Invasive aspergillosis is described more frequently as a complication of neoplastic disease and in immunocompromised patients. Hepatic failure is not a generally recognized risk factor for pulmonary aspergillosis. We report a 3-year-old boy who presented with hepatic failure and pneumonia and whose autopsy revealed liver cirrhosis and pulmonary aspergillosis. [Indian J Gastroenterol;2004;23:147-148]

Key words: Cirrhosis of liver

Pulmonary aspergillosis in children has been generally associated with immunocompromised state, neutropenia, broad-spectrum antimicrobial therapy, and acute organ rejection. We describe a child who presented with hepatic failure and pneumonia in whom autopsy revealed pulmonary aspergillosis.

A 3-year-old boy presented with fever for 15 days, associated with abdominal distension and pedal edema for 7 days, and seizures and altered sensorium for 4 days. He also had dark yellow urine and gum bleeding for 3 days. There was no significant past medical history.

On examination, he was febrile and disoriented. His height was 12 kg and height 96 cm. He was pale and icteric. Abdominal examination revealed shrunken liver with span of 5 cm, and ascites. Respiratory system examination revealed decreased breath sounds on the right side and bilateral scattered crepitations.

Investigations: hemoglobin 5.6 g/dl, WBC count 3900/ cmm (N 20%, L 64%, M 12%, B 3%, E 1%) and platelets 3000/ cmm. Prothrombin time was 33.5 s (control 10.6) and INR 3.23; aPTT was 109 s (control 29.8). Total bilirubin level was 26.9 mg/dl (direct 20.8), total protein 3.5 g/dl (albumin 1.7), AST 4080 U/L, ALT 1020 U/L, and alkaline phosphatase 214 U/L. Serum ceruloplasmin was 5 U/L, serum creatinine 1.1 mg/dl, serum calcium 6.5 mg/dl, serum electrolytes normal. Malarial parasite, Widal test, blood culture, leptospirosis MAT, dengue IgM and HBsAg were negative.

Chest X-ray showed bilateral pulmonary infiltrates, more on the right side, with mild effusion. He was diagnosed to have hepatic failure with encephalopathy and bronchopneumonia, and was started on antibiotics, anti-hepatic coma measures, vitamin K, dextrose infusion, and fresh frozen plasma. He required mechanical ventilation for impending respiratory failure. In spite of the above measures he died within a few hours of admission.

Autopsy showed liver weight of 330 grams. Histology of the liver showed cirrhosis with bridging necrosis, cholestasis, and regenerative changes with fibrosis. Lungs showed foci of intra-alveolar hemorrhage, edema, and septae containing branching hyphae filaments of Aspergillus involving small and medium-sized vessels and pleura (Fig). The postmortem diagnosis was acute liver injury in a child with chronic liver disease with aspergillosis in the lungs and pleura.

Invasive aspergillosis is described more frequently as a complication of neoplastic disease and in immunocompromised patients. Hepatic failure is not a generally recognized risk factor for pulmonary aspergillosis. A few cases of pulmonary aspergillosis as a complication of cirrhosis have been reported. A review of literature revealed that 5 of the 14 previously reported cases of invasive aspergillosis in seemingly immunocompetent hosts were associated with liver disease.

Patients with fulminant hepatic failure have depressed hexose monophosphate shunt and phagocytosis-associated metabolic burst activity, which may increase their risk for severe infections.

Amphotericin B is the drug of choice for pulmonary aspergillosis. The drug may be considered in a child with hepatic failure, if aspergillosis is found in the sputum or if pulmonary infiltrates persist despite broad-spectrum antibiotics.

References
Cerebral venous sinus thrombosis as presenting feature of Crohn’s disease

S C Samal, Sushma Patra,
D Chandrasekhar Reddy, U P Sharma

Global Hospital, Center for Liver Transplantation,
Hyderabad 500 004

Hepatobiliary and vascular manifestations are rare form of extraintestinal manifestations in Crohn’s disease. We report a 20-year-old man in whom cerebral venous sinus thrombosis was the presenting symptom and preceded the diagnosis of Crohn’s disease. [Indian J Gastroenterol 2004;23:148-149]

Key words: Sinus thrombosis, intracranial

The incidence of extraintestinal manifestations of inflammatory bowel disease (IBD) is reported to be 25% to 35%.

Hepatobiliary and vascular manifestations are rare forms of extraintestinal manifestations, occurring in 4% and 1.3% of cases, respectively.

A 20-year-old man presented with headache, vomiting and seizure of three days’ duration. On examination he had pallor and bilateral papilledema without focal neurological deficit. He did not have history of diabetes, hypertension or tuberculosis. Other systemic examination did not reveal any abnormalities. Investigations: CT scan brain and magnetic resonance imaging (MRI) of head (Fig.) showed superior sagittal sinus and left transverse sinus thrombosis; antiphospholipid antibody was negative, homocysteine levels were normal and hemoglobin was 8.9 g/dL. He was better after treatment with phenytoin and oral anticoagulants.

Three months later he presented with jaundice and mild hepatomegaly. Investigations: serum bilirubin 18 mg/dL (direct 13), AST 122 IU/L, ALT 127 IU/L, alkaline phosphatase 427 IU/L. HBsAg, anti HCV antibody, anti HAV IgM, anti HEV IgM were negative. Possibility of drug-induced hepatitis was considered and phenytoin was stopped. On follow up after 3 months he had symptoms of severe anemia with anasarca. Investigations revealed it to be iron deficiency anemia. Mild jaundice was persistent. His alkaline phosphatase was 2842 IU/L.

On questioning, he revealed that he used to pass 2-3 loose stools every day for the last 4-5 years, with occasional blood in the stool. Colonoscopy revealed patchy ulcers in the sigmoid colon and cecum. Biopsy was taken; histology showed

non-cascading granulomas, consistent with Crohn’s disease. ERCP was normal. The patient refused liver biopsy. He was started on 5-ASA and ursodeoxycholic acid. After 6 months he was asymptomatic; he had no loose motions. Hemoglobin was 11.8 g/dL; serum alkaline phosphatase decreased but was still elevated. He was on antiepileptic medication as advised by his neurophysician. A final diagnosis of Crohn’s disease with thrombotic complication and probable pericholangitis was made.

Patients with IBD have an increased risk of thrombosis. They tend to suffer thrombosis earlier in life, as in the present case. In a series of 7199 patients with IBD, 92 patients (1.3%) suffered a thrombotic complication. Of these, only 9 patients had cerebral vessel involvement. Hypercoagulable states may arise from active bowel inflammation, factor V Leiden mutation, and defective methylene tetrahydrofolate reductase. However, in more than one half of the patients no predisposing factor can be identified. In our patient, except antiphospholipid antibody and homocysteine level, other investigations were not done.

Cerebral venous sinus thrombosis is more often encountered than arterial thrombosis and usually occurs in a patient with diagnosed Crohn’s disease. In our patient cerebral thrombotic manifestations preceded the diagnosis of IBD by 6 months.

Thrombosis in IBD is a cause for concern because of the high associated mortality and its occurrence in a relatively young population.

References