

Treatment of radiation retinopathy

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SUMMARY A case of radiation induced retinopathy with neovascularisation arising from the optic disc and retina was treated with panretinal argon laser photocoagulation. Complete regression of the new vessels was observed within 2 weeks of therapy. The pathogenesis of neovascularisation in this condition is discussed.

Radiation retinopathy was first described in 1935¹ and consists of microaneurysms, retinal haemorrhages, cotton-wool patches, perivascular sheathing, exudates, cystoid macular oedema, and new vessels arising from both the optic disc and the retina.²⁻¹⁰ Optic atrophy and central retinal artery occlusion

appeared, there is very little information on the successful treatment of the neovascularisation frequently encountered in this condition. Two cases of radiation retinopathy have been described in which macular oedema was treated by focal light coagulation.^{8,10}



Fig. 1 Radiation retinopathy. Left: Disc and retinal new vessels in the left eye. Right: Sheathing of the inferonasal retinal arteriole in the left eye.

have also been described.^{2-4,6,9,11} Optic disc and retinal neovascularisation may bleed, resulting in vitreous haemorrhage.^{3,12,13} Though many reports on the pathogenesis of radiation retinopathy have

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We report here a case of radiation retinopathy in which optic disc and retinal new vessels were treated successfully with argon laser photocoagulation. We believe this is the first reported case of radiation retinopathy in which optic disc and retinal neovascularisation has regressed after panretinal argon laser photocoagulation.

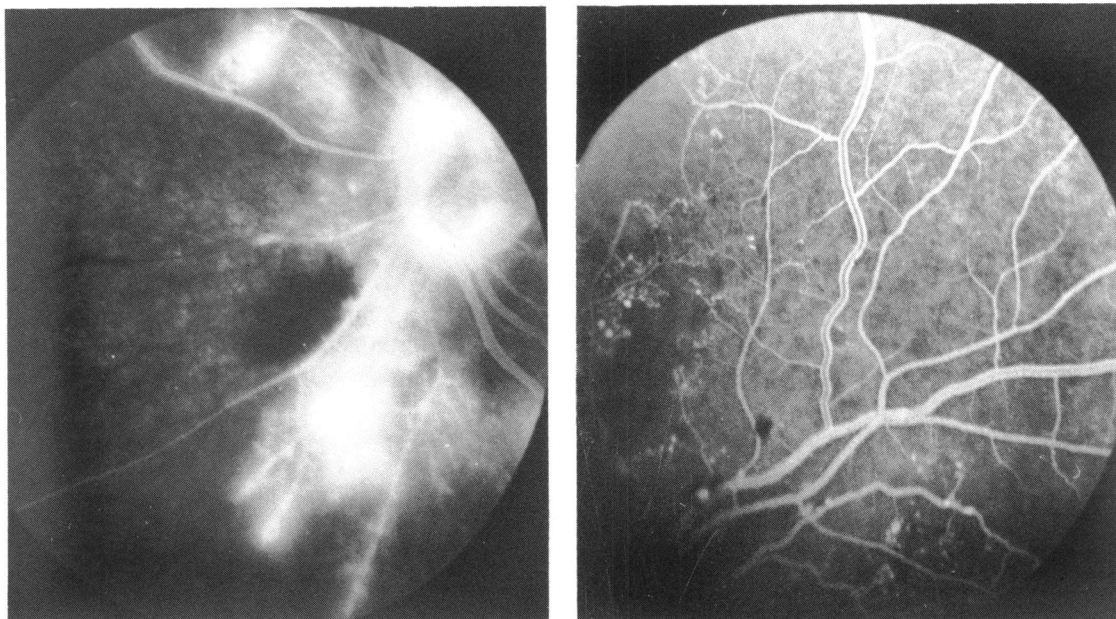


Fig. 2 Fluorescein angiographic view. Left: Extensive capillary closure and leakage from disc and retinal neovascularisation. Right: Dilated capillaries adjacent to the areas of capillary closure.

Case report

A 47-year-old man had a left transantral ethmoidectomy in September 1978 for a rather poorly differentiated small cell carcinoma. After that he received beam-directed supervoltage irradiation to the left paranasal sinuses between 21 November and 21 December 1978. The total dose given was 6000 rads in 4 weeks. Lead shields were placed over the corneas. No ocular complaints were present at the time of radiation.

