in the epigastrium. Ultrasound of the abdomen revealed dilated duodenum with reverse peristalsis. Upper gastrointestinal (GI) endoscopy showed dilated first and second part of the duodenum. The child was resuscitated aggressively. Visible left to right peristalsis and bilious vomiting raised the possibility of an obstructive aetiology. Exploratory laparotomy was performed. It was found that there was partial malrotation of the midgut with dilated incomplete C loop of the duodenum and absent ligament of Treitz. The SMA was posterior to the duodenum, the bands of Ladd were overlying the anterior surface of the duodenum and the mesentery was short and narrow. The bands were divided, mesentery widened and the caecum was replaced and fixed in the right iliac fossa. Postoperative recovery was uneventful. The child started feeding well and gained weight. At 8 months of follow-up, he had gained up to 12 kg.

The second case was a 22-year-old woman who presented with a history of weight loss, vague abdominal pain and distension of 8 years duration. She had undergone surgery for congenital diaphragmatic hernia (CDH) soon after birth. Abdominal examination revealed visible left to right peristalsis. Barium meal showed dilated duodenum and small bowel with markedly delayed emptying. In view of uncertain diagnosis, she was taken up for exploratory laparotomy and was found to have GM with caecum and appendix in the left hypochondrium and there was no duodenojejunal (DJ) flexure. Adhesions were released, mesentery widened and appendectomy was done. The postoperative recovery was good and after 6 months, the patient has gained 17 kg of weight.

Discussion

Congenital bands extending from the right lateral abdominal wall, across the duodenum to the caecum are called the bands of Ladd. These may cause external compression to the duodenum and may be the cause of upper abdominal pain, bloating, loss of appetite and subsequently malnutrition and failure to thrive. In our first case, there was no rotation of the proximal segment, leading to absence of C loop of the duodenum and ligament of Treitz and also the SMA lying posterior to the duodenum. The second case was also of partial GM and it was associated with CDH. The most common congenital anomaly associated with CDH is intestinal malrotation and it is present in almost all cases of CDH.[2]

Presentation of GM after childhood is rare and, as seen in the reported cases, is that of failure to thrive, unexplained weight loss and vague abdominal symptoms.[3] GM is usually not considered as an aetiology because the symptoms are vague and non-specific. This may lead to delay in diagnosis and treatment. For these reasons, it is crucial that all surgeons should have a high index of suspicion for such a late presentation of GM in malnourished patients with upper GI symptoms; and have firm knowledge of intestinal embryology and its anatomical variations.

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References

1. von Flüe M, Herzog U, Ackermann C, Tondelli P, Harder F. Acute and chronic presentation of intestinal nonrotation in adults. *Dis Colon Rectum*. 1994;**37**:192–8.
2. Mattei P. Congenital diaphragmatic hernia. In: Fundamentals of pediatric surgery. *New York, USA: Springer*; 2011:535.
3. Gamblin TC, Stephens RE Jr, Johnson RK, Rothwell M. Adult malrotation: a case report and review of the literature. *Curr Surg*. 2003;**60**:517–20

Intracranial fungal infections in chronic liver disease: a report of two cases

Introduction

We report two patients of advanced cirrhosis who developed intracranial fungal infections, each of them having variable outcome with the treatment administered.

Case report

Case 1:

A 40-year-old man with alcoholic liver disease (Child C cirrhosis; Child–Pugh score 12) presented with complaints of fever for one month and altered sensorium for a week. He was in grade

