CASE REPORT

Acute Neurological Deficits in Young Adults: A Case Report on Spontaneous Arterial Dissection

Shafiq Ur Rehman[1*], Saba Zaidi[1], Muhammad Mubashir[1] and Iman Jauhar[1]

[1]*Department of Neurology, Liaquat National Hospital and Medical College, Karachi, Pakistan*

Abstract

This case report outlines two younger patients who developed severe acute neurological symptoms and were later diagnosed with cerebrovascular events due to suspected arterial dissection. In Case 1, a 40-year-old female presented with dizziness along with severe headache and non-fluent speech. MRI revealed a left parietotemporal infarct, which explains Broca's aphasia. Her CT angiography showed critical stenosis of the left M2 segment of the MCA (middle cerebral artery) associated with an adjacent aneurysm that suggested spontaneous arterial dissection. She was treated with aspirin and atorvastatin, followed by LMWH, which was subsequently discontinued due to aneurysm risk. In Case 2, a 33-year-old male presented with vertigo, vomiting, and unrelenting headache. His examination demonstrates an elevated BP, limb ataxia, and nystagmus. His CT angiography demonstrated left vertebral artery thrombosis, which was suspicious for vertebral artery dissection. MRI confirmed a left cerebellar infarct. Workup showed diabetes, hyperlipidemia, and elevated homocysteine levels. The treatment regimen included insulin, atorvastatin, LMWH, aspirin, and physiotherapy. Because of the advancing hydrocephalus resulting from fourth ventricle compression, a ventriculoperitoneal (VP) shunt was implanted, resulting in a clinical improvement. The cases provide an essential but clinically overlooked explanation for strokes in younger adults that focuses on spontaneous arterial dissections. Early imaging, diagnosis, and intervention are crucial in these cases.

Keywords: *Vertebral artery dissection, atypical stroke etiology, ischemic stroke, middle cerebral artery, young adult, headache.*

INTRODUCTION

Intracranial artery dissection (IAD) is a rare but significant cause of stroke in young adults. This condition is often overlooked due to the inconsistent clinical manifestations. Intradural spontaneous vertebral artery dissections, for instance, account for approximately 3% to 5% of subarachnoid hemorrhages (SAHs), while dissecting aneurysms of the intracranial circulation contribute to approximately 2% to 3% of total cerebral aneurysms [1] clinical features, diagnosis, treatment, and prognosis of intracranial artery dissection (IAD). Cerebral ischemia resulting from intracranial carotid artery dissection is relatively uncommon, with this type representing only approximately 2% of all carotid dissection cases [2].

The cause of IADs is unknown in detail. A few predisposing problems have, however, been trauma, hypertension, dyslipidemia, diabetes mellitus, and connective tissue disorders such as fibromuscular dysplasia, Ehlers-Danlos syndrome, and Marfan's syndrome [3]. Anterior circulation dissections seem to have more common ischemic symptoms, whereas posterior circulation dissections usually present with SAH [1]. The diagnosis is based on neuroimaging techniques such as digital subtraction angiography, MRA, and CT angiography (CTA). They help detect a stenosis, a false lumen, an intimal flap, or an intramural thrombus [4, 5].

**Corresponding author: Shafiq Ur Rehman, Department of Neurology, Liaquat National Hospital and Medical College, Karachi, Pakistan, Email: drshaf98@gmail.com*

Received: April 13, 2025; Revised: June 23, 2025; Accepted: July 01, 2025
DOI: https://doi.org/10.37184/jlnh.2959-1805.3.45

In addition, Vertebral artery dissection (VAD) is a significant cause of ischemic stroke in the posterior circulation, especially in young and middle-aged individuals [6]. There are two distinct types of VAD: spontaneous and traumatic [7]. Depending on the location of the dissection, individuals may experience no symptoms or, more commonly, atypical symptoms such as mild dizziness or headache [8]. The same techniques mentioned above can also be used to diagnose VAD. The treatment approach for ischemic stroke, regardless of whether an IAD or VAD causes it, remains consistent. This includes thrombolysis using tPA (tissue plasminogen activator) followed by antiplatelet therapy and statins [9].

This report describes two young, healthy adults with headaches. One had a left middle cerebral artery infarction due to spontaneous intracranial artery dissection; the other had a left cerebellar infarction from vertebral artery dissection. Both cases showed unique radiographic findings, with no identifiable cause for the dissections.

