Interatrial abscess

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Summary
A large abscess of the interatrial septum developed during the course of acute bacterial endocarditis affecting the aortic valve. Septic involvement of the cardiac conducting system produced atrioventricular dissociation, and subsequent abscess rupture resulted in an aorto-right atrial communication. The clinical presentation of this rare complication of acute bacterial endocarditis is correlated with the post-mortem findings.

Introduction
Myocardial abscesses, a common complication of acute bacterial endocarditis, rarely give rise to localizing symptoms or signs. They are usually small and widespread within the myocardium (Sanson, Slodki and Gruhn, 1963) or adjacent to valve rings (Sheldon and Golden, 1951). Their presence is often masked by the clinical features of severe systemic infection. The incidence and progression of myocardial abscesses are related to the virulence of the organism and the extent of systemic infection. Gram-positive organisms are the most frequent offending bacteria. In a series of 29 cases of myocardial abscesses, derived from 14160 post-mortems, 23 were caused by staphylococci, 3 by streptococci and 2 by pneumococci (Flaxman, 1943). In Sheldon and Golden's study (1951) of 12 cases of valve ring abscesses, pneumococci were found in 9 cases and staphylococci in 3.

The authors have recently encountered a patient with acute staphylococcal endocarditis, who developed a large, interatrial abscess accompanied by an aorta/right atrial fistula and atrio-ventricular dissociation. Study of this rare complication of bacterial endocarditis provides an interesting correlation between extent of cardiac involvement and observed clinical features. Only 2 previous reported cases of myocardial abscess at this site, with similar complications, have been found (Zettner and Irmiere, 1959; Langaker and Svanes, 1973).

Case report
A 33-year-old male was transferred to the Liverpool Regional Cardiac Centre for further treatment of acute bacterial endocarditis. Three weeks before this he had presented to his own general practitioner with malaise, fever and haematuria. He was treated with a number of oral antibiotics including co-trimoxazole, nalidixic acid and cephalaxin, without improvement. Two weeks after the onset of symptoms he became confused, and incontinent of urine and was admitted to Chester City Hospital.

Examination at this stage revealed finger clubbing and multiple splinter haemorrhages of the nail beds. The pulse was regular at 110/min, blood pressure was 130/70 mmHg, the apex beat was of thrusting character in the sixth intercostal space at the mid-clavicular line. Auscultation revealed an ejection systolic murmur (grade 2/4) and an early decrescendo diastolic murmur (grade 2/4) at the left sternal edge. There was no cardiac failure. The chest was clear, the spleen was palpable one finger below the costal margin, and no focal neurological signs were detectable.

He had joined the Royal Navy when aged 18 years but was discharged after 2 years because of the detection of a heart murmur during routine medical examination. There was no history of rheumatic fever, syphilis or hypertension.

ECG on admission demonstrated sinus rhythm with a normal PR interval, a mean frontal QRS vector of 80 and left ventricular hypertrophy. Chest X-ray showed mild cardiomegaly, with clear lung fields. Haemoglobin was 9.9 g/dl WBC 25.0 × 10⁹/l with 90% polymorphs. Blood cultures revealed Staphylococcus aureus. Treatment was initiated with
intravenous benzylpenicillin 24 Mu. daily, probenecid, digoxin and diuretics. He remained confused and pyrexial and serial ECG recordings revealed the development of pathological Q waves in leads II, III and AVF. Three days after admission he developed monofascicular complete heart block with a ventricular rate of 100/min and was transferred to the Liverpool Regional Cardiac Unit.

On admission he was tachypnoeic and pale with a pyrexia of 39°C. The pulse was 100 beats/min, irregular and collapsing in character with a blood-pressure of 140/60 mmHg. There was marked arterial pulsation in the neck, jugular venous engorgement to 4 cm above the clavicle and pistol shot sounds were audible over the dorsalis pedis arteries. The apex beat was diffuse in the left sixth intercostal space at the anterior axillary line and of thrusting character. Auscultation demonstrated a mid-systolic murmur (grade 2/4) and an immediate diastolic murmur (grade 3/4) at the left sternal border. A grade 2/4 low pitched diastolic murmur and a variable fourth heart sound were present at the apex. Fundoscopic examination was normal and there were signs of mild congestive cardiac failure.

