

DISSEMINATED ABDOMINAL MUCORMYCOSIS-AN UNUSUAL CAUSE OF GASTRIC PERFORATION

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ABSTRACT

Mucormycosis forms a group of fatal, opportunistic fungal infections with high morbidity and mortality in vulnerable patients. In this report we discuss a case of disseminated mucormycosis in an immunosuppressed child to emphasize the role of imaging in early diagnosis and surgical planning. However the prognosis is grave despite antifungal and radical surgery.

Key words:

Stomach, Mucormycosis, Computed Tomography

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INTRODUCTION

Mucormycosis, also known as zygomycosis and phycomycosis, is an uncommon opportunistic infection and the gastrointestinal form is the rarest with less than few cases reported in literature. It was first described by Paulltauf in 1885[1] and is one of the most rapidly fatal infections known to mankind. Here we describe a severe form of gastrointestinal mucormycosis in a eight year old child who was on chemotherapy for acute lymphocytic leukemia.

Case Presentation

An eight-year-old child diagnosed with B cell ALL in our hospital three years back and having completed chemotherapy

came with complaints of fever and breathlessness. Initial blood investigations showed neutropenia with a total WBC count of 200 IU/ml and blood culture which showed klebsiella pneumonia. On clinical examination there was reduced air entry with sub costal retraction on the left side. Ultrasound guided left pleural aspiration was done and around 100ml of dark reddish colored fluid was sent for analysis. Ultrasound of the abdomen showed an enlarged liver with multiple echogenic lesions involving both lobes of the liver (Figure 1a) and splenomegaly with a well-defined hyper echogenic lesion involving the inferior pole (Figure 1b). CECT abdomen showed peripheral wedge shaped nonenhancing hypodense lesion in the left lobe of the liver and multiple well



Figure 1a



1b

Figure 1a and 1b Ultrasound of the abdomen shows hepatomegaly with multiple echogenic lesions involving both lobes of the liver (curved arrow) and splenomegaly with a peripheral wedge shaped hyperechoic lesion involving the lower pole of spleen (right arrow)

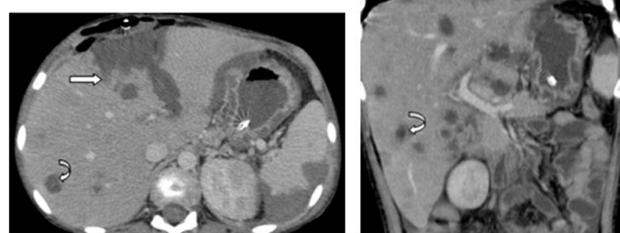


Figure 2a

2b

Figure 2a and 2b Contrast enhanced computed tomography of the abdomen in venous phase shows a peripheral wedge shaped non enhancing hypodense lesion in the left lobe of the liver (right arrow) and well defined peripherally enhancing hypodense lesions involving the right lobe of the liver (curved arrow).

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In addition, multiple infarcts were seen involving the greater curvature of the stomach with breach in the wall and free air tracking along the sub hepatic region (Figure 3).

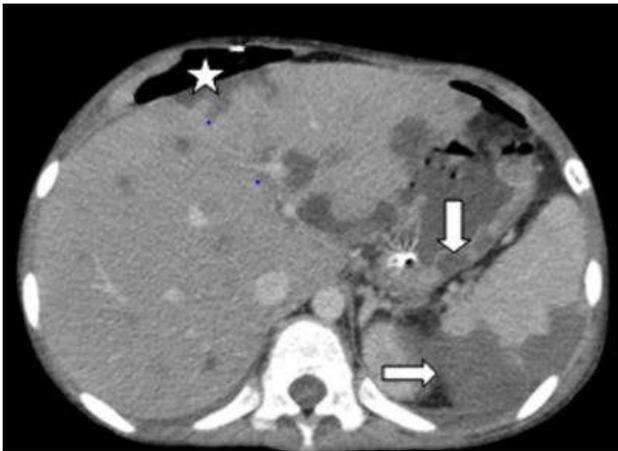


Figure 3

Figure 3 Contrast enhanced computed tomography of the abdomen in venous phase shows non enhancing hypodense lesion involving the greater curvature of the stomach suggestive of infarct (down arrow) with breach in the wall and free air tracking along the sub hepatic region (asterisk). Non enhancing hypodense lesion is seen involving the lower pole of the spleen suggestive of splenic infarct (right arrow).

A nonenhancing hypodense lesion was seen involving the lower pole of the spleen suggestive of splenic infarct (Figure 3). Fusiform aneurysms measuring 6.7 x 4.5 mm was seen in common hepatic artery and measuring 9 x 6.7 mm in the splenic artery (Figure 4a and 4b).

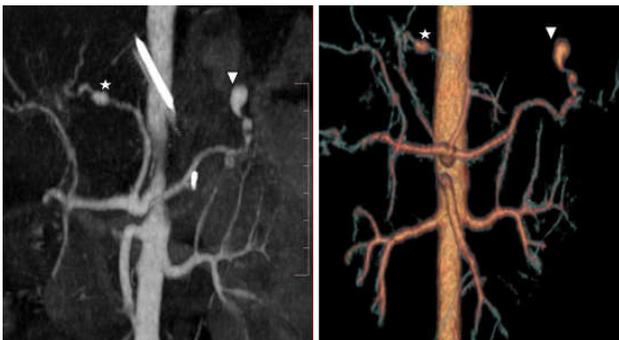


Figure 4a

4b

Figure 4a and 4b Maximum intensity projection and volume rendering images shows fusiform aneurysm of the splenic artery (arrow head) and the common hepatic artery (asterisk).

