Case Reports

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Sinus of Valsalva Pseudoaneurysm as a Sequela to Infective Endocarditis

Pseudoaneurysm is an uncommon sequela of infective endocarditis. We treated a 44-yearold man who had an active case of group B streptococcal infective endocarditis of the aortic valve despite no evidence of valvular dysfunction or vegetation on his initial transesophageal echocardiogram. After completing 6 weeks of intravenous antibiotic therapy, the patient developed a sinus of Valsalva pseudoaneurysm and severe aortic regurgitation caused by partial detachment of the left coronary cusp.

We used a pericardial patch to close the pseudoaneurysm and repair the coronary cusp. This case shows the importance of routine clinical follow-up evaluation in infective endocarditis, even after completion of antibiotic therapy. Late sequelae associated with infective endocarditis or its therapy include recurrent infection, heart failure caused by valvular dysfunction (albeit delayed), and antibiotic toxicity such as aminoglycoside-induced nephropathy and vestibular toxicity. (Tex Heart Inst J 2016;43(1):46-8)

nfective endocarditis (IE) is an infection with potentially devastating consequences. The spread of infection from the valvular structures to the surrounding tissue can result in valvular aneurysms, paravalvular abscesses, and the formation of pseudoaneurysms or fistulous tracts. These rare sequelae can occur subacutely, even after completion of intravenous antibiotic therapy. Consequently, routine clinical follow-up evaluations are important for the early detection and treatment of such events in IE patients. We report the case of a patient who presented with an active case of group B streptococcal endocarditis of the aortic valve, in whom a sinus of Valsalva pseudoaneurysm developed after his apparently successful completion of antibiotic therapy.

Case Report

A 44-year-old previously healthy man, admitted to our hospital for fevers, was found to have persistent group B streptococcal bacteremia. His initial transesophageal echocardiogram (TEE) yielded normal results, with no evidence of valvular disease or vegetation. We treated him with a 6-week course of intravenous ceftriaxone for presumed group B streptococcal IE. His follow-up blood cultures were negative for bacteremia.

One week after his completion of antibiotic therapy, the patient reported new-on-set dyspnea upon exertion, and palpitations. A transthoracic echocardiogram (TTE) showed new severe aortic regurgitation, as well as aneurysmal enlargement of the right sinus of Valsalva. Subsequently, a repeat TEE showed no valvular vegetations but did reveal a pseudoaneurysm (0.8×1.2 cm) at the sinus of Valsalva; the right coronary cusp of the aortic valve prolapsed into the pseudoaneurysm, resulting in severe aortic insufficiency (Fig. 1).

When the patient underwent aortic valve repair, he was found to have partial detachment and erosion of the left coronary cusp of the aortic valve. The pseudoaneurysm, which had formed between the left and right coronary cusps, extended into the soft commissural area (Figs. 2 and 3). We used a pericardial patch to close the cavity and repair the coronary cusp. Our patient's postoperative blood and tissue cultures remained negative for IE, and he had an uneventful postoperative recovery.

Discussion

Infective endocarditis is associated with sequelae of multiorgan systems. These include heart failure caused by valvular dysfunction, multiorgan infarctions caused by

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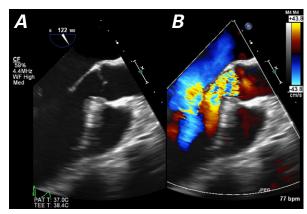


Fig. 1 Transesophageal echocardiograms show severe aortic regurgitation in **A**) 2-dimensional midesophageal long-axis view and **B**) color-Doppler mode.

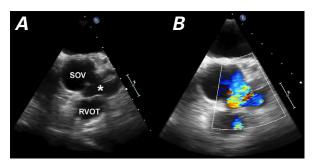


Fig. 2 Transesophageal echocardiograms. A) The 2-dimensional midesophageal short-axis view of the sinus of Valsalva shows the pseudoaneurysm (asterisk). B) Color-Doppler mode shows turbulent flow through the narrowed neck of the pseudoaneurysm (asterisk) that communicates with the aorta.

RVOT = right ventricular outflow tract; SOV = sinus of Valsalva

septic embolization, and infections caused by seeding. On rare occasions, IE can cause pseudoaneurysms of the sinus of Valsalva and the surrounding paravalvular area.¹⁻⁵ Our patient developed a pseudoaneurysm despite his completing 6 weeks of intravenous antibiotic therapy—followed by negative blood cultures and no evidence of valvular insufficiency, vegetation, or paravalvular abscess on the initial TEE.

Acquired pseudoaneurysm of the sinus of Valsalva is often associated with a paravalvular abscess, which is estimated to occur in 28% of IE cases.⁶ Our patient probably first developed a paravalvular abscess, which subsequently formed a communication with the aortic sinus, and then became a pseudoaneurysm. Transesophageal echocardiography is recommended for the early detection of IE and abscesses—except in patients with very small vegetations (<2 mm)—because it has a high sensitivity and specificity of 87% and 94.6%, respectively, versus 28.3% and 98.6% with TTE.^{7,8} Pseudoaneurysms of the sinus of Valsalva most often involve

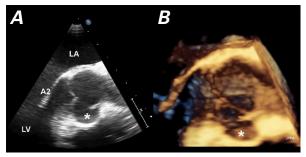


Fig. 3 Transesophageal echocardiograms. A) This 2-dimensional midesophageal long-axis view shows a large pseudoaneurysm (asterisk) arising from between the left and right coronary sinuses of Valsalva. B) This 3-dimensional view shows the pseudoaneurysm (asterisk) and its narrowed neck communicating with the aorta

A2 = mitral valve cusp; LA = left atrium; LV = left ventricle

the right coronary sinus (80%), followed in frequency by the noncoronary sinus (16%), and the left coronary sinus (4%).9 In our patient, the pseudoaneurysm involved both the left and right coronary sinuses. Despite our inability to detect a vegetation by TEE on the aortic valve, our patient developed a pseudoaneurysm and partial detachment of the left coronary cusp. This result indicates that an active infection of the aortic valve and a periannular extension of the valvular infection were probably too small to detect on the initial TEE. We should also note that aortic paravalvular abscesses and pseudoaneurysms involving the sinus of Valsalva rupture in 1.6% of cases, to form aortocavitary fistulae with the adjacent cardiac chamber (atrium or ventricle). These fistulae can cause shunting and hemodynamic deterioration, in addition to the primary valvular insufficiency.¹⁰ Other rare sequelae include the formation of thrombus within the pseudoaneurysm, which can cause peripheral embolization and even occlusion of the coronary artery, thereby requiring urgent revascularization and surgical repair.11

In most patients with IE, medical or surgical therapy (or both) are needed to cure the disease. The American Heart Association recommends routine clinical follow-up evaluation after the initial therapy, given the possibility of late sequelae of IE or its therapy, including recurrent infection, heart failure due to delayed onset of valvular dysfunction, and antibiotic toxicity such as aminoglycoside-induced nephropathy or vestibular toxicity. If patients show evidence of new-onset heart failure, a repeat TTE study is warranted.¹²

This report is important because it draws attention to the possibility that a valvular aneurysmal sequela secondary to IE can manifest itself subacutely, despite the apparent success of antibiotic therapy. It is important to perform routine clinical follow-up evaluation after therapy. In patients who develop these rare valvular sequelae, the aneurysms sometimes rupture, causing further shunting, hemodynamic instability, and the need for urgent surgical evaluation and repair.

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