Branch retinal artery occlusion associated with congenital retinal macrovessel

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A congenital retinal macrovessel (CRM) is a large retinal vessel, usually a vein, which traverses through the central macula and has large tributaries extending on both sides of the horizontal raphe. In the majority of cases, CRM have no effect on visual acuity, although in rare cases, macular hemorrhage, foveolar cysts, serous macular detachment, and the presence of the anomalous vessel in the foveola can affect vision. We describe a case of CRM with decreased vision secondary to a branch retinal artery occlusion (BRAO). To the best of our knowledge, this association has not been reported previously.

Keywords: Congenital retinal macrovessel, aberrant retinal vessels, branch retinal artery occlusion, optical coherence tomography, macular thickness, retinal nerve fiber layer thickness

Introduction

Congenital retinal macrovessels (CRM) are rare, aberrantly large branches of retinal arteries or veins that typically cross the horizontal raphe to either supply or drain the macula.[1] Reduction of vision in the involved eye is rare and has been attributed to serous macular detachment, hemorrhage, foveal cyst, or to the mere presence of the aberrant vessel in the foveal area. We present a case of CRM with decreased vision due to co-existent branch retinal artery occlusion (BRAO) and ensuing hemimacular atrophy.

Case Report

A 45-year-old healthy female presented with decreased vision in her left eye noticed during a routine ophthalmic evaluation. On examination, the best corrected visual acuity (BCVA) was 20/20 in the right eye and 20/80 in the left eye. Anterior segments of both eyes were normal. Fundus examination of the left eye revealed a large aberrant tributary of a dilated tortuous superotemporal vein that branched inferiorly and crossed the horizontal raphe with several tributaries reaching up to the fovea. The inferotemporal artery as well as vein was attenuated [Figure 1a]. The right fundus was normal. The cup:disc ratio was 0.3 in both eyes.

Fluorescein angiography (FA) revealed, in addition, early filling and delayed emptying of the aberrant vein, altered perifoveal capillary network, and foveal avascular zone. Though anastomoses were seen between its branches and that of the superotemporal vein, there were no abnormal leakages or capillary nonperfusion areas [Figure 1b].

Both eyes of the patient were studied using RTVue spectral domain (SD)-OCT (Optovue Inc., Fremont, California, USA). The left macula showed perifoveal and parafoveal thinning in the inferior quadrant, especially of the inner retinal layers [Figures 1c-e]. Also, the retinal nerve fiber layer (RNFL) thickness in the left eye was significantly reduced in the inferotemporal quadrant [Figure 2a]. Standard automated perimetry using 30–2 visual field threshold testing with a size III stimulus (Humphrey Field Analyser II, Humphrey Instruments Inc., San Leandro, CA, USA) revealed a corresponding field defect in the superonasal quadrant [Figure 2b]. The left eye was unremarkable with all values within normal range.

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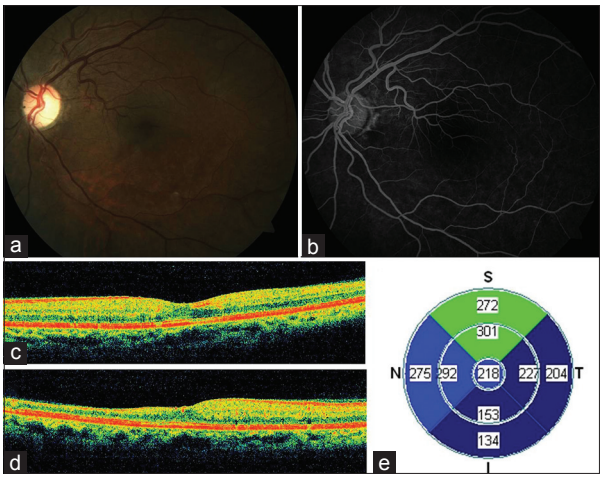

Figure 1: (a) Color fundus photograph of the left eye showing a large aberrant branch of a dilated superotemporal vein reaching up to the fovea (congenital retinal macrovessel). The inferotemporal vessels are attenuated. (b) Fluorescein angiogram showing multiple arteriovenous communications around the CRM. Horizontal (c) and vertical (d) SD-OCT scans through the macula showing marked thinning of the macula inferiorly especially involving the inner retinal layers. A macular thickness map (e) confirms that the superior quadrant is unaffected

A detailed review of the patient's systemic history revealed no abnormality. Complete laboratory testing including hemogram, coagulation profile, erythryocyte sedimentation rate (ESR), and lipid profile were within normal limits. Echocardiography and carotid Doppler examination were unremarkable. The patient was advised to follow up regularly.

Comment

CRMs are typically associated with normal visual acuity and are mostly detected on routine examination. The anomalous vessel is remarkably stable and complications only occur exceptionally as described in literature.[1] Archer *et al*. have classified retinal arteriovenous anastomoses in three groups depending on the caliber of the communicating vessels, the presence of a capillary plexus bridging these vessels, and the grade of visual impairment.[2] This case likely belongs to group 2 of this classification, which includes single or multiple direct arteriovenous communication without capillary bed, as seen on FA.

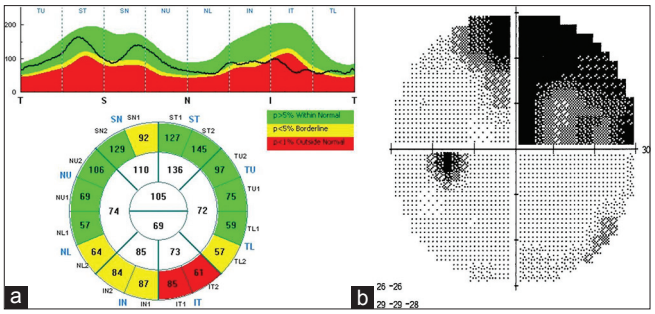

Figure 2: (a) Retinal nerve fiber layer (RNFL) thickness map showing significant reduction in the inferotemporal quadrant. (b) Standard automated perimetry using 30-2 visual field threshold testing showing a corresponding field defect in the superonasal quadrant

In our case, SD-OCT demonstrated segmental (inferior) macular thinning notably of the inner retinal layers along with reduction in the corresponding peripapillary RNFL, consistent with an ischemic insult to this part of the retina (an inferior BRAO).[3] To the best of our knowledge, this is the first reported case of decreased vision due to BRAO associated with a CRM. It has been suggested that decompensation of CRMs can cause a relative ischemia of the macular area due to direct arteriovenous communications which results in chronic, stable visual impairment.[4] This may have been the cause in our patient, with diminution of vision noticed later incidentally.

References

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