Emergency laparotomy showed multiple large perforations involving the greater curvature of the stomach (Figure 5a), massive peritonitis, liver and splenic infarcts and massive hemoperitoneum.



Figure 5a

5b

Figure 5a and 5b Intraoperative image shows a perforated wall of the stomach (right arrow) and the specimen of the gangrenous portion of the stomach (asterisk).

The gangrenous portion of the stomach was resected (Figure 5b) and sent for histopathological analysis which showed features of extensive mucormycosis (6a & 6b). During the

second post operative day the child developed tachycardia, desaturation and succumbed.

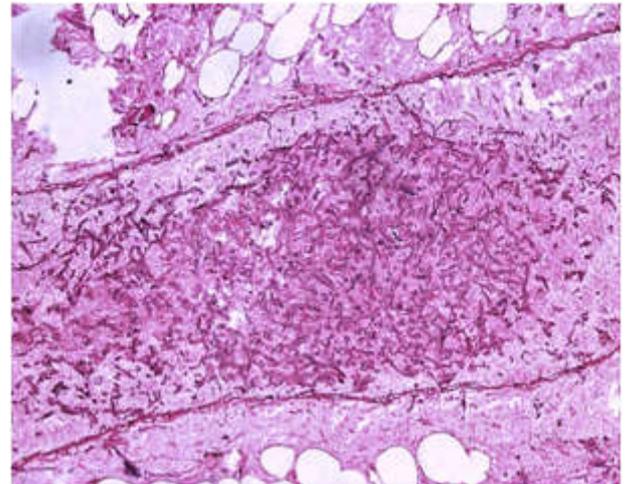


Figure 6a

Figure 6a Photomicrograph of hematoxylin and eosin (x 100) stained biopsy sample of the stomach shows mucormycosis seen in the mucosa.

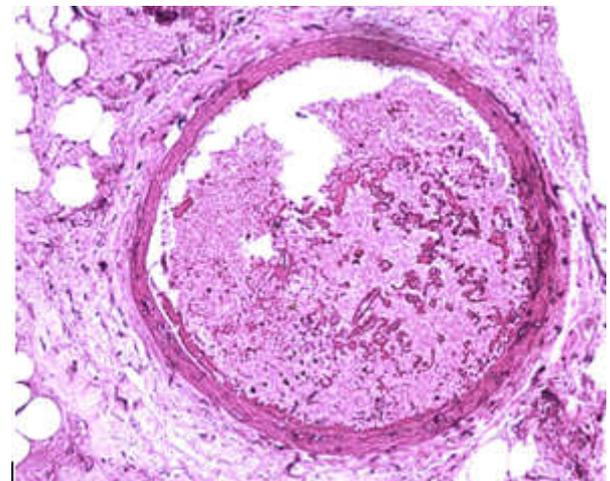


Figure 6b

Figure 6b Photomicrograph of hematoxylin and eosin (x 200) stained biopsy sample shows angioinvasion of mucormycosis.

DISCUSSION

Mucormycosis is a rare fungal infection with high morbidity and mortality. It is caused by the fungi belonging to the Mucorales family which is commonly found in soil and decaying matter. Though it is primarily an airborne infection, other routes of spread have been reported. Only 9% of 185 cases of disseminated mucormycosis have been detected on ante mortem studies [2].

Contaminated foods and prolonged nasogastric intubation is the probable cause of gastrointestinal mucormycosis leading to the development of gastric or colonic ulcers, that facilitate fungal entry.

Mucormycosis can affect any organ, but generally it presents with rhino-orbito-cerebral manifestations or pulmonary manifestations. Patients with diabetic ketoacidosis, lymphoma, transplant recipients, premature infants, immunocompromised patients, trauma and burns victims are more susceptible to this fatal opportunistic infection [3]. Pulmonary and disseminated infections are observed in

lymphoma. A Study by Thomas *et al* shows that about 1% of acute leukemia patients are affected by mucormycosis [4].

Gastrointestinal spread of mucormycosis is known to be one of the rarer and less documented forms of invasion. The stomach is the commonest site of gastro intestinal mucormycosis followed by ileum and colon [5]. Gastrointestinal mucormycosis can also affect the liver and spleen. The common symptoms include nonspecific abdominal pain, abdominal distention associated with nausea and vomiting, fever and hematochezia. The fungi can invade bowel walls and blood vessels, resulting in bowel perforation, peritonitis, sepsis, and massive gastrointestinal hemorrhage, which is a common cause of death [6].

In the early stages, CT may show nonspecific bowel wall thickening, with or without target sign and decreased bowel wall enhancement representing ischemia. Our case, in addition showed micro aneurysm involving the common hepatic artery and the splenic artery with wedge shaped infarcts in the inferior pole of the spleen and the left lobe of the liver. This shows the angioinvasive nature of the fungi; however, a definitive diagnosis can be only made by histopathological examination.

The mortality rate of gastrointestinal mucormycosis is very high, as they are diagnosed at a later stage. The treatment of choice in mucormycosis is antifungal therapy with liposomal Amphotericin-B and aggressive surgical resection.

CONCLUSION

In conclusion, disseminated gastrointestinal mucormycosis is a fatal disease because the diagnosis is often delayed and a thorough knowledge of this rare entity is imperative to both the clinician and the radiologist to reduce the mortality rate in future.

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