Acute Esophageal Necrosis Due to Posterior Inferior Cerebellar Artery Stroke

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Introduction

Strokes involving the cerebellum, especially the medial vermis, can cause a constellation of severe vertiginous symptoms that include nystagmus, vomiting, gait ataxia, and other truncal and limb incoordination. Vomiting secondary to cerebellar stroke can be difficult to control with medications and can lead to significant morbidity. Intractable vomiting after cerebellar stroke may be a precursor for the development of acute esophageal necrosis (AEN). Acute esophageal necrosis (black esophagus) is a rare syndrome in which presumed hypoperfusion predisposes the esophagus to severe injury via reflux of acid and pepsin.[2] Typically, injury occurs in the distal one-third of the esophagus.[3] In this case, we report the development of AEN related to vomiting from posterior inferior cerebellar artery (PICA) stroke.

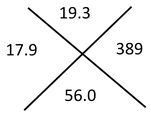
Case Presentation

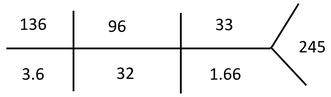
A 59-year-old male with past medical history of essential hypertension, diabetes mellitus type II, and hyperlipidemia presented to the emergency department with complaints of dizziness, nausea, and vomiting lasting over one week. He had previously been evaluated at an urgent care and was diagnosed with an ear infection and given ondansetron for his symptoms of nausea. After persistent bouts of nausea and vomiting, the patient sought further evaluation at the emergency department.

Vital Signs and Physical Exam

T: 98.14 HR 114 BP 115/82 RR 17 SpO2 93% on RA

General appearance: alert, awake, oriented, uncomfortable
Head/Eyes: atraumatic, EOMI, normocephalic, PERRLA
ENT: dry mucosal membrane, poor dentition, normal pharynx
Cardiovascular: tachycardic, normal heart sounds, no murmur
Respiratory: aerating well, clear to auscultation, no distress
Neuro/CNS: alert, oriented X 3, CNII-XII intact, normal speech, no motor deficits, no sensory deficits, horizontal nystagmus





Clinical Course

The patient had an MRI brain which showed a PICA stroke. He was discharged home with aspirin, clopidogrel, and atorvastatin. His nausea was initially well-controlled on diazepam and ondansetron. Two days following discharge, the patient was readmitted due to worsening nausea, vomiting, and vertigo. The patient continued to have refractory retching and vomiting. Despite multiple medications including valium, ondansetron, promethazine, scopolamine patches, and gabapentin, the patient continued to vomit.

On hospital day 2, he developed brisk hematemesis. The patient was taken for emergent upper endoscopy. This revealed LA Grade D esophagitis with the distal half of the esophagus severely inflamed with a sizeable area of black mucosa and friability *(Figure 1).* Due to the severity of the bleeding and inflammation, five hemostatic clips were successfully placed as well as injection of 3 mL of 1:10,000 solution of epinephrine for hemostasis. Post procedure, the patient remained intubated and sedated to allow for adequate control of nausea and to allow healing of the distal esophagus. A pantoprazole drip and TPN were initiated. A J-tube was placed for eventual enteral nutrition. On hospital day 6, surveillance endoscopy again revealed LA Grade D esophagitis but the previously black necrotic tissue in the lower third of the esophagus was now confluent with white exudate *(Figure 2a, 2b).* He was successfully extubated on hospital day 12. The prior intractable nausea and vomiting were now easily controlled with metoclopramide, ondansetron, scopolamine patches, and aluminum-magnesium-hydroxide. He had no further hematemesis.

Endoscopy

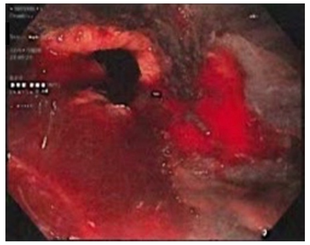


Figure 1. Emergent Endoscopy. LA Grade D esophagitis with near circumferential necrosis of the distal esophagus.

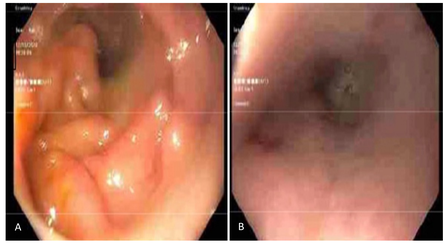


Figure 2. Surveillance endoscopy. Healing necrosis with overlying white exudate. Upper endoscopy performed on hospital day 6 for surveillance.

Discussion

Acute esophageal necrosis is classically described in older men who are critically ill with multiple comorbidities; it is an unusual etiology of hematemesis, especially following a stroke.[1] AEN, though rare, has a significant mortality risk of 38%.[2] Typically, it occurs due to a low-flow vascular state (hypotension, DKA, dehydration, AKI) altering the ability of the esophageal mucosal barrier systems to repair thus predisposing the mucosa to severe surface injury via reflux.[2] The patient's initial presentation of severe nausea and vomiting as well as dehydration likely were the initial events that predisposed his esophagus to such injury leading to hematemesis and discovery of AEN.

This case is a unique presentation of AEN due to a complication of an acute right PICA infarct associated with intractable vomiting. This case demonstrates a multidisciplinary approach to treating a patient with a PICA ischemic stroke accompanied by severe nausea, retching, and hematemesis. Multiple subspecialties including General Surgery, Neurology, Pulmonology, and Gastroenterology collaborated to aid in the successful healing and improvement of the patient's outcome.

Conclusions

This case demonstrates a previously undescribed presentation of acute esophageal necrosis as a complication of a PICA stroke. Our approach using prolonged sedation, mechanical ventilation, and jejunal feedings allowed healing of necrotic esophageal mucosa. This case report may serve as a model for care of this rare syndrome and may stimulate further study.

References

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