MAILBOX

Comparative study of intraoperative mitomycin C and β irradiation in pterygium surgery

EDITOR,—We read with interest the study that compared intraoperative mitomycin C with β irradiation in primary pterygium surgery.[1] The authors rightly commented that long term complications of β irradiation, such as scleral necrosis, may arise more than 10 years after the irradiation.[2] Longer follow ups are necessary to reveal such complications.

We performed primary pterygium excision with intraoperative β irradiation in one eye of six patients between 1988 and 1990. A dose of 1000 rad of β irradiation was applied to the scleral bed intraoperatively and 1 week later. The patients were recently reviewed in our clinic for recurrence and complications. We also performed ultrasound biomicroscopic examination (UBM) for both eyes in each patient, looking for corneal and scleral thinning. Corneal thickness was arbitrarily measured 0.5 mm anterior to the scleral spur at the 12, 3, 6, and 9 o'clock positions of each eye, while the scleral thickness was measured 2 mm posterior to the scleral spur at the same positions.

Mean follow up was 138.0 months. Mean age at time of surgery was 37.5 years (range 32–45 years). All six eyes were right eyes with nasal pterygia in male patients. No recurrence was found, using the same definition. There was neither significant deterioration in visual acuity nor increase in intraocular pressure in any eye. There were no signs of inflammation.

There were no significant differences in the scleral and corneal thickness between the treated nasal position of the operated eye (mean scleral 0.617 (SD 0.112) mm; mean corneal 0.656 (0.076) mm) and the control nasal position of the fellow eye (mean scleral 0.611 (0.030) mm; mean corneal 0.645 (0.044) mm).

Furthermore, there were no significant differences in the mean scleral and corneal thickness between the operated eye (scleral 0.590 (0.077) mm; corneal 0.635 (0.067) mm) and the fellow eye (scleral 0.590 (0.059) mm; corneal 0.624 (0.054) mm). The mean scleral and corneal thicknesses were calculated by averaging the scleral or corneal thickness at the four measured positions in each eye.

It appears that β irradiation is safe, even in the long term. We believe these additional data could supplement the findings by Amano *et al.*

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Visual field defects after vitrectomy with fluid-air exchange

EDITOR,—The paper by Cullinane and Cleary[1] presents an excellent prospective study of peripheral visual field loss in patients undergoing macular hole surgery. The authors compared vitrectomy with complete posterior cortical vitreous peeling to limited vitrectomy with removal of cortical vitreous off the macula, but not off the optic nerve head or the peripheral retina. The authors showed a statistically significant decrease in peripheral visual field defects with the limited vitrectomy technique (0%, 0/22 patients) compared with the complete vitrectomy group (22%, 18/82 patients).

The authors postulated that this difference is due to the avoidance of traction on the optic nerve head during peeling of the posterior hyaloid, thus limiting damage to the peripapillary nerve fibre layer, which they believed would be most severe nasally because of firmer vitreopapillary attachments nasally. This explanation does not take into account the variable position of visual field defects found in other studies based on the position of the infusion cannula. If the infusion cannula is superiorly located, visual field defects occur superiorly, implicating inferior retinal damage.[2] If the infusion cannula is inferonasal, visual field defects occur inferonasally and not inferotemporally.[3] The inferotemporal location of field defects noted in most studies is based on the conventional placement of the infusion cannula inferotemporally in three port vitrectomy, which results in infused air directed towards the superonasal midperipheral retina.

Animal studies show damage to the inner limiting membrane, nerve fibre layer, and ganglion cells of the retina in the path of the pressurised air flow from the infusion cannula.[4 5] This inner retinal damage could be caused by desiccation of the retina[2] or by direct mechanical damage by the pressurised air flow.[4 5] However, humidification of air did not prevent inner retinal damage in animal models,[4 5] and the sharp demarcation between damaged and undamaged retina on electron microscopic studies supports the theory of direct mechanical damage to the inner retina.[4] In addition, decreasing the infusion air pressure also decreased the risk of inner retinal damage.[5] What I think this work by Cullinane and Gleary shows is that leaving the peripheral vitreous in place is another way of protecting the peripheral retina from mechanical damage by pressurised air flow. However, I would be concerned about the potential risk of increased postoperative retinal detachment, which was 10% in the limited vitrectomy group and 4% in the complete vitrectomy group, but was not statistically significant because of small sample size. However, this increased risk of retinal detachment was also a concern in a previous study utilising similar surgical techniques (Brian Conway, Western Association for Vitreoretinal Education Meeting, Maui, Hawaii, 1996).

Because of the studies on retinal damage by pressurised air infusion and the significance of high infusion air pressure, it would be important to know the usual infusion air pressure utilised during fluid-air exchange by the authors, and if the infusion air pressure varied at any point during the period of the study or between the two vitrectomy groups. Currently, in order to minimise retinal damage induced by pressurised air infusion during vitrectomy for any surgical indication requiring fluid-air exchange, I would recommend simply using a low infusion air pressure.

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Sclerotomy complications following pars plana vitrectomy

EDITOR,—The work of West and Gregor again points out the importance of sclerotomy complications following pars plana vitrectomy.[1] They demonstrate that, even in the hands of a skilful and experienced surgeon, vitreous haemorrhage after vitrectomy for diabetic retinopathy is common and requires vitreous cavity washout (VCWO) in 12% of cases. In their series, over half of the eyes had detectable fibrovascular ingrowth (FVI) as the cause of the haemorrhage.

Interestingly, in this case series of 159 eyes, no occurrences of anterior hyaloidal fibrovascular proliferation (AHFP) were noted. Definition of the relation between these two entities has been controversial, to say the least.

Part of the controversy is due to a misunderstanding of the nature and pathogenesis of FVI. As McLeod points out in his editorial, FVI is a term that has been used inadvisedly, suggesting that episcleral tissue grows into the eye through the sclerotomy incision.[2] While episcleral tissue, scleral fibroblasts, and ciliary epithelium all contribute, the majority of the fibroproliferative healing of a sclerotomy originates from the uvea of the ciliary body.[3]

In normal wound healing, early fibrovascular proliferation in the incision is followed by its involution and contraction, with the result being the small scar seen at the internal aspect of a healed sclerotomy.[3] Inevitably, because of the proximity of the vitreous base and anterior hyaloid, vitreous strands are adherent to the wound and fibrous tissue extends a short way into the vitreous body. This tissue may contain blood vessels, even with normal healing. From this perspective, all sclerotomy wounds heal with fibrovascular ingrowth. That is, ingrowth of tissue from the eye wall extends into the vitreous cavity. Fortunately, only in unusual circumstances does this process become exaggerated and result in what clinicians have termed FVI with its concomitant intraocular mischief.[4]