Case 1

A 40-year-old female with no significant medical history presented with severe headache, speech difficulty, and dizziness for one day. Four days earlier, she had an episode of unwitnessed loss of consciousness. Vertigo worsened with head movement. She reported regular sleep and appetite, with no history of seizures, fever, incontinence, oral ulcers, rash, or miscarriages. She was not on any medications. On examination, she was alert

and followed commands. Speech was non-fluent with incomplete sentences; repetition was intact, but naming, reading, and writing were impaired. Motor examination showed normal bulk, tone, power, and reflexes. Plantar responses were flexor bilaterally. Pupillary reactions and cerebellar function were normal.

Based on the patient's acute presentation of severe headache and impairment in speech, investigations were ordered to rule out ischemic infarction. An MRI of the brain was performed, which revealed an area of restricted diffusion in the left parietotemporal region involving the Sylvian fissure, explaining Broca's aphasia (**Fig. 1**). The computed tomography angiography (CTA) imaging revealed critical stenosis of the left M2 segment of the middle cerebral artery (MCA) (**Fig. 2**). Additionally, an aneurysm was observed in the left MCA vascular territory measuring 3.0 x 4.7 mm with the neck measuring 1.9 mm (**Fig. 3**). Based on the presentation of left middle cerebral artery (MCA) stenosis and an aneurysm distal to the stenosis along with the symptom of headache, we considered a likely possibility of spontaneous arterial dissection in our case. The patient was started on a management regimen that included intravenous normal saline 0.9%, oral aspirin 75 mg daily, LMWH 60 mg twice daily, along with oral atorvastatin 20 mg daily for symptomatic relief, oral betahistine 10 mg three times a day, and paracetamol 1 g, administered six hourly. We later stopped the anticoagulation to prevent the risk of aneurysm rupture.

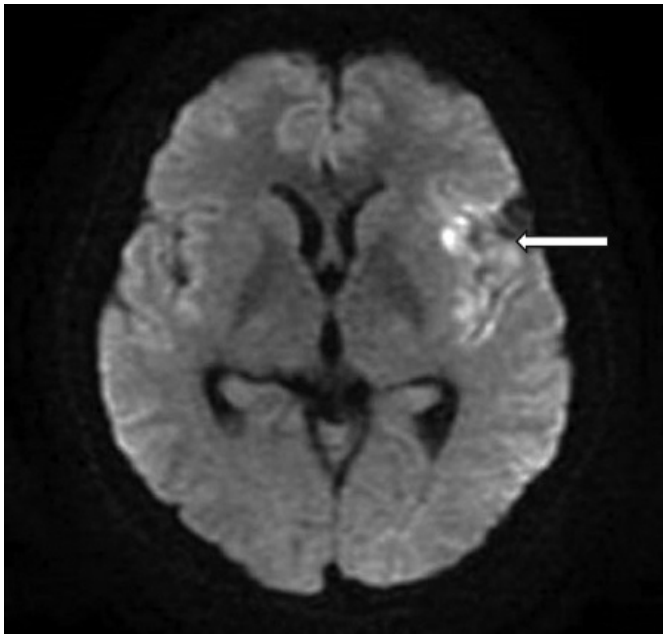

Fig. (1): DWI sequence MRI brain: Left MCA infarction.

