

Pericardial abscess: The corollary of disseminated Methicillin-resistant *Staphylococcus aureus* following diabetic foot ulcer infection

Thai Lun Tan, MRCP¹, Soon Hooi Lim, MRCP¹, Mohd Ruslan Mustapa, Master Int. Med¹, Ganeswary Ramaloo, MBBS²

¹Department of Internal Medicine, Hospital Seri Manjung, Perak, Ministry of Health, Malaysia, ²Department of Radiology, Hospital Seri Manjung, Perak, Ministry of Health, Malaysia

SUMMARY

Methicillin-resistant *Staphylococcus aureus* (MRSA) purulent pericarditis, characterised by frank pus collection or microscopic pyogenic effusion in the pericardium represents the most serious form of pericardial infection. The route of MRSA acquisition in pericardial abscess commonly occurs via the blood stream infection and it is more commonly observed among immunocompromised individuals. To date, diabetic foot ulcer infection rarely disseminates and becomes a nidus for pericardial infection. Herein, we report an unusual case of MRSA pericardial abscess in a 44-year-old man who presented at Hospital Seri Manjung, Malaysia with cardiac tamponade. Past medical history indicated that he was recently treated for infected diabetic foot ulcer with MRSA bacteraemia one week earlier. Despite adequate pericardial drainage and extended parenteral vancomycin therapy, this case ended in fatality on day 42 of admission due to nosocomial infection. It is hoped that this report serves to increase the vigilance among clinicians that diabetic foot ulcer infections have the potential to progress to pericardial abscess in the presence of MRSA bacteraemia, although they may appear seemingly innocuous at presentation. Systemic vancomycin must be instituted promptly when MRSA bacteraemia is confirmed in order to circumvent the propagation of MRSA.

INTRODUCTION

The occurrence of Methicillin-resistant *Staphylococcus aureus* (MRSA) pericardium infection or pericardial abscess has been increasingly being reported in the literature.¹⁻⁴ Pericardial abscess gains notoriety as an unforgiving disease, and if diagnosed late it would progress to fulminant sepsis and cardiac tamponade. Fatality is inevitable if the cardiac tamponade was left untreated. To date, this is the first case of MRSA pericardial abscess that occurred via haematogenous spread which presented as cardiac tamponade following a recent diabetic foot ulcer infection.

CASE REPORT

A 44-year-old man presented at Hospital Seri Manjung, Malaysia with the chief complaint of progressive dyspnoea for two days associated with lethargy and giddiness. Of most concern to us was that he had multiple syncopal episodes at

home. However, he did not complain of fever. His past medical history was significant for long standing type two diabetes mellitus, dyslipidaemia and chronic hepatitis B.

Retrospective review of his clinical notes showed that he was recently discharged 8 days earlier for infected right diabetic foot ulcer with osteomyelitis of the fifth proximal phalanx. He presented with single superficial ulcer measured 2x2cm over lateral aspect of right fifth toe with minimal seropurulent discharge and slough. Right foot X-ray showed osteomyelitic changes over the right fifth toe proximal phalanx. Blood investigation was unremarkable other than raised white cell count measured $13.8 \times 10^3/\mu\text{L}$ with neutrophilia. Wound debridement and bone nibbling were performed. He demonstrated remarkable improvement after operation and empirical intravenous amoxicillin/sulbactam antibiotic. This was evidenced by resolution of fever for more than 48 hours prior to discharge and the total white cell count was declining in trend. Consequently, he was discharged on day four of admission with oral antibiotics and given an outpatient orthopaedic clinic appointment 5 days later. Both bone culture and blood culture taken during admission yielded MRSA later, prior to his clinic appointment. Unfortunately, he defaulted his orthopaedic clinic appointment.

