Case Report
Septic vasculitis secondary to mycotic pseudoaneurysm of the abdominal aorta caused by methicillin-resistant Staphylococcus aureus: a case report

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Introduction
A pseudoaneurysm (PA) is a localized dilatation of an artery due to disruption of the arterial wall which is only contained by the periaortic connective tissue. A mycotic aneurysm may arise following infection of the normal arterial wall or through secondary infection of a pre-existing aneurysm. It is a rare condition. Staphylococcus aureus is a common organism that causes mycotic aneurysms [1]. Vasculitis can be triggered by infections and the spectrum of manifestation varies with the infectious agent.

Case presentation
A 52-year-old male presented with fever with high spikes and severe myalgia with painful soles of five days duration. He had no significant medical or surgical history and denied trauma to the abdomen. He was a manual labourer, working at a construction site and denied recreational drug abuse or smoking but occasionally took alcohol.

On examination, he was febrile (102 °F) with bilateral tender calves and multiple tender, papular vasculitic lesions over the soles and ankles. There was no lymphadenopathy. There was a tender, smooth, firm and non-pulsatile mass in the right upper quadrant of the abdomen. Peripheral pulses were palpable. No cardiac murmurs, splinter haemorrhages or clubbing was seen and other systemic examinations were unremarkable.

The complete blood count showed a neutrophil leukocytosis of $21.59 \times 10^3/ \mu L$ ($1.5 - 8.0 \times 10^3$) without thrombocytopenia or anaemia. The inflammatory markers were high; erythrocyte sedimentation rate (ESR) was 81 mm/hr and C-reactive protein (CRP) was 365 mg/L(<6mg/L). Two blood culture sets became positive for methicillin-resistant S. aureus (MRSA). Contrast enhanced computed tomography (CECT) of the thorax and abdomen revealed a large abdominal aortic pseudoaneurysm with a large luminal thrombus.
Both transthoracic and transoesophageal 2D-echocardiography were negative for vegetations or any valve abnormality. Eye screening was unremarkable. Serology for leptospirosis, rickettsia, retrovirus, syphilis, hepatitis B, hepatitis C, and melioidosis were negative. Biopsy of the skin lesion revealed: dermis showed perivascular inflammation with extension of lymphocytes into the vessel wall and red cell extravasation suggestive of lymphocytic vasculitis. Creatine kinase was 66 U/ L (22-198). Anti-nuclear antibody (ANA), complement, immunoglobulin profile and cryoglobulins were unremarkable. Renal, liver and coagulation profiles were normal. He was treated with teicoplanin and showed marked improvement in general wellbeing and the rashes and fever subsided. He was transferred to the vascular surgical unit and underwent a laparotomy to repair the PA but it was abandoned due to multiple complex adhesions. The patient and his family were counseled and vascular clinic follow up was arranged.

Discussion
Our patient presented with fever, a non-pulsatile abdominal lump and distal lower extremity tender vasculitic rashes. Our prime working diagnosis was infective endocarditis with septic emboli but, clinically, there was no murmur and no vegetation or valvular dysfunction on echocardiography. Differential diagnoses were leptospirosis, rickettsial infection, systemic vasculitis or malignancy. Radiological confirmation of PA with thrombus and blood culture positivity shifted our focus to mycotic PA of the abdominal aorta and its manifestations.

Vasculitis is simply defined as inflammation of a vessel wall and has different clinical manifestations depending on the size of the vessel and associated disease conditions. Vasculitic cutaneous lesions can occur secondary to microorganisms such as Staphylococcus, Streptococcus, Rickettsia, Mycobacterium, Hepatitis B and C and retrovirus. Septic vasculitis is associated with a disseminated infection. The skin lesions seen in septic vasculitis are petechiae, purpura, papules or blisters. There are four pathogenic mechanisms for cutaneous manifestations in...
septic vasculitis i.e. disseminated intravascular coagulation, direct vessel wall invasion by microorganism, immune mediated vasculitis and septic embolism. There may be more than one mechanism involved in an individual case [2].

The typical histology of septic vasculitis shows a perivascular inflammatory cell infiltration – neutrophils, lymphocytes or mixed type, that may extend through the dermis with red cell extravasation. It may be associated with luminal thrombus [2]. The biopsy of our patient showed a lymphocytic vasculitis.

Histologically septic vasculitis differs from leukocytoclastic vasculitis in the following findings; less leukocytoclasis, presence of occlusive luminal thrombi, prominent hemorrhage or red cell extravasation, deep dermal involvement of inflammatory infiltrate and epidermal pustules or necrosis [2]. Our patient had features of septic vasculitis on histology and this was supported by the presence of the mycotic pseudoaneurysm with blood culture positivity for S. aureus. In addition, the vasculitic rashes subsided with antibiotic therapy.

S. aureus is a common organism that causes mycotic aneurysm of the aorta and is associated with small vessel vasculitis of the skin. The possible mechanisms are that S. aureus and its exotoxin are potent stimulators of the immune response including proliferation and differentiation of T and B lymphocytes and immunoglobulin and cytokine production [3]. The probable pathogenic mechanism that caused septic vasculitis in our patient was immune-complex mediated vessel injury as suggested by bilateral symmetrical tender vasculitic rashes involved the feet and resolution with antibiotics. A more generalized rash would have been seen if there was direct bacterial invasion or if it was due to an exotoxin. Septic emboli were unlikely because the histology did not demonstrate the typical features of fibrinoid necrosis of the vessel with neutrophilic infiltration.

The aetiologies of PA are iatrogenic, traumatic, anastomotic or infective [4]. The common presentation of infected aortic PA is a febrile illness with abdominal pain and a pulsatile lump. The most common aetiology for mycotic PA is pre-existing infective endocarditis. Other causes are direct trauma to the vessel, such as vascular catheterization for any intervention or vascular surgery, and persistent bacteraemia due to intravenous drug abuse. The common complications of mycotic PA are life-threatening sepsis, rupture and fatal haemorrhage and systemic embolization [4]. The aetiology for mycotic PA in our patient was unclear. In our patient, MRSA was the causative agent and infection responded well to teicoplanin. Unfortunately, the PA had to be managed conservatively due to inoperable PA.

**Conclusion**

Mycotic PA is a rare condition that can present as a septic vasculitis which is a non-leukocytoclastic vasculitis. Distinctive histopathologic features and a suggestive clinical history can help to make the diagnosis of septic vasculitis.

**References**

