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Case Report

An autopsy case of cardiac tamponade caused by a ruptured ventricular aneurysm associated with acute myocarditis

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ABSTRACT

We report an autopsy case of hemopericardium caused by rupture of a ventricular aneurysm associated with acute myocarditis in an infant boy aged 2 years and 10 months. Three days before his death, the patient developed fever. On the day of death, he described an urge to defecate and attempted to do so in an upright position. While straining to defecate without success for a prolonged period, he stopped breathing and collapsed. On autopsy, his heart weighed 91.7 g and cardiac tamponade was evident, the pericardial cavity being filled with 140 mL of blood that had come from a 1.5-cm-long rupture in a 2.7 × 1.5 cm ventricular aneurysm in the posterior left ventricular wall. Patchy grayish-white discoloration was noted in the myocardium. Histologically, CD3-positive T lymphocytic infiltration accompanied by pronounced macrophage infiltration was observed in the myocardium. Hemorrhagic necrosis was detected in the area of the ventricular aneurysm. Staining for matrix metalloproteinase (MMP) expression revealed abundant MMP-2, MMP-7, and MMP-9. Polymerase chain reaction to detect viruses failed to identify any specific causative viruses in the myocardium. In this case of lymphocytic (viral) and histiocytic myocarditis with pronounced macrophage infiltration and upregulation of MMP expression, myocardial remodeling and associated wall weakening had resulted in formation and rupture of an aneurysm.

1. Introduction

Myocarditis 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 has a wide range of highly variable clinical presentations; thus, the diagnosis is frequently made at autopsy. Autopsy studies report a frequency of myocarditis ranging from 0.11% to 5.55% in the general population [2].

Spontaneous cardiac rupture leading to cardiac tamponade is one of the fatal outcomes of fulminant myocarditis. Spontaneous cardiac rupture is rare, most reported cases being caused by myocardial infarction. Other even rarer causes include myocarditis, mediastinitis, myocardial abscess, angiosarcoma of the heart, Chagas disease, and post-mitral valve replacement surgery. Cardiac tamponade following rupture of the heart occurs very rapidly, resulting in a fatal fall in cardiac output and consequent circulatory collapse. The interval between rupture and collapse is variable, but is usually short [1].

Rupture of the myocardium is classified into the following three types: (i) acute rupture, in which the patient characteristically dies within minutes, precluding hospitalization; (ii) small rupture, for which corrective surgery may be possible provided appropriate interventions occur within a few hours and adequate hemodynamic support is provided; and (iii) chronic rupture, which is associated with formation of a false aneurysm [3,4].

Cardiac tamponade, also termed hemopericardium, is a clinical syndrome caused by accumulation of fluid in the pericardial space, resulting in reduced ventricular filling and subsequent hemodynamic compromise. Rapid accumulation of as little as 200–300 mL of blood can result in a marked increase in pericardial pressure and severely impede cardiac output; rapid accumulation of more than 400 mL can very quickly cause sudden death [5].

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An aneurysm, a localized abnormal dilatation of a blood vessel or the heart, can be congenital or acquired [6]. Ventricular aneurysm formation and rupture is an infrequent complication of acute myocarditis; several case reports of subjects with acute myocarditis and associated left ventricular free wall rupture have been published [1,5,7–10]. However, ventricular aneurysmal changes have not been clearly described in these case reports.

In the present report, we describe a case of cardiac tamponade caused by a ruptured ventricular aneurysm associated with acute myocarditis.

