Cardiac Tamponade as a Rare Initial Manifestation of Systemic Lupus Erythematosus

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Abstract

Systemic lupus erythematosus is a collagen disease that frequently involves the cardiovascular system, the pericardium being the most commonly affected site. This involvement is usually pericardial effusion, majority of the time of mild degree. In a few patients, pericardial involvement may be the initial presentation of systemic lupus erythematosus. Cardiac tamponade is a rare clinical manifestation and very few cases are reported as the initial manifestation of the disease. Here, we report a patient with SLE with cardiac tamponade. Our patient presented with subacute presentation. Echocardiography showed large pericardial effusion with features suggestive of cardiac tamponade. Pericardiocentesis was done. The fluid was hemorrhagic in nature. Fluid analysis was suggestive of exudative effusion. Antinuclear antibody, U1-nRNP, nucleosomes, Sm were found to be positive. Oral prednisolone was started and slowly tapered to a maintenance dose. Azathioprine, hydroxychloroquine, torsemide were added. Patient has responded well and is following up in the OPD.

Keywords: SLE -Systemic lupus erythematosus, CTD – Connective tissue disorder, Cardiac tamponade, Pericardial effusion.

Introduction

Cardiac tamponade in SLE (Systemic Lupus Erythematosus) is a rare manifestation accounting for around 1-2% cases.[1] Although pericarditis is a common phenomenon in SLE, cardiac tamponade is rare.

A 46 years old female patient, came with breathlessness on exertion, anasarca, multiple joint pains, fatigue, recurrent oral ulcers, rash over her cheeks for 2 months. Pulse-120/min, BP-100/60mmHg with severe pulsus paradoxus, CVS- S1, S2 muffled, JVP was raised-10cm with prominent x descent and absent y descent. Other systems were normal.

Observations

She had anemia (Hb- 9.3g/dl), thrombocytopenia (78,000/cu mm) and leukocyte count was normal. Rest lab reports were normal. CXR showed cardiomegaly (Figure 1).

Echocardiography showed large pericardial effusion with features suggestive of cardiac tamponade. Diastolic RV collapse, moderate pulmonary artery hypertension with mild Tricuspid regurgitation. There was also significant mitral valve and tricuspid valve flow variation on inspiration and expiration consistent with haemodynamic compromise (Figure 2 and 3). Emergency Pericardiocentesis was done and pig

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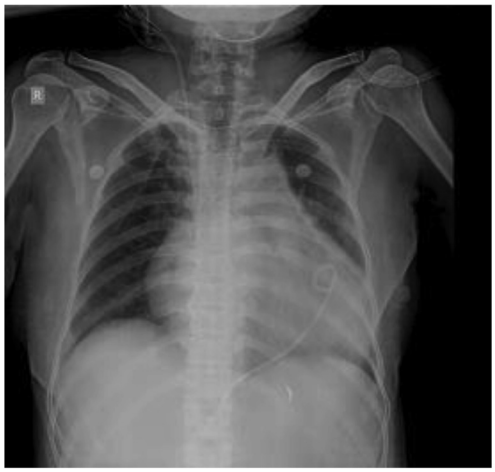

Figure 1

tail catheter was inserted. The fluid was hemorrhagic in nature. In view of hemorrhagic fluid differential diagnosis of tuberculosis and malignancy were kept. Fluid analysis was suggestive of RBC-1.6 million cells. Total cells were 1340/cu mm predominantly lymphocytes. Glucose was 79 mg/dl (serum glucose 98mg/dl) and protein were 5.4gm/dl (Serum total protein7.3gm/dl). Fluid ADA and TB-PCR were negative. Culture of the fluid showed no growth. It was also negative for malignant cells. Cultures of blood/urine/ central venous line tip, pigtail tip showed no growth.

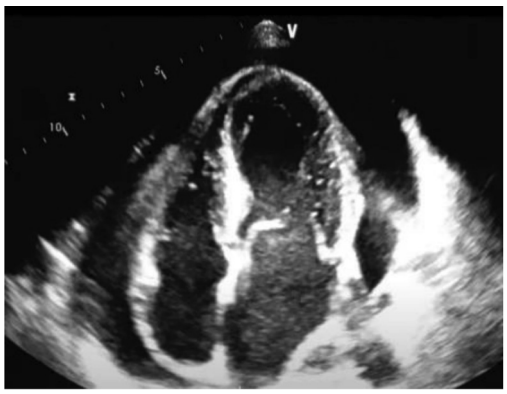

Figure 2: Apical four chamber view showing large pericardial effusion.

