• Review Article •

Ocular manifestations of internal carotid artery dissection

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Abstract

• Internal carotid artery dissection (ICAD) results from disruption of the intima of the arterial wall, and can lead to intrusion of blood into the arterial wall and form an intramural hematoma. The hematoma can compress the true lumen of the vessel, causing functional stenosis or occlusion. The classic triad signs of ICAD include pain in the ipsilateral neck, head and orbital regions; a (partial) Horner syndrome; and cerebral or retinal ischemia. However, not all ICAD patients present with this classic signs. In some cases, ocular manifestations are the initial (and sometimes the only) findings. We summarize the ocular manifestations associated with ICAD in 3 categories: visual symptoms, oculosympathetic palsy, and ocular motor nerve palsy.

• **KEYWORDS:** internal carotid artery dissection; ocular manifestation; oculosympathetic palsy; ocular motor nerve palsy; visual symptoms

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INTRODUCTION

The cervical arteries comprise bilateral internal carotid arteries and vertebral artery. Any damage or thrombosis to these vessels, including dissection, can lead to complications, such as cerebral ischemia, stroke, blindness, or death. Unfortunately, given its rarity and nonspecific symptoms, cervical artery dissections is difficult to make diagnosis. Internal carotid arteries carry blood flow to bilateral eyes, ophthalmologic complaints are frequently the initial (and sometimes the only) presentation of internal carotid artery dissection (ICAD)[1].

Pathophysiology ICAD is one type of cervical artery intimal wall disruption, leading to intramural hematoma formation[2]. The hematoma can expand and compress the true lumen of the vessel, causing functional stenosis or occlusion. The decreased perfusion leads to hemodynamic or embolic ischemia of brain and retina. ICAD are classified as either traumatic or spontaneous.

The classic triad of ICAD consists of pain in the ipsilateral neck, head and orbital regions, (partial) Horner syndrome, and cerebral or retinal ischemia[2]. However, not all ICAD patients present with this triad. In recent years, research studies have reported that ICAD can cause ischemic optic neuropathy (ION), ophthalmic artery occlusion (OIS), and cranial nerve (CN) palsy, which will present primarily as visual complaints and require ophthalmologic work-up.

Epidemiology ICAD has an incidence of 2.5 to 3.0 cases/100 000 per year[3] and it is one of the most important causes in ischemic stroke in young adults[4], usually occurring in 30 to 50 year old patients[5]. It is responsible for up to 25% of ischemic strokes[6]. It is a major cause of cerebrovascular injuries in children, occurring in 20% of cases of pediatric acute ischemic stroke[7]. The pathophysiology of ICAD is poorly understood, but patients with connective tissue diseases and concomitant arterial anomalies such as fibromuscular dysplasia are at higher risk[8].

Method of Examination and Diagnosis Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) contributes to the diagnosis of ICAD, with the evidence of delayed filling of ophthalmic artery and middle cerebral artery or the exact location of dissection; Hypoperfusion in either middle cerebral artery can be seen with transcranial doppler.

Fundus fluorescein angiography (FFA) and indocyanine green angiography (ICGA) are often used to observe retina and choroid. ION is confirmed with dilated ophthalmoscopy (optic disc edema or subsequently optic disc atrophy) or visual field

(optic nerve related visual field defect); FFA may also present delayed filling of optic disc. Central retinal artery occlusion (CRAO) is diagnosed with dilated ophthalmoscopy (severe retinal ischemia and cherry red spot) or FFA (delayed retinal filling and hypoperfusion or location of emboli). Branch retinal artery occlusion (BRAO) is diagnosed with dilated ophthalmoscopy [retinal whitening and associated edema along the distribution of either branch of central retinal artery (CRA)] or FFA (focal retinal arterial blockage or location of emboli). OIS is a sum of ocular hypoperfusion, which is diagnosed with the evidence of FFA (retina hypoperfusion) and ICGA (choroid hypoperfusion), ophthalmoscopy may discover retinal artery narrowing, retinal hemorrhage, optic disc swelling, severe retina edema, *etc*. Slit-lamp examination may also disclose anterior segment ischemia, aqueous flare, iridocyclitis, neovascular glaucoma, *etc*.

Horner syndrome is characterized by miosis, upper eyelid ptosis and facial anhidrosis on the affected side. Miosis is examined especially in darkness and greater anisocoria support the diagnosis of miosis. Upper eyelid ptosis is examined with location of upper eyelid and width of palpebral fissure.

Ocular motility examination is used to check any ocular motor nerve palsy. Complete CN III palsy is characterized by limitation of elevation, depression and adduction, accompanied by ipsilateral dilated pupil and ptosis. CN IV palsy is characterized by vertical diplopia and superior oblique underaction. CN VI palsy is characterized by esotropia and ipsilateral limitation of abduction.

