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Case report

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A CASE OF KNUCKLE SANDWICH IN THE CHOROID (POST TRAUMATIC CHOROIDAL RUPTURE) A RARE CASE REPORT WITH LITERATURE REVIEW

[1] Dr.Vimal J. Vyas and [2,] *Dr. Ashish Pandey

[1]Professor and Head, Department of Ophthalmology, P.D.U Government Medical College, Rajkot, India
[2]Resident and Corresponding author, Department of Ophthalmology, P.D.U Government Medical College, Rajkot, India

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ABSTRACT

We report a rare case of Unilateral Choroidal Rupture in a young male patient occurring following blunt trauma to the eye with the stone that accidently hit his left eye while he was working in his field. Patient presented with pain and blurring of vision in his left eye with restriction of ocular movement in left eye. Funds examination and Optical coherence tomography reveled extensive choroidal rupture with surrounding intraretinal hemorrhage. Patient was being closely monitored for few days for worsening of vision and then was referred to higher centre for further management with explained poor visual prognosis.

Keywords: Choroidal Rupture, intraretinal hemorrhage, poor visual prognosis.

INTRODUCTION

A 30 year old male patient, with no previous co morbidities, complaint of blurring of vision in LEFT eyes since 3 days , which was, sudden in onset, painless and was associated with pain. Patient had history of blunt trauma with a stone while he was working in his field 2 days back. No complaints of headache, fever, nausea , vomiting, giddiness. On examination patient is conscious, and oriented to time, place and person and vitally stable. Patient was having pupil: 2mm , reacting to light with vision in RE = 6/6 and vision in LE= 3mfc with grade 3 RAPD pupil .

On slit lamp examination ,

- Lids/lashes:
  - OS: Small abrasion of upper eyelid, ecchymoses and edema of upper and lower lids, lids not taut, easy to open
  - OD: Normal
- Conjunctiva/sclera:
  - OS: 360 degrees of non-bullous sub conjunctival hemorrhage
  - OD: Clear and quiet
- Cornea:
  - OS: Clear
  - OD: Clear
- Anterior chamber:
  - OS: Deep, 2+ pigmented cell, 1.5 mm layered hyphema
  - OD: Deep and quiet
- Iris:
  - OS: Fixed, dilated (Traumatic mydriasis)
  - OD: Normal
- Lens:
  - OD: Clear, no pseudophacodonesis

Dilated Fundus evaluation showed white- yellow crescent shaped lesion just nasal to fovea extending down in inferior arcade, intra retinal hemorrhages surrounding the optic disc extending into nasal macula with surrounding Berlins edema. And on peripheral examination with sclera depressor 360 degree with scattered pre retinal hemorrhages nasal to optic nerve and inferior to the inferior arcade, no retinal breaks and dialysis. No evidence of any disc edema was seen. Patienr was the started on ofloxacin + dexamethas one eye drops 1 hourly along with antiglaucoma drugs (dorzolamide 2% + timolol 0.5%) for the traumatic hyphema and was closely followed for raised IOP and any vision improvement. On subsequent follow up patient was found to have normalized IOP and his vision also improved to 6mfc , but again on next follow up his IOP RAISED TO 54 mmhg and on gonioscopic evaluation it was found that angle recession was present inferiorly. and was kept on close follow up. After one month on follow up patient complained of decreased vision again and funds photo revealed a choroidal neovasculisation at site of choroidal rupture nasal to fovea. Patient was then referred to higher centre for the VR surgeon opinion and further management.

***Corresponding Author: Dr. Ashish Pandey,**
2Resident and Corresponding author, Department of Ophthalmology, P.D.U Government Medical College, Rajkot, India.