Haemoglobin was 9.5 g/dl, WBC 20 × 10⁹/L, ESR 76, and blood cultures continued to yield a profuse growth of S. aureus. Serial ECG records demonstrated an evolving inferior myocardial infarction pattern with a monofascicular complete heart block and multifocal ventricular ectopic beats.

Flucloxacillin 1.5 g i.v. every 6 hr was added to his existing treatment and digoxin was discontinued. Four days after transfer he developed acute pulmonary oedema and a loud diastolic murmur became audible to the right of the sternum imparting a continuous character to the murmurs. His jugular venous pressure was raised to the angle of the jaw with prominent systolic waves. The pulmonary oedema remained refractory to treatment and he died in asystole 12 hr after its onset.

Post-mortem revealed a bicuspid aortic valve with a large friable vegetation arising from the lower part of the posterior aspect of the valve. The vegetation had spread down the adjacent left ventricular wall and was almost completely occluding the valve lumen. The infective process had tracked around the aortic valve ring and formed an ulcerated mass in the adjacent right atrium with a fistula between the aortic valve and the right atrial cavity (Fig. 1). A soft swelling was present in the adjacent part of the left atrium with early ulceration of the surface of this mass. On incising the swelling, blood-stained pus exuded. The other heart valves were normal. There was no coronary atheroma and no coronary emboli. No macroscopic areas of infarction could be identified in the ventricular myocardium. Examination of the brain revealed an area of haemorrhage into the pia arachnoid membranes over the left parietal lobe, and a necrotic abscess 2 cm in diameter was present in the left occipital lobe.

Discussion

Single or multiple abscesses of the valve rings were demonstrated by careful dissection technique in a clinico-pathological post-mortem study in 86% of patients with treated acute bacterial endocarditis (Sheldon and Golden, 1951). Myocardial abscess formation should be suspected if there is a slow response to antibiotic therapy, for although antibiotics control surface valve infection, they are unable to penetrate valve ring abscesses in adequate concentration to prevent their extension (Sheldon and Golden, 1951). The development of atrio-ventricular conduction disturbances during the course of aortic valve endocarditis indicates deep extension of infection and is associated with a poor prognosis (Roberts and Somerville, 1969). In the present patient there was incomplete response to appropriate antibiotic therapy and ECG evidence of interruption of atrio-ventricular conduction was present. The development of a continuous murmur coincident with acute pulmonary oedema, and prominent systolic pulsation in the jugular veins, shortly before death, suggests that fistulous communication between the aorta and right atrium occurred at this time.

Myocardial abscess formation may arise from lodgement of infected emboli in coronary arteries (Langaker and Svanes, 1973) or by contiguous spread from valve rings. Sheldon and Golden (1951) considered, on histological grounds, that valve ring abscesses may be mycotic aneurysms of the vessels supplying the valve ring. It is possible that the ECG evidence of inferior infarction, in the present patient, was due to an infected coronary embolus, but it is felt that direct extension of the septic process, through the annulus fibrosus of the aortic valve into the atrial septum was the most likely pathogenesis of the intratral abscess. This route was traceable at post-mortem indicating direct spread of infection. The atrio-ventricular node and part of the conducting bundles are contained within the interatrial and membranous septum which were involved in the abscess and fistula formation. A space-occupying lesion in this position would thus give rise to atrio-ventricular conduction disturbance. In other reports of myocardial abscess in this position, complete heart block was present, as in this patient (Zettner and Imiere, 1959; Langaker and Svanes, 1973).

Although myocardial abscess with fistula formation in the interatral septum is rare, its presence may be suspected when a combination of poor response to adequate antibiotic therapy, conduction disturbances and continuous murmurs occur during
the course of bacterial endocarditis affecting the aortic valve.

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References
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