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SLE PANCREATITIS: A RARE PRESENTATION

General Medicine

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ABSTRACT

Systemic Lupus Erythematosus presenting as acute pancreatitis is rare. Incidence of acute pancreatitis is 0.7-4% of SLE patients. SLE pancreatitis is more common in females and third decade of life. We present a case of a 25 year old male who presented with acute pancreatitis and later diagnosed as SLE related after ruling out other possible etiologies. An early recognition and appropriate treatment with immunosuppression may result in favorable outcomes.

KEYWORDS

pancreatitis, systemic lupus erythematosus, necrotizing fasciitis, immunosuppression

INTRODUCTION

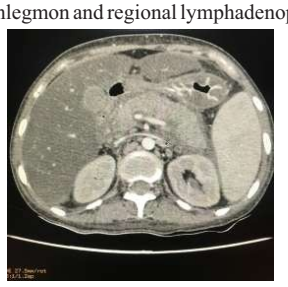
Systemic lupus erythematosus (SLE) is a systemic autoimmune inflammatory disease, with several different clinical manifestations. Its annual incidence is about 5 cases per 100000 inhabitants [1,2]. The prevalence is around 52 cases per 100000 inhabitants. The gastrointestinal tract may be affected, either by the disease itself or by adverse reactions of medications or by opportunistic infections. Clinically, there are four main patterns of gastrointestinal involvement in SLE: mesenteric vasculitis, present in 0.2 to 9.7% of patients; protein-losing gastroenteropathy, with estimated prevalence from 1.9 to 3.2%; intestinal pseudo- obstruction, rare and related to dysfunction of the visceral smooth muscles, enteric nerves and/or visceral automatic nervous system with aperistalsis; and lupus pancreatitis, found in 0.7 to 4% of patients [1,2]. SLE pancreatitis is more common in females and third decade of life. We present a case of a 20 year old female who presented with acute pancreatitis and later diagnosed as SLE related after ruling out other possible etiologies. An early recognition and appropriate treatment with immunosuppression may result in favourable outcomes.

Case Report

A 25 year old male patient presented with a 6 month history of intermittent fever, arthralgias and lethargy, non healing ulcer over the left foot for which local debridement and antibiotic treatment were given. On admission, patient had complaints of epigastric abdominal pain radiating to the back as well as non bilious vomitings.

On examination, patient is febrile, tachycardic, had epigastric tenderness, bowel sounds were absent. Respiratory, cardiovascular and neurologic examination were unremarkable. Local examination of the left foot showed necrotizing fasciitis on the lateral aspect. Her investigations revealed Hb%-9.2 gm/dl,WBC count- 4400 cells/cumm, Platelet count-90000/cumm, RFT showed S.urea-48 mg/dl,S.creatinine-1.8 mg/dl, electrolytes were normal. LFT showed total Bilirubin-2.5 mg/dl,AST-293 U/L,ALT-76 U/L,ALP-367 U/L,S. Amylase was 772 U/L, S. Lipase was 1338 U/L, Corrected S. Calcium was 8.1 mg/dl,S.Albumin-

1.2 gm/dl. Fasting lipid Profile revealed Sr.Cholesterol-156 mg/dl, Sr.Triglycerides-376 mg/dl. Her Abdominal Ultrasonography and CECT Abdomen were consistent with Acute Pancreatitis with peripancreatic phlegmon and regional lymphadenopathy.



CECT abdomen showing acute pancreatitis

Patient was treated conservatively with intravenous fluids and supportive management. Her ANA profile was positive for ANA, dsDNA, anti-histones, anti nucleosomal antibodies supporting the diagnosis of SLE. After ruling out other possible etiologies a provisional diagnosis of SLE pancreatitis was made and patient was started on high dose steroid therapy. During subsequent hospital stay patient condition slowly deteriorated and succumbed to death due to complications of acute pancreatitis and hemolytic anemia during SLE flare.

DISCUSSION

Pancreatitis in the setting of SLE was first reported by Reifenstein, [3] and has an estimated annual incidence of 0.4–1.1 per 1000 patients. Common precipitants of acute pancreatitis include hepatobiliary tract disease-related mechanical obstruction and metabolic insults including alcohol, drugs, hypercalcemia, and hypertriglyceridemia. And a diagnosis of acute pancreatitis due to SLE can be made only after ruling out other possible common etiologies. Patients are typically female, with clinically active SLE developing within the first 2 years of disease onset, as in the case presented here [1]. The pathogenic mechanism is still unclear, but vascular damage may be implied. Necrotizing vasculitis, occlusion of arteries and arterioles by thrombi, intimal thickening and proliferation and immune complex deposition with complement activation in the wall of pancreatic arteries have been postulated [1,4]. Direct in - flammation of the parenchyma may result from autoantibody production or abnormal cellular immune response [5,6]. The mainstay of treatment is corticosteroids and supportive care. The use of steroids has shown a reduction in mortality to 20% as compared to 61% when they are not used [7, 8]. Lupus pancreatitis is associated with high mortality up to 45% as compared to non-lupus pancreatitis with increased overall complications like recurrent pancreatitis (22%), respiratory failure (22%), pleural effusion (18%) and ascites (19%) [9].

CONCLUSION

Though SLE pancreatitis is a rare entity and mortality rates are as high as 40% in patients diagnosed with SLE pancreatitis. Early recognition and appropriate treatment with immunosuppression may result in favourable outcomes.

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