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Cardiac tamponade as initial presentation of systemic lupus erythematosus: A Case Report

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Abstract
Systemic lupus erythematosus (SLE) is an autoimmune disease that can involve any organ system, exhibiting great diversity in presentation. Although pericarditis and pericardial effusion are common cardiac complications of systemic lupus erythematosus (SLE), cardiac tamponade is a very rare initial manifestation of this disease.. We report the case of 8 years old girl from Himachal who presented with cardiac tamponade secondary to pericardial effusion as the initial presenting symptom of SLE.

Keywords: systemic lupus erythematosus, cardiac tamponade, pericarditis, pericardial effusion

Introduction

Systemic lupus erythematosus (SLE) is a multisystem autoimmune connective tissue disorder. Approximately 20% of cases present in childhood. Pericarditis and pericardial effusions in SLE are well recognized cardiac complications in SLE and are usually of mild degree. Cardiac tamponade is a medical emergency that develops when a pericardial effusion reaches a critical amount, limiting cardiac inflow and leading to hemodynamic compromise. Although pericardiocentesis associated with antiin flammatory drugs is the treatment of choice, surgery is indicated in some cases. In this case report we present and discuss pericarditis leading to cardiac tamponade as the initial manifestation of cSLE.

Case Report

8 years old girl, resident of Himachal, presented to emergency with chief complaints of facial puffiness and decreased urine output for 3 days. She had no past medical history however she had progressive weight loss, on-offfever and pain abdomen for 3 months. Shewas first borne to non-consanguineous married parents with no family history of tuberculosis or autoimmune disorder. There was no history of rash, oral ulcers, hair lossor use of any medication.

On examination she had a heart rate of 110/min, R.R - 34/min, B.P of 100/70 mm of Hg and a temperature of 37.8 ℃and was maintaining saturation of 98% on room air. She was underweight and stunted and had pallor with generalized lymphadenopathy. She had doughy abdomen on examination with hepatosplenomegaly. On cardiac examination she had muffled heart sounds with jugular venous distension and pulsus paradoxus with decreased breath sounds on left side on respiratory examination. There was no evidence of joint inflammation/clubbing.

Her chest x ray displayed an enlarged cardiac silhouette following which her echocardiography was performed which was suggestive of massive pericardial effusion and confirmed features of cardiac tamponade with diastolic collapse of RA/RV with intrapericardial fibrinous exudates and left pleural effusion. Echocardiography guided pericardiocentesis was done and around 100 ml of straw-colored fluid was drained which brought immediate relief to patient and patient remained hemodynamically stable.

Based on her history and clinical findings possibilities of disseminated tuberculosis, autoimmune disorders -SLE were considered initially and were evaluated for the same.

Laboratory investigations revealed microcytic hypochromic anemia with normal white cell count and differential. ESR and CRP were raised with values of 120mm/1sthr and 46mg/l respectively. Urine-analysis was done which was suggestive of nephrotic range of proteinuria. Patients serum tested strongly positive for anti -nuclear antibodies (ANA) with 4+, diffuse pattern and also positive for anti-double stranded DNA antibodies (anti-dsDNA). Serum compliments were low with C3 of 46 HIV testing and Mantoux tuberculin skin test was negative with CBNAAT of samples of pericardial fluid /gastric lavage as negative. ADA was within normal range.

The diagnosis of SLE was established based on positive clinical and immunological findings. The patient satisfied 5 of 17 Systemic Lupus International Collaborating Clinics (SLICC) criteria for classifying SLE, namely serositis, renal involvement as persistent proteinuria, low compliment levels and positive serum ANA and positive anti-ds DNA antibodies. Supporting the diagnosis was raised ESR, CRP and generalized lymphadenopathy.

Patient was started on oral corticosteroids at 2 mg/kg/day along with ATT.??? During her course she developed hypertension and her urine analysis showed persistent nephrotic range of proteinuria with hematuria. Her repeat ECHO analysis showed reaccumulating pericardial fluid with features of cardiac tamponade on 10th day of her hospital stay. She was started on methyl prednisolone therapy and echocardiography guided peri cardio centesis was performed, 50 ml of pericardial fluid was aspirated. On day two of injection methylprednisolone child developed oliguria and subsequently renal shut down, planned for

peritoneal dialysis, but child scum to her illness.

Discussion

SLE is one of the most common autoimmune connective tissue diseases in childhood, where it tends to present more severely than in adults. The common initial presentations of cSLE included constitutional symptoms, renal disease, musculoskeletal and cutaneous involvement. Less frequently involved at cSLE presentation were the neuropsychiatric, pulmonary and cardiac systems, with pericarditis reported in 3–24% of cases at presentation.

Few studies have examined cardiac involvement in cSLE and cardiac tamponade in these patients has not been well-defined. In a 10-year retrospective single center study, Oshiro *et al*. examined 31 patients (<10 years) with SLE and found that out of 13 patients who had cardiac involvement, only 2 patients presented with cardiac tamponade.[1]A more recent multicenter cross-sectional study of 155 cSLE patients reported the initial manifestation of cardiac tamponade in 2 cases (1.3%) [2]. This latter figure is consistent with findings in adult SLE (aSLE) where it is reported 1% of patients present in this way [3]. Pericarditis can occur at any time during the disease course but appears to be one of the earlier cardiac manifestations. This is clearly seen in a longitudinal study of 256 cSLE patients (where out of the 39 patients who had pericarditis at any time (mean follow-up time 3.5 ± 3 years), 30 patients presented at diagnosis [4].However data on the prevalence of cardiac tamponade throughout the course of cSLE is lacking. In one aSLE series of 395 patients, 10 patients were found to have cardiac tamponade and in 4 patients it was the initial manifestation [5]. Therefore in aSLE it is likely that cardiac tamponade is truly rare, both as the initial manifestation and throughout the disease course

Above studies revealed the immediate treatment of cardiac tamponade involves withdrawal of pericardial fluid usually by pericardiocentesis, done in all cases except one which was aborted due to a thickened pericardium with adhesions [6]. When compared to surgical drainage, echocardiography y guided pericardiocentesis has been associated with lower morbidity and mortality rates [7]. Some cases required placement of a pericardial drain or less commonly surgical pericardiectomy incase of massive chronic pericardial effusion. Medical management consists of anti-inflammatory medication which typically involves high dose corticosteroids, antimalarials and non-steroidal anti-inflammatory drugs. Follow up is essential to exclude recurrent pericardial effusions and pericardial thickening.

Atypical presentation is common in cSLE and often leads to major diagnostic delay. By allowing for greater weighting of immunologic criteria, the use of the SLICC criteria may be more sensitive for diagnosis in these cases, potentially leading to earlier diagnosis and treatment.

Conclusion

Cardiac tamponade as the initial presentation of cSLE is rare. More females than males presented in this way. One must consider cSLE in the differential diagnosis of pericarditis and cardiac tamponade and perform appropriate testing for rheumatologic disease. Pericardiocentesis should be done when there is hemodynamic compromise. Pigtail insertion under C arm fluoroscopy should be done for massive chronic pericardial effusion.

Conflict of Interest Statement

No conflicts of interest.

Funding

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Ethical Approval

Due permission was taken from institutional ethical committee.

Patient Consent

Written and informed consent was taken from patient for the publication of this case report.

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