Case Report

Indian Journal of Emergency Medicine
Volume 5 Number 2, April - June 2019
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.5219.11

Rare Presentation of a Traumatic B/L Carotid and Vertebral Arteries Dissection; Diagnostic Challenges in Emergency Department

Kangjam Lakshmi Devi[1], Sarat Naidu[2], Dheeraj Bhaskaran Nair[3], Ritu Sabharwal[4], Hrishikesh Chakraborty[5],

Author's Affiliation:
[1]MEM Resident, [2]Attending Consultant, [3]HOD, [4]Attending Consultant, Department of Emergency Medicine, [5]Consultant, Department of Neurosurgery, Max Super Speciality Hospital, Vaishali, Ghaziabad, Uttar Pradesh 201012, India.

Corresponding Author:
Sarat Naidu, Attending Consultant, Department of Emergency Medicine, Max Super Speciality Hospital,Vaishali, Ghaziabad, Uttar Pradesh 201012, India.

E-mail: ritz154@yahoo.com

Received on 22.02.2019,
Accepted on 26.03.2019

Abstract

A 46 years old previously healthy female was brought to emergency department (ED) with alleged history of road traffic accident (RTA) while driving a car one hours back. She was initially assymptomatic for about one hours post accident but later deteriorated both hemodynamically and mentation. After aggressive resuscitation as per ATLS protocol, she underwent multiple radiological imaging studies which revealed bilateral carotid and vertebral arteries dissection and severe traumatic brain injury with bilateral peripheral infarction. She was managed conservatively with mechanical ventilation in the intensive care unit.

Keywords: Road traffic accident; Trauma; Traumatic brain injury; Bilateral carotid artery dissection; Vertebral artery dissection; Multiple brain infarction.

Introduction

Cervical artery consist of four main arteries that supply blood flow to the brain, two carotid arteries and two vertebral arteries. The carotid arteries can be felt on each side of the lower neck, and the vertebral arteries are located in the back of the neck near the spine and cannot be felt on physical examination.

Carotid artery forms the main arterial blood supply to the brain. The "Circle of Willis" begins to form when the right and left internal carotid artery (ICA) enters the cranial cavity and each one divides into two main branches: the Anterior Cerebral Artery (ACA) and Middle Cerebral Artery (MCA). The anterior cerebral arteries are then united and blood can cross flow by the anterior communicating (ACOM) artery The ACAs supply most midline portions of the frontal lobes and superior medial parietal lobes. The MCAs supply most of the lateral surface of the hemisphere, except the superior portion of the parietal lobe (via ACA) and the inferior portion of the temporal lobe and occipital lobe. The ACAs, ACOM, and MCAs form the anterior half, or better known as the anterior circulation of the circle of Willis. Posteriorly, the Basilar Artery (BA), formed by the left and right vertebral arteries, branches into a left and right Posterior Cerebral Artery (PCA), forming the posterior circulation as soon in fig below.

Carotid artery dissection or Vertebral artery dissection is the tear in the arterial wall, forcing blood between the layers of the wall by splitting the layers. They have similar aetiology with carotid artery dissection being twice as common as vertebral artery dissection. It can be caused by major or minor trauma, hyperextension of the neck. And it can also be spontaneous, in which case, genetic, familial, or heritable disorders are likely etiologies [1,2]. It can be unilateral or bilateral. Dissection

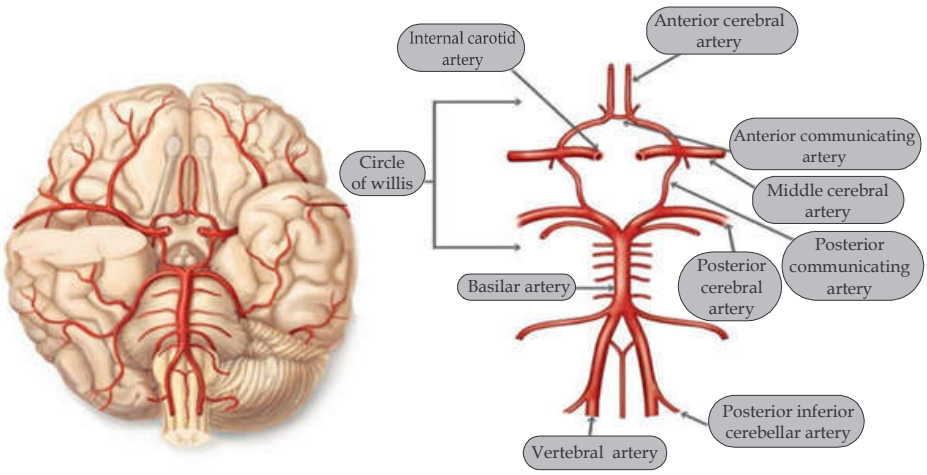

Fig. 1: Carotid artery (Google Courtesy)

of the carotid arteries is rare with recent data suggesting rates in patients with blunt head trauma mainly (high-speed motor vehicle accidents) ranges from less than 1% to 3% [1,3]. Carotid artery and vertebral artery dissection is an important and leading cause of ischemic brain stroke in patients under 45 years of age and the condition is likely to be underdiagnosed.

