

Combined occlusion of branch retinal artery and vein secondary to prepapillary arterial loops

Anuradha Dhawan^{1,2}, Dhananjay Shukla^{1,3}

A middle-aged diabetic and hypertensive man presented with diminished vision in the left eye. Fundus examination revealed prepapillary arterial loops, but with features of venous rather than arterial occlusion. Fluorescein angiography and optical coherence tomography confirmed the presence of a branch retinal vein occlusion along with two branch retinal artery occlusions. The resultant macular edema responded well to intravitreal triamcinolone and laser photocoagulation though the visual improvement was moderate.

Key words: Branch retinal artery occlusion, branch retinal vein occlusion, prepapillary arterial loops

Prepapillary vascular loops are predominantly arterial congenital malformations, which may cause transient or permanent visual acuity or field loss due to arterial occlusion, vitreous hemorrhage or hyphema; these complications may occur spontaneously or following trivial accidents.^[1-3] We report a branch retinal vein along with twin arterial occlusions secondary to prepapillary arterial loops.

Case Report

A 54-year-old man presented with diminished vision OD for 8 weeks. He was diabetic and hypertensive on regular treatment. His best-corrected visual acuity (BCVA) was 20/125 OD and 20/20 OS. Anterior segment and intraocular pressures were normal bilaterally. Fundus examination OD revealed prepapillary vascular loops with superotemporal macular branch vein occlusion (appearing as the sheathed vessel) and cystoid macular edema (CME) OD [Fig. 1]. The occlusion of branch retinal arteries was not obvious: Superotemporal branch retinal artery showed subtle beading at the first branching [asterisk in Fig. 1a]. The occluded

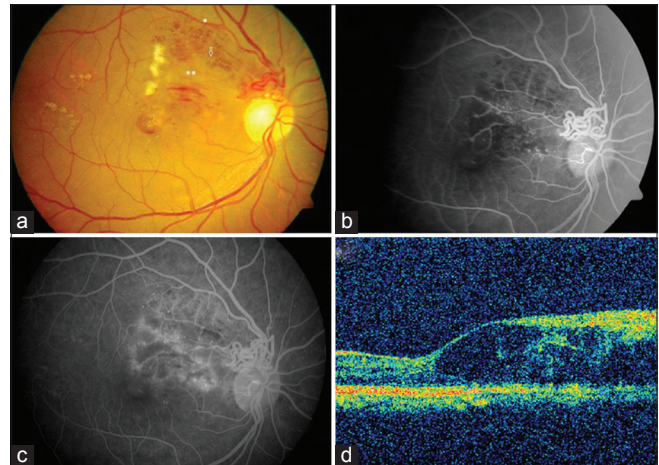


Figure 1: Clinical picture at presentation (a) prepapillary loops with branch vein occlusion (arrow), beaded major branch artery* and macular artery hidden by the retinal edema**. (b and c) Mid-phase fluorescein angiogram reveals the attenuated macular artery and vein and prolonged arteriovenous transit superotemporally. Late phase shows leakage from microaneurysms. (d) Optical coherence tomography (vertical scan) shows superior macular thickening, full-thickness cysts, and inner-retinal hyper-reflectivity

macular branch retinal artery was veiled by the tissue edema [double asterisk, Fig. 1a-b]. Fundus was unremarkable OS. Fluorescein angiogram OD in the early arteriovenous phase confirmed the arterial origin of the prepapillary loop (filling simultaneously with arteries; no lamellar flow) and also revealed capillary nonperfusion extending into the foveal avascular zone [Fig. 1b], and macular leakage in the late phases [Fig. 1c]. No cilioretinal artery was present. Optical coherence tomography (OCT) (time-domain OCT; vertical 10 mm scan) quantified the central macular thickening (506 μm) and also revealed inner retinal opacification, typical of arterial occlusion [Fig. 1d]. An inferior quadrantic field defect was documented OD on perimetry. Systemic evaluation, cardiovascular workup, and referral to a neurophysician revealed no additional pathologies. After obtaining approval of the Institutional Review Board and informed consent of the patient, he received intravitreal triamcinolone acetate (IVTA, 4 mg/0.1 ml) OD, followed by grid laser photocoagulation after 4 weeks. The CME resolved completely (central macular thickness: 172 μm); BCVA improved to 20/80 over 6 weeks. Fundus OD now revealed the occluded arteries and vein in better detail. The occluded macular artery (double asterisk) was attenuated, and the corresponding vein (arrow) was sheathed; inner retinal atrophy (darker area) in the superotemporal area

Access this article online	
Quick Response Code:	Website: www.ijo.in
	DOI: 10.4103/0301-4738.176027

