Radiation Retinopathy and Papillopathy

6.26

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Definition: A slowly progressive occlusive vasculopathy affecting the retina, choroid, and optic nerve, with a delayed onset after irradiation.

Key Features

- Retinal microaneurysms.
- Retinal hemorrhages.
- Retinal telangiectatic vessels.
- Retinal hard exudates.
- Macular edema.
- Cotton–wool spots.
- Optic disc swelling.

Associated Features

- Retinal neovascularization.
- Disc neovascularization.
- Vitreous hemorrhage.
- Iris neovascularization.
- Optic atrophy.
- Occlusive choroidal vasculopathy.
- Polypoidal choroidal vasculopathy.

RADIATION RETINOPATHY

Radiation retinopathy (RR) is characterized by a slowly progressive occlusive vasculopathy with a delayed onset after irradiation.[1] Radiation retinopathy was first described in 1933 by Stallard, who noted retinal exudation, retinal hemorrhage, retinal pigment epithelial changes, and optic disc edema in patients treated with radon seeds for retinal tumors.[2] In 1935, Moore expanded on the initial observations made by Stallard.[3]

RR has been documented after both plaque radiotherapy (also termed *brachytherapy*) and external beam radiotherapy (EBRT) (also termed *teletherapy*) following treatment of intraocular tumors. RR can also develop after EBRT of orbital, paranasal sinus, and intracranial tumors, as well as thyroid ophthalmopathy and orbital inflammation.[1,4] In recent years, a better understanding of the biological effects of ionizing radiation, development of techniques enabling more accurate focusing of radiation to target tissues, and earlier diagnosis and treatment have helped decrease the risk of vision loss resulting from RR.

Pathology and Pathogenesis

Histopathological studies of the eyes with RR confirm the predominantly vascular nature of damage produced by radiation therapy. There is an unequivocal loss of endothelial cells with relative sparing of the pericytes.[1,4,5] This is in contrast to diabetic retinopathy, where the target cell is the pericyte instead of endothelial cells.[5]

Radiation basically has two side effects on tissues.[5,6] First, radiation directly damages the endothelial cell DNA. The endothelial cells undergoing mitosis and proliferating at the time of radiation are most vulnerable and eventually succumb to death. As a result of this insult, the remaining endothelial cells migrate and divide in an effort to compensate for the damaged segments. However, if the damage is extensive, there is a relative endothelial cell deficiency, leading to a vasculopathy characterized by vascular leakage, platelet aggregation, and activation of the clotting cascade. Second, the indirect effect of radiation involves creation of free radicals (OH) from water contained within the cell cytoplasm. Free radicals are highly reactive and induce further DNA damage.[6] The differential sensitivity between endothelial cells and pericytes is the result of direct exposure of endothelial cells to high oxygen and iron found in blood, which generates some of the free radicals.[1,6] Cell damage usually starts on the arterial side of the circulation, where free radical generation is greater because of higher oxygen tension.[1,6] Small retinal vessels are involved first, leading to capillary closure. Larger retinal vessels and choroidal vessels can also be affected later in the course of the disease.

Clinical Features

RR is suspected when there is a history of radiation exposure and ocular examination reveals the characteristic fundus findings. RR has a clinical course similar to diabetic retinopathy.[1,7–9] Therefore, it is prudent to classify RR as nonproliferative radiation retinopathy (NPRR), proliferative radiation retinopathy (PRR), and radiation maculopathy (RM) similar to the classification in diabetic retinopathy.[7] In NPRR, retinal capillary changes, retinal edema, retinal hemorrhages, microaneurysms, telangiectatic vessels, hard exudates, vascular sheathing, and cotton-wool spots are evident. When these nonproliferative changes are present within 3 mm of the foveola, the condition is referred to as RM (Fig. 6.26.1). With the development of widespread areas of capillary nonperfusion, retinal and disc neovascularization develop, leading to PRR. If untreated, PRR can result in vitreous hemorrhage, iris neovascularization, and neovascular glaucoma.

Other manifestations of radiation effects on the posterior globe include radiation choroidopathy, including closure of choroidal vessels, leading to choroidal atrophy, polypoidal choroidal vasculopathy, and choroidal infarction.[7] Radiation-related retinal pigment epitheliopathy is another effect that manifests with mottling of the retinal pigment epithelium (RPE), hyperplasia, and eventual atrophy. The term RR is most often used in the literature to broadly encompass all fundus changes, including NPRR, PRR, and RM, as well as radiation choroidopathy and RPE changes.

RR can develop as early as one month to over 15 years after radiation exposure. However, it most commonly occurs between six months and three years.[4] Gündüz et al. reported that 5% of the 1300 patients treated with plaque radiotherapy had NPRR at 1 year and 42% at 5 years.[7] More serious PRR developed in 1% at 1 year and 8% at 5 years.[7] The Collaborative Ocular Melanoma Study (COMS) confirmed these clinical observations and assessed histopathological evidence of RR in enucleated eyes following plaque radiotherapy for melanoma. The authors of that study found that 55% of eyes had evidence of microvascular abnormalities related to RR.[10] Sagoo et al. found that for juxtapapillary melanoma (posterior margin within 1 mm of the optic disc), the risks of RR and RM were higher at 75% and 65%, respectively, after plaque radiotherapy.[11] These studies were based on ophthalmoscopic observations and done mostly before the availability of optical coherence tomography (OCT). Using OCT, Horgan et al. found an increased incidence of macular edema in 40% of eyes by 1 year and in 61% by 2 years after plaque radiotherapy for choroidal melanoma.[12] For proton beam radiation treatment of juxtapapillary and parapapillary melanomas, those located <1 disc diameter from the optic disc, the 5-year rate of RM was 60%.[13] A more recent study evaluating ciliary body as well as choroidal melanomas treated with proton beam irradiation found a 5-year RR rate of 85%.[14]

Diagnostic Studies

Fluorescein angiography (FA), OCT, and OCT angiography (OCTA) are diagnostic studies that are most helpful in the evaluation of eyes with RR. FA can demonstrate microvascular changes, capillary dropout, retinal nonperfusion, retinal and disc neovascularization, and macular edema[7–9] (see Fig. 6.26.1; Fig. 6.26.2). It is especially useful in classifying RM as