IV encephalopathy. Deep tendon reflexes were asymmetrically exaggerated on the right side. He had polymorphonuclear leukocytosis. Liver function tests showed serum bilirubin level of 2.32 mg/dL (0.3–1.3 mg/dL), aspartate transaminase/alanine transaminase (AST/ALT) of 67/47 IU/mL (<40 IU/mL) and alkaline phosphatase level of 96 IU/mL (<128 IU/mL). Serologies for human immunodeficiency virus (HIV), hepatitis B and hepatitis C were non-reactive. Blood, urine and tracheal aspirate cultures were sterile. There was no evidence of spontaneous bacterial peritonitis. He received lactulose and intravenous (IV) broad-spectrum antibiotics (piperacillin/tazobactam and vancomycin). Computerized tomographic (CT) scan of the brain showed multiple hypodense lesions in the left temporo-parietal region (**Figure 1**). Magnetic resonance imaging (MRI) of the brain revealed multiple bilateral T2W hypointense lesions with diffusion restriction suggestive of multiple abscesses (**Figure 2**) and evidence of sinusitis in the form of soft tissue thickening of the paranasal sinuses (**Figure 3**). Tissue scrapings from the nasal cavity and sinuses showed acutely branched septate hyphae and the cultures grew Aspergillus species. He was started on parenteral voriconazole; however, he did not show improvement and his condition worsened. He received ventilatory support, IV fluids and vasopressors for multiorgan dysfunction. However, the patient could not be salvaged and succumbed to his illness.

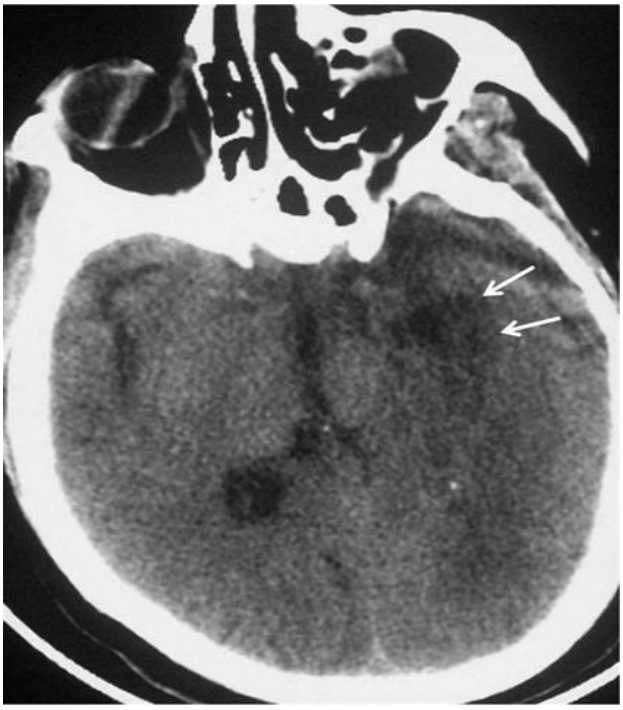

Figure 1: CT scan of the head showing a hypodense lesion suggestive of infarct (white arrows) in the temporoparietal region on the left side.

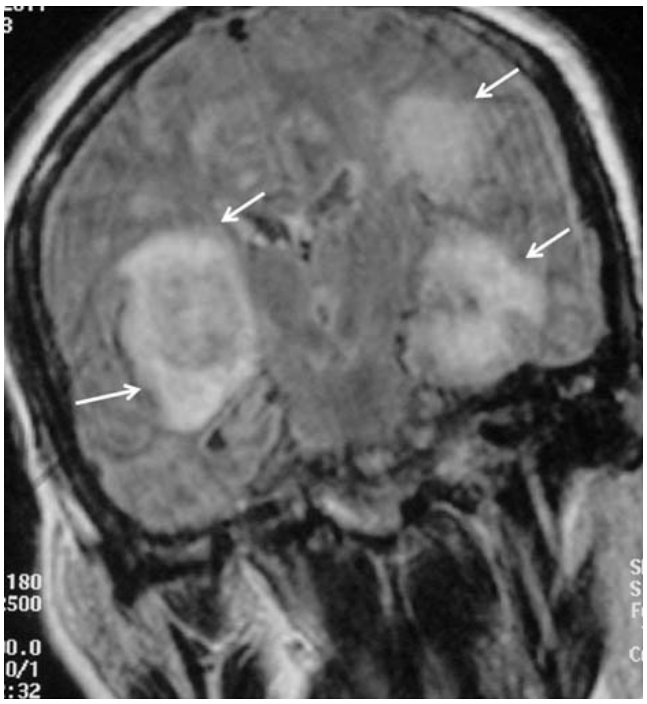

Figure 2: Coronal fluid attenuated inversion recovery (FLAIR) image showing heterogeneously hypointense lesions (white arrows) in bilateral temporoparietal regions with perilesional oedema.

