CASE REPORT

From bone to heart: a case of MRSA osteomyelitis with haematogenous spread to the pericardium

Parinita A Dherange,¹ Sarah Patel,¹ Evbu Enakpene,² Prakash Suryanarayana²

¹Department of Internal Medicine, Banner University Medical Center South, Tucson, Arizona, USA

²Department of Cardiology, University of Arizona, Tucson, Arizona, USA

Correspondence to

Parinita A Dherange,
parinita.dherange@
bannerhealth.com

Accepted 18 November 2015

SUMMARY

We report a case of a 55-year-old woman with a history of type 2 diabetes mellitus, Charcot arthropathy and end-stage renal disease, who presented with a syncopal episode after undergoing haemodialysis. She had a history of methicillin-resistant *Staphylococcus aureus* (MRSA) bacteraemia from an unknown source 3 months earlier, which was treated with an 8-week course of intravenous antibiotics. At the time of presentation to the emergency room, she was found to be in refractory shock. Bedside echocardiogram was performed, which showed moderate pericardial effusion. The effusion was later found to be due to MRSA, which was identified in blood and in pericardial fluid cultures. The patient was successfully treated with intravenous daptomycin for 6 weeks. Acute osteomyelitis of her right foot was the source of the MRSA, for which a right below-knee amputation was ultimately performed.

BACKGROUND

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a pervasive organism that causes life-threatening illnesses.¹ Although there are many cases of skin and soft tissue infections secondary to MRSA, there are only five described cases of purulent pericarditis due to MRSA, and these are in the paediatric literature.^{2,3} Purulent pericarditis is an accumulation of purulent fluid within the pericardial space.⁴ We describe a case of refractory septic shock with pericardial involvement, resulting from MRSA osteomyelitis.

CASE PRESENTATION

A 55-year-old woman was brought to the emergency department after a syncopal episode. Her medical history was significant for type 2 diabetes mellitus, end-stage renal disease requiring maintenance haemodialysis and Charcot arthropathy. Three months prior to the current admission, she was treated for MRSA bacteraemia from an unknown source, with an 8-week course of intravenous daptomycin.

During the index admission, the patient was brought to the emergency room, with syncope. On further evaluation, she was noted to have a temperature of 96.4°F, heart rate of 122 bpm and blood pressure of 96/61 mm Hg. Physical examination was significant for muffled heart sounds and diffuse crackles in bilateral lung fields. She also had local signs of inflammation, including swelling, redness, warmth and tenderness in her right lower extremity, however, she had no signs of inflammation at the site of her arteriovenous (AV) fistula. Thus, the most likely source of infection was the

right lower extremity rather than the AV fistula. She was given intravenous fluids and started on antibiotics. She was then transferred to the intensive care unit, for a higher level of care.

INVESTIGATIONS

Blood tests revealed white cell count of $25.1 \times 10^9/L$ (normal value $3.4\text{--}10.4 \times 10^9/L$) and C reactive protein of 279.2 mg/L (normal value 0.2–5 mg/L).

An initial diagnosis of septic shock due to an infective focus in the right foot was made. The patient was started on vancomycin, linezolid and ceftaroline for the MRSA coverage, given the prior history of MRSA bacteraemia. She was adequately fluid resuscitated and started on norepinephrine, however, she continued to be in refractory shock.

A bedside echocardiogram was performed at this time, showing a large pericardial effusion. A detailed transthoracic echocardiogram performed later showed a moderate-sized pericardial effusion with pericardial fibrinous stranding but no evidence of cardiac tamponade (figures 1 and 2).

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of a hypotensive patient can be formulated by understanding the basic cardiovascular physiology. The first concept to remember is that $\text{pressure} = \text{flow} \times \text{resistance}$, where flow is cardiac output (CO), and resistance is systemic vascular resistance (SVR). Using this concept, presence of hypotension can be explained when at least one of these parameters (CO or SVR) is abnormal. Differential diagnosis for hypotension is shown in figure 3.