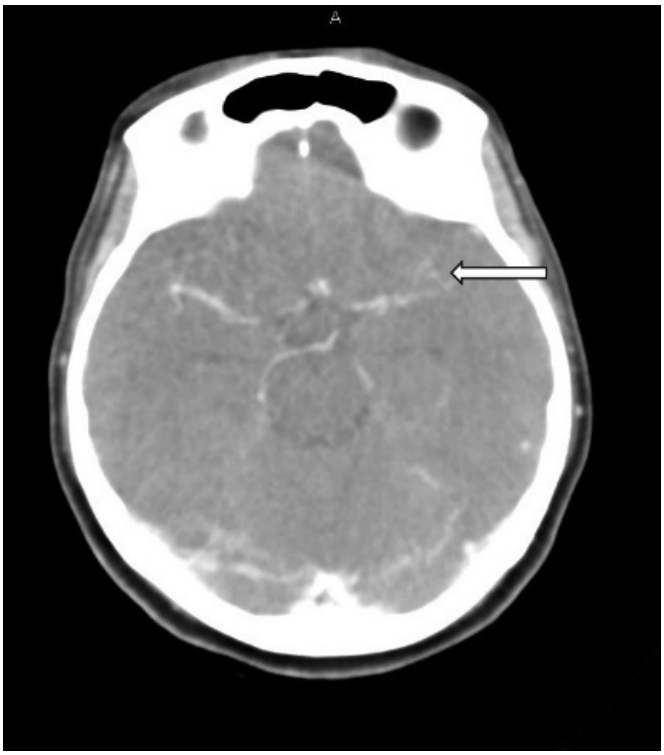

Fig. (2): CTA left MCA critically stenosed (M2 segment).

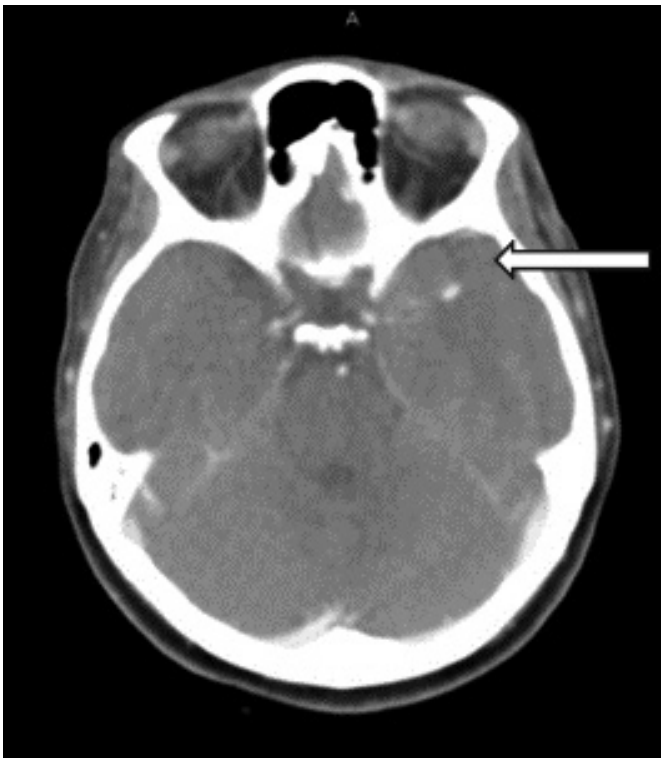

Fig. (3): Left MCA territory aneurysm.

Case 2

A 33-year-old healthy male presented with intractable headache, neck pain, vertigo, and vomiting for 2 days. Upon examination, he was irritable, with a blood pressure of 178/107 mmHg, a pulse of 92 beats/min (regular), and afebrile. Neurologically, he was awake

but lethargic, followed commands, and had left-beating nystagmus. Motor power was 5/5 in all limbs. Mild left-sided finger-nose dysmetria and heel-shin ataxia were noted. Plantar reflexes were down-going, and speech was every day. Imaging studies, including an MRI brain, confirmed a left cerebellar infarct (**Fig. 4**). A CT angiogram neck revealed a reduced caliber of the left vertebral artery from its origin to the C1 vertebrae suggesting its thrombosis (**Fig. 5**). Based on the patient's symptoms, examination findings, and imaging results, the diagnosis of left cerebellar infarction with possible thrombosis of the left vertebral artery was made. In the absence of a comorbid status and severe headache, we made a likely diagnosis of vertebral dissection in this young gentleman. His workup revealed an HbA1c of 8.31% (above 6.5% indicating diabetes), an LDL of 211 mg/dL (130-159), and a homocysteine level of 20.3 micromol/L (5-15). The patient was started on aspirin 75mg daily and therapeutic doses of LMWH 1 mg/kg twice a day to prevent infarct progression.

Additionally, he received atorvastatin 80 mg OD to manage his lipid levels and reduce the risk of future cardiovascular events. Simultaneously, regular insulin was started for blood sugar control. Physiotherapy was initiated to aid recovery, and blood pressure was closely monitored. As his headache persisted, a repeat CT head revealed hydrocephalus due to fourth ventricle compression from left cerebellar infarction. A ventriculoperitoneal (VP) shunt was placed, which improved his overall condition, and he was discharged home.

