Severe acute pancreatitis complicating acute hepatitis A in a child

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Abstract

The case of a young girl who developed acute pancreatitis in the course of acute hepatitis A is reported along with relevant literature review. The rarity of this complication in children [compared to young adults] even in endemic areas where water borne viral hepatitis affects large number of children is highlighted.

INTRODUCTION

The incidence of acute pancreatitis complicating non fulminant acute viral hepatitis1 and that of acute viral hepatitis as etiology of acute pancreatitis2,3 are both low. It is mostly reported in adolescents and young males but rarely in children.4-7 According to incidence, the etiologies of acute pancreatitis are different in children than adults. Infection [including viral], trauma, systemic and metabolic diseases as well as congenital and hereditary pancreatic disorders are common causes in children. A recent pediatric series describes acute pancreatitis to be associated with hepatitis A in only 8.3% cases.3 We report the case of an 8 year old girl with acute hepatitis A who developed acute pancreatitis with fluid collection early in the course but had uneventful recovery with the relevant literature review.

CASE REPORT

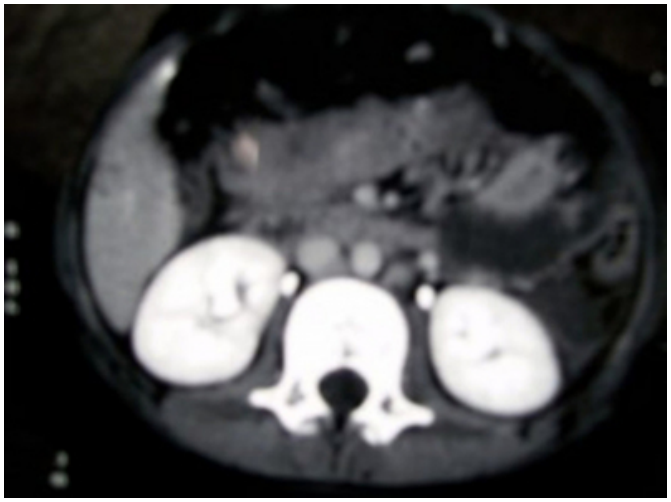
A previously healthy 8 year old girl had a bout of fever with prodrome followed by mild epigastric discomfort. She became icteric after another 2 days. The abdominal discomfort gradually increased in severity and was associated with vomiting which needed hospitalisation of the child on the sixth day. The pain was boring in nature, radiated to the back, persisting throughout the day with the child lying curled up on her side in bed and feeling better when sitting up and stooping forward.

On examination she was icteric and distressed by the pain. Epigastrium was tender, bowel sounds were sluggish but no mass was palpable. Vitals were stable. The family members denied any history of recent abdominal trauma, drug intake, surgery, vaccination, blood transfusion, diarrhoea or worm expulsion, rash, parotid swelling and any family history of hyperlipidemia, gallstone or pancreatic diseases.

Blood investigations were as follows : Hemoglobin 12.8 gm%, total leukocyte count[TLC] 14,900/cu.mm, neutrophil80%,lymphocyte18%, eosinophil2%, serum bilirubin 15.4 [direct 13.1, indirect 2.3] mg/dl, alanine aminotransferase2910 IU/dl, aspartate aminotransferase2500 IU/dl, alkaline phosphatase 318 IU/dl, amylase 771 IU/dl [normal less than 170], lipase 1752 IU/dl [normal less than 30], urea 10 mg/dl, creatinine 0.3 mg/dl, triglyceride 120 mg/dl, calcium 9.8 mg/dl, phosphorus 4.1 mg/dl, C Reactive protein 22 U/dl [normal < 6], malarial parasites and antigens were negative. Serologic studies revealed negative results for Widal test, Dengue serology, HBsAg, AntiHCV, IgMAntiHEV and positive IgMAnti HAV. Radiologic investigations were as follows : USG abdomen – pancreatic edema, thick gallbladder wall but no gallstone or sludge, Contrast enhanced CT Scan abdomen [Fig 1] – edematous pancreatitis with collection in tail of pancreas and left paracolic gutter [Balthazar grade D], bilateral mild pleural effusion, mild hepatomegaly and normal gallbladder. A MRCP study did not reveal any pancreas divisum, choledochal cyst or chronic pancreatitis.

Figure 1

Figure 1. Contrast enhanced CT Scan of abdomen showing collection in tail of pancreas and left paracolic gutter



With conservative treatment the pain and vomiting decreased over next 2 days and she was able to eat food from 3rd day. Repeat blood investigations on day 4 of hospitalisation were : haemoglobin 12.6 gm/dl, TLC 10,600/cu.mm, neutrophil65%, lymphocyte30%, eosinophil3%, monocyte2%, total serum bilirubin 13 gm/dl, aspartate aminotransferase 49 IU/dl, alanine aminotransferase 79 IU/dl, amylase 360 IU/dl, lipase 600 IU/dl. Further course was uneventful and she was discharged after 8 days.

DISCUSSION

Association of acute pancreatitis and hepatitis A is sufficiently rare to be highlighted only as case reports and recently as small case series.1-15 Of all the documented cases of such association, most have been in adolescents and young adults where the pancreatitis was mild, developed early in course of hepatitis and followed uneventful recovery. Reports are even rarer in children below the age of 10 years, only 5 cases [including the present one]4-7 have been cited.

These few childhood cases have been reported from countries endemic for viral hepatitis which is surprising since hepatitis A is a disease primarily of children in these countries. In this context it is worthwhile to note that for a similar endemic hepatitis E virus causing acute hepatitis, although associated occurrence of acute pancreatitis is being increasingly reported in adolescent and young adults,2 but only very recently a single case in a child less than 10 years age have been reported.16

The course of pancreatitis in all these children were like their older counterparts except one7 where the pancreatitis was severe mimicking acute abdomen . In the present case also the pancreatitis developed early in the course of hepatitis and although clinically mild but radiologically it was severe [Balthazar grade D]. Even then the clinical recovery was very quick and uneventful. Similar associated pleural effusion have been previously reported, possibly the result of fluid sequestration associated with acute pancreatitis.5,7

Do these facts mean that children are less prone to pancreatic affliction by the hepatitis viruses A and E? It is seen that compared to those above 10 years of age, the overall incidence of acute pancreatitis (due to all etiologies) below the age of 10 is much less and even in this group of children, there is a decreasing trend in incidence and severity in younger age.3,17,18 This fact may be reflected in case of the viruses also and might possibly be related to [a] Immaturity of the immune system in young children which protects the pancreas from cytokine mediated injury occurring in acute pancreatitis, **low pancreatotropism of the viruses in children, [c] occurrence in only those children who have a genetic predisposition to pancreatitis.**

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