Photocoagulation of raised new vessels by long-duration low-energy argon laser photocoagulation — a preliminary study

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SUMMARY  Direct laser treatment of retinal neovascularisation is indicated when regression has not been brought about by retinal photocoagulation. Current treatment regimens involve multiple laser applications to the neovascular complex. Long-duration low-energy burns have the advantage of slowly heating the tissue without engendering disruption. We report the use of this treatment as a means of occluding the feeder vessels to a neovascular network. The application of argon blue-green laser treatment at 0.1 watt for 60 seconds at two adjacent points on a feeder vessel was found to give rise to permanent vascular occlusion without causing complications.

One of the primary intentions of the development of photocoagulation was to occlude raised retinal new vessels. However, even with the argon laser, with its coherence, selective absorption, and ease of variation of the size and duration of each burn, direct treatment was often found to be ineffective and difficult.1 It was subsequently found that the surrounding retinal damage engendered by the photocoagulation treatment appeared to bring about regression of such aberrant vasculature as a secondary phenomenon. Panphotocoagulation was thus introduced as a means of inducing regression.24 Nevertheless, raised new vessels continue to present problems when panphotocoagulation fails to induce regression.5 Argon laser treatment strategies have thus been devised in which multiple lesions are applied repeatedly to the neovascular network in order to induce regression. Such treatment is time consuming and may be complicated by vitreous haemorrhage,16 retinal holes, and preretinal fibrosis.1

Recently long-duration (10~30 seconds) low energy (0.2~0.4 watt) laser photocoagulation has been advocated as a means of treating choroidal melanoma.17 However, to our knowledge similar treatment has not hitherto been described as being applied to raised new vessels.

We report on a patient with perivasculitis retinaceae (Eales’ disease) in whom repeated photocoagulation of ischaemic retina failed to induce regression of neovascularisation, but for whom the direct application of long-duration low-energy treatment was found to be effective in producing closure of the aberrant vasculature.

Patient and methods

A 29-year-old healthy male presented with sudden loss of vision in the left eye. Clinical examination revealed a vitreous haemorrhage arising from abnormal raised new vessels in the inferonasal quadrant of the left peripheral retina. The aberrant vasculature comprised feeder vessels from the venous side and a fine network of minute vessels and gliosis underlying a detached posterior vitreous face (Fig. 1). Fluorescein angiography showed patent abnormal vessels arising from the venous circulation, which was surrounded by areas of capillary closure (Fig. 2). This late frame showed leakage from the neovascular complex. Unfortunately the patient was violently sick following intravenous fluorescein, and no further fluorescein angiography was performed. Blue-green argon laser photocoagulation was carried out over the area of capillary closure. No direct treatment was administered to the abnormal vasculature at this stage. No regression of the new vessels was engendered by this treatment, and further coagulation of the ischaemic area was performed six weeks later. The patient was followed up for six months but again there was no evidence of regression (Fig. 3).

In view of the risk of recurrent vitreous haemorr-
Fig. 1  *Fundus photograph of neovascular membrane overlying the left inferonasal retina.*

Fig. 2  *Fluorescein angiogram taken at 52 seconds showing peripheral retinal capillary closure and leakage of dye from raised new vessels.*

Fig. 3  *Fundus photograph taken after two sessions of argon laser photocoagulation of the ischaemic retina. There has been no regression of the neovascular membrane.*

Fig. 4  *Fundus photograph taken 48 hours after long-duration low-energy argon blue-green laser photocoagulation of the upper feeder vessel supplying the neovascular membrane.*

Hage direct photocoagulation of the abnormal new vessels was carried out. There were two main feeder vessels which each divided into two main branches; three of these main branches were elevated from the retina. Treatment was applies as follows. Topical local anaesthetic was instilled and a three-mirror contact lens applied. The biophysical medical argon-krypton laser was employed with the time setting adjusted to continuous. The argon blue-green laser was aimed at a point on the abnormal vessel and the foot switch depressed. Two adjacent points along the uppermost vessel were treated with 0.15 watt, with a 50 μm spot size, delivered continuously for 30 seconds. The duration of laser application was timed to the nearest second. (The laser employed has a protective filter between the patient and the operator, and thus the position of the treatment being applied can be visualised throughout.) After
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Fig. 5  Fundus photograph taken six months after long-duration low-energy argon blue-green laser photocoagulation of the three feeder vessels supplying the neovascular membrane. Complete regression of the new vessels has taken place.

this first application the treated vessel was occluded by the following day, but a flame shaped haemorrhage was later observed adjacent to the vessel (Fig. 4).

Consequently for further applications a lower energy setting was used (0.1 watt) with twice the duration of exposure (60 seconds). The two remaining raised vessels were treated by the same method at the same sitting. On this occasion narrowing of both vessels was seen the next day, and closure was observed four days later. Six months after treatment the new vessels remained closed (Fig. 5).

Discussion

Elevated new vessels and vascular loops have hitherto proved difficult to occlude by direct photocoagulation. The light emitted by the argon blue-green laser is absorbed by haemoglobin. It thus provides an appropriate means of occluding new vessels. Treatment strategies at present employed involve the application of repeated, multiple, relatively high-energy low-duration burns to the abnormal vessels. Such vessels are friable and susceptible to rupture, particularly if laser treatment is too heavy. It is thus not surprising that this time consuming mode of treatment is commonly accompanied by haemorrhage, the very complication which the treatment is designed to avoid.

The rate of blood flow in the main retinal vein is between 12 and 14 mm per second as determined by the differential laser Doppler method. Thus, when laser energy is applied to the retinal vein, heat it dispersed by a flowing blood column. For this reason treatment strategies have been designed which begin by occluding the terminal portions of the neovascular fronds in which the rate of blood flow is lower. The rationale of the treatment described in the present study was gradually to heat the venous feeder vessel and thereby induce stasis and thrombosis of the blood column between two points of coagulation. Vessel closure is brought about without vitreous haemorrhage in a single short treatment session.

Experimental work with rabbits has shown moderate swelling of the nerve fibre layer after light coagulation, and it is possible that this phenomenon may contribute to the vessel closure. Vascular spasm may also be a factor in giving rise to narrowing of the vein. Once the laser treatment has brought about stasis within the vessel being treated, it is likely that the heat being taken up by the blood column would be transmitted to the adjacent vessel wall. In addition, the oxyhaemoglobin will become reduced to haemoglobin in a static blood column. Reduced haemoglobin has a higher energy absorption for argon blue-green laser than oxyhaemoglobin, thus potentiating the effect of treatment.

Only lasers in which a protective filter rather than a shutter is fitted can be used for the type of treatment described in this paper. The filter precludes accurate assessment of the visible effect of the retinal burn. The area of whitening is seen only when the laser is switched off. Moreover 'closure' of the vessel being treated is not observed during treatment but is seen shortly afterwards. Thus the time duration of treatment must be empirical and awaits definitive histopathological investigation.

As the time duration of laser application is reduced and the energy level increased, the focal temperature becomes higher. This can cause vaporisation and haemorrhage in the target tissue. Photocoagulation regimens which include initial treatment of the feeder vessel to cause spasm have been described. However, we suggest that by increasing the time and concomitantly reducing the energy the safety margin for treating retinal new vessels is increased. Furthermore, direct closure of retinal new vessels by using multiple laser applications is time consuming for the clinician and is uncomfortable for the patient. On the basis of our experience with this patient we would suggest that any studies designed to delineate the optimum energy and time criteria for vessel closure could begin with the findings we have described.

References


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