Ophthalmic masquerades of the atherosclerotic carotids

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Patients with carotid atherosclerosis can present with ophthalmic symptoms. These symptoms and signs can be due to retinal emboli, hypoperfusion of the retina and choroid, opening up of collateral channels, or chronic hypoperfusion of the globe (ocular ischemic syndrome). These pathological mechanisms can produce many interesting signs and a careful history can bring out important past symptoms pointing toward the carotid as the source of the patient's presenting symptom. Such patients are at high risk for an ischemic stroke, especially in the subsequent few days following their first acute symptom. It is important for clinicians to be familiar with these ophthalmic symptoms and signs caused by carotid atherosclerosis for making an early diagnosis and to take appropriate measures to prevent a stroke. This review elaborates the clinical features, importance, and implications of various ophthalmic symptoms and signs resulting from atherosclerotic carotid artery disease.

Key words: Carotid artery disease, ocular ischemic syndrome, retinal emboli

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Prevalence of asymptomatic extracranial carotid artery disease (>50% stenosis) ranges from 1.5 to 9%in population-based studies,[1-4] with a higher incidence in the elderly.[3,5] Symptoms in the ipsilateral eye can often herald the presence of asymptomatic atherosclerotic carotid disease. Such patients are at a high risk for stroke. The annual stroke risk ranges between 2.0% and 2.8%[6-10] and can reach 8.4% in the presence of higher grades ofipsilateral carotid stenosis.[8,11,12] Bruno[13] noted a 10-fold increase in the annual rate of stroke in men with retinal emboli independent of blood pressure and other vascular risk factors. It is also important to note that the risk of stroke following a transient ischemic attack is maximum in the subsequent 48 h.[14-17] The EXPRESS[18] study and the SOS–TIA study[19] have shown thatearly intervention in such patients is needed to prevent stroke. It is therefore important for the first medical contact to be aware of the importance and implications of the ophthalmic symptoms caused by atherosclerotic carotid artery disease to prevent an impending catastrophic stroke.

Atherosclerosis affects mainly the site of bifurcation of the common carotid artery. The posterior walls of the arteries tend to be more involved. The pathogenesis of atherosclerosis is a decade-long process which results in the formation of the atherosclerotic plaque in the tunica intima.

Falk[20] divided the atherosclerotic plaques into two types, a hard collagen-rich plaque, with barely detectable lipid, and a soft plaque, with a prominent pool of lipid. Soft plaque is more vulnerable because itsthin fibrous cap can rupture.

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When an atherosclerotic plaque ruptures, the contents of the plaque (collagen, lipids, and smooth muscle cells) protrude into the vascular lumen and come in contact with platelets and blood cells. This leads to activation of platelets and the coagulation cascade, resulting in thrombus formation on the atherosclerotic plaque. Such thrombi may cause distal ischemia by various mechanisms as follows: (1) The newly formed thrombus can cause a sudden marked reduction in the vessel diameter, resulting in decreaseddistalperfusion. (2) Apart of the thrombuscan break off and travel distally (thromboembolism) into the ophthalmic artery orenter the cerebral circulation. (3) The thrombus can grow in size and propagate distally and cause occlusion of a vessel. (4) The thrombus can become organized andget incorporated into the diseased vessel, making its lumen narrow and causing chronic distal ischemia.

When the internal carotid artery (ICA) is obstructed, the blood flow to the brain is compensated by the following: (1) collateral circulation from the circle of Willis which brings blood form the contralateral ICA through the anterior communicating artery (ACom) into the ipsilateral middle carotid artery (MCA) and (2) collateral channels openup between the terminal external carotid artery (ECA) branches and the terminal branches of the ophthalmic artery. Through these channels, there is retrograde flow via the ophthalmic artery to ICA. Thus, ophthalmologic symptoms and signs caused by carotid atherosclerotic disease can occur due to either of the following: (1) retinal emboli arising from the atherosclerotic plaque; (2) hypoperfusion of the retinal and choroid due to the ICA stenosis; (3) opening up of collateral channels; and (4) ocular ischemic syndrome (OIS; venous stasis retinopathy).

In this article, we describe the various ophthalmic symptoms and important physical signs caused by carotid atherosclerosis.

