Acute macular neuroretinopathy after blunt ocular trauma: A rare association

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Acute macular neuroretinopathy (AMN) is a deep retinal ischemic manifestation. It has been reported after the use of sympathomimetics, childbirth, bee sting, oral contraceptives, flu-like illness, intravenous contrast agents and bodily trauma not directly involving the eyes. We report a case of AMN following blunt ocular trauma. An 18-year-old male presented with an acute history of blurring of vision following blunt trauma to the right eye. Spectral domain optical coherence tomography (SD-OCT) showed hyperreflectivity of the outer nuclear layer with ellipsoid layer disruption. This report highlights AMN as a manifestation of blunt trauma, presence of which may be an indicator of poor visual prognosis.

Key words: Acute macular neuroretinoathy, AMN, deep retinal ischaemi, ocular trauma, paracentral acute middle maculopathy

Blunt ocular trauma can result in a myriad of findings including commotio retinae, macular hole, choroidal ruptures, retinal and vitreous hemorrhage or retinal detachment.[1] Retinal vascular occlusions have been reported after ocular trauma. Fluorescein angiography is currently the gold standard for imaging ischemia, but its resolution is insufficient to identify isolated loss of the deep capillary plexus. Advancements in optical coherence tomography (OCT) has led to a better understanding of pathophysiology and manifestation of different types of retinal ischemia occurring at various retinal levels. It has led to the recognition of retinal ischemic pathologies like acute macular neuroretinopathy (AMN) and paracentral acute middle maculopathy (PAMM). Acute retinal ischemia presents in various forms depending upon the location and level of vascular occlusion. Blunt trauma has been reported to cause central retinal artery occlusion; however, acute macular neuroretinopathy after trauma is yet unreported. Present case highlights multimodal imaging findings of AMN in a case of blunt ocular trauma.

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Case Report

An 18-year-old male presented to us with an acute history of blurring of vision following blunt trauma to the right eye by a wooden stick 4 days back. Visual acuity in the right eye was counting finger 2 meter, N36 and 20/20, N6 in the left eye. Left eye anterior segment was normal. Conjunctiva was congested with 2 + anterior chamber cells and flare, and 1 + vitreous cells. Right eye had a dilated, non-reacting pupil. Intraocular pressure on goldmann applanation tonometry (GAT) was 33 mm of Hg and 13 mm Hg in the right and left eye respectively. Fundus examination of right eye showed absent foveal reflex. Spectral domain optical coherence tomography (SD-OCT) scan showed hyperreflectivity of the outer nuclear layer (ONL) with a disrupted ellipsoid layer and external limiting membrane with few cystic spaces. [Fig. 1a and b] Multicolor composite image, reflectance and autofluorescence images were unremarkable. Based on above findings, a diagnosis of AMN was made. Patient was managed conservatively with anti-glaucomatous medication; cycloplegics eye drop and topical prednisolone acetate eye drop every hourly for a week, tapering over a period of next one

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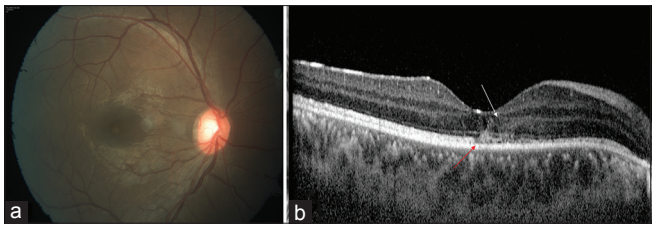

Figure 1: (a) Color fundus photograph of the right eye with no foveal reflex. (b) SD-OCT scan of the right eye showing the hyperreflectivity in the outer nuclear layer [White arrow] with a disrupted ellipsoid layer [Red arrow] and a few cystic spaces

month. The patient reviewed after one month. A repeat OCT showed a restoration of outer retinal layers with a focal external limiting membrane (ELM) disruption and outer nuclear layer thinning [Fig. 2] though the visual acuity in his right eye remained as before. Intraocular pressure on GAT was 19 mm of Hg and 12 mm of Hg respectively with a resolved inflammation.