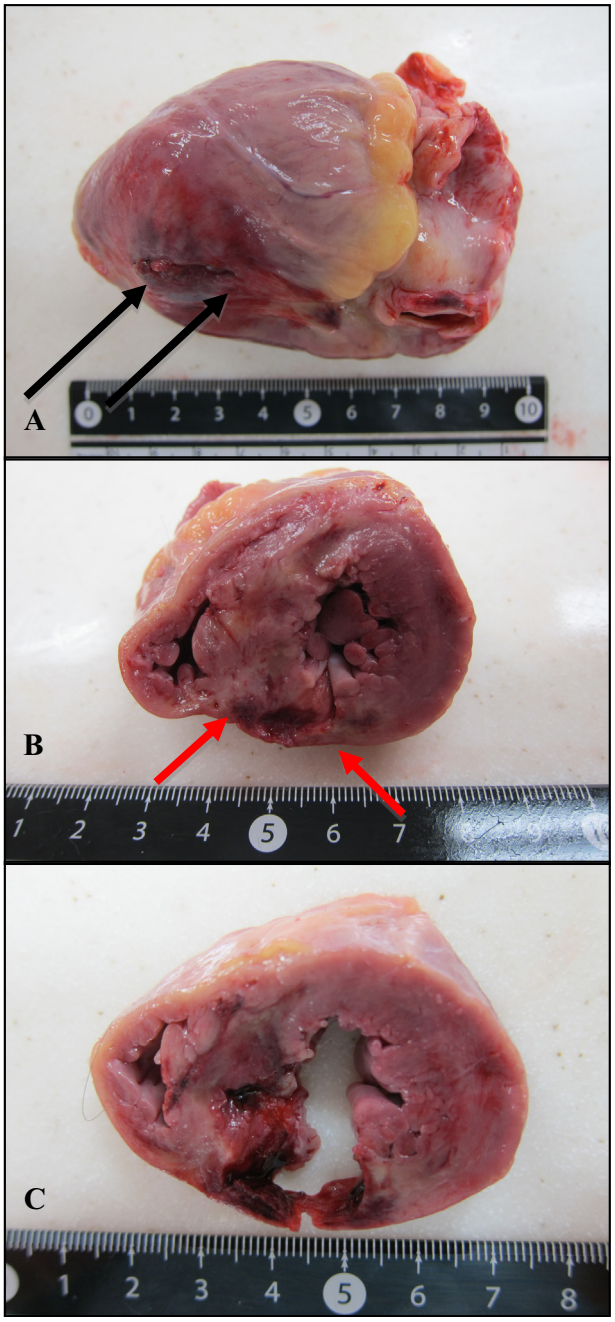

Fig. 1. Macroscopic findings. A and B: A 2.7 × 1.5 cm ventricular aneurysm (red arrows) has formed in the posterior left ventricular wall, accompanied by a 1.5-cm-long rupture (black arrows). C: Patchy grayish-white discoloration and hemorrhagic necrosis is evident in the myocardium from the interventricular septum to the posterior wall.

2. Case report

The case subject was an infant boy aged 2 years and 10 months. Three days before his death, the patient developed fever and was brought to the hospital for assessment, after which he was sent home. The next morning he expressed an urge to defecate and attempted unsuccessfully, despite considerable straining, to do so in an upright position. His breathing became irregular and eventually ceased. He collapsed and an ambulance was called. On arrival at the emergency department, the boy was in a state of cardiopulmonary arrest and could not be resuscitated despite immediate medical intervention. An administrative autopsy was performed on the same day.

2.1. Autopsy findings

The subject was 96 cm in height and weighed approximately 15 kg. No findings suggestive of trauma were detected. The heart weighed 91.7 g and the pericardial cavity was filled with 140 mL of blood mixed with soft blood clots, resulting in an autopsy diagnosis of cardiac tamponade. The blood had come from a 1.5-cm-long rupture in a 2.7 × 1.5 ventricular aneurysm that had formed in the posterior left ventricular wall (Fig. 1). Patchy grayish-white discoloration was noted in the myocardium from

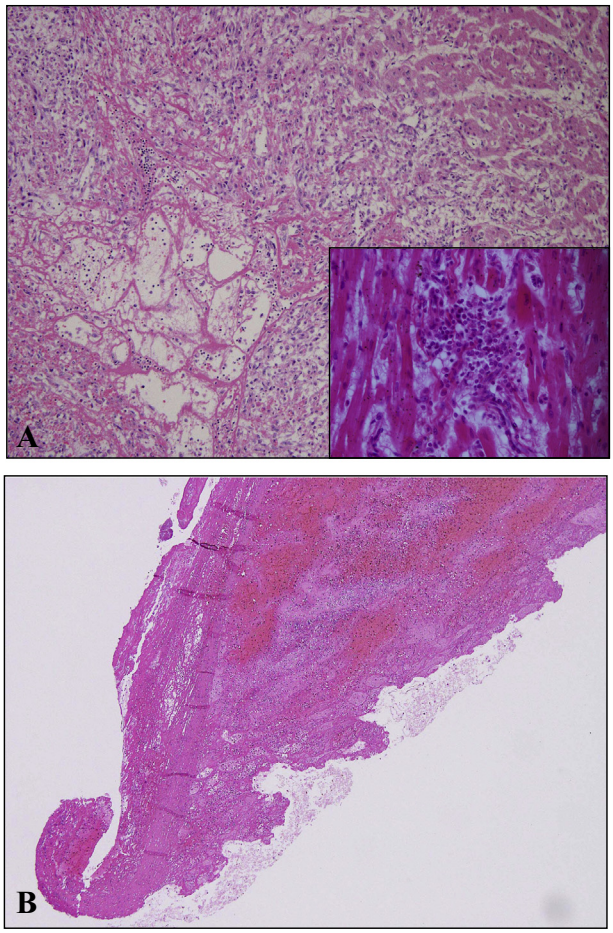

Fig. 2. Histological assessment of the myocardium. A: Lymphocyte infiltration is evident in the myocardium, indicating lymphocytic myocarditis. Pronounced macrophage infiltration is also evident. Inset: Lesion in which lymphocytic infiltration is predominant. B: Hemorrhagic necrosis is present at the rupture site in the ventricular aneurysm. Hematoxylin and eosin stain. (A) 100× and (B) 40×.