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A RARE PRESENTATION OF SYSTEMIC LUPUS ERYTHEMATOSUS-ACUTE PANCREATITIS

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Abstract :

Acute pancreatitis is a rare presentation of systemic lupus erythematosus.Here we present a case who presented to us with acute abdomen ,diagnosed to have acute pancreatitis and later diagnosed to have SLE.Though mortality of acute pancreatitis in SLE is high,our patient was successfully treated and got discharged.

Keyword : Acute pancreatitis,Systemic Lupus Erythematosus

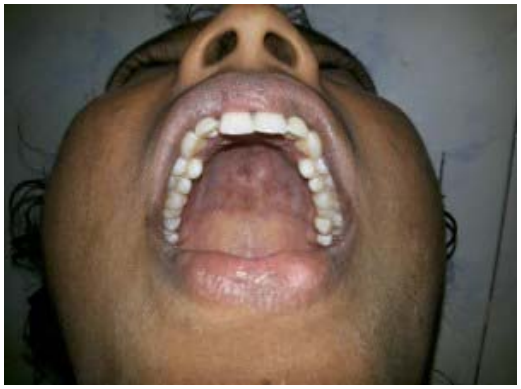
INTRODUCTION:

SLE is an autoimmune disorder that affect a variety of organ systems characterized by a plethora of autoantibodies and immune complex formation and varying clinical presentation. Musculoskeletal, Mucocutaneous and Renal systems are commonly affected.

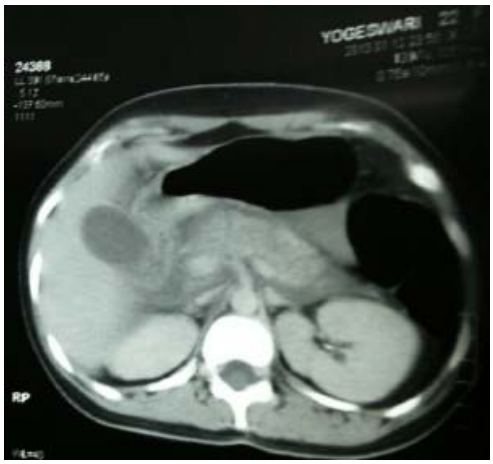
CASE HISTORY:

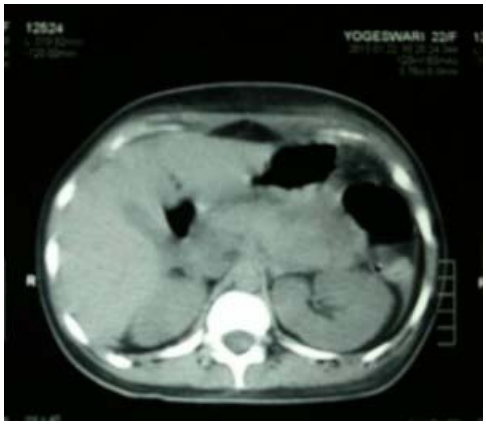
28 yrs old female presented with complaints of sudden onset of severe intractable abdominal pain of 1 week duration. It was predominantly upper abdominal pain, with pain radiating to the back and relieved on bending forward. There was no history of aggravating or relieving factors.History of nonbilious ,non projectile vomiting was present.No history of loosestools or constipation or altered bowelhabits or abdominal distension. On probing she gave history of low grade intermittent fever of 2 months on and off preceding this event.She also gave history of oralulcers, photosensitivity, alopecia and fatigue. She denied history of loss of apetite and loss of weight.There was no history of jointpain, raynauds, muscle weakness, seizures and skin lesions preceding this episode.There was no significant personal , family or drug history. **On examination** she was thin built,pale and febrile. Malar rash was present over both the cheeks and erythematous macular lesions were present over extensor aspect of both arms.She had diffuse hairloss over the scalp and oral cavity revealed an active ulcer over the hard palate..There was diffuse tenderness over the abdomen predominantly over left hypochondrium

There was no free fluid or organomegaly.Other systems were normal.Her vitals were stable with no evidence of hypotension .



