

Sir,
Cavernous sinus thrombosis following prone position anaesthesia

Prone position is commonly used in orthopaedic and neurosurgical procedures. Although there is high risk of systemic complications,¹ ocular complications are relatively rare. We present here a case of unilateral blindness and cavernous sinus thrombosis following cervical spine surgery in prone position. A case is made for proper care in positioning the patient to avoid ocular complications.

Case report

A 76-year-old male underwent surgery for cervical spine stenosis, under general anaesthesia. The patient was in prone position for 5 h, with face supported on a Mayfield head clamp (Figure 1).

Immediately on regaining consciousness, he complained of being unable to see with the left eye. Vision was no perception of light in this eye. The eye was mildly proptotic with lid swelling and conjunctival chemosis. There was complete external ophthalmoplegia. The cornea was hazy with shallow anterior chamber. The pupil was mid-dilated and nonreactive. Fundoscopy showed retinal oedema with cherry-red spot at the fovea. Retinal arterioles and veins had normal calibre. Contrast CT (Brain and Orbits) on the day of surgery was normal. A week later, the disc was pale with attenuated arterioles. Retinal oedema had subsided. By day 14, the disc was atrophic with thread-like arterioles and disorganization of retinal pigment. MRI brain and MRA carotids 1-week post-op were normal. A cerebral four-vein angiogram 2 weeks after the operation showed left cavernous sinus thrombosis, the right sinus was normal (Figure 2). The patient remained systemically well. Systemic work up including blood cultures, cardiological, and haematological work up was normal. He was anticoagulated. Over a 4-month follow-up, the proptosis gradually subsided. There was partial recovery of ocular movements. Vision remained at no perception of light.

Comment

The sequence of ocular events seen in our patient is a rare complication of neurosurgical procedures requiring sitting/prone position anaesthesia and is known as the Hollenhorst syndrome.² It is believed to be due to inadvertent pressure on the affected eye, usually from the headrest²⁻⁶ and has been prevented by changes to the design of the head rest.³

The disturbance of physiology producing these events is seemingly initiated by partial or complete collapse of



Figure 1 Head clamp (Mayfield) used for prone position surgery in this case.

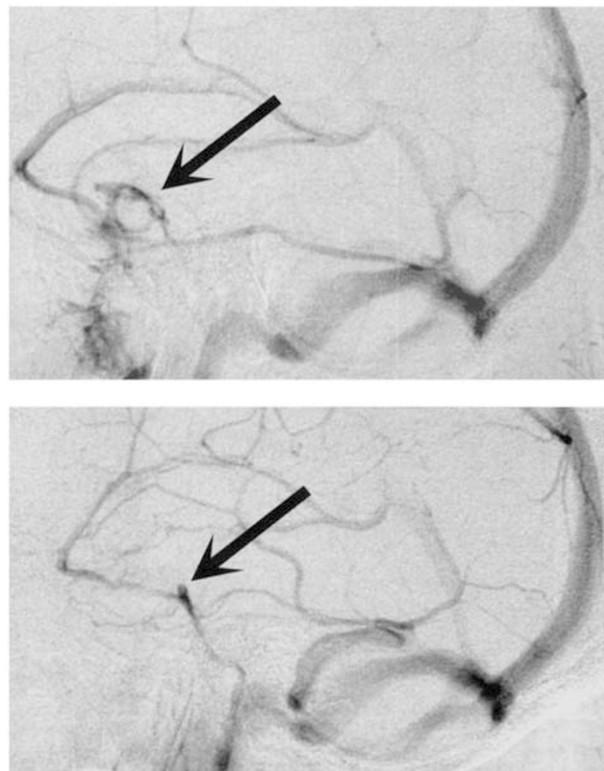


Figure 2 Top. Normal right cavernous sinus (arrow). Bottom. No filling of left cavernous sinus (arrow) indicating cavernous sinus thrombosis.

the orbital arterial and venous channels caused by a tamponade action of the ocular contents. When the external pressure is released, the ischaemic vessels dilate

and there is transudation of fluid into the tissue spaces. This results in orbital oedema, proptosis, paresis of ocular movement and retinal oedema. Retinal arterioles and veins at this stage are of normal calibre or dilated due to the relative anoxia of the vessel walls. The extremely low tolerance of ganglion cells to ischaemia produces the loss of vision.³

The acute stage is followed by gradual absorption of oedema, narrowing and sclerosis of previously ischaemic retinal arterioles, and ganglion cell degeneration producing optic atrophy.³

We are however not aware of any previous report of cavernous sinus thrombosis in this syndrome. Our patient was a healthy male with controlled hypertension. There were no other predisposing factors for venous thrombosis. The cavernous sinus thrombosis in his case was a late event as shown by normal neuroimaging on the day of surgery and 1-week postoperative. It was unilateral and nonprogressive. We believe that stagnation of blood flow and ischaemic damage to vessel walls resulting from prolonged collapse of vascular channels in this patient led to the onset of thrombosis in the orbital circulation with retrograde extension to the cavernous sinus.

This case represents a serious but potentially avoidable sequence of ocular events following prone position surgery. The importance of proper head positioning, such that the eye is not subjected to sustained pressure against the headrest, cannot be overemphasized.

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Sir,
Ocular and systemic side effects of latanoprost

We have read with considerable interest the paper by Konstas *et al.*¹ The authors concluded that latanoprost had a 'similar safety than timolol as monotherapy in the treatment of exfoliation glaucoma (XFG)'. However, according to the paper's Table 3, there were significantly more cases of conjunctival hyperaemia in patients receiving latanoprost compared with those receiving timolol regimen. This can be reasonably explained because prostaglandins including latanoprost, as potent vasodilators, act directly on vascular smooth muscle, attenuating responses of vasoconstrictive stimuli, and enhancing microcirculation,² thereby leading to conjunctival hyperaemia. The latter might be further aggravated by the fact that prostaglandins can promote angiogenesis.

Moreover, in Table 4 there was one case of upper respiratory tract infection in a patient treated with latanoprost. Although this systemic side effect has not reached statistical significance when compared with the timolol group, it can be attributed to latanoprost, because it has been shown that local administration of antiglaucoma drops results in systemic absorption through the nasal mucosa,³ and prostaglandins, released by cyclooxygenase (COX)-2 (one of the key isoenzymes in the production of prostaglandins), induce immune suppression,⁴ thereby leading to defective elimination of pathogens. Notably, disturbances of the immune system can induce disease in virtually any portion of the eye; examples include conjunctivitis, keratitis, keratoconjunctivitis, scleritis, uveitis, optic neuritis, and orbital inflammation. Therefore, patients under latanoprost treatment should be aware of potential systemic and/or topical infective side effects.

From another extreme viewpoint, although the authors have not administered latanoprost for an extended period of time, it should be noted that prostaglandins are implicated both in cell proliferation, and inhibition of immune surveillance; therefore these agents could favour a potential malignant growth. It is already known that