We are reporting a case of an unusually delayed presentation of bilateral carotid artery and bilateral vertebral arteries dissection with bilateral brain infarct after a Road traffic Accident.

Case Study

A previously healthy 46 years old female was brought to the emergency department with alleged history of road traffic accident (RTA) while driving a car with her child in the front passenger seat. She was initially taken to another hospital for initial management. As per her transfer documents, She did not have vomiting, seizures, loss of consciousness nor ENT bleed. She had complaints of dizziness but was conscious and oriented with time, place and person. After some medications there, she came to our emergency department for further management as her dizziness did not subside.

On arrival in our ED 4 hours post-accident, she was conscious and oriented but had light-headedness and generalized fatiguability. Her pulse was 90/min, BP 140/80 mmHg, SpO_2 94% at RA, RBS 99 mg%.

While being examined, she had a seizure (GTCS) which was managed appropriately with benzodiazepines.

She was immediately assessed thoroughly as per ATLS protocol.

She was intubated and put on mechanical ventilator.

She was started on antiepileptics, IV fluids, IV antibiotics and IV sedation and paralytic agents.

NCCT head was done which showed normal findings. (Fig. 1).

X ray C-spine did not reveal any significant abnormality.

It was therefore thought initially that the seizures were due to concussion injury to the brain and was managed conservatively.

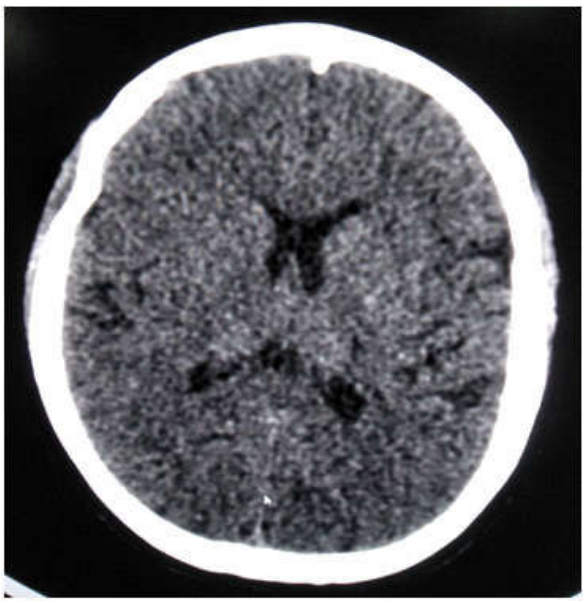

Fig. 1: NCCT Head (normal)

After few hours of elective ventilation, sedation and paralytics were stopped to see response (GCS). After stopping sedation her response was not as good as expected (E1 V1 M2, pupils bilateral sluggishly reacting); In view of prolonged poor GCS, Neurology opinion was sought in this case of trauma. MRI brain showed right MCA, ACA, PCA infarction and left ACA, part of MCA and PCA infarction. (Fig. 2).

After the MRI report of the massive infarct, CT angio brain and neck vessels was done which revealed dissection of bilateral carotid arteries and bilateral vertebral arteries (Fig. 3).

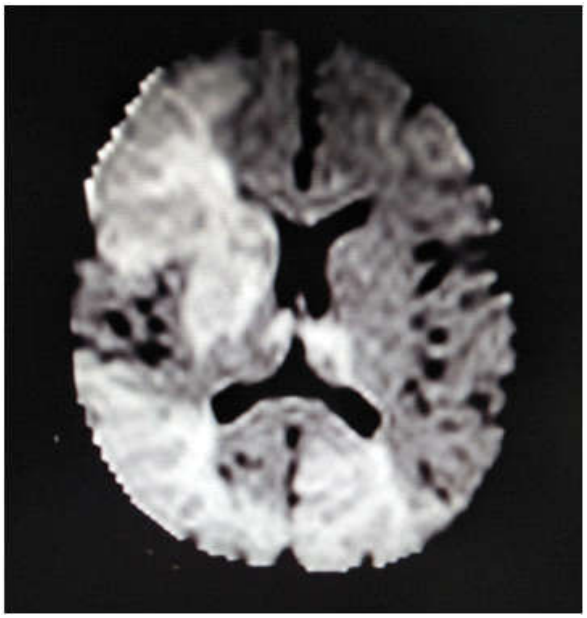

Fig. 2: MRI Brain showing right MCA, ACA, PCA and left ACA, part of ACA and PCA infarction.