Discussion

Blunt trauma to the eye causes commotio retinae due to the shock waves that traverse the eye from the site of impact. Ophthalmoscopically, sheen like retinal whitening appears some hours following injury due to extracellular edema, glial swelling, and photoreceptor outer segment disruption. Histopathologic studies have shown that it is characterized by disruption of photoreceptor outer segments and retinal pigment epithelial damage. In High-speed ultra-high-resolution OCT (UHR-OCT) and SD-OCT, retinal disruption at the level of the outer and inner photoreceptor layers and retinal pigment epithelial layer is seen[2] and is mostly associated with retinal atrophy and pigmentary disturbance.

AMN was first described by Bos and Deutman. It affects healthy women in whom acute paracentral scotomas develop. These patients may present with lobular, reddish brown, wedge-shaped lesions found in the macular region as their principal ophthalmoscopic manifestation. AMN develops near the middle retina at the junction of the outer plexiform (OPL) and ONL, associated with outer macular disruption and ellipsoid layer loss or ELM defect. Two types of AMN lesions have been described with SD-OCT, occurring above and below the OPL. Type 1 hyperreflective bands in the OPL/INL region with subsequent inner nuclear layer (INL) thinning, and Type 2 hyperreflective bands in the OPL/ONL region with subsequent ONL thinning.[3] AMN has been described after the use of oral contraceptives, post-viral illness, vasoconstrictive agents, hypovolemic shock, anemia, thrombocytopenia, terson syndrome, SUSAC syndrome, and dengue fever.[4-8] Microscopically retina has 10 layers.

There are two sources of blood supply to the retina: the central retinal artery and the choroidal blood vessels. The choroid receives the greatest blood flow (65–85%) and is vital for the maintenance of the outer retina. The remaining 20–30% flow to the retina is through the central retinal artery from the optic nerve head to nourish the inner retinal layers. Four different capillary networks are identified in different layers of retina. They are (1) in the nerve fiber layer (2), one in the ganglion cell layer (3), one at the junction of inner plexiform layer and superficial part of inner nuclear layer, and (4) one at the boundary of inner nuclear layer and outer plexiform layer. According to literatures, 1 and 2 form the Superficial Capillary Plexus (SCP) and 3 and 4 form the Deep Capillary Plexus (DCP). Acute ischemia causes axonal swelling and is responsible for the hyperreflective signal in OCT. Animal studies have shown that oxygen tension is highest in choroid then dips as we move towards retina, lowest being at ONL-OPL junction and then rises again at superficial retina. Therefore, OPL appears to be at a watershed zone and very much vulnerable to ischemic damage.[9]

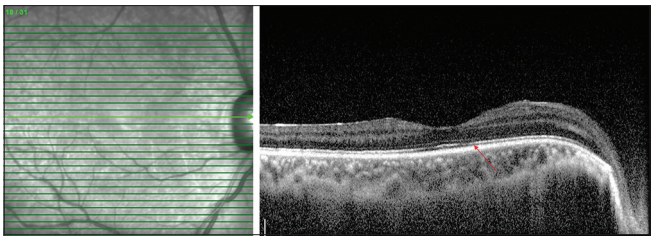

Figure 2: Follow up OCT of right eye showing thinning of outer nuclear layer with persistent focal ELM deficit [Red arrow]

Gillies *et al.* were the first to describe indirect trauma as a possible cause of retinopathy resembling AMN. They suggested a vascular aetiology and hypothesized that a sudden rise of intravascular pressure due to trauma may cause an acute breakdown of the capillary blood–retinal barrier. While the extravascular fluid would be removed quickly, the structural damage to the retina would persist. Nentwich *et al.* in their report on six eyes (five patients) with blunt trauma suggested that indirect trauma can cause changes in the outer retina resembling those seen in AMN.[10] They found a disruption of the inner segment/outer segment (IS/OS) junction with defects at the level of the photoreceptor outer segments, which was in line with the findings in non-traumatic AMN. AMN is a subtle lesion which can be diagnosed only with SD-OCT. It has been reported with many systemic diseases and syndromes like SUSAC syndrome, dengue fever, terson syndrome, anemia, etc., Such varied etiological spectrum of AMN suggests that it could be a clinical sign or manifestation of a group of pathologies and not a specific disease *per se*.

Conclusion

Identifying AMN in a setting of blunt trauma is important as it is an indicator of poor visual prognosis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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