

## Central retinal artery occlusion after peribulbar anesthesia : report of 3 cases

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**Abstract :** We report 3 cases of central retinal artery occlusion following peribulbar anesthesia. Those patients were scheduled for retinal detachment repair, removal of a pterygium, and cataract surgery, respectively. In patient 1, a peribulbar anesthesia was performed with 17 ml of ropivacaine and a compression with a Honan's balloon was maintained at 30 mmHg for 30 min to reduce intra ocular pressure and facilitate intra ocular surgery. In the second patient, 13 ml of ropivacaine was administered without compression of the ocular globe. Patient 3 received 14 ml of mepivacaine and compression was maintained at 30 mmHg for 10 min. In these 3 cases, the surgeon noticed a whitening of the retina during the postoperative period, corresponding with a typical occlusion of the central artery of the retina. Several mechanisms may be cited in an attempt to explain this retinal ischemia : the high volume injected, the speed of injection, a compression of the ocular globe at high pressures for a prolonged period of time, and/or an intrinsic vasoconstricting effect of local anesthetic agents.

**Key words :** Anesthesia ; peribulbar ; complications ; retinal ; artery.

### INTRODUCTION

Peribulbar anesthesia is recognized as a safe regional anesthesia technique that allows carrying out most of ophthalmic surgical procedures (2). Compared to retrobulbar anesthesia, peribulbar anesthesia has increased in popularity, as far as it provides the same anesthetic result and has a lower morbidity (15).

Peribulbar anesthesia can be performed according to the Hamilton's technique (6). The first insertion of the needle (25 Gauge, 32 mm in length) is parallel to the orbital floor at the lateral aspect of the inferior orbital rim (25 mm maximal depth), and the second (25 mm maximal depth too) is performed at the level of the supraorbital notch. The injection must immediately be stopped when tension appears in the ocular globe. As soon as the globe is soft again, injection can be restarted until another sensation of tension into the globe re-

appears. This technique is safe, as far as the incidence of complications has been reported to range from 0.006 to 0.04% (1). Among them, central retinal artery occlusion is even rarer.

Removing an ocular compression with a Honan's balloon that was applied before surgery is associated to a significant reduction of the intra ocular pressure. This technique has been used for a long time, is supposed to facilitate the surgical procedure and is of frequent use. Noteworthy, LING *et al.* recently reported that a Honan's balloon does not appear to make a difference in the effectiveness of peribulbar anesthesia for surgeons or patients (10).

We report here 3 cases of central retinal artery occlusion that occurred after peribulbar anesthesia, with or without Honan's ocular globe compression. All patients gave their oral consent to publication of their history.

### CASE REPORTS

#### Case 1

In 2004, a 77-y-old female, weighing 63 kg, who had complained for a loss of vision for one week in the right eye, was scheduled for retinal detachment repair. Medical history revealed an hypertension treated with the association of perindopril and indapamide, and a depressive syndrome controlled with mianserine. She was given a

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peribulbar anesthesia with a volume of 17 ml of ropivacaine (7.5 mg/ml, 10 ml in the infero-temporal space, 7 ml in the superomedial). A compression with a Honan's balloon was maintained at 30 mmHg for 30 min.

#### Case 2

This case occurred 3 years ago. A 71-y-old female, weighing 79 kg, and scheduled for cataract surgery, was given a peribulbar anesthesia with a volume of 14 ml of mepivacaine (20 mg/ml, infero-temporal 10 ml, superomedial 4 ml). A compression was maintained at 30 mmHg for 10 min. Medical history only noticed a mental depression that was controlled with mianserine and alprazolam.

#### Case 3

More recently, i.e. 2 years ago, a 58-y-old male, weighing 60 kg, with a Recklinghausen's disease, was scheduled for the removal of a pterygium of the left eye. He lost the vision of the right eye because of a neurofibroma in the right optic nerve. The left optic nerve was normal (CT Scan). He was given a peribulbar anesthesia with a volume of 13 ml of ropivacaine (7.5 mg/ml, inferotemporal 10 ml, superomedial 3 ml). No compression was performed.

In these 3 cases, after termination of the surgical procedure, patients complained of a loss of vision. Fundoscopy then revealed a whitening of the retina in the posterior pole, corresponding with a typical occlusion of the central artery of the retina. This was later confirmed by angiography. No signs of exophthalmos, peribulbar hemorrhage or eyelid hemorrhage were noted. Ocular motility, assessed after the disappearance of anesthesia, was preserved. Systemic blood pressure values always remained within the normal range. Doppler ultrasound of the carotids, echocardiograms and electrocardiograms were normal.

