Development of a Subaortic Aneurysm Secondary to Disseminated Tuberculosis in a Child

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Subvalvular aneurysms of the left ventricle are very rare and often the cause is uncertain. Most of the cases of subvalvular left ventricular aneurysms described in the literature are due to congenital weakness of the fibromuscular annuli. We describe a unique case of a child with a tuberculous subaortic aneurysm observed at different stages of development by serial transthoracic echocardiography. The patient underwent successful cardiac surgery after the initial conservative treatment for tuberculosis. (Ann Thorac Surg 2010;90:644–7)

Subvalvular aneurysms of the left ventricle are very rare and often the cause is uncertain. Very little data in the literature describes subvalvular aneurysms in children and most of the data are derived from case reports. We describe a unique case of a human immunodeficiency virus negative child with a tuberculous subaortic aneurysm observed at different stages of development by serial transthoracic echocardiography. The patient underwent successful cardiac surgery after the initial conservative treatment for tuberculosis (TB).

A 3-year-old black boy who was negative for human immunodeficiency virus presented to our institution with a 1-week history of coughing, fever, night sweats, and generalized body swelling. On examination he appeared chronically ill and was underweight for his age. He had generalized lymphadenopathy in the axillary, supraclavicular, cervical, and submandibular regions. He had ascites, hepatomegaly (liver span, 12 cm), and splenomegaly (3 cm below costal margin). The boy was tachypneic with a respiratory rate of 50 breaths/min. He had intercostal and subcostal recession and audible bilateral rhonchi. His heart rate was150 beats/min, with a gallop rhythm present and with muffled heart sounds. There were no murmurs, but he was in congestive cardiac failure. His blood pressure was 80/40 mm Hg.

A chest roentgenogram demonstrated cardiomegaly, a widened mediastinum, and paratracheal lymphadenopathy. The C-reactive protein, lactate dehydrogenase levels, and erythrocyte sedimentation rate were elevated. Echocardiography (Fig 1) revealed normal intracardiac anatomy with a large organized and loculated pericardial effusion. A biopsy of a supraclavicular lymph node demonstrated caseating necrosis with sites of granulomatous inflammation. Epitheloid cell granulomas and Langhans giant cells were also seen. The Ziehl Neelsen stain highlighted occasional acid and alcohol fast bacilli, which confirmed the TB. There were no features of lymphoma or malignancy present. Mycobacterium TB was also isolated on cultures of gastric aspirates. A diagnosis of disseminated TB with organized TB pericarditis was made.

