Acute pancreatitis in hepatitis A infection

A. Lopez Morante, C. Rodriguez de Lope, G. San Miguel and F. Pons Romero

Gastroenterology Unit ‘Marques de Valdecilla’ Medical Center, Faculty of Medicine, Santander, Spain.

Summary: Hepatitis viruses are an uncommon cause of acute pancreatitis. We present the case of a boy with acute pancreatitis complicating viral hepatitis with satisfactory recovery. The finding of IgM-anti HAV antibodies implicates hepatitis A virus as the cause.

Introduction

Several viral infections have been implicated as aetiological factors of acute pancreatitis. The viruses most frequently thought to be responsible are mumps virus (Feldstein et al., 1974; Naficy et al., 1973), Coxsackie B virus (Capner et al., 1975; Ursing, 1973; Tsui et al., 1972), Epstein-Barr virus (Wislocki, 1968), and measles virus (Bunel & Monif, 1972). An association between viral hepatitis and acute pancreatitis has also been observed, although most of the patients concerned had fulminating hepatitis and the causal agent has been identified in only a very few patients (Archod, 1968; Parbhoo et al., 1973; Wands et al., 1973).

We present here a patient with acute pancreatitis associated with acute hepatitis caused by hepatitis A virus (HAV), who made a satisfactory recovery.

Case report

A 12 year old boy with no previous medical history, became ill 8 days before admission with tiredness, general malaise and dark urine. He was admitted to the Emergency Department because of severe epigastric pain of abrupt onset accompanied by nausea and vomiting. On admission, he was afebrile and jaundiced. His abdomen was distended with general tenderness on palpation and no bowel sounds were heard. The liver was enlarged 1 cm below the right margin.

Laboratory investigations included: white blood count 12.8 x 10^9/l (81% polymorphs), haemoglobin 16.3 g/dl, erythrocyte sedimentation rate 25 mm/h, serum amylase: 1200 U/dl (Somogyi) (normal <180); bilirubin: 7.3 mg/dl (normal 0.2–1.2) (conjugated 3.3 mg/dl); aspartate aminotransferase (AST) 2100 U/l (normal 20–40); alanine aminotransferase (ALT) 2208 U/l (normal 20–40); alkaline phosphatase: 350 U/l (normal 38–85). The plasma electrolytes, creatinine, calcium, proteins and lipids were normal apart from an IgM of 515 mg/dl (normal 35–56). Hepatitis B surface antigen (HBsAg) was negative; IgM antibodies to HAV were positive (RIA Abbot).

The serum isoamylases were studied by electrophoresis and there was a single peak caused by amylase P1. The 24-hour urinary amylase content was 27,600 U and the amylase/creatinine clearance ratio was 7.1% (normal <5%).

Plain films of abdomen showed distended small bowel loops with no fluid levels. Ultrasonographic examination of the abdomen showed a layer of ascitic fluid at the base of the lesser sac. The pancreatic region could not be visualized because of interference by gas. Gall-bladder and bile duct were normal.

Treatment was with fluid and electrolyte replacement. Pain ceased after one day and intestinal movement returned two days after. On discharge, AST was 170 U/l, ALT 483 U/l, bilirubin and amylase returned to normal values. Ten days after discharge the AST and ALT values were normal, and a new ultrasonographic examination showed a normal pancreatic region and no ascitic fluid.

Discussion

The mechanism of pancreatic damage by viruses is not known. The cytopathic effect may be direct or it may be mediated through the patient’s immune response. It has also been suggested that viral infections cause oedema of Vater’s ampulla and pancreatic ducts leading to pancreatitis as result of obstruction to the flow of pancreatic fluid (Tsui et al., 1972).

When acute pancreatitis is associated with fulminating hepatitis, the virus may cause tissue damage directly, but there are several other factors which can play an important role in the development of pan-
creatitis and these include acute liver failure, hypotension and drug-induced disease. These factors were not operative in our patient and so the pancreatic damage could only have been caused by HAV.

Shimoda et al. (1980) demonstrated the presence of hepatitis B virus core in the cytoplasm of pancreatic acinar cells in patients infected with this virus. Pancreatic lesions may be caused by immunological mechanisms in a similar way to that in hepatocellular necrosis.

References