## DISCUSSION

### *Incidence*

In our department, the peribulbar procedures are performed by the same 3 anesthetists, all of them having recognized expertise in peribulbar anesthesia. During the last 20 years, 30 000 peribulbar anesthetic procedures were performed in our institution. Three cases of retinal occlusion

occurred, leading to an incidence of  $10^{-4}$ , which can be considered as an extremely rare event.

### *Mechanisms involved*

To elucidate the mechanisms involved in this dramatic complication, one must first consider the anatomy and physiology of the blood supply to the ocular globe and the retina. The blood supply to the choroid comes ultimately from the ophthalmic artery. Although variations exist, posterior branches of this artery give rise to the central retinal artery and to ciliary arteries on each side of the optic nerve. These vessels divide into 2 long posterior ciliary arteries and (about 20) short posterior ciliary arteries. The entry of those arteries into the eye is immediately adjacent to the optic nerve. The short posterior ciliary arteries directly supply the choroid and the long posterior ciliary arteries travel into the suprachoroidal space anteriorly to supply the anterior choroid via recurrent branches. The central retinal artery is the first intraorbital branch of the ophthalmic artery, which enters the optic nerve 8-15 mm behind the globe to supply the retina. Short posterior ciliary arteries branch emerge distally from the ophthalmic artery and supply the choroid. Anatomical variations and factors that influence the blood flow in the optic nerve head have been fully studied by HAYREH (7, 8).

The choroid receives the greatest blood flow (65-85%) and is vital for the maintenance of the outer retina (particularly the photoreceptors). The remaining 20-30% flows to the inner retinal layers through the central retinal artery. The central retinal artery has 4 main branches in the human retina. KOCABIYIK *et al.* showed that the central retinal artery (CRA) arises directly from the ophthalmic artery in more than 90 % of cases (9), but may also arise in common with the medial posterior ciliary artery. DELAËY *et al.* reported that the retina receives its nutrients from retinal and choroidal circulation. Retinal circulation is characterized by a low blood flow as compared to the choroidal flow. The choroidal circulation is mainly controlled by sympathetic innervation and is not autoregulated, while retinal circulation lacks autonomic innervation, shows an efficient autoregulation and is mainly influenced by local factors (4).

### *First possible mechanism : the pressure around and into the ocular globe*

High level pressures around and into the ocular globe for prolonged periods of time may have

compromised retinal blood flow. These high level pressures may have been provoked by the injection of high volumes of the local anesthetic agent, at a high speed, and by the Honan's balloon compression.

The speed of injection and the amount of local anesthetic drug were dosed according to the clinical appreciation of the pressure around the globe by the anesthetist. This speed of injection is possibly of great concern in the perturbation of retinal blood flow but, to our knowledge, it has not been investigated so far. TURUT *et al.* reported 2 cases of ciliary and retinal occlusion following local retrobulbar anesthesia with orbital compression (13). Two other cases of central retinal artery occlusion after peribulbar anesthesia have recently been reported by VINEROVSKY *et al.* (14). LUNG *et al.* have reported that peribulbar anesthesia using a high volume reduces the ocular blood-flow response to the peribulbar anesthesia as compared to using a low volume (12). LOKEN *et al.* described a transient retinal ischemia produced by ocular compression (11). We have injected higher volumes of local anesthetic agent than usually reported for peribulbar anesthesia (3). However, as a precaution, we injected the mixture very slowly and in several steps. When stopping the injection according to tension of the ocular globe, as we did, the injected volume depends on the size of the orbit and differs from one patient to the other. It is not easy to make the link between our technique and the occurrence of retinal ischemia in our patients. This way of proceeding has been followed for 20 years in our institution before experiencing this complication. External ocular compression can also not be cited easily, since one of the above-reported occlusion occurred in the absence of an ocular compression. A way of clarifying the exact role of those factors in precipitating retinal ischemia would have been to measure the intra-ocular pressure before, during and after the injection of the local anesthetic agent.

#### *Second possible mechanism : an intrinsic vasoconstricting effect of the local anesthetic agent*

FRUHSTORFER *et al.*, reported that mepivacaine has a constrictor effect on the post capillary vascular intradermal bed in humans, causing pallor of the area in which it has been injected (5). WIENZEK *et al.* performed ropivacaine applications directly to a rat's tail artery and showed that ropivacaine, at all concentrations, decreased blood flow in comparison with normal saline. Following those results, they do not recommend the use of ropivacaine at high con-

centrations if tissues are supplied by end arteries (16).