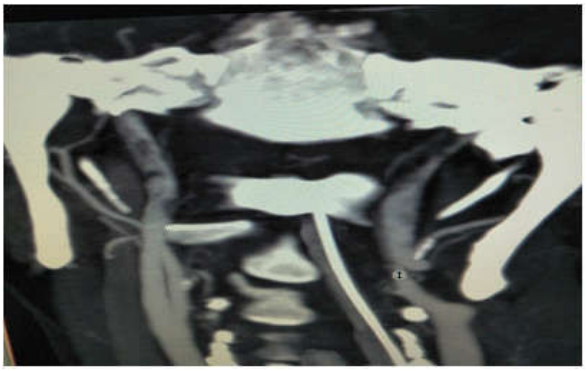

Fig. 3: CT Angio Neck

All other radiological imaging studies were unremarkable.

She started deteriorating hemodynamically, therefore a multi-speciality approach was undertaken, including Neurosurgery, Neurology, and the Surgical trauma team.

Further considering the massive volume of infarction bilaterally, the chances of haemorrhagic conversion was very high if anticoagulation was started; so it was decided to continue elective ventilation and conservative management.

She remained more or less stable for about 3 days in the ICU but started deteriorating from 4th day onwards. She was then taken to another hospital by her family members for further management.

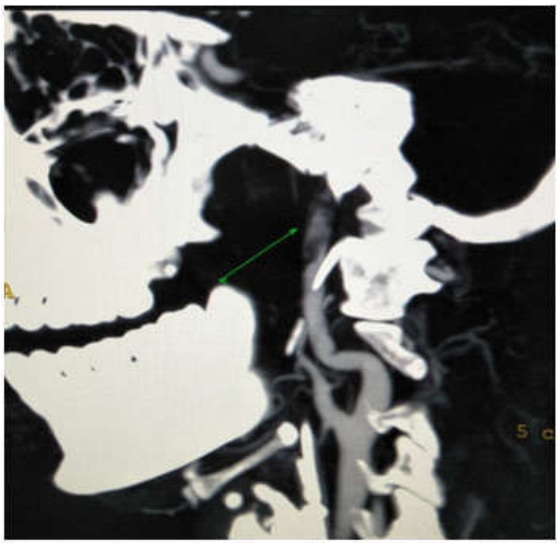

Fig. 4: CT Angio Right side

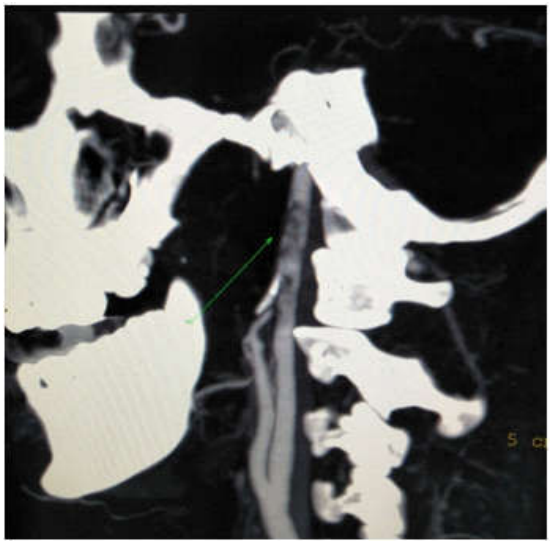

Fig. 5: CT Angio Left side

Discussion

This case report summarizes a very unique case where the patient had severe internal vascular injuries but she had very vague and mild clinical features on examination. It was only after the MRI brain and CT angiography findings that the actual injuries were known.

Normal CT head findings and the delayed presentation of the seizures post-injury also mislead the ER physician; the initial impression was that the patient was in post-ictal state. However the repeated GCS monitoring showed prolonged poor GCS inspite of withholding sedatives and paralytics which made us suspicious of another cause of her altered mental status. This was when Neurological opinion and MRI brain were undertaken.