At presentation, he was afebrile and with poor peripheral perfusion. His blood pressure was 80/46mmHg, heart rate 99 beats per minute and peripheral oxygen saturation 98% under room air. Lungs were clear and neither muffled heart sound nor pulsus paradoxus was appreciated. The previous ulcer debridement site was clean. Chest X-ray showed enlarged cardiac silhouette (Figure 1). ECG showed sinus rhythm with diminished QRS complexes. Full blood count showed leucocytosis with total white cell counts $12.8 \times 10^3/\mu\text{L}$, neutrophils $9.27 \times 10^3/\mu\text{L}$. His urea was 32.4mmol/L and creatinine 386 $\mu\text{mol/L}$. A provisional diagnosis of septic shock secondary to occult sepsis was established and he was admitted to the medical ward.

Despite adequate fluid resuscitation and parenteral vancomycin, he remained in refractory septic shock. Emergency bedside echocardiogram was performed the following the day of admission in ward which detected circumferential pericardial effusion measured 5cm in

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Corresponding Author: Thai Lun Tan

Email: tanthailun@gmail.com

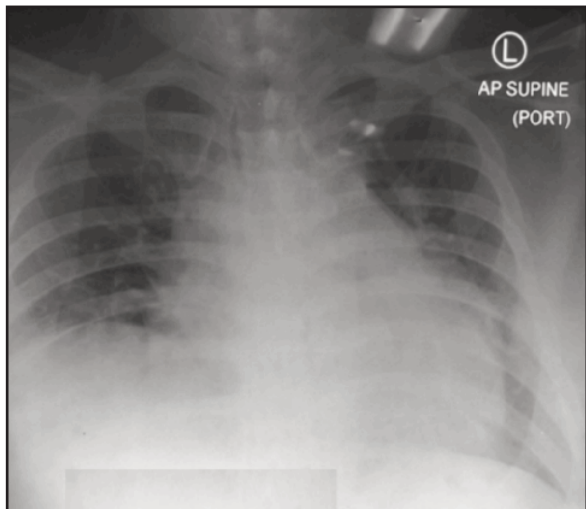


Fig. 1: Chest X Ray at arrival showed cardiomegaly with bilateral pleural effusions.

diameter with diastolic collapse of right ventricle and plethoric inferior vena cava. There was no septation, fibrin or loculation detected. Cardiac tamponade was diagnosed and emergent pericardiocentesis was performed under ultrasound guidance. An intercostal approach was undertaken as it provided a better view in our patient who was obese and we drained 300cc of frank pus (Figure 2). Angiocatheter was left *in situ* for continuous pericardial drainage. Following that, he was monitored in cardiac intensive care unit and a remarkable clinical improvement was observed after pericardial drainage. The pericardial fluid analysis showed copious pus cells, protein 6,030mg/dL, lactate dehydrogenase 14873U/L and it grew MRSA. MRSA pericardial abscess was diagnosed and parenteral vancomycin was continued.

Sequential echocardiogram showed static pericardial effusion measuring 0.5 to 0.6cm in diameter with no regional wall motion abnormality and left ventricular ejection fraction was preserved. Pericardial drainage catheter was removed on day six of antibiotics as there was no more effluent. Serial serum vancomycin levels were within therapeutic range. Blood culture during admission was negative. Unfortunately, he developed worsening of dyspnoea two weeks later which prompted further re-evaluation. Ultrasound of the abdomen did not detect intraabdominal collection. CT pulmonary angiogram and CECT thorax showed bilateral pleural effusion with residual pericardial effusion, without evidence of pulmonary embolism or lungs abscess. Thoracocentesis was performed over left lung which demonstrated a more pronounced pleural effusions to exclude empyema and also for symptoms relief. The pleural fluid analysis showed exudative fluid and the pleural fluid culture was negative. Frusemide infusion was commenced to relieve pulmonary congestion and intravenous tazobactam/piperacillin was administered.

Antibiotics was escalated to IV meropenem later, alongside IV vancomycin in view of worsening clinical condition and the opinion of the visiting infectious disease physician was sought regarding this challenging case. There was no indication for cardiothoracic surgery in view of serial



Fig. 2: Purulent pericardial fluid drained during emergency pericardiocentesis.

echocardiogram demonstrated only minimal residual pericardial effusion and the ventricular functions were satisfactory. Despite maximal medical therapy, his conditions continued to worsen with multi-organ failure and he passed away on day 42 of admission.