HEALED PALATAL ULCER Investigations revealed a Haemoglobin of **8.6gm/dl,**Total count of 4600cell/mm3and Differential count, N 53,L42,E 3**,Platelets-50,000**and **ESR 116mm/hr**.The Peripheral smear was normal and Direct Coombs test was negative All biochemical investigations were normal except SGOT which was 69IU/L,,CPK was 490IU/L,,LDH 497IU/L.**Serum amylase** was **2660IU/L**,**S.Lipase** was **2139IU/L** .Lipids were normal.Urine analysis was normal Her immunological profile revealed positive Antinuclear antibody titre 1:160 by Hep2and speckled pattern by indirect immunofluorescence and extractable nuclear antigen was positive for anti Sm ,anti-Ro antibodies.Her anti dsDNA was 1:80 positive and complements were normal,anticardiolipin antibodies was negative and Lupus anticoagulant was not detected.Her blood and urine cultures were negative. Fever profile was negative.Ultrasound abdomen showed minimal free fluid. **CT abdomen** showed bulky Pancreas with loss of attenuation,Peripancreatic fat stranding ,Gastric lesser curvature thickening,.thickening of bilateral pericolic fat and fluid in pericolic space and bilateral





CT ABDOMEN-ACUTE PANCREATITIS

CT ABDOMEN-RESOLVING PANCREATITIS

With the above clinical ,immunological ,radiological,lab investigation a diagnosis of **Systemic lupus erythematous with acute pancreatitis** was made.She satisfied 6/11 of ACR criteria. Her SLEDAI score was 14. She was treated as in patient with high dose steroids ,antibiotics and intravenous fluids. patient well being improved, abdominal pain subsided,she was started on oral feeds.she was discharged after 20 days with oral prednisolone and chloroquine.Opinions obtained were-Haematology –Microcytic Hypochromic Anemia,

Dermatology-SLE Acute skin lesions,Medical gastroenterology –Acute Pancreatitis,Nephrology- Nil active intervention CT ABDOMEN was repeated before discharge and it showed peripancreatic collection and minimal Peripancreatic strands .Compared to old film the collection had decreased and radiological picture was suggestive of resolving pancreatitis. Repeat amylase was 230 IU/l and serum lipase 213 IU/l. This case is presented because of rare presentation of acute pancreatitis as the first presenting manifestation of SLE.At present patient is doing well.She is on low dose steroids, chloroquine and MMF and is on regular follow up at our department.No further episodes of abdominal pain.

DISCUSSION :

Acute pancreatitis is a rare but life threatning complication of SLE The estimated annual incidence is 0.4–1.1/1000 lupus patients. . It is estimated that 30.5% of asymptomatic SLE patients have hyperamylasemia.The pathogenic mechanism of SLE-related pancreatitis is multifactorial complex and unclear. It could be either due to a vascular phenomenon or an autoimmune inflammatory reaction.Most common is an autoimmune reaction involving abnormal cellular immune response or antibody reaction rather than vasculitis,that is responsible for the intense inflammatory reaction leading to acute panreatitis.[1] Postulated mechanism of vascular damage includes necrotizing vasculitis, occlusion of arteries and arterioles by thrombi resulting from severe hypertension or antiphospholipid syndrome, intimal thickening, proliferation and immune complex deposition with complement activation in the wall of pancreatic arteries.[1] Other traditional predisposing factors include Hypertriglyceridemia, steroids and Azathioprine. 57% of SLE related pancreatitis develop complication if not treated promptly and mortality rate is 45% with fatal complications compared to 3% without complications.[2,3] High lupus activity is associated with increased mortality and also patients with concurrent central nervous system and cardiac involvement. Risk factors for increased mortality include increased serum creatinine, hypoalbuminemia, anti- ds DNA antibodies, thrombocytopenia, low complement, hypocalcemia, hyperglycemia and elevated liver enzymes. Mortality was 100% in patients developing circulatory shock or acute renal failure, 87% in patients developing respiratory insufficiency, and 77% in patients with infections. About 22% of patients may experience recurrent acute pancreatitis attacks, while 12% of patients develop pancreatic pseudocysts and 5%-14% become chronic. The treatment of SLE pancreatitis is with steroids. Administration of steroids is somewhat controversial, as steroids have been implicated as a cause of SLE pancreatitis.[4] Recent studies have shown that the toxic effect of steroids on the pancreas is probably negligible, whilst their immunosuppressive effect is essential for the improvement of the pancreatitis.[5] Immunosuppressive medications such as azathioprine and cyclophosphamide can be used with steroids. In severe cases plasmapheresis and intravenous immunoglobulin can be used.

CONCLUSION

Pancreatitis should be suspected in any SLE patient with abdominal pain though uncommon. Pancreatitis may be the initial manifestation of SLE. Mechanical and toxic-metabolic etiologies should be ruled out. Acute pancreatitis is often associated with increased SLE activity. In most cases, the onset of

pancreatitis appears unrelated to previous treatment with steroids or azathioprine. Mortality rate appears to be higher than in non-SLE associated pancreatitis. Mortality is related to both the presence of active SLE and several biochemical abnormalities.It should also be remembered that SLE patients may develop pancreatitis secondary to non SLE-related causes, such as biliary stones or alcohol ingestion.

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