Annals of Stroke and Vascular Neurology

Case Report

Stop Yelling: A Rare Cause of Internal Carotid Artery Dissection (ICAD) in a Young Professional

Alyssa Caplan[1] and Atul Sharma[2]*

[1]Department of Internal Medicine, St. Mary's Medical Center, CA, USA

[2]Department of Medicine, University of California, CA, USA

Abstract

Internal Carotid Artery Dissection (ICAD) is a tear involving the intimal layer of the blood vessel resulting in intramural hematoma or aneurysmal dilatation. Although ICAD is an uncommon entity and the clinical presentations are often benign, dissections are the most common cause of stroke in young patients, accounting for about 10% of all strokes in this population. Although typically following minor trauma, spontaneous ICADs often occur in young and previously healthy individuals, and can be easily missed by clinicians given their variable presentation. Our case describes spontaneous ICAD in a healthy 41-year-old male. He presented with 1 week history of left sided headache, which began after a stressful business related telephone call during which he endorsed a prolonged period of yelling. This case highlights the need to consider ICAD in young patients presenting with ipsilateral headache, appropriate work-up and management.

Keywords: ICA dissection; Carotid artery dissection; ICAD; Stroke

Case Presentation

A healthy 41-year-old right-handed male presented to the emergency department with left-sided headache. One week prior to admission, he experienced pain behind his left ear that began abruptly after a stressful business-related telephone call, during which he reported yelling for a prolonged period of time after losing a large sum of money in the stock market. The pain behind his left ear, although localized at first, subsequently migrated behind his left eye and left temporal region. He also endorsed subjective left eye drooping with associated diplopia when looking to the left. No changes in speech, gait, or focal weakness were initially observed. The night prior to his presentation to the ED, patient developed tingling sensation in the sole of his left foot. On the morning of his presentation to the hospital he developed ataxic gait where he found himself veering to the right. When he arrived to the ED, all his symptoms had resolved except for diplopia and left sided headache. Patient denied any history of head/neck trauma, including motor vehicle accidents, injuries or falls. He additionally denied vigorous coughing, heavy lifting, retching, riding roller coasters or chiropractor manipulations.

Family history

Mother died of a brain aneurysm at age 60 and his brother suffered multiple strokes at age 50.

Citation: Caplan A, Sharma A. Stop Yelling: A Rare Cause of Internal Carotid Artery Dissection (ICAD) in a Young Professional. Ann Stroke Vasc Neurol. 2020;1(1):1002.

Publisher Name: Medtext Publications LLC

Manuscript compiled: Oct 09th, 2020

***Corresponding author:** Atul Sharma, Department of Medicine, University of California, San Francisco, CA, USA, E-mail: atul.sharma@ucsf.edu

Social history

Moderate alcohol use but denies any drug (IV and non - IV drugs) or tobacco use.

Physical examination

Vital Signs: Within normal limits.

General: WDWN Male in no acute distress.

HEENT: TMs Normal; See neuro section for eye exam.

Pulmonary: CTA bilaterally.

Cardiac: RRR; No murmurs, gallops or rubs.

Neuro: Mental status/Psych: A & O × 3. Fluent language. Naming intact too high and low frequency objects. Repetition intact. Followed 2 step commands. Memory intact to the details of the history.

Cranial nerves: VFFTC. PERRL. Diplopia on far left gaze. Facial strength and sensation intact and symmetric. Tongue and palate midline. Traps full. No dysarthria. Allodynia over left forehead, scalp, and neck.

Motor: Normal bulk and tone. No pronator drifts. Finger and foot taps fast and symmetric.

Strength: 5/5 throughout all muscle groups.

Reflexes: 2+ throughout.

Sensory: Intact to light touch, vibration, and pin prick throughout. Romberg negative.

Gait: Normal narrow based casual gait. Able to walk on toes, heels, and tandem.

Labs

No significant derangements.

Imaging

CT Angiogram (CTA) of head/neck showed L cervical ICA dissection extending into the petrous portion (Figure 1). Non contrast

Brain MRI showed no evidence of an infarct. MRI demonstrated intramural hematoma with elevated T1 signal around left high cervical ICA extending into the petrous portion, consistent with ICA dissection (Figure 2). No cervical spine fractures were noted on the imaging studies.

