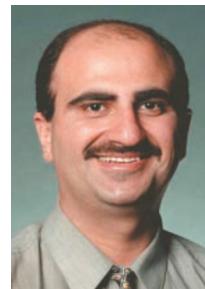


Intracardiac Fistulae: A Rare Complication of Infective Endocarditis

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ABSTRACT

We present the case of a diabetic gentleman who was admitted to the hospital with an infected right foot. Swabs were positive for *Staphylococcus aureus* and *Pseudomonas aeruginosa*. His right big toe was amputated. Postoperatively, the patient experienced recurrent episodes of chest pain. He was therefore transferred to the coronary care unit, where he deteriorated rapidly. The patient was subsequently transferred to intensive care. Transthoracic and transesophageal echocardiograms revealed evidence of aortic dissection, but this finding was not confirmed in a computed tomography scan. The patient subsequently experienced cardiac arrest and died. The postmortem examination revealed no aortic dissection but did show a vegetation on the mitral valve with a fistula that tracked into a ruptured epicardium.

INTRODUCTION

Infective endocarditis (IE) was first described by Sir William Osler during his Gulstonian Lectures, which were delivered at the Royal College of Physicians in 1885. Although most commonly associated with a disease process involving the native valve leaflets, IE may also affect the chordae, the myocardium, and implanted shunts, valves, and conduits. Perianular extension of the infection into the adjacent myocardium is a serious complication associated with increased patient mortality; it therefore predisposes a patient to a greater need for surgical intervention. Tissue necrosis and pyogenesis may lead to the formation of an abscess cavity. The weakened and necrotic myocardial tissue may come under increased pressure from an expanding space-occupying lesion and rupture, thereby creating fistulous communications.

Perianular extension of IE occurs in approximately 10% to 40% of native valve IE cases and is more commonly seen in the aortic position [Graupner 2002]. Echocardiography,

particularly transesophageal echocardiography (TEE), is the preferred investigation modality for valvular infections [Kang 2009].

CASE REPORT

A 67-year-old man was initially transferred to the vascular unit of our hospital with a 2-month history of a poorly managed infected right foot. He was known to have non-insulin-dependent diabetes with poorly controlled glucose levels (hemoglobin A1c value of 10.9% on arrival). His medical history also included hypertension, osteoarthritis, and being an ex-smoker. Swabs from the infected toe were positive for *Staphylococcus aureus* and *Pseudomonas aeruginosa*. His complete blood count showed an elevated white cell count ($16 \times 10^3/\mu\text{L}$) but was otherwise unremarkable. His C-reactive protein serum level was also elevated (235 mg/L). The patient was commenced on 2 g flucloxacillin administered intravenously over 6 hours and 500 mg ciprofloxacin administered orally every 12 hours. He subsequently underwent debridement and amputation of his right big toe. There were no immediate postsurgical events.

Postoperatively on the surgical ward, the patient was continued on the same antibiotic regimen, and daily dressings were administered. Three days after the amputation, the patient experienced 2 episodes of chest pain overnight, with radiation to the jaw. These episodes were accompanied by small decreases in blood pressure. No electrocardiographic changes were noted. The patient's condition was managed with aspirin, fluids, and analgesia. The next morning, the patient experienced a third episode of chest pain, along with hypotension and hypoxia. He was afebrile but was short of breath, with a blood pressure of 70/50 mm Hg and an oxygen saturation of 62% on room air. There were no electrocardiographic changes, and the troponin I concentration was not elevated. The patient's chest radiograph was normal on initial admission. He was transferred to the coronary care unit with a provisional diagnosis of postoperative myocardial ischemia. Dopamine and heparin infusions were commenced. The intensive care unit (ICU) team became involved, and a repeat chest radiograph showed a widened mediastinum (Figure 1).

The patient deteriorated further later that morning. He was then intubated and transferred to the ICU with a suspicion of

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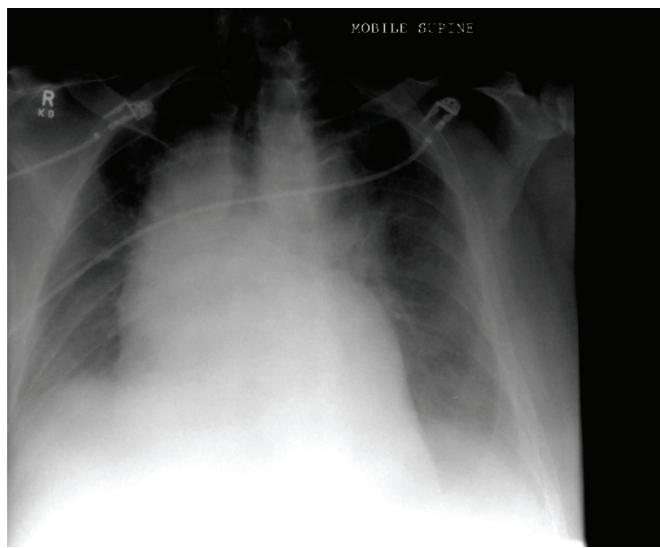


Figure 1. Chest radiograph showing a widened mediastinum.