Symptoms and Signs Due to Retinal Emboli

For unclear reasons, an embolus originating in the carotid artery is more likely to enter the ophthalmic artery, which is

the first branch, than the cerebral vessels.[21-23] Once an embolus from the carotid arteryenters the ophthalmic artery, it can cause any of these four conditions: (1) transient monocular visual loss (TMVL); (2) central retinal artery occlusion (CRAO); (3) central retinal artery branch occlusions (CRABO); and (4) anterior ischemic optic neuropathy (AION).

Transient Monocular Visual Loss

It is also called transient monocular blindness (TMB) and amaurosis fugax. The classical description of TMVL caused by a retinal embolus is of a "curtain" or "shade" descending over the vision.[24] The possible explanation for this is that the arteries which supply the inferior retina are more tortuous, have smaller diameters, and take a longer course, hence the inferior segments of the retina are more prone to ischemia.[25] Rarely, the visual loss may be associated with scintillations or the sensation of color.[26] Episodes generally last from seconds to minutes, rarely more than 15 min.[27,28]

By the time a patient with a TMVL seeks medical help, the symptoms may have resolved completely and the neurological and ophthalmologic examination can be normal. If the patient is seen during an attack of TMVL, the pupil will be amaurotic and the retinal vessels will appear collapsed. Embolic particles may be seen in the ipsilateral eye in up to one-fifth of patients with carotid strokes.[29] For a proper examination, the pupils should be dilated to see the peripheral retina. Different types of microemboli can be seen in the retinal arterioles during or between attacks of TMVL.[30] Hollenhorst plaques are cholesterol crystals from an eroded atheroma and can be seen as irregular, bright glistening retractile, orange-yellow plaques. Platelet-fibrin emboli from a carotid thrombus can be seen as grayish-white nonrefractile plugs. Rarely, ovoid nonrefractile grayish-white calcific emboli can also be seen. Fluorescein angiography can be helpful if the ophthalmoscopic examination is negative. It canshow leakage of dye in regions of arteries damaged by crystal emboli.

In patients with TMVL, the clinical features most predictive of high-grade (>70%) carotid stenosis included rapid onset of symptoms, altitudinal pattern of onset or resolution, and symptom duration of 1–10 min.[28]

Central Retinal Artery Occlusion

When an embolus enters the CRA, it tends to lodge at the lamina cribrosa which is the narrowest part of this artery. These patients present with sudden, painless visual loss and anabnormal pupillary reaction [afferent pupillary defect (APD)] can appear within seconds. Subsequently, there will be edema of the superficial retina at the posterior pole, giving ita yellow-white appearance. In the region of the foveola, the retina is extremely thin, allowing the underlying choroid to be seen as the "cherry-red spot."[31]

Slowing of the retinal circulation with segmentation of the blood column and slow streaming of flow in the veinscauses box carappearance to be seen.[32]

Central Retinal Artery Branch Occlusions

The CRA is an end artery with no anastomotic connections. It divides into four major branches supplying non-overlapping sectors of the retina. When an embolus enters and blocks it, the area of retina that they supply infarcts. CRABO are more often attributable to severe ICA disease than CRAOs.[33-35] The visual loss will be focal and corresponds to the portion of the retina that is ischemic. CRABO may affect only the upper, lower, nasal, or temporal portion,[26,36,37] andmay present assmall central or paracentral scotomas. On ophthalmoscopy, the portion of the retina will have a ground glass like appearance due to ischemic edema. Fluorescein angiography can pinpoint the occlusion.

Anterior Ischemic Optic Neuropathy

Unlike the above conditions, AION is caused by hypoperfusion of the optic nerve head due to ischemia to the anterior part of the optic nerve. Rarely, an ICA occlusion with thrombus extending to the carotid siphon can block the ophthalmic artery causing AION.[27] Painless monocular visual loss is the usual symptom. If the ischemia involves the anterior part of the optic nerve, the examination will show edema of the optic disc and small splinter hemorrhages at the disc margins. If the ischemia affects the posterior part of the optic nerve, then the fundus examination can be normal.

Symptoms and Signs Due to Hypoperfusion of the Retina and Choroid

ICA narrowing can cause hypoperfusion of the retina and the choroid. This can result in some interesting symptoms. Patients can experience an iris diaphragm type of TMVL wheretheconstriction of the visual fields start form theperiphery and move towards the center.[24] This TMVL of hypoperfusion can occur in many specific clinical settings.

