e-ISSN: 2807-7970

AKSONA

Volume 4 Number 1, January 2024

Successfully Procedure Endovascular Carotid Angioplasty Stent in a Case of Neck Stab Wound with Pseudoaneurysm, Recurrent Transient Ischemic Attack, and Horner Syndrome: A Case Report

Pinto Desti Ramadhoni[1], **Hawari Martanusa**[2]

[1] Department of Neurology, Faculty of Medicine, Universitas Sriwijaya; Dr. Mohammad Hoesin Hospital, Palembang, Indonesia

[2] Department of Neurology, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia

Article info

Article History:
Received Aug 22, 2023
Revised Oct 27, 2023
Accepted Nov 3, 2023
Published Jan 31, 2024

Keywords:
Carotid stenting
Dissecting aneurysm
Pseudoaneurysm
Transient ischemic attack

ABSTRACT

Introduction: Neck stab wounds can lead to various vascular complications, including pseudoaneurysm or carotid artery dissection. Proper diagnosis and management can prevent recurrent strokes. **Case:** We reported a case of a 14-year-old boy with a previous left neck stab wound who developed recurrent transient ischemic attacks (TIAs) accompanied by Horner's syndrome and headache following a traumatic common carotid artery dissection. An angiographic examination revealed a pseudoaneurysm or dissection of the left common carotid artery. The patient was initially treated with oral anticoagulant therapy, but he continued to experience recurrent TIA. The patient was then treated endovascularly with a carotid angioplasty stent to restore the compromised carotid artery. Following the endovascular procedure, the patient experienced no complications, showed improvement in Horner syndrome, and had no headache. During the follow-up period, the patient had no more TIAs. **Conclusion:** Endovascular carotid angioplasty stents are an effective and safe way to treat pseudoaneurysm or dissection of the carotid artery, especially in cases presenting with recurrent TIAs and Horner syndrome. The observed clinical improvement following the procedure indicates significant benefits in enhancing the patient's quality of life and preventing complications from recurrent TIA episodes.

Corresponding Author
Pinto Desti Ramadhoni
Department of Neurology, Faculty of Medicine, Universitas Sriwijaya; Dr. Mohammad Hoesin Hospital, Palembang, Indonesia
email: pinto@fk.unsri.ac.id

INTRODUCTION

Arterial dissection is characterized by a tear in the arterial wall, specifically in the tunica intima, resulting in the separation of the vascular layers and the accumulation of blood within the arterial wall.[1,2] It is estimated that approximately 20% of arterial dissections lead to ischemic stroke in young adults, with a prevalence of up to 20% in this population and an annual incidence rate ranging from 2.6 to 2.9 per 100,000 individuals.[3] The most common type is extracranial internal carotid artery dissection, which happens about 2 to 3 cm above the bifurcation and causes most ischemic strokes (2.5% of all first-ever strokes). The common carotid artery dissection (CCAD), on the other hand, is rare.[4] A study reported that 177 of 8,800 stroke patients had extracranial internal carotid (ICAD) or vertebral artery dissection (VAD), but no cases of CCAD were reported. Another literature review spanning from 1960 to 2010 found 47 cases of CCAD, 11 of which were classified as traumatic CCAD.[5,6] However, in these reported traumatic CCAD cases, no sharp trauma as the cause was identified.

Horner syndrome is another manifestation of carotid artery dissection. It has the classic triad of unilateral ptosis, miosis, and anhidrosis as a result of sympathetic innervation disruption (oculosympathetic paresis). Horner syndrome occurs in around 25% of carotid dissection cases.[7] This suggests that Horner syndrome is an uncommon manifestation of carotid dissection. Clinically, Horner syndrome can serve as a significant "red flag" indicating ocular sympathetic pathway dysfunction.[8] This complicated and convoluted pathway encompasses various anatomical structures, and lesions along this pathway can manifest as Horner syndrome in patients. While Horner syndrome with minor symptoms may be difficult to detect, its identification is crucial in diagnosing potentially life-threatening lesions in the head, neck, and chest.