The use of Glasgow Coma Scale (GCS) is very important for monitoring any patient involved in trauma. The GCS provides an objective, reliable way in quantifying consciousness and is particularly valuable when it is used for continuous evaluation [1]. Despite a normal CT brain, MRA brain showed right MCA, ACA, PCA infarction and left ACA, part of MCA and PCA infarction. Futher imaging was obtained with CT angio brain and neck which is suggestive of bilateral corotid artery dissection and thrombosis.

Although the cause of internal carotid artery dissection remains elusive, it can be due to mechanical forces (eg, trauma, blunt injury, and stretching) and underlying arteriopathies (eg, Ehlers-Danlos syndrome IV and other connective tissue disorders and aberrations). The probable mechanism of injury for most internal carotid injuries is rapid deceleration, hyperextension and rotation of the neck, which stretches the internal carotid artery over the upper cervical vertebrae, leading an tear in the tunica intima or directly within the tunica media with blood flow into the arterial wall, creating an intramural hematoma that leads a thrombus [2,3,4].

Carotid artery dissection is always very serious and life threathening condition. Post-traumatic carotid dissection is rather rare and can be asymptomatic at the initial stage and is usually diagnosed late. It can cause cerebral hypoperfusion and embolism which can lead to stroke [5]. And there are no specific guidelines or screening protocol for detection of carotid injury in the absence of symptoms after Road Traffic Accidents. Hence the diagnosis of carotid artery injury before the development of neurological symptoms is a significant challenge in Emergency Department [6].

The incidence of carotid artery dissection as a result of blunt injuries (mainly high-speed motor vehicle accidents) ranges from less than 1% to 3% and it is a common cause of ischemic stroke in patients younger than 45 years and accounts for as many as 25% of ischemic strokes in young and middle-aged patients. In a review of the literature, only 16 cases of documented blunt traumatic bilateral internal carotid artery dissections were found, one of these was associated with bilateral traumatic disruptions of the vertebral arteries [7,8,9].

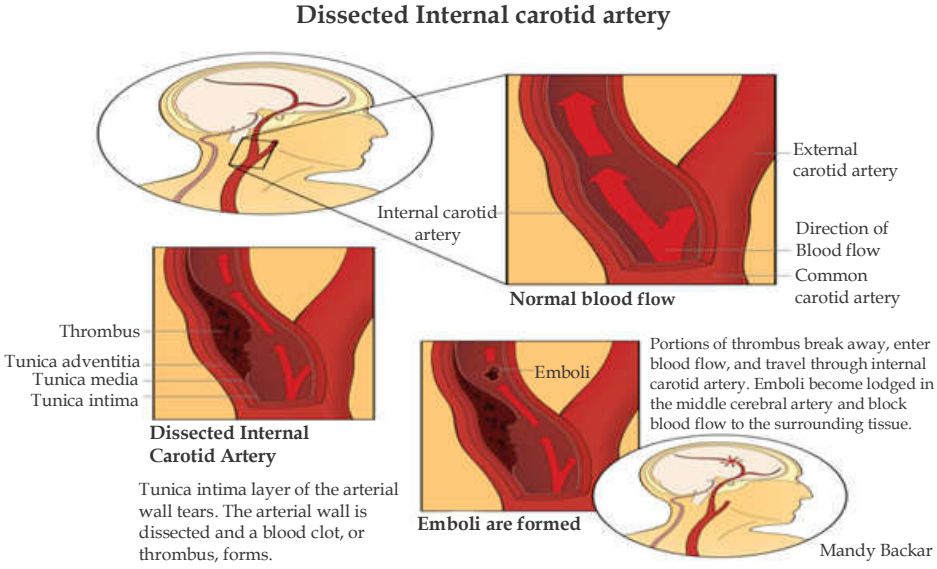

Fig. 6: Internal Carotid Artery Dissection (Google Courtesy)

The classical presentation are Localised headache, mostly around one of the eyes, neck pain, decreased pupil size with drooping of the upper eyelid (Horner syndrome). Patients detected early with mild neurological deficits recovered well with treatment, while those with profound neurological deficits and delayed diagnoses had poor prognosis [10].

The diagnosis is quite challenging and it is made because of awareness of its clinical manisfestations and of advances in non-invasive imaging like color duplex ultrasonography, CT angiography, MRI and MR angiography, and conventional catheter angiography.

Initial computed tomography (CT) of the head is usually warranted, depending on the patient's presentation as we also performed in our case but was found to be normal. The primary indication for carotid angiography is any neurologic deficit that cannot be explained by head CT findings.