1. **Bright light amaurosis**

   The TMVL is brought on after exposure to bright light (retinal claudication). The symptom can persist for seconds to hours after the light exposure.[29,38,39]

   The mechanism is severe retinal ischemia causing a delay in a regeneration of visual pigments in the pigment epithelial layer of the retina. With bilateral carotid disease, this can present as asymmetrical visual loss occurring with bright light exposure.[40]

2. **Position-induced TMVL**

   Episodes of TMVL may be induced by changes in posture or neck positio n that temporarily reduce blood flow through the already stenosed artery.[29]

3. **Postprandial transient visual loss[41]**

   The TMVL following a heavy meal can be secondary to shunting of blood tothe mesenteric arteries.

4. **Exercise-induced TMVL[42]**

5. **TMVL followingfacialheating[43]**

   Theprobable mechanism in this is thought to be diversion of blood to a dilated external carotid vascular bed.

In such suspected cases of ICA narrowing, a carotid or ocular bruit may be heard on auscultation. A carotid bruit due to carotid stenosis may be heard in the following locations: (1) common carotid region – base of neck; (2) carotid bifurcation region – level of the superior thyroid cartilage; and (3) ICA region – angle of jaw. It should be recalled that cardiac murmurs

can radiate to the neck vessels. So, a careful cardiac auscultation should be done, and if any cardiac murmurs are heard, they should be followed up into the neck. If there is an associated carotid bruit, then a change in pitch and/or intensity can be noted.

A carotid bruit is usually heard at the origin of the ICAwhen the stenosis is ≥70% of the lumen diameter and/or the residual lumen diameter is ≤1.5 mm.[44]

It should be noted that a carotid bruit may be heard often in asymptomatic patients, but if heard in the setting of a suspected carotid stenosis, it warrants further investigation.

Occasionally, a carotid bruit may be absent in a patient with a "tight" stenosis. This may be due to a "slow-flow" state.[45]

The orbit can be auscultatedwith the bell of the stethoscopeto look for ocular bruits. The patient should be either lying or sitting. Patients should be instructed to open the opposite eye to reduce blinking noise. The opposite eye should also be fixated to reduce the movement of the globe during auscultation. Both the sides should be auscultated. In carotid stenosis, an ocular bruit may be heard on the contralateral side. The increased flow through the patent contralateral ICA may be the cause.[45,46]

It should be borne in mind that just palpation of the carotid arteries may not be adequate. On palpation, only the common carotid artery pulse can be felt and an ICA occlusion which is posteriorly placed after the bifurcation cannot be ruled out. The left carotid artery is usually posterior and deeper than the right, so there may be a mild asymmetry, normallythe right being more prominent than the left.

Symptoms and Signs Due to Opening Up of Collateral Channels

Theopening up of ECA–ICAcollateral channels via the ophthalmic artery can cause many signs and symptoms which can be picked up by a careful vascular examination [Fig. 1].

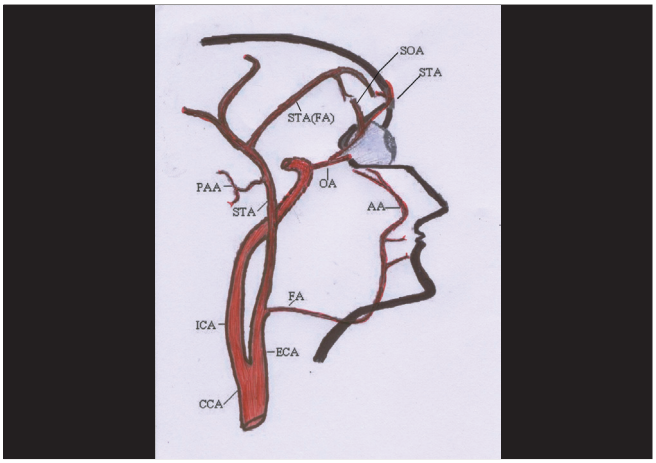

Figure 1: Important internal and external carotid arterial connections around the orbit CCA (Common carotid artery) ECA (external carotid artery) ICA (internal carotid artery) FA(facial artery) AA (angular artery) STA (superficial temporal artery) PAA (Preauricular artery) STA (FA) (Frontal artery–branch of superficial temporal artery)OA (ophthalmic artery) SOA (supraorbital artery)STA (supratrochlear artery)