Sources of bacteraemia can be classified as intravascular (intravascular lines and endocarditis), gastrointestinal, respiratory, urinary, central nervous system, osteoarticular, skin and soft tissues or unknown.

Risk factors for MRSA infection include antibiotic use, HIV infection, haemodialysis and long-term care facilities.

TREATMENT

Given the haemodynamic compromise, an emergent pericardiocentesis was performed, which drained purulent fluid. A pigtail catheter was left in place and daily flushing with saline solution was performed. The surgical team was consulted. No further surgical intervention was recommended due to improving purulent pericardial effusion as seen on serial echocardiograms. The pigtail catheter placed in the pericardial space was removed after

CrossMark

To cite: Dherange PA, Patel S, Enakpene E, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-211410

Figure 1 (A) Parasternal long axis image demonstrating circumferential pericardial effusion (white arrows) measuring 1.3 cm during end diastole. Note the absence of collapse of RVOT region. (B) Subcostal view showing moderate-sized circumferential pericardial effusion during systole. (C) Zoomed in image of subcostal view demonstrating anteriorly located moderate-sized pericardial effusion with fibrinous strand (blue arrow). Also note prominent density in the AV groove region, most likely due to epicardial fat (black asterisk). (LV, left ventricle; RV, right ventricle; RVOT, right ventricular outflow tract).

5–7 days without any complications. Blood and pericardial fluid cultures were found to be positive for MRSA. This led to a search for the primary source of sepsis. MRI of the right ankle was obtained given the patient's history of Charcot arthropathy and also due to severe tenderness on palpation of the right ankle during examination. MRI revealed a massively distended synovial fluid complex with possible superimposed infection (figure 4). An orthopaedics consult was obtained and a bedside arthroscopy followed by incision and drainage was performed. Ultimately, a right below-knee amputation had to be performed. Wound biopsy and culture from the right fourth metatarsal revealed growth of MRSA and pathology confirmed acute osteomyelitis. The patient was initially started on ceftaroline for the MRSA infection, however, she developed thrombocytopenia, which was thought to be a side effect of ceftaroline. She was later discharged on intravenous daptomycin for 8 weeks.

OUTCOME AND FOLLOW-UP

The patient was discharged in a stable condition from the hospital to a skilled nursing facility. She completed a total of 8 weeks of daptomycin. She was noted to be doing well on follow-up

clinic visits. A transthoracic echocardiogram performed after completion of her antibiotics showed no pericardial effusion.

DISCUSSION

Pericarditis due to MRSA is rare, especially in the post-antibiotic era.⁴ Most cases of purulent pericarditis currently are associated with nosocomial bloodstream infections such as in the setting of haemodialysis, thoracic surgery, or immunosuppression. Our patient had diabetes mellitus with end-stage renal disease on chronic haemodialysis, and subsequently developed MRSA infection due to haematogenous spread causing the rare purulent pericarditis.

In a study from Spain, only 33 cases of 593 000 inpatient hospitalisations were reported as those of purulent pericarditis. In the pre-antibiotic era, the majority of patients with purulent pericarditis had a primary infection due to pneumonia or endocarditis. There were reported cases of purulent pericarditis due to bacteraemia, central nervous system infections and bone or skin infections.⁴

There are several different mechanisms by which purulent pericarditis may occur: direct spread from an intrathoracic aetiology (including contamination from trauma or thoracic surgery), haematogenous spread, or extension from a subdiaphragmatic source.⁵ Any organism can cause pericarditis. However, staphylococci and streptococci are most common.³ In the post-antibiotic era, many patients have a pre-existing pericardial disease due to uraemic, neoplastic, or collagen vascular disease.⁴ Conditions that increase the incidence of purulent pericarditis include alcohol abuse, immunosuppression and acute chest trauma.³

Purulent pericarditis typically presents as an acute illness characterised by high fever in virtually all patients whereas chest pain may be present in only 25–37% of patients.^{4 6} Other signs and symptoms include tachycardia, cough and pericardial friction rub. About 42–77% of the patients develop cardiac tamponade, which can lead to sudden cardiac decompensation.⁶ MRSA purulent pericarditis as an aetiology for refractory shock has a high mortality rate of up to 60%. Index of suspicion of staphylococcal pericarditis should be high in an immunosuppressive patient as was the case in our patient.

