Journal of Acute Disease

Case Report

jadweb.org

doi: 10.4103/jad.jad_140_24

Cavernous sinus syndrome due to cavernous internal carotid artery dissection: A case report

Felicity Mishan Ng Yiwey[1], Lim Thiam Hou[1]✉, Lyon Huong Ian Bing[2], Edwin Pheng Chin Meng[1]

[1]*Department of Ophthalmology, Hospital Sibu, Sarawak, Malaysia*

[2]*Department of Radiology, Hospital Sibu, Sarawak, Malaysia*

ABSTRACT

Rationale: Cavernous internal carotid artery dissection (ICAD) is a rare cause of cavernous sinus syndrome, presenting diagnostic challenges, particularly in resource-limited settings.

Patient's concern: A 43-year-old man presented with sudden, painless left eye blindness, partial ptosis and reduced facial sensation. Examination revealed a relative afferent pupillary defect, anisocoria, mild ophthalmoplegia and a pale optic disc. Initial computed tomography imaging was unremarkable, and lumbar puncture suggested meningoencephalitis. Despite treatment, his condition deteriorated to complete ptosis and total ophthalmoplegia. Urgent magnetic resonance imaging and computed tomography angiography confirmed cavernous internal carotid artery dissection and multiple areas of infarction.

Diagnosis: Cavernous sinus syndrome secondary to cavernous ICAD with non-arteritic ischemic optic neuropathy, total ophthalmoplegia and trigeminal nerve palsy.

Interventions: The patient initially received treatment for meningoencephalitis. Upon confirming the diagnosis of ICAD, he declined surgical intervention.

Outcomes: Irreversible left eye blindness.

Lessons: This case highlights the progressive nature of cavernous ICAD, the critical role of advanced imaging in diagnosis, and the need for clinical vigilance in resource-limited settings for timely diagnosis and intervention.

KEYWORDS: Internal carotid artery dissection; Non-arteritic ischemic optic neuropathy; Ophthalmoplegia; Trigeminal nerve palsy

1. Introduction

Classic presentations of internal carotid artery dissection (ICAD) include symptoms such as ipsilateral headache, neck pain, facial or eye pain. Horner's syndrome and stroke-like symptoms are also common[1].

In this case report, we highlight the unique manifestations of cavernous ICAD leading to cavernous sinus syndrome (CSS), characterized by the combination of non-arteritic ischemic optic neuropathy, progressive total ophthalmoplegia, and trigeminal nerve palsy. Recognizing these atypical signs is crucial for timely diagnosis and effective management. We also discuss the imaging modalities best suited for identifying this complex condition.

2. Case report

A 43-year-old man with underlying poorly controlled diabetes

✉To whom correspondence may be addressed. E-mail: houlth@gmail.com

How to cite this article: Yiwey, FMN, Hou LT, Bing LHI, Meng EPC. Cavernous sinus syndrome due to cavernous internal carotid artery dissection: A case report. J Acute Dis 2025; 14; 13.

Article history: Received 29 December 2024; Revision 1 January 2025; Accepted 22 May 2025; Available online 16 July 2025