Given that traumatic carotid artery dissection and Horner syndrome are so rare and rarely happen in everyday life, it is very important for physicians to be able to spot the signs of a suspected carotid dissection and apply appropriate therapeutic strategies for this uncommon condition. The purpose of this case report was to better understand the clinical manifestation of carotid artery dissection following a stab wound and to explore endovascular management as an effective option for stroke prevention.

CASE

A 14-year-old boy presented with a gradual onset of headaches and visual disturbances. He had minor eyelid drooping as well as double vision and glare vision in his left eye. He also had dry skin on the left side of his face. These symptoms appeared two weeks after he was stabbed in the left neck with a knife. The patient also experienced intermittent pulsating headaches on the left side of his head and neck, particularly in the temporal region, but no visual problems.

The patient's headaches intensified five weeks after that traumatic occurrence, and he had his first transient ischemic attack (TIA). The TIA appeared as a sudden onset of communication difficulties and right-side weakness, which resolved within three hours. A normal CT scan was obtained during the radiological examination. The patient was initially treated with antiplatelet therapy. A second TIA occurrence happened two weeks later, characterized by sudden weakness on the right side of his body that disappeared within four hours. A normal CT scan was obtained again, and the patient was subsequently given anticoagulant medication. The patient received medical assistance at our hospital following these two TIA occurrences in order to discover the cause of the TIAs.

The patient experienced another incident of right-limb weakness before coming to our hospital, which improved within two hours. Upon clinical examination, minimal left eye ptosis and miosis were observed, along with anhidrosis on the left side of his face. Other neurological functions were within normal limits. A scar from a previous traumatic event was found on the left anterior side of the neck. A normal CT scan was obtained (Figure 1).

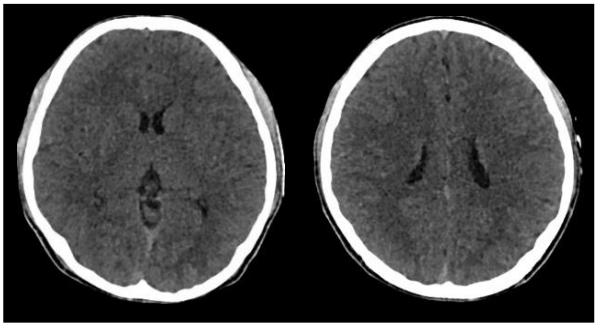

Figure. 1 – Normal head NCCT

A post-surgical scar in the shape of an inverted "L" of approximately 7 cm in length was discovered during physical examination (Figure 2). Neurological assessment revealed unequal pupil sizes (4 mm and 2 mm) and ptosis in the patient's left eye (Figure 3).

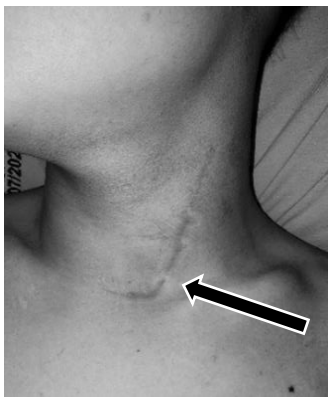

Figure 2. Post-surgical scar on regio colli sinistra (post-traumatic penetrating injury).

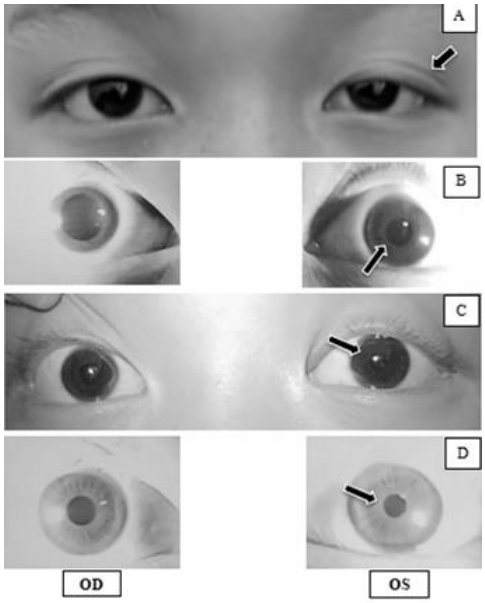

Figure 3. Minimal ptosis on the left eye. (A) the diameter of the left eye is smaller compared to the right eye in dim light (just before it was given direct light. (B) direct light reflex (+) on both eyes, miosis presents on left eye (C, D)