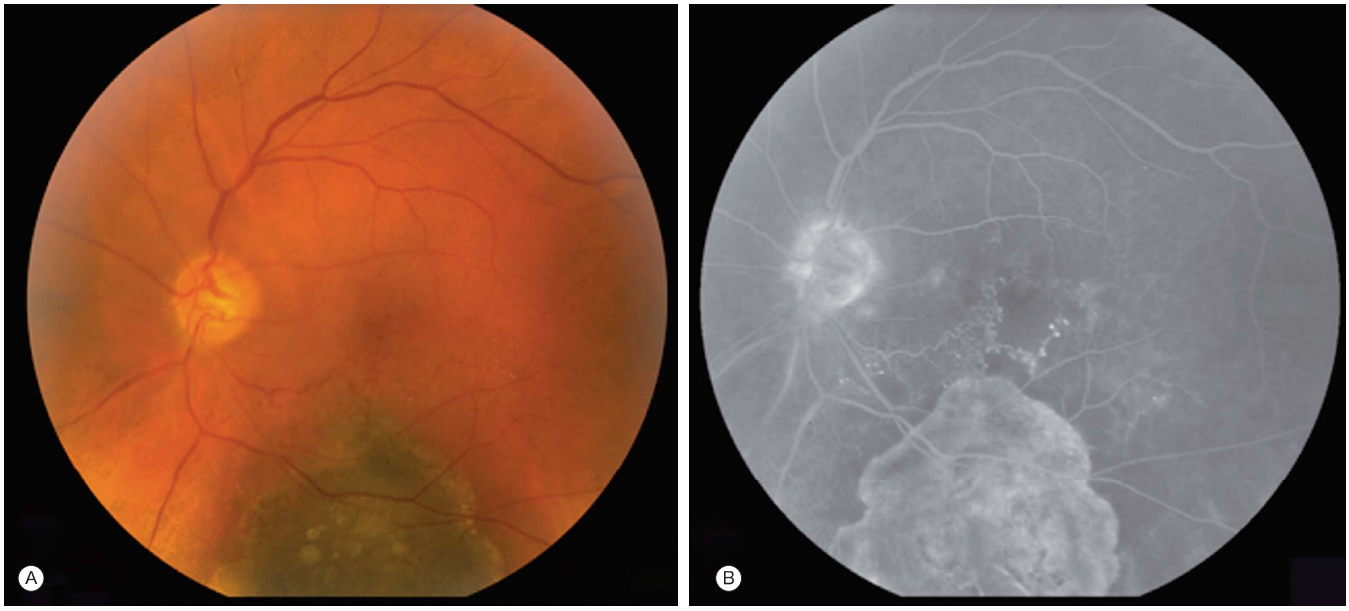


Fig. 6.26.1 Radiation maculopathy following plaque radiotherapy for choroidal melanoma (A) showing regressed melanoma inferiorly and retinal telangiectasia, microaneurysms, and exudation in the inferior macula. On fluorescein angiography (B), macular telangiectasia, microaneurysms, and nonperfusion are found. Staining of the optic disc implies radiation-related damage.

