Internal carotid artery dissection presenting with monocular superior quadrantanopsia

Jennifer Chu, M.D.[1] and Eroboghene Ubogu, M.D.[1]

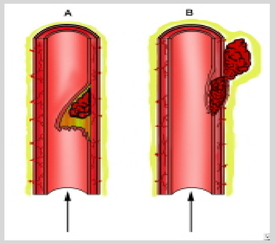
[1]Department of Neurology, Baylor College of Medicine, Houston, Texas

Objective

To gain familiarity with unique presentations of internal carotid artery dissection

Background

Cervical artery dissection is a major cause of non-atherosclerotic ischemic stroke in adults. The pathogenesis of arterial dissection is usually multi-factorial. Traumatic dissection is the result of either external mechanical injury or trivial trauma related to a movement or abrupt change in head position. While spontaneous dissections have no definitive precipitating factor; associations with connective tissue disorders, systemic hypertension, smoking, diabetes mellitus, a history of cerebral infarction, hyperlipidemia, cerebral and abdominal aortic aneurysms, use of oral contraceptives, or a family history of arterial dissection, have been described. Conventional catheter angiography is considered the gold standard for the diagnosis of arterial dissection, although noninvasive methods such as computed tomography angiography (CTA) and magnetic resonance angiography (MRA) are increasingly used. Early recognition of a cervical arterial dissection is essential, because prompt anticoagulant or anti-platelet therapy or endovascular intervention, or combination thereof reduces the risk for infarction, neurologic disability, and death. Internal carotid artery dissection presenting as transient monocular visual loss is uncommonly described.



Arterial dissection

(A) Tear and elevation of intima from wall of artery, resulting in luminal stenosis. This creates a blind pouch that predisposes to thrombus formation.

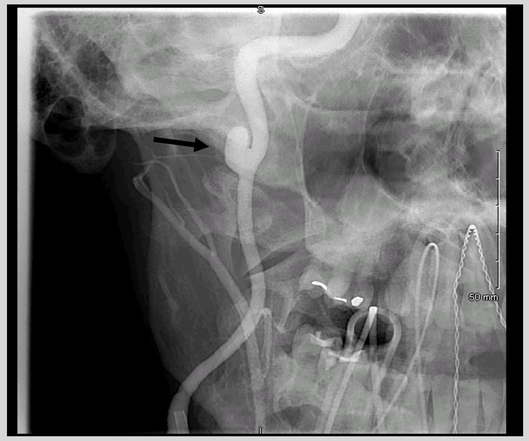
(B) Subadventitial dissection represents hemorrhage between media and adventitia which can extravasate through adventitia, resulting in pseudoaneurysm or fistula formation.

Design/Methods

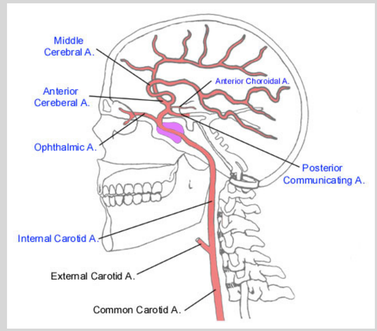
Case report

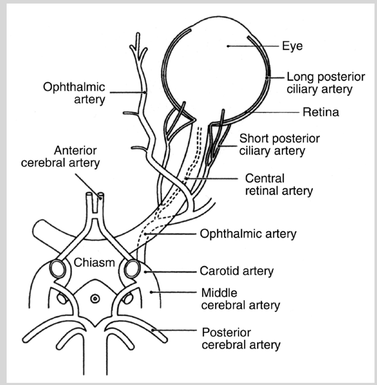
Results

A 65-year-old male with hyperlipidemia, prior myocardial infarction and history of ventricular tachycardia, presented with headache and intermittent vision loss involving the upper outer quadrant of his right eye. Patient was a marathon runner and trampoliner since his youth. He had been active, swimming several laps a day for exercise and was in good health. On the evening prior to his admission, he reported sudden transient episodes of seeing only parts of the room and that people's faces were blacked out in the upper outer quarter of his right eye. He also had a brief episode of left hand clumsiness while trying to button his sleeve followed by intermittent dull left sided headaches. Emergent head CT and CTA of the head and neck demonstrated a 12 mm wide neck pseudoaneurysm of the distal right cervical internal carotid artery. A heparin infusion was initiated. He started to see spots of flickering lights in his left eye afterwards. Catheter angiography confirmed the dissection and a right internal carotid artery stent was placed with subsequent resolution of the visual phenomena. The patient was likely initially experiencing symptoms secondary to emboli traveling upward from the right internal carotid artery dissection at the atlanto-occipital junction to the retinal artery causing transient ischemia to the inferior nasal retina, followed by possible right lateral frontal cortex ischemia causing left hand clumsiness.



Vascular Anatomy





Conclusions

An internal carotid artery dissection may present in varying ways dependent on the route of microemboli travel and associated ischemia. In this case, the patient experienced transient monocular superior temporal quadrantanopsia followed by a scintillating scotoma in the other eye. Angiography demonstrated a right internal carotid artery dissection with pseudoaneurysm, implying that retinal migraine was unlikely. Recurrent neurologic symptoms or deficits anatomically associated with a carotid dissection requires prompt detailed evaluation to determine the etiology for these symptoms/ deficits and to deduce whether endovascular intervention (e.g. stenting) is warranted.

Summary

1. Internal carotid artery dissections can present with transient ischemic attacks reflecting artery-to-artery embolization from the dissection site or hypoperfusion from arterial narrowing.
2. It is uncommon for these dissections to be associated with transient migrainous phenomena although there have been previous reports.
3. Clinical features, visual field testing, and radiographic testing (including angiography) should aid to distinguish retinal ischemia secondary to internal carotid artery dissection from retinal migraine
4. With recurrent or persistent neurologic symptoms after internal carotid artery dissection, anti-platelet, anticoagulation or endovascular intervention to reduce the likelihood of embolization and chronic stenosis should be considered even in cases with less widespread involvement.

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