Management

He was started on aspirin 325 mg daily.

Discussion

Carotid artery dissection is a common cause of stroke in young individuals [1,2]. Internal Carotid Artery Dissection (ICAD) is defined as tearing of the intimal layer of the artery due to a variety of etiologies. The shear leads to intramural hematoma, which creates stenosis and clot formation and ultimately can cause strokes [3]. Because ICAD is uncommon with variable clinical presentations and a common cause of stroke in young patients, it is imperative that this diagnosis is on the differential for those presenting with ipsilateral headaches.

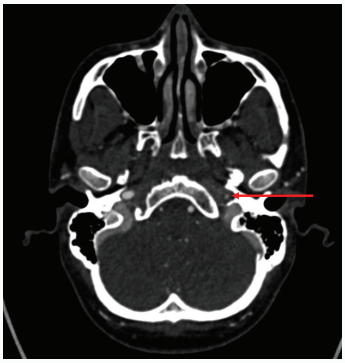

Figure 1: Contrast enhanced CT axial.

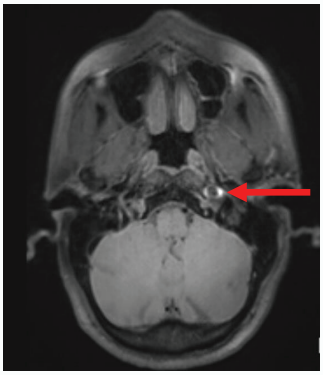

Figure 2: T1-weighted MRI, axial.

Causes

Carotid artery dissection often occur secondary to trauma, usually mild, including but not limited to, nose blowing, coughing, and sudden neck turning, especially hyperflexion resulting in stretching of the ICA over the spinal vertebrae [4,5]. Other more commonly known causes include motor vehicle accidents and chiropractic manipulations. Inherited connective tissue disorders such as Ehlers-Danlos syndrome, Marfan syndrome, fibromuscular dysplasia, and osteogenesis imperfecta have also been associated with spontaneous ICAD [6]. The exact etiology of ICAD in our patient is unclear; however, we postulate that it is related to his prolonged episode of yelling. In reviewing the literature, we found one case report detailing yelling (a fan cheering loudly at a sporting event) as a mechanism of ICAD [7]. The previous case report hypothesized that a strong vocal output may be an independent risk factor for developing ICAD.

Symptoms

Initial symptoms can be non-specific and variable making the diagnosis difficult [8]. The extracranial portion of the ICA is the most commonly affected vessel (>90%); dissections of the intracranial portion, although less common culprit, are associated with greater neurological deficits and have a poorer prognosis [9].

Approximately two-thirds of patients with carotid artery dissection presents with ipsilateral headache, face, or neck pain as seen in our patient [10]. Non-specific features of headache can often resemble cluster or migraine headaches [11]. Although strongly associated with ICAD, acute painful Horner Syndrome (miosis and ptosis without anhidrosis), is only present in about 50% of the cases [11]. Interestingly, our patient reported subjective left eye drooping, which had resolved by the time he presented to the hospital.

Other focal neurologic symptoms including weakness or numbness on one side of the body, speech disturbances, dysphagia, pulsing sound in an ear, and abnormal or loss of sense of taste can also be present. Internal carotid or vertebral artery dissections complicated by development of pseudoaneurysm may exhibit local symptoms due to compression of adjacent structures. For example, cranial nerves XII, IX and X are more commonly affected in carotid artery dissections due to its close proximity to the vessel. Rupture of pseudoaneurysm resulting in subarachnoid hemorrhage is a feared complication in carotid artery dissection. This often presents with an acute onset severe thunderclap headache, is a medical emergency, and is associated with high morbidity and mortality [12].

Workup/Imaging

A clinician should have a high index of suspicion for carotid artery dissection based on thorough history and physical examination to make the diagnosis. CT angiogram of head and neck is most commonly performed as it can be concurrently done with CT head to assess for acute stroke or intracranial hemorrhage [13]. The sensitivity of CT angiography in detecting ICAD is 80% to 95% [14]; however, this imaging modality is associated with radiation exposure and technical challenges [15].