Hematological examination, echocardiography, and carotid doppler were performed to identify risk factors for the patient's TIA attack. The laboratory examinations revealed that the results were normal. As for carotid ultrasound revealed a tear in the artery on the left side of the neck. it was suggested that there was a ±0.80cm gap in the LCCA wall bordering the left thyroid lobe, which then appeared to form a sac with a diameter of ±0.94cm. These findings suggested analyzing the image of LCCA with the differential diagnosis of the image of pseudoaneurysm. (Figure 4)

The patient underwent CT-angiography examinations. From the results suggested, there were pseudoaneurysms on the common carotid artery at the level of vertebrae cervical 7. (Figure 5)

Based on the clinical presentation and findings, we hypothesize that the patient's headache, Horner syndrome, and recurrent TIAs were caused by the identified dissecting aneurysm. Considering the ongoing recurrence of attacks despite antiplatelet and anticoagulant medication, the decision was made to proceed with carotid angioplasty stenting. This intervention was performed in order to prevent more occurrences and improve the patient's condition.

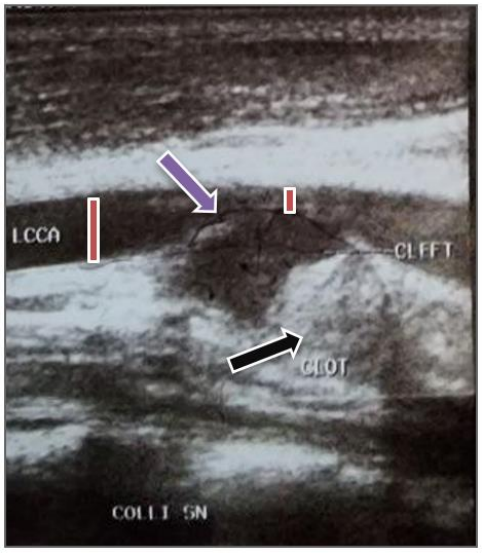

Figure 4. Cleft (purple arrow) due to blood vessel rupture, clot formation (black arrow); accompanied by narrowing of blood vessel lumen diameter (red line) in LCCA.

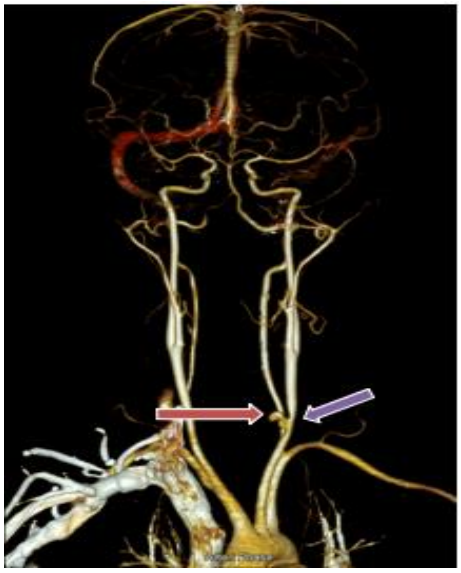

Figure 5. Head and neck CTA showing pseudoaneurysm (red arrow) and stenosis (purple arrow) in LCCA

Digital subtraction angiography confirmed the presence of stenosis along with a dissecting/pseudoaneurysm in the left common carotid artery. The lesion was successfully treated with the placement of a 7 mm dual-layer carotid stent. This intervention improved the lumen size and reduced flow into the pseudoaneurysm (Figure 6). No complications were observed following the carotid angioplasty stent (CAS) procedure. The patient was

discharged with a prescription for dual antiplatelet therapy.