1. **Delay/asymmetry of ECA pulses:** If the common carotid artery is blocked, then the branches of theipsilateral ECA, the facial artery (felt along the angle of the lower jaw), the preauricular artery (felt anterior to the ear), and the superficial temporal artery (felt in the temporal region) will be diminished/delayed. The face on that side of the block will feel cool to touch. Both the sides should be felt simultaneously to detect this asymmetry.
2. **ABC (angular, brow, cheek) pulses:** When the ICA is occluded before its ophthalmic artery branch, there will be increased flow in the ECA branches to open up collateral vessels around the orbit. This augmented flow can be felt by palpation atthe inner **a**ngle of the eye, the brow, and cheek.[47]
3. **Frontal artery sign:** Normally, there is antegrade flow of blood form the ICA to the ophthalmic artery and then to its supratrochlear and supraorbital (frontal) branches. In severe carotid stenosis, blood formsuperficial temporal artery (ECA) flows retrograde through supratrochlear and supraorbital branches into the ophthalmic artery and enters the ICA system.[48,49] Normally, if the supratrochlear and supraorbital (frontal) pulses are seen on the forehead, they can be blocked by applying pressure at the brow (as they are being filled antegrade from the ophthalmic artery). In cases of severe carotid stenosis where these arterieshave retrograde flow into the ophthalmic artery, applying pressure on the brow will not diminish them, but pressure on the superficial temporal artery on the forehead will obliterate them. This is called the frontal artery sign.[50]
4. **Dilated episcleral arteries** may be seen with ipsilateral carotid artery occlusion.[51] This is caused by ECA orbital collateral channels which cause retrograde filling of the ophthalmic arteries.
5. **Olivarius' external carotid sign:** Increased prominence and pulsation of the branches of the superficial temporal artery, especially the frontal, in occlusion of the ipsilateral ICA.[52] Rarely, a subjective bruit over the ipsilateral ear may be the complaint. Occlusion of ipsilateral ICA can cause compensatory hyperperfusion of the ECA. This can cause a subjective bruit over the ipsilateral ear (due to increased flow across the temporal artery). Digital compression on the temporal artery can eliminate this bruit.[53]

Symptoms and Signs Due to Chronicois (Venous Stasis Retinopathy, Hypotensive Retinopathy)

Initially, this condition was called "venous stasis retinopathy." Since mild central retinal vein obstruction was also designated as "venous stasis retinopathy," the term "ocular ischemic syndrome" is now used for changes in the eyes caused by chronic ipsilateral carotid stenosis.[54,55]

It is postulated that the decreased vascular perfusion caused by carotid stenosisresults in tissue hypoxia and increased ocular ischemia, leading to neovascularization.[56,57] Reported frequencies range between 5% and 21% in series of patients with carotid artery stenosis or occlusion.[58,59] The patient with OIS can present with pain or visual loss. In 90% of cases, visual loss occurs gradually over a period of weeks

to months;in rare instances, it may be abrupt.[60] Sudden visual loss may occur when the Intra ocular pressure (IOP) suddenly exceeds the perfusion pressure within the CRA, causing a CRAO. The possible mechanisms for pain in OIS are ischemia to the globe, increased intraocular pressure secondary to neovascular glaucoma, or dural ischemia. It is usually a dull ache over the brow and eyes. This pain usually is relieved on assuming a supine position.[61,62]

An ophthalmic examination may show rubeosis iridis (iris neovascularization) in two-third of patients.[63] It is interesting to note that in chronic OIS, a raised IOP is rare even if the anterior chamber angle is closed because of ciliary body hypoperfusion and decreased aqueous production. Other findings in this condition could be mature cataracts, especially if the ICA occlusion is longstanding. Retinal examination can show pale, narrow, and thread-like retinal arteries, and the retinal veins can appear dilated with occasional beading.

Retinal hemorrhages (dot and blot) and microaneurysms can be seen mainly in the midperiphery, and vitreous or subhyaloid hemorrhage can be seen following rupture of the new vessels. Varying degrees of optic nerve and retinal revascularization and retinal cherry-red spot can be seen.[63,64]

It should be noted that most of the changes caused by OIS can be seen with diabetes and hypertension. Marked asymmetry of findings (more marked in the side of the occluded carotid) and disproportion in the extent of anterior and posterior segment pathology can be a clue that the changes are more likely due to carotid narrowing.

Signs of Stroke

It is very important not to miss out signs and symptoms of a stroke in patients presenting with visual symptoms and known or suspected carotid stenosis. Very often being preoccupied with his/her visual symptoms, the patient may not have noted subtle symptoms of the stroke.