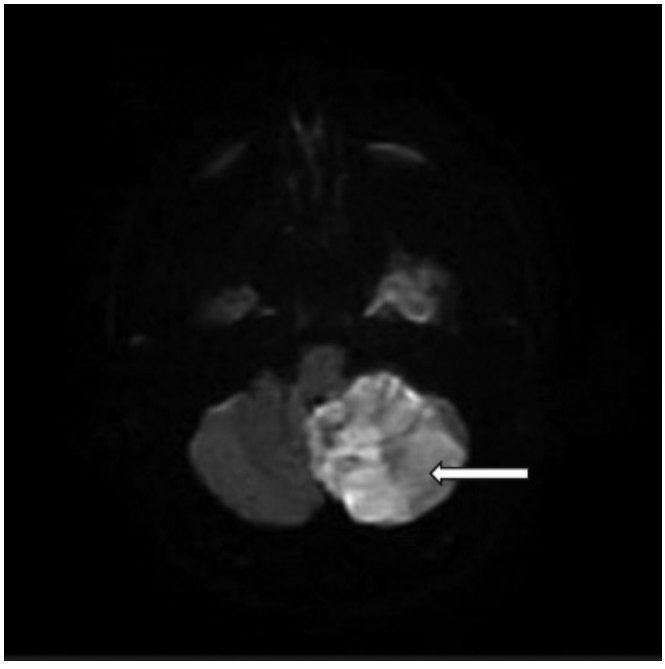

Fig. (4): DWI sequence MRI brain left cerebellar infarction.

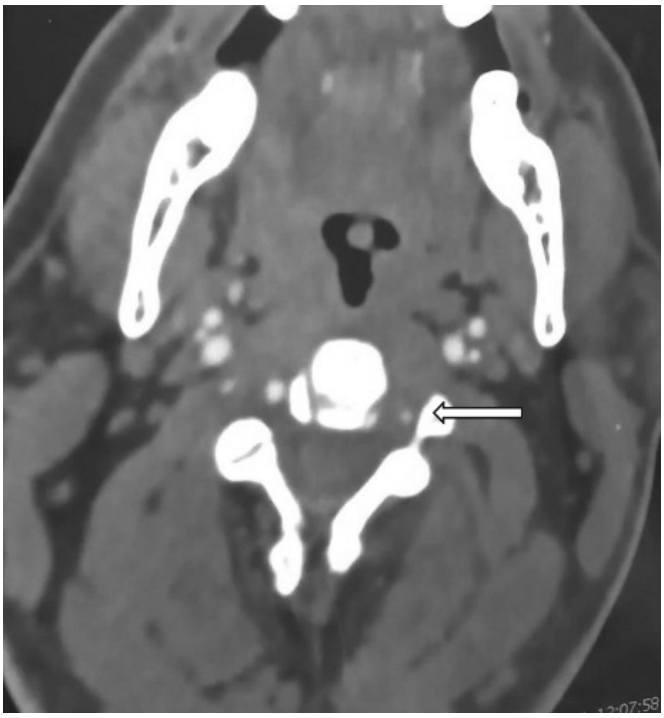

Fig. (5): Left vertebral artery reduced in caliber from its origin till the C1 vertebra.

DISCUSSION

Spontaneous arterial dissection, whether intracranial or extracranial, is a concerning cause of stroke in young individuals. In our initial case report, a woman with no prior medical conditions presented with sudden non-fluent aphasia. Extensive stroke workup ruled out other potential causes such as cardioembolic or extracranial carotid artery disease. Imaging revealed thrombosis of the left middle cerebral artery (MCA) with an accompanying aneurysm distal to the thrombosis, strongly suggesting middle cerebral artery dissection (MCAD). Isolated MCAD is a rarely reported cause of stroke [9] and remains less understood compared to dissections involving the intracranial internal carotid artery (ICA). Most cases of MCAD involve the M1 segment of the MCA due to its location adjacent to the sphenoid wing; however, in our case, the M2 segment was involved [10]. Headache is commonly reported in cases of spontaneous arterial dissection, and manifestations typically include ischemic infarction in the involved territory, primarily when associated with aneurysms, due to flow stagnation and peripheral embolization [9, 11]. Vessel wall MRI (VW-MRI) and time-of-flight MR angiography (TOF-MRA) are now commonly used non-invasive tools for diagnosing intracranial artery dissection (IAD) [12]. Digital subtraction angiography (DSA), although sensitive, is typically reserved for subarachnoid hemorrhage, unclear findings, or when planning an intervention. A confirmed diagnosis often

needs both wall and lumen imaging, along with follow-up scans. In our case, CT angiography showed segmental M2 stenosis, suggesting arterial dissection [13].

