Fluorescein Angiographic Features, Natural Course and Treatment of Radiation Retinopathy

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Summary

Fifteen patients who developed retinopathy following radiotherapy for cephalic tumours were studied by fluorescein angiography. Nine patients with unilateral or bilateral retinopathy had serial angiograms at six monthly or yearly intervals. Angiography revealed a wide range of retinal microvascular changes which were graded 1-4 according to the extent and degree of capillary malformation, incompetence and closure. All patients showed slow progression of retinopathy with remodelling of the affected microvasculature and increased capillary fallout and leakage to dye.

The earliest retinopathic changes were capillary dilatation and closure and microaneurysm formation. Telangiectatic-like vessels were a feature of the established retinopathy and probably represented collateral channels which bordered sites of capillary occlusion.

Two patients with progressive macular oedema and declining vision responded favourably to focal laser photocoagulation which returned a measure of competence to some residual dilated capillaries.

The first fluorescein angiographic features of radiation retinopathy were described by Chee¹ and Gass² who reported a microvasculopathy characterised by capillary incompetence and closure. In 1970, Hayreh³ studied three patients with choroidal melanoma treated by Cobalt 60 applicators and documented a wide range of angiographic changes in the retinal vasculature and outer retina. Bagan and Hollenhorst⁴ reported six cases of radiation retinopathy but angiographic changes were only described in one patient. Brown *et al.*⁵ reviewed the angiographic changes in the retinae of patients receiving external beam or plaque therapy for intraocular tumours and identified vascular occlusion as the key pathophysiological event

leading to the development of radiation retinopathy.

Thompson *et al.*⁶ studied seven patients with radiation retinopathy and highlighted the value of fluorescein angiography in accurately identifying the microvascular alterations. Ehlers and Kaae⁷ also used fluorescein angiography to determine the extent of retinal and choroidal vaso-occlusive changes in infants receiving radiotherapy for retinoblastoma. Lommatzsch *et al.*^{8,9} and Tarkkanen and Laatikainen¹⁰ documented the regression of choroidal melanomas treated with Ru 106/Rh 106 plaques using angiographic techniques. They reported the disappearance of most choroidal and retinal vessels within the irradiated area and noted that chorioretinal

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vascular occlusion and retinal pigment epithelial atrophy continued for one year or more following irradiation.

Several authors using fluorescein angiography have commented on the inexorable nature of radiation retinopathy,^{5,6,11,12} although some have noted its progression may be slow.⁴ Natural regression of the disease process has also been reported in a few instances.^{13,14,15}

Most studies of radiation retinopathy to this time are either case reports or single observations in small cohorts of patients receiving ocular brachytherapy or teletherapy. In most instances the disease process is advanced or end-stage and few descriptions of the early vascular changes have been available. Sequential investigations of retinopathy have been reported from time to time;^{4,6,15} however, the natural course of the vasculopathy, particularly the early stages, has not been studied in detail. This paper describes the fluorescein angiographic changes in the retinae of patients receiving cephalic radiation emphasising the initial vascular alterations and documenting the natural course of the disease process. The modifying effects of laser photocoagulation on the natural progression of the vasculopathy are also examined.

Materials and Methods

Fifteen patients who developed retinopathy following cephalic radiation in Northern Ireland between 1972 and 1988 were studied by fluorescein angiography. The tumour pathology, tumour sites, radiation doses and methodology are documented in Table I.

Each patient had a full assessment of visual functions, ocular biomicroscopy and fundus examination. Fluorescein angiograms were obtained with a Zeiss fundus camera following an intravenous injection of 5 ml of 20% sodium fluorescein. High quality angiograms in one patient permitted a quantitative assessment of microvascular changes using a Kodak Image Analyser.

Nine patients with retinopathy were followed, and serial angiograms taken at six month or one year intervals. In six patients review was not possible due to advancing lens or corneal opacities or deteriorating health. The time of follow-up varied between 13 and

58.5 months, (mean follow-up time 25.8 months) (Table II).

