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 × 10^3/μL) but was otherwise unremarkable. His C-reactive protein serum level was also elevated (235 mg/L). The patient was commenced on 2 g flucloxacillin administrated 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...
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 \textit{S. aureus}. A subsequent urgent TEE evaluation again revealed a suspicious 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...
an acute cardiac tamponade. We inserted a pigtail cardiocen-
tesis catheter, which drained 800 mL of blood.

The patient then experienced cardiac arrest. Cardiopul-
monary resuscitation was commenced, but the patient unfor-
nortunately 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 abscesses 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 differ-
ent clinical settings, depending on the site of communication. Refractory heart failure appears to be a consistent clinical pic-
ture [Ananthasubramanium 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

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**REFERENCES**


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