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implantation, are more frequent when the IOL is implanted in the sulcus.1–3 Some cases of pigment-dispersion syndrome after single-piece AcrySof IOL implantation have been reported in the literature. In all of them, at least one haptic was inserted into the sulcus ciliaris.4,5

However, in our case, the IOL was implanted in the capsular bag after uncomplicated cataract surgery. The UBM showed that the haptic causing the pigment dispersion was inside the capsular bag. Although retraction of the capsule would be the most likely mechanism in this case, there was no capsular fibrosis and the pigment dispersion began in the immediate postoperative period. It is possible that the relatively sharp and square edge of this IOL in this case favored the pigment dispersion in the same way as described in cases of sulcus IOL implantation.

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Cilio-retinal perfusion in concurrent cilio-retinal and central retinal vein occlusions

A 73-year-old man with sudden visual loss in his left eye presented with a mildly hemorrhagic central retinal vein occlusion (CRVO) and a cloudy white macular area in the same eye. Visual acuities were right, 6/6, and left, counting fingers. There was a left relative afferent pupillary defect. The rest of the ocular examination was unremarkable. The patient took no medication and had no previous medical history. At the 3-month review, visual acuity in the left eye remained at counting fingers. The CRVO signs had reversed and there was thinned arterial vascularity in the inferior macula (Fig. 1).

Given the 4-quadrant hemorrhages, diffusely engorged veins, and an area of well-defined macular pallor, a diagnosis of CRVO with concurrent cilio-retinal artery obstruction (CIO) was made. This distinct pattern of retinovascular signs was first reported tentatively by Oosterhuis in 1968.1 In recent times, this curious entity has continued to prompt debate. Specifically, there has been contention over the identity of the vessel responsible for the macular arterial infarction. Having contributed to the first definitive paper on this subject in 1976, McLeod has challenged the impression of some recent observers who have encountered the same pattern of fundus signs. These authors have concluded that the macular artery is an ordinary off-shoot of the retinal arterial tree, which has coincidentally suffered thrombo-occlusive blockage against a background of coexistent CRVO.2,3

McLeod has refuted this conclusion by referring to the fundus angiogram to prove that the occluded arterial vessel in the macula is actually a cilio-retinal (and not an ordinary macular) arteriole. He has described how fluorescein per-
fusion of the artery in question occurs in time with the choroid, thereby confirming that it is a cilioretinal vessel, a branch that is typically part of the choroidal tree.4

However, this idea that an occluded cilioretinal artery should fill normally (that is, in time with the choroid) seems counterintuitive.2 Rather, a relatively blocked cilioretinal vessel might be expected to show delayed fluorescein perfusion, or indeed, a lack of filling with dye. Likewise, this pathophysiological paradox of normal fluorescein perfusion of an occluded cilioretinal vessel is not in agreement with the mechanistic theory put forward for concurrent CIO/CRVO.

In the proposed mechanism, raised intravascular pressure after CRVO is said to transmit backward through the capillary beds toward the central retinal and cilioretinal arteries. This back-pressure slows blood flow in the cilioretinal artery preferentially because the cilioretinal vessel has a lower perfusion pressure than the central retinal artery.5 Hence, the suppression of cilioretinal artery perfusion (by CRVO) should be apparent as a relatively delayed filling of the cilioretinal vessel with fluorescein. This logical prediction was borne out in our patient. Here, the cilioretinal artery arose from the inferotemporal edge of the disc (5 o’clock) and coursed as a major vessel passing inferior to the fovea, with an associated territory of pallor on either side. Fluorescein perfusion of this vessel was seen at as late as 34.5 seconds, against a background of diffuse arterial filling (Fig. 2).

Our finding (of delayed cilioretinal filling) differs from the common documentation of cilioretinal perfusion occurring in time with the choroid in cases of combined CRVO/CIO.4 Thus, the observation reported here is an interesting departure from relatively conventional angiographic signs. Since the choroidal arterial tree perfused normally in our patient, the selectively delayed filling of the contiguous cilioretinal artery was perhaps related to localized hemodynamic dysfunction within this vessel. For instance, ischemic retinal tissues release vasodilatory mediators that induce the opening up of vessels in the affected territory.5 Where this intrinsic tissue-response is suboptimal in a given retinal microenvironment, any reflex vasodilatation would be insufficient (particularly under the duress of back-pressure hemodynamics), such that cilioretinal territory hypoperfusion and eventual vascular shrinkage are the outcome.

Similarly, the flow of blood into a cilioretinal system might be delayed to an unusual extent if rather more capillary beds are connected to it than is common (i.e., an anatomical variation), and so exert a larger cumulative back-pressure than is typical in this disorder. The latter speculation is based on the finding that this patient had a large cilioretinal artery, the extinction of which ultimately led to diffuse macular infarction and irredeemable visual acuity.

Other factors have also been implicated in the perfusion characteristics of concurrent CRVO/CIO. Thus, observed angiographic patterns can be influenced by anatomical factors, such as the precise origin of the cilioretinal artery (ophthalmic or posterior ciliary). Hemodynamic variations, such as the phase of the cardiac cycle that is in progress when fluorescein reaches arterial branch-points, also have a bearing on the angiogram.2 Evidently, a whole host of factors can introduce nuances into the eventual angiographic picture, and their interplay means that cilioretinal perfusion on the angiogram can manifest in a number of ways.

As such, with reference to the seminal publication by McLeod and Ring more than 30 years ago,6 this report is a consecutive documentation in the literature of unambiguously delayed fluorescein perfusion of a cilioretinal artery in concurrent cilioretinal and central vein occlusions.

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