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CASE REPORT

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Massive haematemesis complicated with an acute ischaemic stroke due to internal carotid artery pseudoaneurysm

Fernando MSPK[1]*, Selvarathnam G[2], Pradeepan J[2], Sujanitha V[2], Rajendran N[1]

Abstract

Haematemesis, commonly associated with upper gastrointestinal disorders, can rarely result from cranial vascular anomalies like internal carotid artery aneurysms (ICAAs). ICAA rupture may cause fatal nasopharyngeal epistaxis, with swallowed blood presenting as hematemesis, posing a diagnostic challenge. We report a 75-year-old woman with life-threatening haematemesis, epistaxis, and right hemiplegia. Despite negative findings from otolaryngological and gastrointestinal evaluations, CT angiography identified a left internal carotid pseudoaneurysm. This case emphasizes the importance of considering cranial vascular anomalies in patients with unexplained epistaxis and haematemesis. Endovascular stent embolisation is recommended for managing ICAAs while preserving arterial integrity.

Keywords: massive haematemesis, ischaemic stroke, internal carotid artery pseudoaneurisym

Introduction

Haematemesis is typically a manifestation of upper gastrointestinal tract bleeding.(1) Cranial vascular anomalies such as internal carotid artery aneurysm, can rarely present as epistaxis and haematemesis, due to their anatomical contiguity with the pharynx and esophagus.(2) Epistaxis is associated with 0.55% of cases, who presents with haematemesis. We present a case of a 75-year-old woman who experienced life-threatening massive haematemesis and epistaxis secondary to an extracranial internal carotid artery pseudoaneurysm rupture, leading to haemodynamic instability causing cerebral watershed infarctions.

Case presentation

A 75-year-old woman presented with reduced level of consciousness following massive haematemesis. She had experienced epistaxis for three days prior to this presentation. She is a known patient with type 2 diabetes mellitus and hypertension, both reasonably controlled. She denied any upper respiratory tract infections, head trauma or ENT surgeries prior to this event. She was drowsy with a Glasgow Coma Scale (GCS) score of 12/15, severely pale, and haemodynamically unstable upon presentation (BP 70/40 mmHg, HR 120 bpm with low volume). She required urgent resuscitation with fluids, multiple blood transfusions, and subsequent inotrope support. Neurological examination revealed right-sided hemiplegia with reduced tone, reflexes, and grade 3/5 motor weakness. Rest of the systemic examination was unremarkable.

Haematological investigation revealed a low hemoglobin level of 7.4 g/dL. Rest of the laboratory investigations were unremarkable as shown in table 1.

*Correspondence:

MSPK Fernando
Teaching hospital Jaffna, Sri Lanka
E-mail: sandunpulasthi@yahoo.com
Phone: +94777938730

Full list of author information is available at the end of the article

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Table 1 - Summary of laboratory investigations

Blood investigation	Result
Full blood count	WBC-16 x 10^9 /L Hb- 7.4 g/dL PLT- 314x 10^9 /L
Liver functions	AST- 45 U/L (15-43) ALT 56 U/L (11-63)
Renal functions	Serum Creatinine - 86 micomol/L (65-90)
Serum electrolytes	Na-136 mmol/L (136-145) K- 3.8mmol/L (3.5-5.1)
APTT	34 s (25-35 s)
PT/INR	PT- 12s (11-13 s) INR- 1.08
Upper GI endoscopy	Reflux esophagitis, no active bleeding or altered blood
NCCT brain	Left MCA-PCA and MCA-ACA watershed infarctions
CT angiography	PA of C3 segment of left ICA at foramen lacerum associated with bone remodeling. Small aneurysm at the junction of petrous and cervical segment of left ICA

WBC-white blood cell, PLT-platelets, HB-hemoglobin, ALT- alanine aminotransferase, AST- aspartate aminotransferase, PT- prothrombin time, INR- international normalized ratio, MCA- middle cerebral artery, PCA- posterior cerebral artery, ACA-anterior cerebral artery, PA- pseudoaneurysm, ICA- internal carotid artery

