Gastric Zygomycosis (Mucormycosis)
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Citation

Abstract
Gastric Zygomycosis (Mucormycosis) is an uncommon fungal infection occurring in different settings of immunosuppression. A high index of suspicion and aggressive treatment may lead to successful outcome.

A patient of gastric Zygomycosis in a renal transplant recipient is reported. Diagnosis was established on histology and confirmed by culture.

INTRODUCTION
Gastric Zygomycosis (Mucormycosis) is rare and occurs predominantly in immunosuppressed individuals due to ingestion of contaminated food or drink. Diabetes and renal transplant are occasionally associated with Zygomycosis. Though earlier reported to have fatal outcome in 98% of patients, successful outcome with medical and surgical therapy is sometimes achieved. We report a case of Gastric Zygomycosis in a post transplant patient who had widespread disease and died shortly after diagnosis.

CASE HISTORY
A 48 year-old male type – II diabetic for seven years with end-stage renal disease underwent renal transplantation from his cousin. Induction of immunosuppression was established with Basilaximab 20 mgs on day 0 and day 4. Prednisolone was administered at 20 mg/Kg and cyclosporine was adjusted to maintain serum levels of 200-250 mg/l. His graft function normalized on day 4. Patient complained of abdominal pain on day 5 with distention and discomfort and treated symptomatically. Ultrasound abdomen and serum amylase were normal. Upper GI endoscopy revealed an erosion on the posterior wall of the stomach. Biopsy was not performed. His pain worsened. A CT-scan done on day 10 revealed pancreatitis with collection in the lesser sac and left pleural effusion. He was treated with Sandostatin 100 mgs subcutaneously 8th hourly. On 26th day chest tube was inserted by video assisted thoracoscopy. Pleural fluid analysis revealed neutrophils. Stains for AFB and fungus were negative.

Immunosuppression was reduced to steroids alone. On 40th post operative day laparotomy was performed. On opening the abdomen post wall of the stomach was necrotic and was excised from the gastro esophageal junction to the middle of greater curvature, with subsequent gastrostomy.

Macroscopic examination revealed a 2x8 cm segment of stomach, brownish black and membranous, with a 3-mm thickness. Due to discoloration, it was not evident which surface corresponded to the mucosa or the serosa.

On microscopic examination, routine H&E stains revealed non-viable gastric mucosa, with extensive necrosis and numerous fungal hyphae. These were broad with irregular thickness and irregular branching. There was vascular invasion. Silver methenamine stains confirmed the morphology (Figure 1). Culture revealed growth of Mucormycosis sp.
On day 42 repeat CT-scan with oral contrast showed evidence of gastrocolic fistula with leakage of contrast into peritoneal cavity. Patient reexplored with subsequent omentectomy and splenectomy. Patient developed hypotension and cardiac arrest during surgery dying shortly after. In the autopsy, there was extensive splenic infarction with vascular invasion of Mucormycosis organisms.

DISCUSSION

The vast majority of Human Zygomycosis is caused by the members of the family Mucoraceae that includes Rhizopus, Mucor, Absidia and Rhizomucor. The morphology of all of them in tissue is similar and indistinguishable from one another; culture identifies them separately and hence Zygomycosis is the preferred term to Mucormycosis when identified in tissue without culture confirmation. Zygomycetes are opportunistic pathogens and require breakdown in immune defenses that lead to neutropenia or neutrophil dysfunction. Neutrophil dysfunction occurs in ketoacidosis and neutropenia is produced by immune suppression induced by transplantation. The patient described in this report had identifiable risk factor of immunosuppression after renal transplantation. The other known risk factors associated with gastric Mucormycosis include malnutrition and peptic ulcer. The pathology ranges from colonization of peptic ulcers to infiltrative disease with vascular invasion and dissemination. It involves blood vessels producing thrombus and infarction leading to perforation of hollow viscous like stomach or intestines. In our patient, diagnosis was established on histology, which showed necrotic material with neutrophilic infiltration. There was histologically demonstrable angioinvasion that lead to spread of infection into omentum and spleen. The patient succumbed to the disease despite medical and surgical treatment. High index of suspicion, early diagnosis, aggressive surgical debridement and prompt antifungal therapy, correction of underlying conditions may improve the outcome.

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References

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