CASE REPORT

Group G streptococcal sepsis, septic arthritis and myositis in a patient with severe oral ulcerations

Wu Deng,¹ Laurie Farricielli²

¹University of California, Irvine, California, USA

²Department of Internal Medicine, Scottsdale Health Care, Shea Hospital, Scottsdale, Arizona, USA

Correspondence to

Dr Laurie Farricielli,
lauriefarricellimd@gmail.com

SUMMARY

Lancefield group G streptococci (GGS) are a relatively less common cause of streptococcal infections but the incidence of which has been reported to increase in the recent years. Similar to group A streptococci, GGS produce localised and invasive infections. Streptococcal myositis is a very rare but highly fatal infection of muscles generally caused by group A streptococci. We report a case of sepsis, migrating septic arthritis and diffuse myositis caused by β -haemolytic GGS. It is an unusual case of diffuse β -haemolytic GGS myositis involving multiple muscle groups in a patient who demonstrated no skin lesions or sign of streptococcal toxic shock syndrome. The patient responded well to intravenous antibiotics without surgical intervention and experienced full recovery.

BACKGROUND

Streptococcal infections are very common in clinical practice. Although Lancefield group A streptococci (GAS) and group B streptococci are the most common cause of streptococcal infections, multiple studies have reported that group G streptococci (GGS) share many microbiological characteristics with GAS and produce similar invasive infections.^{1 2}

Despite being carried by a variety of animals and birds, GGS have been identified as part of the normal flora of human skin, oral cavity, nasopharynx, gastrointestinal tract and vagina.¹ It has been widely accepted that group G species generally cause opportunistic and nosocomial infections in patients with underlying medical conditions.^{3 4} However, animal exposure, as a cause of GGS infection, has also been reported.⁵

We report a case of GGS sepsis, diffuse myositis and septic arthritis in a patient with severe oral ulcerations of unclear aetiology. Streptococcal myositis is a very rare but fatal diffuse muscle infection that is generally caused by GAS.⁶ This case unusually presented with diffuse β -haemolytic GGS myositis involving multiple muscle groups, and there were no skin lesions or signs of streptococcal toxic shock syndrome (STSS), which were always present in the previously reported cases of streptococcal myositis. The patient responded to intravenous antibiotics without surgical debridement.

CASE PRESENTATION

A 65-year-old Caucasian man was admitted to our hospital with fever, shaking chills, general weakness, back pain, generalised myalgias and

polyarthralgias. The patient developed acute onset of pain in the right hip and thigh while at work 3 days prior to the admission, which was associated with general weakness, lethargy, dry cough and subjective fever. The next day he experienced pain in his left hip and left shoulder and improving pain in the right hip. The patient went to an emergency care and was treated with oseltamivir based on a positive influenza A nasopharyngeal swab. Twelve hours later, he developed progressive pain in his right shoulder and back, generalised myalgias, fever, shaking chills, nausea, vomiting and continued weakness. The patient also reported pain in his mouth and odynophagia. The medical history included well-controlled hypertension, hyperlipidaemia, non-obstructive coronary artery disease, atrial fibrillation with successful cardioversion, gastro-oesophageal reflux disease, partial thyroidectomy, bilateral inguinal hernia repair and appendectomy. He admitted taking 1–2 alcoholic drinks daily and occasionally smoked a pipe. He denied a history of recurrent infections, sexual transmitted diseases, joint disease, animal contact or intravenous drug use.