Figure 3. A computed tomography scan of the thorax did not reveal any obvious aortic dissection. Note bilateral pleural effusions.

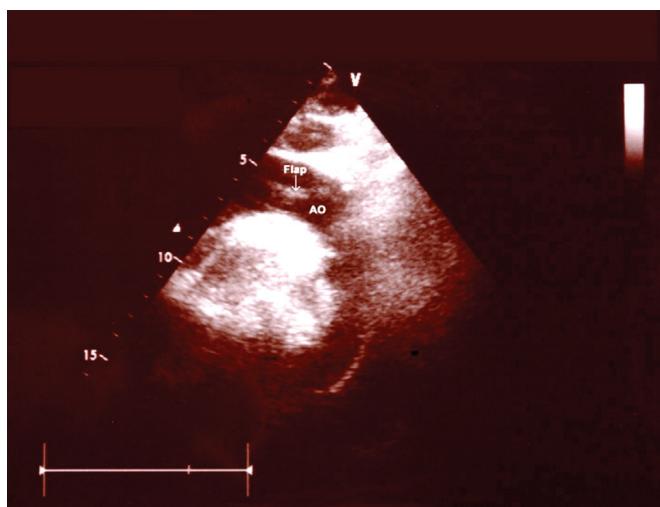


Figure 2. Transthoracic echocardiogram showing the suspicious dissection flap on the ascending aorta. AO indicates ascending aorta.

acute aortic dissection. A transthoracic echocardiogram performed prior to transfer revealed a suspicious dissection flap in the ascending aorta (Figure 2). The cardiothoracic unit was then consulted; hence, we became involved in the patient's care.

In the ICU, the patient was stabilized with a noradrenaline infusion. Once stable, he underwent a computed tomography (CT) scan of his chest and abdomen. No aortic dissection was found, but there were bilateral pleural effusions and a mild pericardial effusion (Figure 3).

Bilateral intercostal drains were inserted, and serosanguinous fluid was drained. A subsequent diagnostic pericardiocentesis procedure further revealed turbid bloodstained fluid, a culture of which revealed infection by *S aureus*. A subsequent urgent TEE evaluation again revealed a suspicious

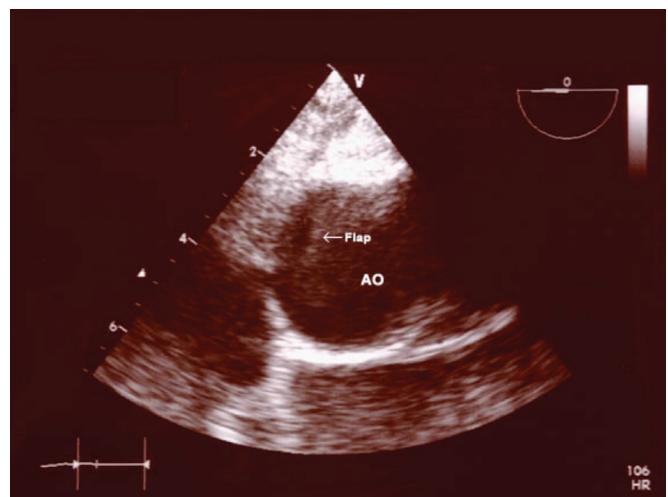


Figure 4. Transesophageal echocardiography scan, again revealing the suspicious flap on the ascending aorta. AO indicates ascending aorta.

dissection flap in the ascending aorta (Figure 4). Color Doppler studies were done but did not show a clear flow within the suspected aortic intimal flap area. This finding added to the confusion regarding reaching a definitive diagnosis.

