

Case Report: Group B Streptococcus Related Lower Limb Necrotizing Fasciitis, Complicated by Purulent Pericarditis and Cardiac Tamponade

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Abstract

Background: Necrotizing soft tissue infections (NSTI) are severe and rapidly progressive. Rarely, Group B Streptococcus (GBS) can cause NSTI, majority due to an immunocompromised state. Even more uncommon is pericardial involvement following NSTI of a non-adjacent structure.

Case Report: We report a challenging case of NSTI of the lower limbs due to GBS, with acute pericardial dissemination leading to cardiac tamponade. Bedside echocardiography revealed a massive pericardial effusion, measuring largest at 2 cm in depth, with evidence of both right atrial and ventricular collapse, leading to an urgent pericardiocentesis being performed which revealed turbid-looking aspirate. Urgent gram staining revealed moderate amounts of pus cells with occasional gram positive cocci. Wound debridement was performed on day 3 of admission, and tissue cultures were taken peri-operatively. Cultures from blood, pericardial aspirate and tissue aspirate were positive for Streptococcus Agalactiae. Unfortunately, the patient deteriorated post-operatively due to extensive blood loss and overwhelming septicemia and succumbed to his illness 72 hours after.

Conclusions: This case highlights the rare possibility of cardiac involvement in cases of NSTI, and the possibility of cardiac tamponade causing cardiogenic shock masquerading alongside septic shock, and reminds clinicians on the importance of combining clinical acumen and appropriate ancillary testing to facilitate early detection of a fatal condition.

Keywords: Case Report, Group B Streptococcus, Streptococcus Agalactiae, Cardiac Tamponade, Purulent Pericarditis, Pericarditis

Abbreviations: GBS: Group B Streptococcus ECG: Electrocardiogram

Background

Necrotising soft tissue infections (NSTI) are severe and rapidly progressive, requiring rapid recognitions and early, often surgically-based, management. Mono-microbial types of NSTI (i.e. Type 2 NSTI), which amounts for 20 to 30% of overall cases, are often linked to invasive Group A Streptococcus or *Staphylococcus Aureus* infections¹. Rarely, Group B Streptococcus (GBS), also known as *Streptococcus Agalactiae*, are implicated². We report a unique case of NSTI of the lower limbs due to GBS, with acute pericardial dissemination leading to cardiac tamponade, leading to a diagnostic dilemma due to co-existing cardiogenic and septic shock.

Case Report

A 51-year old gentleman of Chinese ethnicity presented with right foot pain and swelling over 2 weeks, associated with chest pain and shortness of breath during that period. He had a 10-year history of poorly controlled diabetes mellitus with a HbA1c level of 8.8 %, hypertension and dyslipidaemia.

He was hypotensive on arrival, with a blood pressure of 91/60 mmHg and hypoxic, requiring high flow oxygen of 15L/min to maintain saturations at 100%. Otherwise other vitals were stable, pulse rate being 72 beats per minutes, respiratory rate 24 breaths per minute and a temperature of 37.4 degrees Celsius.

Clinical examination revealed a gangrenous lateral two toes extending into the lateral malleolus on the right foot, with evidence of pus discharge and associated warmth and crepitus up to hindfoot level on palpation. There was also evidence of dry gangrene in the fourth toe of the left foot, with presence of a small puncture at dorsum of foot with pus discharge. Similarly, crepitus was felt up to midfoot level on palpation of the left side. Bilateral dorsalis pedis and posterior tibialis pulses were palpable but feeble.

Table 1: Blood Investigations on Admission

Blood Test	Results	Blood Test	Results
White Cell Count	26.99 x10 ⁹ /L	Alkaline Phosphatase	168 U/L
Neutrophil	90.30%	Alanine Aminotransferase	37 U/L
Lymphocyte	4.50%	Aspartate Aminotransferase	40 U/L
Platelets	210 x10 ⁹ /L	Sodium	121 mmol/L
Hemoglobin	10.0 g/dL	Potassium	7.6 mmol/L
Lactate Dehydrogenase	441 U/L	Urea	40.5 mmol/L
International Normalised Ratio	1.2	Creatinine	323 µmol/L
Activated Partial Thromboplastin Time	36.5 s	Creatinine Kinase	43 U/L
Prothrombin Time	14.6 s	Total Bilirubin	21 µmol/L

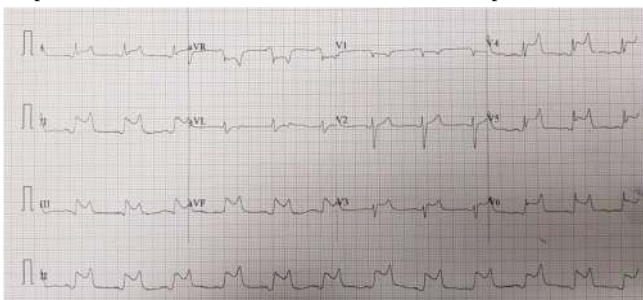
Figure 1a & 1b: Radiography of the (1a) left foot and (1b) right foot respectively, demonstrating gas within soft tissue bilaterally.



Figure 2: Chest radiography demonstrating cardiomegaly and globular-shaped heart, with loss of left-sided cardiophrenic and costophrenic angles.



Figure 3: Electrocardiogram demonstrating widespread saddle-shaped ST-elevation, consistent with pericarditis.