DISCUSSION

Etiology/Epidemiology/Mechanism

Choroidal rupture, first described by von Graefe in 1854, is a break in the choroid, Bruch's membrane, and the retinal pigment epithelium (RPE). This usually occurs secondary to blunt or penetrating ocular injury. Studies have found up to 5-10% of cases of blunt ocular trauma result in choroidal rupture [Williams *et al.,* 1990]. Choroidal rupture can be classified as direct or indirect depending on the mechanism of action. A direct choroidal rupture occurs anteriorly at the site of impact, generally parallel to the ora serrata and peripheral retina. In contrast, an indirect choroidal rupture occurs away from the site of impact, more posterior, and often concentric to the optic disc in a crescent shape. An overwhelming majority (~80%) of choroidal ruptures are indirect [Williams *et al.,* 1990]. The exact mechanism behind a choroidal rupture remain unknown but is thought to be related to mechanical forces occurring during trauma. Anteroposterior deformation of the globe expands the eye equatorially. This causes a sheering force that radiates concentrically from the nerve. The additional tensile strength of the sclera and the elasticity of the retina make these structures relatively resistant to rupture. In contrast, Bruch's membrane is relatively less elastic and therefore breaks more easily, along with the choroid and RPE [Williams *et al.,* 1990;

Dubinski and Sharma, 2006; Wyszynski, *et al.,* 1988]. Patients with pseudoxanthoma elasticum have been shown to have brittle Bruch's membrane, thereby making them more susceptible to choroidal rupture following relatively minor trauma to the eye [Williams *et al.,* 1990].

Histopathology

Histopathologic analysis of choroidal ruptures reveals early hemorrhage - often there is extensive choroidal, sub retinal, and vitreous hemorrhage [Williams *et al.,* 1990; Aguilar and Green, 1984]. The overlying retina is generally continuous but may show early thinning or atrophy. As early as four days post-injury, fibroblastic activity begins and is usually present between days 6-14. This is followed by extensive scarring extending into the sub retinal space, the retina, and as far as the inner retina and/or vitreous by weeks 3-4. Hyperplasia of the retinal pigment epithelium is also a common finding at 3-4 weeks post-rupture [Aguilar and Green, 1984]. Choroidal neovascularization is a common occurrence in the healing process post-choroidal rupture, but in most instances the new blood vessels regress. However, in some cases the choroidal neovascularization (CNV) persists and becomes a sight-threatening complication [Aguilar and Green, 1984].

Signs/Symptoms

Depending on the location of the rupture, the degree of associated choroidal or vitreous hemorrhage, and other associated injuries, visual acuity from choroidal ruptures can range from 20/20 to worse than 20/400. Patients with sub foveal or perifoveal lesions generally have the worst presenting visual acuity. The ruptures clinically appear as white to yellow-red crescent-shaped lesions that are often wider in the center and tapered at the ends. In addition, there is usually associated retinal edema and scattered hemorrhage. In most instances, there is a single rupture, though approximately 25% of affected eyes will contain multiple ruptures. Choroidal ruptures are located temporal to the disc in about 80% of eyes [Wyszynski, *et al.,* 1988]. A study by Ament et al., which examined 111 cases of traumatic choroidal rupture, found ruptures occurred in the macula in approximately 68% of cases, with 37% of cases being foveal and 31% being extrafoveal [Ament *et al.,* 2006]. Choroidal ruptures typically develop a fibrotic or gliotic appearing scar within a few weeks, followed by hyperpigmentation at the margins of the healed lesions. These lesions should be monitored closely for development of CNV. It is important to instruct patients to report any changes in central vision, which may include reduced visual acuity or metamorphopsia. Other reported complications from choroidal rupture include late hemorrhagic detachment of the retinal pigmented epithelium [Gitter and Slusher, 1968], serous detachment of the macula [Fuller and Gitter, 1973], or chorioretinal vascular [8] anastomoses. Visual outcomes following choroidal rupture are variable. Wood et al. reported that 17 of their 30 patients recovered with 20/40 best corrected visual acuity or better after an average 4.5 year length of follow-up [Wood and Richardson, 1990]. Secrétan and colleagues demonstrated similar results in 79 of their patients after a median follow up of 4 years [Secrétan *et al.,* 1998]. In contrast, a larger study by Ament et al. [Wood and Richardson, 1990] found only 34% of eyes (of 111 patients) recovered driving vision (20/40 or better) at median follow up of 20 months. The median visual acuity at last evaluation was 20/100. Recovery of driving vision was seen in 59% of eyes with peripheral choroidal ruptures, 22% with macular choroidal ruptures, 38% without CNV, 8% with CNV, 39% without retinal detachment, and 7% with retinal detachments. Ament et al. [Ament *et al.,* 2006] found that failure to recover driving vision was significantly correlated with foveal location of the rupture, multiple ruptures, and poor baseline visual acuity (less than 20/40).

