ACUTE PANCREATITIS- ATYPICAL INITIAL MANIFESTATION OF SLE: A CASE REPORT

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ABSTRACT-

There are very few literatures on Acute Pancreatitis as an initial manifestation of SLE. SLE is a multisystem autoimmune disease which can present from classical to atypical forms. This case reports a 29-year-old female with SLE whose early presentation was abdominal pain, abdominal distension with vomiting and fever, elevated pancreatic enzymes with pancreatic enlargement in USG and other deranged reports for which the use of steroids led to reduction in clinical symptoms indicating the importance of early diagnosis and management resulting in favorable outcomes.

KEYWORDS searched for were SYSTEMIC LUPUS ERYTHEMATOSUS, Abdominal pain and PANCREATITIS.

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INTRODUCTION

SLE is a multisystemic autoimmune disorder characterized by a broad range of manifestations and findings of antibodies in the blood directed against one or more components of cell nuclei [10]. It has diverse clinical manifestations like mesenteric vasculitis (0.2-9.7%), protein losing enteropathy (1.9-3.2%), rare forms like intestinal pseudo-obstruction, lupus pancreatitis (0.7-4%). Lupus pancreatitis is more common in females in the third decade of life [1,2]. The American Rheumatism Association recommends 4 of the following 11 revised criteria for the diagnosis of SLE: malar rash, discoid rash, photosensitivity, oral ulcers, arthritis, serositis, renal disorders, neurological disorders, hematological disorders, immunologic disorders on serologic testing and anti-nuclear antibodies [10]. Pancreatitis as an initial manifestation of SLE is rare [11]. Acute pancreatitis is a rare but life-threatening complication of SLE [12]. Activity of SLE, multiple organ systems involvement may attribute to severity and mortality of Acute pancreatitis [12]. Appropriate glucocorticoid treatment leads to better prognosis in majority of SLE patients with Acute pancreatitis [12].

CASE REPORT

A 29 years old female, farmer by occupation of low socioeconomic status and no known comorbidities presented with abdominal pain since 4 days which was acute in onset, dull aching in nature, more in the epigastric region which radiated to back associated with nausea and vomiting 2-

3 episodes containing food particles, non-bilious, non-blood tinged. She had 1 episode of low-grade fever, not associated with chills or rigors, relieved on taking antipyretics. Patient also complained of generalized weakness, reduced sleep and loss of appetite for 5 days. Patient denied using any long-term drug or any past medical illness. Patient also complained of multiple large and small joint pain for 15 days.

On examination patient had pallor, was afebrile, pulse rate was 114/min, blood pressure was 110/70mmHg. She had angel wing nail changes with possibility of lichen planus due to SLE as primary cause. Her chest sounds were clear with no added sounds. She had no murmur or rub. Neurologically she was conscious and oriented to time place and person. On per abdominal examination her abdomen was soft, tenderness was present more in epigastric region with minimal guarding, no rigidity.

