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CASE REPORT
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ASCITES AS THE EARLY PRESENTING SIGN OF SYSTEMIC LUPUS ERYTHEMATOSUS

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ABSTRACT
Ascites is rare and uncommon presenting sign in systemic Lupus Erythematosus (SLE). A 17-year old female presented with fever, decreased appetitie and abdominal distention and was found to have enlarged axillary lymph nodes, anemia, thrombocytopenia, deranged LFTs and raised ADA on asciitc fluid. Initially treated with antituberculous therapy but later on she was found to have positive antibodies for SLE. She was treated with methylprednisolone and improved.

Keywords: SLE, ascites, serositis, lupus peritonitis

INTRODUCTION
Systemic Lupus Erythematosus (SLE) is an autoimmune multisystem disease that can affect any organ system of body and has variable presentation in each patient. Its diagnosis can be easily missed if the initial presentation is unusual, and for this reason it is considered as one of the great imitators of medicine.[1] Serositis in SLE occurs only in 9.42% patients as an initial manifestation, and ascites is rare.[2] However, a local study in Pakistan showed that serositis was an early manifestation of SLE in 39.3% patients.[3]
This is the case report of a young girl with ascites

CASE REPORT
A 17 year old female patient admitted in Medicine Department with fever (101F), decreased appetite, weight loss of 10kg for the last 2 months, mild abdominal pain and abdominal distention for the last 15 days. She had history of watery loose stools and vomiting 3 months ago for which she took treatment from local clinic. She had history of occasional productive cough with white sputum 2 months ago. She also gave history of patchy hairfall. However there was no history of recurrent oral ulcers, photosensitivity, dry eyes, dry mouth, joint pains, myalgia, shortness of breath, fatigue, bruises, burning micturition, foamy/frothy urine, orthopnea, paroxysmal nocturnal dyspnea and wheezing. There was no history of lumps/bumps on body, rash, tenesmus, fecal urgency, hand or feet swelling. On further inquiry, her elder sister had asthma and her cousin had intestinal tuberculosis. Patient was living in slums and drinking unfiltered tap water.
On examination, she was hemodynamically stable. Pallor positive. Good orodental hygiene. Palpable axillary and anterior cervical lymph nodes 1.5x1.5 cm. No signs of respiratory distress. Chest

auscultation revealed mild bilateral ronchi. Abdominal examination revealed soft distended abdomen with audible bowel sounds and positive shifting dullness. Mild 1+ pitting pedal edema. No sacral edema. No rash or bruises.

Laboratory studies revealed elevated ESR (ESR: 101), anemia (hemoglobin: 9.0 g/dL), leucopenia (WBCs: 3.73), thrombocytopenia (platelets: 111). Liver function test showed normal bilirubin levels, elevated aminotransferases with ratio reversal (AST: 247U/L ; ALT: 78U/L), hypoalbuminemia (albumin: 1.1g/dL), elevated alkaline phosphatase (ALP: 624 U/L) and elevated gamma GT (GGT: 814 U/L). Serology for hepatitis B, C and HIV was negative. Serum creatinine was 0.4 mg/dL. Urine complete examination revealed 2+ protein, 3+ blood, numerous RBCs, 2-4 pus cells and granular casts. Echocardiography normal. Raised ADA (144.3 U/L) on ascitic fluid. HRCT Chest showed mild left pleural effusion, enlarged bilateral axillary lymph nodes and prominent subcentimeter mediastinal lymph nodes. (Figure 1). CT Abdomen and pelvis with contrast showed gross ascites and enlarged bilateral inguinal lymph nodes (figure 2). Urine analysis showed 2+ protein. Elevated spot urine protein creatinine ratio 7840mg/g (normal < 200mg/g). Normal serum ceruloplasmin. ANA by ELISA was weak positive. Lymph node biopsy, Anti-DsDNA, ENA profile and ascitic fluid complete analysis were awaited. Initially patient was started antituberculous therapy according to her weight in view of raised ADA on ascitic fluid. On 4th admission day, she had yellow sclera and dark colored urine. Serology for hepatitis A and E were sent, and serology for hepatitis E came out reactive. LFTs were repeated that showed elevated bilirubin (Total: 1.4mg/dL, Direct: 1.5 mg/dL), elevated aminotransferases with ratio reversal (AST: 209U/L ; ALT: 65U/L), hypoalbuminemia (albumin: 1.3g/dL), elevated alkaline phosphatase (ALP: 686U/L) and elevated gamma GT (GGT: 1063U/L).