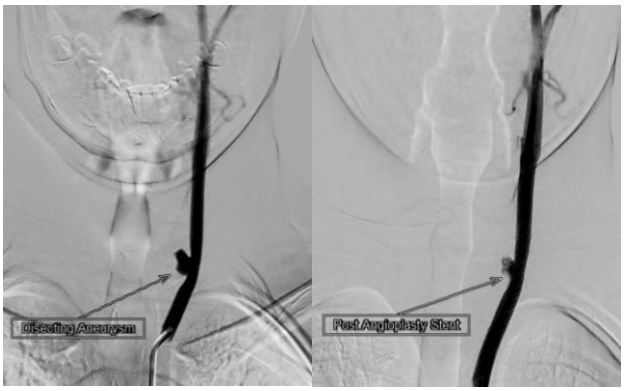

Figure 6. DSA of cerebral angiography showed an aneurysm formation with lumen stenosis implicating dissecting aneurysm (left) before angioplasty. Slow flow of the blood to the aneurysm with improvement of lumen stenosis after angioplasty (right).

During the 3-month follow-up period, the patient showed significant improvement in the symptoms of Horner syndrome. Additionally, there were no further reports of headaches or occurrences of stroke or TIA. After 3 months of dual antiplatelet therapy, the patient's regimen was transitioned to a single 80 mg dose of aspirin.

DISCUSSION

A stab wound commonly causes a dissecting pseudoaneurysm because it disrupts the layers of the arterial wall, resulting in the formation of a false aneurysm. The mechanism includes a direct injury to the arterial wall, which can happen when a sharp object penetrates the tissue and lacerates the blood vessel.[9] Generally, the patient presents with headache, neck, or face pain, as well as partial Horner's syndrome and a cerebral ischemic event.[10]

A stab wound is caused when a sharp item, such as a knife, pierces the skin and underlying tissues, including the arterial wall. The forceful puncture and subsequent removal might create tearing or shearing forces on the arterial wall, resulting in damage to the artery's inner layers. The injury disrupts the artery wall's integrity, leading to the formation of a dissecting pseudoaneurysm. Blood seeps through the tear, forming a false lumen that connects to the arterial lumen. Blood accumulates inside the arterial wall layer, forming a bulging sac-like structure. This pseudoaneurysm may continue to expand over time, compressing adjacent structures or impairing blood flow through the afflicted artery.[11,12]

The underlying pathophysiology of blood vessel dissection involves the formation of a gap between the layers of the blood vessel. Blood enters the space rightt below the tunica intima immediately after the initial tear, leading in the creation of an intramural hematoma (also known as a false lumen or pseudolumen).[12] This can lead to the creation of pseudo-occlusion or thrombus, which can embolize the distal blood vessel or form a pseudoaneurysm. In cases of transient ischemic attack (TIA), the loose thrombus and temporary blockage of anterior circulation blood vessels can lead to symptoms such as aphasia and weakness in the right arm. [3,13]

Horner syndrome can arise as a result of a common carotid artery injury or dissection that disrupts or compresses second-order oculosympathetic nerve fibers. This is due to the failure of the superior tarsal muscle, which is responsible for raising the upper eyelid and receiving sympathetic nerve supply. Horner syndrome causes milder ptosis than oculomotor (CN III) palsy, which supplies the levator palpebrae superioris.[14,15] The denervation of the superior tarsal muscle partially explains the observed ptosis. Additionally, the disruption of sympathetic nerve supply causes mydriasis (pupil dilation), whereas the unimpeded parasympathetic supply causes miosis (pupil constriction).[16] The pupillary light reflex and accommodation responses remain unaffected since they do not rely on sympathetic nerve supply.[17] Another characteristic feature is ipsilateral anhidrosis, which is dependent on the extent of the sympathetic supply disruption. The ipsilateral side of the body is affected by anhidrosis caused by first-order neuronal lesions because the sympathetic supply comes from the central nervous system.

The underlying pathophysiology of headaches in carotid dissection is related to the continual pressure exerted on the dissected carotid blood vessels, which causes stretching and pulsing pain.[18] The pain usually correlates to the affected area of carotid dissection, with temporal pain in carotid dissections and occipital pain in vertebral dissections (Figure 7).

