The diagnosis can be challenging since it may not be possible to distinguish AFNAC from ulcerative colitis or Crohns' disease since they can have a similar presentation.[1-3] Clinical symptoms and signs, laboratory investigations, and radiology remain inconclusive in distinguishing between them. The presence of trophozoites, especially if showing erythrophagocytosis (ingested red blood cells in the cytoplasm) in fresh stool samples of symptomatic patients, was typically considered diagnostic; recently, erythrophagocytosis has been reported in non-pathogenic trophozoites too. Serological tests for amoebiasis are of doubtful value in endemic areas as they cannot distinguish between prior and present infection; antigen detection in stool and molecular techniques using polymerase chain reaction are highly sensitive, but not easily accessible. In the absence of these tests, the only means of definitely establishing the diagnosis may be a demonstration of trophozoites of E. histolytica on histopathology.[4]

Surgery should be expedited in AFNAC.[1-3] Bowel involvement dictates the extent of the colonic resection; primary anastomosis is usually precluded since the colon is very friable, and it is safer to resect and exteriorize the proximal and distal bowel to be restored at a later date.[1-3,5] Amoebicidal therapy (metronidazole followed by luminal agents such as diloxanide furoate) should be given in suspected cases, and continued if the diagnosis is confirmed.

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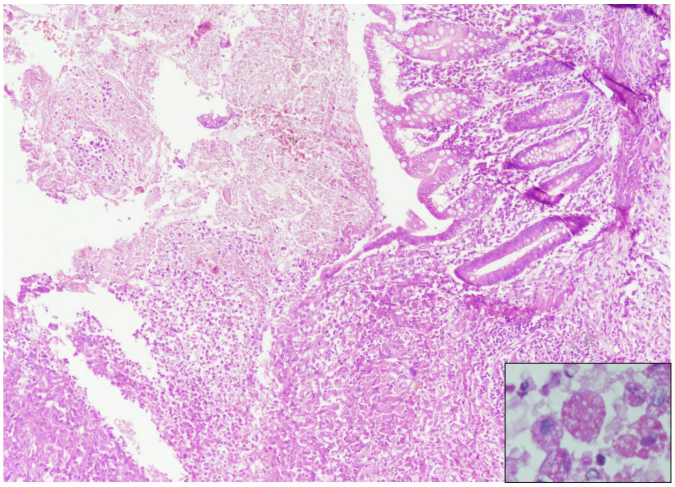

Figure 2: Photomicrograph showing intestinal epithelium lined tissue with necrotic slough having trophozoites of Entamoeba (HE-x-100) Inset shows Entamoeba trophozoites with ingested RBCs (PAS-x-400).

Acute Severe Pancreatitis and Bilateral Renal Cortical Necrosis

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It is well known that acute pancreatitis can be complicated by renal failure due to various mechanisms. In most cases, acute renal failure is due to acute tubular necrosis, and kidneys recover entirely with supportive care[1]. However, acute renal cortical necrosis complicating acute pancreatitis is a rare event, and recovery is unlikely.

Only a few cases are reported in the literature. We present a case of acute necrotizing pancreatitis complicated by the rare complication of acute renal cortical necrosis, showing characteristic imaging features.

Case Report

A 30-year-old male with no previous illness presented to trauma center with a history of road traffic injury. The airway was patent, breathing and circulation were normal. Glasgow Coma Score (GCS) was 15. On physical examination, there was tenderness in the epigastrium. Focused assessment with sonography in trauma (FAST) was positive for free fluid in Morrison's pouch and pelvis. CECT torso was performed, which showed diffuse enlargement of the pancreas with heterogeneous enhancement and peripancreatic inflammation (**Figure 1**). Also, Bilateral kidneys showed diffuse cortical hypodense areas and a normal enhancing renal medulla suggestive of renal cortical necrosis (**Figure 2**). Subsequently, laboratory investigations revealed raised serum creatinine (5mg/dl) and amylase (700 U/L) levels. Over the next three days, the patient developed anuria. He received supportive management, antibiotics, and dialysis support. He is being evaluated for renal transplantation since there was no improvement in renal function during the hospital course.