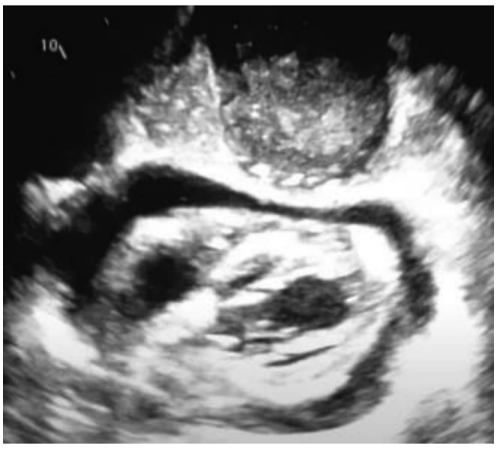

Figure 3: Subcostal view showing right ventricular collapse due to large effusion.

ANA by IFA- positive (5.4). ANA BLOT- U1-nRNP, Sm- strong positive, nucleosomes-positive. RF, CRP-neg.

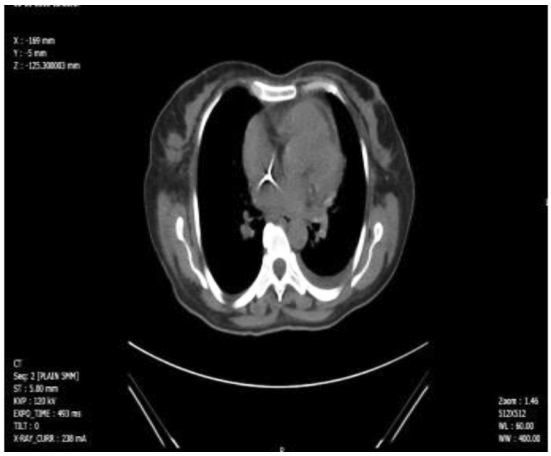

Figure 4

HRCT Thorax showed bilateral pleural effusion, pericardial effusion. No evidence of malignancy (Figure 4).

Follow up Echocardiography showed mild effusion, RA, RV Dilated, moderate PAH.

Discussion

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

The cardiac manifestation of SLE includes pericarditis, myocarditis, endocarditis and conduction system abnormalities. Although pericarditis is common, cardiac tamponade, particularly as an initial form of presentation, is rare.

In our case, the patient had a subacute presentation where cardiac tamponade occurs over weeks. Most common presenting symptoms in subacute cardiac tamponade include dyspnoea, chest discomfort, peripheral oedema and fatigability.[3] These symptoms bear a resemblance to several presentations of fluid retention such as heart failure and renal failure. Complete physical examinations and appropriate investigations should be performed mainly before the

initiation of diuretic therapy as diuretics could actually worsen cardiac tamponade by reducing cardiac filling pressure.

Early recognition of cardiac tamponade is supreme as delayed pericardiocentesis can be fatal. 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.

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 7 of the 11 criteria for a definite diagnosis of SLE specifically, cutaneous involvement in the form of malar rash, oral ulcer, synovitis in the form of >2 joint involvement, serositis in the form of pericardial effusion and pleural effusion, thrombocytopenia, positive Anti-Sm, and antinuclear antibody titres.

Oral prednisolone 1mg/kg/day was started and slowly tapered to a maintenance dose of 10mg/day. Azathioprine 50mg, hydroxychloroquine, torsemide, Bosentan 62.5mg were added. Patient has responded well and is following up in the OPD.

Conclusion

Cardiac tamponade as an initial presentation of SLE is rare. Thorough physical examination and appropriate investigations with a high index of suspicion are keys to diagnosis of cause of cardiac tamponade. Judicious use of diuretic for symptoms of fluid retention is important during initial period of presentation as diuretics may worsen the hemodymics by reducing cardiac filling pressure. Also, in a pre-existing hypertensive patient with cardia tamponade can tend to be hypertensive even in tamponade due to high beta-adrenergic stimulation. Although cardiac manifestation is common late feature of SLE, cardiac tamponade as an initial manifestation of SLE is rarely reported.

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