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Case Report

CASE REPORT

Aorto-cavitary fistula to the left ventricle with severe aortic regurgitation as a complication of prosthetic valve infective endocarditis: a novel report

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Abstract

Infective endocarditis can present in different clinical forms and lead to a variety of complications depending on the affected valvular and perivalvular structures. We describe a case of a 74-year-old male who developed an aortic-cavitary fistulous tract as a complication of prosthetic aortic valve infective endocarditis. Transesophageal echocardiography (TEE) revealed an aorto-cavitary fistula (ACF) connecting the aortic root with the left ventricle, creating an intracardiac shunt, which resulted in severe aortic regurgitation physiology. The patient underwent surgery with successful exclusion of the ACF. ACF is an unusual complication of infective endocarditis that creates an abnormal communication between the aortic root and the heart chambers, establishing an intracardiac shunt. This case highlights that physicians should be aware of the possibility of rare cardiac complications in infective endocarditis. TEE is a valid diagnostic test for ACF.

INTRODUCTION

Infective endocarditis (IE) is an infectious process of the endocardial surface of the heart, most commonly involving the valvular apparatus [1]. The annual incidence of IE is three to nine cases per 100 000 in developed countries like the USA [2]. Patients with prosthetic valves, cardiac devices, unrepaired cyanotic heart disease, intravenous drug use, prolonged bacteremia and history of endocarditis are at the highest risk of developing endocarditis.

The vast majority of IE is caused by *Staphylococci* or *Streptococci*. Well-known cardiac complications due to endocarditis include perivalvular abscess [3], dehiscence of prosthetic valves, conduction abnormalities [4], heart failure [1], cerebrovascular accidents and central nervous system infections [5].

This case report describes a rare complication whereby IE originated an aorto-cavitary fistula (ACF), which is formed when an abscess or pseudoaneurysm involving the sinus of Valsalva ruptures, resulting in the formation of a communication between the aortic root and the cardiac chambers, endangering adequate hemodynamics by the formation of an intracardiac shunt.

CASE REPORT

We present the case of a 74-year-old male who was admitted to the hospital after presenting to the emergency department (ED) with the chief complaint of right arm weakness and difficulty in

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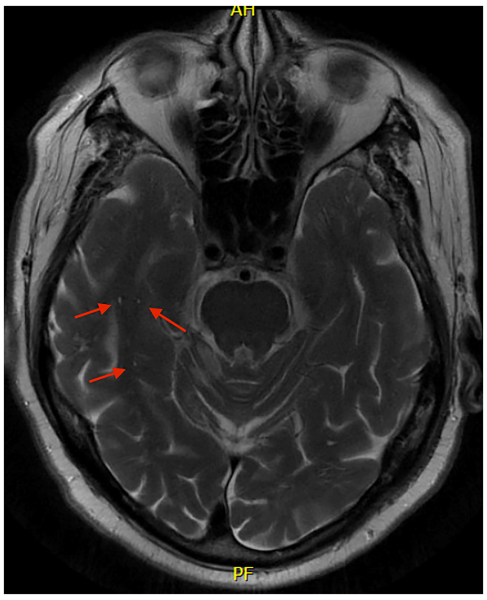

Figure 1: Brain MRI. Findings of disseminated punctate ischemic lesions (red arrows), suggestive of multiple acute ischemic strokes secondary to embolic phenomenon.

speaking. His past medical history included diabetes mellitus, hypertension, persistent atrial fibrillation on long-term anticoagulation with rivaroxaban, heart failure with reduced ejection fraction of 30% and aortic stenosis with two previous bioprosthetic surgical aortic valve replacements in 2004 and 2014. Two months prior to admission, he was diagnosed with IE caused by Group B *Streptococcus* involving the aortic bioprosthetic valve. Initially, he presented with complaints of intermittent febrile episodes and night diaphoresis for approximately 8 weeks. At that time, the decision was made to treat him conservatively with oral antibiotics and close clinical monitoring. Blood cultures were negative in two different occasions, and the patient was discharged on stable conditions. However, a day prior to his second hospital admission, the patient noted sudden onset right upper extremity weakness (MRC power grade 3) and dysarthria. Upon further evaluation in the ED, a magnetic resonance imaging (MRI) of the brain revealed multiple embolic strokes (Fig. 1).

Transthoracic echocardiography and computed tomography angiography (CTA) of the chest revealed nonspecific structural changes with images compatible with a possible aortic perivalvular abscess/pseudoaneurysm (Fig. 2). Transesophageal echocardiography (TEE) confirmed a perivalvular cavity around the aortic root, and this cavity was communicating with the aorta. Furthermore, the cavity was also in communication with the left ventricle (LV), creating a dynamic of retrograde flow, with blood passing from the aorta to the LV through the fistulous tract (Fig. 3). As a result, the patient developed an ACF due to perivalvular endocarditis, which resulted in symptomatic severe aortic valve regurgitation.