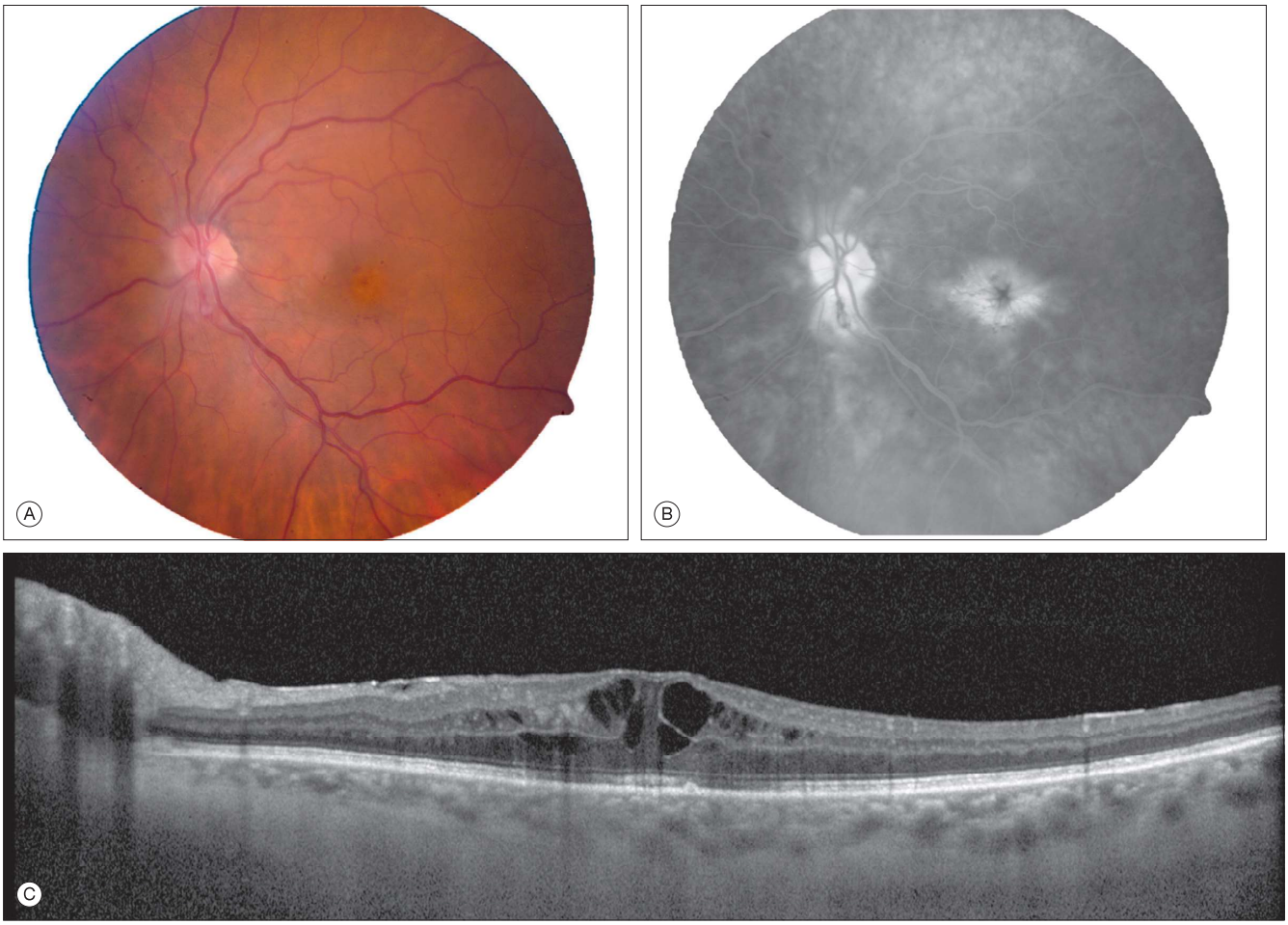


Fig. 6.26.2 Radiation maculopathy with retinal telangiectasia and hemorrhage (A), fluorescein angiographic petalloid staining (B), and optical coherence tomographic outer plexiform cystoid edema (C).

ischemic versus nonischemic. In eyes with ischemic maculopathy, there is little chance for visual improvement with currently available treatment methods. Eyes with nonischemic maculopathy are amenable to treatment, as discussed below.

Using FA, radiation-related macular edema is classified into diffuse, focal, and mixed types based on the pattern of dye leakage. This classification is useful if grid or focal laser treatments are to be applied. Indocyanine green angiography (ICGA) can demonstrate areas of choroidal hypoperfusion; however, this method of angiography is not routinely used in the evaluation of RR.

OCT is a noninvasive procedure that has the advantages of providing quantitative measurement of macular edema. OCT is an important modality in evaluating presymptomatic radiation-induced macular edema (see Fig. 6.26.2). A study by Horgan et al. found that OCT detected evidence of macular edema approximately 5 months earlier than in the case of clinically detected RM.[12] The median visual acuity at the time of OCT-evident macular edema was 20/40. Horgan et al. proposed a five-grade scale for radiation macular edema based on OCT findings:

Grade 1 – noncystoid edema extrafoveolar.
Grade 2 – cystoid edema extrafoveolar.
Grade 3 – noncystoid edema foveolar.
Grade 4 – cystoid edema foveolar.
Grade 5 – cystoid edema foveolar severe, simulating serous retinal detachment.

Increasing grade of macular edema was found to be associated with worse visual acuity. With the introduction of spectral domain OCT (SD-OCT) technology, better central foveal thickness measurements and superior visualization of the retinal layers became possible.

As a further step in diagnosis of RM, OCTA has come into use. OCTA is a noninvasive technology that can visualize both the superficial and deep capillary plexuses of the retina as well as choroidal vasculature in a noninvasive manner without injection of dye. In contrast, FA can visualize only the superficial retinal plexus and requires dye injection.[15,16] OCTA can demonstrate enlargement of the foveal avascular zone and reduction in parafoveal capillary density of both superficial and deep capillary plexuses as well as parafoveal nonperfusion, capillary dropout, microaneurysms and telengiectasia in eyes with choroidal melanoma after plaque radiotherapy, even in the absence of clinical and SD-OCT evidence of radiation maculopathy.[15,16] The deeper capillary plexus and choroidal vasculature may be affected more because radiation is delivered from the scleral side in plaque radiotherapy (Figs. 6.26.3 and 6.26.4). Therefore OCTA is currently the most sensitive method for detection of early RM and probably for assessment of its severity as well.

Electroretinography (ERG) studies are rarely used in the evaluation of RR and RM. ERG reflects the mass response of retina and may demonstrate decreased amplitudes in widespread RR.

Risk Factors

Brachytherapy

Tumor type, thickness, location, radioisotope type, and radiation parameters are important factors to consider in the development of RR following brachytherapy. With plaque radiotherapy, the risk of RR is related to total radiation dose. Total radiation dose is, in turn, related to tumor type. In the treatment of posterior uveal (ciliary body and choroidal) melanoma, therapeutic apical tumor doses range from 80 to 100 Gy, whereas in circumscribed choroidal hemangioma the apical dose is only approximately 20–25 Gy.

Total radiation doses that result in 5% and 50% complication rates at 5 years are defined as tissue tolerance doses—TD_5 and TD_{50}. The retina has a TD_5 value of 45 Gy and a TD_{50} value of 60 Gy.[17] First, with the therapeutic apical doses used for uveal melanoma, the retina and the macula are inevitably exposed to radiation doses in excess of the normal tissue tolerance doses discussed above. Therefore, RR is seen more often after brachytherapy for choroidal melanoma than that for similar-sized choroidal hemangioma because of the higher radiation dose used in the treatment for melanoma. Second, tumor thickness >4 mm has also been associated with a greater risk of RM.[18] The spillover radiation dose delivered to the surrounding retina is greater with increasing tumor thickness. Third, tumor location is a factor in the development of visually significant RM. Brown et al. found that RM did not develop if the fovea received a dose of ≤50 Gy in brachytherapy.[8] A subsequent study by Gündüz et al. validated this finding.[7] With brachytherapy for peripheral tumors, the fovea receives a smaller radiation dose, and the risk for RM is less. A number of radioactive isotopes, including cobalt-60, iridium-192, ruthenium-106, iodine-125, and pallidium-103, have been used in ocular brachytherapy. Of these, the use of cobalt-60 and iridium-192 has been almost totally abandoned because of the higher energy and marked radiation side effects of these radioisotopes. Radiation parameters including tumor apex dose >90 Gy, apex dose rate >75 cGy/hr, and base dose rate >260 cGy/hr were found to lead to RR on a univariate model in one study.[7] Coexisting morbidities, including diabetes mellitus, and concurrent chemotherapy increase the risk of RR in both teletherapy and brachytherapy.[4]

Teletherapy

Total radiation dose, fractionation, field design, and type and rate of administration are factors that affect the development of RR after EBRT. RR has