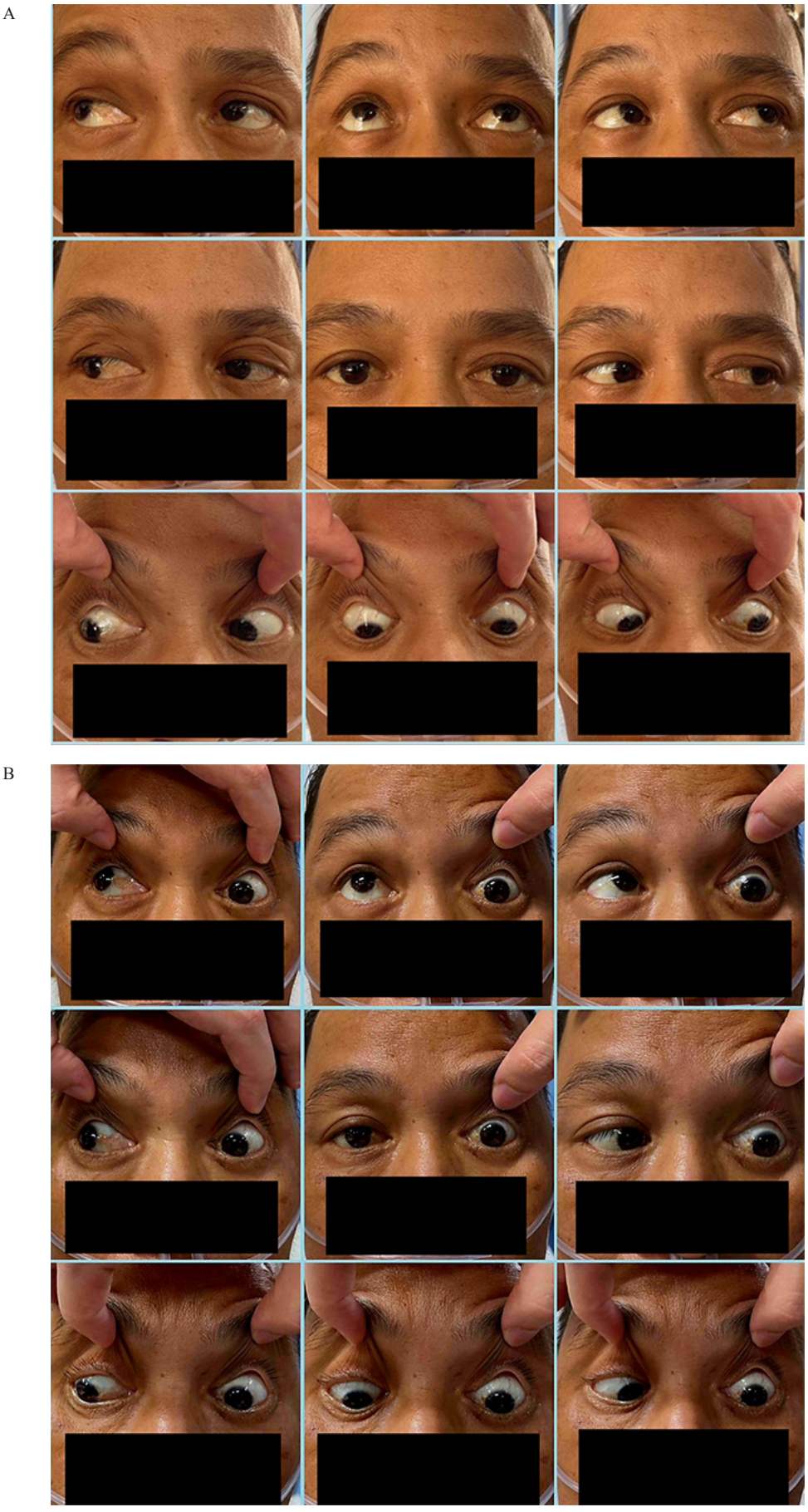

Figure 1. Cavernous sinus syndrome in a 43-year-old man presented with sudden, painless left eye blindness, partial ptosis and reduced facial sensation A) LE 9- gaze images taken on presentation demonstrating left eye mildly restricted extraocular movements. B) LE 9- gaze images taken only 3 days later to show complete left eye ophthalmoplegia.