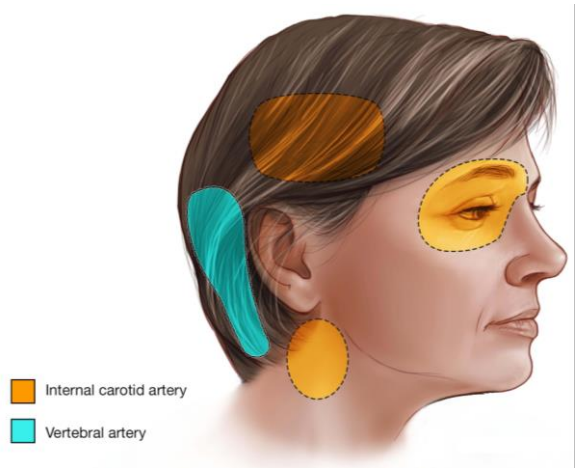

Figure 7. Headache or neck pain locations with internal carotid (orange) and vertebral artery dissection (green)[12]

A carotid ultrasound examination was performed on the patient during therapy at the time of the first TIA attack, which revealed a dissection of the LCCA and the finding of a cleft in the vessel wall. This supported the clinical suspicion of a previous carotid artery dissection. According to the guideline for the management of stroke or TIA in extracranial carotid or vertebral dissection, antithrombotic therapy for 3 months can be given to prevent stroke or TIA recurrence (Class I; Level of Evidence: C), with the drug of choice being aspirin or warfarin (Class II B; Level of Evidence: B).[19]

The patient had previously been treated with an anticoagulant as a preventative antithrombotic since the first TIA attack, but the TIA recurrence happened two weeks later. According to the AHA ASA 2021 Ischemic Stroke Prevention guideline, patients with stroke or TIA and extracranial carotid or vertebral artery dissection who have recurrent events despite antithrombotic therapy and endovascular therapy can be considered to prevent recurrent stroke or TIA (Class: IIb; Level of Evidence: C). Therefore, patients should receive endovascular therapy.[19] Endovascular therapy may be considered to close the pseudoaneurysm and dilate the carotid lumen. Carotid stenting is an option to prevent intramural thrombus formation and widen the diameter of the stenosed carotid artery.[20]

The patient was then treated as an outpatient with dual antiplatelet therapy and monitored for one month. The main goal of management was to prevent recurrent stroke or TIA, whereas the secondary goal was clinical improvement, especially Horner syndrome. After a one-month examination, it was discovered that the stroke had not recurred, but the ptosis complaint had improved slightly. Glare and double vision were reported to have disappeared once the present pupil size was iso-corrected. Horner syndrome improvement is reported to occur within 3–6 months.

CONCLUSSION

In conclusion, extracranial carotid artery dissection is a condition that should not be ignored in the differential diagnosis of post-traumatic headache cases. This needs to be caught early and diagnosed correctly so that stroke or transient ischemic attack prevention management can be given to reduce the incidence of post-dissection stroke and the occurrence of severe or even permanent neurological deficits. Arterial dissection requires considerable deliberation before deciding on a course of treatment, with antiplatelet therapies, anticoagulants, and endovascular therapy all being possibilities. In this case, carotid stenting was successful for the improvement of Horner's syndrome, and the patient has not experienced a recurrence of stroke or transient ischemic attack in the past month.

Acknowledgment
We would like to express our sincere gratitude to the institutions and individuals that contributed to the development and completion of this manuscript. We extend our heartfelt appreciation to the Department of Neurology at the Mohammad Hoesin General Hospital and the Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia, for their unwavering support and provision of resources throughout the course of this study.

Conflict of Interest
All authors have no conflict of interest.

Funding
No funding sources.

Author Contribution
PDR contributed to the collection of data, data analysis, interpretation, supervision of the study, and writing the manuscript; HM contributed to the writing of the manuscript and the collection of data.