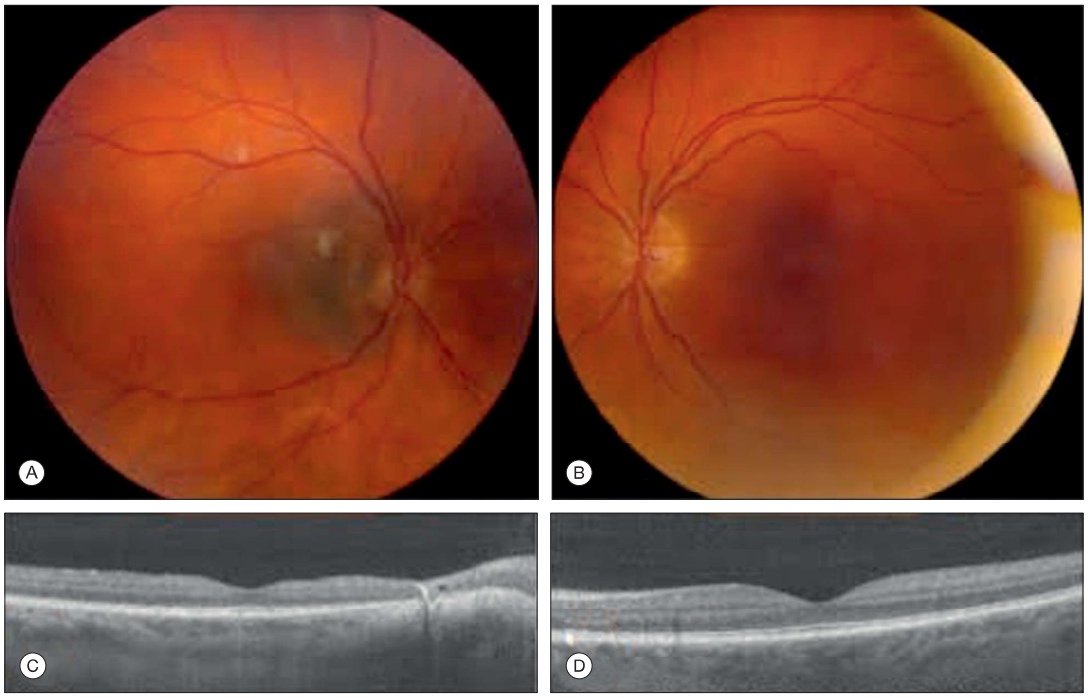


Fig. 6.26.3 Mild radiation maculopathy after plaque radiotherapy. Color fundus photographs show temporally located small, biopsy-proven irradiated juxtapapillary choroidal melanoma in the right eye (A) and normal fellow eye (B). Note the nerve fiber layer infarctions in the papillomacular region and superior macular region of the right eye (A). Spectral-domain optical coherence tomography (SD-OCT) of the right eye after irradiation shows regular foveal contour and normal central macular thickness with no evidence of intraretinal edema or subretinal fluid (C). There is retinal thinning in the papillomacular region from previous subretinal fluid and the vertical bright line through the retina marks the site of needle biopsy (C). Normal SD-OCT findings in the left eye (D).

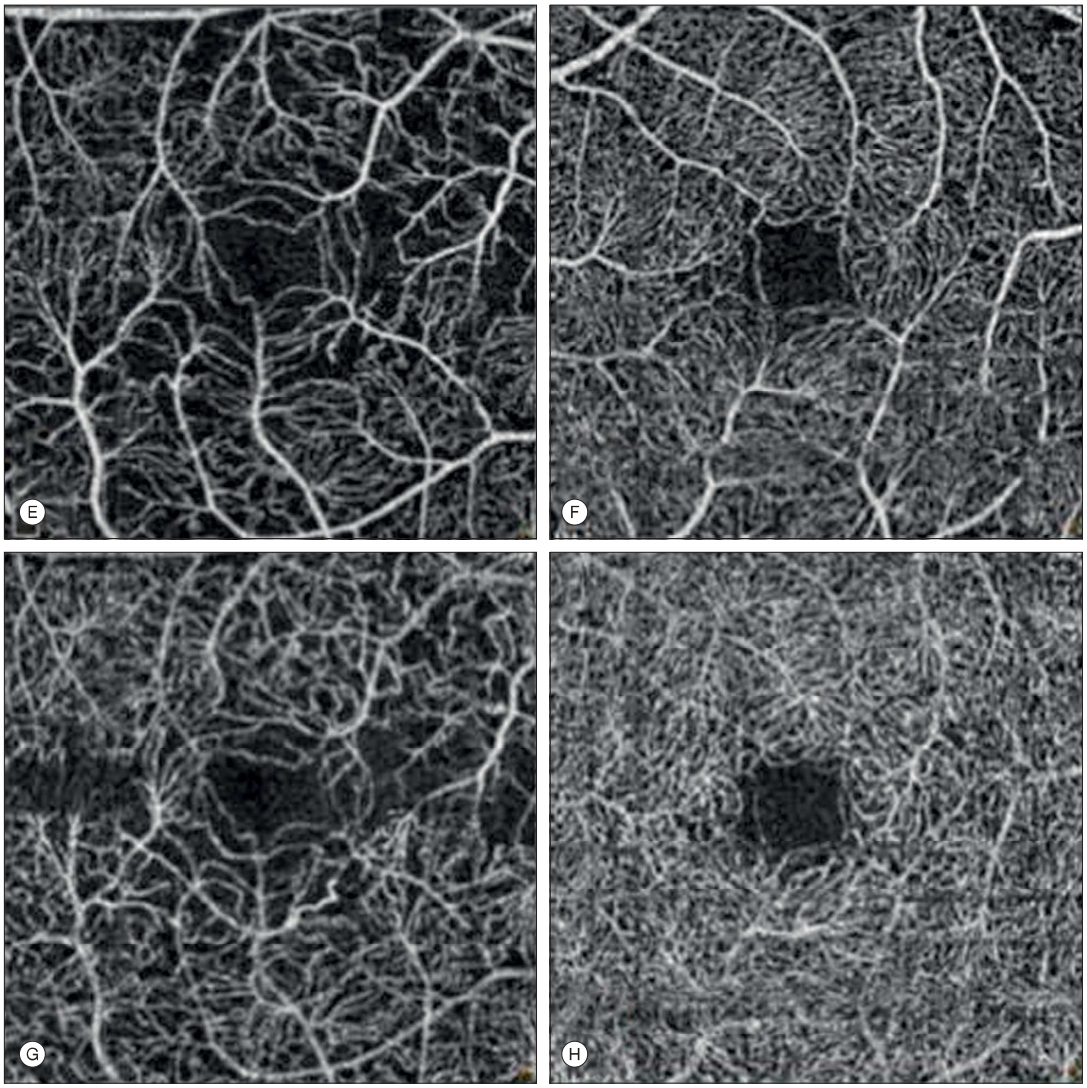


Fig. 6.26.3, cont'd Optical coherence tomography angiography (OCTA; 3-mm scan) of the superficial capillary plexus shows decreased capillary density and widening of the foveal avascular zone (FAZ) in the irradiated right eye (E) compared with the normal fellow eye (F). OCTA (3-mm scan) of deep capillary plexus shows similar findings, including decreased capillary density and widening of the FAZ in the irradiated right eye (G) compared with the normal fellow eye (H).