The diagnosis of purulent pericarditis has improved significantly with the widespread availability of two-dimensional echocardiography and CT. The diagnosis of purulent pericarditis

Figure 2 Doppler image of mitral inflow with respiration: There is no evidence of respirophasic variation to suggest cardiac tamponade (MV, mitral valve).

Figure 3 Differential diagnosis for hypotension (CO, cardiac output; CVP, central venous pressure; MBP, mean blood pressure; LV, left ventricle; RV, right ventricle; SVR, systemic vascular resistance).

Figure 4 End-stage Charcot arthropathy of the right ankle, hindfoot and midfoot, with an associated massively distended synovial fluid complex (white arrows) and possible superimposed infection.

needs to be established by obtaining pericardial fluid for culture and microscopy. This will help the clinician in instituting a pathogen-specific goal-directed therapy.

Primary source control represents a key component of successful treatment. Failing to identify the primary source of infection may lead to inadequate treatment and recurrent infections.

Survival in cases of purulent pericarditis rests on early empiric antibiotic therapy and pericardial drainage.⁷ In some patients, pericardiocentesis can result in poor drainage or loculations of thick pericardial fluid in the pericardial space. In such cases, subxiphoid pericardiotomy and pericardial window with manual lysis of adhesions and loculations allows complete drainage.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

Learning points

- ▶ Purulent pericarditis is usually secondary to complication of another infection and thus a high level of suspicion is needed to make a diagnosis.
- ▶ Methicillin-resistant *Staphylococcus aureus* purulent pericarditis-related refractory septic shock has a mortality rate of 60%.
- ▶ Index of suspicion for staphylococcal pericarditis should be high in an immunosuppressive patient.
- ▶ Rapid diagnostic attempts are important as diagnosis can provide source control, which may then result in decreased future complications.
- ▶ Completion of antibiotic therapy based on appropriate sensitivity studies is of great importance, and eventually helps in decreasing the mortality.

REFERENCES

- 1 Dhanoa A, Singh VA, Mansor A, *et al.* Acute haematogenous community-acquired methicillin-resistant *Staphylococcus aureus* osteomyelitis in an adult: case report and review of literature. *BMC Infect Dis* 2012;12:270.
- 2 Rallidis LS, Baroutsis KN, Pentzeridis PC. Successful treatment of staphylococcal pericarditis. *Hellenic J Cardiol* 2003;44:230–3.
- 3 Raju S, Kumar A, Joseph R, *et al.* Cardiac tamponade secondary to methicillin-resistant *Staphylococcus aureus* pericarditis. *Chest J* 2012;142:357A.
- 4 Sagrista-Sauleda J, Barrabes JA, Permanyer-Miralda G, *et al.* Purulent pericarditis: review of a 20-year experience in a general hospital. *J Am Coll Cardiol* 1993;22:1661–5.
- 5 Kang HR, Kim SR, Lee EJ, *et al.* Primary methicillin-resistant *Staphylococcus aureus* pericarditis in a patient undergoing hemodialysis. *Soonchunhyang Med Sci* 2014;20:42–4.
- 6 Rubin RH, Moellering RC. Clinical, microbiologic and therapeutic aspects of purulent pericarditis. *Am J Med* 1975;59:68–78.
- 7 Maisch B, Seferović PM, Ristić AD, *et al.* Guidelines on the diagnosis and management of pericardial diseases executive summary; The Task force on the diagnosis and management of pericardial diseases of the European society of cardiology. *Eur Heart J* 2004;25:587–610.

Copyright 2015 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow