Sir,

Pancreatic disease has been found frequently in adults infected with HIV. It has been reported in children from two centers in USA with an incidence ranging from 17 to 23 per cent. It has been associated with pentamidine isothionate exposure, CD4 cell count <100 cells/μl, infective agents such as cytomegalovirus, cryptosporidium, Pneumocystis carinii pneumonia and Mycobacterium avium intracellulare. It has also been reported in patients taking didanosine (ddI) as part of their antiretroviral treatment. However, HIV itself is known to cause hyperamylasaemia and hyperlipasaemia in HIV infected patients. We present a case of an HIV infected child with subclinical pancreatic dysfunction being reported from India.

A 3 yr old girl born of non-consanguineous marriage was referred to Pediatric & Perinatal HIV clinic of B.J. Wadia Hospital for Children, Mumbai, in March 2004 in view of a positive HIV ELISA test. She had repeated episodes of pneumonia in the past and required hospitalizations for the same. She was also treated for pulmonary Koch’s for 1 yr at the age of 2 yr. At present, she had fever, respiratory distress and cough since preceding 10 days. On examination, she had Grade III clubbing with tachypnoea and chronic skin dermatitis. She had bronchial breathing in right axillary region with cardiomegaly and hepatosplenomegaly. She had failure to thrive with weight of 9 kg (<5th centile) and height of 84.5 cm (<5th centile). Her investigations revealed severe anaemia (haemoglobin of 4 g/dl) with white cell count of 13,000/μl and raised ESR (57 mm at end of 1 h). Her sputum culture grew Candida albicans and X-ray chest showed bilateral patchy consolidation. Mantoux test was negative. Echocardiography revealed moderate pulmonary hypertension with dilated right atrium, right ventricle and main pulmonary artery with left ventricular ejection fraction of 60 per cent. High resolution CT scan of the chest revealed consolidation in right upper lobe and both lower lobes with enlarged mediastinal and hilar lymph nodes. Her HIV status was reconfirmed by a repeat HIV ELISA test by a different kit (DETECT-MC) which was also positive and vertical transmission was confirmed by testing both parents for HIV who were found to be positive. She was given intravenous antibiotics, fluconazole and trimethoprim-sulphamethoxole prophylaxis. Furosemide, arginine and aspirin were started for her pulmonary hypertension. In view of severe anaemia, packed cell transfusions were given. Pneumonia did not respond to antibiotics and in view of non-response with failure to thrive and generalized lymphadenopathy, she was again started on 4 drug antituberculous therapy (ATT) as TB is endemic in India and is the commonest cause of chronic pneumonia in our country.

On follow up after 2 months, she still did not have any clinical improvement. Her CD4 count was 670 cells/μl and CD4 per cent was 11.8 with CD4:CD8 ratio of 0.14 suggestive of severe immunosuppression. Her baseline liver enzymes and renal function tests were normal. She was now shifted to 2 drugs ATT (INH + rifampicin) and antiretroviral therapy (ART) consisting of zidovudine (AZT), lamivudine (3TC) and nevirapine (NVP) was started. She responded clinically, had a weight gain of 2 kg in 1 month and fever, cough subsided and X-ray chest showed an improvement. However, her serum biochemistry at the end of one month of ART showed a high amylase (1301 IU/l). Her serum lipase was also elevated (413...
Ultrasound of the abdomen showed bulky pancreas with peripancreatic and splenic lymphadenopathy. Clinically she had no abdominal pain or vomiting. There was no parotid enlargement. In view of subclinical pancreatitis, she was advised stopping ART. ATT was continued. Serial amylase and lipase levels over the next 5 months remained elevated (Fig.) though the child clinically remained asymptomatic. Arginine which is known to cause pancreatic dysfunction, was stopped. Her serum lactate was normal. In view of peripancreatic lymphadenopathy, a short course of steroids was given for 15 days but there was no decrease in the size of the nodes and hence was omitted. In view of asymptomatic pancreatic enzyme elevation which was due to HIV infection itself, ART was restarted in December 2005 after a period of 8 months with serial monitoring of pancreatic enzymes. Again, the child had a weight gain of 2 kg in one month and there was a gradual decrease in the serum amylase levels and ultrasound of the abdomen showed normal pancreas with decrease in the size of the abdominal lymph nodes. The child is on regular follow up till date.

The aetiology of pancreatic dysfunction in HIV is multifactorial. A study from New York has found the prevalence of hyperamylasaemia and hyperlipasaemia among randomly selected asymptomatic HIV-infected adults to be 28 and 18 per cent respectively. Similar findings were noted in our patient where even after a period of 1 yr of initial detection of hyperamylasaemia and hyperlipasaemia, the child had no clinical manifestations of pancreatitis. Hyperamylasaemia may be seen with salivary involvement and hence elevated serum lipase may be a better indicator for identifying such patients. Our patient had no salivary gland enlargement and had both elevated lipase and amylase with bulky pancreas on initial ultrasound examination suggestive of pancreatic involvement though child was clinically asymptomatic for the same. Though opportunistic infections and drugs have been implicated with pancreatitis, it has been found that HIV can lead to pancreatic disease and can occur not only in patients with AIDS, but in asymptomatic carriers and in patients with lymphadenopathy syndrome. Our patient had pulmonary tuberculosis but abnormal levels of pancreatic enzymes did not come to normal with antituberculous therapy even though X-ray chest showed an improvement. Through a few cases have been reported with pancreatic disease and miliary TB, TB leading to pancreatitis is extremely rare. Also mitochondrial damage due to ART was ruled out in
view of normal blood lactate. Other drugs that can lead to pancreatic abnormality were also omitted but there was no improvement in the biochemical parameters suggestive that the pancreatic abnormality in this child was due to HIV as amylase levels remained elevated even after 8 months of stopping ART initially. Subsequently after treatment with ART, there was a marked decrease in serum amylase levels. However, it is not possible to comment whether the reduction in serum levels of pancreatic enzymes was due to the ART or was a spontaneous regression.

To conclude, pancreatic abnormality may occur in HIV infected children, therefore, serum levels of pancreatic enzymes needs to be closely monitored if antiretroviral therapy is continued in such cases.

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References