

# Central Retinal Artery Occlusion after Carotid Endarterectomy

Dear Editor,

A 61-year-old man with a history of amaurosis fugax and 90% stenosis of the right internal carotid artery underwent carotid endarterectomy under local anaesthetic. At the time of reperfusion, he developed sudden loss of vision in the right eye.

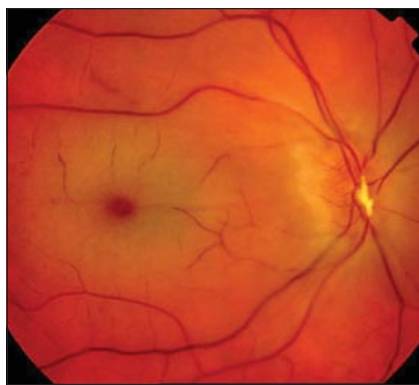
The visual acuity was hand movements in the affected eye and an afferent pupillary defect was noted. Fundus examination revealed a large saddle embolus in the central retinal artery (Figure 1).

Immediate treatment with vigorous ocular massage, in-the-bag inhalation, intravenous diamox, and paracentesis was initiated. The vision failed to improve because of the anatomic configuration

of the embolus. The embolus probably originated from the residual fragments of the removed atheromatous plaque. Loss of vision with this surgery is extremely rare<sup>1</sup> and this is the first case of a saddle

embolus causing central retinal artery occlusion after this procedure. We suggest that this possibility is raised when obtaining informed consent.

**Figure 1. Saddle embolus in the central retinal artery**



## Reference

1. Nucci C, Martelli E, Appolloni A, Palma S. A case of central retinal artery occlusion after carotid endarterectomy. *Eye* 1997;11 (Pt 5):755-757.

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## Comment

These authors have provided a graphic example of a devastating ophthalmic complication of non-ocular surgery.

Central retinal artery occlusion (CRAO) has been reported to occur in relation to embolic phenomena following carotid vessel surgery,<sup>1</sup> spinal surgery,<sup>2</sup> renal surgery,<sup>3</sup> cardiac catheterisation,<sup>4</sup> and even manipulation of the neck.<sup>5</sup> While most cases have either documented or presumed emboli, it is also well known that prolonged pressure on the globe during surgery (typically surgery under general anaesthesia in the prone position) can also cause CRAO.<sup>6</sup> Most anaesthetists are aware of this and take great pains to position the patient appropriately. Interestingly, Gaunt et al, in their evaluation of the effects on vision of carotid endarterectomy in 100 consecutive patients, found only 1 new retinal embolus (as opposed to 6 patients with pre-existing retinal emboli).<sup>7</sup>

Besides including CRAO as a potential complication of surgery when obtaining informed consent, as the authors remind us, it might be worthwhile for patients undergoing surgery for which there is a risk for CRAO to have their visual function evaluated before and immediately after the surgery. While acute interventions for CRAO such as anterior chamber paracentesis, ocular massage, inhalation of carbogen, acetazolamide, intra-arterial thrombolysis, and hyperbaric oxygen have had limited success, it could be argued that patients who have just undergone surgery present an opportunity for early detection and intervention. In addition, besides CRAO, ischaemic optic neuropathy and cerebral ischaemia are also documented causes of visual loss following major surgery and should, where appropriate, be considered when obtaining informed consent.

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# Endogenous Endophthalmitis and Duke's Diagnostic Criteria for Evaluation of Infective Endocarditis

Dear Editor,

Due to the highly variable clinical manifestations of infective endocarditis, Duke's system of major and minor diagnostic criteria is widely used to establish the diagnosis.<sup>1</sup> Documentation of 2 major criteria, 1 major and 3 minor criteria, or 5 minor criteria identifies the case as 'definite' infective endocarditis. Major arterial emboli resulting in cerebral, splenic, and renal abscesses and septic pulmonary infarcts are considered to be minor criteria of vascular phenomena in the Duke's diagnostic system.<sup>1</sup> For obvious reasons, minor emboli to capillaries and non-specific peripheral signs that may be due to tiny emboli are excluded. However, the exclusion of endogenous endophthalmitis from diagnostic consideration in Duke's diagnostic strategy remains hard to justify. We have recently encountered a patient in whom an occurrence of endophthalmitis alerted the physician to the possibility of infective endocarditis, but endophthalmitis by itself does not count towards the evaluation using Duke's system. This case was only later categorised as 'definite' infective endocarditis when Roth's spot was detected in the other eye allowing fulfilment of the required number of criteria.

We feel that for the following reasons, based on clinical as well as anatomical realities, that endophthalmitis should be incorporated in the minor criteria of Duke's system. Endogenous endophthalmitis is known to be associated with infective endocarditis.<sup>2,3</sup> Endogenous endophthalmitis can even constitute initial evidence of an intracardiac septic process.<sup>4</sup> Endogenous endophthalmitis is fundamentally an intraocular infection resulting from haematogenous bacterial spread.<sup>2</sup> That endogenous endophthalmitis is an indication of an intravascular septic focus is evident from the fact that the right eye is more commonly affected than the left, perhaps due to more direct flow to the right carotid artery.<sup>4</sup> Ophthalmic circulation is an offshoot of the internal carotid circulation. Developmentally, the eye itself is an offshoot of the brain.<sup>5</sup> Whereas cerebral abscess is accorded the status of a minor criterion in the Duke's diagnostic system, the occurrence of endogenous endophthalmitis from embolisation in same area of the circulation does not receive similar diagnostic status.

In light of the above arguments, we propose that for evaluation of patients suspected to have infective endocarditis according to Duke's diagnostic criteria, endogenous infective endophthalmitis should

be considered on a par with other embolic vascular phenomena such as cerebral abscess. However, the addition of new criteria would require a careful prospective evaluation to enhance diagnostic sensitivity without compromising specificity.

