sarcoidosis, if bile duct damage is present it is less conspicuous; granulomas are abundant and well-formed. Lobular granuloma commonly occur in sarcoidosis and are rare in PBC.

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A fatal case of disseminated aspergillosis in an immune-competent patient- An autopsy case report

Introduction

Invasive aspergillosis has emerged as a cause of severe infection in immune-compromised patients. The lungs are primarily affected, likely due to its portal of entry through the respiratory tract. Very few cases in the literature show primary involvement of the gastrointestinal tract.[1] Also disseminated aspergillosis in the immune-competent state is rare.[2-3]

Case report

A 70-year old male patient, admitted to our tertiary care hospital, had chief complaints of pain in abdomen and inability to pass stools for 8 days. He had no complaints of fever, nausea, vomiting, yellowish discoloration of urine or stools or any significant past surgical or medical history of tuberculosis, diabetes mellitus, hypertension or bronchial asthma. He was afebrile and conscious with pulse of 98 beats/min and blood pressure of 110/70 mmHg. His abdomen was distended and tender, with guarding and absent bowel sounds. His hemoglobin was 10.7 g/dL, WBC count of 10,700/mm^3 (Polymorphs-89%, Lymphocytes-8%, Eosinophils-3%) and platelet count of 1.43 x10^5/mm^3. Random blood sugar level was 167g/dL. The serology status of the patient was negative for HIV, HCV and HBsAg. Ultrasonography of the abdomen showed distended bowel loops with gas shadows, suggestive of partial intestinal obstruction. Immediate emergency laparotomy showed 0.5 cm x 0.5 cm perforation, 50 cm proximal from the ileocaecal junction with dense adhesions and perforative peritonitis.

A primary closure of the perforation site was performedwith drain in situ. The postoperative general condition was moderate and the patient was afebrile. But, a leak from the drain was noted. Re-exploration showed new serosal tear of the small intestine, for which primary suturing was done. His general conditions were stable. From post-operative day five, he developed intermittent spikes of high bloodpressure. Electrocardiogram showed depression in leads II and III. Troponin-T was positive. He developed pleural effusion with consolidation, bilaterally. WBC counts increased to 18,360/mm^3. Tracheal SCAST was positive for Klebsiella pneumonia species. He died on twelfth post-operative day.

At autopsy, the stomach fundus showed a large necrotic ulcer, with multiple small ulcers along its lesser curvature. Small and large intestine showed pre-gangrenous changes with serosa covered with greenish yellow exudates and thinned out, dusky mucosa containing altered blood (**Figure 1**). Also, the small intestine showed suture-site covered with similar exudates and few tiny small ulcers. Bilateral lungs showed target lesions, characteristic of angio-invasive aspergillosis (**Figure 2**). The heart showed small endocardial vegetations

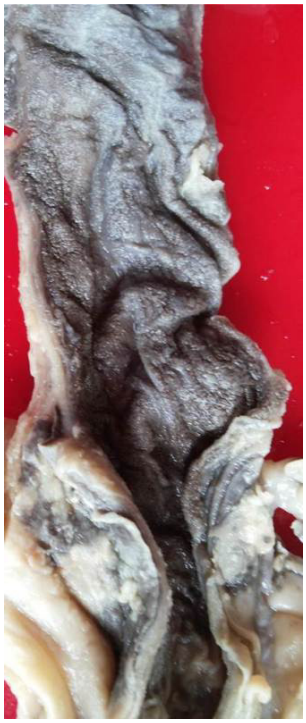

Figure 1: Small intestine showing pre gangrenous changes

Figure 2: Targetoid lesions in the lung characteristic of angio invasive aspergillosis

with left anterior descending coronary artery showing 100% block. Liver and spleen capsules showed focal yellowish exudates. There was no focus of infection in the brain.

Microscopy of the small intestine showed features of gangrene with perforation, superadded infection and the presence of slender septate, acutely branched fungal filaments of Aspergillus, which were further confirmed with special stain of Gomori Methanamine Silver (GMS) (**Figure 3**).