Upper gastrointestinal endoscopy (UGIE) and ultrasound abdomen were normal. Otolaryngology examination for the evaluation of epistaxis did not reveal any source of bleeding. Non-contrast computed tomography (NCCT) of the brain (figure-1) revealed watershed cerebral infarctions involving left anterior, middle and posterior cerebral artery territories. Massive haematemesis resulting in haemodynamic compromise and significant haemoglobin drop despite normal otolaryngeal and endoscopic examination, led us to consider the rare possibility of bleeding from cranial vascular anomalies. CT angiography of cranial vessels (figure-2) confirmed the presence of a pseudoaneurysm of the left internal carotid artery in the C3 (lacerum) segment and a small aneurysm in C2 (petrous) segment of ICA. Digital subtraction angiography (DSA) and endovascular coil embolization of the pseudoaneurysm were recommended but were not performed due to patient's and family's concerns.

She was discharged with a follow up plan to initiate antiplatelets for the management of the cerebral infarctions.Antiplatelet therapy was not started during this presentation due to the life-threatening massive bleeding. The patient and the family members were informed about the possibility of life-threatening re-bleeding and the prognosis if it occurs. They understood and accepted the clinical circumstance and opted for conservative management.

Discussion

Although haematemesis is typically a manifestation of upper GI disorders, it can also be a rare presentation of cranial vascular anomalies, such as ICAAs. The rupture of these aneurysms may cause bleeding into sphenoid sinus, resulting in fatal posterior nasopharyngeal epistaxis.(4) Blood from nasopharyngeal sources can be swallowed and manifest as haematemesis or malena. This clinical scenario is extremely rare and has been scarcely reported in the literature.(5)

ICAAs are a rare presentation commonly seen in females and the incidence would increase with advanced age.(6) They are mostly considered asymptomatic. However, some may develop compressive symptoms due to the increasing size of the aneurysm, which can compress adjacent cranial nerves. Others may cause thromboembolism or massive bleeding due to the rupture of these aneurysms.(7) These aneurysms can either be true aneurysms, involving all three layers of arterial wall, causing dilatation of intact arterial wall, or pseudoaneurysms, which have only a fibrous cap or a blood clot. True ICAAs occur at the carotid bifurcation or proximal ICA, while pseudoaneurysms can be seen near suture lines or traumatic areas. The skull base areas most commonly affected by ICAAs' are the ethmoid, sphenoid and frontal bones.(8)

