Correspondence


Is irradiation a justifiable treatment of choroidal melanoma?

Sir, In reply to the letter by Drs Char et al., the answer to the question, ‘at what stage in the natural history of the disease must intervention occur to avoid development of metastasis,’ is: at the earliest feasible one, preferably before the 7 mm diameter stage (see paragraphs 3 of previous replies).¹ The answer on ‘the most effective means of treating this tumour’ is: enucleation at the earliest feasible stage; this eliminates any (further) dissemination. Irradiation of melanomas has been a regular practice for about 15–20 years. The reason that ‘there are no definitive data that demonstrate that either irradiation or enucleation is superior in preventing tumour-related mortality’ is the surprising lack of reported >10-year survival results of irradiated patients. Only one clinic has published prospective 10-year comparative survival results of all primary enucleated and irradiated patients in the same period.¹

So far 39 calculated doubling times of uveal melanomas have been reported. Paragraph 4 of our reply to Drs Zimmerman et al.¹ explains why it is warranted to assume that nearly all metastatic deaths within 6-1 years after treatment are a consequence of pre-existing dissemination.

More than 50% of 153 microscopically studied irradiated melanomas did not reveal any necrosis, while 94% contained viable tumour tissue.¹ One wonders that ophthalmic radiotherapists, not being trained pathologists, claim to be able to interpret better the viability of melanoma cells, which presumably they have not studied, than trained pathologists who have studied these tumours. Charet al. state that they have not observed mitoses in some irradiated melanomas. Other pathologists and we have observed mitoses in proton beam and in ruthenium irradiated melanomas. Besides, a body is legally dead because of the presence of signs of death, not because vital signs are absent.

The findings by Gass in his 10-year follow-up of all patients have never been ‘shown to be invalid’. The main criticism is the greater number of anteriorly located melanomas in the irradiation group. Many believe, for no reason, that anterior melanomas have a worse prognosis. Weinhaus et al.¹ found that ‘patients with juxtapapillary melanomas had a worse prognosis than those with tumors in other locations’.

Our statement ‘almost all survival statistics after irradiation are still based on follow-up periods of a few months to no more than three to four years’ was correct at the time it was submitted (see paragraphs 7-9 of reply to Dr Oosterhuis et al.).

Our statement that ‘patients have the right to be treated by enucleation which eliminates any further dissemination’ is progressively becoming more supportable. Death from metastases, disseminated after irradiation, cannot manifest itself before six years after therapy. ‘Short-term analyses’ of treated patients are irrelevant. Survival rates after enucleation, irradiation, and observation are identical during the first six post-treatment years if the selection conditions have been identical. Results more than six years after irradiation have been published only twice; both were highly unfavourable.¹

After enucleation any (further) dissemination is prevented. After irradiation major parts of the remaining tumour tissue (69% after a two-year follow-up period) continue their growth and will shed cell emboli, eventually causing metastatic death after more than six years.

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References


Familial exudative vitreoretinopathy (FEVR) and platelet dysfunction

Sir, Chaudhuri et al.¹ reported abnormal platelet aggregation in patients from two families with familial exudative vitreoretinopathy (FEVR). However, Gole et al.² reported no platelet aggregation defects in a patient with incontinentia pigmenti, a separate syndrome with a phenotypically similar proliferative retinopathy. These two conflicting findings are at odds with regard to association between FEVR and platelet function.

1 Chaudhuri et al.² reported abnormal platelet aggregation in patients from two families with familial exudative vitreoretinopathy (FEVR). However, Gole et al.² reported no platelet aggregation defects in a patient with incontinentia pigmenti, a separate syndrome with a phenotypically similar proliferative retinopathy. These two conflicting findings are at odds with regard to association between FEVR and platelet function.
We have performed platelet aggregation studies on two patients with FEVR—a 7-year-old boy with total blindness and his 14-year-old female cousin, who is mildly affected. The boy’s total blindness at 3 months of age has been described elsewhere. Neither subject had been exposed to any antiplatelet agents within the two weeks prior to the study. Coagulation studies were conducted and the results compared with the normal ranges of values in our laboratory. Platelet aggregation to added arachidonic acid (1.5 mmol/l), adrenaline (5 μmol/l), collagen (1-0 mg/l), ristocetin (1.5 g/l, 1.2 g/l, and 0.5 g/l) and adenosine diphosphate (ADP) (1 and 10 μmol/l) was within normal limits in each patient as compared with that of healthy controls. Platelet counts and morphology were normal, as were prothrombin times, partial thromboplastin times, and fibrinogen levels. Factor VIII and von Willebrand’s factor were also within the normal range in each patient. The only abnormality noticed was the boy’s bleeding time of 13 minutes (normal range 2–9 minutes) and his lack of adenosine triphosphate (ATP) secretion as measured by the luciferin-luciferase method using a lumi-aggregator. This finding is consistent with storage pool disease. The cousin’s bleeding time and ATP secretion were normal.

Although we found no abnormality of platelet aggregation in response to arachidonic acid in either patient, the prolonged bleeding time and storage pool disease in the boy may have aggravated his condition, resulting in his total blindness, which brought him to medical attention originally. An additional feature of his history may be significant. Shortly after his birth his mother reported being treated with high doses of aspirin for endomtritis while breast feeding him. The combination of FEVR, his storage pool disease, and aspirin exposure may explain his severe clinical course. While Chaudhuri et al.’s report of platelet aggregation defects in response to arachidonic acid has yet to be verified in other FEVR patients, this case supports the contention that platelet abnormalities may contribute to the severity of FEVR.

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References

Call it lenteectomy
Sir, Congratulations to Drs Grossman and Peyman on the report of their very successful application of this procedure. Following Dr Peyman’s pioneering work in this area we adopted his procedure and have been similarly gratified with a low rate of complications and very successful results.

Dr Peyman has been a persistent advocate of the term ‘pars plicata’, which is anatomically correct. Now if we can just get him to call it a lenteectomy, then he will have it 100% proper. (Ectopia lentis is another condition which is very well treated by his procedure.)

PAUL E ROMANO
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References

Sir, I appreciate the kind comments of Dr Romano on our paper. His suggestion is correct, and we will use the proper terminology.

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Eye protection for welders
Sir, Mr G P H Brittain emphasises the need for adequate eye protection at all times for amateur welding especially using the MIG welder (metal arc inert gas welder). Fortunately the two patients reported on by Mr Brittain had a full visual recovery. We reported a case of foveal injury by an amateur welder which produced permanent visual loss, further emphasising the potential seriousness of the injury that can occur.

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References

Obituary

Professor T Krwawicz

Professor Tadeusz Krwawicz, who died in September 1988 at the age of 78 years, was born in Lwow in 1910.