The patient was transferred to a tertiary care center for further surgical evaluation. His physical examination was unremarkable, except for a slight right facial droop and mild dysarthria. All initial laboratory including new sets of blood cultures were unremarkable. He continued empiric antibiotic therapy with ceftriaxone. Sacubitril/valsartan and metoprolol were discontinued due to concerns of rapid progression to complete heart block in the setting of a perivalvular cavity.

He was evaluated by a multidisciplinary team including specialists in cardiology, cardiothoracic surgery, neurology and infectious disease. The consensus was that patient's presentation was likely caused by a perivalvular abscess/pseudoaneurysm, which was further complicated by the formation of an ACF, requiring rapid surgical intervention. Coronary angiogram was performed as part of the preoperative evaluation, revealing smooth eccentric narrowing (50–60%) of the ostial/proximal left circumflex (LCx), which raised the suspicion of possible external compression from the known aortic root pseudoaneurysm/perivalvular abscess seen on CTA (Fig. 4).

TEE in the operating room evidenced a large posterior aortic pseudoaneurysm underneath the left coronary artery, corresponding to the narrowing of the LCx. This was then confirmed by surgical exploration and visual inspection. Active infection with pus secretion was noticed around the aortic annulus, with small vegetations on the prosthesis. The surgical procedure consisted of a third cardiac redo surgery with full sternotomy, explantation of previous prosthetic aortic valve and exclusion of the ACF. The aortic root and valve were replaced with a 24 mm homograft, and the LCx was reimplanted.

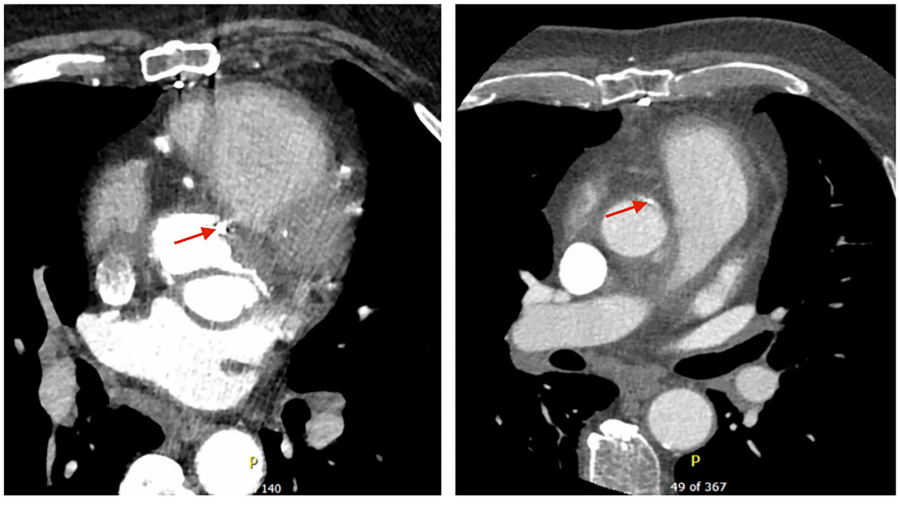

Figure 2: Computed tomography angiography (CTA) of the chest. Nonspecific structural changes of the ascending aorta at the level of the sinotubular junction with images compatible with a possible aortic perivalvular abscess/pseudoaneurysm.