Initial blood investigations are highlighted in Table 1. HIV Antibody, Hepatitis B Surface Antigen and Hepatitis C Antibody serology were all negative. Lower limb radiography revealed evidence of gaseous shadows bilaterally (Figure 1). The clinical and radiological findings were consistent with necrotising soft tissue infection of bilateral feet, and the patient was advised for extensive wound debridement and possible amputation of the affected sites during an orthopaedic consult.

However, on closer review of the chest radiography, there was evidence of cardiomegaly with a globular-shaped heart (Figure 2). His electrocardiogram on arrival, revealed diffuse ST-segment elevations on majority of leads, ST-segment depression on lead aVR consistent with pericarditis (Figure 3).

Figure 4: Parasternal Long Axis view of bedside echocardiography showing evidence of pericardial effusion and right atrial and ventricular collapse

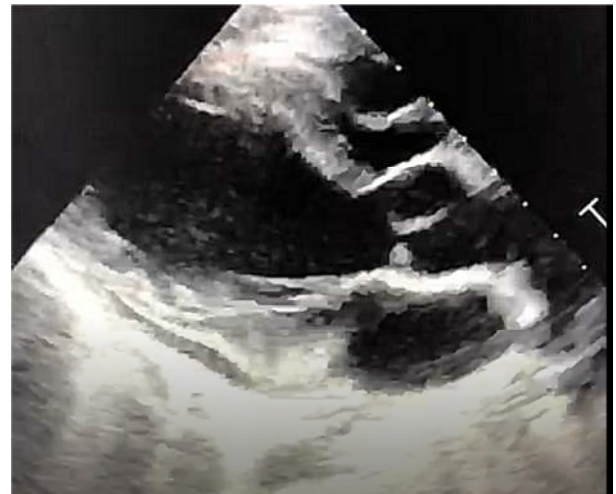


Figure 5: Purulent pericardial aspirate via pericardiocentesis



A bedside echocardiogram was performed, revealing a massive pericardial effusion, measuring largest at 2 cm in depth, with evidence of both right atrial and ventricular collapse (Figure 4).

An urgent pericardiocentesis was performed, under echocardiographic guidance, which revealed turbid-looking aspirate (Figure 5). Urgent microscopic analysis revealed 45 Cells per mm³, majority of which were lymphocytes, and gram stain showed moderate amounts of pus cells with occasional gram positive cocci. Pericardial fluid was negative for acid-fast bacilli.

A repeat transthoracic echocardiogram was performed post-pericardial drain insertion, revealing minimal remnant pericardial fluid, with the pericardial drain in-situ, and no evidence of any mass or vegetation. Unfortunately, a transoesophageal echocardiography and Computed Tomography (CT) imaging of the mediastinum (to rule out mediastinitis and pneumonitis) was not performed, as management of the NSTI took precedence.

The patient was started on intravenous antibiotics, both tazobactam-piperacillin and clindamycin. There was a delay in performing limb saving wound debridement as the patient was reluctant for invasive management, but had later consented to the procedure which was performed only on day 3 of admission. Tissue cultures were taken peri-operatively. Unfortunately, the patient deteriorated post-operatively due to extensive blood loss and overwhelming septicaemia and succumbed to his illness 72 hours after. Subsequently, it was revealed that cultures from blood, pericardial aspirate and tissue aspirate were positive for GBS infection.

Discussion

GBS is a common microorganism, often colonising the gastrointestinal and reproductive tract³. Rarely, GBS colonises the skin and can cause necrotising fasciitis, i.e. necrotising soft tissue infection (NSTI), with only 22 cases having been reported in the past ii. Majority of these patients are either immunocompromised or have other predisposing factors including recent thoracic intervention or trauma^{4, 5}.

GBS-related infections of cardiac structures are rare, as a whole, with 2 to 3% of cases presenting as native valve endocarditis and far less as pericarditis, mycotic aneurysms and intraventricular abscesses³. Parikh *et al* reviewed the types of microorganisms isolated from purulent pericarditis samples and revealed that only 5% were due to streptococcal organism, *sans Streptococcus Pneumoniae*, possibly less so due to GBS⁶. Our literature search revealed only one case of GBS-related purulent pericarditis reported although the case was not linked in any way to a NSTI to our knowledge⁷.

Our case was unique as, at the time of writing, there were no other reports of GBS-related lower limb NSTI in combination with mediastinal involvement. There have been only a handful of cases of necrotising fasciitis reported with mediastinal involvement, the majority of which were supra-diaphragmatic with only one reporting NSTI of the lower limb due to *Aspergillus* infection^{8, 9}.

The similarity in culture results obtained from blood, tissue aspirate and the pericardial fluid in our patient suggest dissemination of GBS from the NSTI, possibly via a haematogenous route, although bacterial-related pericardial

dissemination can also occur via direct spread from infected foci from neighbouring intra-thoracic structures or sub-diaphragmatic⁴. The possibility of multi-routed spread should remind clinicians that, albeit rare, mediastinal involvement in NSTI is a possible complication of such disease.

Conclusion

This case highlights the rare possibility of cardiac involvement in cases of NSTI, and the possibility of cardiac tamponade causing cardiogenic shock masquerading alongside septic shock. It also highlights the importance of combining clinical findings with ancillary testing, including bedside echocardiography, when faced with challenging cases of sepsis to help look for possible foci of infection.

Competing Interests

None declared

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