In a busy clinical setting, the FAST approach[65,66] is an easy way to recognize and remember the most common signs of a stroke. Face – Check the patient's face for any facial droop or drooping of the eyes. Arms – Ask the patient to liftup both his arms and to keep it horizontally for 10 seconds, with the palms facing the ceiling and eyes closed. The arm on the affected side will start to drift down in 10 seconds if there is subtle weakness. Speech – Look for any slurring of speech and whether the patient is able to understand your speech. Time – If any of these signs are present, do not waste time. The patient may be having a stroke and should be rushed to a hospital.

It is important to pick up these early signs of stroke since the patient may be a candidate for thrombolytic therapy if still within the window period.

Conclusion

Often ophthalmologic symptoms and findings may be the only manifestation in patients with carotid atherosclerotic disease. It is important for ophthalmologists to be familiar with these symptoms and signs. So, high-risk patients for an ischemic stroke can be picked up and referred for early intervention.

References

1. Willeit J, Kiechl S. Prevalence of risk factors of asymptomatic extracranial carotid artery atherosclerosis. A population based study. Arterioscler Thromb Vasc Biol 1993; 13:661-8.
2. Prati P, Vanuzzo D, Casaroli M, Di Chiara A, De Biasi F, Feruglio GA, *et al.* Prevalence and determinants of carotid atherosclerosis in a general population. Stroke 1992; 23:1705-11.
3. O'Leary DH, Polak JF, Kronmal RA, Kittner SJ, Bond MG, Wolfson SK, *et al.* Distribution and correlates of sonographically detected carotid artery disease in the Cardiovascular Health Study. The CHS Collaborative Research Group. Stroke 1992; 23:1752-60.
4. Pujia A, Rubba P, Spencer MP. Prevalence of extracranial carotid artery disease detectable by echo- Doppler in an elderly population. Stroke 1992; 23:818-22.
5. Fine-Edelstein JS, Wolf PA, O'Leary DH, Poehlman DH, Belanger AJ, Kase CS *et al.* Precursors of extracranial carotid atherosclerosis in the Framingham Study. Neurology 1994; 44:1046-50.
6. Hurwitz BJ, Heyma A, Wilkinson WE. Comparison of amaurosis fugax and transient cerebral ischemia: A prospective clinical and arteriographic study. Ann Neurol 1985; 18:698-704.
7. Donders RC. Clinical features of transient monocular blindness and the likelihood of atherosclerotic lesions of the internal carotid artery. J Neurol Neurosurg Psychiatry 2001; 71:247-9.
8. Poole CJ, Ross Russell RW. Mortality and stroke after amaurosis fugax. J Neurol Neurosurg Psychiatry 1985; 48:902-5.
9. Wilterdink JL, Easton JD. Vascular event rates in patients with atherosclerotic cerebrovascular disease. Arch Neurol 1992; 49:857-63.
10. Hankey GJ, Slattery JM, Warlow CP. The prognosis of hospital referred transient ischaemic attacks. J Neurol Neurosurg Psychiatry 1991; 54:793-802.
11. Benavente O, Eliasziw M, Streifler JY. Prognosis aftertransientmonocular blindness associated with carotid artery stenosis. N Engl J Med 2001; 345:1084-90.
12. Streifler JY, Eliasziw M, Benavente OR. The risk of stroke inpatients with first ever retinal vs hemispheric transient ischemicattacksand high grade carotid stenosis. North Americansymptomatic CarotidEndarterectomy Trial. Arch Neurol 1995; 52:246-9.
13. Bruno A, Jones WL, Austin JK. Vascular outcome in men with asymptomatic retinal cholesterol emboli. A cohort study. *Ann Intern Med* 1995; 122:249-53.
14. Streifler JY, Eliasziw M, Benavente OL. The risk of stroke in patients with first-ever retinal vs hemispheric transient ischemic attacks and high-grade carotid stenosis. North American Symptomatic Carotid Endarterectomy Trial. Arch Neurol 1995; 52:246-9.
15. Johnston SC, Gress DR, Browner WS, Sidney S. Short-term prognosis after emergency department diagnosis of TIA. JAMA 2000; 284:2901-6.
16. Hill MD, Yiannakoulias N, Jeerakathil T. The high risk of stroke immediately after transient ischemic attack: A population-based study. Neurology 2004; 62:2015-20.
17. Wu CM, McLaughlin K, Lorenzetti DL. Early risk of stroke after transient ischemic attack: A systematic review and meta-analysis. Arch Intern Med 2007; 167:2417-22.
18. Rothwell PM, Giles MF, ChandrathevaA. Effect of urgent Treatment of transient ischaemic attack and minor stroke on earlyrecurrent stroke (EXPRESS study): A prospective population-basedsequential comparison. Lancet 2007; 370:1432-42.
19. Lavallee PC, Meseguer E, Abboud H. A transient ischaemic attack clinic with round-the-clock access (SOS-TIA): Feasibility and effects. Lancet Neurol 2007; 6:953-60.
20. Falk E. Coronary thrombosis: Pathogenesis and clinical manifestations. Am J Cardiol 1991; 68:28B-35B.

