Abstract:
Cardiac tamponade is a life threatening emergency caused by the accumulation of fluid in pericardial cavity causing obstruction to blood flow. Malignancy and tuberculosis in developing countries are the usual causes of cardiac tamponade. SLE, a connective tissue disorder affecting multiple systems can rarely present with cardiac tamponade. Common cardiac manifestation of SLE is pericarditis. We are presenting the case report of a 35 year old female patient who got admitted with pericardial effusion with tamponade. On further evaluation the cause of effusion was found out to be SLE. She was treated with prednisolone hydroxychloroquine and showed excellent response.

Keyword: Cardiac tamponade, Pericardial effusion, Systemic lupus erythematosus.

INTRODUCTION
Though Cardiac involvement is common in SLE its unusual to see cardiac tamponade as the presenting clinical manifestation. Pericarditis is the most common cardiac manifestation of SLE. It occurs in 60% patients with SLE. We are presenting the case report of a 35 year old female presented with massive pericardial effusion, the cause of which was later found out to be SLE.

CASE REPORT
35 year old female patient admitted in our hospital with history of breathlessness - grade 1 for the past one month , progressing to grade 4 for the five days. No h/o chest pain, palpitation, decreased urine output. No h/o any significant illness in the past & no history of any medicine intake. No h/o weight loss or fever. Menstrual & Obstetric histories were normal. O/E: The patient was conscious, oriented, dyspnoeic, had pedal edema, pallor. BP was 100/70 mmHg & PR was100/mt. Cardiovascular system examination revealed elevated JVP, heart sounds were decreased in intensity and there was no murmurs or added sounds. Chest was clear. Other system examination didn’t show any positive findings. Chest-X ray showed massive pericardial effusion with minimal bilateral pleural effusion. An ECG was taken which showed low voltage complexes & T wave inversion in all leads. An Echocardiogram was done which
ventricular diastolic collapse. Cardio thoracic surgeons opinion was taken 800 ml of pericardial fluid was drained. A pericardial window was created to pleural space & intercostal drain was put. Then investigations were send to find out the cause of pericardial effusion. Complete hemogram showed hemoglobin concentration of 9.2 gm%. Other cells were normal. ESR was 70mm/hr. Peripheral smear showed normocytic normochromic anemia. Mantoux test was negative. RBS, urea, creatinine levels were normal. Urine routine examination was normal & casts were absent. HIV-ELISA was non-reactive. Thyroid function test was normal. CT chest was normal except for pericardial & pleural effusion. CT abdomen was normal. Pericardial fluid analysis showed a protein concentration of 4.1/litre. LDH was 610 IU/ml. ADA, PCR were negative for tuberculosis & ANA was positive in pericardial fluid. Cytology for malignant cells were negative. ANA analysis in serum was done which turned out to be positive. Anti ds-DNA was done and it was strongly positive. Coombs test was done & was positive. As 4 out of 11 criteria for SLE was positive and thus a diagnosis of SLE was made by ACR criteria. The patient was started on prednisolone 60mg/d & hydroxychloroquine 400mg/d. Patient responded and by the end of 10th day drain was minimal. Chest tube was removed & patient was discharged on 12th day.

**Discussion**

The accumulation of fluid in pericardial cavity in a quantity sufficient to cause serious obstruction to inflow of blood into the ventricles results in tamponade. Common causes for effusion progressing to tamponade are bacterial (including TB), fungal & HIV infections & neoplasms. SLE is a connective tissue disorder which involves almost all systems including cardiovascular system. The cardiac manifestations of SLE includes myocarditis, pericarditis, endocarditis, conduction abnormalities etc. Pericardial effusion in SLE may occur either due to direct pericardial involvement or may occur as uremic pericarditis secondary to lupus nephritis. Cardiac manifestation as the presenting manifestation of SLE is rare, out of which tamponade is rarest. Our patient didn’t have signs of any other major system involvement. The diagnosis of SLE was made after evaluating the causes for pericardial effusion with cardiac tamponade among which SLE is very unusual. Tuberculosis was one of the most important consideration which was ruled out as pericardial fluid ADA, PCR & Mantoux test were negative. ANA positivity & strongly positive anti ds-DNA were in favour of SLE. Cytology for malignant cells was also negative in pericardial fluid. Hypothyroidism, though a rare cause, was also ruled out. For the diagnosis of SLE revised ACR criteria is used. As per this criteria 4 out of 11 should be positive to make a diagnosis of SLE. It has got 95% specificity & 75% sensitivity. Features positive in our patient were serositis, hematological disorder in the form of auto-immune hemolytic anemia, immunological disorder.
(anti-ds-DNA), anti-nuclear antibody positivity & thus a definitive diagnosis of SLE was made. Lupus nephritis was ruled out as blood urea, serum creatinine & urine examination for protein, casts & sediments were normal. Regarding the management, pericardiocentesis is the initial treatment of choice. Patients with mild tamponade can be treated with a course of NSAIDS & or colchicine & monitored. Patients with autoimmune disease can be treated with corticosteroids. Our patient was treated with pericardiocentesis, high dose steroids & hydroxychloroquine to which she responded excellently.

REFERENCES

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