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Sir,

Central retinal vein obstruction with cilio-retinal infarction

In a letter to your journal, Singh[1] described a 28-year-old woman with central retinal vein obstruction (CRVO) and infarction of the inner retina in the territory of a superior macular arteriole. He felt that the affected arteriole was a branch of the central retinal artery (CRA) rather than a cilio-retinal arteriole, even though the vessel filled with fluorescein before the CRA proper and its four immediate branches. One cannot be certain of the origin of such a vessel without anatomical proof,[2] but equally it is difficult to reconcile such early dye filling with an origin other than from the posterior ciliary arterial (as opposed to the CRA) circulation. Early cilio-retinal dye filling in cases of CRVO with cilio-retinal infarction has previously been reported[3,4] along with a 'pulsatile' dye transit (or intermittent retrograde flow) in the cilio-retinal arterioles.[3–6] These angiographic features, and especially the paradox of early dye filling with impaired perfusion, help in revealing the nature of the relationship between CRVO and cilio-retinal infarction where similar haemodynamic principles to those underlying sphygmomanometry and ophthalmodynomometry apply.[5,7]

Consider firstly the effect of CRVO on inner retinal perfusion in an eye with a cilio-retinal arteriole arising directly from the ophthalmic artery near the origin of the CRA. The perfusion pressure (arteriovenous gradient) in the cilio-retinal territory would essentially be the same as that in the CRA territory and, provided the intraluminal CRV pressure did not exceed the diastolic ophthalmic artery pressure, this would most likely be sufficient to maintain the viability of the entire inner retina. Next, consider the effect of CRVO in an eye with a cilio-retinal arteriole arising as a branch of a major vessel in the choroid. The perfusion pressure in such a cilio-retinal arteriole will reflect the systolic and diastolic arterial pressures in the parent choroidal artery (part of the 'high-flow, low-resistance' circulation unaffected by CRVO) and not the ophthalmic arterial pressures. Hence, 'pulsatile' flow (ie intermittent reversal of flow) in the cilio-retinal territory might arise, perfusion being below the level needed to sustain inner retinal viability. Nevertheless, the cilio-retinal dye front might still precede that in the CRA. Whether the dye front precedes,[1,3,4] coincides with[5,6] or follows[5,6] CRA filling will depend on the phase of the cardiac cycle at which the bolus of fluorescein arrives at the arterial branch point during a particular angiographic study. In practice, the site of origin and branching pattern of the posterior ciliary circulation vary considerably[2] but may be crucial to the outcome of CRVO.

In young individuals, the retinal capillaries are capable of withstanding high transmural pressure gradients after (partial) CRVO, so there may be little by way of retinal oedema or haemorrhage (unless provoked by paracentesis[8]). Thus, the symptomatic presentation of unilateral CRVO in Singh's[1] patient most likely depended on her possessing not only a macular cilio-retinal arteriole but also the one derived from a choroidal artery (rather than the one with a more direct origin from the ophthalmic artery).

References

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Sir,

Reply

I would like to thank Professor McLeod for the interest shown in this case report and for comments regarding the nature of arterial occlusion in eyes with central retinal vein occlusion (CRVO).

The reason that led me to conclude that this was a branch artery as opposed to a cilio-retinal artery was the