Two patients had laser photocoagulation for progressive retinopathy and persistent macular oedema. As the affected microvasculature involved the central macula in both instances, the Argon green laser was chosen. Photocoagulation was applied to microvascular lesions, particularly microaneurysms and telangiectatic-like capillaries and nearby retina. Areas of ischaemic and poorly perfused retina were also treated. No burns were placed within 750 μm of the centre of the foveola. The first treated eye received a total of 448 burns (100 $\mu\text{m} \times 0.1 \text{ sec} \times 0.3 \text{ W}$) in two sessions. The second treated eye received 250 burns (100 $\mu\text{m} \times 0.1 \text{ sec} \times 0.18 \text{ W}$). Supplementary treatment of 181 burns (100 $\mu\text{m} \times 0.1 \text{ sec} \times 0.2 \text{ W}$) was given to the second patient after three months.

Results

In most patients with established radiation retinopathy angiography demonstrated striking and characteristic changes in the architecture and structure of the retinal microvasculature with varying degrees of capillary incompetence and closure. Typically, the retinopathy was concentrated in the macular and peripapillary regions; however, two patients in whom the radiation field was eccentric to the eye developed asymmetrical retinopathy corresponding to the respective fields of irradiation. One of these patients, (Case 3), a diabetic without discernible retinopathy showed widespread microaneurysms, haemorrhages and ischaemia in the irradiated superior retina of the right eye (Fig. 1). The inferior retinae displayed little or no evidence of retinopathy. The second patient (Case 15) with asymmetrical retinopathy had received radiation for a malignant melanoma of the nose and the inferior retina had been included in the radiation field. Subsequently this patient presented with extensive areas of ischaemia in the inferior retina associated with preretinal neovascularisation.

Three patients showed evidence of retinal pigment epithelial degenerative changes particularly atrophy and proliferation. A further patient who received direct orbital irradiation demonstrated a large subretinal

Table 1 Radiation retinopathy—a fluorescein angiographic study

Case No.	Patient	Eye	Tumour site	Radiation, dose fractionation	Date	Chemotherapy	Systemic disease	Angiography initial date
1	A.C.	R	Nasopharynx	5250 cGy/25f	Sept 1981			23.9.86
2	B.C.	R	(L) Nose and	5000 cGy/25f	Jan 1984			7.5.85
		L	Ethmoid					29.1.87
3	E.D.	R	(L) Parieto-temporal lobe	4000 cGy/30f	Nov 1984	CCNU*	Diabetes mellitus 1979	11.10.86
4	H.D.	L	(L) Lacrimal sac	2750 cGy/20f	Aug 1983	Vincristine, Cyclophosphamide, 5-FU†		9.5.88
5	A.H.	R	(R) Orbit	3350 cGy/15f	Jan 1972			26.3.87
6	D.H.	R	(R) Orbit	4800 cGy/20f	Dec 1978	Actinomycin D Vincristine, Cyclophosphamide		17.10.88
7	S.J.	R	Nasopharynx	5000 cGy/25f	March 1978			27.5.86
		L						27.5.86
8	H.K.	L	(L) Orbit	3417 cGy/15f	Nov 1981			27.4.88
9	H.M.	R	(R) Maxillary antrum Ethmoid	4350 cGy/20f	Oct 1977			3.6.86
10	J.M.	R	(R) Orbit	3500 cGy/19f	Jan 1981		Diabetes mellitus 1987	28.4.88
11	A.M.	R	Nasopharynx	5250 cGy/25f	Feb 1978			6.1.87
		L						6.1.87
12	N.O.	L	(L) Lacrimal sac	5500 cGy/25f 2400 cGy/25f	Feb 1988		Diabetes mellitus 1987	7.12.87 7.12.88
13	J.O.	R	Nasopharynx	4424 cGy/24f	Oct 1983	5FU†	Diabetes mellitus 1982	30.6.86 30.6.86
		L						6.1.87
14	M.S.	L	(L) Maxillary antrum	5460 cGy/26f	Nov 1979			6.1.87
15	A.T.	L	(L) Nose	5000 cGy/25f	Jan 1981	Tamoxifen (for breast Ca)		24.6.86

* CCNU 1-(2-Chlorethyl)-3-Cyclohexyl-1-Nitrosurea.