The intrinsic vasoconstricting effect of some local anesthetic agents can theoretically lead to a vasospasm of the central retinal artery, particularly if those agents are in contact with the portion of the artery that lays outside the optic nerve. Although the occlusion of the central retinal artery might have been coincidental, its temporal occurrence with the peribulbar anesthesia suggests some relationship.

#### CONCLUSIONS

These three cases of central retinal artery occlusion following peribulbar anesthesia outline important points. First, this complication, although rare, may occur despite the large experience of the performer. Second, the involved mechanism is not clear. The high injected volume, the speed of injection, a prolonged external compression of the ocular globe at a high pressure, and, finally, an eventual intrinsic vasoconstricting effect of ropivacaine and mepivacaine may be cited. Although further studies are needed to clarify these elements, it is wise to recommend the use of local anesthetic agents without any intrinsic vasoconstricting effect, and to perform peribulbar anesthesia cautiously with a low volume of local anesthetic agent, and in several steps.

#### References

1. Arnold P. N., *Prospective study of a single-injection peribulbar technique*, J. CATARACT REFRACT. SURG., **18**, 157-161, 1992.
2. Davis D. B., Mandel M.R., *Posterior peribulbar anesthesia : an alternative to retrobulbar anesthesia*, J. CATARACT REFRACT. SURG., **12**, 182-184, 1986.
3. Davis P. L., O'Connor J. P., *Peribulbar block for cataract surgery : a prospective double-blind study of two local anesthetics*, CAN. J. OPHTHALMOL., **24**, 155-158, 1989.
4. Delaey C., Van de Voorde J., *Regulatory mechanisms in the retinal and choroidal circulation*, OPHTHALMIC RES., **32**, 249-256, 2000.
5. Fruhstorfer H., Wagener G., *Effects of intradermal lignocaine and mepivacaine on human cutaneous circulation in areas with histamine-induced neurogenic inflammation*, BR. J. ANAESTH., **70**, 167-172, 1993.
6. Hamilton R. C., *Techniques of orbital regional anaesthesia*, BR. J. ANAESTH., **75**, 88-92, 1995.
7. Hayreh S. S., *Blood flow in the optic nerve head and factors that may influence it*, PROG. RETIN. EYE RES., **20**, 595-624, 2001.
8. Hayreh S. S., *Posterior ciliary artery circulation in health and disease : the Weisenfeld lecture*, INVEST. OPHTHALMOL. VIS. SCI., **45**, 749-757, 2004.
9. Kocabiyik N., Yalcin B., Ozan H., *The morphometric analysis of the central retinal artery*, OPHTHALMIC PHYSIOL. OPT., **25**, 375-378, 2005.

10. Ling R., Beigi B., Quinn A., Jacob J., *Effect of Honan balloon compression on peribulbar anesthesia adequacy in cataract surgery*, J. CATARACT REFRACT. SURG., **28**, 113-117, 2002.
11. Loken R. G., Coupland S. G., Deschenes M. C., *The electroretinogram during orbital compression following intraorbital regional block for cataract surgery*, CAN. J. ANAESTH., **41**, 802-806, 1994.
12. Lung S., Luksch A., Weigert G., Georgopoulos M., Menapace R., Polska E., Garhofer G., Findl O., Schmetterer L., *Influence of infusion volume on the ocular hemodynamic effects of peribulbar anesthesia*, J. CATARACT REFRACT. SURG., **32**, 1509-1512, 2006.
13. Turut P., Dumont P., Dhedin G., Bego B., *[Ciliary and retinal occlusion following local retrobulbar anesthesia with orbital compression (2 cases)]*, BULL. SOC. OPHTHALMOL. FR., **86**, 865-6, 868, 1986.
14. Vinerovsky A., Rath E. Z., Rehany U., Rumelt S., *Central retinal artery occlusion after peribulbar anesthesia*, J. CATARACT REFRACT. SURG., **30**, 913-915, 2004.
15. Weiss J. L., Deichman C. B., *A comparison of retrobulbar and periocular anesthesia for cataract surgery*, ARCH. OPHTHALMOL., **107**, 96-98, 1989.
16. Wienzek H., Freise H., Giesler I., Van Aken H. K., Sielenkaemper A. W., *Altered blood flow in terminal vessels after local application of ropivacaine and prilocaine*, REG. ANESTH. PAIN MED., **32**, 233-239, 2007.