Case Report

Sudden visual loss due to posterior ischemic optic neuropathy following craniotomy for a ruptured intracranial aneurysm

Kishor A. Choudhari, Anupama A. Pherwani*
Department of Neurosurgery, Royal Victoria Hospital, Regional Neurosciences Unit, Belfast BT12 8BA, *Department of Ophthalmology and Visual Sciences, Queens Medical Centre, Nottingham University Hospitals, Nottingham NG7 2UH, United Kingdom

The authors report a rare case of acute ipsilateral blindness that occurred after a standard fronto-temporal craniotomy for aneurysm in supine position. Posterior ischemic optic neuropathy caused by external pressure on the ipsilateral eye, its differentials and subsequent medico-legal implications are discussed.

Key words: Aneurysm surgery, blindness following craniotomy, Ischemic optic neuropathy, pterional craniotomy, visual loss

Introduction

Sudden visual loss after an uncomplicated craniotomy can be a devastating aftermath. This complication is recognized following prolonged spinal surgeries performed in prone position. The authors report a case where an uncomplicated standard fronto-temporal craniotomy performed in supine position for a ruptured anterior communicating artery (AComA) aneurysm resulted in permanent visual loss in the ipsilateral eye. Various aetio-pathological mechanisms and medico-legal consequences are discussed.

Case Report

A 47-year-old lady presented with CT-proven Grade I sub-arachnoid and intraventricular hemorrhage. A CT angiogram and digital subtraction angiogram (DSA) were highly suggestive of a small aneurysm arising at AComA complex with vasospasm around anterior cerebral vessels [Figure 1]. The patient was surgically explored through a standard right pterional craniotomy in supine position.

Although the CSF was xanthochromic, no aneurysm was detected, the AComA complex being unremarkable. The AComA itself was slightly bulbous but not aneurysmal [Figure 2]. The craniotomy- was essentially a negative exploration. A DSA and MRI/MRA carried out 10 days later after the spasm was resolved revealed no vascular abnormality.

The patient woke up from surgery fully alert without any neurological deficits except complaining of inability to see with her right eye (RE). External examination of the eye was unremarkable. There was no swelling, discoloration, or chemosis. Visual acuity was reduced to perception of light and the anterior segment examination revealed an afferent pupillary defect in the affected eye. Fundoscopy showed no abnormalities of the optic disc and retina. A CT and later MRI scan performed postoperatively did not show any new intra-cranial or

Figure 1: An A-P view of right carotid angiogram suggestive of an aneurysm at the anterior communicating artery (black arrow)
intra-orbital pathology. Although the patient made an excellent neurological recovery, vision in the right eye remained only up to finger-counting eight weeks later with fundoscopy showing pallor of the optic nerve head.

**Discussion**

Sudden monocular visual loss following uncomplicated cranial operations performed in supine position is rare. This phenomenon is more recognized after spinal surgeries performed in prone position. We wonder whether this problem is more common in Asians with a flattened nasal bridge. Unfortunately, when it occurs, irrespective of its cause, it is usually irreversible.[1]

In our case, several mechanisms for this rare phenomenon were considered as discussed below:

- **Thrombosis of central retinal artery due to spasm and/or surgical manipulation** was considered as a possibility. The central retinal artery is an end-artery. Spasm of vessels supplying intracranial optic nerve could possibly lead to ischemic optic neuropathy.[2]
- **Occlusion of the central retinal artery** is known to occur after prolonged hypotension or severe blood loss.[3,4] Our patient was however normotensive throughout with minimal blood loss. There were no other significant medical predisposing illnesses like atherosclerosis or diabetes.
- **Intra-operative technical error** could result in mechanical or thermal damage to the intra-cranial portion of the optic nerve. In our case, however, there was not inadvertent direct trauma to the optic nerve noticed to explain the blindness.
- **Lastly, intra-operative external pressure on the eyeball** causing posterior ischemic optic neuropathy was a possibility. Excessive protective eye-padding, a bulky wet swab buckled over the eyeball and underneath the scalp flap intended to keep the flap moist, pressure exerted by the spring hooks used to retract the scalp inferiorly to facilitate a low craniotomy, could, alone or in combination, result in raised orbital venous pressure with prolonged ischemia of the intra-orbital structures [Figure 3]. We believe prolonged external pressure on the eyeball for over three hours was the main cause of our patient’s blindness. However, it is likely that the pathogenic mechanism was multifactorial and as a cumulative effect of more than one of the theories postulated above.

Aneurysms in the region of AComA and internal carotid arteries are known to present with pre-existing visual deficits of compressive etiology.[5-8] These pre-operative deficits usually improve following successful surgical decompression. Even if the aneurysm ruptures into the optic apparatus, substantial delayed recovery can be expected.[9] Unfortunately, iatrogenic post-operative blindness following neurosurgical procedures is associated with poor visual prognosis.[1,10] This phenomenon is more recognized after spinal surgeries performed in prone position especially when the head is rested on a horse-shoe.[11,12] Kang and co-workers recommend using protective eye-shields rather than bulky and spongy eye-pads to prevent posterior ischemic optic neuropathy.[11,12] From our experience, we also recommend the same in preference to bulky eye-pads.

In spite of being disappointed with this tragic complication of causing irreversible visual deficit in our patient, the authors feel it necessary to highlight this complication for two main reasons. Firstly, it can potentially occur after any improperly executed fronto-temporal craniotomy that seems preventable in retrospect. Secondly, due to its rarity, the patients are not routinely warned of this complication. When it happens, it overshadows an otherwise uneventful neurological recovery and can potentially pose adverse medico-legal implications for the operating team as well as the treating Institution.

**Figure 2:** Intra-operative microscopic photograph depicting both optic nerves and AComA complex. ON- Optic Nerve, ICA- Internal carotid artery, AComA – Anterior communicating artery. Small arrows - intact perforators supplying the optic apparatus

**Figure 3:** Intra-operative photograph of right pterional craniotomy. Small Black arrows - Fishhook retracting the scalp flap. Medium sized black arrows - wet bulky sponge buckled underneath the scalp flap over the padded eyeball
References


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