† 5FU 5-Fluorouracil.

neovascular membrane at the macula. Details of the angiographic findings and follow-up period for each patient are listed in Table II.

1. Grading of angiographic changes

All varieties of reported microvascular response to radiation were identified in this study, and 15 patients (20 eyes) were classified according to the degree and extent of the microvascular changes.

Grade 1 Retinopathy

Two eyes demonstrated small foci of dilated and irregular retinal capillaries in association with isolated or small clusters of microaneurysms. These eyes also showed subtle evidence of capillary closure, although there was no detectable microvascular incompetence or

fluid accumulation within the retina (Fig. 2). Visual functions were good in these patients i.e. 6/6 or better.

Grade 2 Retinopathy

Seven eyes showed multiple foci of dilated and telangiectatic-like capillaries and zones of capillary closure up to one optic disc area in dimension. This group also showed numerous microaneurysms and focal leakage of dye from defective capillaries in later phase angiograms (Fig. 3a, b). In some patients fluorescein leakage was associated with clinically observable retinal oedema, although cystoid macular changes were absent and visual acuity remained relatively good, i.e. 6/9 or better. One patient (two eyes) in this group (Case 13) was diagnosed as suffering from

Table II Radiation retinopathy—a fluorescein angiographic study

Case No.	Eye	Initial angiographic findings	Final angiographic findings	Follow-up period	Retinopathy grade
1	R	Small area, irregular capillaries 2 microaneurysms. Focal capillary closure	Large single microaneurysm increased capillary irregularity, further capillary closure	32 months	1
2	R	Focal capillary dilatation and closure, microaneurysms and intraretinal haemorrhages. Focal RPE changes	Increased area capillary dilatation and number of microaneurysms. Fusiform capillary loops further capillary fallout. No vascular incompetence	31 months	1
	L	Focal macular and perimacular inner retinal ischaemia, intraretinal haemorrhage, widespread capillary dilatation, distortion and incompetence. Multiple microaneurysms	Incomplete reperfusion ischaemic retina, absorption haemorrhage. Development telangiectatic-like channels. Macular oedema	58 months	3
3	R	Widespread capillary defects IRMA, microaneurysms and inner retinal ischaemia superior retina. Arteriolar and venular occlusion.			4
4	L	Dilated irregular incompetent capillaries. Microaneurysms, capillary closure, RPE scar, inferior macula	Slight increase extent capillary abnormalities and microaneurysms. Dye leakage and retinal staining more intense	13 months	2
5	R	Extensive inner retinal ischaemia, IRMA, patent and thrombosed microaneurysms. Macular RPE changes, subretinal neovascularisation			3
6	R	Dilated, disorganised macular capillaries. Microaneurysms IRMA, telangiectatic-like vessels, cystoid macular oedema, retinal ischaemia			3
7	R	Dilated, irregular capillaries. Multiple microaneurysms, capillary closure and staining of defective capillary bed, retinal oedema	Further capillary closure, thrombosis and formation microaneurysms increased dye leakage	37 months	2
	L	Scattered dilated and incompetent caps, focal leakage of dye into retina. Microaneurysms and focal capillary closure	Small increase in microaneurysms and area of vascular staining and incompetence	38.5 months	2
8	L	Dilated telangiectatic like capillaries, microaneurysms, focal retinal ischaemia	Further closure capillary bed, reperfusion of some radicals within ischaemic retina	17 months	3
9	R	Dilated, incompetent capillaries, microaneurysms, choroidal vascular stasis, macular oedema			3
10	R	Telangiectatic-like vessels, microaneurysms cystoid macular oedema. Retinal ischaemia. Venular and arteriolar staining	Extension of microvascular abnormalities, increased capillary incompetence. Some reperfusion of ischaemic retina	14 months	3
11	R	Widespread capillary closure, IRMA's and telangiectatic-like channels. Irregular arterioles and venules, macular oedema	Further closure of abnormal dilated capillaries, additional haemorrhages and increased microvascular leakage	28 months	4
	L	Dilated telangiectatic capillaries. Microaneurysms, early leakage capillary closure	Additional microaneurysms increased microvascular incompetence		2
12	L	Microaneurysms focal RPE defects	Gross macular oedema, ischaemia, staining venules and arterioles	11 months	4
	R	Microaneurysms small foci, incompetent, occluded vessels	Further microaneurysms, increased microvascular leakage		2
13	R	Moderate background diabetic retinopathy. Focal capillary closure and leakage	Intensification of retinopathy, increased capillary leakage	9 months	2
	L	Microaneurysms, capillary leakage	Increased microaneurysms and dye leakage		2
14	R	Telangiectatic-like macular vessels, microaneurysms, focal capillary closure macular oedema			3
15	L	Telangiectatic-like channels, extensive retinal ischaemia, preretinal neovascularization			4

