Seeing beyond the gut: An unusual cause of massive hematemesis

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ABSTRACT
We report the clinical details, imaging findings, and management of a 74-year-old male who had recurrent episodes of massive hematemesis secondary to rupture of a cavernous internal carotid artery (ICA) aneurysm. Ruptured ICA aneurysms may present with epistaxis. However, intracranial aneurysmal rupture with hematemesis as the presenting complaint has not been described previously in the literature. In this case report we describe the pathophysiology of cerebral aneurysm as a cause of hematemesis and its management.

INTRODUCTION
Hematemesis is vomiting of blood, which generally occurs secondary to oral cavity, esophageal, gastric, duodenal, and systemic diseases. We describe a very rare cranial vascular cause of hematemesis in this report and discuss the imaging findings and its treatment.

CASE PRESENTATION
A 74-year-old man experienced multiple episodes of hematemesis of quantity approximately 1,500 ml, over a span of 24 h. Initially he was taken to a local hospital where fluid resuscitation was done and he was transferred to our centre on inotropic support for further management. Upper gastrointestinal (UGI) endoscopy was done in view of hematemesis, which was normal. Patient was evaluated further with computed tomography (CT) abdominal angiography, which revealed no evidence of any mucosal or submucosal pathology in relation to the UGI tract. Patient developed two more episodes of hematemesis one on third and the other on seventh day of hospital admission. Repeat UGI endoscopy revealed fresh blood trickling from nasopharynx and oropharynx and clotted blood in esophagus and stomach [Figure 1]. In view of fresh blood in nasopharynx, CT angiography of neck and paranasal air sinuses was performed. The sphenoid sinus and posterior ethmoidal air cells showed support for further management. Upper gastrointestinal (UGI) endoscopy was done in view of hematemesis, which was normal. Patient was evaluated further with computed tomography (CT) abdominal angiography, which revealed no evidence of any mucosal or submucosal pathology in relation to the UGI tract. Patient developed two more episodes of hematemesis one on third and the other on seventh day of hospital admission. Repeat UGI endoscopy revealed fresh blood trickling from nasopharynx and oropharynx and clotted blood in esophagus and stomach [Figure 1]. In view of fresh blood in nasopharynx, CT angiography of neck and paranasal air sinuses was performed. The sphenoid sinus and posterior ethmoidal air cells showed support for further management. Upper gastrointestinal (UGI) endoscopy was done in view of hematemesis, which was normal. Patient was evaluated further with computed tomography (CT) abdominal angiography, which revealed no evidence of any mucosal or submucosal pathology in relation to the UGI tract. Patient developed two more episodes of hematemesis one on third and the other on seventh day of hospital admission. Repeat UGI endoscopy revealed fresh blood trickling from nasopharynx and oropharynx and clotted blood in esophagus and stomach [Figure 1]. In view of fresh blood in nasopharynx, CT angiography of neck and paranasal air sinuses was performed. The sphenoid sinus and posterior ethmoidal air cells showed support for further management. Upper gastrointestinal (UGI) endoscopy was done in view of hematemesis, which was normal. Patient was evaluated further with computed tomography (CT) abdominal angiography, which revealed no evidence of any mucosal or submucosal pathology in relation to the UGI tract. Patient developed two more episodes of hematemesis one on third and the other on seventh day of hospital admission. Repeat UGI endoscopy revealed fresh blood trickling from nasopharynx and oropharynx and clotted blood in esophagus and stomach [Figure 1]. In view of fresh blood in nasopharynx, CT angiography of neck and paranasal air sinuses was performed. The sphenoid sinus and posterior ethmoidal air cells showed support for further management. Upper gastrointestinal (UGI) endoscopy was done in view of hematemesis, which was normal. Patient was evaluated further with computed tomography (CT) abdominal angiography, which revealed no evidence of any mucosal or submucosal pathology in relation to the UGI tract. Patient developed two more episodes of hematemesis one on third and the other on seventh day of hospital admission. Repeat UGI endoscopy revealed fresh blood trickling from nasopharynx and oropharynx and clotted blood in esophagus and stomach [Figure 1]. In view of fresh blood in nasopharynx, CT angiography of neck and paranasal air sinuses was performed. The sphenoid sinus and posterior ethmoidal air cells showed
areas of hyperdensities indicating hemorrhage. In addition, CT angiography revealed a large saccular aneurysm within right sphenoid sinus arising from cavernous ICA [Figure 2]. The aneurysm was projecting medially into sphenoid sinus with breach in the lateral sinus wall. Digital subtraction angiography revealed a wide necked saccular aneurysm arising from medial wall of proximal cavernous ICA measuring $17 \times 15$ mm with its neck measuring 8 mm. It is projecting medially and anteriorly with teat in its medial aspect. Manual cross-compression test done during angiography showed good cross-flow across Anterior Communicating Artery (AcomA) into right middle cerebral artery (MCA) and anterior cerebral artery (ACA) branches with venous delay of less than 2 s in right cerebral circulation [Figure 3]. In view of wide neck ruptured right cavernous ICA aneurysm, patient was given definitive options for surgical external carotid artery-middle cerebral artery bypass, endovascular flow diverter placement, and parent artery occlusion. Patient has given consent for flow diverter placement with additional coiling. However, on the day of the procedure, the angiogram revealed partial thrombosis of the ICA aneurysm with extension of thrombus into ICA. Hence, a decision for parent artery occlusion was taken. An 8 Fr femoral sheath was inserted in the right femoral artery. A 6 Fr Neuron Max (Penumbra, Inc. Alameda, USA) guiding sheath was placed in right proximal cervical common carotid artery. A 5 Fr Navien guiding catheter (Covidien Rendezvous, Paris, France) was placed in right proximal cervical internal carotid artery. A balloon occlusion test was performed which demonstrated good cross-flow across AcomA into right MCA and ACA branches with venous delay of less than 2 s. An Echelon10 (ev3, Toledo Way, Irvine, USA) micro-catheter was advanced coaxially through the guiding catheter into the aneurysm and segmental trapping of the aneurysm and petrous ICA was done using Axium detachable coils (ev3, Toledo Way, Irvine, USA). Check angiogram showed complete non-opacification of the aneurysm with good cross-flow across AcomA [Figure 4]. The procedure was uneventful and the patient was discharged in a stable neurological condition. On follow-up, over a period of 6 months there had been no further episodes of hematemesis.

