DOI: https://doi.org/10.4038/ajim.v4i1.235

CASE REPORT

Asian J Intern Med. 2025 Jan-Feb; 4(1)

Ischaemic stroke and intracranial haemorrhage from an internal carotid artery aneurysm: a management dilemma

Fernando MSPK[1]*, Selvarathnam G[2], Pradeepan J[2], Sujanitha V[2], Rajendran N[3]

Abstract

Internal carotid artery aneurysms (ICAAs) are rare and predominantly affect older females. Though often asymptomatic, larger ICAAs may cause compressive symptoms, thromboembolism, ischaemic stroke, transient ischaemic attacks (TIA), or massive bleeding upon rupture. The prevalence of ischaemic stroke or TIA in ICAA patients ranges from 3-6.3%. Contributing factors include congenital conditions, trauma, cranial surgeries, radiation, and infections. We report a 42-year-old woman with an acute frontal lobe infarction due to an ICAA, which subsequently led to fatal intracranial haemorrhage. Early diagnosis and timely intervention are critical to reduce the mortality and morbidity associated with ICAAs and their complications.

Keywords: internal carotid artery aneurysms, ischaemic stroke, intracranial haemorrhage

Introduction

Internal carotid artery aneurysms (ICAA) are a rare occurrence more commonly seen in females and has an increased risk with advancing age.(1) Unruptured intracranial aneurysms (UIA) are reported in 1.5% -1.8% of the general population.(2) While Most patients with ICAAs' are asymptomatic, some may exhibit compressive symptoms due the large aneurysms with compression of the adjacent cranial nerves. Others may cause thromboembolism leading to ischaemic stroke or transient ischaemic attacks (TIA) or massive bleeding due to the rupture of ICAA. (3) The prevalence of ischaemic stroke or TIA is 3-6.3% in patients with ICAA .(4)

The main aetiologies of ICAAs' are congenital, head trauma, cranial surgeries, radiation and infections such as tuberculosis, chronic otitis media, invasive fungal sinusitis or osteomyelitis of the skull base.(5) Non-traumatic ICAAs' are extremely rare. Digital subtraction angiography is considered as the gold standard diagnostic tool.(6)

We report a case of a 42-year-old woman with an acute frontal lobe infarction in the presence of ICAA which was later complicated with a fatal intracranial haemorrhage.

Case presentation

A 42-year-old woman presented with an acute onset headache for one day. She denied having fever, limb weakness or slurred speech. She did not have recent head trauma, but attributed it to mechanical type pain. . She was previously healthy and did not have a family history of vascular anomalies or coagulopathies. She was a non-smoker and did not consume alcohol or illicit drugs.

Upon admission she was afebrile and alert, her blood pressure was 130/80 mmHg and pulse rate was 84 bpm. Abdominal, cardiovascular and respiratory examinations were unremarkable. Her neurological examination was normal with preserved tone, reflexes, power in bilateral upper and lower limbs

*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

The Official Journal of
Sri Lanka College of Internal Medicine

and negative Babinski reflex. There were no signs of meningeal irritation, and her Glasgow coma scale was 15/15.

Her haematological, biochemical investigations and coagulation profile were normal. Two-dimensional echocardiogram and electrocardiogram were also normal. Summary of investigations are shown in table1.

Despite adequate analgesics, her headache gradually worsened. The non-contrast computed tomography (NCCT) brain was done, which revealed the presence of an acute infarction involving the left frontal lobe. She was started on aspirin 75 mg and atorvastatin 40 mg. Magnetic resonance imaging of the brain and cranial angiography were arranged to rule out underlying vascular anomalies. It revealed the acute frontal lobe infarction (figure-1) and left ICAA at the level of siphon (figure-2). It was later confirmed by DSA as a left ICA saccular aneurysm at the communicating segment. After excluding possible risk factors, it was decided that the ICAA was responsible for the ischaemic stroke in the index case. After withholding antiplatelets she was transferred for endovascular coil embolization which was scheduled to be done in two weeks due to limited resources. While waiting for endovascular intervention she was admitted to the emergency department one week after the discharge with a history of fall and reduced level of consciousness. An urgent NCCT-brain revealed the presence of an intracranial haemorrhage (ICH) (figure 3) and subarachnoid hemorrhage (SAH)(Figure-4) with a GCS of 7/15. She was intubated and transferred for neurosurgical intensive care where she succumbed to the disease one week later.

