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Case Report

Tubercular splenic abscess and recurrent acute on chronic tubercular pancreatitis: a rare coincidence in an immune competent child

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ABSTRACT

Tubercular splenic abscess in association with chronic tubercular pancreatitis is scarcely reported in an immune competent child. 12 years old boy had presented with recurrent attacks of upper abdominal pain radiating towards left shoulder. He had multiple hospitalizations with similar complaints. The child had associated anorexia and significant weight loss during the period of his illness. Clinical examination revealed tenderness and muscle guard over the epigastric region. The pancreatic enzymes were markedly raised. Abdominal CT scan had suggested chronic pancreatitis with multiple splenic abscesses. The cartridge- based nucleic acid amplification test (CBNAAT) of the pus aspirated from the abscess confirmed the presence of pan sensitive mycobacterium tuberculosis. Image guided fine needle aspiration biopsy revealed the presence of caseating granulomatous lesion. Treatment with anti-tubercular drugs led to complete clinical recovery and normalization of pancreatic enzymes at follow up. An immunocompetent child had chronic tubercular pancreatitis associated with tubercular splenic abscess.

Keywords: Tubercular pancreatitis, Tubercular splenic abscess, Immune competent child

INTRODUCTION

Tuberculosis still remains one of the major health problems in developing countries. Among the extra pulmonary sites of involvement, tuberculosis of the spleen and Pancreas are exceedingly rare. Retrospective case series review of adult literature has quoted a very low incidence.[1] Extensive search of contemporary paediatric literature had revealed a mere handful of cases where isolsted tubercular splenic abscess were reported in immune competent children. Though at times associated with acute pancreatitis, formation of a splenic abscess in chronic pancreatitis is rare. The abscesses associated with pancreatitis are most frequently localized either in pancreas itself or in liver.[2] An isolated splenic abscess particularly in an immune competent child has been rarely reported.[3] This is a unique coincidence where recurrent acute exacerbations of chronic tubercular pancreatitis were complicated by multiple tubercular splenic abscess in an immune competent child.

CASE REPORT

A 12 years old boy had presented with recurrent upper abdominal pain, which was radiating towards the left shoulder. These were associated with intermittent fever, anorexia, nausea and weight loss. The child had six hospital admissions in one year presenting with similar complaints. Every time the clinical examination revealed marked epigastric tenderness and muscle guard. The pancreatic enzymes were always significantly raised (Table 1) with serum amylase ranging between 226 and 564 U/L and serum lipase between 466 and 1021 U/L. The liver function tests and lipid profiles were normal. The blood glucose was always normal and the highest glycosylated hemoglobin (HbAic) was 5.8. The total WBC count was ranging between 8800 and 12600 with

neutrophilia. The CRP was always elevated. The PT an APTT were normal. The immunoglobulin assay, absolute CD4, CD8 count and the CD4:CD8 were within normal range. The serology for HIV and the anti-hepatitis An IgM, HBsAg and anti-hepatitis E IgM were negative. There was marginal elevation of fecal fat excretion (25 g/d, normal<7 g/d). The genetic analysis for PRSS gene mutation for hereditary pancreatitis was negative and the sweat chloride was normal. The ultrasound revealed bulky pancreas, pancreatic calcification and peri pancreatic collection. The CT scan (Figure 1) showed atrophic pancreas as well as multiple ill-defined enhancing hypo dense lesions with central fluid in upper and mid poles of the spleen and tail of the pancreas with peri-splenic collection suggestive of splenic abscess. MRCP features suggested acute exacerbation of chronic pancreatitis with inflammatory stenosis at distal end of common bile duct.

Table 1: Trend of various investigations performed during multiple hospitalizations.

Investigations	Dec 2017	Feb 2018	May 2018	Aug 2018	Oct 2018	Dec 2018	Feb 2019
TC per microlitre	8800	7400	7650	7420	10900	11200	12600
CRP (mg/dl)	42	38	64	29	56	62	86
Amylase (U/l)	226	308	264	356	234	462	564
Lipase (U/l)	466	502	564	640	796	802	1021
Sugar fasting (mg/dl)	86	92		106		74	82
HbAic	5.2		5.6		5.4		5.8
Fecal fat over 24 hours (N<7g)		25 g		28 g		26 g	
CD4, CD8,		Normal			Normal		
Immunoglobulin assay		Normal			Normal		
Cholesterol mg/dl	117		124		114		120
Triglycerides mg/dl	72		82		78		86

