CASE REPORT

A rare case of pyogenic pericarditis secondary to *Streptococcus constellatus*Sehem Ghazala,¹ Todd Rabkin Golden,² Sumaya Farran,³ Tirdad T Zangeneh³¹Department of Internal Medicine, University of Arizona, Tucson, Arizona, USA²Department of Medicine, University of Arizona College of Medicine, Tucson, Arizona, USA³Infectious Diseases, The University of Arizona College of Medicine, Tucson, Arizona, USA

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Accepted 16 March 2018

SUMMARY

We report an extremely rare case of purulent pericarditis caused by the normally commensal oral flora, *Streptococcus constellatus*, a viridans *Streptococcal* species and member of the *S. anginosus* group (previously also known by the eponymous '*S. milleri*', for American Willoughby Dayton Miller). This case is a previously healthy 71-year-old immunocompetent woman from Arizona who presented with a 5-day history of progressive shortness of breath and chest tightness, and subjective fever and chills, but without history of nausea, vomiting, night sweats, recent travel, autoimmune disease or sick contacts. Early recognition and intervention of purulent pericarditis allow patients like the one outlined in this case to achieve full recovery.

BACKGROUND

We report a very rare case of purulent pyogenic pericarditis caused by *Streptococcus constellatus* in an immunocompetent patient. The *S. anginosus* groups (SAGs), including *S. constellatus*, are normally part of the commensal mucosal flora of the oropharynx, gastrointestinal and genitourinary tracts; however, more recently recognised, it can rarely be pathogenic in a variety of body sites.

CASE PRESENTATION

A 71-year-old woman from Arizona with no medical history presented after 5 days of progressive shortness of breath and chest tightness, both of which worsened while lying down. She reported subjective fever and chills but denied night sweats, recent travel or sick contacts. There was no history of autoimmune disease. She denied weight loss, nausea, vomiting, urinary symptoms or abdominal pain. On presentation, her temperature was 37.2°C, pulse was irregularly irregular at 123 beats/min, blood pressure 105/56 mm Hg, respiratory rate 22/min and oxygen saturation 93% on room air. Physical examination was significant for elevated jugular venous pressure, decreased breath sounds bilaterally, irregular tachycardia with distant heart sounds, and there was no peripheral oedema.

INVESTIGATIONS

ECG showed atrial fibrillation with rapid ventricular response and was without ST abnormality. Chest X-ray was significant for bilateral pleural effusions. Bedside ultrasound indicated possible pericardial effusion, and formal echocardiogram revealed a small pericardial effusion with

left ventricular ejection fraction of 55% with no definite criteria for tamponade (figure 1). The patient was started on a heparin drip and metoprolol. Complete blood count was significant for leucocytosis of $14.7 \times 10^9/L$ with an elevated absolute neutrophil count. Electrolytes and renal function were normal. Troponins were negative. C reactive protein was elevated at 30.54 mg/dL (<0.6); erythrocyte sedimentation rate was 86 mm/hour (0–30) and ferritin 1184 ng/dL (5–204). CT demonstrated bilateral pleural effusions and pericardial effusion (figure 2), without findings of pneumonia. The patient underwent left-sided thoracentesis yielding exudative fluid with a pH of 7.45, which was negative for cytology, adenosine deaminase, gram stain and cultures. She developed chest pain and dyspnoea and a limited echo showed increased pericardial effusion, without evidence of tamponade. Significantly, a broad infection, including blood cultures and autoimmune workup, was negative. However, it is important to mention that blood cultures were obtained after the administration of antibiotics. Subsequent daily limited echocardiography showed further increase in pericardial effusion, and pericardiocentesis of 300 mL cloudy fluid was performed via a subxiphoid approach with a pigtail catheter placement that was removed after 36 hours, which improved the patient's shortness of breath and chest pressure. Her heart rate was converted to sinus with cardioversion after ruling out left atrial appendage thrombus via transoesophageal echocardiogram followed by treatment with amiodarone and metoprolol, after which the patient reported significant improvement in her symptoms. Interestingly, the pericardial fluid, negative by cytology, grew *S. constellatus*, sensitive to ceftriaxone. She underwent transoesophageal echocardiography that was negative for valvular vegetation. Given *S. constellatus* is a known oral flora, CT of the face was obtained which revealed a 1 cm periosteal abscess at the left maxillary second molar (figure 3). We believe that the infection was haematogenous spread from the periosteal abscess. However, other mechanisms include direct spread through the retropharyngeal space.

DIFFERENTIAL DIAGNOSIS

- Autoimmune pericarditis.
- Viral pericarditis.
- Tuberculosis.

To cite: Ghazala S, Golden TR, Farran S, et al. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-223804

Figure 1 Echocardiogram revealed a small pericardial effusion.

OUTCOME AND FOLLOW-UP

The patient was discharged home on ceftriaxone for a total of 4 weeks and outpatient follow-up with infectious disease, cardiology and dentistry. At 4 weeks' follow-up, the patient reported a full resolution of symptoms, and her echocardiogram was normal without evidence of pericardial effusion.

