**Case Report:**

Pancreatic Pseudocyst as Presenting Feature in Systemic Lupus Erythematosus

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**Abstract:** We report of a case of 16-year-old young tribal girl from rural Maharashtra who had fever, abdominal pain and vomiting. She was first diagnosed with pseudocyst of pancreas by computerized tomography and then with Systemic lupus erythematosus after further investigations. There has been no reported case of SLE and pancreatic pseudocysts here in India, who was treated conservatively.

**Key Words:** Systemic lupus erythematosus; Pseudocyst; Pancreas

**Introduction:**

Systemic lupus erythematosus (SLE) is a multi-system, autoimmune disorder characterized by a broad range of manifestations. The initial manifestations of SLE, however, can involve many organ systems either singly or in combination, which frequently makes diagnosis difficult. Acute pancreatitis is a well known but rare manifestation of SLE which could lead to the development of pancreatic pseudocysts. Abdominal pain is the commonest symptom in pancreatitis (88% of patients) followed by nausea and vomiting but as many SLE patients present with musculoskeletal pain and abdominal symptoms a high index of suspicion will be needed in diagnosing SLE associated pancreatitis or pseudocyst of pancreas.[1] In our literature search, there has been no reported case of SLE and pancreatic pseudocysts here in the India. We report a 16-year-old young girl with fever, abdominal pain and vomiting who was first diagnosed with pseudocyst of pancreas and then with SLE, after further investigations.

**Case Report:**

A 16 years old young tribal girl from rural Maharashtra came to emergency department of our hospital with a 6 days' history of abdominal pain, loss of appetite, occasional nausea and vomiting. She also had a 3 days' history of fever and generalized weakness. On further questioning, she also complained of thinning and loss of hair and weight loss of 3kgs in the past 2 months. Her menstrual cycles were regular. Her family history was unremarkable for diabetes, hypertension and auto-immune disorders. Her body temperature was 38 degree celsius, pulse rate was 100 beats per minute, blood pressure was 120/80mmHg. She had no skin lesions. Her abdomen was soft with a firm and tender smooth surfaced palpable mass in the epigastric region.

On investigations, complete blood count showed a white cell count of 13000/cmm, with 78% neutrophils, 20% lymphocytes, and 2% monocytes. Hemoglobin was 8.9 mg/dL, hematocrit 33%, and platelets 112×10^9/L, ESR was 52 mm in first hour. Her blood chemistry was as follows: sodium 126 mmol/L, potassium 4.4 mmol/L, chloride 97 mmol/L, bicarbonate 22.5 mmol/L, calcium 9.5mg%, blood urea 26mg/dL, serum creatinine 0.7 mg/dL, and glucose 98 mg/dL. Her liver function revealed bilirubin 1.8 mg%, aspartate aminotransferase 63 U/L, alanine aminotransferase 16 U/L, protein 5.6 g/dL, and albumin 3.7 g/L. Her lipid profile and urine examination were normal. Blood culture was negative.

CT scan of the abdomen showed a large pancreatic pseudocyst arising from the body and tail of the pancreas (Fig 1). There was no normal tissue in the body or tail of the pancreas but the head appeared normal. There were no gall stones and the biliary tract was normal.
CT-guided percutaneous drainage of the pancreatic cystic structure revealed brown fluid with red blood cell and high amylase and LDH content (344 U/L and 1,670 U/L, respectively). These findings were consistent with a pancreatic pseudocyst. Cytologic analysis of the pseudocyst fluid did not reveal any malignant cells. Fluid was drained by placing a pigtail catheter percutaneously into the fluid cavity. Her pain abdomen subsided after two days.

On clinical suspicion of systemic lupus erythematosus, we got the antinuclear antibody (ANA) and anti-double-stranded DNA (dsDNA) antibody tests done, which were strongly positive, suggesting a diagnosis of systemic lupus erythematosus with pseudo pancreatic cyst. We treated her conservatively as there was no sign of complication and progression of size of the cyst assessed sonologically. She was put on prednisolone 1mg/kg body weight and she made significant recovery.

Discussion:

Patients with SLE can have manifestations of digestive system, but pancreatitis is rare, and such acute pancreatitis as an initial manifestation is even rarer. Very few such cases have been documented in the literature.[2-6] No reports are available regarding pseudopancreatic cyst as the early feature of SLE. Exact mechanisms involved in the origin of SLE pancreatitis are not clear. This may result from vasculitis, microthrombi, anti-pancreas autoantibody, side-effects of medicines, intimal thickening and virus infection. Most lupus pancreatitis is found in patients with long-standing SLE who have multi-organ involvement and are already on steroid, diuretic, or immunosuppressive therapy, all of which have been implicated in the etiology of pancreatitis. Since the initial description of SLE pancreatitis, whether steroid or SLE is the primary cause has been controversial. This etiological diagnosis assumes importance when decisions regarding steroids as preferred therapy need to be taken.

Our patient was never diagnosed as SLE or pancreatitis before the admission. On abdominal imaging, she was found to have pseudopancreatic cyst, a known complications of pancreatitis characterised by localized fluid collection surrounded by a wall of fibrous tissue that is not lined by epithelium.[7] The pathogenesis of pseudocysts seems to stem from disruptions of the pancreatic duct due to pancreatitis or trauma followed by extravasation of pancreatic secretions. In case of acute pancreatitis if fluid collection persists more than 4-6 weeks, and is well-defined by a wall of fibrous or granulation tissue, can one say that an acute pseudocyst has appeared. Such a pseudocyst usually contains enzymatic fluid and necrotic debris. In chronic pancreatitis, pathogenesis is less well understood and proposed mechanisms may be as a consequence of an acute exacerbation of the underlying disease and/or blockage of a major branch of the pancreatic duct by a protein plug, calculus or localized fibrosis.[8]

In this case, the classical features of pancreatitis may have been masked due to underlying SLE. General physicians should be aware of the different ways in which SLE can present to ensure its early diagnosis and treatment or at least to halt the progression/complication of this disease.

References: