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Case Report

Mysterious vision loss in a child

Abstract

Objective: Retinal vascular occlusions are a rare event in children and, if they do occur, secondary to other systemic or ocular diseases. Idiopathic central retinal artery occlusion (CRAO) in pediatric population has been sparsely reported in literature. We describe an unusual case of a 10-year-old girl who presented with findings suggestive of a previously reperfused central retinal artery occlusion (CRAO) in the right eye.
Methods: A 10-year-old girl presented with decreased vision in the right eye (counting fingers), which was detected incidentally. Clinical features and ocular investigations (optical coherence tomography, fluorescein angiography and electroretinography) pointed to a possible central retinal artery occlusion with reperfusion. An extensive systemic work-up to find out the underlying cause failed to reveal any abnormality.
Results: No treatment was initiated due to the delayed presentation. The child was closely followed up with no change in vision over one year.
Conclusion: The case emphasizes the role of multi-modal imaging in the diagnosis of such unusual presentations. It also stresses on the need to do a thorough multi-system evaluation in any case of vascular occlusion as it can be lifesaving. This case also emphasizes the need to routinely screen kids at school for visual defects from a very young age.

Keywords: idiopathic, central retinal artery occlusion, multi-modal imaging

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Introduction

The incidence of central retinal artery occlusion (CRAO) in people under the age of 30 years has been estimated to be less than 1 in 50,000 and this number is much lower in children under the age of 10 years [1]. Age related atherosclerosis is a major cause for CRAO in adults aged >55 years. However, in children occurrence of CRAO almost always points towards an underlying systemic or ocular cause like emboli from cardiovascular sources, hypercoagulable states, migraine or anomalous disc vasculature [1]. A major challenge in children is that the diagnosis is often delayed in unilateral cases as children do not notice visual loss in one eye. We present an interesting case of a 10-year-old girl who presented with a possible re-perfused CRAO which was detected incidentally. Multi-modal imaging helped in arriving at the diagnosis. Extensive work-up did not reveal an underlying cause.

Case description

A 10-year-old girl was incidentally detected to have decreased vision in the right eye (RE) during a school-screening program. Neither the child nor the parents had noticed the visual loss. There was no history of trauma, ocular surgery, amaurosis or any major illness in the past. Visual acuity was counting fingers in the RE and 20/20 in left eye (LE). There was a relative afferent pupillary defect in the RE with normal intraocular pressure in both eyes. Dilated fundus examination of the RE showed pale optic disc with marked attenuation of arterioles and mild pigmentary mottling in the mid-peripheral retina (Figure 1A). There were no anomalous disc vessels or subretinal tracts. The LE fundus was unremarkable (Figure 1B). Enhanced depth optical coherence tomography (EDI-OCT) scans showed thinning of inner retinal layers with intact outer retinal layers and choroid in the RE (Figure 1C). Fundus fluorescein angiography (FFA) showed a normal choroidal flush, arm-retina time of 17 seconds and arteriovenous (AV) transit time of 10 seconds in the RE. FFA was normal in the LE (Figure 2A–E). Full field electroretinogram (ERG) showed an electronegative pattern and reduced oscillatory potentials in the RE. Scotopic and photopic ERG showed diminution of b wave amplitude in the RE (Scotopic: 39 ⌈V in RE and 291 ⌈V in LE; Photopic: 22.2 ⌈V and 120 ⌈V in the LE) (Figure 3). Systemic examination did not show signs of any underlying systemic disorder.
Complete hemogram, peripheral smear, erythrocyte sedimentation rate (ESR), blood sugars, renal function tests, electrocardiogram (ECG), echocardiography, lipid profile, hypercoagulability screen (bleeding and clotting times, prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrinogen levels, serum homocystiene levels, antithrombin III activity, proteins C and S

