CASE REPORT

HIV and Pancreatitis

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Abstract

Pancreatic disorders are rare with HIV infection. We report here a case of pancreatitis as initial manifestation of AIDS.

Key Words

Pancreatitis, AIDS

Introduction

Gastrointestinal manifestations are common during the course of Human Immunodeficiency virus (HIV) infection (1). Thirty-five percent of HIV positive patients have gastrointestinal complaints on presentation (2). Pancreatitis and other pancreatic disorders are rare in HIV infected individuals (3). We report a patient who had acute pancreatitis as initial manifestation of acquired immunodeficiency syndrome (AIDS). This is the first report of its kind.

Case Report

A 40 year old female was admitted to the hospital with acute abdominal pain and vomiting. She was nondiabetic and had no addiction. Her general physical examination was normal. Systemic examination revealed normal chest and cardiovascular system. She had epigastric tenderness with no organomegaly or ascites. A clinical diagnosis of acute pancreatitis was made. Investigations like CBC, urine analysis, kidney and liver function tests, lipid profile and repeated blood sugar were normal. Chest radiograph was normal. Abdominal ultrasonography revealed an oedematous pancreas. There was no mass. Liver, gallbladder, spleen were normal with no ascites. Liver function tests were normal. Her serum amylase was 2600 IU/L. She was managed conservatively with Ryle tube suction, intravenous fluids and antibiotics. The patient improved and was discharged from the hospital on tenth day of admission. One week later the patient was readmitted with odynophagia, dysphagia and yellowish discoloration of eyes. On examination she had diffuse oropharyngeal candidiasis, deep jaundice, stage II hepatic encephalopathy and hepatomegaly. Cardiovascular and chest examination was normal. Her CBC, renal function tests and x-ray chest were normal. Urine analysis showed increased urobilinogen. Liver function tests revealed serum billrubin of 22mg% (D-16, ID-6), AST 600 IU/L, ALT-300 IU/L ALP 15 KA units/L, S. Albumin 3.5g% and globulins 2g%. Serum amylase was 132 IU/L. Abdominal ultrasonography showed hepatomegaly, normal pancreas and spleen and no ascites. HBsAg was negative. Rapid HIV test was

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positive and ELISA for HIV was also positive. HIV positivity was confirmed by Western Blot. (CD 4 counts were not done). Serology for toxoplasmosis, rubella, CMV and Herpes virus was negative. A diagnosis of AIDS with oropharyngo- esophageal candidiasis with viral hepatitis with stage II encephalopathy was made. The patient developed fulminant hepatitis with hepatic coma and died within 48 hours of admission, despite intensive therapy for hepatic coma and oropharyngeal candidiasis.

Discussion

Pancreatitis is rare in HIV infected individuals. In a series involving 7806 patients, the incidence of abdominal pain and pancreatitis was 5% after use of Dideoxynosine (4). In another large autopsy series. Kaposi's sarcoma and lymphoma were found to involve pancreas in 8% of AIDS patients but they were usually asymptomatic (5,6). Focal fat necrosis, acinar dilatations, fibrosis and cancer infiltration were encountered in 50 - 70% of cases in a reproted series (7). The incidence of hyperamylasemia is significantly greater than the incidence of pancreatitis in HIV infected individuals. It was noted in 46% of patients by Zarro and co-workers (8). The differential diagnosis of pancreatitis in HIV infected patients includes medications or malignancy (Kaposi's Sarcoma or Lymphoma) or infections (CMV, MAC, cryptosporidiasis, cryptococcosis, toxoplasmosis). Our patient was neither on any medication nor had cholelithiasis. Hypertriglyceridemia which is common in patients of AIDS and is associated with pancreatitis was not present in this patient (9). The presentation of pancreatitis in HIV positive patients is no different from that in general population except with following significant differences. A high frequency of medication associated pancreatitis; a low frequency of gall stones, a high frequency of HIV related causes (most commonly by HIV related drugs) (10). The suspicion of AIDS was

Viral hepatitis has been recognized as an important cause of morbidity in the setting of HIV infection since the beginning of AIDS era (11). Hepatitis virus B (HBV) serology is positive in 90% of AIDS patients and 10-20% are HBs Ag positive (chronic carrier state). Since our patient died within 48 hours of hospital admission serology for HBC and E could not be done and the cause of hepatitis could not be established, though the patient was HBs Ag negative.

Oral candidiasis was one of the first reported opportunistic infections in AIDS (12). In HIV infected individuals the development of oral candidiasis is associated with an increased risk of progression to AIDS or death, as also happened in our patient (13). The prognostic significance of oropharyngeal candidiasis has been confirmed by many subsequent studies (13-15).

In the developing world, nearly all HIV infection in women have been acquired heterosexually, in contrast to the situation in USA; This is speculated to be caused by their husband's greater ability to buy sex. African women's greatest risk of HIV infection is conferred by her husband (16).

The course of HIV infection among women does not differ substantially from that in men. Although a few small studies have found different incidence rates for specific opportunistic infections and malignancies in women compared with men, the overall differences seem to be minimal with the exception of esophageal candidiasis, Kaposi's sarcoma and genitourinary cancer, and infections occuring 30-50% more common among women (17). Esophageal candidiasis has been found to be most common AIDS indicator illness in addition to Kaposi's sarcoma (occuring eight times more frequently among men and opportunistic herpes simplex infection32% more common in women (18). The present case is of interest because of its rarity and progression to fatal full blown AIDS within days of development of candidlasis and hepatitis.

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