The lungs showed features of acute invasive aspergillosis with lung infarction and pulmonary vessels showing septic

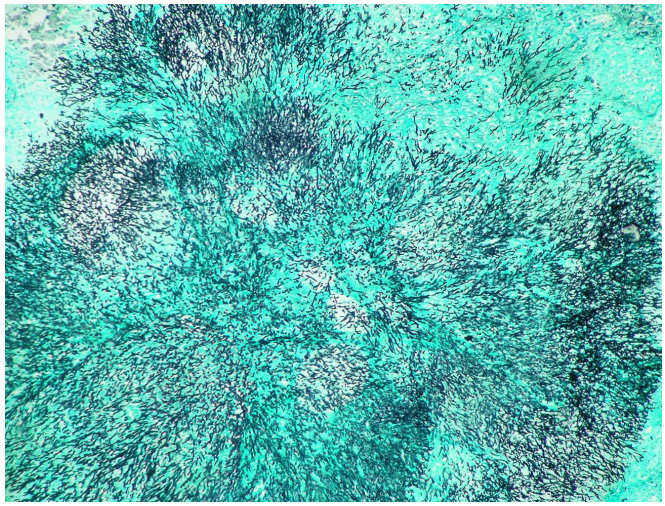

Figure 3: Slender septate acutely branched fungal filaments of Aspergillus confirmed by special stain of Gomori Methanamine Silver (GMS)

fungal fibrin thrombi. The cardiac vegetation, seen on gross examination, corresponded to septic infective endocarditis with the presence of few fungal filaments. The left anterior descending coronary artery showed an 80% fibro-calcific plaque. The liver and spleen capsules were thickened with moderate mixed inflammation.

The final cause of death was disseminated aspergillosis involving the gastrointestinal tract, lungs and heart with acute coronary insufficiency.

Discussion

Few reports in the literature show gastrointestinal aspergillosis, without pulmonary involvement as the initial presentation of disseminated disease.[4] Involvement of extrapulmonary sites such as the skin, brain, gastrointestinal tract, heart and kidney are rare.

Gastrointestinal aspergillosis may be clinically asymptomatic or rarely present as a localized tumour like mass (aspergilloma) or as locally infiltrative lesions causing intestinal ischemia and obstruction. The gastro-intestinal tract can be involved via esophageal or bowel mucosal ulceration or via dissemination from a primary pulmonary aspergillosis. The aspergillus spores cannot survive in the normal gastric mucosa. However, ulceration and a necrotic tissue background provides a ready environment for the germinating spores. Many patients of gastrointestinal aspergillosis are asymptomatic. Invasive aspergillosis should be considered in the differential diagnosis of an otherwise undiagnosed febrile respiratory illness, even in immune-competent patients.[5]

Considering the acute course of the disease in our patient, it seems likely that gastro-intestinal aspergillosis preceded the pulmonary and cardiac aspergillosis. The gastro-intestinal ulcers may have been the primary site from which further infective dissemination may have occurred. Subsequently, these ulcers perforated and peritonitis developed. Also, there was no evidence of immune-compromised status of our patient.

Thus, this is a very rare clinical presentation of fatal invasive aspergillosis in an immune-competent elderly male.

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Megacystic microcolon intestinal hypoperistalsis syndrome with mydriasis in a male child

Introduction

Megacystis microcolon intestinal hypoperistalsis syndrome (MMIHS) is a rare, congenital disease affecting smooth muscle peristalsis mainly in the gastrointestinal tract and urinary bladder. Also known as Berdon disease, as first described by W E Berdon and coworkers[1], is a severe and often lethal form of neonatal pseudo obstruction. It is characterized by hypoperistalsis or a peristalsis of the gastrointestinal (GI) tract, malrotation, microcolon, and genitourinary abnormalities namely, a non-obstructed dilated bladder[2]. The prognosis is extremely poor, with most patients dying within the first 6 months without treatment[3].Although the disease is often diagnosed in female infants, we describe a male child with late diagnosis in childhood with bilateral mydriasis.

Caser report

A 6 year old male child admitted with recurrent vomiting, abdominal pain, and failure to thrive since 1 year of age. Patient had normal growth velocity upto age of 1 year. After that weight and height did not increase as compared to other sibling. However language and motor milestones were normal. Patient had abdominal distention since last 1 year which was painless and more in the central and lower abdomen. His weight was 13 kilograms and height was 80 cm, both of which were below 3rd percentile for his age .His birth weight was 2.7 kilograms and was full term. He had one elder brother, who wasnormal.On examination patient had distended abdomen which was tympanic without evidence of any free fluid or organomegaly. Haematological and biochemical investigations showed mild anaemia. Abdominal X ray showed dilated bowel loops without significant air fluid levels or free gas. Barium study showed slow transit of barium in dilated bowel loops with normal ileocaecal valve and colon. CECT abdomen suggestive of dilated small bowel loops with moderate enlargement of left pelvicalyceal system, left entire ureter & over distended bladder. DMSA renal scan of both right and left kidney showed normal functioning cortical mass with no evidence of any cortical defect. Micturating cystourethrogram showed distended bladder with smooth outline without any evidence of residual urine, filling defect, diverticulum, vesicoureteral reflux with normal anterior and posterior urethral valves. Upper G.I endoscopy showed dilated esophagus, dilated stomach and dilated duodenum. However there was no evidence of any mechanical obstruction. Esophageal manometry was done to