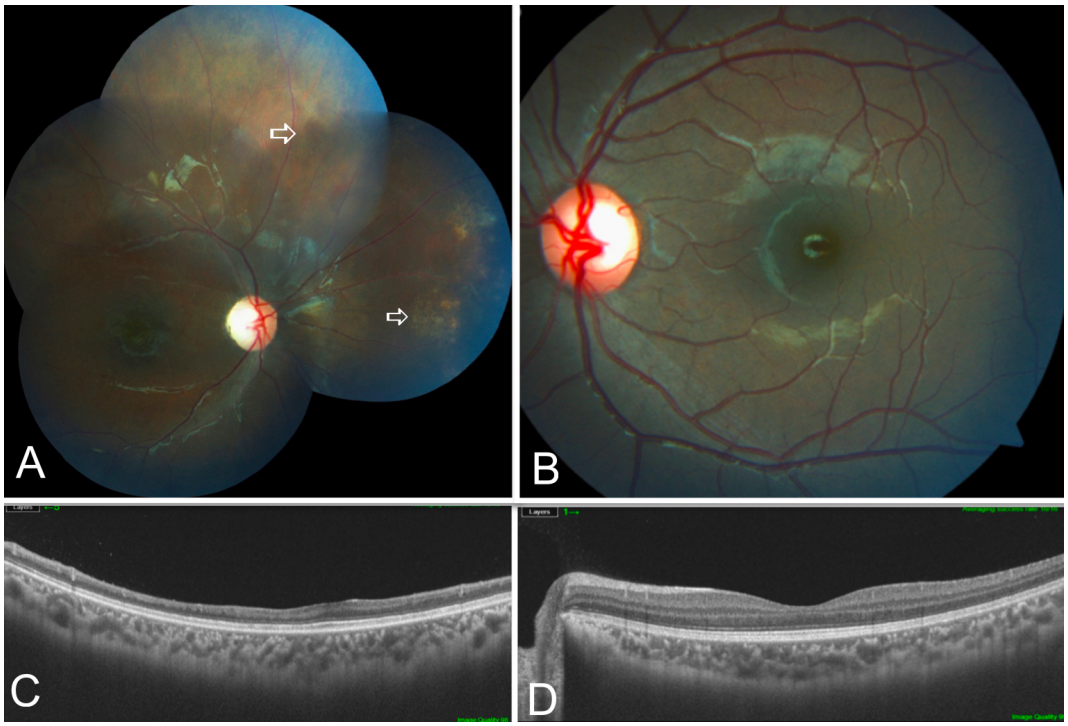


Figure 1: (A) Fundus photo of the right eye showing disc pallor with vascular attenuation and mild pigmentary changes in the retina (arrows). There are no anomalous disc vessels or subretinal tracts. (B, D) Fundus photo and optical coherence tomography (OCT) scan of the left eye was normal. (C) Macular OCT scan showing inner retinal layer thinning with intact outer layers and choroid in the right eye.

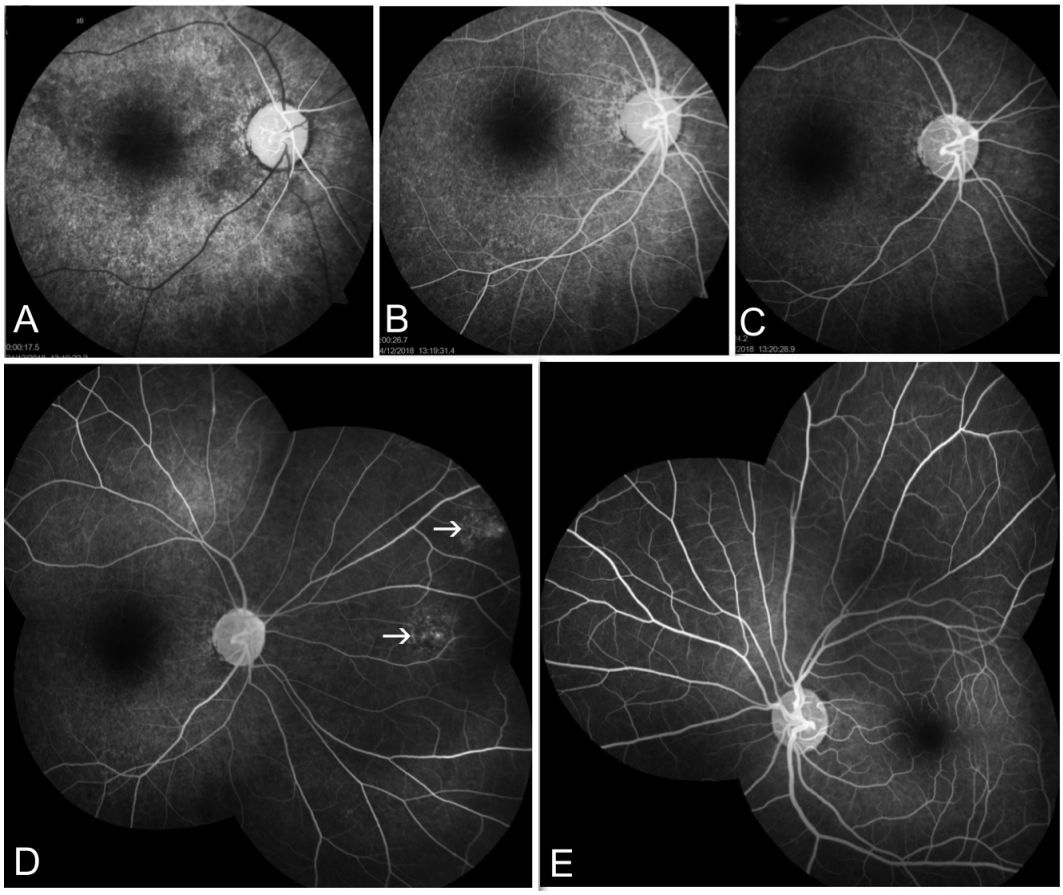


Figure 2: (A-C) Fundus fluorescein angiography showing an arm-retina time of 17 seconds and arterio-venous (AV) transit time of 10 seconds in the right eye. (D) A few window defects seen in the periphery of the right eye (arrows). (E) FFA of the left eye was normal.

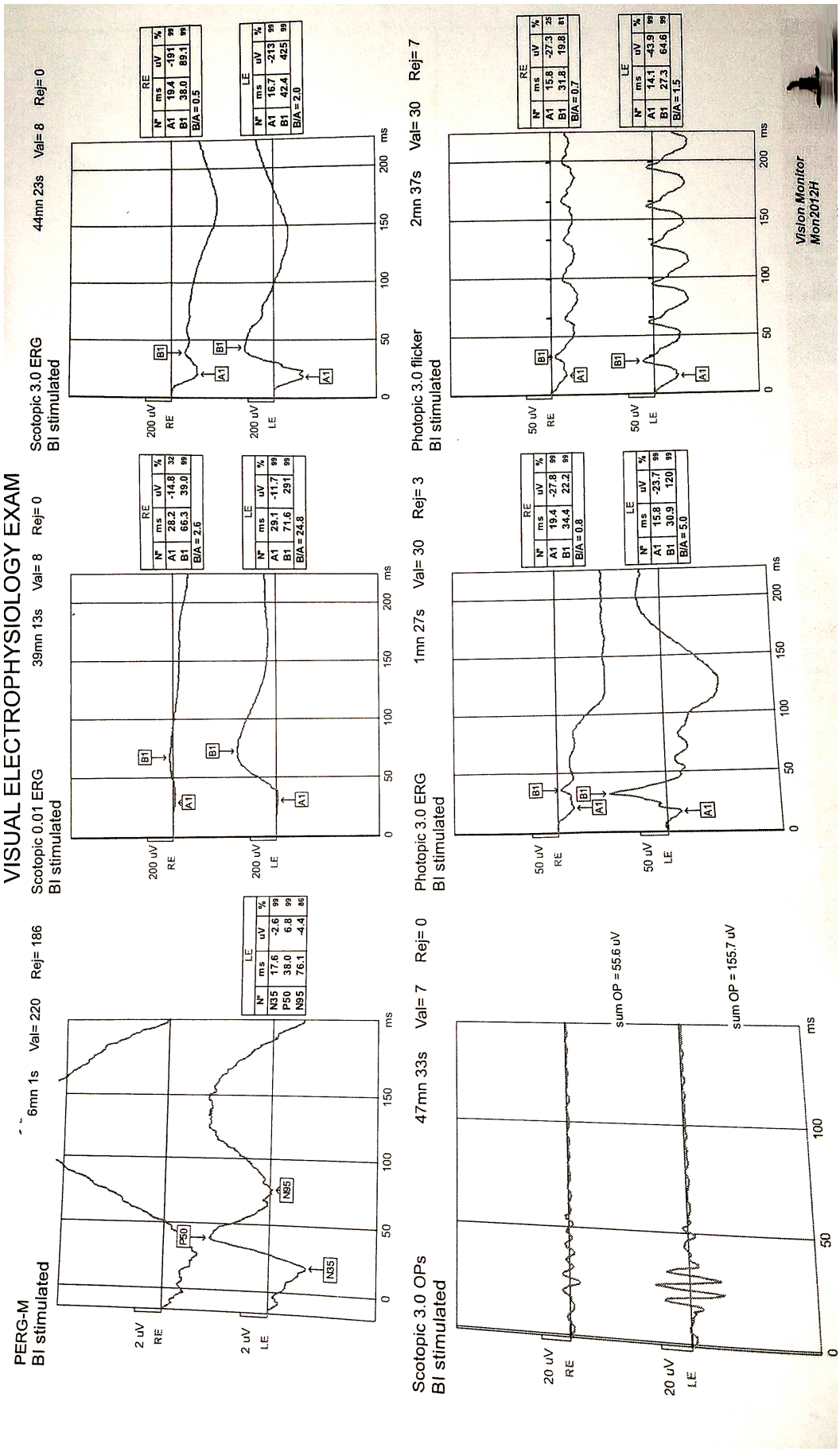


Figure 3: Electroretinography (ERG) showed decreased scotopic and photopic amplitudes with an electronegative pattern and decreased oscillatory potentials in the right eye. Left eye ERG was normal.

levels, factorV-Leiden mutation), autoantibody screen (anti-dsDNA, ANA, ANCA, anti-histone and antiphospholipid antibodies), hemoglobin electrophoresis, HIV, treponema pallidum hemagglutinantion (TPHA) test, serum antibodies for toxoplasma gondii, mantoux, chest X-ray, carotid doppler, contrast enhanced MRI and MR-angiography of the brain and orbit were all normal.

Discussion

Given the incidental detection of profound unilateral visual loss and the absence of a clearly documented acute onset, the exact timing of the presumed vascular event could not be established. The differential diagnoses considered were central retinal artery occlusion (CRAO) with subsequent reperfusion, ophthalmic artery occlusion, and diffuse unilateral subacute neuroretinitis (DUSN). Ophthalmic artery occlusion was considered less likely because the choroidal circulation was preserved, with a normal choroidal flush on fluorescein angiography. DUSN was also considered because it may present with optic disc pallor, retinal pigmentary changes and visual dysfunction; however, DUSN typically demonstrates involvement of the outer retina, whereas in our patient OCT demonstrated predominantly inner retinal thinning with preservation of the outer retinal layers. The ERG abnormalities were also predominantly consistent with inner retinal dysfunction. The combination of marked retinal arterial attenuation, inner retinal layer thinning, an electronegative ERG pattern, and preserved choroidal circulation favored a previous CRAO with subsequent reperfusion. However, because the patient presented after the presumed acute event and the timing of onset was unknown, the diagnosis should be interpreted as a presumed reperfused CRAO rather than an observed acute occlusion. No intervention was planned because of the delayed presentation. Follow-up after one year showed no change in the visual acuity or fundus examination.

Retinal arterial occlusions are a rare event in pediatric population and are usually secondary to underlying systemic or ocular problems like emboli from cardiovascular sources, hypercoagulable states, migraine or anomalous disc vasculature [1]. CRAO has also been described in association with collagen vascular and neoplastic diseases, herpes zoster ophthalmicus and varicella infections and orbital trauma [2], [3]. No cause was found in our patient in spite of an extensive multi-system evaluation. Diffuse unilateral subacute neuroretinitis (DUSN) and posttraumatic chorioretinopathy can have a similar clinical presentation but the OCT and ERG in these cases show both outer and inner retinal layer involvement as against what we noted in the present case [4].