DISCUSSION

Members of the *Streptococcus* genus are gram-positive, non-motile, non-sporeforming, catalase-negative cocci that occur in pairs or chains. *S. viridans* is a heterogeneous group of species that are normally commensal flora of the oropharynx as well as the upper respiratory, gastrointestinal and female genital tracts.¹ *Viridans* (from Latin for green, *viridis*) refers to the green discolouration of blood agar produced by alpha haemolysis, and modern classification (on the basis of the 16S rRNA gene sequences) separates these species into five groups: *mitis*, *mutans*, *salivarius*, *sanguinis* and *anginosus*.^{2,3}

The SAG (comprised three species: *S. anginosus*, *S. intermedius* and *S. constellatus*) are facultative, non-motile anaerobes

Figure 2 Chest CT demonstrated pericardial effusion.

Figure 3 Face CT revealed a 9.8 mm periosteal abscess at the left maxillary second molar.

with variable Lancefield group positivity (F, C, A and G in order of frequency) and may be alpha, beta or non-haemolytic, with non-beta-haemolytic strains more common.^{3,4} This group has pathogenic potential to contribute to a variety of human infections including soft-tissue infections, deep tissue abscesses and pleuropulmonary infections, with extension to surrounding tissues.^{5–7} Invasive SAG infection has been described in patients of all ages with higher risk associated with alcohol abuse and male sex in some studies.^{8–12} Pooled data of seven studies totaling 215 patients with SAG bacteraemia found an all-cause mortality rate of 16%.⁵ Of all invasive pyogenic streptococcal disease 40%–50% may be associated with SAG.¹¹ Unique among *viridans Streptococci*, SAG is associated with serious pyogenic infection and abscess formation particularly of the brain, liver, abdomen and lungs, but has rarely been described as a cause of purulent pericarditis; however, purulent pericarditis secondary to SAG is rapidly life threatening.^{13–17} Only 20 cases of pericardial infection secondary to SAG have been reported worldwide, all in the past four decades.^{17,18} SAG pathogenicity is not fully understood but includes cell surface adhesins, exotoxin production, evasion of phagocytosis and anaerobic organism synergism in polymicrobial infections.^{3,19} SAG polymicrobial abscesses are often coinfecting with *Eikenella* and *Fusobacterium* species in the head and neck, and *Enterobacteriaceae* and *Bacteroides* species within the abdomen; however, most abdominal cultures are polymicrobial while most head and neck cultures grow only SAG.^{9,16,19} Broad-spectrum beta-lactams are frequently used to treat polymicrobial abscesses with several weeks of intravenous therapy often needed and additionally, drainage of SAG abscesses should be attempted when appropriate.^{20–22} Also, key to the management of SAG infection is investigation to locate the primary abscess as well as secondary sites.²³

The cornerstones of treatment for purulent pericarditis include early recognition, pericardial fluid drainage and appropriate antibiotics with broad antibiotic coverage until fluid cultures are reported as well as critical care support, and additional intervention may be needed with loculation.^{17 18} Since the introduction of antibiotic therapy decades ago, purulent pericarditis has become uncommon; however, prognosis is poor without early diagnosis and treatment as it may lead to cardiac tamponade and haemodynamic collapse, and its mortality rate may approach 100%, with diagnosis most often made at autopsy.^{17 24–28} Predisposing factors for pericardial infection include chest trauma or surgery, pre-existing pericardial disease, uraemia, collagen vascular disease, alcohol abuse, malignancy and immune suppression.²⁹ In addition to *Streptococcus*, purulent pericarditis is commonly caused by *Staphylococcus*, *Pneumococcus*, *Haemophilus* and *Mycobacterium tuberculosis*.³⁰ Many of the recently reported cases of purulent pericarditis have occurred in individuals who, unlike our patient, were immunocompromised.^{31 32}

It is clinically significant to identify *S. viridans* to the species level, yet until the 2000s this did not occur due to confusion regarding classification and identification, non-automated laboratory techniques at the time and low awareness of this clinical significance, as well as less antibiotic resistance concerns.^{3 5 9 33–35} Therefore, there is currently very little data published on this topic, yet SAG has been repeatedly described as important yet elusive and unappreciated.^{35–37}

Learning points

- *Streptococcus constellatus*, along with *S. anginosus* and *S. intermedius* the other two species constituting the *S. anginosus* group, is normally a commensal mucosal flora; however, when pathogenic, it has a propensity for serious pyogenic infection and abscess formation in the lung, brain and abdominal organs.
- Once identified, *S. anginosus* group infection management warrants investigation of a primary abscess and additional sites.
- Identification of *S. viridans* to the species level is of clinical significance as an understanding of its prevalence and pathogenicity becomes elucidated, and there are no longer technological and taxonomy barriers hindering this identification.

Contributors SG: primary care of patient, wrote hospital course and consented patient. TRG: wrote the discussion. SF: helped in writing and/or editing the article. Also, consultant service (ID). TTZ: attending on service, played a role in manuscript revision and supervision of the case.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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