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Natural history of non-ischemic central retinal vein occlusion versus iatrogenic intervention

Sir,

I read with interest the report by Issa and Qasem on the occurrence of central retinal vein occlusion (CRVO) in association with thrombotic thrombocytopenic purpura (TTP) in a 45-year-old man.

Given that their patient had an absent relative afferent pupillary defect from the outset and showed visual recovery from an acuity of 6/60 to 6/12 over 12 weeks, there were sufficient grounds to diagnose a "non-ischemic" CRVO. Their inference that a combination of macular laser and systemic medical therapy favorably influenced the outcome of (non-ischemic) CRVO in their patient cannot be delivered with confidence. As such, their concluding sentiments are questionable because the natural history of a "non-ischemic CRVO" is one that characteristically carries a good prognosis, such that most eyes eventually enjoy good visual acuity.

To recapitulate: the visual fate of an eye that suffers a CRVO is chiefly determined by whether the insult is of an "ischemic" or "non-ischemic" nature. This dichotomous classification is helpful in predicting prognosis and applies regardless of the underlying etio-pathogenesis of a CRVO. Hayreh has already made the point that since the natural history for non-ischemic CRVO is resoundingly benign, it is imprecise and misleading to congratulate ourselves on the effectiveness of any interventions undertaken in parallel with the usual timecourse for clinical improvement.

In both variants of CRVO it is believed that there is a blockage of the central retinal vein, but in "non-ischemic" cases there is a relatively superior blood flow owing to a better availability of collateral venous channels.

Secondly, the application of macular laser at 12 weeks is also a questionable decision in this case, bearing in mind that the resolution of macular edema (as evidenced by the improvement in visual acuity) was already progressing acceptably. Clearance of edema at such a rate signifies the survival of a macular microcirculation with healthy hemodynamics. Hence it is fallacious to commemorate any laser treatment applied towards the final phase of visual recovery, when the preamble was already very satisfactory. Indeed, it would have been preferable to wait for a further four to six weeks to observe the subsequent course of visual acuity before intervening with laser photocoagulation or another anti-edema treatment. Any supposed concerns regarding the creation of "chronic macular edema" beyond 12 weeks (and consequent permanent degeneration of macular structure) are not tenable in this instance given the prevailing behavior of this patient’s CRVO.

Rather counter-intuitively, the overriding belief at present is that thrombus-clearing or preventing strategies are seemingly ineffective in CRVO. A similar opinion has been expressed regarding the efficacy of hemodilution techniques in cases where raised blood viscosity has been considered relevant to the pathogenesis of CRVO. Germane too in this regard is the observation that CRVOs can occur in patients on therapeutic levels of anticoagulant therapy.

Thus the proposed effectiveness of anti-thrombotic and laser treatment in modulating this patient’s CRVO is difficult to reconcile with the observation that (even when left alone) eyes that have suffered a non-ischemic CRVO overwhelmingly tend to fare well. Whether the described iatrogenic input actually improved the outcome for this single patient is therefore an eminently debatable point. And importantly, when considering therapeutic strategies we must first acknowledge the natural history of the disease process under scrutiny to introduce equilibrium into the discussion and allow a better informed interpretation.

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References

Authors’ reply

Sir,

We would like to thank the author for his interest in our case report. We agree with the author that it is important to make a distinction between ischemic and non-ischemic central retinal vein occlusion (CRVO). The case report we described would
indeed fit the diagnosis of a non-ischemic CRVO. However, in our experience and other authors’ report, CRVOs which are associated with thrombotic thrombocytopenic purpura do not usually resolve quickly.[3] Even if the retinal hemorrhages and the venous stasis resolve gradually, the residual macular edema becomes chronic and usually does not improve.

It might seem to the reader that we were a bit hasty in applying the laser treatment and that we could have waited a bit longer to see if the edema would resolve spontaneously. However, on clinical grounds, we did not feel that the edema was resolving as quickly as the retinal hemorrhages and venous stasis. At that time, we believed that complete spontaneous resolution of the macular edema was unlikely. Another fact was that the patient had an active life and wished for a quicker restoration of vision. This was the first time that we have tried macular grid laser treatment in such a case and the quicker resolution of the patient’s macular edema and restoration of vision to 6/6 was in favor of our judgment.

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