CASE REPORT

Cardiac tamponade: an initial presentation of SLE

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SUMMARY

A 45-year-old woman presented with dyspnoea, chest pain, orthopnoea and bilateral leg oedema. On admission, she was found to have nephrotic syndrome and global pericardial effusion with impending tamponade for which pericardiocentesis was performed. The diagnosis of systemic lupus erythematosus was made based on the clinical and biochemical findings. She was also started on dialysis and immunosuppressants for lupus nephritis.

BACKGROUND

Systemic lupus erythematosus (SLE) is a multisystemic disease which can affect virtually any bodily organ. Pericarditis and pericardial effusion are well-recognised cardiac complications of SLE, with pericardial effusions seen in up to 50% of patients with SLE.¹ However, cardiac tamponade has been rarely described especially as an initial presenting feature of SLE. In this case, a patient with undiagnosed SLE presents with features of nephrotic syndrome and global pericardial effusion which has resulted in cardiac tamponade.

It is imperative that cardiac tamponade is diagnosed promptly through thorough physical examinations and appropriate investigations as early pericardiocentesis can be lifesaving. Injudicious use of diuretics for fluid retention should be avoided as they could reduce cardiac filling and worsen cardiac tamponade.

CASE PRESENTATION

A 45-year-old Nigerian woman presented to a district general hospital with 4-week history of worsening bilateral leg oedema, abdominal distension and oliguria. Over the 3 weeks prior to admission, she also noticed a non-productive cough and orthopnoea for which she was started on furosemide in community by her GP. Since then, she had developed worsening dyspnoea with sharp, intermittent left-sided chest pain which warranted her admission to hospital. The pain was worse on exertion and coughing, and resolved spontaneously. Each episode lasted from 15 min to 5 h. She also complained of pruritus and described three episodes of rectal bleeding with streaks of fresh red blood over the 3 days prior to admission. She admitted a travel history to Africa last year.

She has a history of hypertension for which she takes lisinopril (10 mg twice daily). She was diagnosed with diabetes 1 year ago which had been diet-controlled. She also suffers from migraine.

On examination, she had periorbital oedema and bilateral peripheral oedema up to her knees. There was a palpable cervical lymph node. She was

afebrile, tachycardic and hypertensive with heart rate of 118 and blood pressure of 189/83. Jugular venous pressure was raised at 4 cm with visible carotid pulsations. Auscultation of heart sounds revealed a loud S1 with a possible diastolic murmur. There was reduced air entry bilaterally on examination of the chest. The abdomen was distended with generalised tenderness and shifting dullness. There was also a vasculitic rash on her toes.

INVESTIGATIONS

Blood tests revealed microcytic anaemia (haemoglobin 7.0 g/dL) and thrombocytopenia (platelets $121 \times 10^9/L$). Renal functions were deranged with raised urea (22 mmol/L) and creatinine (139 $\mu\text{mol/L}$). She was hyperkalaemic (5.9 mmol/L). Albumin was low (19 g/L). C-reactive protein was normal. Blood film examination showed hypochromia, microcytosis, presence of schistocytes and microspherocytes with marked anisopoikilocytosis. Reticulocyte count was normal.

Urinalysis showed ++++ protein and ++ blood. Nephrotic syndrome was therefore observed clinically with a triad of proteinuria, hypoalbuminaemia and oedema. ECG showed sinus tachycardia with small complexes. Chest X-ray (figure 1) revealed cardiomegaly with bilateral pleural effusions. The initial differential diagnoses at the time include glomerulonephritides and causes of haemolytic anaemia, such as microangiopathic haemolytic anaemia.

In view of the presence of a new murmur, renal failure with haematuria and a vasculitic rash, a transthoracic echocardiogram was arranged urgently for suspected infective endocarditis. The echocardiogram (figure 2) revealed a moderate-to-large global pericardial effusion of a maximum of 2.3 cm adjacent to the right atrium free wall. There were right ventricle outflow tract collapse and exaggerated right atrium free wall motion suggestive of early tamponade. There was also significant mitral valve and tricuspid valve flow variation on inspiration and expiration consistent with haemodynamic compromise. No obvious vegetation was seen.

After discussion with a nephrologist and a cardiologist, she was transferred to a tertiary centre for urgent pericardial drainage which drained 500 mL of blood-stained fluid.

Pericardial fluid analysis showed a raised level of protein (38 g/L) and lactate dehydrogenase (190 g/L) with a normal level of glucose (7.5 mmol/L) suggestive of possible exudate aetiology. Microscopy and culture showed no growth and was negative for tuberculosis. Cytology showed no epithelial or mesothelial cells.

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Figure 1 Chest X-ray cardiomegaly with bilateral pleural effusion.

In view of the yield of blood-stained pericardial fluids, a malignant effusion was suspected. An ultrasound scan of the right breast was organised because of examination findings of some right-upper breast firmness to palpation and significant axillary lymphadenopathy. The ultrasound scan showed dense parenchyma but no definite intramammary lesion. Significant abnormal lymph nodes were identified in the right axilla. Biopsy was not taken at that time as she was thrombocytopenic with disordered clotting.

A CT scan of chest/abdomen/pelvis was then organised to rule out a malignant process. The scan showed bilateral pleural effusions, some dependent changes in the adjacent lung bases, moderate pericardial effusion and generalised subcutaneous oedema. No focal abnormality was seen apart from a bulky 9 cm subserosal fibroid.