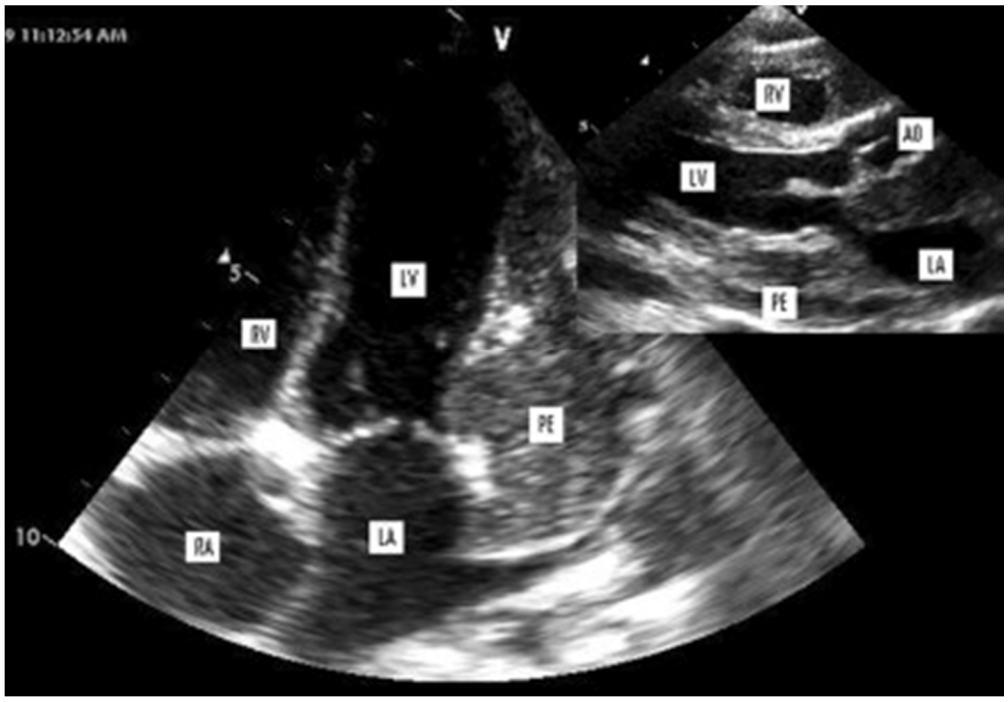

Fig 1. Apical four-chamber view and parasternal long-axis view echocardiogram (inset) demonstrating the organized loculated pericardial effusion. (AO = aorta; LA = left atrium; LV = left ventricle; PE = loculated organized pericardial effusion; RA = right atrium; RV = right ventricle.)

The patient was treated with anti-TB drugs (isoniazid, rifampicin, pyrazinamide, and ethambutol), prednisone, and appropriate cardiac failure therapy. His clinical condition improved with resolution of the generalized edema and ascites and with a decreased size of his lymph nodes. A repeat echocardiogram (Fig 2) after 6 weeks of treatment demonstrated a small pouch (2 mm × 3 mm) in the subaortic area with mild aortic regurgitation. Cardiac surgery was not considered at this stage because of the active TB and the possibility of friable tissue. He was closely monitored while receiving TB treatment, and he remained clinically asymptomatic. Another follow-up echocardiogram (Fig 3) 3 months later demonstrated a large subaortic aneurysm just below the left coronary cusp that measured 30 mm × 24 mm and compressed the left atrium. The neck of the aneurysm measured 5 mm and a color Doppler echocardiogram demonstrated flow in and out of the aneurysm from the left ventricle. The pulsed wave spectral Doppler echocardiogram demonstrated restrictive bidirectional flow into the aneurysm with a flow velocity of 4 m/s. There was no distortion of the aortic valve and only mild aortic regurgitation was evident. These findings were confirmed on transesophageal echocardiography (Fig 4). A left ventriculogram (Fig 5) demonstrated the large aneurysm clearly, which also appeared to be compressing the left coronary artery.