Early carotid and intracranial angiography should be performed when there is high suspection of carotid artery lesion involvement and it is more appropriate as initial screening modality in cases of severe trauma. Although, the golden standard for identifying a possible dissection is Digital Subtraction Angiography (DSA) as it offers a very high diangostic performances of 97% for vascular injuries. However, DSA is an invasive method and not readily available in all institutions, hence CT scan combined with CT angiography would be more appropriate in identifying carotid dissection patient with severe trauma. "String signs", also known as "angiographic string sign" or "carotid string sign" indicating constrictions of the lumen, creates a string like appearance found in CT angiography indicating carotid artery dissection, ICA thrombosis and pre-occlusive atherosclerosis at the carotid bifurcation.

MRI with MRA could be an alternative for early detection of traumatic carotid artery detection specially due to blunt trauma and also it provides additional information about concomitant injuries such as infarction, brain injury or skull fractures as in our case also MRI report showed right MCA, ACA, PCA and left ACA, part of MCA and PCA infraction. The diagnostic quality of CT angiography and MRI has been demonstrated in this case report with several excellent images obtained. Hence both modalities can be used in side by side to aid management in carotid artery dissection [8,11,12].

Treatment of carotid artery dissection depends on the patient's symptoms. In general, asymptomatic patients with low grade dissections are typically treated conservatively with medical management and close imaging observation.

Anticoagulation remains the mainstay of medical treatment as studies have shown that ischemic event can be avoided in most cases if early diagnosis and treatment with anticoagulants is implemented but do not initiate anticoagulation in trauma patients without first ruling out intracranial hemorrhage (ICH) and extracranial sources of hemorrhage. Anticoagulation with antiplatelet (intravenous heparin followed by warfarin) has generally been accepted as adequate medical management for preventing thromboembolic complications [12,13].

Conclusion

Bilateral carotid artery dissection is rare but it can cause serious life threatening condition after high-speed motor vehicle accidents. CT angiography and MRI are the gold standard for early diagnosis. The initial clinical and radiological picture may be normal or there may be a delay in symptom presentation on first arrival; so it is crucial for emergency physicians to aggressively search for this injury in the presence of blunt cranial trauma. It is therefore challenging for emergency physicians to diagnose carotid dissection early on time, potentially delaying definitive medical management.

References

1. Park K, Park J, Hwang S, Im S, Shin W, Kim B. Vertebral Artery Dissection: Natural History, Clinical Features and Therapeutic Considerations. J Korean Neurosurg 2008;44:109-15.
2. Cleveland Clinic. Cervical (Carotid or Vertebral) Artery Dissection. 2017. https://my.clevelandclinic.org/health/diseases/16857-cervical-carotid-or-vertebral-artery-dissection. Accessed 12 April 2018.
3. Srivastava A, Bradley M, Kelly M. Bilateral Carotid Artery Dissection after High Impact Road Traffic Accident. Radiology Case. 2008;2(5):23-28.
4. Townend BS, Traves L, Crimmins D. Bilateral spontaneous carotid artery dissection. J. Clin. Neurosci. 2005;12(5):592-94.
5. Galyfos G, Filis K, Sigala F, Sianou A. Traumatic Carotid Artery Dissection: A Different Entity without Specific Guidelines. Vasc Spec Int. 2016;32(1):1-5. http://dx.doi.org/10.5758/vsi.2016.32.1.1
6. Akala A. Carotid Artery Dissection. FOAM EM RSS. 2014. http://www.foamem.com. Accessed 12 April 2018.

7. Weerakkody Y, Gaillard F, et al. Internal carotid artery dissection. Radiopaedia. https://radiopaedia.org. Accessed 18 April 2018.
8. Zohrabian D. Carotid Artery Dissection Clinical Presentation. Emergency Medicine in Drugs and Diseases column of Medscape. Dec. 28 2017. https://emedicine.medscape.com/article/757906-clinical. Accessed 2 April 2018.
9. Ballard JL, Bunt TJ, Fitzpatrick B, Malone JM. Bilateral traumatic internal carotid artery dissections: Case report. J Vasc Surg 1992;15:431-35.
10. Samra NS, Ravi AK, Johnson LW, Williams M. Traumatic Bilateral Carotid Artery Dissection. J La State Med Soc 2011;163:26-28.
11. Watridge CB, Muhlbauer MS, Lowery RD. Traumatic carotid artery dissection: diagnosis and treatment. Journal of Neurosurgery. 1989; 71:854-57.
12. Zohrabian D. Carotid Artery Dissection Treatment & Management. 2017. https://emedicine.medscape.com. Accessed 2 April 2018.
13. Zohrabian D. Carotid Artery Dissection. Emergency Medicine in Drugs and Diseases column of Medscape. Dec. 28 2017. https://emedicine.medscape.com/article/757906-overview. Accessed 2 April 2018.