* RPE Retinal pigment epithelium. IRMA Intraretinal microvascular abnormality.

Fig. 1. Venous phase angiogram right posterior fundus, Case 3. Ischaemic retinopathy is largely confined to the irradiated superior fundus.

diabetes mellitus one year prior to receiving radiotherapy and chemotherapy for a nasopharyngeal tumour. It was not possible to determine whether the retinopathy was pri-

marily due to the metabolic or radiational insult, although it is likely that it was a response to the combined or synergistic effects of radiation, chemotherapy and diabetes.

Grade 3 Retinopathy

Seven eyes in this group were characterised by widespread capillary dilatation, telangiectatic-like channels, microvascular incompetence and significant areas of capillary closure (1–4 disc areas). All patients had significant macular oedema with or without cystoid macular degenerative changes (Fig. 4). Microaneurysms and intraretinal microvascular abnormalities commonly occurred at the border of perfused and non-perfused retina. Eyes in this group typically had poor visual acuity, i.e. five eyes had vision of 6/18 or worse. One patient in this group was diagnosed a diabetic six years following radiation to the right orbit (Case 10). The retinopathy was severe and unilateral and was judged to be primarily radiation in type, although the metabolic

Fig. 2a. Mid-venous phase angiogram, right posterior fundus, Case 1. Capillary irregularities and microaneurysms in the superior macula (arrows) represent early radiational changes. Retinal pigment epithelial changes are present at the foveola.

Fig. 2b. Angiogram right fundus, Case 1, 32 months after that shown Fig. 2a. There is evidence of capillary fusion and closure. The wall of a dilated fusiform capillary stains with dye (arrow).

Fig. 3a. Venous-phase angiogram right posterior fundus Case 7. The macular microvasculature is grossly dilated and displays multiple microaneurysms of varying size. Small foci of capillary nonperfusion are evident.

Fig. 3b. Late phase angiogram of fundus shown Fig. 3a. Many dilated channels and microaneurysms stain and leak fluorescein into the nearby retina.

Fig. 3c. Venous-phase angiogram of fundus shown Fig. 3a, taken 37 months later. There is extension and intensification of retinopathy with capillary remodelling and appearance and disappearance of microaneurysms. The area of nonperfused retina has increased.

Fig. 3d. Late phase angiogram of fundus shown Fig. 3c. Dye leakage has increased compared with Fig. 3b.

Fig. 3e. Venous phase angiogram following laser photocoagulation to fundus, shown above, 2 months after angiograms Figs. 3c and d. Some defective capillaries are occluded and others reduced in calibre. Microaneurysms are reduced in number.

Fig. 3f. Late phase angiogram, fundus shown Fig. 3e. Dye leakage is reduced except at one untreated area superonasal to foveola. This was subsequently treated.

abnormality may have been a contributory factor.