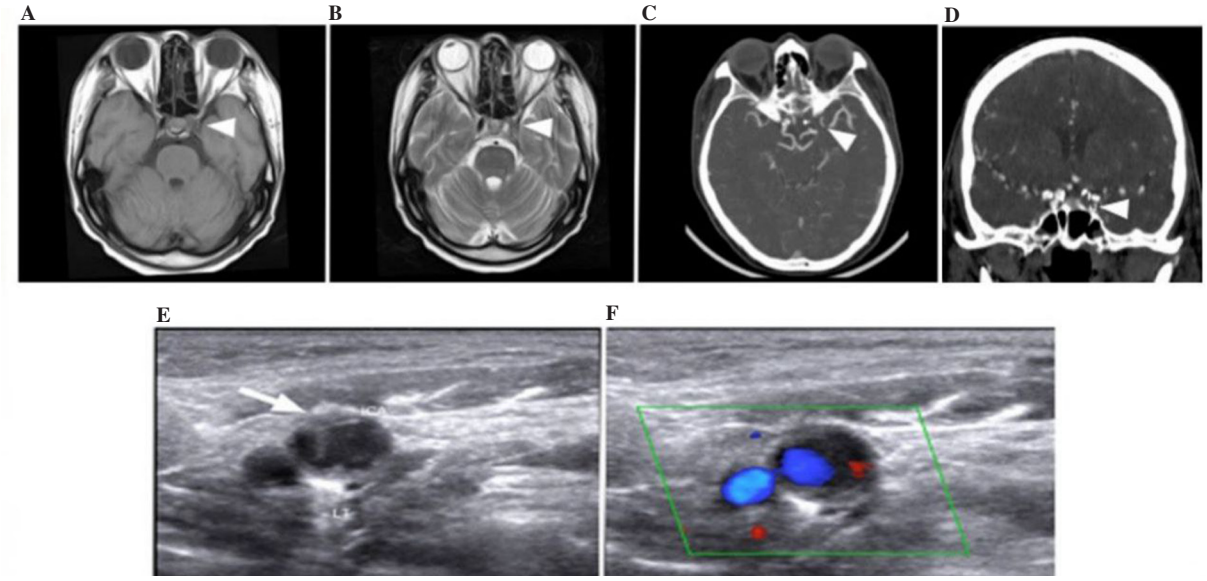

Figure 2. Selected MRI brain images A (T1W) & B (T2W): white arrowhead shows loss of flow void signal at cavernous segment of left ICA. Selected CT angiogram images C&D: white arrowhead shows non-opacification of cavernous segment of left ICA. Complementary US carotid Doppler E&F: white arrow shows intimal flap in left ICA with partial loss of doppler signal within indicating dissection of vertical segment of left ICA. MRI: magnetic resonance imaging. ICA: internal carotid artery. CT: computed tomography.

mellitus, hypertension and dyslipidemia- who is also a frequent defaulter to follow-up presented with sudden onset of left eye (LE) blindness which was preceded by partial ptosis. He had been admitted the night before for central chest pain, shortness of breath, and diarrhea, and was being treated for acute coronary syndrome and gastrointestinal sepsis. He did not report symptoms of eye pain or redness, and there were no signs of elevated intracranial pressure. On examination, his visual acuity was no perception to light in all quadrants, and a grade 4 LE relative afferent pupillary defect with ipsilateral partial ptosis and anisocoria (3/5 mm). Extraocular movements were initially mildly restricted in all directions (-0.5 to -1), as demonstrated in Figure 1. He also exhibited reduced sensation to the left side of his face with a temporally pale optic disc of the LE. In retrospect, these findings likely represented early manifestations of cavernous sinus syndrome, although the underlying etiology remained unclear at that point. A written consent from the patient's spouse for the writing of this case report was obtained.

An initial contrast enhanced computed tomography (CT) imaging revealed no brain lesions or pathology. This was followed by a lumbar puncture which showed mildly raised cerebrospinal fluid cell counts, leading us to a preliminary diagnosis of possible meningoencephalitis. Despite treatment, his symptoms progressed rapidly leading to the development of complete ptosis and total ophthalmoplegia within a matter of days, as depicted in Figure 1B.

An urgent magnetic resonance imaging (MRI) was then performed which revealed a proximal intracranial cavernous ICAD. These findings were further corroborated by a CT angiogram of the brain and carotids, as illustrated in Figure 2.