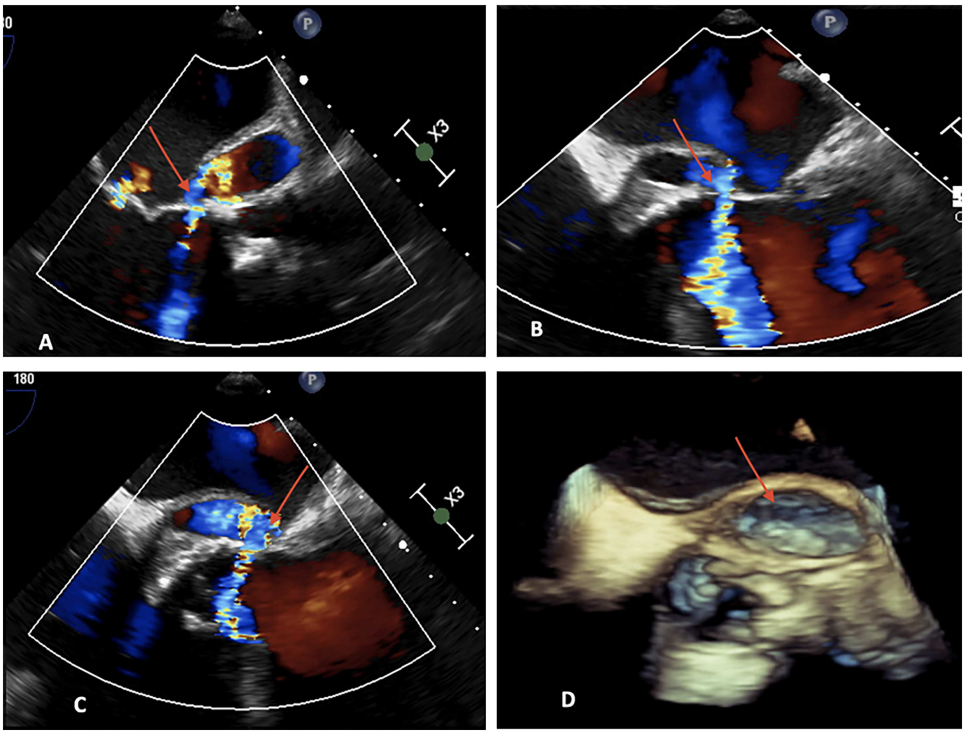

Figure 3: Transesophageal echocardiography (TEE). Perivalvular cavity around the aortic root communicating with the aorta, with presence of retrograde blood flow resulting in severe (4+) aortic valve regurgitation. (**A**) Midesophageal long-axis view with color Doppler showing significant blood flow from the ACF to the left ventricle cavity. (**B** and **C**) Midesophageal five-chamber view with color Doppler showing significant blood flow from the aortic root to the ACF. (**D**) 3D transesophageal echocardiography with visualization of the ACF.

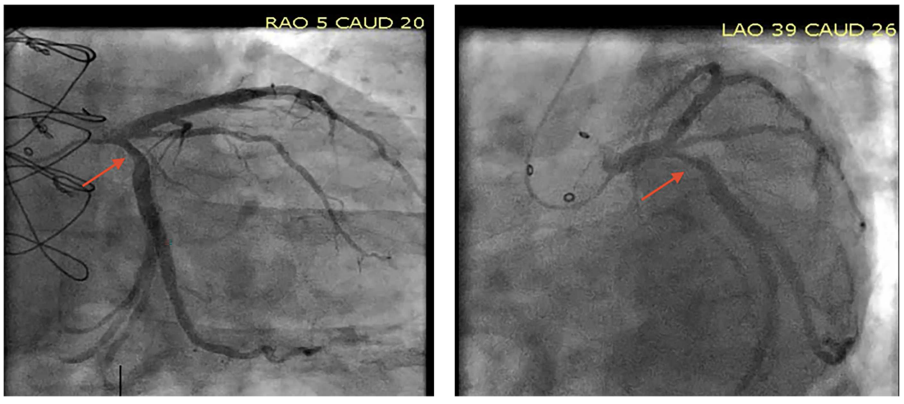

Figure 4: Left heart catheterization with coronary angiography. Eccentric narrowing (50–60%) of the ostial/proximal LCx. The left anterior descending artery (LAD) has mild luminal irregularities. Ramus intermedius branch is present, also with mild luminal irregularities.

The patient recovered without significant complications, and he went home with intravenous ceftriaxone through a peripherally inserted central catheter for a total of 6 weeks, and any further oral antibiotic therapy was not required. Close outpatient monitoring with follow-up with cardiology, neurology, infectious disease and primary care provider was arranged in the next week. All neurological manifestations were entirely resolved with treatment and rehabilitation.

DISCUSSION

A well-documented complication of IE is the development of periannular abscesses [6]. However, cases describing the progression of an abscess into a pseudoaneurysm that ruptures internally, while connecting the aortic root with the heart chambers, and resulting in a subsequent ACF are reported far less common [7]. ACF is caused by the extension and infiltration of the pyogenic and necrotic tissue from the perivalvular abscess into the cardiac chambers. This results in the expansion and rupture of the weakened necrotic myocardium, creating intracardiac fistulous communication. It is possible to develop an intracardiac communication leading to blood flow dynamics similar to those seen in valvulopathies.

Prior to Anguera *et al.* [6], the data was limited exclusively to case reports. According to this study, the prevalence of ACF is as high as 5.8% in individuals in whom endocarditis infects a prosthetic valve. TEE and cardiac MRI are valid imaging modalities for the detection of ACF in native and prosthetic valves [8].

Physicians should consider discontinuation of medications with negative chronotropic properties such as beta blockers and calcium channel blockers, as ACF has been associated with the development conduction abnormalities like complete heart block [9]. The mechanism of atrioventricular conduction disorder in ACF is the result of inflammation from the bacterial colonization of the valve and surrounding tissues, and the electric conduction system of the heart is no exception. ACFs usually originate from the sinus of Valsalva, increasing the risk of invasive coronary angiography. Other imaging modalities to evaluate the coronary arteries such as coronary CTA may be considered in similar circumstances.

Surgery to repair the fistula is advised. Practitioners should have a high suspicion for cardiac pathology in patients presenting with IE and should not hesitate to consider urgent surgical intervention [10]. However, patients who are severely hemodynamically unstable or those who have multiple comorbidities are not candidates to undergo surgery. Percutaneous procedure is another option in high-risk patients when active infection is healed with antibiotics.

CONFLICT OF INTEREST

None declared.

FUNDING

None declared.

ETHICAL APPROVAL AND CONSENT

This case report does not require ethical approval. The patient consented and gave permission for publication of this data.

DATA AVAILABILITY

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

GUARANTOR

Alejandro Sanchez-Nadales, MD.

NOTES

The authors have no relationships relevant to the contents of this paper to disclose. Informed consent was obtained for this case.

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