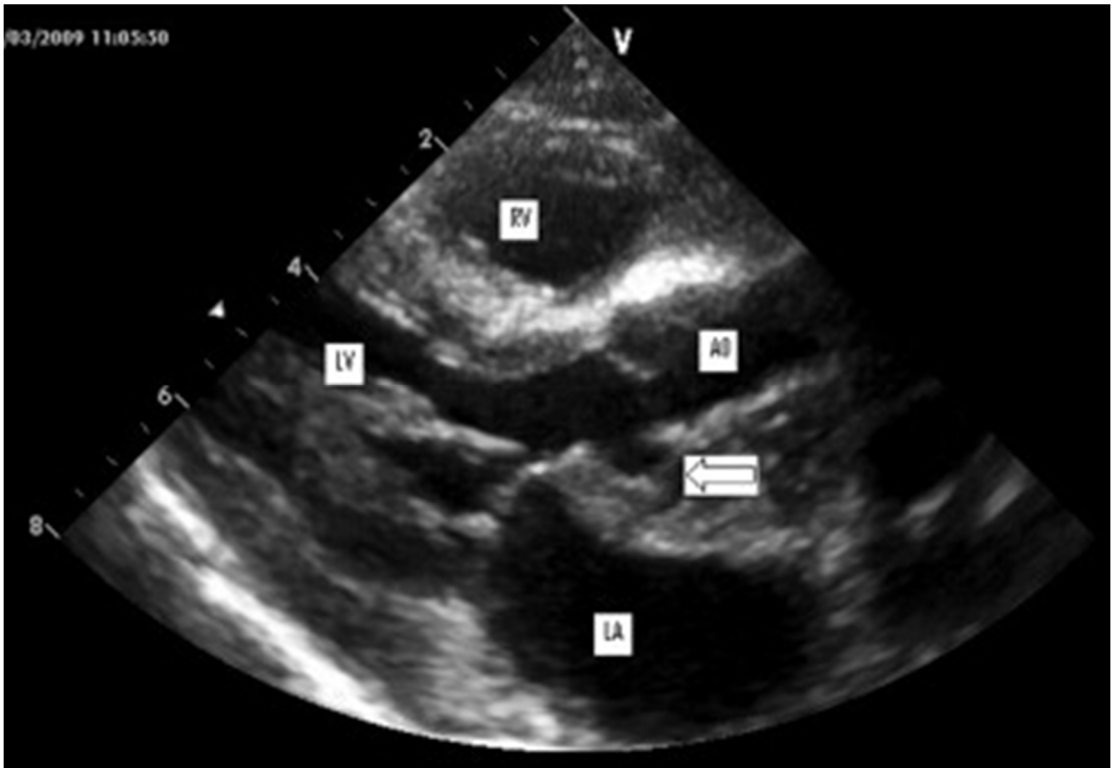

Fig 2. Repeat transthoracic echocardiogram 6 weeks after presentation. Parasternal long axis view demonstrating a small pouch in the subaortic area (arrow). (AO = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.)

The boy underwent successful cardiac surgery 5 months after TB therapy was initiated. There was extensive fibrosis in the thoracic cavity and pericardial space. The aneurysm was approached through an incision in the ascending aorta and through the aortic valve. The neck of the aneurysm was sutured and closed to isolate the aneurysm, and blood was allowed to resorb spontaneously. Due to the extensive fibrosis and the posterior position of the aneurysm, evacuation was deemed too risky. The boy was doing well 4 months after the surgery and had completed the anti-TB therapy.

Comment

Subvalvular left ventricular aneurysms can either be subaortic or submitral. The submitral aneurysms are more common than the subaortic type [1]. Subaortic and submitral aneurysms arise in the fibro-muscular rings of the aortic valve and mitral valve annuli, respectively [2].

Most of the cases of subvalvular left ventricular aneurysms described in the literature are due to congenital weakness of the fibro-muscular annuli. Chesler and colleagues [2] postulate that a dehiscence of the fibro-muscular union will result in aneurysm formation. The majority of these patients are of African ancestry, although similar conditions have been found to a lesser degree

