Internal carotid artery dissection presenting with monocular superior quadrantanopsia

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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 combination thereof reduces the risk for infarction, neurologic disability, and death. Internal carotid artery dissection presenting as transient monocular visual loss is uncommonly described.

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 quadrantopsia 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.

References