

Case Report

MASSIVE ASCITES DUE TO LUPUS PERITONITIS: A RARE PRESENTATION OF SYSTEMIC LUPUS ERYTHEMATOSUS

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ABSTRACT

Polyserositis is a common finding among the wide range of manifestations of SLE patients. Approximately 16% of SLE patients have pleuritis and pericarditis, but peritoneal involvement is extremely rare, and its presentation as massive ascites is even a rare condition. Here we report a case of initially diagnosed drug induced lupus evolving with systemic manifestations as lupus nephritis and massive ascites due to lupus peritonitis. Exhaustive diagnostic investigation was performed including imaging and ascitic fluid analysis to rule out any organic cause or infection.

Keywords: *Lupus, Peritonitis, Ascites*

INTRODUCTION

Systemic lupus erythematosus is an autoimmune disease characterized by involvement of multiple organs. The incidence of disease is 10 folds higher among females compared to males and incidence peaked in the population aged 25-44 years. Polyserositis is a common finding among the wide range of manifestations of SLE patients. Approximately 16% of SLE patients have pleuritis and pericarditis, but peritoneal involvement is extremely rare, and its presentation as massive ascites is even a rare condition. Here we report a case of initially diagnosed drug induced lupus evolving with systemic manifestations as lupus nephritis and massive ascites due to lupus peritonitis.

CASES

35 years old female previously diagnosed as drug induced lupus with skin involvement on Hydroxychloroquines 200 mg once a day & a old pulmonary tuberculosis now presented with complaints of fever and sudden abdominal distension from last 10 days. Fever was high grade, intermittent with no chills and rigor. H/O loose stools 7-8 episodes/day, cramping pain abdomen. There was no history of vomiting. History of bilateral lower limb swelling and shortness of breath was also present. There was no history of chest pain, palpitations. On examination patient had facial puffiness, pallor, bilateral symmetrical pitting pedal edema. Systemic examination revealed tense abdominal distension and shifting dullness suggestive of massive ascites but no tenderness. Hepatomegaly was present but no splenomegaly. On chest examination percussion note was dull and breath sounds were diminished in bilateral lower lobes suggestive of bilateral pleural effusion. On basis of history & examination a provisional diagnosis of SLE with polyserositis was made and plan was to look for acute disease activity. But as patient had fever (high grade) so infection was also to be ruled out, so all set of investigation were planned. Patient's Hemoglobin was 7.2g/dl, TLC-4300 cells/mm³, PLT-2 Lac/mm³. Liver function tests were normal except albumin which was low with a value of 1.6 gm/dl. KFTs showed Urea -106mg/dl & Creatinine-1.1mg/dl, Electrolytes were normal. Her urine routine examination showed 10-15 RBCs/hpf, 5-10 pus cells/hpf, granular casts, proteinuria 3+. 24 hour urinary protein excretion of 1800mg/day. USG whole abdomen showed Liver normal in size & shape & coarse in echotexture, normal portal vein diameter, with free fluid in peritoneal cavity (massive ascites) & right sided pleural effusion. CECT whole abdomen confirmed the ultrasonographic findings with nothing suggestive of any perforation, lymph nodes etc. Serum ANA was positive & double stranded DNA was 832.8 IU/ml. Her direct coomb's test came out to be positive. Viral markers were negative. To look for staging of lupus nephritis-renal biopsy was done which revealed diffuse lupus nephritis displaying active lesion (cellular crescents, endocapillary proliferation with neutrophil exudation, wire loop subendothelial deposits) suggestive of

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ISN/RPS class IV-G. There were also features of patchy acute tubular injury with interstitial edema. To look out infective etiology as patient had fever & also had old pulmonary tuberculosis further investigation were planned. Ascitic fluid analysis showed TLC of 1500, DLC – Polys- 70%, Lymphocytes- 30%, Total Protein-3.2gm, Glu-93mg/dl. Ascitic fluid AFB-negative, ADA Normal, gram stain and culture was sterile & gene expert was also negative. Pleural fluid examination was normal showing transudative effusion. So a diagnosis of lupus peritonitis was made. Cultures done from body fluids and blood were all negative except Urine culture which grew-E.coli with a colony count of 10^4 . So a final diagnosis of SLE with lupus nephritis stage IV with lupus peritonitis with UTI was made. Patient was started on 3rd generation cephalosporins & carbapenems to which she responded and became afebrile. Repeat urine analysis showed 1-2 pus cells/HPF and culture was sterile. Now patient was planned for immune-suppression & High dose steroids (pulse therapy) with cyclophosphamide was given. Ascitic fluid analysis was repeated which showed a decrease in TLC count to 50-100 cells, P- 40%, L-60%, negative culture and negative gene expert. As she also had protein loss with albumin of 1.6 so albumin replacement was done. Patient was also started on prophylaxis with septran, acyclovir & fluconazole. After 6-7 days of cyclophosphamide, she again started having fever urine r/e – 1-2 pus cells but culture grew E.coli. Antibiotics were given as per sensitivity. Patient developed sepsis and started having bleeding from multiple sites due to sepsis with DIC & succumbed to death.