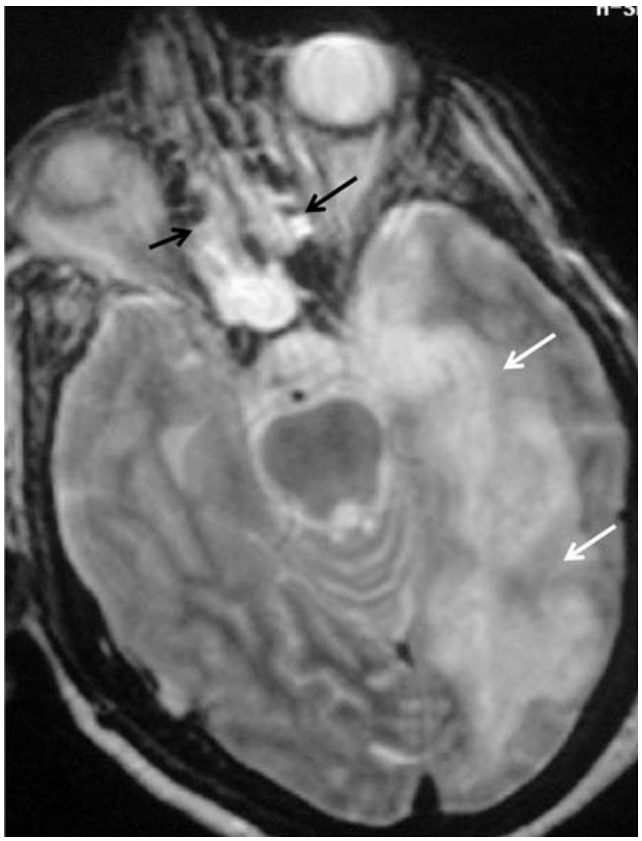

Figure 3: Axial T_2W image showing iso- to hypointense lesion (white arrows) in the left temporal lobe with perilesional oedema, associated right sphenoid and B/L ethmoid sinusitis (black arrows).

Case 2:

A 45-year-old man was suffering from cirrhosis of the liver due to chronic alcoholism (Child B; Child–Pugh score 7). He presented with fever and gait ataxia for the past 7 days. Cerebrospinal fluid (CSF) examination revealed: total cell count of 160 (70% lymphocytes)/μL (0–5 mononuclear cells); protein level of 167 mg/dL (15–50 mg/dL), sugar 14 mg/dL (40–70 mg/dL); adenosine deaminase 6 IU/mL (<10 IU/mL). CSF cultures were sterile and tuberculosis-polymerase chain reaction (TB–PCR) was negative. CSF cryptococcal antigen (by latex agglutination method) was positive with a titre of 1:8. Serology for HIV was non-reactive and CD4 count was 535 cells/mm^3. MRI of the brain revealed conglomerate ring-enhancing lesions in the peritrigonal area and right periventricular region, minimal perifocal oedema and ependymal enhancement (**Figure 4**) with focal parenchymal abnormal enhancement also seen in B/L medial temporal lobes (**Figure 5**). Serology for toxoplasmosis was negative. The patient was started on liposomal amphotericin and subsequently parenteral voriconazole was given. He showed improvement with this treatment and was discharged. He was doing well at 4 months of follow-up.

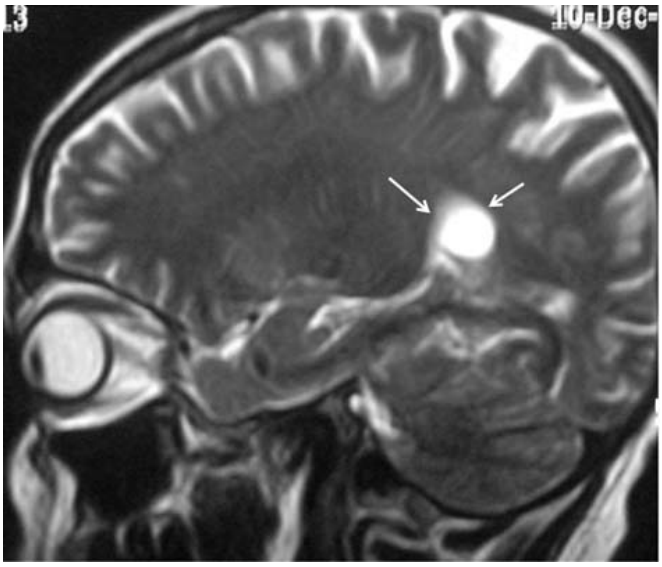

Figure 4: MRI of the brain showing a ring-enhancing lesion in the peritrigonal area with minimal perifocal oedema and ependymal enhancement (white arrows).