Grade 4 Retinopathy

Four eyes demonstrated widespread disorganisation of the retinal microvasculature with extensive inner retinal ischaemia. Non-perfused retina was in excess of four disc areas, and one eye in this group developed preretinal neovascularisation and vitreous haemorrhage (Fig. 5). Two eyes with ischaemic maculopathy had visions of 6/60 and 3/60; however, two eyes with retinal ischaemia eccentric to the fovea retained vision of 6/6 and 6/12.

Two patients in this group were diabetic and documented as having early background diabetic retinopathy prior to radiotherapy (Cases 3 and 12). The development of extensive ischaemic retinopathy in each case after radiotherapy reflects the combined microvascular insult of the two disease processes. The effects were particularly severe in case 12

Fig. 4a. Mid-venous phase angiogram left posterior fundus (Case 2). There is advanced radiation retinopathy with focal areas of retinal nonperfusion, dilated and incompetent capillaries, and intraretinal haemorrhage.

Fig. 4b. Venous phase angiogram fundus shown Fig. 4a, 18 months later. There is some reperfusion of ischaemic retina and development of incompetent telangiectatic-like channel over much of the nasal and superior macula.

where radiotherapy for a left lacrimal sac carcinoma resulted in severe ischaemia of the posterior retina at nine months and rubeosis iridis within twelve months of treatment.

2. Sequential study of radiation retinopathy

All nine patients (14 eyes) followed sequentially in this study demonstrated intensification and extension of retinopathy. No patient demonstrated any reversal of pathology or improvement in retinal microvascular competence, although attempts to recanalise non-perfused capillary beds were occasionally observed (Fig. 4). The progression of microvascular pathology varied in degree and extent (Table II), although the rate of change was typically slow, and in this series only one patient showed sufficient deterioration in the capillary bed to be regraded (Case 7, Fig. 3).

Over the period of observation all patients showed additional capillary closure and a net increase in the number of microaneurysms. In some eyes there was clear evidence of microaneurysm thrombosis and atrophy. All patients in groups 2 to 4 showed more microvascular incompetence with time as judged by the intensification of macular fluorescence in late phase angiograms. In one patient (Case 7) it was possible to quantify some of the retinal vascular changes and estimate the

area of microvascular non-perfusion using image analysis of high-quality angiograms. Over a period of three years this patient demonstrated a 50% nett increase in the number of microaneurysms and a 70% nett increase in the area of non-perfused retina in spite of a considerable turnover in the number of microaneurysms, and abortive attempts at revascularisation of ischaemic retina (Fig. 3a and 3c).

Three eyes followed in this study suffered loss of central vision concomitant with advancement of retinopathy, intraretinal fluid accumulation and macular ischaemia.

3. Treatment

Two patients in the present study received argon (green) laser photocoagulation for severe or advancing maculopathy associated with deterioration of vision. Focal laser photocoagulation was applied to areas of deformed and incompetent capillaries and regions of non-perfused retina (Fig. 3e, f). Both patients showed improvement in visual acuity (Case 7, 6/18 to 6/9) (Case 10, 6/36 to 6/12–2) within two to three months of therapy, and improvement was maintained at one year post treatment in each case.

Post-treatment angiography revealed that some deformed and telangiectatic vessels adjacent to photocoagulation burns collapsed

Fig. 5. Late venous phase angiogram of inferonasal fundus (case 15). There are multiple microaneurysms, extensive inner retinal ischaemia and a hyperfluorescent preretinal neovascular membrane.

and failed to perfuse with dye. Other dilated capillaries were dramatically reduced in calibre, and demonstrated increased competence to fluorescein. Some microaneurysms either decreased in size or disappeared, and in some instances became less permeable to dye (Fig. 3e, f). Photocoagulation did not result in any observable reperfusion of ischaemic retina. One patient with widespread retinal ischaemia, preretinal neovascularisation and vitreous haemorrhage was advised to have laser therapy but declined treatment.