Discussion

Formation of thrombosis within an aneurysm was detected in 76% of aneurysms of diameters over 25 mm and 48% in the 20-25mm range.(8) The main risk factors are the ratio between aneurysmal volume and the neck size, age of the aneurysm and intrasaccular changes such as reduced blood flow, increased viscosity and turbulent blood flow which lead to endothelial damage causing platelet aggregation and thrombi formation. This would result in distal embolization which could clinically manifest as an ischaemic stroke or TIA.(9)

Long term follow-up of the patients with ischemic stroke due to ICAAs revealed that the recurrent rate was low, and symptoms are transient, carrying a good prognosis. Surgical intervention in patients with ICAA revealed a lower recurrence rate but failed to

Table 1 - Summary of laboratory and other investigation

Investigation	Result
Full blood count	WBC-8x 10^9/L, Hb- 11.2 g/dL, PLT- 345x 10^9/L
Liver functions	AST- 23 U/L (15-43) ALT 42 U/L (11-63)
Renal functions	S. Cr - 76 micomol/L (65-90)
Serum electrolytes	Na-137 mmol/L (136-145) K- 4.2 mmol/L (3.5-5.1)
APTT	34 s (25-35 s)
PT/INR	PT- 11.4s (11-13 s) INR- 0.95
Anti cardiolipin antibody	Negative
Beta 2 glycoprotein antibody	Negative
Urine, serum homocysteine	Normal
NCCT brain	Left frontal lobe infarction
MR-angiography	Left sided ICA aneurysm at the siphon
DSA	Saccular aneurysm of Left ICA