The patient was then started on antiplatelet therapy but visual rehabilitation remains poor to date as he had refused any surgical interventions in view of his poor comorbid status.

3. Discussion

ICAD is an important cause of stroke. However, most reported cases of ICAD strokes involve the extracranial carotid or vertebral arteries. In contrast, spontaneous, non-traumatic intracranial dissection of the ICA, as depicted in this case, is relatively uncommon. Details pertaining to its profile of risk factors, clinical manifestations and imaging abnormalities have not been extensively documented in literature. According to Pessin *et al*, this condition remains a rare and often under-recognized cause of CSS[2,3].

The above case presented a significant diagnostic challenge, highlighting the complexities of CSS secondary to ICAD. The patient's poorly controlled vascular risk factors, including diabetes mellitus, hypertension and dyslipidemia, along with a history of poor compliance with medications and routine follow-ups, likely contributed to chronic vascular endothelial damage, weakening of the vascular layers, and the eventual development of an ICAD[4].

His initial symptoms localized a lesion to cranial nerves Ⅱ and Ⅲ, which raised suspicion for a compressive intracranial lesion affecting both nerves. As his CT scan returned clear, his condition deteriorated rapidly, involving cranial nerves Ⅳ and Ⅵ with complete ptosis

within days. These evolving symptoms were highly suggestive of a cavernous sinus syndrome, prompting further imaging studies. MRI, which was validated by the CT angiogram revealed a cavernous ICAD, leading to the diagnosis of CSS secondary to cavernous ICAD.

Through our literature search, we found that the prevalence of CSS arising specifically from an ICAD is exceedingly rare. In most cases, CSS is typically caused by tumours, trauma or inflammatory diseases. Vascular causes, like an ICAD are much less common in clinical practice.

The cavernous sinus houses cranial nerves Ⅲ, Ⅳ, Ⅴ1, Ⅴ2 and Ⅵ along with the ICA. In this case, the pathomechanism involved the compression and dysfunction of these structures within the cavernous sinus, caused by the expanding arterial wall of the dissected ICA[5]. This explains our patient's evolving symptoms, with the dissection worsening over time. Additionally, the non-arteritic ischemic optic neuropathy can be explained from the compromised blood supply to the optic nerve from the dissection[6].

Interestingly, the ischemic brain changes were not made apparent through the first sets of CT imaging. It was only after the deterioration of CN Ⅲ palsy with CN Ⅳ and Ⅵ involvement that an MRI brain and CT angiogram were ordered, revealing the ICA lesions along with areas of corresponding infarct.

We postulate that the ICAD happened gradually and worsened during the two examinations. This would also explain the gradual, but rapidly worsening symptoms over that period of time.

Our experience with this case and from existing research from Hakimi R and Sivakumar S suggests that the MRI is a superior modality for visualizing an ICAD compared to a CT. It has also been established as an essential screening tool for dissections[7,8]. However, in resource limited settings, a CT angiogram remains a valuable diagnostic tool due to its widespread availability in most hospital settings and its rapid acquisition especially for clinically unstable patients, as it enables faster imaging without compromising patient monitoring[7]. Although a CT angiography (CTA) is typically recommended following a positive MRI or CT angiogram, it was not performed in our case due to the patient's poor comorbid state[8].

In resource-limited settings like ours, challenges were encountered in accessing advanced imaging, highlighting the need for greater awareness of arterial dissections as a potential cause of CSS. Enhancing clinical knowledge is crucial for ensuring timely diagnosis and management of such critical conditions.

In centers without MRI access, serial CT/CTA imaging can be considered when clinical suspicion remains high but initial scans are inconclusive. Where neither CT nor MRI is available, carotid Doppler ultrasound may serve as an alternative, particularly for identifying features such as an intimal flap, severe stenosis or a double lumen sign[9]. However, the utility of carotid Doppler ultrasound is limited, as much of the data available is outdated due to the widespread availability of more advanced imaging techniques.