Routine investigations initially showed that her hemoglobin was low-8 gm/dl TLC- 8500 Platelets- 1 lakh/ cu mm and patient developed a picture of pancytopenia after 2 days (Hb was 7.2gm/dl, TLC was 3500 and Platelets were 60000/ cu mm). On peripheral smear- moderate degree Normocytic Normochromic anemia with moderate thrombocytopenia, lymphopenia, eosinopenia was seen with Retic count- 0.5% and ESR- 26 mm/hour. Her coagulation profile was deranged. Patient had hypocalcemia-6.9 mg%, hypomagnesemia-1.8 mg%, hypokalemia-2.9 mg%. Her sr. creatinine was 2.2, sr. urea was 62. Her pancreatic enzymes were raised, sr. lipase was 540, sr. amylase was 386, LDH was 697. She had Euthyroid status- sr. TSH was 0.75, CRP was 116.20 mg/l and Dengue profile and WIDAL test were negative. Her LFTs were SGPT-82, total bilirubin (direct +indirect) was 2.1(1.8+0.3). HIV, HCV, HBsAg, HAV, HEV were negative. Urine r/m showed Albumin- +2, sugar-absent ,2-3 pus cells, 7-8 RBCs, 7-8 Epithelial cells. Her USG Whole abdomen showed Moderate ascites, Gall bladder sludge with peri GB cuffing secondary to ascites, mildly heterogenous, pancreatic body-1.8 cm, head-2.3 cm; bilateral kidney showed mildly raised renal cortical echotexture. Her MSCT Scan of Abdomen and Pelvis with Contrast showed liver- 17.5cm enlarged, normal in density and echotexture s/o hepatomegaly. Pancreas appeared bulky. Pancreatic head measured 3.5cm and tail 3cm. Moderate to gross ascites with bilateral pleural effusion was seen. Ascitic fluid examination showed turbid appearance, pale yellow, sugar- 61 protein- 2.9 albumin- 1.6 ADA-25u/l LDH-869u/l Total count-4500 lymphocyte-10 polymorph- 60 mesothelial cells-30 in the background of RBC. On KOH stain no fungal element seen. SAAG ratio was 0.5 which was suggestive of exudative origin. Urinary protein concentration was 189.6mg/dl, urinary creatinine was 43.2 mg/dl, UPCR showed 4388.89mg/gm creatinine. Her Connective tissue workup showed ANA was Positive +3 (homogenous nucleus, Cytoplasmic positivity also seen) with SS-A +, ds DNA +, Nucleosomes ++, Histones ++. Presence of ds DNA Nucleosomes and clinical features suggest possibility of SLE. The diagnosis was made by clinical history and examination, biochemical tests and radiological interventions.

The patient was kept nil per oral, IV fluids were given. She was started on antibiotics (Injectable Cefepime plus Tazobactam & Metronidazole), oral steroids at 1mg/kg/day, antiemetics, analgesics, antipyretics, multivitamins with iron, folic acid, pancreatic enzymes supplements. Her hypokalemia, hypomagnesemia and hypocalcemia were corrected. The patient was symptomatically better.

CONCLUSION-

SLE as a cause of pancreatitis should be considered actively in case of acute abdominal pain after ruling out more common etiologies of pancreatitis like alcohol, gall stones, hypercalcemia,

hypertriglyceridemia, certain drugs. ACUTE PANCREATITIS is a rare but life-threatening presentation of SLE. The mainstay treatment of pancreatitis is steroids but it's use is controversial as it is implicated in the cause.

DISCUSSION-

Pancreatitis in the setting of SLE was first reported by Reifenstein [7]. The incidence of acute pancreatitis in SLE varies from 0.7% to 4% and presents as initial manifestation in 22% and within first 2 years of diagnosis of SLE in 60% of all SLE-induced pancreatitis [1, 2]. Alcohol, gall stones, hypertriglyceridemia, hyperparathyroidism, recent viral infection, psychosis and congenital anomalies of pancreas like pancreatic divisum and annular pancreas are common causes of pancreatitis [13]. Acute pancreatitis as an initial manifestation in SLE is multifactorial and may be related to complement activation and vasculitis with microthrombi formation in arteries and arterioles resulting in vascular damage or direct involvement of pancreatic parenchyma by autoantibodies and cellular immune response [1, 3]. The etiology may be acute pancreatitis as initial presentation or later during the course of disease due to either uncontrolled disease activity or drug toxicity. The clinical manifestations of SLE pancreatitis can range from subclinical (an elevation of pancreatic enzymes without clinical symptoms) to acute pancreatitis or chronic pancreatitis (self-limiting) disease course [13]. Patients with SLE and acute pancreatitis have higher systemic lupus erythematosus disease activity index (SLEDAI) score with increased incidence of multiorgan involvement like skin (46%), renal (35%), articular (43%), hematological (24%) and central nervous system (21%) [3, 4]. The case discussed here was of a young female with pancreatitis as the first presentation of SLE with skin, articular and renal involvement. The lupus pancreatitis, unlike non-lupus pancreatitis, has higher incidence of leucopenia (59%) instead of leukocytosis [4]. 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%) [5]. Treatment with azathioprine and glucocorticoids reduces mortality [4,8,9]. The diagnosis in this patient was made by clinical features and examination, biochemical lab tests and radiological interventions along with positive ANA titer and dsDNA status. The use of steroids has shown a reduction in mortality to 20% as compared to 61% when not used [6]. We administered steroids at 1 mg/kg/day and tapered it, over time the proteinuria also subsided along with abdominal pain. The patient showed improvement in her symptoms.

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