been seen following EBRT doses ranging from 15 to 60 Gy. However, RR usually occurs with doses >45 Gy. Hyperfractionation is associated with a decreased incidence of RR,[19] and patients who received fractions of <1.9 Gy are unlikely to develop RR, as found in one study.[19] When the treatment site is closer to the eye, the incidence of RR is higher. With respect to proton beam radiotherapy of posterior uveal melanoma, the risk factors for development of RR include posterior tumor margin <3 mm to fovea and optic nerve, larger tumor base diameter, and increased tumor thickness, as reported in one study.[14]

Differential Diagnosis

RR can be clinically indistinguishable from changes seen in patients with diabetic retinopathy. Other conditions that need to be considered in the top differential diagnosis include branch retinal vein occlusion, central retinal vein occlusion, hypertensive retinopathy, Coats' disease, and perifoveal telangiectasia.

Treatment

Prevention, if possible, is better than treatment. Unfortunately, RR is an inevitable side effect of radiotherapy, and complete prevention is not possible. However, a number of strategies have been developed to decrease the dose of radiation and hence the risk of development of RR because once macular ischemia develops, vision loss is irreversible.

Reducing the Dose of Radiation

For brachytherapy, the use of collimating plaques as well as eccentric positioning of the plaques so that the posterior plaque edge is aligned with the posterior tumor margin may help decrease the radiation dose to the macula. Further measures include de-escalating (lowering) the apex tumor dose (to 63 Gy) in an effort to reduce the total radiation dose to macula[20] and filling the vitreous cavity with silicone oil to attenuate intraocular radiation.[21] For teletherapy, appropriate shielding of the ocular structures during EBRT can minimize radiation exposure. As previously mentioned, hyperfractionation of EBRT has been associated with decreased incidence of RR.

Laser Photocoagulation

Focal/grid macular laser photocoagulation has been used to treat RM, sector scatter and panretinal laser for severe NPRR and PRR, and circumferential laser around the tumor to prevent or delay RM and RR. Focal/grid macular laser photocoagulation has been used to treat RM with variable success. Studies by Kinyoun et al. and Hykin et al. demonstrated a benefit in visual acuity following treatment.[9,22] However, the effect was not sustained with longer follow-up in the study by Hykin et al.[22]

Sector scatter and panretinal laser photocoagulation has been widely used to treat NPRR and PRR after plaque radiotherapy (Fig. 6.26.5). Bianciotto et al. reported that 66% of eyes that underwent panretinal photocoagulation had complete resolution of PRR.[23] In a study by Finger et al., eyes with choroidal melanoma treated by plaque radiotherapy received