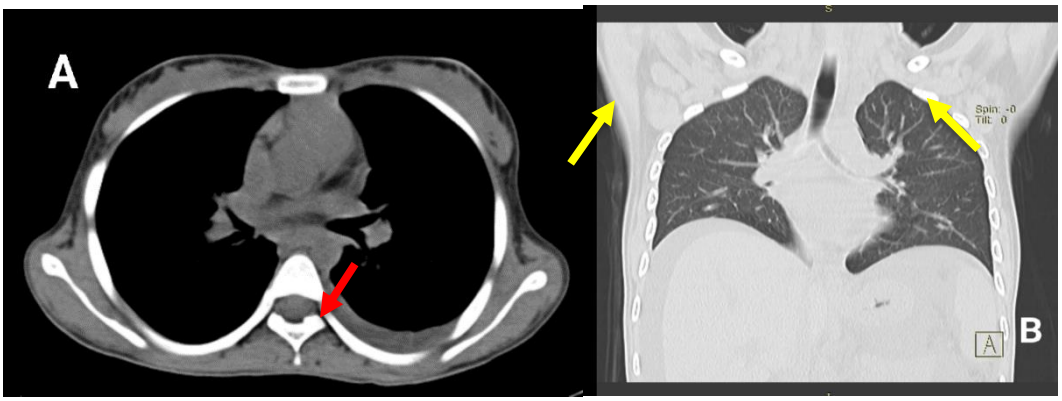

Figure 1. HRCT Chest

Axial view (panel A): left pleural effusion shown by red arrow.
Coronal view (panel B): enlarged bilateral axillary lymph nodes shown by yellow arrow