REFERENCES

1. Birawa ABP, Amalia L. Stroke pada usia muda. *Cermin Dunia Kedokt*. 2015;42(10):737–9. [Journal]
2. Bax M, Romanov V, Junday K, Giannoulatou E, Martinac B, Kovacic JC, et al. Arterial dissections: Common features and new perspectives. *Front Cardiovasc Med*. 2022; 9. doi: 10.3389/fcvm.2022.1055862
3. Caplan L, Biller J. Uncommon causes of stroke. 3rd ed. Cambridge University Press; 2018. [Book]
4. Gaillard F, Baba Y, Thwala S. Internal carotid artery dissection. In: Radiopaedia.org. Radiopaedia.org; 2023. doi: 10.53347/rID-6692
5. Kil JS, Lee MK, Eom KS. Common carotid artery dissection in multiple extracranial injury: A case report. *Korean J Neurotrauma*. 2018; 14(1):28–31. doi: 10.13004/kjnt.2018.14.1.28
6. Boucher P, Robidoux S, Chahine S. Cervical artery dissections: Factors that influence causation determination in litigated cases. *J Forensic Leg Med*. 2018; 58:169–78. doi: 10.1016/j.jflm.2018.06.007
7. Martin TJ. Horner syndrome: A clinical review. *ACS Chem Neurosci*. 2018; 9(2):177–86. doi: 10.1021/acschemneuro.7b00405
8. Abbas A, Manjila S, Singh M, Belle V, Chandar K, Miller JP. Johann Friedrich Horner and the repeated discovery of oculosympathoparesis. *Neurosurgery*. 2015; 77(3):486–91. doi: 10.1227/NEU.0000000000000832
9. Biller J, Sacco RL, Albuquerque FC, Demaerschalk BM, Fayad P, Long PH, et al. Cervical arterial dissections and association with cervical manipulative therapy: A statement for healthcare professionals from the american heart association/american stroke association. *Stroke*. 2014; 45(10):3155–74. doi: 10.1161/STR.0000000000000016
10. Bond KM, Krings T, Lanzino G, Brinjikji W. Intracranial dissections: A pictorial review of pathophysiology, imaging features, and natural history. *J Neuroradiol*. 2021; 48(3):176–

88. doi: 10.1016/j.neurad.2020.03.007
11. Keser Z, Meschia JF, Lanzino G. Craniocervical artery dissections: A concise review for clinicians. *Mayo Clin Proc*. 2022; 97(4):777–83. doi: 10.1016/j.mayocp.2022.02.007
12. Keser Z, Chiang C-C, Benson JC, Pezzini A, Lanzino G. Cervical artery dissections: Etiopathogenesis and management. *Vasc Health Risk Manag*. 2022; 18:685–700. doi: 10.2147/VHRM.S362844
13. Debette S, Compter A, Labeyrie M-A, Uyttenboogaart M, Metso TM, Majersik JJ, et al. Epidemiology, pathophysiology, diagnosis, and management of intracranial artery dissection. *Lancet Neurol*. 2015; 14(6):640–54. doi: 10.1016/S1474-4422(15)00009-5
14. Khan Z, Bollu PC. Horner syndrome. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023. [Book]
15. Han J, Park SY, Lee J-Y. Nationwide population-based incidence and etiologies of pediatric and adult Horner syndrome. *J Neurol*. 2021; 268(4):1276–83. doi: 10.1007/s00415-020-10270-2
16. Hall JE. Guyton and Hall textbook of medical physiology. 13th ed. Philadelphia: Elsevier Inc; 2016. 515–519 p. [Book]
17. Kanagalingam S, Miller NR. Horner syndrome: Clinical perspectives. *Eye Brain*. 2015; 7:35–46. doi: 10.2147/EB.S63633
18. Chen SY, Zipfel GJ, Wick CC. Internal carotid artery dissection causing pulsatile tinnitus. *Am J Otolaryngol*. 2019; 40(1):121–3. doi: 10.1016/j.amjoto.2018.09.008
19. Kleindorfer DO, Towfighi A, Chaturvedi S, Cockroft KM, Gutierrez J, Lombardi-Hill D, et al. 2021 Guideline for the prevention of stroke in patients with stroke and transient ischemic attack: A guideline from the American Heart Association/American Stroke Association. *Stroke*. 2021; 52(7):e364–467. doi: 10.1161/STR.0000000000000375
20. Vellimana AK, Lavie J, Chatterjee AR. Endovascular considerations in traumatic injury of the carotid and vertebral arteries. *Semin Intervent Radiol*. 2021; 38(1):53–63. doi: 10.1055/s-0041-1724008