There have been very few reports of pediatric idiopathic retinal vascular occlusion in literature [5], [6], [7], [8], [9]. In all these case reports, the presentation was acute with clinical features typical of acute RAO. Our case was unique in regards to the age of the patient and the subacute presentation. We hypothesize two possible mechanisms that could have caused a CRAO in this child: 1) a transient hypercoagulable state (as can happen during an episode of high fever due to dehydration) 2) a past episode of post-viral vasculitis as reported by Lee et al. [9]. A normal AV transit time on FFA suggests that reperfusion has occurred following the CRAO. However, the vision failed to improve because complete CRAO has shown to cause irreversible retinal ganglion cell death after just 12–15 min [10]. Hence, even though the blood supply maybe restored to the retina after this window period, we may not see a functional improvement. But this should not prevent the ophthalmologist from doing a thorough work-up for the cause as this can be lifesaving for the child.

Conclusion

This report describes an unusual case of unilateral visual loss in a child with multimodal findings suggestive of a previously reperfused CRAO. As the visual loss was detected incidentally and its onset was unknown, the diagnosis remains presumptive. Multimodal imaging and electrophysiology, along with exclusion of other causes, supported a previous retinal arterial occlusive event with reperfusion. Further studies are needed to determine the long-term prognosis and risk to the fellow eye.

Notes

Conference presentation

The material was presented at the annual virtual meeting of the Vitreo-Retina Society of India, 2020.

Competing interests

The authors declare that they have no competing interests.

References

1. Brown GC, Magargal LE, Shields JA, Goldberg RE, Walsh PN. Retinal arterial obstruction in children and young adults. Ophthalmology. 1981 Jan;88(1):18-25. DOI: 10.1016/s0161-6420(81)35080-5
2. Cohen RG, Hedges TR 3rd, Duker JS. Central retinal artery occlusion in a child with T-cell lymphoma. Am J Ophthalmol. 1995 Jul;120(1):118-20. DOI: 10.1016/s0002-9394(14)73772-9
3. Atmaca LS, Ozmert E. Optic neuropathy and central retinal artery occlusion in a patient with herpes zoster ophthalmicus. Ann Ophthalmol. 1992 Feb;24(2):50-3.
4. Audo I, Webster AR, Bird AC, Holder GE, Kidd MN. Progressive retinal dysfunction in diffuse unilateral subacute neuroretinitis. Br J Ophthalmol. 2006 Jun;90(6):793-4. DOI: 10.1136/bjo.2005.085548

5. Ganesh A, Mitra S, Koul RL, Venugopalan P. Spontaneous ophthalmic artery occlusion in a child. J Pediatr Ophthalmol Strabismus. 2001 May-Jun;38(3):183-5. DOI: 10.3928/0191-3913-20010501-16

6. Manayath GJ, Shah PK, Narendran V, Morris RJ. Idiopathic pediatric retinal artery occlusion. Indian J Ophthalmol. 2010 Mar-Apr;58(2):151-2. DOI: 10.4103/0301-4738.60091

7. Delhiwala KS, Rao C. Cystoid Macular Edema with Idiopathic Acute Central Retinal Artery Occlusion in a Healthy Child. J Ophthalmic Vis Res. 2017 Jul-Sep;12(3):348-50. DOI: 10.4103/jovr.jovr_211_16

8. Sebban AI, Sullivan TJ, Davison MB. Branch retinal artery occlusion in a child. Aust N Z J Ophthalmol. 1996 Aug;24(3):283-6. DOI: 10.1111/j.1442-9071.1996.tb01594.x

9. Lee WB, Pearson PA, Moreman K. Central retinal artery occlusion and disc edema in a child. J AAPOS. 2002 Aug;6(4):264-5. DOI: 10.1067/mpa.2002.124903

10. Tobalem S, Schutz JS, Chronopoulos A. Central retinal artery occlusion - rethinking retinal survival time. BMC Ophthalmol. 2018 Apr 18;18(1):101. DOI: 10.1186/s12886-018-0768-4

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Please cite as
Shyamala DD, Slesser D, Ganne P. Mysterious vision loss in a child. GMS Ophthalmol Cases. 2026;16:Doc11.
DOI: 10.3205/oc000275, URN: urn:nbn:de:0183-oc0002750

This article is freely available from
https://doi.org/10.3205/oc000275

Published: *2026-11-18*

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