CASE REPORT

Successful treatment of community-acquired methicillin-resistant *Staphylococcus aureus* purulent myopericarditis

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Accepted 29 September 2017

SUMMARY

A previously healthy 48-year-old active duty man, who had been treated for an elbow abscess 3 weeks earlier, presented to an emergency department in Bahrain with tachycardia, pericardial friction rub and jugular venous distention. Cardiac tamponade was confirmed on transthoracic echocardiogram and he was taken for emergent pericardiocentesis. Pericardial fluid cultures grew community-acquired methicillin-resistant *Staphylococcus aureus*. Despite ongoing treatment with intravenous vancomycin, he developed a recurrent fibrinous pericardial effusion and constrictive pericarditis requiring pericardiectomy. Though he initially did well postoperatively, he developed drug reaction with eosinophilia and systemic symptoms syndrome in response to vancomycin. He was transitioned to ceftaroline and started on high-dose steroids. He recovered during a week-long admission and was discharged home. Several weeks later at follow-up he was doing well and had resumed moderate intensity exercise.

BACKGROUND

Purulent pericarditis is an infection of the pericardial space associated with pus found on gross examination or tissue microscopy and most commonly occurs in the context of bacterial pneumonia or empyema.¹ Bacterial purulent pericarditis is rare, encompassing <1% of pericardial disease, is often complicated by pericardial constriction and cardiac tamponade, and is associated with significant mortality.^{1,2} Classically described mechanisms of disease transmission include contiguous extension of intrathoracic processes, haematogenous spread, penetrating chest wall injury, surgical wounds, oesophageal rupture with fistula formation, retropharyngeal abscess and seeding from hepatic/subdiaphragmatic abscesses.¹⁻⁴ In one western case series, the most common causative organisms cultured were staphylococci, streptococcus and pneumococcus.³ Community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) purulent pericarditis appears to be even more uncommon having been described in a few case reports. Here we present a case of CA-MRSA purulent myopericarditis successfully managed with ceftaroline and pericardiectomy.

CASE PRESENTATION

A previously healthy 48-year-old active duty man presented to an emergency department in Bahrain with intermittent fevers, dyspnoea, positional chest pain and reported a history of several syncopal episodes. Three weeks prior, he had been treated for an elbow abscess with an oral antibiotic and incision and drainage. He denied any history of medical problems, was not taking any medication and had no known allergies. He also denied any illicit drug use or risky sexual behaviour. Notable physical findings included tachycardia to 120 beats/min, a pericardial friction rub, jugular venous distention and dullness to percussion over the left subscapular area, also known as Ewart's sign. Laboratory examination revealed an elevated troponin level of 3.26 ng/mL, a white cell count of $13 \times 10^3/\text{mm}^3$ and a C-reactive protein level of 49.3 mg/L. A chest X-ray revealed an enlarged cardiac silhouette in the shape of a flask, also known as a 'water bottle heart'. A subsequent transthoracic echocardiogram (TTE) revealed a large pericardial effusion with the diastolic collapse of the right ventricle, respiratory ventricular interdependence and inferior vena cava dilatation with the loss of respiratory variation, confirming a diagnosis of cardiac tamponade. He underwent emergent pericardiocentesis, with 750 mL of grossly purulent fluid removed, and was admitted to the intensive care unit. Pericardial fluid and blood cultures grew MRSA with the isolate sensitive to minocycline, moxifloxacin, sulfamethoxazole/trimethoprim, clindamycin, rifampin, vancomycin, linezolid and mupirocin. Screenings for HIV, tuberculosis and several other infectious and inflammatory aetiologies were negative. He was started on intravenous vancomycin, but in transition to the USA, he developed recurrent fevers and tachycardia. A repeat TTE showed a reaccumulated pericardial effusion with dense fibrinous loculations and concurrent constrictive pericarditis. He was taken for total pericardiectomy where surgery revealed that the pericardium was grossly oedematous and densely adherent to the heart with visible purulent pockets over the left lateral portion. The entire pericardium was successfully removed. Postoperatively he required intra-aortic balloon pump, high-dose epinephrine, norepinephrine and vasopressin. He recovered well and was discharged home after 1 week.