Ocular Presentation Thus far, only a few articles have discussed the spectrum of ophthalmologic involvement in ICAD and most of them have been case limited to case reports. The present study reviewed all the existing literature and summarizes the ophthalmologic symptoms and signs associated with ICAD. We divided these symptoms into 3 categories: visual symptoms, oculosympathetic palsies, and ocular motor nerve palsy.

Visual symptoms The visual symptoms caused by ICAD can range from transient monocular visual loss (TMVL)[9] to permanent losses, resulting from ION[10], CRAO/BRAO[11], or OIS[12].

TMVL, also mentioned as amaurosis fugax, is characterized by prompt visual loss that may last for a few minutes and then disappear. There are four mechanisms that may produce TMVL: embolic, hypoperfusion, angiospasm, and idiopathic[9,13].

TMVL has been reported in 6% to 38% of patients with ICAD[9]. Patients also described their TMVL as "moving candles in the night" or "bright sparkling water drops"[14]. Fluctuating TMVL may due to unstable hemodynamics caused by ICAD and might be evoked by posture changes. Some patients complained of visual loss when they were trying to sit up, which might be caused by a fall of systemic blood pressure worsening the hypoperfusion[15].

Biousse *et al*[10] reported on 146 patients with ICAD, and 41 of them had one or multiple episodes of TMVL, 31 had episodes coincident with ipsilateral facial, head, or cervical pain and 13 had them with Horner syndrome. Additionally, 23 of the patients described their TMVL as "scintillations", "flashing lights", "bright spots" and "scintillating scotoma". In 14 of the 23 patients, these symptoms were related to postural changes or exposure to bright lights. Moreover, 14 of these patients experienced nonreversible cerebral infarctions within a mean of 5.7d after the first episode of transient visual loss.

Abed *et al*[13] reported a 38-year-old woman with right ICAD presenting a transient loss of vision in the right eye with a preceding right-sided neck pain which emphasized the setting of TMVL and preceding neck pain suggesting the possibility of ICAD. Kim *et al*[9] published a 33-year-old woman with left ICAD presenting with transient monocular blindness in left eye following acute-onset left headache. Monocular blindness developed episodically only in the upright position and was relieved quickly by lying down but still a relative afferent pupillary defect (RAPD) was noticed. Both signs disappeared after treatment with carotid stent. This case suggested even in supine position, hemodynamics was still unstable caused by ICAD.

ION is an infarction of the optic nerve, and is the most frequent cause of acute optic neuropathy in patients older than 50y. ION was defined as a sudden onset of visual loss in the eye, with no presence of retinal and vascular abnormalities in the fundus, and with a sectorial visual field defect. ION according to the ischemic damage location can be classified as optic nerve head ION-anterior ischemic optic neuropathy (AION) and retrobulbar ION-posterior ischemic optic neuropathy (PION). A diagnosis of AION is made if the initial fundus showed disc edema[16] and PION if there is an acute visual loss without initial disc edema but with subsequently optic disc atrophy. ION, according to etiology, can be classified into arterial ION caused by giant cell arteritis and nonarterial ION, occupying 90% of all ION. ICAD is rarely a reason for ION.

According to the literature, in 4% of cases, ICAD occurs with ION[17]. Biousse *et al*[18] summarized the clinical features of ION caused by ICAD differing from those of nonarterial anterior ION: 1) the mean age is younger in ICAD (44y, range 33-51y); 2) 5 patients had severe ipsilateral orbital pain which is a prominent symptom of ICAD; 3) 5 patients experienced previous recurrent episodes of transient monocular blindness lasting 3-8d (mean 5.3d) before permanent visual loss caused by ION; and 4) Horner syndrome is common in ICAD associated with ION. Finally, ION with ICAD has a

normal erythrocyte sedimentation rate (ESR), and it is not associated with diseases such as hypertension, diabetes, or hyperlipidemia.

Biousse *et al*[10] reported 146 patients with ICAD and 4/146 (2.74%) were cases of ION (2 of AION and 2 of PION). ION occurred after a mean interval of 5.25d (range 3-8d) after the presenting symptoms of ICAD (1 patient with headache, 2 patients with TMVL and 1 patient with hemispheric transient ischemic attack). Associated with ION, 1 patient had Horner syndrome, 2 patients had a severe ipsilateral headache and orbital pain. Kerty[15] reported 28 patients with ICAD and found 1/28 had ION, combining with Horner syndrome. Tsai and Sun[16] reported a 55-year-old man with right ICAD complaining of right eye blindness [with no light perception (NLP)], and with normal ophthalmoscopy but a depigmentation patch in the papillomacular bundle. After a month the patient developed proptosis, ocular motility limitation, and conjunctival congestion. Koch *et al*[19] and Correa and Martinez[17] reported cases where the patients suffered traumatic ICAD. Both patients presented with visual loss and RAPD in the ipsilateral eye, with normal retinal and optic disc findings. One of the patients developed optic disc pallor after 3mo.

