



Left Ventricular Aneurysm in a Four-Year-Old Child: A Diagnostic Challenge

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Abstract

We describe spontaneous rupture of a congenital left ventricular (LV) aneurysm with subsequent tamponade and cardiac arrest in a 4-year-old male with staphylococcal septicemia. Emergency resuscitation, thoracotomy, and oversewing were successfully undertaken in the pediatric intensive care unit. There was complete cardiovascular recovery without adverse neurodevelopmental sequelae. This article details the difficulties in determining the etiology of ventricular aneurysms but highlights the importance of attempting to do so, particularly in distinguishing between congenital and acquired forms. Congenital aneurysms are usually a stable pathology; mycotic aneurysms are not and should be managed emergently, as survival after rupture is rare.

Keywords

left ventricle, congenital, aneurysm, cardiac arrest, ECMO

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Case Report

A previously healthy 4-year-old male was transferred from the Cook Islands to a tertiary children's hospital in Auckland, New Zealand, for treatment of staphylococcal septicemia. For a month, he had been managed with antibiotic treatment of tibial osteomyelitis involving the left knee and extensive cavitating pneumonia. Both arthroscopic washings and peripheral blood cultures were positive for *Staphylococcus aureus*.

Transthoracic echocardiography (TTE) revealed a large aneurysm arising from the left ventricle (LV; Figure 1). A computed tomogram (CT) was requested to identify whether there was involvement of the coronaries and this demonstrated LV dilatation with normal function. There was a 59-mm diameter thin-walled aneurysm, with paradoxical motion, arising from the diagonal surface of the LV cavity, with a 6.1 mm communication (Figure 2). This was separated from the left anterior descending and major diagonal coronary arteries. There was no evidence of intraluminal debris or thrombus and no pericardial abnormality apart from a very small simple effusion. Motion and contrast enhancement of the myocardium directly adjacent to the neck of the aneurysm was normal.

Lack of radiological evidence of local inflammation suggested a congenital ventricular aneurysm rather than an acute mycotic or false aneurysm. However, it was recognized that

coinfection of a preexisting congenital lesion could not be excluded.

The LV dilatation indicated significant volume overload due to the aneurysm. Despite the concerns of rupture, the child was not taken immediately for surgery due to the severity of his pneumonia. It was decided that his condition be optimized in the pediatric intensive care unit prior to surgical repair (PICU).

On day 2, after admission to PICU, there was an acute deterioration with profound bradycardia and hypotension and subsequent cardiac arrest. Cardiac tamponade was suspected and an emergency thoracotomy performed in the PICU. Extensive blood was evacuated from the pericardial space, a hole in the LV aneurysm was identified, and hemorrhage was controlled with digital compression of the aneurysm sac against the

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Abbreviations and Acronyms

CT	computed tomogram
ECMO	extracorporeal membrane oxygenation
LV	left ventricular
PICU	Paediatric Intensive Care Unit
TTE	transthoracic echocardiography

sternum while simultaneously applying internal cardiac massage. A second surgeon instituted cardiopulmonary bypass via a midline sternotomy. The communication between the aneurysm and the left ventricular cavity was oversewn directly. The sac was observed to be thin walled, with minimal inflammation consistent with a congenital aneurysm, as opposed to a mycotic or false aneurysm. The aneurysm was not resected and no specimen could be sent for histopathology.

Total duration of cardiac arrest was approximately 100 minutes and ventricular function appeared poor before going on with bypass. The patient was electively placed on extracorporeal membrane oxygenation (ECMO) via an open sternum, and moderate hypothermia (34°C) was maintained for 48 hours to enhance neuroprotection.

The patient was separated from ECMO uneventfully on day 3 and chest closure was delayed until day 5 because of tissue edema. He was extubated onto BiPAP (bi-level positive airway pressure) on day 7 but required a prolonged period of noninvasive ventilatory support as a result of extensive cavitating staphylococcal pneumonia. He spent a total of 56 days in the PICU as a result of pulmonary disease, before being transferred to the ward. Review after discharge by the pediatric developmental service revealed normal fine and gross motor skills, age-appropriate intellect, problem-solving capabilities, behavior, and social skills. The TTE at day 90 demonstrated normal left ventricular size and function.