MRA along with T1 axial cervical MRI with fat saturation technique is favored due to high sensitivity and specificity (84% and 99% respectively) [16], ability to accurately visualize an intramural hematoma and absence of radiation exposure [17]. Carotid ultrasonography with color Doppler is another imaging modality that can be utilized, but it is operator dependent and has poor diagnostic value in intracranial carotid dissection compared to extracranial carotid dissection [18].

Management

Prevention of strokes is the hallmark for the treatment of ICAD. However, there is some controversy as to whether anticoagulation is superior to antiplatelet therapy in preventing strokes in this subgroup of patients. Most studies have not shown any difference between the two groups [19]. Cervical Artery Dissection in Stroke Study (CADISS) further proved that there are no differences in the efficacy of antiplatelet and anticoagulation in preventing strokes (Figure 3) [20].

	Intention-to-treat population				Per-protocol population			
	Antiplatelet group (n=126)	Anticoagulant group (n=124)	OR (95% CI)*	p value	Antiplatelet group (n=101)	Anticoagulant group (n=96)	OR (95% CI)*	p value
psilateral stroke or death	3 (2%)	1 (1%)	0·335 (0·006–4·233)	0·63	3 (3%)	1 (1%)	0·346 (0·006–4·390)	0·66
Secondary endpoints								
Any stroke or death	3 (2%)	1 (1%)	0·335 (0·006–4·233)	0·63	3 (3%)	1 (1%)	0·346 (0·006–4·390)	0·66
Any stroke, death, or major bleed	3 (3%)	2 (2%)	0·673 (0·055–5·983)	1·00	3 (3%)	2 (2%)	0·696 (0·057–6·220)	1·00
Any stroke	3 (2%)	1 (1%)	0·335 (0·006–4·233)	0·63	3 (3%)	1 (1%)	0·346 (0·006–4·390)	0·66
Ipsilateral stroke, TIA, or death	4 (3%)	5 (4%)	1·280 (0·268–6·614)	0·98	4 (4%)	4 (4%)	1·054 (0·190–5·835)	1·00
Any stroke or TIA	5 (4%)	5 (4%)	1·017 (0·228–4·540)	1·00	5 (5%)	4 (4%)	0·836 (0·161–4·015)	1·00
Major bleeding	0 (0%)	1 (1%)	..	..	0 (0%)	1 (1%)	..	..
Death	0 (0%)	0 (0%)	..	..	0 (0%)	0 (0%)	..	..

ata for presence of residual stenosis (>50%) at 3 months have not yet been analysed. OR=odds ratio. TIA=transient ischaemic attack. *Tested with exact logistic regression.

able 2: Outcomes within 3 months

Figure 3: CADISS Trial. Antiplatelet versus anticoagulant comparison.

The majority of carotid and vertebral artery dissections heal spontaneously and management with antithrombotic agents, including aspirin and clopidogrel are often sufficient to prevent thromboembolic complications. The American Heart Association recommends Aspirin 75 mg to 325 mg daily over anticoagulation with coumadin [21].

In patients with unstable lesions, progressive neurological deficits and/or further strokes despite treatment, endovascular stenting has been shown to have a 99% success rate [22]. Similarly, if the patient exhibits symptoms of cerebral ischemia, severe narrowing of the arterial lumen, or an unstable plaque, IV heparin is frequently utilized, followed by warfarin [23].

Given the stable presentation exhibited by our patient, he was only treated with aspirin with close outpatient follow up.

Conclusion

ICAD is an important cause of stroke, especially among young individuals. Due to variable presentations and subtle symptoms, a clinician should have a high index of suspicion for the diagnosis. Prompt imaging study with CTA or MRI/MRA should be performed. Since embolism from thrombus forming at the dissection site is thought to play a significant role in stroke pathogenesis, treatment with antiplatelet agent or anticoagulation should be rendered with close outpatient follow-up.