In our second case, a young man with no comorbidities or history of neck trauma or drug abuse presented with a left cerebellar infarction and severe headache. An urgent stroke workup was initiated. Initial echocardiography raised suspicion for aortic dissection, but CTA of the aorta was standard. CTA of the neck revealed critical stenosis of the left extracranial vertebral artery, suggesting cerebellar infarction secondary to spontaneous vertebral artery dissection.

Vertebral artery dissection is classified into two types based on its location: subintimal and subadventitial. In subintimal dissection, a tear in the inner arterial layer narrows the lumen, potentially causing thromboembolism and posterior circulation infarction, as seen in our case. In subadventitial dissection, a tear in the outer layer of the vessel leads to aneurysmal dilation, which can compress surrounding structures or rupture, causing subarachnoid hemorrhage [6].

In addition to trauma or hyperextension movements of the neck, the literature cites several other potential causes of vertebral artery dissection, including hypertension, migraine, fibromuscular dysplasia, homocystinuria, vasculitis, syphilis, Marfan's syndrome, and Ehlers-Danlos syndrome [3]. While detailed physical examination and laboratory workup ruled out most potential causes in both cases, neither patient had a family history of similar conditions. However, in the second case, the patient had significant hyperlipidemia and persistently elevated blood pressure during hospitalisation, which may have been previously unknown and potentially triggered the disease. Alternatively, these findings could be secondary to his severe and persistent headache.

Regarding the management of our described cases, thrombolysis was not an option due to the elapsed window period. While the safety of intravenous thrombolysis for acute ischemic stroke caused by extracranial carotid artery dissection has been investigated, its safety for intracranial dissections remains uncertain. Therefore, both patients were initiated on single antiplatelet therapy following neuroimaging.

A systematic review on the management of spontaneous arterial dissection highlighted hesitancy in using anticoagulation due to the risk of worsening intramural hematoma. However, some experts suggest that anticoagulation may slow the progression of thrombosis, potentially preventing the formation of fusiform aneurysms. Altered protein expression and activation can also contribute to arterial wall weakening, blockage, and aneurysm rupture [12]. In our management, therapeutic anticoagulation was initiated for both patients. However, it was discontinued in the first patient due to the presence of an aneurysm, and in the second patient due to the absence of radiological evidence of a free-floating thrombus.

CONCLUSION

The two cases highlight spontaneous arterial dissection as a significant but frequently unrecognized cause of ischemic stroke in young adults with acute headache and neurological deficits. Neuroimaging is essential in the early diagnosis of this condition, especially in the absence of traditional stroke risk factors or causes. Individualized treatment, including antithrombotic and hemorrhagic risk, along with aggressive management of vascular risk factors and neurosurgical treatment, can result in a good outcome for this group of patients. Increased awareness of this condition may result in a better prognosis for this group of patients.

CONSENT FOR PUBLICATION

Written informed consent was taken from both patients to publish this case.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

ACKNOWLEDGEMENTS

Declared none.

AUTHORS' CONTRIBUTION

Saba Zaidi contributed to case management, data collection, and initial drafting of the manuscript.

Muhammad Mubashir contributed to the literature review, image selection, and clinical interpretation.

Iman Jauhar contributed to the discussion, writing, and the critical review of the manuscript.

Shafiq Ur Rehman (corresponding author) conceptualised the report, coordinated the manuscript development, edited the final draft, and managed communication with the journal.

All authors have read and approved the final version of the manuscript.

GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the authors used generative AI tools (*e.g.*, ChatGPT, OpenAI) in a limited manner to obtain language suggestions and for minor proofreading in some parts of the manuscript. After

using these tools, the authors carefully reviewed and edited the content as needed and take full responsibility for the integrity, originality, and accuracy of the final published article.