21. Mead GE, Lewis SC, Wardlaw JM, Dennis MS. Comparison of risk factors in patients with transient and prolonged eye and brain ischemic syndromes. Stroke 2002; 33:2383-90.
22. Anderson DC, Kappelle LJ, Eliasziw M. Occurrence of hemispheric and retinal ischemia in atrial fibrillation compared with carotid stenosis. Stroke 2002; 33:1963-7.
23. Hurwitz BJ, Heyman A, Wilkinson WE. Comparison of amaurosis fugaxandtransient cerebral ischemia: A prospective clinical and arteriographic study. Ann Neurol 1985; 8:698-704.
24. Bruno A, Corbet JJ, Biller J. Transient monocular visual loss patterns and associated vascular abnormalities. Stroke 1990; 21:34-9.
25. Caplan LR, Warren MJ, in Brain Embolism; Informa Healthcare USA, Inc 2006: p 81-82.
26. Burde RM. Amaurosis fugax. An overview. J Clin Neuroophthalmoogy 1989; 9:185-9.
27. Fisher CM. 'Transient monocular blindness 'versus' amaurosis fugax'. Neurology 1989; 39:1622-4.
28. Donders RC; Dutch TMB Study Group. Clinical features of transient monocular blindness and the likelihood of atherosclerotic lesions of the internal carotid artery. J Neurol Neurosurg Psychiatry 2001; 71:247-9.
29. Caplan LR, Warren MJ, in Brain Embolism; Informa Healthcare USA, Inc 2006: p 87-88.
30. Younge BR. The significance of retinal emboli. J Clin Neuroophthalmology 1989; 9:190-4.
31. Sharma S, ten Hove MW, Pinkerton RM, Cruess AF. Interobserver agreement in the evaluation of acute retinal artery occlusion. Can J Ophthalmol 1997; 32:441-4.
32. Hayreh SS. Carotid artery insufficiecy. In: Gold DH, Weingeist TA, editors. The eye in systemic diseases. Philadelphia: JB Lippincot; 1990. p. 64.
33. Merchut MP, Gupta SR, Naheedy MH. The relation of retinal artery occlusion and carotid artery stenosis. Stroke 1988; 19:1239-42.
34. Savino PJ, Glaser JS, Cassady J. Retinal stroke: Is the patient at risk? Arch Opthalmol 1977; 95:1185-9.
35. Chawluck JB, Kushner MJ, Bank WJ. Atherosclerotic carotid artery disease in patients with retinal ischemic syndromes. Neurology 1988; 38:858-63.
36. Marshall J, Meadows S. The natural history of amaurosis fugax. Brain 1968; 91:419-34.
37. Bogousslavsky J, Regli F, Zografos L, Uske A. Opticocerebral syndrome: Simultaneous hemodynamic infarction ofoptic nerve and brain. Neurology 1987; 37:263-8.
38. Lord RS. Transient monocular blindness. Aust N Z J Ophthalmol 1990; 18:299-305.
39. Furlan AJ, Whisnant JP, Kearns TP. Unilateral visual loss in bright light. An unusual symptom of carotid artery occlusive disease. Arch Neurol 1979; 36:675-6.
40. Kaiboriboon K, Piriyawat P, Selhorst JB. Light-induced amaurosis fugax. Am J Ophthalmol 2001; 131:674-6.
41. Levin LA, Mootha VV. Postprandial transient visual loss. A symptom of critical carotid stenosis. Ophthalmology 1997; 104:397-401.
42. Kofoed PK, Milea D, Larsen M. Transient monocular blindness precipitated by sexual intercourse. Br J Ophthalmol 2009; 93:1199, 1250. [Epub 2008 Jul 25].
43. Russell RW, Page NG. Critical perfusion of brain and retina. Brain 1983; 106:419-34.
44. Caplan LR.In: Caplan's stroke A clinical approach. 4th ed. Philadelphia: Saunders; 2009. p. 64.
45. Pessin MS, Panis W, Prager RJ, Millan VG, Scott RM. Auscultation of cervical and ocular bruits in extracranial carotid occlusive disease: A clinical and angiographic study. Stroke 1983; 14;246-9.
