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Case Report

Endocarditis and myocardial abscess caused by group B *Streptococcus*

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Introduction: Group B *Streptococcus* (GBS) is a rare infectious endocarditis. Patients with GBS infective endocarditis have a high rate of local and systemic complications.

Case presentation: A 30-year-old male presented to the Emergency Department with fever, chills, fatigue and a recent onset of symptoms suggestive of stroke following a bout of pyelonephritis. Echocardiography confirmed a diagnosis of endocarditis and blood cultures grew GBS. Antibiotic therapy was initiated with penicillin G and gentamicin. Urological evaluation revealed a urethral stricture. He was taken to the operating room on hospital day 10 for the debridement of his aortic annulus, reconstruction of his aortic root and replacement of his aortic valve. On post-operative day 7, he died of sudden cardiac arrest. A large myocardial abscess located within the interventricular septum was identified post-mortem.

Conclusion: Recurrent or complicated urinary tract infections are rare among the young male population. Without evaluation and treatment for the underlying pathology, patients are at risk of developing antimicrobial-resistant infections, which may disseminate rapidly. Although a common pathogen of the urinary tract, GBS is a rare infectious agent for endocarditis. We propose urethral stricture as a risk factor for developing GBS endocarditis. Operative timing for these infections can be challenging; however, urgent and radical surgical debridement appears to yield favourable results.

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Introduction

Group B *Streptococcus* (GBS) is a β-haemolytic, Gram-positive coccus and is a pathogen mainly of the genitourinary tract and soft tissues. Endocarditis is rare and typically is seen in immunocompromised individuals. These are aggressive infections with high levels of systemic complications and mortality rates, which may be explained by the pathogen's lack of fibrinolysin (Sambola *et al.*, 2002; Chang & Cunha, 2004). Myocardial abscess is a rare complication, with only two current reports (Scully *et al.*, 1987; Chang & Cunha, 2004). Whilst medical management may appear beneficial in the absence of indications for emergent surgery, urgent and radical surgical debridement may improve prognosis (Scully *et al.*, 1987; Sambola *et al.*, 2002; Prendergast & Tornos, 2010; Kang *et al.*, 2012) We report a case of GBS infectious endocarditis complicated by left ventricular–aortic valve discontinuity and myocardial abscess in a young, previously healthy male.

Abbreviations: GBS, group B *Streptococcus*; UTI, urinary tract infection.

Case report

A 30-year-old man was evaluated for fever, and flank and abdominal pain with vomiting in the Emergency Department. He was diagnosed with pyelonephritis and sent home on oral levofloxacin.

Four days later, he presented to the Emergency Department with new-onset facial weakness and slurred speech suggestive of stroke. His past medical history included recurrent urinary tract infections (UTIs) for 2 years' duration. A review of systems revealed an acute history of fever, chills, appetite change and fatigue, which developed in the intervening 4 days. On examination, he had facial asymmetry, difficultly speaking, a 2/6 diastolic murmur at the right second interspace and flank tenderness. The rest of his neurological, musculoskeletal and skin examination was unremarkable.

Computed tomography imaging of the brain confirmed a recent infarct. Later, left pleuritic chest pain, shortness of breath and tachypnoea developed. A chest X-ray was unremarkable, and computed tomography revealed pericardial effusion but no pulmonary emboli. A transthoracic echocardiogram revealed aortic valve vegetations with severe aortic insufficiency and mild aortic root dilation. Blood cultures grew GBS, and his antibiotic regimen was switched from empiric vancomycin and ceftriaxone to high-dose penicillin G and gentamicin. Surgical and neurological consultation was obtained for aortic valve replacement. The timing of valve replacement because of his recent stroke was discussed, in particular the risks of haemorrhagic conversion with systemic anticoagulation during surgery and cardiopulmonary bypass. Urological consultation was sought in light of his recurrent UTIs, which revealed a urethral stricture. Cardiac surgery was performed on hospital day 10.

At the time of surgery, a large, bloody pericardial effusion with fibrinous pericarditis was noted. The aortic valve demonstrated dehiscence of the annulus at the commissure between the non-coronary and right coronary cusps with vegetations on the ventricular side of the leaflets. The left cusp and annulus were spared. Pus was expressed from the abscess extending downward towards the septum, after which the valve leaflets and annulus were debrided in the customary manner. The annulus was then reconstructed utilizing fresh, autologous pericardium, and a mechanical valve was fitted and then placed.