4. Conclusion

This case highlights the complexity of diagnosing CSS secondary to cavernous ICAD, a rare and often under-recognized condition. The patient's initial presentation and subsequent evolution of symptoms had led to a delay in recognizing the syndrome, illustrating the importance of considering vascular causes when common etiologies such as trauma and tumours are ruled out. His gradual worsening of neurological deficits correlates with the progression of the arterial dissection, emphasizing the dynamic nature of this condition.

The case also illustrates the role of advanced imaging like MRI and CTA in the early and accurate detection of ICAD MRI is the superior tool in visualizing subtle vascular abnormalities, however the convenient access to a CTA makes it an invaluable tool in acute settings[6-8].

In resource limited facilities like ours, where access to these imaging modalities may be unavailable or delayed, heightened clinical awareness is crucial. There is also a need for a multidisciplinary collaboration to address the diagnostic and therapeutic challenges associated with ICAD effectively.

Our case report also aims to contribute to the limited literature available on intracranial, cavernous ICAD, emphasizing the importance of increased awareness and early intervention to improve outcomes in patients with similar presentations.

Conflict of interest statement

The authors have no conflicts of interest.

Funding

This study received no extramural funding.

Acknowledgement

We extend our gratitude to the Director General of Health Malaysia for granting permission to publish this article.

Authors' contributions

FMY&LTH: Conception and design of study; FMY: Definition of intellectual content and literature search; LHIB: Data acquisition&data analysis and/or interpretation; FMY&EPCM: Drafting of manuscript and/or critical revision; LTH: Approval of final version of manuscript.

Publisher's note

The Publisher of the *Journal* remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Edited by Liu JJ

References

[1] Goodfriend SD, Tadi P, Koury R. Carotid artery dissection 2022. In: StatPearls. Treasure Island (FL): StatPearls Publishing. [Online] Available from: https://pubmed.ncbi.nlm.nih.gov/28613585/ [Accessed September 18, 2024].

[2] Chaves C, Estol C, Esnaola MM, Gorson K, O'Donoghue M, de Witt LD, et al. Arterial dissection is now recognized as a common cause of ischemic stroke in young and middle-aged adults. *Arch Neurol* 2002; **59**(6): 977-981.

[3] Pessin MS, Adelman LA, Barbas NR. Spontaneous intracranial carotid artery dissection. *Stroke* 1989; **20**(8): 1100-1103.

[4] Levy D, Sharma S, Grigorova Y, Farci F, Le JL. Aortic Dissection 2024. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing. [Online] Available from: https://www.ncbi.nlm.nih.gov/books/NBK441963/ [Accessed April 7, 2025].

[5] Bakan AA, Alkan A, Kurtcan S, Aralaşmak A, Tokdemir S, Mehdi E, et al. Cavernous sinus: A comprehensive review of its anatomy, pathologic conditions, and imaging features. *Clin Neuroradiol* 2014; **25**(2): 109-125.

[6] Salvetat ML, Pellegrini F, Spadea L, Salati C, Zeppieri M. Non-arteritic anterior ischemic optic neuropathy (NA-AION): A comprehensive overview. *Vision (Basel)* 2023; **7**(4): 72.

[7] Siddiqui A, Kim N, Kogan M, Natarajan S, Sawyer R, Snyder K. Third nerve palsy following carotid artery dissection and posterior cerebral artery thrombectomy: Case report and review of the literature. *Surg Neurol Int* 2014; **5**(14): 497-500.

[8] Hakimi R, Sivakumar S. Imaging of carotid dissection. *Curr Pain and Headache Rep* 2019; **23**(1): 2.

[9] Gardner DJ, Gosink BB, Kallman CE. Internal carotid artery dissections: Duplex ultrasound imaging. *J Ultrasound Med* 1991; **10**(11): 607-614.