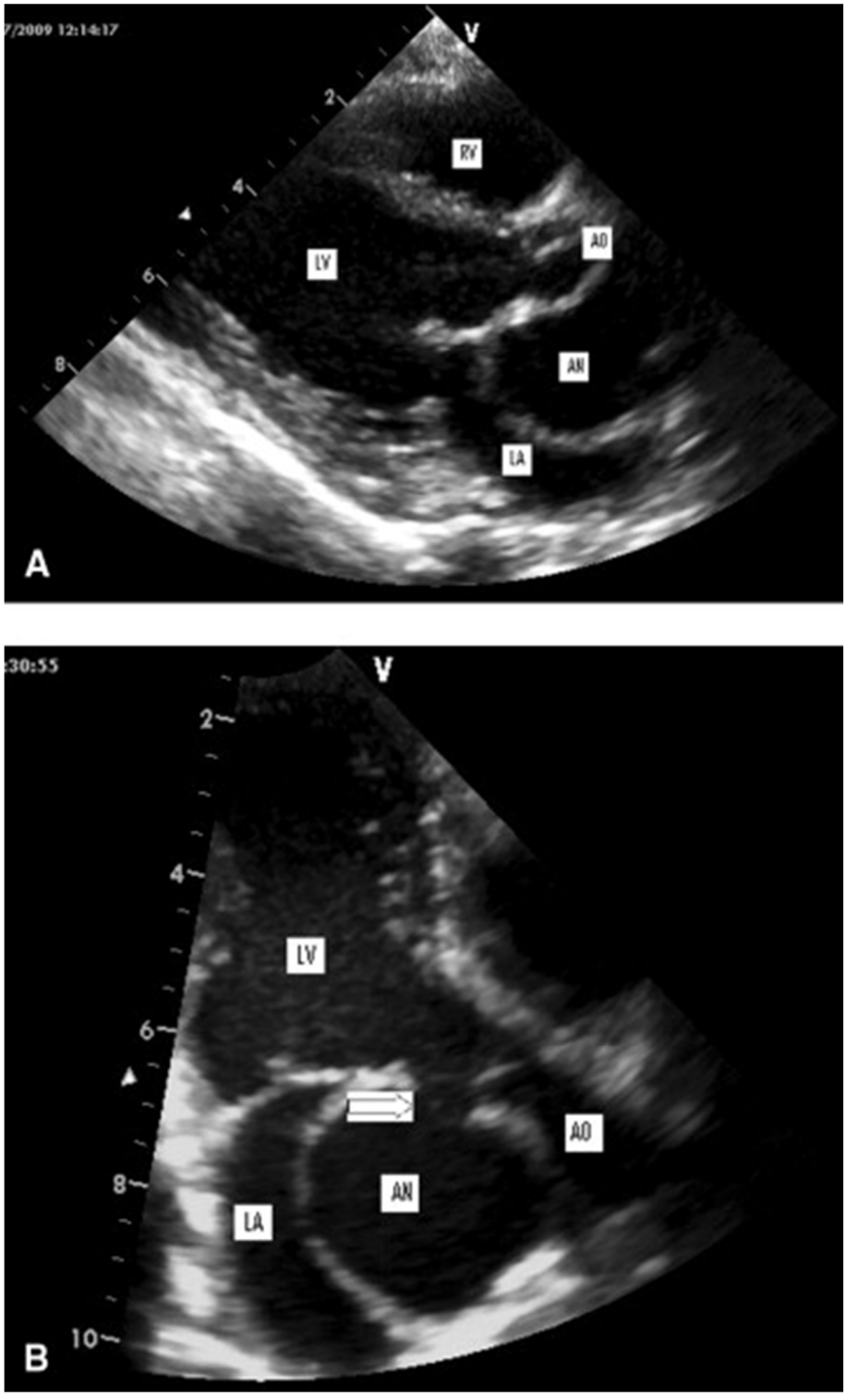

Fig 3. Repeat transthoracic echocardiogram 3 months after presentation. (A) Modified parasternal long-axis view demonstrating a large subaortic aneurysm. (B) Modified apical three-chamber view demonstrating the large subaortic aneurysm compressing the left atrium (LA). The opening of the aneurysm is seen just below the aortic valve (arrow). (AN = aneurysm; AO = aorta; LV = left ventricle; RV = right ventricle.)