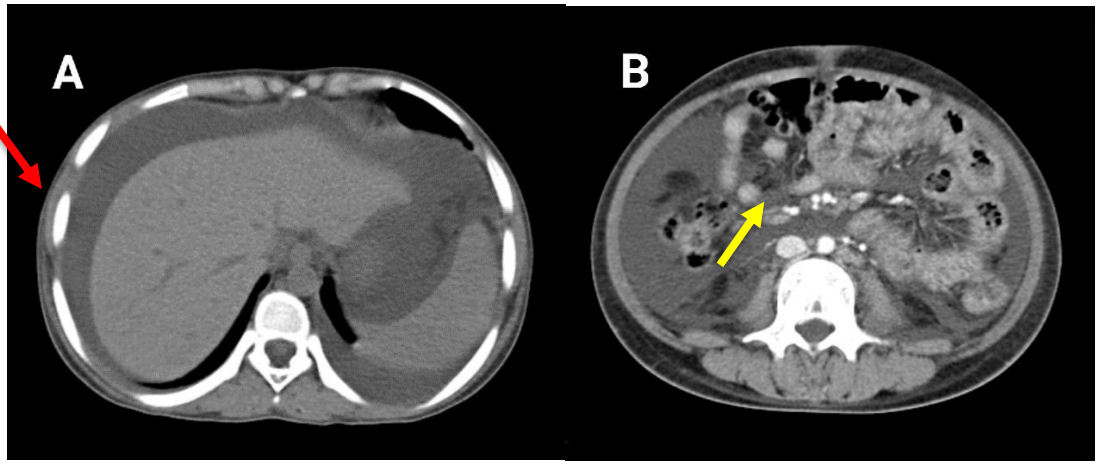

Figure 2. CT scan Abdomen

Axial view (without contrast; panel A): moderate to gross ascites shown by red arrow
Axial view (with contrast; panel B): enlarged paraortic lymph nodes shown by yellow arrow
Gastroenterology opinion taken and they asked to monitor LFTs and continue ATT. Repeat serial LFTs showed decreasing trend. Ascitic fluid analysis showed transudative ascites. ATT was continued. Thyroid profile done which showed hypothyroidism (TSH:10.8uIU/mL, Free T4:0.46ng/dL, Free T3: 1.0pg/mL), so thyroxine started. On 13th day of admission, anti-DsDNA came positive. ENA profile by Quantrix revealed positive antibodies for SLE (Table 1). Direct coombs test was weak positive.

Table 1 ENA QUANTRIX 25 IgG

Antibodies	**Result (U/mL)**
Anti Nucleosome	75
Anti Ds DNA	74
Anti Histones	8
Anti Sm	1
Anti Ribosome	3
Anti PCNA	3
Anti Rnp Igg 68Kd/A/C	1
Anti Sm/Rnp	1
Anti – Ro (Ss-A) 60Kd	22
Anti – Ro (Ss-A) 52Kd	2
Anti-La (Ss-B)	2
Anti-Scl-70	3
Anti CENP-A/B	7
Anti Ku	59
Anti Pm/Scl	1
Anti Srp 54	1
Anti Jo-1	1
Anti PL-7	1
Anti PL-12	1

Patient was shifted under Rheumatology care. Therapy was started with hydroxycholorquine 200mg every 24 hourly and IV methylprednisolone 100mg every 24 hourly for 5 days and then increased to 250mg 12 hourly. The report of right axillary lymph node biopsy showed reactive lymphoid hyperplasia but no evidence of casseous necrosis and malignancy. So antituberculous therapy was stopped. Patient developed cellulitis at the biopsy site which was managed conservatively with IV antibiotics, and dressing done. Patient improved clinically after IV methylprednisolone and hydroxycholorquine. Repeat urine complete examination showed 1+ protein, 14-16 RBCs and no casts. CBC and LFTs were in improving trend. Patient was discharged home on oral prednisolone and hydroxycholorquine, with followup in OPD. Renal biopsy and cyclophosphamide infusion in plan.

DISCUSSION

Systemic lupus erythematosus is a chronic autoimmune disorder that can affect any organ system of body[4]. The most common presenting features of SLE are fatigue (91.8%), joint pains/arthralgia (90.2%), fever (88.5%), oral ulcers (86.9%), alopecia/patchy hairfall (86.9%), malar rash (83.6%),

and photosensitivity (80.3%). Among systemic manifestations, renal involvement is most common (75.4%).[5] However, out of these symptoms, this patient only had patchy hairfall and fever. When thorough workup was carried out, it was revealed that renal system in this patient was also affected, however, patient had no signs and symptoms of renal impairment. According to EULAR (European League Against Rheumatism) / ACR (American College of Rheumatology) 2019 criteria of SLE, pleural and pericardial effusions are specifically included as serosal manifestations, but they have not mentioned peritoneal serositis in the criteria.[6]

Approximately 10% patients diagnosed with SLE[7] have ascites due to inflammation of peritoneum termed as lupus peritonitis. Ascites in SLE is exudative. Lupus exudative ascites is a diagnosis of exclusion, and other causes of exudative ascites such as nephrotic syndrome, intestinal/peritoneal tuberculosis, peritoneal carcinomatosis and pancreatitis should be investigated first. However in this patient ascitic fluid was transudative. All other possible causes of ascites were excluded in our patient. The underlying mechanism of ascites in SLE is not fully understood. However a proposed theory is inflammation of the peritoneum leading to increased permeability and accumulation of exudative peritoneal fluid.[7] The ascites usually responds well to glucocorticoids as is the case in our patient.

CONCLUSION

Patients diagnosed with SLE rarely present with ascites. A clinican must consider serositis secondary to SLE in the differential diagnosis of ascites when all other possible causes of ascites have been ruled out.

PATIENT CONSENT

Informed consent taken from the attendant of patient.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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