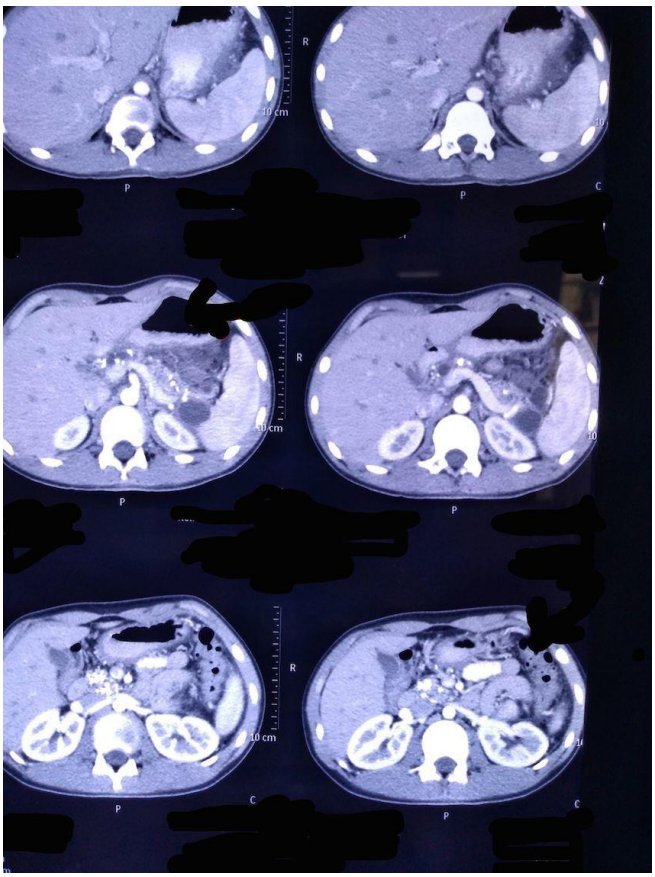

Figure 1: CT Scan showing chronic pancreatitis and multiple hypodense splenic abscess.

A single setting USG guided aspiration of one of the bigger abscess cavities had revealed straw colored exudative fluid. The protein in the aspirated fluid was>4.5 gm while the amylase and lipase were normal, proving that the abscess did not have any pancreatic connection. The cell count was markedly raised (>1000) with a mixture of polymorphs and lymphocytes. The culture of the aspirated fluid had revealed the presence of multiple methicillin sensitive staphylococcus aureus and mycobacterium tuberculosis. There were no fungal elements isolated. The CBNAAT was also positive (pan sensitive mycobacterium tuberculosis) proving a tubercular splenic abscess with probable bacterial super infection. The child was treated with broad spectrum intravenous antibiotics as per the sensitivity pattern. He was also prescribed anti tuberculosis drugs (2 months of INH, rifampicin, pyrazinamide and ethambutol followed by 4 months of INH and rifampicin). Pancreatic enzyme supplementation was continued and he had made complete resolution of clinical symptoms within 3 months with satisfactory weight gain.

DISCUSSION

An isolated splenic vein thrombosis, intra splenic pseudocyst, splenic hematoma and severe bleeding from eroded splenic vessels.[4] are known splenic complications of acute on chronic pancreatitis. Though an intra splenic abscess has been described in connection with acute pancreatitis, but it is unknown in chronic calcifying pancreatitis.[5] Tuberculosis is a multi-systemic disease

with lung being the commonest site of involvement. In developing countries like India, all primary care physicians should be aware of both the pulmonary and extra pulmonary manifestations of Tuberculosis. Extra pulmonary disease accounts for almost 15-20% of all cases of tuberculosis.[6] Splenic tuberculosis was first described in 1846 by Coley, however it remains an extremely rare form of tuberculosis particularly in immune competent hosts. There are only a few case reports of splenic tuberculosis in immune competent patients.[7] This child had chronic calcifying pancreatitis with recurrent acute exacerbations along with multiple splenic abscess of tubercular etiology. He was not immune deficient and there was no evidence of either significant exocrine or endocrine pancreatic insufficiency apart from mild steatorrhoea. The question remained whether tuberculosis was the cause of the chronic pancreatitis in this child. The points in favor of tubercular pancreatitis were the chronic history, the presence of systemic signs like weight loss and anorexia. However recurrent acute exacerbations and absence of characteristic mass lesion in pancreatic imaging went against the diagnosis of pancreatic tuberculosis. To clinh the diagnosis and the etiology of chronic pancreatitis, an image guided needle biopsy of the pancreas was performed. This had revealed presence of granuloma with caseation. The resolution of clinical and biochemical features of pancreatitis did support the diagnosis of tubercular pancreatitis with splenic abscess. Appropriate treatment with antibiotics and anti-tubercular medications, had resulted in complete recovery and no further recurrence in follow up.

CONCLUSION

Pancreas and spleen are two of the least common sites of extra pulmonary tuberculosis. The usual sites of abscess formation associated with pancreatitis are pancreas itself and liver. Multiple splenic abscess assiciated with recurrent acute exacerbation of chronic tubercular pancreatitis in an otherwise immune competent child is very unusual.

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