By the early afternoon, the patient was still clinically and hemodynamically stable with ICU support. Because of the discrepancy between the echocardiography and CT results, a decision was made to perform a magnetic resonance imaging (MRI) evaluation to clarify the suspicion that an aortic dissection had occurred. In the MRI unit, the patient experienced

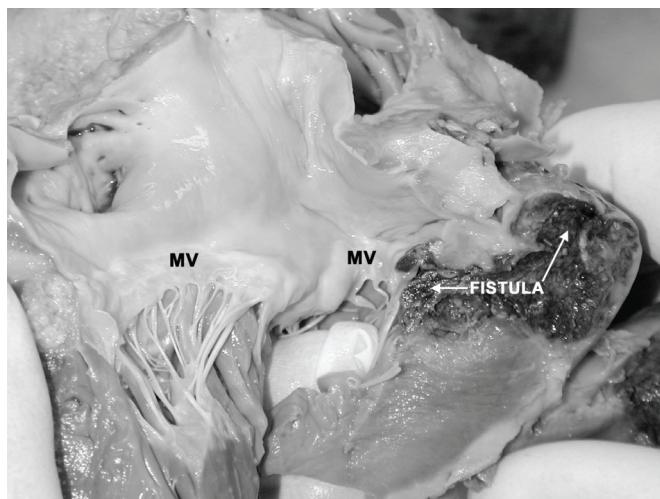


Figure 5. Autopsy specimen of the heart showing the fistula tract from the mitral valve (MV) to the epicardium.

an acute cardiac tamponade. We inserted a pigtail cardiocentesis catheter, which drained 800 mL of blood.

The patient then experienced cardiac arrest. Cardiopulmonary resuscitation was commenced, but the patient unfortunately did not survive. A postmortem examination revealed severe pericarditis and a vegetation on the mitral valve that connected to a fistula tract extending to the epicardium, which had then ruptured and bled (Figure 5). There was no aortic dissection.

DISCUSSION

Intracardiac fistulae are uncommon conditions of the heart. Known causes include congenital causes, trauma (penetrating or blunt), myocardial infarction, tumor, an iatrogenic cause, and, as in this case, infection. The incidence of IE is estimated to be 4 in 100,000 patient-years [Mylonakis 2001]. One study found that fistulae occurred in 8.5% of IE cases, all of which had perivalvular involvement [Baumgartner 2000].

The most common causative organism in IE is *Streptococcus*, followed by *Staphylococcus*, especially *S aureus* [Tak 2002]. Staphylococci strains are usually more virulent, however, and so have a higher propensity to cause perivalvular abscess and fistula formation [Baumgartner 2000; Anguera 2005]. Other organisms that should be considered are the HACEK group of bacteria (*Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Eikenella*, and *Kingella*). This group of fastidious gram-negative bacilli shows limited proliferation under conventional culture conditions [Lester 2007].

IE complicated by fistula formation can present in different clinical settings, depending on the site of communication. Refractory heart failure appears to be a consistent clinical picture [Ananthasubramanian 2005]. Another clinical feature that should alert the physician to the possibility of periannular complications is the occurrence of electrical-conduction disturbances, such as atrioventricular block or a newly developed bundle branch block. In fact, electrocardiogram evidence of

a newly developed atrioventricular block has an 88% positive predictive value for abscess formation [Graupner 2002]; however, clinical features are usually difficult to interpret.

A definitive diagnosis from imaging is therefore necessary. TEE is a superior method of investigation for detecting endocarditis vegetations. The literature generally reports sensitivity values ranging from 85% to 100% [Shapiro 1994; Reynolds 2003; Lester 2007]; however, the specificity of TEE evaluation may not be sufficiently high for differentiating septic vegetations from aseptic vegetations, such as Libman-Sacks [Evangelista 2004]. The specificity has been recorded to be approximately 88% to 100% [Ryan 2000]. For detecting perivalvular abscesses, TEE has a sensitivity and specificity of 87% and 95%, respectively [Daniel 1991]. With continuous wave Doppler, communications within the heart can be discerned [Shanewise 1996]. In the present case, the suspicious flap may have been the proximal end of the mitro-epicardial fistula; however, abscesses and their complications on the mitral valve are more difficult to detect than those involving the aortic valve (57% versus 86%) [Daniel 1991].

IE is mainly treated medically. The principal indications for surgical intervention include heart failure, a poorly controlled infection, embolisms, large vegetations, perivalvar extension, valvar obstruction, and an unstable prosthesis [Delahaye 2004]. If a fistula is diagnosed, surgical repair should be considered. Such surgery includes simple closure or patch repair with either pericardium or synthetic materials. The choice of technique is usually made intraoperatively, depending on the site of damage [Anguera 2005]. Valve replacement may occasionally be required.

This report illustrates the difficulty in interpreting a mitral perivalvular fistula and the limitations of TEE in detecting low-pressure flows, as through the mitral valve.

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