Discussion

Radiation retinopathy subsequent to both brachytherapy and teletherapy has been the subject of fluorescein angiographic investigation by several workers. The early studies of Chee,¹ Gass² and Hayreh³ demonstrated that all components of the retinal microvasculature could be affected, and that angiography was useful in identifying microvascular pathology and demonstrating changes in vascular perfusion and competence. Subsequent angiographic investigations have also confirmed that the outer retina and choroidal circulation could be implicated in the disease process, and that photocoagulation could limit some of the sight-threatening complications of end-stage retinopathy.^{4,5,6,7,16,17}

The progressive nature of radiation retinopathy has been emphasised; however, the natural course of clinical radiation retinopathy is still poorly understood, largely due to the difficulties encountered in mounting longitudinal studies in patients who are often ill, who have a poor life expectancy, and who occasionally develop corneal and lens opacities limiting observation of the ocular fundus.¹⁸ The long latent period between irradiation and development of retinopathy and the typically slow progression of microvascular changes also make sequential analysis of the vasculopathy difficult and hinder prospective investigations. Furthermore, as early microvascular pathology is not usually associated with significant loss of vision, patients with early retinal changes are unlikely to be identified unless screening procedures are instituted.

The retinal microvascular alterations induced by ionizing radiation are observed

most accurately in patients undergoing radiotherapy for extraocular tumours and orbital disease, e.g. lymphoma and Grave's Disease, as radiotherapy of intraocular tumours introduces not only the impact of ionising radiation on the retinal vessels but also the secondary effects of the tumour and its postradiation necrotic products on the retinal neuropile and microvasculature. Brachytherapy for intraocular tumours also causes local obliteration of the choroidal circulation^{3,8,9,10} and profound alterations in the nearby retinal pigment epithelium^{3,9} which may influence the form and function of the nearby retinal microvasculature.

This study was successful in identifying early radiation retinopathy and monitoring the development of microvascular changes as a large number of patients receiving substantial ocular radiation were screened and a significant proportion of these had early pathology. Furthermore, some patients with bilateral retinopathy had advanced disease in one eye but only early or incipient changes in the fellow eye which were particularly suitable for angiographic analysis. Also, in most patients radiotherapy was successful in containing the primary malignant disease process thereby facilitating long term observation.

The earliest identifiable changes of radiation retinopathy (Grade 1) were focal capillary closure and irregular dilatation of the neighbouring microvasculature. The affected vessels were typically competent in early disease although their walls often stained with dye in late angiography. Retinal oedema was not a feature and visual functions were not measurably affected. With time, further localised collapse of the capillary bed occurred, with development of additional dilated irregular capillaries. These dilated and telangiectatic-like vessels probably represent capillary collaterals which divert blood from defunct microvascular units.

The precise aetiology of capillary closure is unknown, however, it is likely that ionizing radiation directly affects vascular endothelial cell viability or its potential to replicate, and the subsequent failure of the capillary endothelium precipitates platelet deposition, capillary thrombosis and focal atrophy of the retinal microvasculature.^{3,5,7,14} Microaneu-

rysms were also a feature of early retinopathy and typically formed in dilated capillaries which bordered perfused and non-perfused capillary beds. Their formation may be due to the effects of a local increase in intra-luminal pressure on channels which have been structurally compromised by the loss of either endothelial cells or pericytes.

As early radiation retinopathy advanced, a significant part of the capillary bed, usually including the macula, became involved (Grade 2). Dilated, tortuous and fusiform capillaries were the principal angiographic feature and microvascular incompetence occurred as judged by fluorescein leakage. Despite the presence of dye leakage, intraretinal accumulation of fluid was often not pronounced and central visual functions were frequently surprisingly good despite involvement of the macular and perifoveolar circulations. The absence of clinically discernible intraretinal fluid accumulation is probably a measure of the efficiency of neighbouring intact capillaries and local retinal pigment epithelium in dehydrating the affected retina.