Discussion

Congenital ventricular diverticula and aneurysms are rare in children and present considerable diagnostic problems. Etiology has, however, important implications for management.¹

False aneurysms have a high rate of rupture, whereas true and congenital aneurysms do not and may usually be managed expectantly. Congenital ventricular aneurysms associated with complications such as arrhythmia and cardiac failure suggest instability and are best treated surgically.¹ Mycotic aneurysms have a high rate of rupture, and surgery should also be performed early.^{2,3} Overall, unstable pathologies have a high risk of rupture and survival from this is rare.⁴

Histopathological examination reveals congenital ventricular diverticula to be characterized by synchronous contractility within the 3 myocardial layers.⁵ Congenital ventricular aneurysms are characterized by fibrotic tissue with akinesis or paradoxical systolic motion of the aneurysm sac and a variable size connection to the ventricle.^{5,6} Coexisting heart or midline thoracoabdominal defects are absent.^{1,6} The prognosis for children

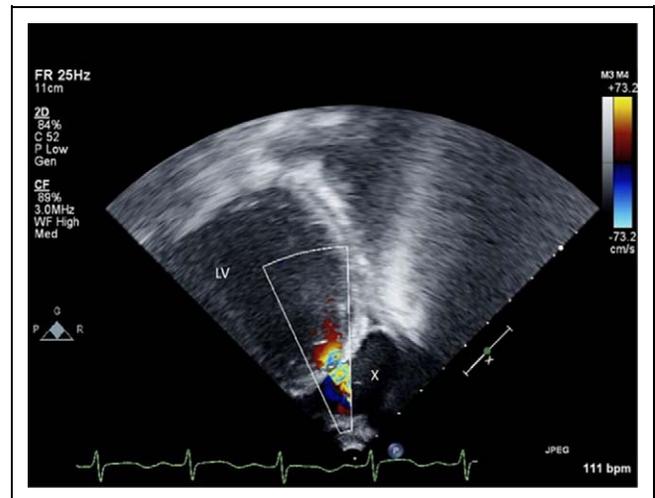


Figure 1. Echocardiography illustrating flow of blood from the left ventricle (LV) into the aneurysm (X) via a narrow communication.



Figure 2. Volume-rendered three dimensional study of the aneurysm arising from the left ventricle (LV) apex, clearly separate from the left anterior descending and major diagonal coronary arteries.

with such lesions is variable. A case series of 18 children undertaken by Hamaoka et al observed only 1 child requiring surgical intervention for intractable arrhythmias, and all others having no detectable progression over time.

In this case, the absence of markers of inflammation on imaging was consistent with a congenital aneurysm. Congenital etiology was also suggested by normal motion and contrast enhancement of the myocardium at the entrance and the thick-walled nature of the aneurysm. However, a mycotic aneurysm or secondary infection of a congenital aneurysm could not be excluded. Unfortunately, the aneurysm ruptured before it could be repaired, the cause of which is unknown. It is possible that the increased cardiac output secondary to severe sepsis may have

increased aneurysmal shearing forces and caused rapid distension and enlargement of the aneurysm sac. Secondary infection may also have weakened the wall of the aneurysm.

Beare et al reported the first case of a false LV aneurysm³ in 1967. The aneurysm occurred in a child with osteomyelitis of the femur. The aneurysm was considered mycotic as it appeared in an area of previously normal myocardium with 3 small communications to the LV cavity. The aneurysm was repaired during a period of clinical stability. Pathological examination revealed no inflammatory tissue or cardiac muscle. Children rarely survive rupture of an LV aneurysm completely unscathed. Westaby et al reported the case of a 15-year-old girl with staphylococcal endocarditis who developed a mycotic aneurysm after emergency mitral valve repair. The aneurysm subsequently ruptured causing tamponade.⁷ This aneurysm was not considered reparable due to the involvement of the circumflex artery and was managed expectantly using a left ventricular assist device to allow LV unloading and scar tissue development.⁸ Cardiac function remained suboptimal after this episode but neurological function was not documented in the report.

In summary, this case report illustrates the difficulty in timing the surgical intervention when an aneurysm is detected in the context of severe systemic infection. Elucidating the etiology of the aneurysm is not easy but, in this case, the radiological diagnosis suggesting congenital etiology possibly led to a false sense of security. Any sign of cardiovascular instability warrants cardiosurgical intervention planning. Radiological evidence of mycoses, such as abnormal contrast enhancement, should expedite surgical management. A diagnosis of mycotic aneurysm would have been an indication for urgent surgical intervention despite the respiratory infection. We believe that the most likely etiology of the lesion was congenital and that the combination of severe sepsis and LV aneurysm (of any cause) lowers the threshold for immediate surgical repair.

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Declaration of Conflicting Interests

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