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To cite: DeYoung H, Bloom A, Tamayo S. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-221931

Figure 1 Above is a single sagittal cardiac MRI image from delayed enhancement sequence at the cardiac apex. There is increased subepicardial enhancement that is indicative of a myocarditis secondary CA-MRSA. Follow-up cardiac MRI performed 5 months after discharge showed complete resolution of subepicardial enhancement. AR, anterior right; CA-MRSA, community-acquired methicillin-resistant *Staphylococcus aureus*; H, heat; F, foot; PL, posterior left.

After an additional week at home, he developed a rash and dyspnoea prompting re-evaluation. He was found to have a morbilliform rash, facial oedema, eosinophilia and elevated liver-associated enzymes concerning for drug reaction with eosinophilia and systemic symptoms (DRESS) syndrome and concurrent acutely decompensated heart failure. He was started on high-dose oral prednisone, intravenous furosemide and transitioned to intravenous daptomycin. His rash and eosinophilia worsened, requiring a change from daptomycin to intravenous ceftaroline. Right and left cardiac catheterisations revealed an occluded obtuse marginal branch of the circumflex artery. Mildly elevated right ventricular filling pressures in a dip-and-plateau pattern concerning for residual fibrosed pericardium were also seen. His rash, eosinophilia and dyspnoea improved dramatically over the next 6 days, and no bacteraemia was detected on blood cultures throughout his admission. He was discharged with a peripherally inserted central catheter for receipt of intravenous ceftaroline for three additional weeks. He was also continued on furosemide and a prednisone taper for five additional weeks, and was started on carvedilol, lisinopril, atorvastatin and aspirin.

Page 4 of 12 shortly after discharge, a cardiac MRI revealed that his left ventricular ejection fraction was 30% with subepicardial enhancement inferiorly towards the apex, suggestive of likely CA-MRSA-induced myocarditis (figure 1). Transmural enhancement from the base to the inferolateral wall was also detected, consistent with a previous undiagnosed myocardial infarction.

After completing courses of ceftaroline and prednisone he reported improving symptoms. Five months later, cardiac MRI revealed the absence of the subepicardial enhancement and his ejection fraction had improved to 47%. He had also resumed moderate intensity exercise.

INVESTIGATIONS

Complete blood count, complete metabolic panel, troponins, blood cultures, pericardial fluid cultures, chest X-ray, TTE and cardiac MRI.

TREATMENT

Pericardiocentesis, pericardiectomy, ceftaroline 600mg every 6 hours and prednisone 60 mg each day.

OUTCOME AND FOLLOW-UP

The patient received ceftaroline at home for four total weeks along with prednisone taper. A cardiac MRI at follow-up showed complete resolution of previous subepicardial enhancement concerning CA-MRSA myocarditis.

DISCUSSION

We believe that the patient described in this case acquired purulent pericarditis through haematogenous spread, one of the mechanisms of acquisition previously described. One of the unique aspects of this case is that the patient was entirely healthy prior to infection. Based on his history and predeployment workup, he did not have any medical conditions that would predispose to dissemination of CA-MRSA and the development of purulent myopericarditis.

While the majority of previous cases describe the use of vancomycin for CA-MRSA purulent pericarditis, this is only the second case where the patient was successfully treated with ceftaroline. In comparison, this case is unique in that the patient developed DRESS syndrome in response to vancomycin requiring transition to ceftaroline, rather than oxacarbazepine.⁵ Ceftaroline is a fifth-generation cephalosporin with a strong affinity for penicillin-binding protein 2a (PBP2a), a modified penicillin-binding protein in MRSA, providing its spectrum of activity against MRSA. Ceftaroline is FDA approved for the treatment of MRSA skin and soft tissue infections and bacterial pneumonia. Ceftaroline use has also been described in pneumonia, endocarditis, osteomyelitis and bacteraemia, and once in a similar case report mentioned previously for the treatment of MRSA purulent pericarditis after the development of DRESS syndrome.⁵⁻¹⁰

