Case Report

A case of SLE with pancreatitis

Karan Jain*, Amit A. Palange, Vijayashree S. Gokhale, Arjun Lal Kakrani

Department of Medicine, Dr. D.Y. Patil Medical College and Hospital, Pimpri, Pune, Maharashtra, India

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*Correspondence:
Dr. Karan Jain,
E-mail: karanjain503@gmail.com

ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic, autoimmune inflammatory disease characterized by the presence of autoantibodies, immune complex formation, and multiple organ system involvement. Gastrointestinal manifestations are common in SLE patients, but acute pancreatitis is rare. Here we present a case of a 23 yrs. old male who came to the medicine OPD with the chief complaints of pain in abdomen, swelling all over the body since, 8 days and multiple joint pain for 1 and half months. On examination he was febrile pallor present with anasarca, periorbital edema with heliotrope around both eyes. Dry and xerotic skin over the face, butterfly rash present. Blood investigations, USG and CECT suggestive of acute pancreatitis. Patient was treated in ICU for pancreatitis. Patient was found to be hypothyroid and treated with thyroid supplements. ANA BLOT was suggestive of SLE. Renal biopsy showed diffuse proliferative lupus nephritis. Hence our patient had pancreatitis possibly due to SLE induced hypertriglyceridemia.

Keywords: Acute pancreatitis, Anasarca, Butterfly rash, Hypertiglyceridemia, Hypothyroidism, Lupus nephritis, Renal biopsy, SLE

INTRODUCTION

Systemic lupus erythematosus is an autoimmune disease in which organs and cells undergo damage initially mediated by tissue-binding autoantibodies and immune complex.1

In most patients, autoantibodies are present few years before the first clinical symptom appears. Patients present with fever, weight loss and mild lymphadenopathy which suggest active inflammatory disease. Raynauds phenomenon is common with arthralgia or arthritis. Rash is common in SLE and is classically precipitated by exposure to UV light (sunlight), classical butterfly rash, subacute cutaneous lupus erythematosus, discoid lupus lesions. The most common manifestation in cardiovascular system is pericarditis and myocarditis. Nausea with vomiting and diarrhea can be the manifestation of an SLE flare. There can be diffuse abdominal pain due to autoimmune peritonitis and/or intestinal vasculitis. Acute pancreatitis as the presentation of SLE is a rare occurrence.3

CASE REPORT

A 23 yrs. old male with no other known comorbidities came to the medicine OPD with the chief complaints of swelling all over the body for 8 days and multiple joint pain for 1 and half months. He had pain in the both shoulder, wrist, knee, ankle and proximal and middle interphalangeal joints. With morning stiffness lasting for 1 and half hours. He developed high grade fever on day of admission. No h/o decreased urine output. Not a k/c/o DM/ HTN/ TB/ Bronchial asthma. Herein h/o any substance abuse. On examination he was febrile pallor present with anasarca, Periorbital edema with heliotrope around both eyes. Dry and xerotic skin over the face, butterfly rash present. USG abdomen and pelvis showed focal pancreatitis involving the tail of pancreas and right sided pleural effusion.
Contrast Enhanced Computed Tomography of abdomen and pelvis: Hepatomegaly with bulky tail of pancreas with fat stranding and minimal free fluid in the pelvis. Pancreatitis was managed by keeping him nil by mouth and adequate IV hydration. Patient was found to be hypothyroid and treated with thyroid supplements.

ANA BLOT: SS-A, Nucleosomes were strongly positive, Ro-52, ds-DNA; Histones were positive-ANCA, P-ANCA: negative. Complement C3, C4 reduced. Renal biopsy showed diffuse proliferative lupus nephritis. ISN/RPS class 4 (A)(G), Activity score: 14/24, Chronicity score: 0/12.

We treated the patient with adequate IV hydration, antibiotics, pulse therapy of methyl prednisolone, cyclophosphamide, thyrroxine, statins. The patient improved and is on regular follow up.

Table 1: Lab investigations.