REFERENCES

1. Sikkema T, Uyttenboogaart M, Eshghi O, De Keyser J, Brouns R, van Dijk JMC, *et al.* Intracranial artery dissection. Eur J Neurol 2014; 21(6): 820-6.
DOI: https://doi.org/10.1111/ene.12384 PMID: 24824740
2. Shimizu H, Ono T, Abe T, Hokari M, Egashira Y, Shimonaga K, *et al.* Current treatment results of intracranial carotid artery dissection causing cerebral ischemia: a Japanese nationwide survey. Neurol Med Chir (Tokyo) 2023; 63(2): 80-9.
DOI: https://doi.org/10.2176/jns-nmc.2022-0249 PMID: 36599430
3. Li S, Yan B, Kaye A, Mitchell P, Dowling R, Collins M, *et al.* Prognosis of intracranial dissection relates to site and presenting features. J Clin Neurosci 2011; 18(6): 789-93.
DOI: https://doi.org/10.1016/j.jocn.2010.11.006 PMID: 21507658
4. Hosoya T, Adachi M, Yamaguchi K, Haku T, Kayama T, Kato T. Clinical and neuroradiological features of intracranial vertebrobasilar artery dissection. Stroke 1999; 30(5): 1083-90.
DOI: https://doi.org/10.1161/01.str.30.5.1083 PMID: 10229748
5. Schaefer FKW, Schaefer PJ, Altjohann C, Bourne M, Decobelli F, Goyen M, *et al.* A multicenter, site-independent, blinded study to compare the diagnostic accuracy of contrast-enhanced magnetic resonance angiography using 1.0M gadobutrol (Gadovist) to intraarterial digital subtraction angiography in body arteries. Eur J Radiol 2007; 61(2): 315-23.
DOI: https://doi.org/10.1016/j.ejrad.2006.09.002 PMID: 17074459
6. Zhao J, Luo B, Yao X, Zhang X, He D, Cai L, *et al.* Cerebellar infarction caused by vertebral artery dissection: A case report. Medicine (Baltimore) 2023; 102(24): e34033.
DOI: https://doi.org/10.1097/md.0000000000034033 PMID: 37327265
7. Gottesman RF, Sharma P, Robinson KA, Arnan M, Tsui M, Ladha K, *et al.* Clinical characteristics of symptomatic vertebral artery dissection: a systematic review. Neurologist 2012; 18(5): 245-54.
DOI: https://doi.org/10.1097/nrl.0b013e31826754e1 PMID: 22931728
8. Hurford R, Sekhar A, Hughes TAT, Muir KW. Diagnosis and management of acute ischaemic stroke. Pract Neurol 2020; 20(4): 304-16.
DOI: https://doi.org/10.1136/practneurol-2020-002557 PMID: 32507747
9. Biller J, Hingtgen WL, Adams Jr HP, Smoker WR, Godersky JC, Toffol GJ. Cervicocephalic arterial dissections: A ten-year experience. Arch Neurol 1986; 43(12): 1234-8.
DOI: https://doi.org/10.1001/archneur.1986.00520120019010 PMID: 3778258
10. Lin CH, Jeng JS, Yip PK. Middle cerebral artery dissections: differences between isolated and extended dissections of internal carotid artery. J Neurol Sci 2005; 235(1-2): 37-44.
DOI: https://doi.org/10.1016/j.jns.2005.03.047 PMID: 15946687
11. Asaithambi G, Saravanapavan P, Rastogi V, Khan S, Bidari S, Khanna AY, *et al.* Isolated middle cerebral artery dissection: a systematic review. Int J Emerg Med 2014; 7(1): 44.
DOI: https://doi.org/10.1186/s12245-014-0044-1 PMID: 25593617
12. Pini R, Faggioli G, Lodato M, Campana F, Vacirca A, Gallitto E, *et al.* Medical and interventional outcome of dissection of the cervical arteries: Systematic review and meta-analysis. J Vasc Surg 2024; 80(3): 913-921.e13.
DOI: https://doi.org/10.1016/j.jvs.2024.04.036 PMID: 38636608
13. Yadav KL, Gupta A, Swarnkar CP, Mannan N. Role of vessel wall magnetic resonance imaging in the diagnosis of intracranial vasculopathies. Cureus 2024; 16(11): e73714.
DOI: https://doi.org/10.7759/cureus.73714 PMID: 39677081