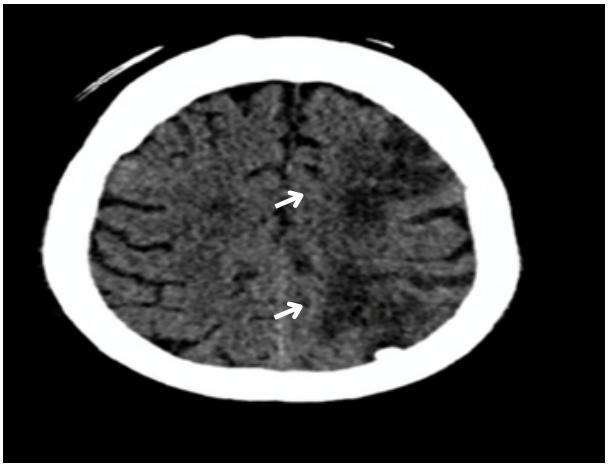

Figure 1 - NCCT brain showing left MCA-PCA and MCA-ACA watershed infarctions

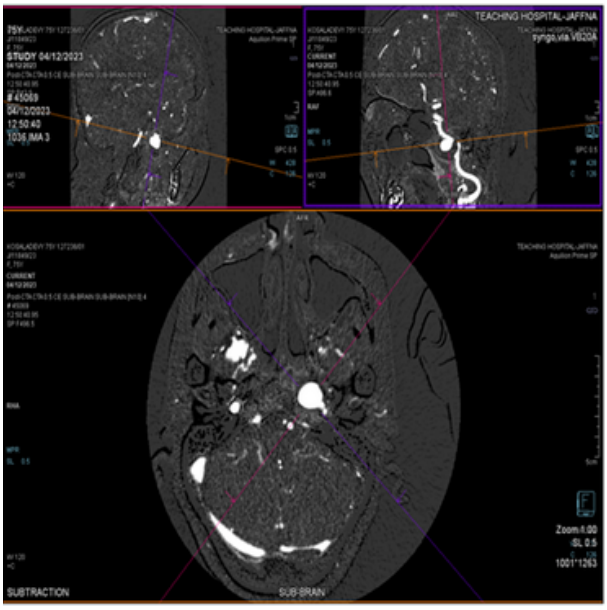

Figure 2 - CT angiogram showing left ICA pseudoaneurysm in C3 segment

The aetiologies of ICAA include head trauma, cranial surgeries, congenital factors, radiation therapy, and infections such as tuberculosis, chronic otitis media, skull base osteomyelitis, or invasive fungal sinusitis. (9) Non-traumatic aneurysms are extremely rare, and present a diagnostic challenge, especially in cases presenting with massive epistaxis or haematemesis as in our case.(10)

Bleeding into sphenoid sinus causing epistaxis following the rupture of an ICAA can be recurrent, with the severity of bleeding increasing with each occurrence, leading to a mortality rate of 30-50 % of cases. It typically takes about 3 days to 6 months from the formation of an ICAA until clinical symptoms manifest. However, the majority (50-80%) of patients will exhibit clinical symptoms within 3 weeks.(11) These minor symptoms at the initial presentation can cause a delay in seeking medical treatment, contributing to a higher mortality among patients with ICAAs. Lehmann et al.(12) has reviewed 36 cases of ICAA from 1950 to 2006 and reported a mortality rate of 22 %. When untreated, the mortality rate significantly rose to 71.4%. Unilateral blindness, orbital fracture and massive epistaxis form a clinical triad that is pathognomonic for ICAA. Digital Subtraction Angiography is considered the gold standard diagnostic test to confirm ICAA.(13)

Management of ICAA or pseudoaneurysms can be either surgical or endovascular. Endovascular therapy with detachable balloon or coil embolization is currently the preferred approach, as surgical ligation and clipping can be relatively difficult due to anatomical constraints and the fragility of the lesion. (14) Occlusion of the parent vessel, whether surgically or endovascularly, could result in severe cerebrovascular events such as strokes if there is an absence of collateral circulation in the circle of Willis. Therefore, it is essential to confirm the adequacy of collateral circulation by performing an occlusion test with a non-detachable balloon. If the occlusion test is successful, the parent vessel can be occluded; if the test is unsuccessful, a vascular bypass may be considered to address this issue. Nevertheless, 5-22% of cases with a successful occlusion test still develop major neurological deficits, such as strokes.(15) Endovascular stent-assisted embolization is considered the preferred approach, as it allows for proximal and distal occlusion of the pseudoaneurysm, leading to the complete exclusion of the lesion from the arterial circulation while preserving the patency of the ICA. A post-embolization DSA would be useful to demonstrate the occlusion of the pseudoaneurysm.(13)

Our patient's presentation can be explained by possible cerebral hypoperfusion secondary to massive bleeding from the left ICA pseudoaneurysm. As the otolaryngeal and upper gastro endoscopic examination did not locate the source of bleeding, the co-existing epistaxis and haematemesis suggested the possibility of vascular bleeding from an adjacent vascular focus. Therefore, cranial vessel CT angiography was performed, which confirmed the rare presentation of the left ICA pseudoaneurysm involving the C3 (lacerum) and a small aneurysm in

the C2 (petrous) segment of the ICA. We believe that the rupture of ICA pseudoaneurysm caused the posterior nasopharyngeal epistaxis, and the collected blood in the posterior pharynx manifested as massive haematemesis, causing haemodynamic instability and watershed cerebral infarctions.

She was discharged with a follow up plan to initiate antiplatelets for the management of the cerebral infarctions. Antiplatelet therapy was not started during this presentation due to the life-threatening massive bleeding. The patient and the family members were informed about the possibility of life-threatening re-bleeding and the prognosis if it occurs. They understood and accepted the clinical circumstance and opted for conservative management.

Conclusion

An ICA pseudoaneurysm can rarely cause life-threatening epistaxis or haematemesis due to rupture into the sphenoid sinus and posterior pharynx. Accurate clinical judgment and proper investigations are vital for diagnosing the source of bleeding and providing prompt treatment to reduce mortality and morbidity.

Declarations

Conflicts of interest
The authors declare that they have no conflicts of interest

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Author details
[1]Teaching Hospital Jaffna, Sri Lanka
[2]Faculty of Medicine, University of Jaffna, Sri Lanka

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