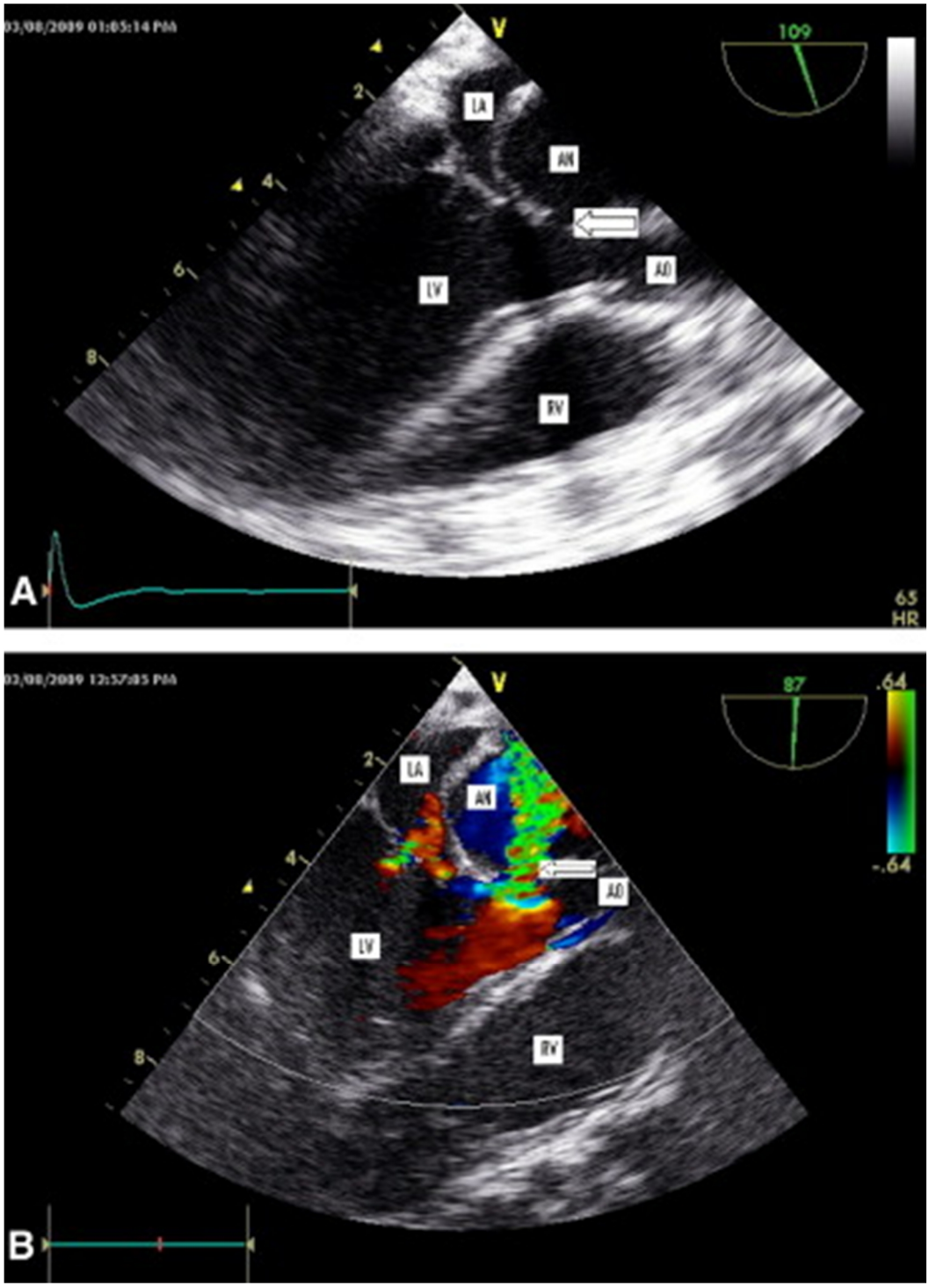

Fig 4. Transesophageal echocardiogram: (A) demonstrating the large subaortic aneurysm compressing the left atrium and narrow neck (arrow). (B) With color Doppler showing the restrictive flow through the neck of the aneurysm (arrow). (AN = aneurysm; AO = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.)