On admission, his blood pressure, heart rate, oral temperature and respiratory rate were 144/79 mm Hg, 77/min, 38.4°C and 25/min, respectively. The physical examination revealed dry oral mucosa and a swollen tongue. On deeper inspection, multiple oral ulcers were noted. There was severe tenderness and decreased range of motion of bilateral hips, spine, acromioclavicular and glenohumeral joints and tenderness to deep palpation of knees and paraspinal muscles. No lymphadenopathy or generalised rashes were found. Laboratory tests revealed white cell count $1.3 \times 10^3/\mu\text{L}$ with 87.3% neutrophils, platelets $132 \times 10^3/\mu\text{L}$, 622 IU/L creatine kinase (CK), CK-MB and troponin-1 within normal range, serum protein 5.7 g/dL, 2.5 g/dL albumin, 30.5 mg/dL C reactive protein and erythrocyte sedimentation rate 85 mm/h. The urine analysis and *Influenza* nasopharyngeal swab were negative. Screening for gonorrhoea, *Chlamydia*, HIV, rheumatoid arthritis, antinuclear antibodies, teichoic acid antibodies and antistreptolysin O were all negative; no evidence of pulmonary infiltrate or infectious endocarditis was found. Two separate blood cultures grew β -haemolytic group G *Streptococcus*. An indium-111 white cell count scan was performed, which showed an increased uptake at shoulders and faint white cell count accumulation at the anterior aspect of his right distal femur. MRI showed marked

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periarticular cellulitis of bilateral shoulders involving the anterior, dorsal and posterior soft tissues, myositis involving bilateral trapezius muscles and the left deltoid muscle, bilateral small joint effusion in the acromioclavicular and glenohumeral joints, pronounced subdeltoid bursitis of the right shoulder, extensive inflammation of the iliopsoas and dorsal paraspinal musculature extending from the thoracic spine into the upper sacral region. The representative images were shown in figure 1. Fluoroscopically guided aspiration of synovial fluids was performed and yielded cloudy, purulent fluids from the left hip, right glenohumeral and right acromioclavicular joints. The analysis of synovial fluid of the left hip and right glenohumeral joints showed white cell count of 11 333 and 128 750/mm³, red blood cell count 556 and 28 000/mm³, respectively, the Gram stain showed a few Gram-positive cocci in clusters. Culture of the right acromioclavicular joints grew β -haemolytic group *G Streptococcus*. The synovial fluid cultures of other joints were negative. No crystals were found.

The patient had worsening odynophagia and decreased oral intake the second day after admission. In order to identify the aetiology of oral ulcerations and odynophagia, oesophagogastroduodenoscopy was performed, which showed a hiatal hernia and Barrett's oesophagus. No infectious aetiology was found. The patient refused further invasive workups.

TREATMENT

The patient was treated empirically with vancomycin on admission and later switched to intravenous ceftriaxone for 6 weeks.

OUTCOME AND FOLLOW-UP

Following the antibiotic treatment, the patient experienced a significant improvement in joint pain and healing of oral ulceration. The outpatient follow-up of the complete blood count and indium-111 white cell count scan after 6 weeks of the treatment showed no evidence of inflammation.

DISCUSSION

Although GAS has been the most common cause of streptococcal infections, multiple studies have reported an increasing incidence of GGS infection in the recent years.⁷ GGS has been known to share many microbiological similarities with GAS and cause similar infections including pharyngitis, skin and soft tissue infections (SSTIs), meningitis, respiratory tract infection, septic arthritis, endocarditis and sepsis.⁷ Most of the patients with blood stream GGS infections had underlying medical conditions, the most common being malignancy, immunocompromised state, cardiovascular disease, diabetes mellitus, cirrhosis, alcoholism, bone and joint disease.⁴

Figure 1 T2 fat suppressed MRI of shoulders and spine. (A) The axial image of the right shoulder shows diffuse oedema, myositis and fluid collection. (B) The coronal image of the left shoulder showing diffuse oedema involving the trapezius muscle and other soft tissues. (C) The sagittal image of spine showing diffuse inflammation in dorsal paraspinal musculature.

Several clinical studies have investigated the SSTIs caused by GGS. Compared with GAS, GGS are more frequently associated with skin lesions.⁸ *Streptococcus canis* and *Streptococcus dysgalactiae* subsp. *equisimilis* (SDSE) are the species of GGS family that commonly cause SSTIs. A retrospective study of 54 patients with *S canis* infection showed that 64.8% of the patients presented with SSTIs, mainly on the lower limbs.⁹ Multiple clinical studies have indicated that the primary presentation of SDSE infections are SSTIs, including pyoderma, cellulitis, wound infections, abscesses, erysipelas and necrotising fasciitis.^{1 10–12} The invasive SSTIs, such as necrotising fasciitis, are commonly complicated by STSS.^{1 2}