On 14 November 1980 he was seen in the ophthalmic casualty department of Leicester Royal Infirmary with sudden onset of blurring of vision in the left eye. On examination vision in the right eye was 6/6 and in the left reduced to 6/18. Anterior segment examination of both eyes was normal, with clear lenses. Left fundus examination revealed vitreous haemorrhage obscuring the fundus details. Sheathing of the inferonasal retinal arteriole was seen in the right fundus. The vitreous haemorrhage in the left eye cleared with bed rest, at which time the left fundus showed the classical findings of radiation retinopathy. In the posterior pole microaneurysms, scattered nerve fibres, and deeper retinal haemorrhages were observed. In addition new vessels were seen arising from the optic disc as well as from the retina above and below the disc. There was extensive sheathing of the inferonasal retinal arteriole (Fig. 1).

Fluorescein angiography revealed large areas of capillary closure in the nasal retina both above and below the disc. Some dilated capillaries were observed adjacent to these areas of capillary closure. Leakage of fluorescein from disc and retinal new vessels was also present (Fig. 2).

The patient was observed closely without therapeutic intervention. A month later examination of the left eye revealed fresh vitreous haemorrhage and obvious increase in the size and number of retinal and optic disc new vessels. Because of the worsening of the retinal picture panretinal argon laser photocoagulation was carried out in the left eye. The patient received 2090 burns of 500 μm spot size in one sitting. No direct treatment of new vessels was performed. Two weeks later repeat fluorescein angiography revealed complete regression of peripheral and optic disc new vessels (Fig. 3). During the next 4 months follow-up examination showed no recurrence of the neovascularisation.

Discussion

Clinically apparent retinopathy is a delayed complication of radiation therapy.⁴ The changes usually appear after a latent period that varies between 18 months and 3 years, though extremes of one month to 15 years have been reported.¹⁴ It may follow either local irradiation for retinoblastoma or malignant melanoma

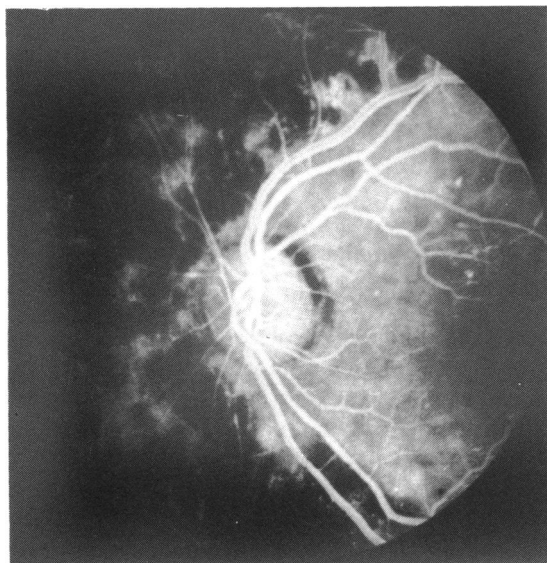


Fig. 3 Fluorescein angiography shows complete regression of disc and retinal new vessels following panretinal photocoagulation.

of the choroid^{1 4 14 15} or irradiation to lesions adjacent to the eye such as paranasal sinus tumours.^{6 8 13} Patients vary in the response of their retinal vessels to radiation, but it is agreed that approximately 6000 rads is sufficient to produce the retinopathy.^{6 16}

Postradiation retinopathy is an ischaemic retinopathy in which sheathing and occlusion of retinal vessels occur.¹⁷ Fluorescein angiography reveals microaneurysms, areas of capillary closure, and leakage from optic disc and retinal new vessels.³ The clinical picture and that of fluorescein angiography resemble those seen in diabetic retinopathy and other forms of retinal vascular occlusive disease.³ Histologically the affected vessels have thickened hyalinised walls, and the lumen may be occluded by a fine fibrillary material which is PAS-negative.¹¹ The exact cause of the optic disc and retinal neovascularisation in radiation retinopathy is unknown. According to Martin and Reese⁵ a possible factor in the formation of telangiectasia and new vessels is atresia of a small portion of the normal smaller veins and capillaries as a result of irradiation, with consequent dilatation of the vessels to provide collateral circulation. Another explanation may be the presence of a vasoproliferative factor produced by the hypoxic retina similar to that factor thought to cause the lesions in diabetic retinopathy.¹⁸ This latter theory is substantiated by the presence of extensive areas of capillary closure in

the nasal retina seen in our case, with the new vessels probably resulting from the extensive retinal hypoxia. Further evidence to support the theory of a vasoproliferative factor is the dramatic response of the disc vessels to retinal ablation with laser photocoagulation.

Panretinal photocoagulation of optic disc new vessels in this condition destroys areas of hypoxic retina. The rationale of this treatment is that if hypoxic retina is made anoxic the stimulus for neovascularisation will be eliminated.¹⁷ In the present case the complete disappearance of optic disc and retinal new vessels within 2 weeks of panretinal laser photocoagulation supports the argument for using this mode of therapy.

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