Results from autoimmune screen for suspected glomerulonephritides became available and revealed positive antinuclear antibody (1:640), antidouble stranded DNA (1006 u/mL) and anti-Ro antibody titres (60 kd antibody of 225 AU/mL and 52 kd antibody of 51 AU/mL) suggestive of SLE. Cytoplasmic antineutrophil cytoplasmic antibody: proteinase 3 (C-ANCA: PR3) was positive (117 AU/mL) and per-nuclear antineutrophil cytoplasmic antibody: myeloperoxidase (P-ANCA: MPO) was negative (0.04 AU/mL). Complement levels including C3 (0.28 g/L) and C4 (0.04 g/L) were both low. Hepatitis B serology, antiglomerular basement membrane antibody and protein electrophoresis were negative.

Figure 2 Echocardiogram global pericardial effusion.

The patient was transfused for associated anaemia and was started on dialysis, as she became anuric. Renal biopsy was taken which showed class IV nephritis with mild scarring, hypercellular glomeruli, mesangial-endocapillary hypercellularity and acute interstitial inflammation. No necrosis or crescents were seen.

Her renal function deteriorated after she was started on immunosuppressants including cyclophosphamide, rituximab and mycophenolate with prophylactic nystatin and co-trimoxazole for lupus nephritis. An ultrasound scan of the kidneys was organised. It showed a diffuse increase in cortical echogenicity suggestive of some degree of intrinsic renal disease. Both kidneys were mildly enlarged with no hydronephrosis seen.

An MRI of the brain was organised due to clinical findings of complex ophthalmoplegia with diplopia raising the suspicion of cerebritis. It was however reported normal apart from some microangiopathic changes.

Echocardiogram following dialysis showed mild pericardial effusion with no evidence of tamponade. Her urine output remained at approximately 300 mL/day. She was discharged on the 18th day with oral mycophenolate (500 mg twice daily) and prednisolone (20 mg once daily) with outpatient dialysis and follow-up at the SLE clinic.

DISCUSSION

Pericardial effusion can accompany any pericardial disease. Its aetiology includes postmyocardial infarction or cardiac surgery, autoimmune, metastatic malignancy, renal failure with uraemia, aortic dissection and pericarditis which can be viral, bacterial or tuberculous. Pericardial tamponade occurs when the associated effusion exerts a significant intrapericardial pressure which offsets the myocardial transmural pressure to impede cardiac filling.²

Cardiac tamponade can be classified into acute, subacute, low pressure and regional. In our case, the patient had a subacute presentation where cardiac tamponade occurs over days to weeks. Common symptoms in subacute cardiac tamponade include dyspnoea, chest discomfort, peripheral oedema and fatigability.³ These symptoms resemble many presentations of fluid retention such as heart failure and renal failure. Thorough physical examinations and appropriate investigations should be performed particularly before the initiation of diuretic therapy as diuretics could potentially exacerbate cardiac tamponade by reducing cardiac filling pressure.

Early recognition of cardiac tamponade is paramount as delayed pericardiocentesis can be life-threatening. Sinus tachycardia and elevated jugular venous pressure are common signs found in tamponade. An enlarged cardiac silhouette with clear lung fields on a chest X-ray suggests the presence of pericardial effusion with the accumulation of at least 200 mL of pericardial fluid in the pericardial space.² It has also been reported that low QRS voltage on an ECG is a specific manifestation of cardiac tamponade, not of the effusion.⁴ Echocardiogram in this case demonstrates features which are characteristic of cardiac tamponade. These include cardiac chamber collapse, flow variation and inferior vena cava plethora (dilatation of inferior vena cava with <50% change on inspiration).⁵

Contrary to the well-known Beck's triad of falling blood pressure, rising jugular venous pressure and a small and quiet heart, hypertensive cardiac tamponade has been recognised as a variant form of tamponade due to excessive β -adrenergic drive in patients with antecedent hypertension.^{2 6 7} The diagnosis of cardiac tamponade should therefore not be neglected even though patients are hypertensive on presentation.

Further investigations including an autoimmune screen and imaging were carried out to establish the cause of pericardial effusion although uraemia could be a potential cause. Pericardial fluid analysis suggested a possible exudative effusion of an infective, inflammatory or malignant aetiology. Negative results from microbiology and cytology exclude an infective or malignant effusion. A breast examination and a subsequent CT scan were also performed to rule out a malignant process. Positive anti-dsDNA and antinuclear antibody titres were key criteria to our diagnosis of SLE.

Diagnosis of SLE is made using the American College of Rheumatology criteria if 4 or more of the 11 criteria are present, either serially or simultaneously, during any interval of observation.^{8 9} In our case, the patient meets 4 of the 11 criteria for a definite diagnosis of SLE namely proteinuria, pericardial effusion, positive anti-dsDNA and antinuclear antibody titres. Nevertheless, she does not meet the criteria of haemolytic anaemia with reticulocytosis or severe thrombocytopenia ($<100 \times 10^9/L$). Hypocomplementaemia also suggests the presence of active disease.

Learning points

- ▶ Cardiac tamponade is a rare initial presenting feature of systemic lupus erythematosus.
- ▶ Injudicious use of diuretics for fluid retention should be avoided as they could worsen cardiac tamponade by reducing cardiac filling.
- ▶ Thorough physical examinations and appropriate investigations with a high index of suspicion are keys to diagnosis of cardiac tamponade in patients with unexplained fluid retention.
- ▶ Patients with pre-existing hypertension may be hypertensive despite cardiac tamponade.

Competing interests None.

Patient consent Obtained.

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