Bilateral avascular necrosis of femoral heads in a case of alcohol-related acute on chronic pancreatitis

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A 40-year-old man with no prior comorbidities presented to our emergency department with a 3-week history of moderate epigastric pain radiating to the back, exacerbated by food intake and partially relieved by oral analgesics, accompanied by recurrent non-bilious vomiting for the past week. He had no history of jaundice, fever, diarrhea, constipation, abdominal distension, or dysuria. However, he had a history of chronic alcohol consumption for the past 5 years, with his most recent intake 1 day before the onset of abdominal pain. He denied any history of smoking or recreational drug use. On examination, he was tachycardic with a heart rate of 112 beats/min, blood pressure of 130/90 mmHg, and oxygen saturation of 98% on room air. Abdominal examination revealed marked tenderness and guarding. Routine investigations revealed leukocytosis (white blood cell count: 15,500 cells/mm^3), an elevated C-reactive protein level of 78 mg/dL, and a raised serum lipase of 980 IU/mL. Renal and liver function tests were largely within normal limits. Abdominal ultrasound showed a grade I fatty liver, a distended gallbladder without calculi, and a heterogeneous pancreas with peripancreatic fluid collections. His serum calcium was 9.8 mg/dL, and his triglycerides were 350 mg/dL. Diagnosis of ethanol-related acute pancreatitis was made, and he was managed conservatively with nil per oral, intravenous fluid therapy, analgesics, and multivitamin supplementation. In view of persistent pain not responding to analgesics and systemic inflammatory response syndrome, a contrast-enhanced computed tomography (CECT) of the abdomen was performed. It revealed focal calcifications in the pancreatic head, a dilated main pancreatic duct with suspected ductal disruption at the pancreatic body, and associated peripancreatic necrosis. A pseudocyst measuring 8 × 6 × 5 cm was identified adjacent to the left lobe of the liver and anterior to the lesser curvature of the stomach. In addition, bilateral avascular necrosis (AVN) of the femoral heads was noted (Fig. 1). Based on CECT findings, the diagnosis was revised to acute-on-chronic pancreatitis with disrupted pancreatic duct syndrome, associated pseudocyst formation, and bilateral AVN of the hips (Fig. 2). The patient was started on intravenous antibiotics (piperacillin–tazobactam) due to the possibility of an infected pseudocyst, and EUS-guided cystogastrostomy was performed using two 7 French plastic stents. During the same session, endoscopic retrograde cholangiopancreatography was also carried out, which demonstrated a disruption of the pancreatic duct in the body region, with absent opacification of the main pancreatic duct distal to the site of disruption. Attempted guidewire negotiation into the distal body and tail of the pancreas was unsuccessful. A pancreatic sphincterotomy was then performed, followed by stenting of the pancreatic duct involving the head and proximal body. The patient's pain and vomiting improved significantly within 48 h following the cystogastrostomy, and he was able to tolerate an oral diet. An orthopedic consultation was sought for bilateral AVN of the hip joints. Since the patient reported no hip pain or difficulty walking, conservative management was advised, including cessation of alcohol, avoidance of weight-bearing activities, calcium and Vitamin D supplementation, bisphosphonates, and administration of analgesics if required.

AVN of the femoral head is a form of aseptic osteonecrosis caused by disruption of the blood supply to the proximal femur, leading to osteocyte death [1]. Several pathogenic mechanisms may result in ischemia and AVN, including vascular interruption, intravascular occlusion, and intraosseous extravascular compression [2]. Common causes of AVN of the femur include trauma (femur neck fracture, dislocation, etc.), prothrombotic conditions, chronic steroid use, smoking, chronic ethanol consumption, sickle cell hemoglobinopathy, vasculitis, acute pancreatitis, etc. [3]. In our patient, both alcohol use and pancreatitis are potential etiological factors contributing to femoral head AVN. The incidence of aseptic necrosis among regular alcohol consumers has been reported to be as high as 5.3%. Alcohol-induced osteonecrosis is not well understood but most likely stems from bone marrow fat cell hypertrophy

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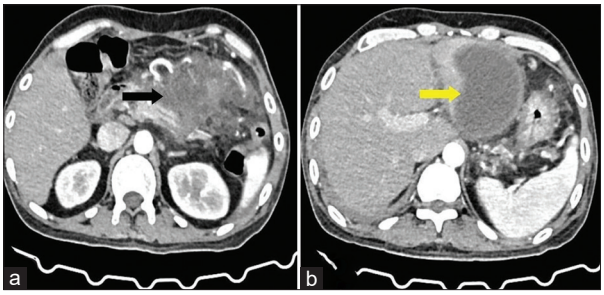

Figure 1: (a) Contrast-enhanced computed tomography abdomen showing pancreatic and peripancreatic necrosis (black arrow), (b) showing pseudocyst in relation to left lobe of liver (yellow arrow)

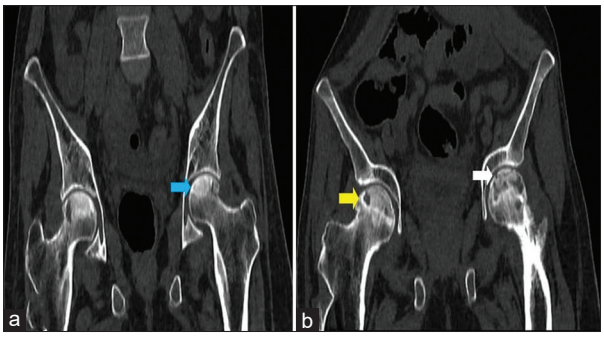

Figure 2: (a) Computed tomography abdomen (bone window) showing sclerosis of bilateral femur head (blue arrow) (b) showing cortical break (white arrow) and subchondral cysts (yellow arrow)

and proliferation, increased intraosseous pressure, and subsequent lack of perfusion [4]. Pancreatitis is an uncommon cause of long bone osteonecrosis, with only very few cases documented in the literature. It can lead to fat necrosis within the bone marrow, and the resulting changes may compress or distort adjacent intramedullary arteries, eventually leading to aseptic necrosis or infarction of the femoral head [5]. Management of femur head AVN depends on the symptoms and stage of disease. The index patient is asymptomatic and has been advised for conservative management. This case highlights the importance of considering rare systemic complications such as AVN in patients with acute-on-chronic pancreatitis, especially in the presence of chronic alcohol use. Early identification and multidisciplinary management are crucial for optimal outcomes.

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