CRA is a branch of the ophthalmic artery and is an end-artery[20]. Its occlusion will cause CRAO or BRAO. ICAD is a rare reason but cause severe consequence of retinal artery occlusion resulting in a visual loss to light perception (LP) or NLP, which is common to cause a permenent blindness even in a short time.

CRAO may result from a sudden hypoperfusion in the ophthalmic artery caused by ICAD, which shows a hypoperfusion in both retina and choroid in FFA. It is different from CRAO caused by arterial embolization in elderly people with systemic diseases. All patients with CRAO should be questioned about ipsilateral head and neck pain or other extraocular symptoms[9]. And all patients with CRAO and ipsilateral pain of head and neck should have careful blood pressure control to prevent aggravation of the ocular injury.

Mokhtari *et al*[11] reported 2 women with ICAD suddenly having visual losses. During the preceding week, one complained about ipsilateral neck pain, a TMVL, and pulsatile tinnitus, whereas the other complained about an ipsilateral headache and tinnitus. Both patients had affected eyes with visual acuity of LP, severe retinal edema, cherry-red spots, and one of them having an RAPD.

McDonough *et al*[21] reported a case of a 34-year-old man with left ICAD complaining of two episodes of monocular transient visual loss in the left eye. Left visual acuity was 20/25 with an RAPD. Confrontation fields and ophthalmoscopy discovered a BRAO. There was no combining anisocoric, ptosis or ocular motor dysfunction present. FFA disclosed nothing but immediately after the FFA, the patient suddenly had a symptomatic hypotensive bradycardic episode, causing visual acuity of left eye to deteriorate to NLP.

OIS is defined for the sum of ocular symptoms and signs that may accompany carotid artery occlusive or stenotic disease. OIS is caused by ocular hypoperfusion due to stenosis or occlusion of the common or internal carotid arteries[22]. OIS is a rare condition, but its complications may lead to irreversible vision loss.

ICAD is one type of carotid artery stenosis that leads to OIS. It mostly affects the ophthalmic artery, central retina artery, and ciliary artery and causes ischemic symptoms of the eye[23]. Affected patients may experience a variety of manifestations including visiual loss, rubeosis of the iris, neovascular glaucoma, normal-tension glaucoma, increased introcular pressure (IOP), iridocyclitis, asymmetric cataracts, atrophy of the iris and sluggish reaction to light, dilated but not tortuous veins, intraretinal hemorrhages, vitreous hemorrhages, macular edema, optic disc atrophy, microaneurysms, neovascularization of the optic disk and retina, and collateral vessels at the optic nerve head[22,24]. There is also a term “orbital infarction syndrome” that denotes ischemic injury to the globe and ocular adnexa, typically presenting with external ophthalmoplegia, proptosis, lid edema, with retinal and choroidal infarction[25]. Patients having retinopathy with marked asymmetry should be urgently evaluated because approximately 20% of these patients will have hemodynamically significant carotid artery stenoses. Once OIS is suspected, the presence of ICAD should be considered.

Duker *et al*[12] and Takaki *et al*[26] reported patient with ICAD who had sudden visual losses as the initial sign, after which ophthalmoscopy showed retinal artery narrowing, with scattered blot hemorrhages in one patient and optic disc swelling and severe retina edema in the other. One patient also had changes in the iris consistent with neovascularization aqueous flare.

Oculosympathetic palsy (Horner syndrome) Horner syndrome is caused by an interruption in any point of the oculosympathetic pathway from the central nervous system to the eyeball. When the syndrome is complete, affected patients exhibit a triad consisting in miosis (anisocoria greater in darkness), upper eyelid ptosis and facial anhidrosis on the affected side[27], occasional ipsilateral conjunctival injection, and apparent enophthalmos[28]. A partial Horner syndrome can consist of miosis and ptosis. Painful Horner syndrome is defined as Horner syndrome coincident with ipsilateral pain at neck, head and the orbital origin. The gold standard diagnostic test is anisocoria of >1 mm 45min after instilling two drops of 4% or 10% cocaine into each eye[29].