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## Comment

We thank Ali et al for their interesting correspondence on *Endogenous endophthalmitis and Duke's diagnostic criteria for evaluation of infective endocarditis*.

The clinical criteria for the diagnosis of infective endocarditis has been changed and improved during the past decade — the most popular of which include Duke's

criteria in 1994,<sup>1</sup> von Reyn's criteria in 1994,<sup>2</sup> and the proposed modified Duke's criteria in 1997.<sup>3</sup> Von Reyn's classification includes 'vascular phenomena' as 'possible' diagnosis of infective endocarditis and Duke's criteria places 'Roth's spots' as an immunological phenomenon — a 'minor' criterion in the clinical diagnosis of infective endocarditis. The eponymous

Roth's spots classically associated with infective endocarditis are now known to be white-centred haemorrhages — an area of central infarct with surrounding haemorrhage.

This can be seen in any retinal vasculitic manifestation of systemic thromboembolic disease, including septicaemia and vascular coagulopathies.

Infective endocarditis can present in the eye in a variety of ways, both as a manifestation or a complication. Medical students are routinely taught to look for Roth's spots in patients suspected to have infective endocarditis, but not to look for infective choroiditis or endogenous endophthalmitis, a severe sight-threatening complication that can present insidiously as mild blurring of vision, redness, or floaters to the general practitioner or as sudden painful blurring of vision to the ophthalmologist. Standard ophthalmology teaching mandates a full systemic assessment and evaluation for foci of infection(s) in any patient presenting with endogenous endophthalmitis. This includes a cardiology assessment, often with echocardiogram (transthoracic or transoesophageal) to exclude endocarditis.

Although endophthalmitis is not a frequent metastatic complication of endocarditis,<sup>4</sup> review of the literature shows that there are more than 20 reports of patients initially presenting with endogenous endophthalmitis caused by a wide variety of organisms from fungal to bacterial (especially *Streptococcus* spp).<sup>5-6</sup> Visual prognosis is generally poor but there have

been reports of good outcomes with early diagnosis and aggressive systemic and local therapy with intravenous, intravitreal, and topical antibiotics. The role of early therapeutic vitrectomy for endogenous endophthalmitis, although advocated by some authors, is still generally controversial and usually reserved for medically-fit patients in whom the infection is fulminant or unresponsive to medical therapy.

It is therefore a timely reminder to internists and ophthalmologists to perform a thorough systemic evaluation for any life-threatening foci of infection in any patient who presents with endogenous endophthalmitis. Although endophthalmitis does not strictly fall into the categories of 'vascular' or 'immunological' phenomena of Duke's criteria, it is a result of septic emboli to the ophthalmic arterial system, and therefore should be eligible as an addition to Duke's minor criteria. However further analysis to determine the sensitivity and specificity of the criterion after such an inclusion will be necessary.

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## Disciform Keratitis following Trauma Ab Interno by a Slit Knife Sterilised in a Dry Formaldehyde Vapour Cabinet

Dear Editor,

Following phacoemulsification through a temporal limbal tunnel incision with rigid posterior chamber intraocular lens implantation in a 75-year-old woman, the patient experienced persistent central corneal oedema. The configuration of oedema was disciform, involving the central 5-mm zone. There was epithelial and stromal oedema accompanied by mild to moderate

heterogeneous small keratic precipitates and anterior chamber reaction. The eye was treated with topical steroids, non-steroidal anti-inflammatory drugs, cycloplegics, and hypertonic sodium chloride ointment. The oedema gradually resolved over 2 months, leaving a faint ring of stromal opacification behind. Paracentrally and just to the temporal edge of the disciform lesion, a penetration site/scar compatible with the shape of a 3.2 slit knife was identifiable following

the resolution of the oedema. The knife must have penetrated the central cornea at the time of keratectomy, ab interno. It is noteworthy that the patient had no history of ocular herpes and no (relative) hypoesthesia was detectable.

At some of the ophthalmic theatres in Iran, disposable knives are kept in a container partially filled with paraformaldehyde tablets for at least 24 hours for a second use. At the time of reuse, they are irrigated

with normal saline. The hypothesis for this case is that remnants of paraformaldehyde were implanted following inadvertent trauma by the blunt knife and this induced endotheliitis. Although trauma alone can induce posterior annular keratopathy (traumatic corneal endothelial ring)<sup>1</sup> and corneal contusion, this mechanism does not explain all of the features observed, namely, associated iriditis, long recovery period, and disciform configuration. Formaldehyde is known to be toxic and a potent allergen<sup>2</sup> and intense keratitis complicated by opacities, myopic shift, and induced

astigmatism have already been reported following photorefractive keratectomy in which paraformaldehyde tablets were used for disinfection of the excimer laser iris cone.<sup>3</sup> It should be stated that it is believed that the use of paraformaldehyde tablets as a sterilising method should be abandoned.<sup>4</sup>

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### Comment

The postulated mechanism of the central cornea oedema is that the knife had penetrated the central cornea and left some paraformaldehyde in the endothelium and/or stroma. One would think that only the tip of the knife would touch the endothelium in the central cornea, and since the whole tip and width of the knife went through the epithelium, stroma, and endothelium of the temporal limbal wound, why is it that a similar oedema was not seen in this wound? It would be intuitive to think that most of the paraformaldehyde would

be deposited on the temporal wound as the knife enters the eye.

Is it possible that this is a case of herpetic disciform keratitis? Trauma and surgery have been reported to cause a reactivation of herpetic disease.<sup>1-4</sup>

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