Outcome and follow-up

On post-operative day 7, the patient developed atrial fibrillation associated with chest pain. He was started on a Cardizem drip but shortly thereafter developed an episode of nausea and vomiting, and subsequently went into asystolic cardiac arrest. Aggressive resuscitation efforts were unsuccessful and the patient died on post-operative day 7.

The autopsy was limited to the heart and lungs. The pericardial sac was thickened by a dark-red haemorrhage, ranging from 0.5 to 1.5 cm in thickness. Gross examination of the heart revealed a massively enlarged, 700 g heart covered by dark-red, haemorrhagic, 'shaggy' fibrinous adhesions (Fig. 1). Both the mechanical valve and surgical access were intact and healing without evidence of dehiscence or infection.

The interventricular septum, immediately inferior to the aortic valve, contained a $5.0 \times 3.0 \times 1.0$ cm abscess cavity that bulged focally into the pulmonary outflow tract (Fig. 1). The surrounding myocardium exhibited extensive

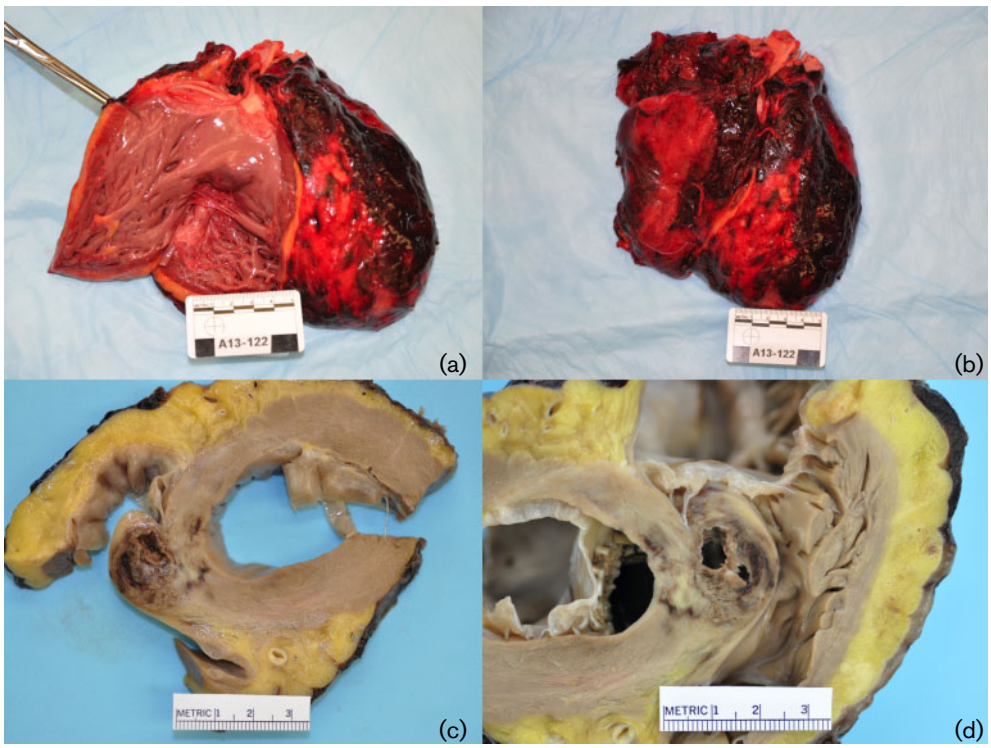

Fig. 1. (a) Abscess bulging into the pulmonary outflow tract. (b) Anterior surface covered in a dark-red thrombus. (c) Superior view showing focal transmural necrosis adjacent to the abscess. (d) Inferior view showing the abscess inferior to the placed valve.

focal transmural necrosis. The pathological diagnosis was ruled as sudden cardiac death due to bacterial endocarditis with necrosis and abscess formation within the interventricular septum and aortic valve ring.