There are many possible etiologies along the three-neuron pathway to cause Horner syndrome. However, a few potentially life threatening causes including ICAD must always be ruled out. In ICAD, eccentric expansion of the bulged arterial wall may cause Horner syndrome[30]. And a painful Horner syndrome occurred in 91% of all Horner syndrome related to ICAD[31]. As many as 40%-58% of patients with ICAD will develop an ipsilateral Horner syndrome[31-32]. Once a patient complains about a sudden onset of ptosis, the ophthamologist should pay attention to their pupil diameters and ask about ipsilateral neck and head pain. If necessary, a cocaine test should be carried out and a neurology consultation obtained.

Lyrer *et al*[30] analyzed 496 patients with ICAD. Horner syndrome was present in 191 patients (38.5%), and 89 of the 191 had cervical pain, whereas 144 of the 191 had headaches. Horner syndrome (+) ICAD patients presented less often with stroke, transient ischemic attacks (TIA), bilateral or occlusive dissections, or severe strokes, and had a better functional 3-month outcome. They also were less likely to have new strokes than Horner syndrome (-) ICAD patients. Horner syndrome was defined as presence of pupillary miosis and blepharoptosis with or without facial anhidrosis.

Biousse *et al*[10] studied 146 patients with ICAD and 65 of them had unilateral and ipsilaterapainful Horner syndrome, involving the face, head, or lateral part of the neck. Isolated painful Horner syndrome was found in 32 patients, who had no ocular or cerebral ischemic symptoms. The other 33 patients had transient or permanent ocular or ischemic symptoms, whereas 13 of the 65 patients had irrevesible cerebral infarctions within 6.7d after the onset of Horner syndrome.

Kerty[15] reported the ophthalmic features that occurred in 28 patients with extracranial ICAD; 23 of them developed oculosympathetic paresis (the most frequent sign) while 2 presented with Horner syndrome as the only indication. Horner syndrome in ICAD patients is postganglionic and is caused by involvement of small nutritive vessels. Orssaud *et al*[33] and Villalba Martinez *et al*[34] reported a case of patient presenting with unilateral non-reactive enlargement of a pupil. MRI disclosed ICAD, and Horner syndrome occurred, suggesting that the initial episode of mydriasis was caused by an oculosympathetic spasm, a rare dysfunction that occurs when the sympathetic nerve is irritated. These spasms have equal value as HS for the diagnosis of ICAD.

Ocular motor nerve palsy Single or multiple cranial nerve palsies may occur as isolated phenomena but are often noted in conjunction with other manifestations of ICAD. Cranial nerves dysfunction may be involved in 6% to 12% of patients with ICAD and the affected ocular motor nerves are mostly CN III, CN IV and CN VI[35].

CN involvement can be explained by mechanical compression, stretching of the nerve by expanded arteries, or compromise of nutrient arterial flow to the related nerve[14,36]. Isolated CN palsies may not be recognized as a clinical presentation of extracranial ICAD[35], and such patients should be evaluated by further tests especially when there is associated ipsilateral headache or facial pain.

Mokri *et al*[36] studied 190 patients with ICAD, and 23 of these patients had CN palsies. Of these, 7 had CN V involvement, 2 had CN III involvement but no Horner syndrome, 2 others had CN VI involvement (1 of whom had Horner syndrome), and 1 had CN IV involvement. All 5 of these patients had headaches, and 4 in these 5 patients had headaches and ocular motor palsies that were the major manifestations of ICAD. One patient had ipsilateral CN V involvement, and 2 had additional oculosympathetic palsies. A syndrome of hemicrania and CN V palsy and Horner syndrome was noted in 4 patients.

Schievink *et al*[37] reported a study of 155 patients with ICAD and found 4 patients with oculomotor palsy. Of these, 1 had CN VI involvement, 2 had CN III involvement, and 1 had CN IV involvement. Also, 3 of these patients had ipsilateral and 1 had bilateral headache or facial pain, and 3 had oculosympathetic palsies. None of the patients had any cerebral or retinal ischemic symptoms. Biousse *et al*[10] reported 146 patients with ICAD, and reported only 1 patient with transient CN VI palsy.

CONCLUSION

The above ophthalmologic manifestations are all associated with ICAD. These symptoms or signs are frequently associated with and are often the presenting features in ICAD, which should be systemically studied by the ophthalmologist who is the first to examine and diagnose the patient. But at this point, underdiagnoses and misdiagnoses still occur. It is important to recognize and treat individuals who present with this condition because cerebral and retinal ischemic strokes can occur in 1/3 of these patients after a mean of 5.7d[10] following initial development of the ocular symptoms. Once the typical signs are discovered, the treating ophthalmologist should recognize the ocular manifestations may be a harbinger of ICAD and therefore refer these patients to neurology emergently, because their occurrence should prompt investigations to diagnose ICAD and begin early treatment if indicated.

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