Streptococcal myositis, which is characterised by muscle necrosis without abscess formation or primary inflammation of subcutaneous tissue and skin, is a rare but highly fatal infection of the muscle caused by invasive GAS.⁶ Only a few cases of GGS myositis were reported in the literature. Four reported cases presented with a local abscess and/or localised pyomyositis.^{2 13 14} A case of diffuse streptococcal myositis, reported by Wagner *et al*,¹⁵ was a patient with diabetes who initially presented with local skin blisters, followed by erysipelas-like skin lesions, haemorrhagic bullae and STSS. Although muscle involvement was reported in two reviews of GGS infections, the presenting symptoms and signs were not reported.^{8 12} In this case, the patient demonstrated diffuse muscle damages, manifested by significantly elevated serum CK and extensive inflammatory MRI signals within multiple muscle groups. Interestingly, this patient demonstrated no skin lesions or STSS, which were present in a previously reported case. Moreover, the myositis of this patient responded to intravenous antibiotics without surgical debridement, which is generally needed for the treatment of myositis.⁶ Although the pathogenesis of streptococcal myositis has not been fully understood, previous studies have suggested that the deep tissue invasion of *Streptococcus* might be attributable to some virulence factors such as streptolysins, fibrinolysins and streptococcal pyrogenic exotoxins.^{2 15} We postulate that certain virulent factor(s) produced by the GGS strain of this patient could be related to his unusual presentation. Future study will focus on further characterisation of the GGS strain isolated from this patient.

Recently, disruption of the cutaneous barrier has been found to be a very common predisposing factor for GGS bacteraemia.⁷ One study reported that, in patients with GGS bacteraemia, skin is the suspected path of entry in 76%, gastrointestinal tract 8.1%, urogenital tract 5.2% and unknown 10.7%.¹⁰ Another study identified that 56% of patients who developed GGS bacteraemia related to underlying malignancies had a certain type of skin or gastrointestinal tract disruption, suggesting that breach of the epithelial barrier of skin or mucosa is important for invasion of GGS.¹⁶ In the present case, the patient had no evidence of skin disruptions or underlying malignancy except for ulcerations in the mouth, where there is known GGS colonisation. We consider the oral ulcerations as the most likely port of entry of GGS bacteraemia. The oral herpes infection has been associated with streptococcal bacteraemia.¹⁷ We were not able to identify the aetiology of the oral ulcers of this case because the patient refused the biopsy and culture of the ulcers. The patient also had cardiovascular disease, which is a known predisposing condition for invasive GGS infection.

Most of the GGS strains are sensitive to penicillins, vancomycin, erythromycin and cephalosporins.¹⁸ The patient was empirically treated with vancomycin and then switched to ceftriaxone base on the sensitivity study. Although this patient had bacteraemia and severe septic infections involving multiple

joints and muscles, the treatment protocol for STSS was not initiated given his stable haemodynamic status. Clindamycin, a lincosamide antibiotic that inhibits bacteria protein synthesis by binding to the 50S subunits of ribosome, has been shown to inhibit the synthesis of streptococcal virulence factors including exotoxin A, exotoxin B and M protein.^{19–21} Clindamycin in combination with β -lactam antibiotics has been recommended for the management of STSS and invasive streptococcal infections.^{22 23} The effectiveness of clindamycin has been reported in the treatment of GGS infection.²⁴ Although the patient responded well to the treatment with ceftriaxone alone, clindamycin in combination with ceftriaxone could be another treatment regimen of choice.

Learning points

- ▶ Diffuse myositis involving multiple muscle groups is a rare clinical manifestation in patients with group G streptococci (GGS) sepsis and septic arthritis.
- ▶ Diffuse myositis in this case responded to intravenous antibiotics without surgical intervention.
- ▶ Disruption of the mucous membrane by oral ulceration might possibly be a potential port of entry of GGS bacteraemia.

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Contributors WD was collected and analysed the patient data, and was a major contributor in writing the manuscript. LF was the primary care physician of the patient and supervised the information collection, data interpretation and manuscript preparation.

Competing interests None.

Patient consent Obtained.

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