46. Fisher CM. Cranial bruit associated with occlusion of the internal carotid artery. Neurology 1957; 7:299-306.
47. Fisher CM. Facial pulses in internal carotid artery occlusion. Neurology 1970; 20:476-8.
48. Sayuri F, Kaoruko K, Hidemitsu N, Yoshihiro S, Norikiyo N. Classification of ophthalmic artery flow in patients with occlusive carotid artery disease. Jpn J Ophthalmol 2006; 50:224-8.
49. Renzo B, Carlo P. Collateral cerebral circulation through the ophthalmic artery and its efficiency in internal carotid occlusion. Br J Radiol 1955; 28:462-9.
50. Caplan LR. The frontal artery sign: A bedside indicator of internal carotid occlusive disease. N Engl J Med 1973; 288:1008-9.
51. Countee RW, Gnanadev A, Chavis P. Dilated episcleral arteries: A significant physical finding in assessment of patients with cerebrovascular insufficiency. Stroke 1978; 9:42-5.
52. Olivarius Bde F. The external carotid sign. Acta Neurol Scand 1965; 41:539-50.
53. Gregory YC. Elimination of subjective bruit with compression of temporal artery: New physical sign indicative of internal carotid artery occlusion. Stroke 1984; 15:903-4.
54. Brown GC, Magargal LE. The ocular ischemic syndrome. Clinical, fluorescein angiographic and carotid angiographic features. Int Ophthalmol 1988; 11:239-51.
55. Bull DA, Fante RG, Hunter GC, VanDalen J, Lee D, Bernhard VM, *et al.* Correlation of ophthalmic findings with carotid artery stenosis. J Cardiovasc Surg (Torino) 1992; 33:401-6.
56. Kahn M, Green WR, Knox DL, Miller NR. Ocular features of carotid occlusive disease. Retina 1986; 6:239-52.
57. Smith VH. Pressure changes in the ophthalmic artery after carotid occlusion (an experimental study in the rabbit). Br J ophthalmo 1961; 45:1-26.
58. Sarkies NJC, Schilling JS, Ross Russell RW. Fluorescein angiography in carotid disease. Trans Ophthalmol Soc U K 1986; 105:489-93.
59. Kersemakers P, Beintema M, Lodder J. Venous stasis retinopathy unlikely results from internal carotid artery obstruction alone. Cerebrovasc Dis 1992; 2:305-7.
60. Kerty E, Eide N, Horven I. Oculatr hemodynamic changes in patient with high –grade carotid occlusive disease and development of chronic ocular ischemia. II. clinical findings. Acta Ophthalmol Scand 1995; 73:72-6.
61. Brown GC, Green WR. The ocular ischemic syndrome. Curr Opin Ophthalmol 1994; 5:14-20.
62. Mizener JB, Podhajsky P, Hayreh SS. Ocular ischemic syndrome. Ophthalmology 1997; 104:859-64.
63. Brown GC, Magargal LE, Simeone FA, Goldberg RE, Federman JL, Benson WE. Arterial obstruction and ocular neovascularizarion. Ophthalmology 1982; 89:139-46.
64. Kearns TP, Hollenhorst RW. Venous stasis Retinopathy of occlusive disease of the carotid artery. Proc Staff Meet Mayo Clin 1963; 38:304-12.
65. Kleindorfer D, Miller R, Moomaw CJ, Alwell K, Bordeick JP, Knoury J, *et al.* Designing a message for public education regarding stroke: Does "FAST" capture enough stroke? Stroke 2007; 38:2864-8.
66. Nor AM, McAllister C, Louw SJ, Dyker AG, Davis M, Jenkinson D, *et al.* Agreement between ambulance paramedic- and physician-recorded neurological signs with face arm speech test (FAST) in acute stroke patients. Stroke 2004; 35:1355-9.

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