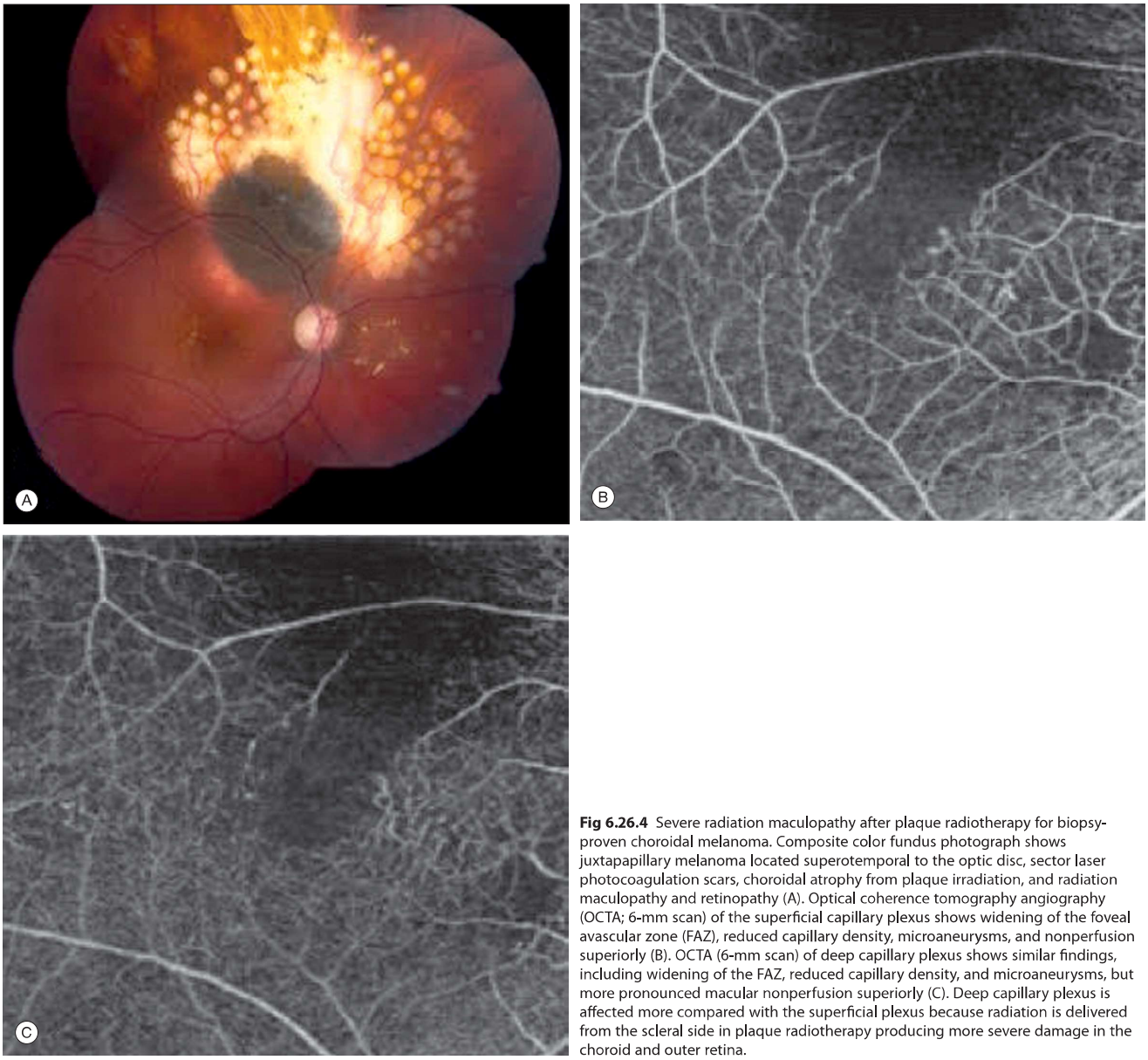


Fig 6.26.4 Severe radiation maculopathy after plaque radiotherapy for biopsy-proven choroidal melanoma. Composite color fundus photograph shows juxtapapillary melanoma located superotemporal to the optic disc, sector laser photocoagulation scars, choroidal atrophy from plaque irradiation, and radiation maculopathy and retinopathy (A). Optical coherence tomography angiography (OCTA; 6-mm scan) of the superficial capillary plexus shows widening of the foveal avascular zone (FAZ), reduced capillary density, microaneurysms, and nonperfusion superiorly (B). OCTA (6-mm scan) of deep capillary plexus shows similar findings, including widening of the FAZ, reduced capillary density, and microaneurysms, but more pronounced macular nonperfusion superiorly (C). Deep capillary plexus is affected more compared with the superficial plexus because radiation is delivered from the scleral side in plaque radiotherapy producing more severe damage in the choroid and outer retina.

sector scatter laser photocoagulation at the first sign of retinopathy, proliferative or nonproliferative.[24] With plaque brachytherapy, an area of ischemia develops in and 2–3 mm surrounding the location of the plaque. Treating this area and any further areas of intraretinal microangiopathy or neovascularization with scatter laser photocoagulation caused regression of retinopathy in 64% of eyes. In the same study, a subset of eyes were treated prophylactically with scatter laser photocoagulation without clinical evidence of RR, and none lost more than three lines of vision at final follow-up.[24] Materin et al. similarly used prophylactic circumferential sector laser photocoagulation and sub-Tenon's membrane triamcinolone injection in eyes with choroidal melanoma managed by plaque radiotherapy and concluded that this treatment combination was safe and effective to decrease the occurrence of RM.[25]

Intravitreal Anti–Vascular Endothelial Growth Factor Agents

Uveal melanomas produce high levels of vascular endothelial growth factor (VEGF) in vitreous and humor aqueous and that larger tumors are associated with higher levels of VEGF.[26] Therefore, trace macular edema may even be present before treatment. Several small case series and retrospective reports examined the use of intravitreal bevacizumab (anti-VEGF agent) in the treatment of RM utilizing time-domain OCT (TD-OCT). Many of these studies demonstrated a reduction in macular edema following intravitreal bevacizumab.[27] However, sustained decrease in macular edema often required multiple injections, and visual acuity was not found to improve significantly. More recent studies concentrated on early SD-OCT diagnosis and prompt treatment of RM. In one such report, 50% of the patients had stable or improved vision at 3 years with repeated intravitreal anti-VEGF treatment.[28] As a further step, Shah et al. studied the prophylactic effect of anti-VEGF therapy on RM. Those authors reported that patients receiving intravitreal bevacizumab injections every 4 months (a total of 7 injections) after plaque radiotherapy for uveal melanoma demonstrated OCT-evident macular edema and vision loss less frequently at the end of 2 years compared with controls who did not receive treatment.[29] This study showed that prophylactic periodic anti-VEGF therapy leads to long-term stabilization of radiation vasculopathy. Other anti-VEGF agents, including ranibizumab and aflibercept, can also be used for the treatment of RM. Finger et al. found that continuous anti-VEGF therapy with either bevacizumab or ranibizumab was well-tolerated with no RPE atrophy and preserved vision in 80% of patients with RM over a 10-year period.[30] The situation may be similar to exudative age-related macular degeneration where continuous

