Leptospirosis As A Rare Cause Of Acute Pancreatitis
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Abstract
Acute pancreatitis is pancreatic inflammation that may be followed by clinical and biological restitution of the gland if the primary cause is eliminated. Multiple causes of pancreatitis are known, most commonly alcohol and cholelithiasis. Infectious causes include mumps, Coxsackie B, CMV and Cryptococcus. We present a case of pancreatitis caused by leptospirosis, which is a rare cause.

CASE
A 45-year-old Muslim male, driver by occupation, presented with severe pain in the abdomen for 2 days. The pain started in the epigastric region and was severe with radiation to the back and bilious vomiting. He had mild fever.

There was no history of abdominal trauma or such an episode in the past. The patient was not alcoholic, nor did he have any history of hyperlipidemia. He was non-diabetic and did not give a history of passing bulky foul-smelling stools (steatorrhea).

On examination, he had tachycardia, tachypnea and mild hypotension. His abdomen showed marked tenderness and guarding in the epigastric region. The patient was in paralytic ileus on admission. There was no evidence of pleural effusion. He did not have any icterus on admission, but he developed icterus after 4-5 days of admission. He was passing concentrated urine with hematuria. Urine output started decreasing after a 2-3 days of admission.

On admission, the patient had leucocytosis (Total White Blood Count: 16,500). The serum bilirubin level was 1.6mg%; but after 5 days the level rose to 4.5mg%. Blood urea levels increased to 76mg% and the serum creatinine levels rose from 1.7mg% to 3.5mg% after 5 days. There were raised levels of serum amylase and lipase. Serum calcium and lipid profile was normal. Urine showed the presence of 20-25 RBCs per high-power field.

Abdominal x-ray showed typical ground-glass appearance. Ultrasonography revealed features suggestive of pancreatitis. The entire biliary tree was within normal limits. CT scan (plain + contrast) was suggestive of fluid collection in the abdomen and edematous pancreas with peri-pancreatic fat stranding.

Investigations for Dengue and Leptospirosis were carried out on the 8th day of admission, which showed positive IgM titres for leptospirosis.

The patient was started on conservative management for pancreatitis with antibiotics (Cefotaxim, Amikacin, Metronidazole), Octreotide and Pantoprazole. Continuous nasogastric tube aspiration was started and urine output was monitored by Foley’s catheter. On detection of leptospirosis, cefotaxim was substituted with amoxycillin.

The patient had paralytic ileus which resolved after 10 days. Pancreatitis completely resolved and the patient was discharged from the hospital.

Figure 1
Figure 1: C.T. scan of the abdomen showing edema and necrosis of the pancreas.

DISCUSSION
Acute pancreatitis is an inflammatory disease of the pancreas that is associated with little or no fibrosis of the gland. It can be initiated by several factors including gallstones, alcohol, trauma and infections and in some cases it is hereditary.
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The generally prevalent belief today is that pancreatitis begins with activation of digestive zymogens inside the acinar cells which cause acinar cell injury. Recent studies suggest that the ultimate severity of resulting pancreatitis may be determined by events that occur subsequent to acinar cell injury (1).

Clinical features include severe pain radiating to the back, nausea, vomiting, tachycardia, tachypnoea, hypotension and hypothermia. There may be guarding, decreased bowel sounds, Cullen’s sign and Grey-Turner’s sign due to retroperitoneal hemorrhage.

Several markers of severity including early prognostic signs (2,3), serum markers and CT scans are commonly used to assess the severity of the disease (4). CT scanning is used to distinguish milder (non-necrotic) forms of the disease from more severe necrotizing or infected pancreatitis (5).

Treatment is supportive and has the important aim of “resting” the pancreas through restriction of oral food and fluids. The long-held opinion that antibiotic prophylaxis in necrotizing pancreatitis is of little use, has been altered by recent studies showing a beneficial effect with metronidazole, imipenam and third generation cephalosporin.

Leptospirosis is considered to be the most widespread of zoonoses, being regularly present in all continents except Antarctica. The first recognized leptospiral disease of human beings was spirochetal jaundice described by Weil (1886). The causative agent of Weil’s disease was isolated in 1915 by Inada and named Leptospira icterohaemorrhagiae.

Incubation period is about 10 days (range 2-26 days). Pathogenic leptospires survive for long periods in convoluted tubules of the kidneys in natural hosts, multiply and are shed in urine.

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Patients. Purpuric hemorrhages may occur on skin or mucosa. Albuminuria is a constant feature.

This patient presented with icteric leptospirosis or Weil's syndrome which is characterized by complications like decreased renal function, pulmonary complications, jaundice and hemorrhagic manifestations. Only 5-10% of patients have this severe form. Pancreatitis is one of the rare complications of leptospirosis.

The most consistent pathologic finding in leptospirosis is vasculitis of capillaries manifested by endothelial edema, necrosis and lymphocytic infiltration. The exact mechanism of acute pancreatitis in leptospirosis is not well understood.

Small vessel vasculitis and ischemic injury leading to activation of proteolytic enzymes and auto-digestion is the possible mechanism.

Hyper-amylasemia in leptospirosis even without pancreatitis is observed because of renal failure and inactivation of the reticulo-endothelial system of the liver which impedes the clearance of amylase. Lipase is known to have high specificity (97%-99%) for the diagnosis of pancreatitis and is not significantly affected by change of glomerular filtration rate.

Diagnosis is by examination of blood or urine for leptospira or serological tests for antibodies. Leptospires are sensitive to penicillins and tetracyclins; but for treatment to be effective, it should be started early in the course of disease.

References
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