Cilioretinal infarction as a sequel to central retinal vein occlusion in a patient exposed to thrombogenic medication

Dear Editor,

Kannan and colleagues reported a case in which cilioretinal arterial occlusion was ascribed to cocaine.[1] One could appreciate a lack of cogency in their analysis. The authors too confessed their perplexity on observing acute cilioretinal infarction (CRI) some 3 days after cocaine exposure. A misperception of cardinal signs led to inferences that merit further consideration. The supposition – vascular insufficiency focally in the cilioarterial tree induced by cocaine – is revisited, and the thesis expanded by providing deeper insights.

On the supplied fundus images, there are retinal hemorrhages discernible across the posterior pole, which are visibly contemporaneous with CRI. The hemorrhages are present in the upper and lower retinal hemispheres on either side of the macular raphe. The area of macular pallor denotes cilioretinal insufficiency whereas the hemorrhages in the posterior retina are consistent with central venous obstruction. Indeed, this is the quintessential signature of a retinovascular cascade: central retinal vein occlusion (CRVO) of nonischemic type with secondary evocation of CRI. Retrograde transmission of intravascular backpressure after CRVO suppresses perfusion of the cilioarterial system (which has lower pressures than the adjoining central retinal artery).

Such *hypoperfusion* of the cilioarterial territory is expressed as macular infarction (that is, CRI). Failure to identify this clinical constellation, acronymed here as CRVO-CRI, is a renowned pitfall.[2] For those not versed with this entity, a recourse to landmark monographs will clarify this distinctive presentation.[3] Also noteworthy is that the CRVO in a "CRVO-CRI tandem" is often nonischemic. Consequently, the CRVO signs may be subtle (expressed as "venous stasis retinopathy"), as exemplified by this typical case. This is the precise way in which I have met this combined retinal vasculopathy. Another exquisite feature is observable on the angiogram : the paradoxically rapid ingress of dye into a cilioretinal bed that should have sluggish perfusion owing to CRVO-CRI. This occurrence is elucidated when one appreciates the hemodynamics and anatomical variations of the chorioretinal tree.[4] Thus dye progression is influenced by (a) oscillatory fluxes related to the cardiac cycle, (b) the intensity of retinovenous backpressure, and (c) variations within the angioarchitecture from which emanates the cilioretinal artery. Such parameters vary from one CRVO-CRI event to another and accordingly produce variation in angiographic filling of the cilioretinal system.

The notion was also advanced in this report that the retinal hemorrhages arose as a result of systemic hypertension from cocaine exposure. Hemorrhages were described in the fundus affected by cilioretinal occlusion. Only if hemorrhages had developed in *both* fundi could one surmise that cocaine generated transitory malignant hypertension in this young patient.

A salient omission from the pathophysiological considerations was the detail of contraceptive use. This is a defined risk factor for retinovascular occlusion.[5] I have assessed young adults with retinovascular events in whom the only detectable risk factor was the contraceptive pill.

Both contraceptive hormones and cocaine have separately been implicated in the pathogenesis of CRVO. Hence the plausible rendition is that these two agents, singly or synergistically, were the genesis of a CRVO-CRI cascade in this young patient. As epilogue, one must emphasise that there is the potential for macular infarction (CRI) in any acute CRVO fundus with cilioretinal feeders. However, the actual likelihood of CRI is determined by the trio of factors outlined earlier. Lastly, the pharmacokinetics of cocaine (as recognized by the authors) are such that selective cilioretinal spasm is improbable 3 days after cocaine exposure. The authors were sensitive to this point in their notably ambivalent conclusions.

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