy develops due to extensive myocardial injury[11].

Myocardium is weakened and more vulnerable to rupture in 1st phase of acute myocarditis[11], which happened in the present case.

Symptoms of acute myocarditis are vague in many patients; often starting with flu-like symptoms, either of the upper respiratory or gastrointestinal tracts, before any cardiac symptoms appear. The prognosis in acute myocarditis is generally good because left ventricular function improves in most cases[5].

Endomyocardial biopsy is the diagnostic tool in clinically suspicious cases of myocarditis. The histopathologic diagnosis of myocarditis as specified by the Dallas' criteria requires an inflammatory infiltrate and associated myocyte necrosis and damage not characteristic of an ischaemic event. It commonly occurs in lateral wall of left ventricle.[1]

In cases of virus induced inflammatory alterations of the myocardium, the infiltrates are predominantly lymphocytes and macrophages[12,13].

Viral serology, cultures and even DNA hybridization techniques almost fail to detect a virus, especially during the early phase[14]; they are of little assistance in establishing a diagnosis. For the Forensic pathologist, the diagnosis of viral myocarditis rests in the histologic appearance of lymphocytic inflammatory cell infiltrate with myocyte necrosis[15].

This case report highlights the importance of histological analysis during autopsies, so as to help families come to terms with death in a previously healthy relative. Immunohistochemistry and molecular biological techniques can be used for confirmation of viral myocarditis.

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