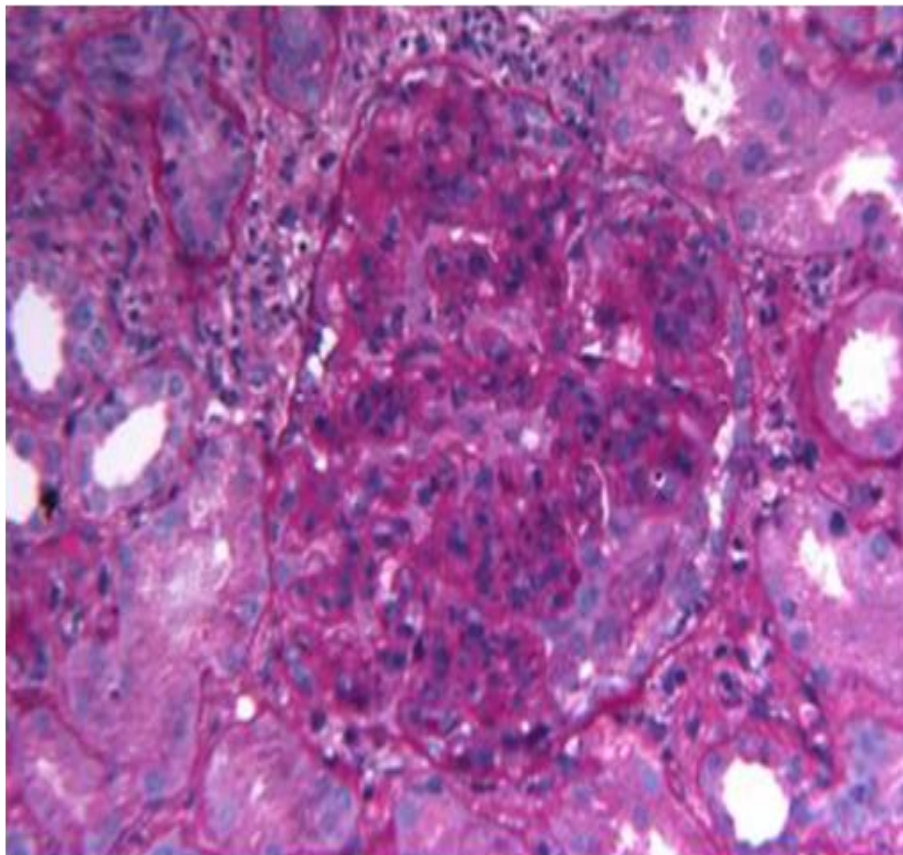


Figure 1: Kidney, Needle Biopsy:

1. Features are Compatible with Diffuse Lupus Nephritis Displaying Active Lesions (Cellular Crescents, Endocapillary Proliferation with Neutrophil Exudation, Wire Loop Subendothelial Deposits, Interstitial Inflammation) and Focal Secondary Segmental Tuft Sclerosis (in One Glomerulus).
2. ISN/RPS Class IV-G (A/C).
3. NIH Indices of Activity 13/24, Chronicity 1/12.
4. There are Features of Patchy Acute Tubular Injury with Interstitial Oedema.

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DISCUSSION

Systemic lupus erythematosus is an autoimmune disorder with clinical manifestations that can affect any organ system (Rahman and Isenberg, 2008). Inflammation of pleural and pericardial serous membranes is relatively common constituting one of the 11 American colleges of rheumatology criteria for classification of SLE (Tan *et al.*, 1982).

A prospective study done with 1000 European patients of SLE found that frequency of serositis was 16% (Man and Mok, 2005). Peritoneal serositis with massive ascites is extremely rare presentation of SLE. There are so many gastrointestinal manifestations of SLE but these are often underestimated as some are nonspecific and in some abdominal symptoms are absent (Tian and Zang, 2010). Whenever a patient of SLE presents with abdominal symptoms along with abdominal distension, many differential causes need to be considered like mesenteric vasculitis, protein losing enteropathy, intestinal pseudo obstruction, acute pancreatitis, or spontaneous bacterial peritonitis. Ascites can be acute or chronic, with or without abdominal pain. Common factors implicated for development of ascites are: portal hypertension or peritoneal disease. In portal hypertension, ascites is formed by increase in vascular hydrostatic pressure, classically resulting in a transudative ascites. However, in damage of peritoneal tissue (inflammation or neoplastic, vessel permeability is altered, originating a protein rich exudative fluid (Hou and Sanyal, 2009). So, a specific diagnostic approach is initiated by use of diagnostic paracentesis. CT scan of abdomen should also be done to rule out other causes as lupus peritonitis is diagnosis of exclusion (Chng *et al.*, 2010). Our patient was also thoroughly investigated and all other causes including spontaneous bacterial peritonitis, perforation peritonitis, intestinal pseudo obstruction were ruled out. The case was different from previous ones as massive ascites has rarely been seen in patients with lupus peritonitis and its acute presentation has also been reported only in few cases (Habib- Agahi *et al.*, 2007).

Conclusion

Even though lupus peritonitis is diagnosis of exclusion, extensive investigations should be done to rule out other causes.

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