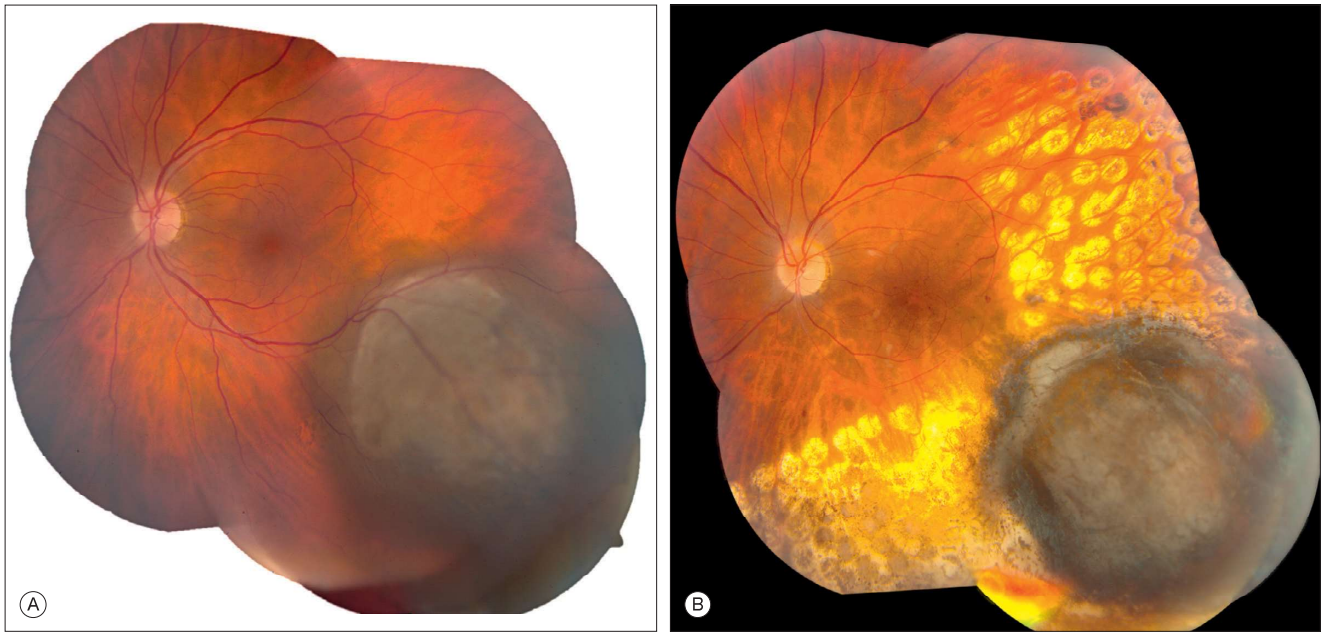


Fig. 6.26.5 Medium-sized choroidal melanoma at the inferotemporal equator of the left eye (A) before treatment. After plaque radiotherapy (B), the melanoma has regressed and radiation maculopathy with nerve fiber layer infarcts in the macular region is noted. Sector panretinal photocoagulation was performed for proliferative radiation retinopathy, leaving minimal vitreous hemorrhage near the melanoma scar.

fixed-interval dosing of intravitreal anti-VEGF therapy seems to provide better vision stabilization or improvement status compared to as-needed regimens.[31] Finger et al. also pointed out that higher doses of anti-VEGF (2.5 mg versus 1.25 mg for bevacizumab and 2.0 mg versus 0.5 mg for ranibizumab) and shorter injection intervals may be used in refractory RM cases.[30] Tachyphylaxis or resistance to one anti-VEGF agent after an initial responsive phase may occur and requires switching to another anti-VEGF agent. Intravitreal anti-VEGF therapy also appears effective in reducing radiation-related neovascularization and vitreous hemorrhage in addition to RM, and therefore it may have a disease-modifying activity in these circumstances.[32]

Corticosteroids

Triamcinolone acetonide is thought to downregulate various cytokines and decrease capillary permeability. It is used to treat macular edema secondary to various retinal pathologies and has similarly been used to treat RM. One study found that intravitreal triamcinolone acetonide (4 mg/0.1 mL) does transiently reduce macular edema and improve visual acuity.[33] A randomized controlled trial assessed the efficacy of periocular triamcinolone (40 mg) given at the time of plaque application, 4 months and 8 months later in preventing radiation maculopathy.[34] At the 18-month follow-up, eyes treated with periocular triamcinolone demonstrated less evidence of macular edema on OCT and less vision loss compared with the control group. The study reported similar rates of elevated intraocular pressure and cataract progression between the two groups.[34] Patients with severe, massive cystoid edema that prove to be refractory to three sequential injections of anti-VEGF agents may also benefit from consolidation with intravitreal triamcinolone.[35] Although triamcinolone may decrease macular edema, permanent inner segment ellipsoid band changes may limit visual recovery. Use of intravitreal dexamethasone implant has also been reported in RM.[36]

Other Treatments

There are case reports of successful treatment of RR using photodynamic therapy as well as hyperbaric oxygen and oral pentoxifylline.[37–39] Advanced PRR complicated by vitreous hemorrhage and/or tractional retinal detachment may require pars plana vitrectomy for removing blood and reattaching the retina.[40]

Prognosis

Mild NPRR, characterized by subtle capillary dropout and occasional microaneurysms may not significantly affect vision and could remain stable for years. Patients with advanced RM and PRR can present with markedly decreased vision. In the COMS, it was estimated that 43% of patients would have a visual acuity of 20/200 or less 3 years after plaque radiotherapy.[41] In an analysis of 1106 eyes with uveal melanoma treated with plaque radiotherapy, Shields et al. found loss of visual acuity (≥5 Snellen lines) in 33% of eyes at 5 years and 69% at 10 years.[42] A later analysis by the same group for 354 large melanomas (tumor thickness >8 mm), revealed visual acuity of 20/200 or worse in 57% of the eyes by 5 years and 89% by 10 years.[43]

RADIATION PAPILLOPATHY

Radiation papillopathy (RP) or radiation optic neuropathy, similar to RR, may be seen after both brachytherapy and teletherapy. Interestingly, almost all patients who suffer from acute RP have accompanying RR, especially RM.

Pathology and Pathogenesis

Radiation injury includes both vascular and neuropathic effects.[4] The creation of radiation-induced free radicals affects both the vascular endothelial cells and neuroglial cells. The induction of genetic mutations in glial cells by ionizing radiation can lead to demyelinization and necrotic degeneration.[4] Comorbidities, including diabetes mellitus, and concurrent chemotherapy are risk factors that increase the severity of disease. The dose of radiation and latent period for the development of RP are similar to those for RR. RP usually develops with optic disc doses above 60 Gy and fraction size >2 Gy.[44]

Clinical Features

RP presents with disc hyperemia, disc swelling, peripapillary edema, hard exudates, hemorrhage, and nerve fiber layer infarcts in the acute phase (Figs. 6.26.6 and 6.26.7). Rarely, RP can present with a central retinal vein occlusion secondary to disc edema. Optic nerve head swelling can persist for weeks or months but eventually leads to optic atrophy. This pattern of presentation simulates an anterior ischemic optic neuropathy. However, other rare cases of radiation optic nerve damage may simulate posterior ischemic optic neuropathy. These cases present with decreased visual acuity and relatively normal appearance of the fundus in the early stages, with optic atrophy developing later. This latter presentation usually occurs after teletherapy of the eye or adjacent structures. Because the optic nerve is confined within the scleral canal and dura, RP-associated exudative