DISCUSSION

The nascence and spread of MRSA was first described in 1960.⁵ MRSA bacteraemia, which is defined as presence of MRSA pathogen in the blood stream, has the potential to metastasize and cause multi-foci infection due to the expression of cell wall-anchored surface (CWS) protein. These CWS proteins perform multiple function, including adhesion to host cells and tissues, as well as invasion of a wide-ranged of cell types or tissues in the host.⁶

Pericardial abscess, albeit rare, should be one of the differentials in the diagnostic sieve for individuals presenting with refractory shock. Recent publications have reported increasing involvement of the pericardium as the site of MRSA seeding, which could lead to pericardial abscess and ultimately to cardiac tamponade if diagnosed late.^{1,4} Symptoms of pericardial abscess are vague and it could occur in the absence of fever as illustrated in our patient. Not uncommonly, syncopal attacks may be the presenting symptoms which occur due to profound cerebral hypoperfusion. The widespread adoption of point-of-care ultrasound in evaluation of individuals with refractory shock could be life-saving in establishing the diagnosis of cardiac tamponade or pericardial abscess and to render goal-directed interventions.⁷

Missed MRSA bacteraemia carries important clinical implications as MRSA has the propensity to disseminate to other organ system. The occurrence of MRSA pericardial abscess in our patient was the corollary of untreated MRSA bacteraemia as he was discharged before the formal blood culture result was available. The ensuing pericardial abscess provides evidence that source control via surgical debridement for infected diabetic foot ulcer (DFU) alone is

inadequate when there is a concomitant MRSA bacteraemia; as the latter warrants an extended systemic vancomycin therapy to prevent metastatic infection.

This report also underscores the importance of maintaining high index of suspicion for blood stream infection when treating immunocompromised individuals. The pitfall in this case was that bacteraemia was unsuspected during first hospitalisation as signs and symptoms of systemic infection was not apparent. This observation affirms that among a subset of immunocompromised patients, they may appear well and afebrile during presentation despite having ongoing blood stream infection. This could be attributed to the impaired innate immune system, which causes such individuals to not be able to mount a normal systemic inflammatory response and therefore they may remain afebrile during the beginning of blood stream infection.⁸ Hence, this might create an illusion that lead to dismissal of the systemic infection in such individuals, especially when the foci of infection had been successfully treated with surgical intervention. Considering the above, we recommend that blood culture be considered as the gold standard test in ruling out systemic infection in such individuals.

An effective patient recall system should be in place in order to enable early return of the patient to hospital in the event of missed or wrong diagnosis.⁹ This is based on the grounds that rapid clinical deterioration could happen if blood stream infection was missed and untreated, especially among immunocompromised patients who are vulnerable to severe adverse complications. A close collaboration between clinicians and microbiological laboratory which could act as a safety-netting mechanism would be able to prevent this occurrence. In addition, proactive tracing and review of blood cultures during hospitalization similarly play important roles in ensuring that all blood stream infections are treated appropriately and if it was missed during admission, patient should be recalled urgently to enable prompt institution of systemic antibiotics.

MRSA pericardial abscess seldom occurs alone, and it commonly results from haematogenous spread from a remote source, or direct extension from intrathoracic or subdiaphragmatic etiology (such as trauma or thoracic surgery). Our patient is the first case of MRSA pericardial abscess following infected DFU with untreated MRSA bacteraemia. In light of these observations, we recommend echocardiogram evaluation be done in all MRSA bacteraemia individuals, not only to exclude vegetation alone, but also to detect the presence of pericardial effusion, which could be a precursor of an evolving pericardial abscess. For the latter, serial echocardiographic assessment is recommended if the aetiology of pericardial effusion is not

evident initially. Contrast Enhanced Computed Tomography (CECT) of the thorax could also serve as an adjunct to echocardiogram to look for collections or micro-abscess in the adjacent structures such as lungs, myocardium and septic aneurysm of intrathoracic major vessels, especially when the source is not apparent in the beginning of the disease.¹⁰

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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