Other previous cases of CA-MRSA purulent pericarditis describe patients with ages ranging from 19 to 60 years. The majority of these cases had patients with ongoing predisposing conditions including pseudoaneurysm, obesity, diabetes mellitus type 2, cocaine use, non-small cell lung cancer and DRESS syndrome. Sources included skin or soft tissue infections and community acquired pneumonia. Antibiotic treatment most commonly involved vancomycin, though others included gentamicin, meropenem, rifampin, amikacin and one case with ceftaroline. Pericardiocentesis was performed in five of seven cases and pericardial window and pericardiectomy were performed in two separate cases.^{8 11-16}

Purulent pericarditis often presents with only fever or with fulminant decompensated sepsis and always presents acutely. Other commonly reported symptoms include chills and tachycardia. Symptoms such as chest pain, pericardial friction rub, orthopnoea, oedema and hypotension have been reported, but are less common. Isolated purulent pericarditis is rare, mostly commonly reported in patients with mixed connective tissue disease, renal failure, alcoholism, immunosuppression, malignancy and HIV.^{1 2} Aggressive management is necessary as morbidity and mortality are profound if untreated. When treated, however, approximately 85% of patients survive. Bacterial infection in conjunction with cardiac complaints necessitates

immediate empiric intravenous antibiotics. Though TTE and pericardiocentesis with pericardial fluid cultures are essential for diagnosis and the treatment of cardiac tamponade, these interventions should not delay the administration of antibiotics. Visualising frank purulence is nearly 100% sensitive, and associated with increased risk of recurrence, further emphasising the need for prompt and aggressive management. Pericardial fluid samples should be sent for (1) Gram Stain; (2) acid-fast bacillus stain; (3) fungal stain; (4) aerobic, anaerobic and fungal cultures; (5) complete cell count with differential fluid analysis for glucose, protein content, lactate dehydrogenase levels and adenosine deaminase levels.^{2 17 18}

Adequate drainage for purulent pericarditis is vital in order to normalise haemodynamics and achieve source control. Reaccumulation of pericardial fluid is not uncommon, as seen in this case and requires re-evaluation with TTE and likely pericardiocentesis. The development of loculations and viscous fibrin deposition or constrictive pericarditis make treatment challenging. Fibrin deposition may be treated with intrapericardial infusions of streptokinase or urokinase in conjunction with systemic corticosteroids to lyse loculations.^{2 19 20} This can be performed with placement of percutaneous catheter, pericardiectomy and placement of pericardial catheter or in conjunction with pericardiectomy. Similarly, and as highlighted in this case, constrictive pericarditis may dictate the need for pericardiectomy.^{1 2}

In conclusion, purulent pericarditis is a life-threatening condition that requires prompt recognition and treatment. It is imperative that patients with cardiac-related complaints in conjunction with bacterial infection receive immediate antibiotics and further investigation with a TTE. Lastly, in patients who cannot tolerate

vancomycin or daptomycin, ceftaroline may be a viable treatment option.

Contributors All authors: conception, design, analysis and interpretation of data; revised it critically for important intellectual content; read and approved the final manuscript to be published. HDY: drafted the article.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- Methicillin-resistant *Staphylococcus aureus* (MRSA) skin and soft tissue infections may have serious implications if ignored or treated inappropriately.
- Purulent pericarditis is a rare, but should be considered in a patient presenting with signs of infection and cardiac complaints.
- MRSA purulent pericarditis is very challenging to treat due to MRSA's ability to form fibrinous deposits and loculations.
- Patients should be screened for recurrent effusions. If constrictive pericarditis develops patients may require pericardiectomy.
- Ceftaroline may be an effective alternative to vancomycin or daptomycin in the treatment of purulent pericarditis, especially if there is a concern for the development of drug reaction with eosinophilia and systemic symptoms syndrome.

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