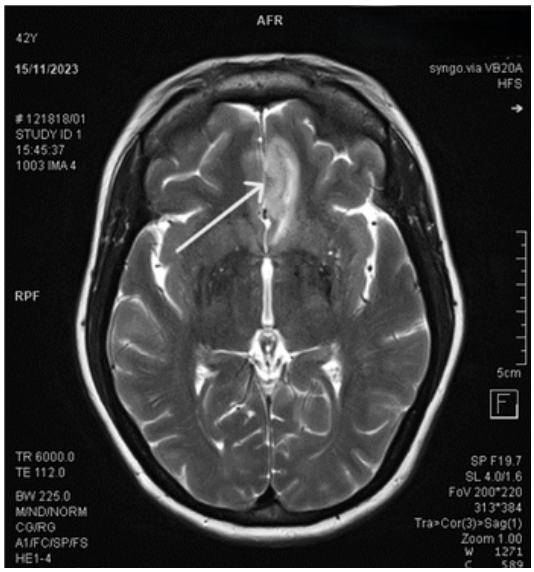

Figure 1 - MRI brain (arrow) showing left-sided gyri recti infarction

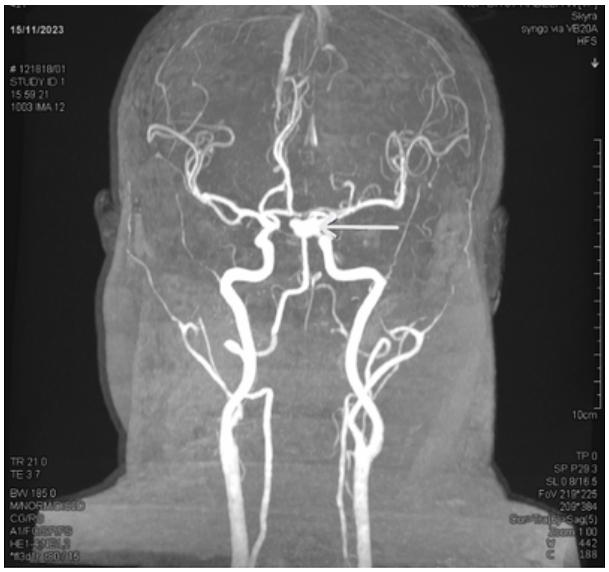

Figure 2 - MRA showing Left internal carotid artery aneurysm at the siphon

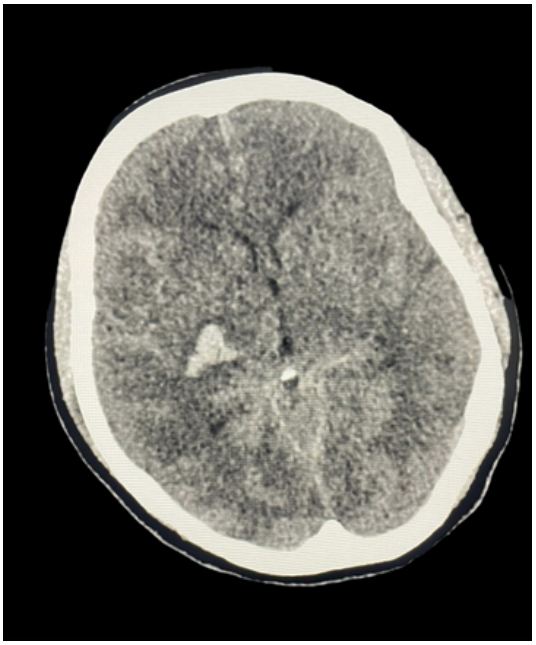

Figure 3 - NCCT brain revealing intracranial hemorrhage on the right side

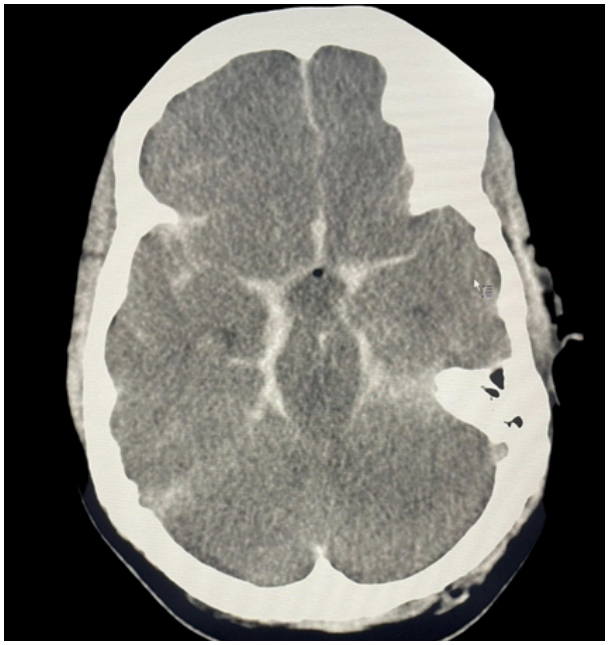

Figure 4 - NCCT brain showing diffuse SAH

demonstrate superiority over conservative management due to the intra and post operative complications.(10) Antiplatelets such as aspirin inhibit platelet aggregation within the aneurysmal sac, reducing the risk of thrombosis. The risk of SAH due to aspirin is not clearly identified yet.(11) Antiplatelets are required to be withdrawn 1 week prior to surgical intervention and can be restarted 2 days after the surgery. Low molecular dextran and nimodipine could also be used to improve microcirculation.(12)

The large ICAAs carry a higher risk of rupture. The annual rupture risk is 100% for diameters of >40 mm, 34.3% for 25-29 mm, 30% for 17 mm and 0.3% 3mm. Past history of SAH, uncontrolled hypertension and their location in the anterior communicating artery are independent risk factors of rupture which warrant follow-up imaging at appropriate intervals and early surgical intervention.(13)

The management of ICAAs' can be occlusive or reconstructive.(14) Occlusion of the parent artery can be achieved by surgical ligation or endovascularly by balloon catheters or coil embolization. Due to the anatomical inaccessibility and fragility of aneurysms, endovascular approach is preferable currently over surgical ligation or clipping. Occlusion of the parent vessel can lead to adverse effects such as ischaemic strokes if there is not enough collateral blood flow in the circle of Willis. Therefore, a temporary balloon occlusive test is performed to demonstrate the collateral flow. If the test is positive, it is safe to proceed with the occlusive surgery either endovascularly or surgically.(15) If the test is negative reconstructive surgery could be performed by placing a flow diverter with or without embolization.(16) Despite a positive occlusive test, 5-22% of patients may develop adverse events such as ischaemic strokes.(17)

In literature, there has been a young man with an ICAA who presented with an embolic stroke following a long-distance race. It was considered that heavy physical exertion, Valsalva maneuvers and acute dehydration could precipitate the formation of thrombosis within the saccular aneurysms.(18) The index case also presented with a thromboembolism following physical exertion, making primary physicians consider the possibility of vascular anomalies if patients present with ischaemic strokes in such circumstances.

The index case presented with acute severe headache following a physical exertion, in the absence of any other neurological deficit. As she did not respond to conventional analgesics and experienced worsening of symptoms. NCCT brain followed by MRI and MRA revealed the presence of acute ischemic stroke and left sided ICAA at the siphon, which was later confirmed by DSA. While waiting for the endovascular therapy she was admitted to the emergency department with an ICH and succumbed to the disease while receiving ICU care.