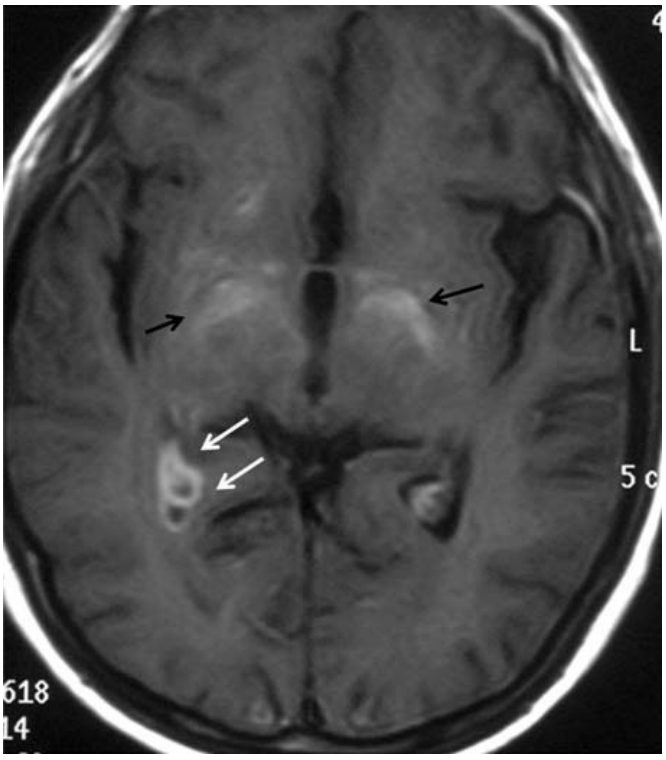

Figure 5: Axial post-gadolinium T_1-weighted image showing ring-enhancing lesions in the right periventricular location (white arrows). Focal parenchymal abnormal enhancement is also seen in the B/L medial temporal lobes (black arrows).

Discussion

Liver cirrhosis is an immunocompromised state. It predisposes to a variety of infections with 30% mortality.[1] One-third of admissions among patients with cirrhosis are due to bacterial infections.[2] The following defences are impaired in cirrhosis leading to increased susceptibility to infections: (i) Liver kupffer cells and sinusoidal endothelial cells (account 90% of reticulo-endothelial system);[3] (ii) leukocyte activation, chemotaxis, phagocytosis, cytokine secretion, oxidative burst and microbicidal activity;[4] (iii) peripheral CD4 T-lymphocytes, complement levels, immunoglobulins.[5-7] Associated portosystemic shunting, hypersplenism, malnutrition, alcoholism, and immunosuppressive medication[5-7] further increase the susceptibility to infections. Additional risk factors predisposing to invasive fungal infections are exposure to broad-spectrum antimicrobials, cancer chemotherapy, indwelling vascular catheters, total parenteral nutrition, prior gastrointestinal surgery, renal failure, haemodialysis, transplantation and diabetes.[8-10] Candida, Aspergillus, Mucor, Cryptococcus neoformans are frequently encountered causing peritonitis, meningitis, pneumonia, cholangitis and blood stream and urinary tract infections.

The diagnosis of intracranial fungal infections in cirrhosis remains challenging and is often missed due to non-specific clinical signs, poor yield of the fungal cultures and limited information provided on radiodiagnosis. Clinicians ought to be aware of the possibility of fungal infections in cirrhosis as these may contribute to adverse outcomes.