**Discussion**

Hematemesis is the vomiting of blood. The source is generally the UGI tract, typically above the ligament of Treitz. Swallowed blood from a nasal or pharyngeal source can be mistaken for gastrointestinal bleeding. Epistaxis was responsible for the 0.55% cases of hematemesis and malena.$^{[2]}$

Aneurysms that involve the cavernous ICA are rare, with a reported prevalence of 3–5% of all intracranial aneurysms$^{[3]}$ and

![Figure 1](image1.png)  
**Figure 1:** (a) Endoscopic image depicting fresh blood in oropharynx (long arrow). (b) Blood clot in fundus of the stomach (short arrow)

![Figure 2](image2.png)  
**Figure 2:** (a) Axial sections of CT at the level of orbits demonstrating hyper densities (long arrow) with the posterior ethmoid air cells and sphenoid sinus indicating hemosinus. (b) Coronal reconstructed images of contrast enhanced CT at the level of sphenoid sinus demonstrating an aneurysm (short arrow) arising from the right cavernous ICA projecting medially with erosion of sphenoid sinus

![Figure 3](image3.png)  
**Figure 3:** (a) Anteroposterior projection of digital subtraction angiography showing a large aneurysm arising from the cavernous ICA with a teat in its superomedial aspect (black arrow). (b) Anteroposterior projection with left ICA injection showing good cross flow across anterior communicating artery on manual compression of right cervical ICA

![Figure 4](image4.png)  
**Figure 4:** (a) Anteroposterior fluoroscopic projection showing coils (arrow) in petrous ICA and proximal cavernous ICA (b) Post-coiling anteroposterior projection of digital subtraction angiography with left ICA injection showing good cross-flow across anterior communicating artery into right middle cerebral artery and anterior cerebral artery branches with complete non-opacification of the aneurysm
approximately 15% of those arising from the internal carotid artery. Since extradural they can grow large in size, unruptured cavernous ICA aneurysms typically present with pressure symptoms due to mass effect on adjacent cranial nerves such as ophthalmoplegia, facial pain, and numbness. Ruptured cavernous ICA aneurysms often produce a spontaneous carotid-cavernous fistula. Cavernous ICA aneurysmal rupture presenting with epistaxis and subarachnoid hemorrhage is very rare. Cervical and extracranial carotid aneurysms may cause hematemesis due to their anatomical contiguity with the upper gastrointestinal tract including pharynx and esophagus. To the best of our knowledge, rupture of intracranial ICA aneurysm or cerebral aneurysm of any location, as the cause of massive hematemesis has not been reported in the English literature. Nasopharyngeal epistaxis may be caused by direct rupture of an ICA aneurysm into the sphenoid sinus as the cavernous ICA has a very close relationship with the sphenoid sinus, bulging into lateral sinus wall. In a study conducted by Renn et al. the bony covering over the cavernous ICA within the sphenoid sinus was less than 1 mm in 66% of cases, and in 4% of the cases, there was no bony covering. The thin interface of bone between these structures provides a free space where aneurysm may enlarge to a considerable size. When these aneurysms rupture, it may result in fatal posterior nasopharyngeal epistaxis. Swallowed blood from nasal or pharyngeal source can be mistaken for GI bleeding, as in the index case. The unusual clinical finding in the index case was that he never had frank epistaxis during his clinical course.

Many treatment strategies have been used in the management of cavernous ICA aneurysms. Treatment of ICA aneurysms can be either occlusive or reconstructive. Oclusive strategies include parent artery ligation surgically or by endovascular techniques. Occlusion of the ICA would lead to cerebrovascular events, unless a balloon occlusion test or manual cross-compression test is performed in advance to demonstrate cross-flow via circle of willis. If a temporary occlusion test is successful, parent artery occlusion is an option. Reconstructive strategies include flow diverter placement with or without additional coil embolization. In our case parent artery occlusion was done using endovascular approach using coils, as there was good cross-flow across AcomA which was demonstrated on balloon occlusion test. Flow diverter placement was not considered in our case as the aneurysm showed interval change in morphology and was partially thrombosed with extension of thrombus into cavernous ICA.

Ruptured intracranial ICA aneurysms can be considered as one of the rare differential diagnosis of hematemesis especially if the bleeding source cannot be identified in the upper gastrointestinal tract. Nasopharyngeal epistaxis arising from the cavernous ICA aneurysm is secondary to erosion into sphenoid sinus. Nasopharyngeal bleed if swallowed can masquerade as hematemesis. Endovascular techniques may provide safe and effective outcome for the management of ICA aneurysm.

Declaration of patient consent
The authors certify that appropriate patient consent was obtained.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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