References

1. Xianjun H, Zhiming Z. A systematic review of endovascular management of internal carotid artery dissections. Interv Neurol. 2013;1(3-4):164-70.
2. Smajlovic D. Strokes in young adults: epidemiology and prevention. Vasc Health Risk Manag. 2015;11:157-64.
3. Anyama B, Treitl D, Wessell J, Solomon R, Rosenthal AA. Delayed Stroke following Blunt Neck Trauma: A Case Illustration with Recommendations for Diagnosis and Treatment. Case Rep Emerg Med. 2017;2017:3931985.
4. Thanvi B, Munshi SK, Dawson SL, Robinson TG. Carotid and vertebral artery dissection syndromes. Postgrad Med J. 2005;81(956):383-8.
5. Carotid Dissection. Carotid Dissection - UCLA Neurosurgery Los Angeles: USA.
6. Kim YK, Schulman S. Cervical artery dissection: pathology, epidemiology and management. Thromb Res. 2009;123(6):810-21.
7. Hirji N, Ionides A. Internal carotid artery dissection secondary to excessive vocal output. BMJ Case Rep. 2011;2011:bcr0120113710.
8. Giroud M, Gras P, Dumas R, Becker F. Spontaneous vertebral artery dissection initially revealed by a pain in one upper arm. Stroke. 1993;24(3):480-1.
9. Guillon B, Lévy C, Bousser MG. Internal carotid artery dissection: an update. J Neurol Sci. 1998;153(2):146-58.
10. Kasravi N, Leung A, Silver I, Burneo JG. Dissection of the internal carotid artery causing Horner syndrome and palsy of cranial nerve XII. CMAJ. 2010;182(9):E373-7.
11. Debette S, Leys D. Cervical-artery dissections: Predisposing factors, diagnosis, and outcome. Lancet Neurol. 2009;8:668-78.
12. Blum CA, Yaghi S. Cervical Artery Dissection: A Review of the Epidemiology, Pathophysiology, Treatment, and Outcome. Arch Neurosci. 2015;2(4):e26670.
13. Goodfriend SD, Tadi P, Koury R. Carotid Artery Dissection. StatPearls. 2020.
14. Mansoor M, Sexton C. Imaging of carotid artery dissection. J Community Hosp Intern Med Perspect. 2012.
15. Leclerc X, Godefroy O, Salhi A, Lucas C, Leys D, Pruvo JP. Helical ct for the diagnosis of extracranial internal carotid artery dissection. Stroke. 1996;27(3):461-6.
16. Le´vy C, Laissy JP, Raveau V, Amarenco P, Servois V, Bousser MG, et al. Carotid and vertebral artery dissections: Threedimensional time-of-flight MR angiography and MR imaging versus conventional angiography. Radiology. 1994;190(1):97-103.
17. Provenzale JM. Mri and mra for evaluation of dissection of craniocerebral arteries: Lessons from the medical literature. Emerg Radiol. 2009;16:185-93.
18. Benninger DH, Georgiadis D, Gandjour J, Baumgartner RW. Accuracy of color duplex ultrasound diagnosis of spontaneous carotid dissection causing ischemia. Stroke. 2006;37(2):377-81.
19. Markus HS, Levi C, King A, Madigan J, Norris J. Cervical Artery Dissection in Stroke Study (CADISS) Investigators. Antiplatelet Therapy vs Anticoagulation Therapy in Cervical Artery Dissection: The Cervical Artery Dissection in Stroke Study (CADISS) Randomized Clinical Trial Final Results. JAMA Neurol. 2019;76(6):657-64.
20. American Academy of Neurology. Guideline on the management of patients with extracranial carotid and vertebral artery disease. Dallas, USA: ANN Publications. 2011.
21. Moon K, Albuquerque FC, Cole T, Gross BA, McDougall CG. Stroke prevention by endovascular treatment of carotid and vertebral artery dissections. J Neurointerv Surg. 2017;9(10):952-7.
22. Peng J, Liu Z, Luo C, Chen L, Hou X, Xiao L, et al. Treatment of Cervical Artery Dissection: Antithrombotics, Thrombolysis, and Endovascular Therapy. Biomed Res Int. 2017;2017:3072098.
23. Brown SC, Falcone GJ, Hebert RM, Yaghi S, Mac Grory B, Stretz C. Stenting for Acute Carotid Artery Dissection. Stroke. 2020;51(1):e3-6.