<table>
<thead>
<tr>
<th>Investigations</th>
<th>Results</th>
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<tbody>
<tr>
<td>Haemoglobin</td>
<td>6.4 gm/dl</td>
</tr>
<tr>
<td>Urine protein</td>
<td>+</td>
</tr>
<tr>
<td>Serum triglyceride</td>
<td>522 mg%</td>
</tr>
<tr>
<td>Serum amylase/ lipase</td>
<td>284/90 iu/l</td>
</tr>
<tr>
<td></td>
<td>↓</td>
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<td></td>
<td>733/1500 iu/l</td>
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DISCUSSION

SLE is an autoimmune disease involving multiple organs in which injury is mainly caused by deposition of immune complexes and binding of antibodies to various cells and tissues. Common in 2nd – 3rd decade Female: Male – 9; 1 (our patient was a 23 yrs. male). Gastrointestinal, manifestations are common in SLE patients, but acute pancreatitis is rare.1,2 The incidence of clinical acute pancreatitis associated with SLE varies from 0.7 to 4%, with the diagnosis of pancreatitis is based on clinical, laboratory and CT criteria.3 The pathogenic mechanism of SLE-related acute Pancreatitis however is very complex and multifactorial. Vascular damage (including vasculitis, intimal thickening, occlusion of arteries, and arterioles) by Immune complex deposition. Autoantibody production, abnormal cellular immune response, and drug toxicity may be responsible for the development of pancreatitis.4

The Hopkins lupus cohort reported the largest case series with 63 SLE attribute pancreatitis out of 1740 SLE patients (3.5%), and a Taiwan series reported 40 out of 2976 SLE patients (1.34%).5,6 In a study conducted by yanlong yang, yujin ye, liqiuqiang, et al., 27 out of 4053 SLE patients were diagnosed as SLE-related Acute pancreatitis, with an overall prevalence of 0.67%, annual incidence of 0.56% and mortality of 37.04%.

SLE patients with acute pancreatitis presented with higher SLE Disease activity index score SLEDAI (21.70 ± 10.32 versus 16.17 ± 7.51, P = 0.03), more organ systems involvement (5.70 ± 1.56 versus 3.96 ± 1.15, P = 0.001), and higher mortality (37.04% versus 0, P = 0.001), compared to patients without acute pancreatitis.7 Severe acute pancreatitis (SAP) patients had a significant higher mortality rate compared to mil (MAP) (75% versus 21.05%, P = 0.014), 16 SLE - related Acute pancreatitis patients received intensive glucocorticoid treatment, 75% of them exhibited favorable prognosis.8

In accordance with other literatures, the manifestations of SLE - related acute pancreatitis in this patient was nonspecific and similar to non – SLE acute pancreatitis. Abdominal pain (92.59%), fever (77.78%), and nausea / vomiting (74.07%) were the most common symptoms. These symptoms could also be attributed to other gastrointestinal disease or adverse reactions of medication and may lead to misdiagnosis in general practice. It was reported that the rate of misdiagnosis of AP in SLE was upto 88.6%.9

In Hopkins cohort, appropriate treatment with corticosteroids added a survival benefit in SLE – related acute pancreatitis.5 Our patient did not receive glucocorticoids. There is still a controversy over steroid treatment in SLE-related acute pancreatitis. Increasingly accumulated evidence showed that steroids do not trigger acute pancreatitis or cause increased mortality on acute pancreatitis, but instead, they have a possible therapeutic effect on SLE – related pancreatitis.8,10

CONCLUSION

SLE - related acute pancreatitis is rare, but associated with high – mortality rate, which is even higher in those severe acute pancreatitis with multiple organ system involvement. Activity of SLE, hematological system, renal, and liver injury in SLE patients may attribute to the mortality of acute pancreatitis. Early diagnosis of acute pancreatitis in SLE patients, especially those with abdominal pain, and appropriate glucocorticosteroid treatment is beneficial for a better therapeutic outcome in the majority of patients.

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REFERENCES