Discussion

Although usually associated with post-partum endocarditis prior to the antibiotic era, cases of GBS have increased in adults, mainly in elderly patients or in those with risk factors that include chronic immunosuppressive diseases such as diabetes mellitus, alcoholism, intravenous drug use, neoplasia and human immunodeficiency virus infection (Sambola *et al.*, 2002; Chang & Cunha, 2004). Common identifiable sources for GBS include soft-tissue infection, gynaecological infection and UTIs.

Here, we report a case of GBS infectious endocarditis complicated by left ventricular–aortic valve discontinuity and myocardial abscess in a young, previously healthy male. Whilst he had no pre-existing cardiac pathology, the history of frequent UTIs secondary to a urethral stricture probably accounts for the offending organism. To date, to the best of our knowledge, only two other cases of GBS endocarditis complicated by myocardial abscess formation and perivalvular abscess have been reported in the literature (Scully *et al.*, 1987; Chang & Cunha, 2004).

UTIs are rare in the young male population. Whilst uncomplicated UTIs can be managed with a 7-day antibiotic regimen, young males who present with fever, pyelonephritis and/or recurrent infection should have a thorough urological evaluation. Without correction of the underlying abnormality, patients are prone to antimicrobial-resistant infections, which carry high rates of treatment failure (Grabe *et al.*, 2008). Had our patient's urethral stricture been identified earlier, he probably would not have developed endocarditis. This case represents the need for a low threshold for urological referral for young males experiencing either repeated or complicated upper UTIs. A high index of suspicion for underlying urological pathology is warranted. Without a thorough urological evaluation, patients are at risk of developing infections that are resistant to antibiotics, and these infections may disseminate rapidly.

Infections with GBS tend to be aggressive, with frequent local and systemic complications. Embolization is common, which may be explained by the large, friable, valvular vegetations from the organism's persistence within the fibrin clot due to the organism's lack of fibrinolysin. Complications include major systemic embolization (34%), heart failure (28%), abscess formation (2%) and heart block (3%) (Chang & Cunha, 2004). Our patient did not have any of the usual predisposing risk factors, yet experienced all of these complications.

Operative timing in endocarditis is controversial, but the trend is towards early intervention after diagnosis to avoid complications (Prendergast & Tornos, 2010; Kang *et al.*, 2012). Once systemic complications such as stroke occur, the timing becomes more difficult. Systemic heparinization has the potential to exacerbate neurological injury due to intracranial haemorrhage. Conversely, waiting for effective antibiotic therapy may be preferred in the absence of heart failure or rhythm disturbances (Scully *et al.*, 1987; Sambola *et al.*, 2002; Prendergast & Tornos, 2010; Takahashi *et al.*, 2013). These issues influenced the interventional timing for our patient, whose risk of haemorrhagic conversion was weighed against the risk of further pre-operative complications. Although no absolute indications were present for urgent surgery pre-operatively, delaying surgery may have resulted in a more aggressive, localized infection involving not just the aortic leaflets but also the annulus and intraventricular septum.

Despite radical debridement, our patient developed a large myocardial abscess within the septum that was only identified post-mortem. The septal abscess was not identified on pre-operative or intra-operative echocardiography, nor was it visualized at the time of surgery. This suggests that the abscess propagated post-operatively, leading to eventual heart block and rupture. The abscess spread inferiorly from the placed valve and repaired annulus, exemplifying the aggressive nature of GBS endocarditis. On post-mortem examination, the pericardial patch was intact, supporting the aggressive debridement and choice of autologous pericardium for patching, as recommended by Takahashi *et al.* (2013).

A high index of suspicion of underlying urological pathology is needed for any young male developing either recurrent or complicated UTIs. Without intervention of the pre-existing pathology, a life-threatening infection may develop. Although not a novel organism for the urinary tract, GBS endocarditis secondary to urethral stricture has, to the best of our knowledge, never been reported. We propose urethral strictures as a risk factor for the development of GBS endocarditis. Management of GBS endocarditis is problematic; the lack of fibrinolysin makes these infections exceptionally aggressive, prone to embolization and difficult to eradicate with antibiotics. Aggressive and early surgical debridement is essential. GBS endocarditis patients who experience heart failure, embolization and/or peri-/myocardial involvement are likely to benefit from urgent radical surgical intervention rather than elective surgical repair following a period of antibiotic therapy. Earlier recognition and treatment of our patient's underlying reason for recurrent UTIs could have potentially prevented this devastating complication.

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