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References

1. Tandon P, Garcia-Tsao G. Bacterial infections, sepsis, and multiorgan failure in cirrhosis. *Semin Liver Dis*. 2008;**28**:26–42.
2. Fernández J, Navasa M, Gómez J, Colmenero J, Vila J, Arroyo V, et al. Bacterial infections in cirrhosis: epidemiological changes with invasive procedures and norûoxacin prophylaxis. *Hepatology*. 2002;**35**:140–8.
3. Ghassemi S, Garcia-Tsao G. Prevention and treatment of infections in patients with cirrhosis. *Best Pract Res Clin Gastroenterol*. 2007;**21**:77–93.
4. Wasmuth HE, Kunz D, Yagmur E, Timmer-Stranghoner A, Vidacek D, Siewert E, et al. Patients with acute on chronic liver failure display "sepsis-like" immune paralysis. *J Hepatol*. 2005;**42**:195–201.
5. Lombardo L, Capaldi A, Poccardi G, Vineis P. Peripheral blood CD3 and CD4 T-lymphocyte reduction correlates with severity of liver cirrhosis. *Int J Clin Lab Res*. 1995;**25**:153–6.
6. Ono Y, Watanabe T, Matsumoto K, Ito T, Kunji O, Goldstein E. Opsonophagocytic dysfunction in patients with liver cirrhosis and low responses to tumor necrosis factor-alpha and lipopolysaccharide in patients' blood. *J Infect Chemother*. 2004;**10**:200–7.
7. Gomez F, Ruiz P, Schreiber AD. Impaired function of macrophage Fc gamma receptors and bacterial infection in alcoholic cirrhosis. *N Engl J Med*. 1994;**331**:1122–8.
8. Concia E, Azzini AM, Conti M. Epidemiology, incidence and risk factors for invasive candidiasis in high-risk patients. *Drugs*. 2009;**69**:5–14.
9. Meersseman W, Lagrou K, Maertens J, Van Wijngaerden E. Invasive aspergillosis in the intensive care unit. *Clin Infect Dis*. 2007;**45**:205–16.
10. Falcone M, Massetti AP, Russo A, Vullo V, Venditti M. Invasive aspergillosisis in patients with liver disease. *Med Mycol*. 2011;**49**:406–13.

Endometriosis causing small bowel obstruction

Introduction

Endometriosis refers to the presence of endometrial stroma and glands outside the uterine cavity and musculature.[1] Ectopic implants can occur anywhere in the body. Intraperitoneal sites include the ovaries (30%), uterosacral and large ligaments (18%–24%), fallopian tubes (20%) and gastrointestinal tract. Extraperitoneal sites include the cervix (0.5%), vagina and abdominal scars.[2] Endometriosis is diagnosed in 1% of patients undergoing major surgery for any gynaecological indication.[3]

Endometriosis has protean clinical manifestations that include pelvic pain (often more severe during menstruation), dysmenorrhoea, infertility, deep dyspareunia, cyclical bowel or bladder symptoms and abnormal menstrual bleeding. Symptoms are initially cyclical (40%) and become permanent when the lesions progress. Bowel endometriosis (BE) can mimic irritable bowel syndrome, infectious diseases, ischaemic enteritis/colitis, inflammatory bowel disease and neoplasms. BE presents with relapsing bouts of abdominal pain, abdominal distention, tenesmus, constipation, diarrhoea, rectal bleeding and pain during defaecation.[3] BE should be suspected in young, nulliparous patients with abdominal pain, cyclical symptoms in the initial phase along with signs of obstruction.

Case report

A 43-year-old woman presented with recurrent periumbilical colicky pain for the past 6 months, associated with bilious vomiting and constipation, each episode lasting for about 1 week. There was no past history of diarrhoea, gastrointestinal bleed, jaundice, fever, weight loss or surgery. She had two children and her menstrual cycles were regular.

General physical examination was unremarkable. Abdominal examination showed generalized distension with visible intestinal peristalsis. There was epigastric and periumbilical tenderness. Investigations revealed normal haemogram and liver and renal function tests. Abdominal X-ray showed multiple air–fluid levels with prominent proximal small bowel loops suggestive of distal small bowel obstruction. Abdominal contrast–enhanced computed tomography (CT) scan showed similar findings with a short segment distal ileal stricture (**Figure 1**).