Conclusion

Prompt diagnosis of the intracranial artery aneurysms in ischaemic strokes, especially without major risk factors, is crucial. Timely surgical or endovascular interventions minimize mortality and morbidity. The case highlights the importance of timely intervention and necessity to optimize management strategies for ICAAs.

Declarations

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

Funding
None

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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

References

1. Imaizumi Y, Mizutani T, Shimizu K, et al. Detection rates and sites of unruptured intracranial aneurysms according to sex and age: an analysis of MR angiography–based brain examinations of 4070 healthy Japanese adults. Journal of Neurosurgery. 2018 Apr 6;1(aop):1-6.
2. Vlak MH, Algra A, Brandenburg R, et al. Prevalence of unruptured intracranial aneurysms, with emphasis on sex, age, comorbidity, country, and time period: a systematic review and meta-analysis. The Lancet Neurology. 2011 Jul 1;10(7):626-36.
3. El-Sabrout R, Cooley DA. Extracranial carotid artery aneurysms: Texas Heart Institute experience. Journal of vascular surgery. 2000 Apr 1;31(4):702-12.

4. Qureshi AI, Mohammad Y, Yahia AM, et al. Ischemic events associated with unruptured intracranial aneurysms: multicenter clinical study and review of the literature. Neurosurgery. 2000 Feb 1;46(2):282
5. Azar MM, Assi R, Patel N, et al. Fungal mycotic aneurysm of the internal carotid artery associated with sphenoid sinusitis in an immunocompromised patient: a case report and review of the literature. Mycopathologia. 2016 Jun;181:425-33.
6. Wei Zhang C, Dong Xie X, You C, et al. Endovascular treatment of traumatic pseudoaneurysm presenting as intractable epistaxis. Korean J Radiol. 2010 Nov;11(6):603-11.
7. Shono Y, Sugimori H, Matsuo R, et al. Safety of antithrombotic therapy for patients with acute ischemic stroke harboring unruptured intracranial aneurysm. International Journal of Stroke. 2018 Oct;13(7):734-42.
8. Rosta L, Battaglia R, Pasqualin A, et al. Italian cooperative study on giant intracranial aneurysms: 2. Radiological data. InProceedings of the 8 th European Congress of Neurosurgery Barcelona, September 6–11, 1987: Intraoperative and Posttraumatic Monitoring and Brain Protection—Cerebro-vascular Lesions—Intracranial Tumours—Benign Intracranial Cystic Lesions, Hydrocephalus, CSF-Volumes—Central Pain Syndromes 1988 (pp. 53-59). Springer Vienna.
9. Black SP, German WJ. Observations on the relationship between the volume and the size of the orifice of experimental aneurysms. Journal of neurosurgery. 1960 Nov 1;17(6):984-90.
10. Qureshi AI, Mohammad Y, Yahia AM, et al. Ischemic events associated with unruptured intracranial aneurysms: multicenter clinical study and review of the literature. Neurosurgery. 2000 Feb 1;46(2):282.
11. Mokin M, Darkhabani Z, Binning MJ, et al. Small unruptured partially thrombosed aneurysms and stroke: report of three cases and review of the literature. Journal of NeuroInterventional Surgery. 2012 May 1;4(3):e6-
12. Diener HC. Stroke prevention: antiplatelet and antithrombolytic therapy. Neurologic clinics. 2000 May 1;18(2):343-55.
13. Ikawa F, Morita A, Tominari S, et al. Rupture risk of small unruptured cerebral aneurysms. Journal of neurosurgery. 2019 Jan 25;132(1):69-78.
14. Van Rooij WJ. Endovascular treatment of cavernous sinus aneurysms. American journal of neuroradiology. 2012 Feb 1;33(2):323-6.
15. Bavinzski G, Killer M, Knosp E, et al. False aneurysms of the intracavernous carotid artery—report of 7 cases. Acta neurochirurgica. 1997 Jan;139:37-43.
16. Zanaty M, Chalouhi N, Starke RM, et al. Flow diversion versus conventional treatment for carotid cavernous aneurysms. Stroke. 2014 Sep;45(9):2656-61.
17. Whisenant JT, Kadkhodayan Y, Cross DT, et al. Incidence and mechanisms of stroke after permanent carotid artery occlusion following temporary occlusion testing. Journal of neurointerventional surgery. 2015 Jun 1;7(6):395-401.
18. Shimada T, Toyoda K, Hagiwara N, et al. Recurrent embolic stroke originating from an internal carotid aneurysm in a young adult. Journal of the neurological sciences. 2005 May 15;232(1-2):115-7.

Received: 15 Oct 2024 **Accepted:** 09 Feb 2025