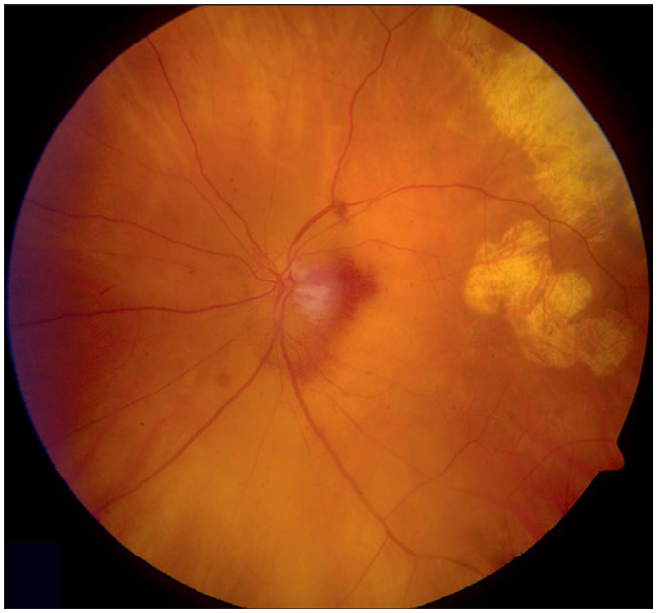


Fig. 6.26.6 Radiation papillopathy following plaque radiotherapy for choroidal melanoma showing disc edema, telangiectasia, and hemorrhage. Note the adjacent retinopathy with vascular thinning and dilation along the superotemporal vessels.

vasculopathy and edema occur within a confined space and lead to a visually devastating course. Some cases of RP may be associated with disc neovascularization that apparently arises from regional ischemia.[45]

Diagnostic Studies

FA demonstrates that the optic disc and surrounding retinal areas are hypofluorescent in the early phase. In the late phase, there is usually leakage over the optic nerve. These findings can be explained on the basis of vascular incompetence leading to capillary closure and leakage. Preliminary data show that OCT can be used in the diagnosis and follow-up of patients with RP with features of optic disc swelling, increased optic disc thickness, and decreased peripapillary retinal thickness.[46] OCTA may show abnormal vessels on the optic disc. Decreased amplitudes and increased latency can be seen on flash visual evoked potential testing.

Differential Diagnosis

RP should be differentiated from all causes of optic nerve swelling including nonarteritic optic neuropathy, arteritic (giant cell) optic neuropathy, optic neuritis, tumor invasion of the optic nerve, and papilledema.

Treatment

There is no proven effective treatment for RP. It is usually managed with only observation. In rare cases, oral corticosteroids may be helpful to decrease pain and degree of visual loss resulting from RP. Shields et al. used intravitreal injection of 4 mg/1 mL triamcinolone acetonide, given 4 months apart, to treat acute RP.[47] Those authors concluded that intravitreal triamcinolone acetonide was effective in the short term to save and, in some cases, modestly improve vision. Finger and Chin reported that intravitreal injections of bevacizumab (1.25 mg/0.05 mL) given every 6–8 weeks lead to improved vision and reduced hemorrhage and disc edema in RP.[45]

Prognosis

RP generally has a poor visual outcome. Vision loss is characteristically severe with hand motions to no light perception. However, there are examples of spontaneous improvement in vision in some cases.[48]

SUMMARY

RR and RP may develop after radiation exposure of any type and have characteristic funduscopic features. FA, OCT, and OCTA are currently the most helpful studies that aid in early diagnosis, treatment planning, and subsequent follow-up. For RR treatment, laser photocoagulation and intravitreal injections of anti-VEGF medications or triamcinolone acetonide are used. Prophylactic treatment before development of RR and RM leads to better prognosis for vision. Intravitreal injections of the above drugs can also be used for RP, although in most cases little benefit in visual acuity has been demonstrated.

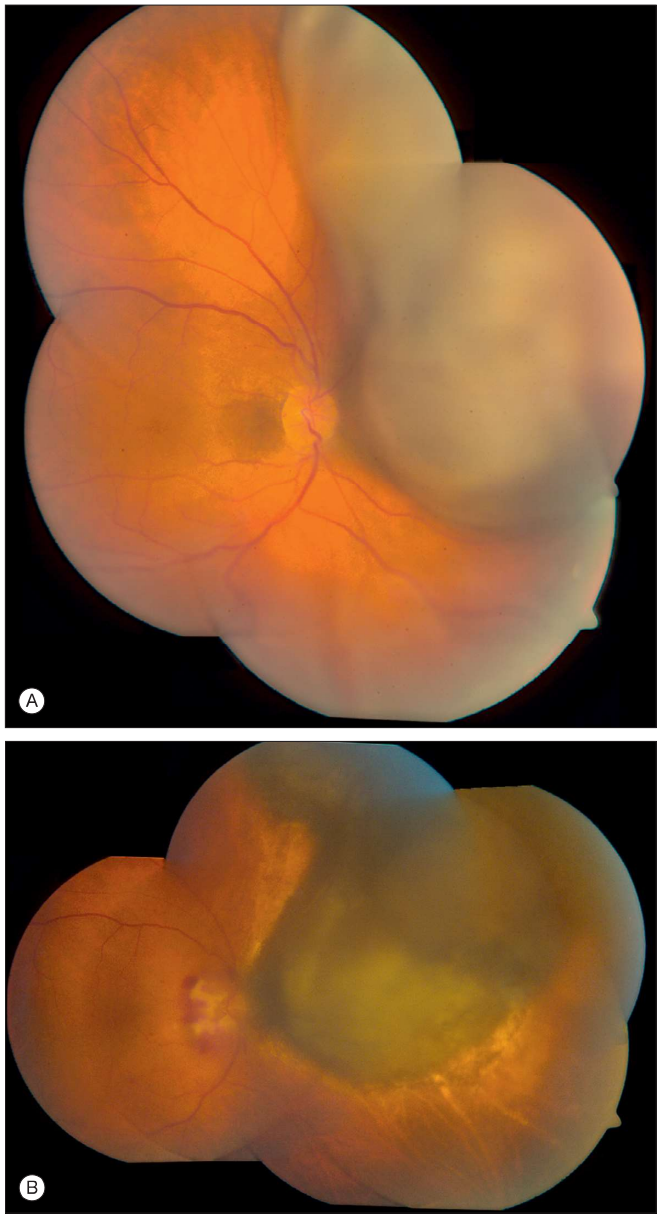


Fig. 6.26.7 Large-sized choroidal melanoma in the nasal quadrant of the right eye (A) before treatment. After plaque radiotherapy (B), the melanoma has regressed and radiation papillopathy with disc edema and hemorrhage is noted.

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