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Acute Pancreatitis Worsening Acute Hepatitis E

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ABSTRACT

The Hepatotropic viruses primarily invade the liver tissue but clinical and pathological involvement of other organ systems also occur. Pancreas is one of the organs that involved during the course of acute viral hepatitis E. Acute pancreatitis complicating fulminant hepatitis has been well recognized but acute pancreatitis occurring in nonfulminant hepatitis is very rare. Here we are presenting a case of 18 year old young male who presented with acute viral hepatitis E later on which was complicated by acute pancreatitis. The diagnosis of hepatitis E was made by detection of anti-hepatitis E IgM antibody. Thus the patients with acute viral hepatitis with severe abdominal pain should be suspected with acute pancreatitis and all the investigations like serum lipase, serum amylase, USG abdomen and CECT abdomen should be done to rule out this complication.

INTRODUCTION

Acute pancreatitis is a sudden inflammation that usually lasts for a short time. It may range from mild discomfort to a severe, life-threatening illness. Most people with acute pancreatitis recover completely after getting the right treatment. In severe cases, acute pancreatitis can result in bleeding into the gland, serious tissue damage, infection, and cyst formation. Severe pancreatitis can also harm other vital organs such as the heart, lungs, and kidneys.

The association between infectious hepatitis and acute pancreatitis was first reported in 1944 by lisney. Most cases of pancreatitis are associated with hepatitis A and hepatitis B infection. Hepatitis E has recently been described as a causative agent of acute pancreatitis mostly in endemic areas, but overt pancreatitis has only been reported generally in association with cases of fulminant hepatic failure. However recently it has come into focus that pancreatitis may also occur in hepatitis in mild to moderate severity.

CASE REPORT

18 year old male came to hospital with high grade fever, recurrent vomiting, severe pain abdomen, breathing difficulty and decrease oral intake for 7 days. On examination patient's vitals were stable and generalized tenderness was present on per abdomen examination. There was no H/O alcohol use, gall stone disease, any drug intake or trauma. He was further investigated and his laboratory reports revealed serum bilirubin total 4.36mg/dl(N=.30-1.20), serum bilirubin direct 3.33mg/dl (N=0.00-0.20), SGOT=2358IU/L (N=0.0-40.0) ,SGPT=3669.1IU/L(N=0.0-41), serum alkaline phosphatase=166.0IU/L(N=40.0-130.0),GGTP=55.0IU/L(8.0-61.0),S.Amylase=48.0U/L(N=28.0-100.0),

pase=49.4U/l(N=13.0-60.0), CXR and ECG did not reveal any abnormality. Ultrasound abdomen revealed normal liver size, normal pancreas, spleen borderline enlarged(125mm), gall bladder shows collapsed lumen with diffuse wall edema and few periportal lymphnodes seen suggestive of infective hepatitis and HEV IGM(ELISA) was positive with titre of 11.11. Patient was treated conservatively and his LFT were serially monitored. On 7th day of hospitalization patient complaining of severe pain abdomen, non passage of flatus and stools. On examination his BP was 90/60, generalized tenderness was present on per abdomen examination.

He was further investigated and found to have serum bilirubin total 12.96mg/dl(N=.30-1.20), serum bilirubin direct 11.60mg/dl (N=0.00-0.20), SGOT=3474 IU/L (N=0.0-40.0) , SGPT=3521.1IU/L (N=0.0-41), serum alkaline phosphatase=194.0IU/L(N=40.0-130.0), S. Amylase=247.2 IU/L, S.lipase=397.4U/l(N=13.0-60.0),S. Lipids=WNL, Ionic calcium was WNL, TLC 16200, blood culture sterile and CECT Whole abdomen revealed bulky pancreas, diffuse gall bladder wall edema with pericholecystic fluid, borderline splenomegaly, moderate ascites, b/l minimal pleural effusion. Patient with moderate hepatitis was complicated by acute pancreatitis, he was serially monitored and treated conservatively and discharged in astable condition.

DISCUSSION

Viral causes of pancreatitis are well established. The viruses most frequently associated with acute pancreatitis are mumps, Coxsackie, rubella, Epstein-Barr virus, cytomegalovirus, and varicella-zoster virus. In adults mumps virus is one of the most commonly associated etiological agents and pancreatitis can occur even in the absence of parotitis But association of pancreatitis with viral hepatitis is not a common entity. A substantial number of these cases were found in fulminant hepatic failure but mild to moderately severe Hepatitis E presenting with acute pancreatitis is very rare. Cholelithiasis and gastric ulcers are the other causes of severe abdominal pain. Majority of the patients recover with conservative management.

The exact incidence of pancreatitis in viral hepatitis is not known. Necropsy studies have identified pancreatic involvement in 12-40% of fatal hepatitis cases and most of the statistics comes from autopsy examination. Joshi et al in 1957 found 3.7% of their cases having viral hepatitis in 108 autopsied cases of acute pancreatitis. Joske et al noted 8 cases of acute viral hepatitis out of 90 patients of acute pancreatitis.

The mechanism of pancreatitis in patients with non fulminant acute viral hepatitis. It is proposed to be 1) Either an immune response or direct cytotoxicity against the infected acinar cells. 2) Due to oedemaof ampulla of vater. 3) DIC associated with acute liver failure leading to pancreatitis. There is no direct evidence for the routes by which hepatitis viruses reach the pancreas however the proposed routes are most likely blood or bile.

In conclusion acute pancreatitis is not uncommon in fulminant hepatitis but in patient with mild to moderate acute viral hepatitis presenting with pain abdomen, acute pancreatitis should be kept as a possibility and has a good prognosis.

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