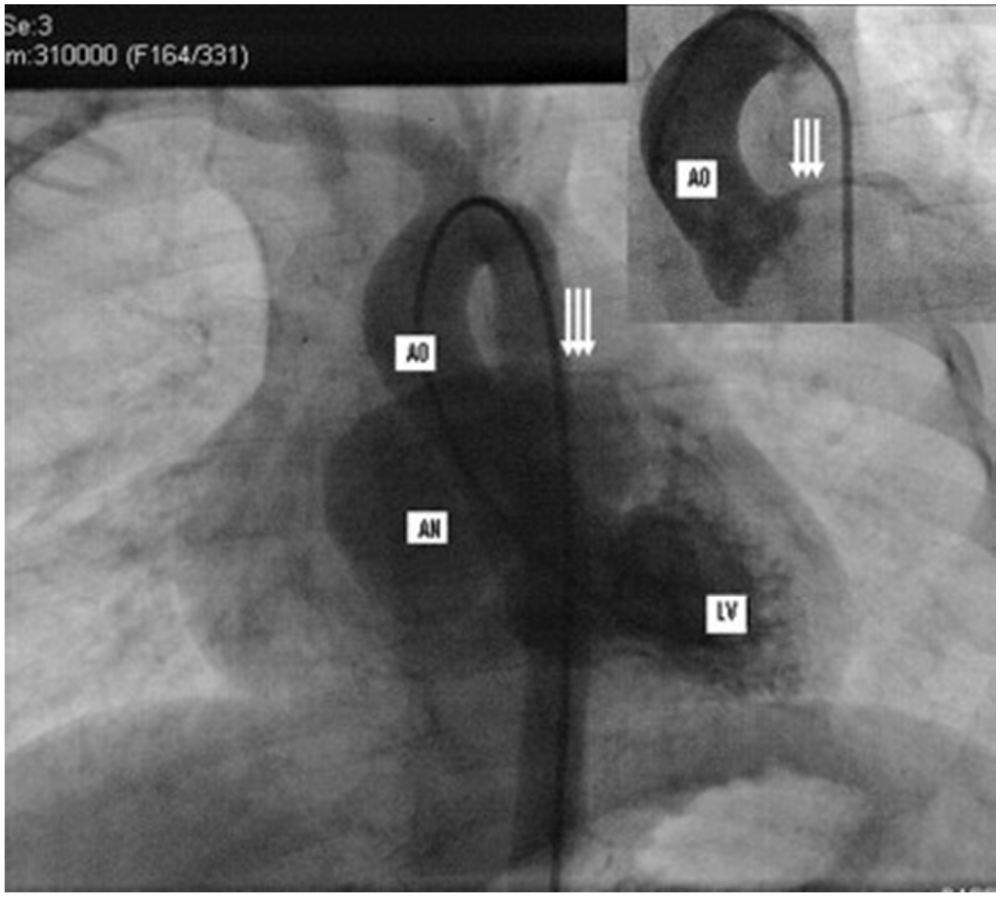

Fig 5. Left ventriculogram and ascending aortogram (inset) demonstrating the large aneurysm compressing on the left coronary artery (arrows). (AO = aorta; AN = aneurysm; LV = left ventricle.)

in other race groups [[1], [3] and [4]]. They have also been described as a complication in patients with infective endocarditis, tuberculosis, and syphilis [[4], [5], [6] and [7]]. Du Toit and colleagues [6] in their series of 12 patients found an association with rheumatic carditis in 2 patients. The causal effect of all these infections has always been speculative, as it may just reflect the high incidence of the infections in endemic areas, rather than a causal mechanism [6].

Our patient had disseminated mycobacterium TB infection with cardiac involvement. There was no aneurysm noted on the initial echocardiogram, thus excluding a congenital origin. The aneurysm was initially identified as a small pouch 6 weeks after presentation. By 4 months the small pouch had grown into a large aneurysm requiring surgical intervention.

We believe this case is the first report in which a congenital cause could be excluded by echocardiogram, and aneurysm development was linked to disseminated TB. We also believe that this is the youngest patient reported with a subaortic aneurysm secondary to TB. Disseminated TB is often associated with acquired immunodeficiency syndrome, but our patient was negative for human immunodeficiency virus and was immunocompetent. There were no vegetations identified on any of the valves to suggest infective endocarditis as the cause.

Subaortic aneurysms must be distinguished from sinus of Valsalva aneurysms. The aortic valve and the sinuses were normal, both on transesophageal echocardiogram and on angiogram, excluding a sinus of Valsalva aneurysm.

Surgical repair is the treatment of choice for large subaortic aneurysms, which are at risk of rupture, calcification, and infective endocarditis if left untreated [8]. Large aneurysms have been reported to compress the coronary arteries, causing myocardial infarction and sudden death [[1] and [2]], or to compress the conduction system, causing heart blocks [9]. The ventriculogram in our patient demonstrated compression of the left coronary artery, although the electrocardiogram did not reveal any ischemic changes.

Transthoracic and transesophageal echocardiography played a pivotal role in identifying, monitoring, and planning surgical intervention in our patient.

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