Further progression of retinopathy is characterised by the closure of additional capillary beds, formation of intraretinal microvascular abnormalities¹⁸ and the development of additional microaneurysms (Grade 3). A quantitative analysis of such effects in one patient revealed that the number of microaneurysms doubled in three years and that the area of capillary-free retina also increased by a factor of about two. More generalised microvascular incompetence was associated with clinically evident retinal oedema, and where the foveola was implicated, visual functions declined sharply.

End stage radiation retinopathy (Grade 4) is characterised by generalised microvascular disorganisation, inner retinal ischaemia and capillary incompetence often leading to cystoid macular oedema. The degree of inner retinal ischaemia in Grade 4 eyes may be sufficient to promote preretinal neovascularisation. Other authors have also described papillary neovascularisation,^{6,18,19} vitreous haemorrhage,^{16,19} retinal detachment, rubeosis iridis and secondary glaucoma in such eyes which usually have received high doses of ocular radiation.^{16,19}

Angiographic evidence of choroidal closure or atrophy has mostly been described in patients having radioactive plaque therapy for choroidal melanomas and retinoblastomas^{7,9,10}. The only angiographic changes in the choroid following teletherapy were noted by Thompson *et al.*⁶ who described delayed choroidal filling in patients receiving radiation for nasopharyngeal tumours. Choroidal vascular stasis, however, was only encountered in hypertensive patients and was not attributed specifically to radiation damage. The present study has demonstrated angiographically that the choroidal circulation can be compromised by gratuitous external beam irradiation of the eye, and that subretinal neovascularisation may occur in association with radiation retinopathy. Whether any causal relationship exists between choroidal neovascularisation and radiotherapy is not known; however, degenerative changes induced in the retinal pigment epithelium and outer retina by radiation may encourage the development of subretinal neovascularisation particularly in ageing patients with evidence of drusen formation.

This study has confirmed that laser photocoagulation can have a beneficial effect on radiation-induced maculopathy and suggests that patients who benefit most from photocoagulation are those with early to moderate macular oedema, without cystoid degenerative changes and who retain moderate visual functions, i.e. 6/24 or better. Kinyoun *et al.*¹⁶ and others^{6,17,19} have noted that laser photocoagulation not only has a positive effect on radiation maculopathy but also serves to contain or eliminate preretinal and papillary neovascularisation, thereby reducing the incidence of vitreous haemorrhage and retinal detachment.

The mechanism whereby laser photocoagulation improves retinal vascular competence is not known, although this study and others confirm that judicious photocoagulation effectively occludes and ablates some incompetent capillaries and microaneurysms. This study also demonstrates that laser photocoagulation indirectly reduces the calibre of dilated and deformed capillaries, possibly by diminishing cell density in the outer retina in the vicinity of the affected microvasculature.²⁰

Theoretically the depopulated and attenuated retina will have lower metabolic requirements and may permit better oxygenation of the inner retina and its vascular cells from the choroidal circulation.^{21,22} Photocoagulation burns may also create defects in the retinal pigment epithelial layer, producing conduits which facilitate the drainage of excess retinal fluid into the choroidal circulation.²³ Alternatively, it could be argued that, following laser photocoagulation, the rejuvenated retinal pigment epithelium has more effective pumping properties and is consequently able to dehydrate the oedematous retina. Nevertheless, irrespective of the mechanism whereby laser photocoagulation improves microvascular function in radiation retinopathy it is clearly an important therapeutic tool, particularly in early maculopathy and vasoproliferative disease. Accordingly, we would suggest that patients receiving in excess of 3,000 cGy to the eye should be regularly reviewed and that patients receiving greater than 5,000 cGy to the eye should be closely observed. Ophthalmologists should also liaise with radiotherapists carrying out cephalic radiation, particularly to patients with nasopharyngeal and paranasal sinus tumours, both to acquaint the radiotherapist regarding the likelihood of radiation retinopathy and to encourage where possible effective screening of the eye during treatment of these malignant tumours.

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