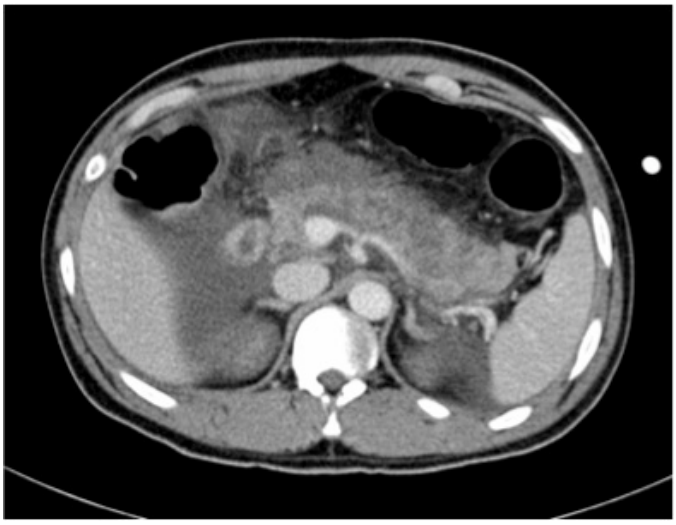

Figure 1. Axial CECT scan image showing diffusely enlarged pancreas with heterogeneous enhancement and peripancreatic inflammation.

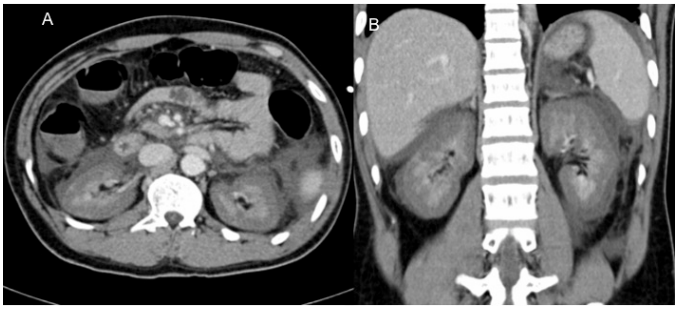

Figure 2. Axial (A) and Coronal 3D reformatted MPR (B) CT images showing diffuse cortical hypodense areas involving both kidneys and a normal enhancing renal medulla suggestive of renal cortical necrosis.

Discussion

A serious complication of severe acute pancreatitis is acute renal failure, an important indicator of morbidity and mortality in critically ill patients

Acute renal failure may result from various mechanisms such as hypoxemia, impaired renal perfusion, the release of pancreatic enzymes from the inflamed pancreas causing damage to renal microcirculation, or abdominal compartment syndrome. Other possible theories, such as the release of endotoxins and reactive oxygen species, have been implicated in the pathophysiology of renal injury[2].

Bilateral cortical necrosis is a rare complication of severe acute pancreatitis; only a few cases of bilateral cortical necrosis following acute pancreatitis were reported in the literature. Cortical necrosis commonly results from reduced perfusion within microcirculation of the renal cortex following septic shock or hypovolemia. However, the cause remains mysterious in normotensive patients, is ascribed due to the release of vasoactive or cytotoxic substances during pancreatitis[3].

Imaging features are characteristic; contrast-enhanced CT shows a non-enhancing renal cortex and a normal enhancing renal medulla described as reverse rim sign[4]. Sometime, a very thin rim of contrast enhancement i.e., cortical rim sign, may be seen and should not be mistaken for adequate perfusion[5]. These imaging features

correlate with the death of all cells in the cortex and congestion of the medulla on histology[1,6]. The capsule and a thin rim of the peripheral cortex (1-2 mm) are spared because of its separate capsular blood supply [7].

In the majority of the cases, acute renal failure is due to acute tubular necrosis, and kidneys recover completely with supportive care. However, acute renal cortical necrosis complicating acute pancreatitis is a rare event, and recovery is unlikely.

The prognosis is very poor, nearly most end up in renal replacement therapy after a variable period. The majority of patients with the diffuse type of cortical necrosis will not recover renal function, while patients with patchy cortical necrosis may show some improvement in renal function but gradually develop the end-stage renal disease and only curative treatment is renal transplantation[8].

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Hepatic Epithelioid Angiomyolipoma of the Liver: A Diagnostic Dilemma

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Epithelioid angiomyolipoma (AML) is included in group of the perivascular epithelioid cell (PEC) tumors, known as PEComas. These tumors are strongly associated with tuberous sclerosis. Most of these cases have been reported in kidney, and liver involvement is rare, with less than 50 cases described in the literature. Most cases of hepatic angiomyolipoma are misdiagnosed as adenomas or other diseases on radiology as some lack definite adipose tissue components. The final diagnosis could be reached through histology and immunohistochemistry testing.

Case Report

A 30-year-old female presented with vague abdominal pain and on evaluation was found to have a liver space-occupying lesion on ultrasound. Lab investigations showed anemia with hemoglobin of 10.5 g/dl (12-15.0 g/dl). Liver and kidney function tests were within normal limits. Contrast-enhanced CT abdomen revealed a large homogeneously hypodense mass without calcification or hemorrhage, measuring 8×7×5.5 cm in the left lobe of liver involving caudate lobe. The mass showed heterogenous centripetal arterial phase enhancement and remained slightly hyperdense, compared to normal parenchyma, on hepatic and delayed phases. A possibility of hepatic