¹Retina-Vitreous Service, Aravind Eye Hospital and Postgraduate Institute of Ophthalmology, Madurai, Tamil Nadu, ²Centre for Sight Group of Eye Hospitals, New Delhi, ³Retina-Vitreous Service, Ratan Jyoti Netralaya, Gwalior, Madhya Pradesh, India

Correspondence to: Dr. Dhananjay Shukla, Retina-Vitreous Service, Ratan Jyoti Netralaya, 18, Vikas Nagar, Gwalior - 474 002, Madhya Pradesh, India. E-mail: daksh66@gmail.com

Manuscript received: 23.04.15; **Revision accepted:** 30.11.15

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Cite this article as: Dhawan A, Shukla D. Combined occlusion of branch retinal artery and vein secondary to prepapillary arterial loops. Indian J Ophthalmol 2015;63:917-8.

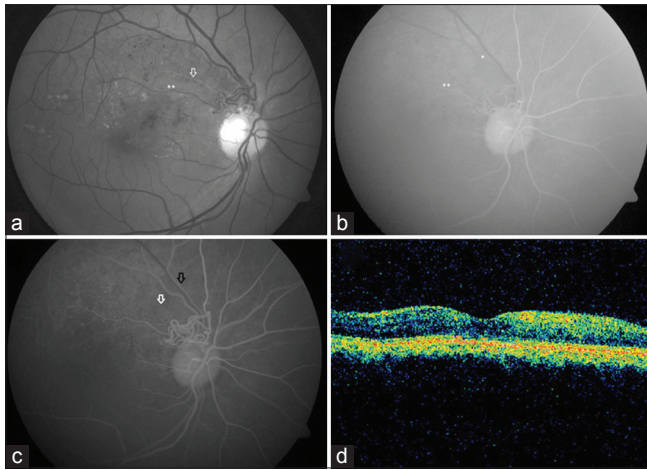


Figure 2: Clinical profile 10 weeks posttreatment (a) the occluded macular artery** and vein (arrow) are attenuated. (b and c) Early-phase fluorescein angiogram: No dye in superotemporal artery* and vein; delayed filling of macular artery** and loop. Late phase shows obliterated macular vein (white arrow) and retrograde flow in superotemporal vein (black arrow) from perfused areas. (d) Macular edema resolved; retinal opacification persists

was also well-evident [Fig. 2a]. A repeat fluorescein angiogram revealed better details of the retinal vasculature; delayed filling was clearly evident in the major superotemporal branch artery (asterisk), the macular artery (double asterisk) as well as the prepapillary loop [Fig. 2b]. The late phase angiogram showed that the sheathed macular vein (white arrow) was completely obliterated (no flow) [Fig. 2c]. A stable retinal and functional status were maintained over the next 18 months [Fig. 2d]. There was a transient rise in intraocular pressure after IVTA, which was managed by topical antiglaucoma medication.

Discussion

Prepapillary vascular loops are embryologically derived from the retinal arterial system in 95% of the cases and are vulnerable to occlusion due to reduced blood flow and increased turbulence.^[2-4] They can be differentiated from arteriovenous malformation which fill rapidly before arterial filling, and from venous loops which display slower, lamellar filling of the dye.^[5] Since the patient presented to us 8 weeks after the visual decline, cilioretinal anastomoses secondary to vascular occlusions were also a possibility, which could be excluded by the size, elevation, location and filling pattern of the loops. Cilioretinal anastomoses tend to be smaller, flatter, circumpapillary, and fill at a slower rate.^[6] In view of the patient's age and systemic hypertension, a coexistence of primary arteriosclerotic branch vein occlusion with the

prepapillary loop was another possibility. The absence of arteriosclerotic and arteriovenous crossing changes elsewhere in the fundus of the affected or in the fellow eye made this possibility unlikely.

There are several reports of prepapillary loops causing arterial occlusions, which are essentially untreatable.^[1-3] We are not aware of any previous report of combined occlusion of branch retinal vein and two branch arteries due to prepapillary arterial loops. Mechanical endothelial damage by hemodynamic turbulence in the convoluted arteries of the loop probably precipitated the intra-arterial clot formation, aggravated by systemic hypertension and diabetes. The macular branch vein was either directly compressed by the loop, or shared with sclerosed macular artery a common adventitial sheath, hidden by the loop. We attempted treatment in spite of the arterial component because the visual loss was moderate, and could be explained by CME, which showed a predominance of macular thickening (venous component) over retinal opacification (arterial component) on OCT. The macular edema responded well to IVTA and laser treatment with significant and stable visual improvement. Venous occlusions can also occur following prepapillary arterial loops, and when present, may be amenable to laser photocoagulation and pharmacotherapy.

Acknowledgment

This study was performed by the authors during their tenure at Retina-vitroeous Service, Aravind Eye Hospital and Postgraduate Institute, Madurai, India.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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