A unusual consequence of meningococcal sepsis: A case of autoimmune constrictive pericarditis
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Aim:
To illustrate to the acute physician that complications can arise even after successfully treated meningococcal disease.

Introduction:
Neisseria meningitidis is a gram-negative diplococcus that occurs as a natural coloniser of the nasopharynx.1 This can result in invasive meningococcal disease, including meningococcal septicaemia and meningitis.2 Common presenting symptoms include fever, dyspnoea, chest pain, malaise, arthralgia, chills, abdominal pain, nausea, vomiting, diarrhoea, generalised myalgia and cough.3 Cases of constrictive pericarditis as a direct consequence of meningococcal infection in adults are exceptionally rare.4,5 Here we report a case of constrictive pericarditis with multi-system involvement in a patient who was treated with antibiotics and pericardial aspiration and subsequently underwent surgical pericardiectomy.

Case:
A 73-year old male was admitted to a District General Hospital whilst on holiday with fever, chills, rigors and symptoms of lethargy and tiredness with a subsequent collapse. He had a past medical history of hypertension but was otherwise fit and well. He was a non-smoker with occasional alcohol consumption. Blood cultures subsequently grew Neisseria meningitides W135 and he was treated for 9 days with intravenous Cefotaxime.

He was subsequently admitted to our Acute Medical Unit 2 weeks later with dyspnoea and a vasculitic rash over his lower limbs. On initial clinical assessment he was afebrile but tachycardic with a heart rate of 130 beats per minute, with no signs of haemodynamic instability. Clinical examination revealed a pericardial friction rub, signs of a left sided pleural effusion and peripheral oedema. Neurological examination did not reveal any neck stiffness or other signs of meningism.

Initial clinical investigations revealed raised inflammatory markers (neutrophil leukocytosis, WBC 13.8 X 10^9/L, Neutrophils 10.3 X 10^9/L; C-reactive protein 65 mg/L, erythrocyte sedimentation rate 100mm in the 1st hour). He was found to be in atrial fibrillation with a rapid ventricular response at a rate of 130 beats per minute on electrocardiography (ECG), and chest radiograph confirmed the presence of bi- basal pleural effusions (Figure 1).

After consultation with the Microbiologists, he was treated with intravenous fluids and Cefotaxime and Gentamicin for presumptive recurrence of meningococcal disease. Rate control of his atrial fibrillation was achieved with intravenous Digoxin. A pleural aspiration was performed which revealed signs of a sterile lymphocytic effusion.

Skin biopsy of his rash confirmed the presence of a leukocytoclastic vasculitis negative for IgG, IgA and C3 on immunofluorescence. A full autoimmune screen and septic screen were unremarkable.

Trans-thoracic echocardiography demonstrated a 2.9cm pericardial effusion with thickened pericardium (4mm) and mild right ventricular systolic dysfunction suggestive of constrictive pericarditis. No other echocardiographic features of cardiac tamponade were observed. Tropinol performed during admission was negative (<0.07 µg/l).

CT scan of the chest, abdomen and pelvis showed the presence of diffuse serous disease, with thickened pericardium, moderate pericardial effusion measuring up to 27mm, moderate-size pleural effusions, and multiple small reactive mediastinal adenopathy (Figure 2). CT head was organised to look for underlying space-occupying lesion; this revealed bilateral basal ganglia calcification but no other lesion. He clinically improved and was discharged with a view to outpatient follow-up.

However, he was subsequently readmitted to the Acute Medical Unit with worsening shortness of breath on exertion; he was found to be in NYHA Class III heart failure. He was started on an intravenous Furosemide infusion. After consultation with Cardiology, a pericardial aspiration was performed, which drained approximately 280mls of serous fluid. Repeat echocardiography showed an improvement in the effusion but persistence of a thickened pericardium.

He improved clinically and was discharged on a course of oral diuretics.

Outpatient cardiac MRI showed features of constrictive pericarditis. He underwent cardiac catheterisation which showed no evidence of flow-limiting coronary artery disease. Despite medical treatment he remained symptomatic and eventually underwent a surgical pericardiectomy.

Discussion:
Autoimmune constrictive pericarditis is a rare but recognised complication of meningococcal bacteraemia, occurring 6-16 days after complete antibiotic treatment5. Acute physicians should consider this possibility when patients present with symptoms suggestive of pericardial disease following treatment.2 Management includes a combination of antibiotics and aspiration of pericardial fluid for symptomatic benefit6. Immunosuppression with corticosteroids is indicated in resistant cases once active infection has been excluded.8

References:

Figure 1: Chest radiograph taken on admission

Figure 2: CT chest demonstrating pericardial thickening with effusion

Figure 3: Surgical pericardiectomy in progress

http://acessurgery.com/A6/Select/Search/AC6%25bC%25bCResults.aspx?searchId=pericardiectomy&locale=en&pericardiectom &1=0&2=0&searchStatus=0&searchTerm=pericardiectomy&searchSource=imagevideos&searchType=

Histology of the pericardial tissue was consistent with dense fibrosis and constrictive pericarditis with no bacterial growth or evidence of acid-fast bacilli in the pericardial tissue.

He remains under regular follow-up by the Cardiologists.