Choroidal Neovascularization

Choroidal neovascularization (CNV) occurs in many cases of choroidal rupture. Approximately 5-25% of choroidal ruptures develop persistent CNV with substantial loss of central vision [Youssri Al and Young, 2002; Wood and Richardson, 1990]. Risk factors for the development of CNV as a sequela of choroidal ruptures are still debated, though most authors agree that macular ruptures are significantly associated with CNV formation as opposed to ruptures in the peripheral retina. Some studies demonstrated that ruptures closer to the fovea were more likely to develop CNV than ruptures farther from the fovea. For example, Secrétan et al. [Secrétan *et al.,* 1998] found that ruptures located closer to the foveal avascular zone were more likely to develop neovascularization than ruptures located peripherally (>1500 um from the foveal avascular zone). If neovascularization were to occur, it was more likely seen in the first year after rupture (~82% of eyes) [Secrétan *et al.,* 1998]. This suggests that follow up should ideally occur more frequently in the first year after traumatic choroidal rupture. Table 1 summarizes key features that may help predict CNV formation in patients with choroidal ruptures.

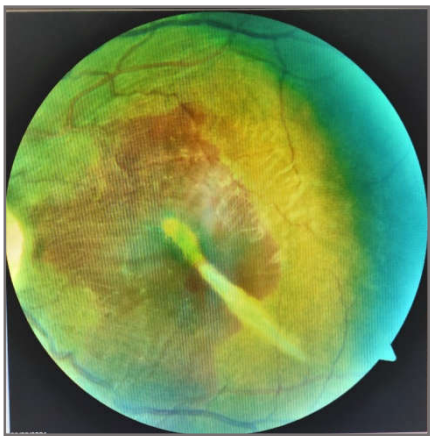
Risk Factors for CNV Formation After Choroidal Rupture [Ament *et al.,* 2006; Secrétan et al., 1998]

Risk Factors
• Longer Length of Rupture (>2.35 mm)
• Location of Rupture (Macula > Peripheral)
• Age of Patient (>65)
• Within First Year of Rupture
• Ruptures Closer to Fovea

Imaging

A variety of imaging modalities can help with visualization of choroidal ruptures that may be difficult to see initially via ophthalmoscopy. Often, there is hemorrhage, commotio retinae, and/or retinal pigment epithelium abnormalities that may obscure the rupture. It is also not unusual to have ruptures that are too small to visualize clinically. Optical coherence tomography (OCT) usually shows a loss of continuity of the RPE layer. Generally, two types of patterns are seen on OCT. The first pattern may show a forward protrusion of the RPE-choriocapillaris layer with an acute dome or pyramid shape. The second type may show a posteriorly concave area of disruption of the RPE-choriocapillaris layer [Nair *et al.,* 2013]. Funds auto fluorescence typically reveals hypo auto fluorescence in the area of the rupture from loss of RPE tissue in addition to blockage by sub retinal hemorrhage. There is hyper auto fluorescence at the rim of the lesion, likely secondary to RPE hyperplasia at the rupture margins . Fluoresce in angiography (FA) can also be helpful in some cases. Typical findings on FA include early hypo fluorescence from disruption of the choroid and choroiocapillaris in the area of the rupture with staining in late frames due to dye leakage into the scar. Fluoresce in angiography can also be useful in determining the presence of choroidal neovascularization. Indocyanine green angiography (ICG) may be more useful than FA due to less interference from the overlying hemorrhage, which is generally seen early in choroidal rupture. In addition, the choroid has markedly improved visualization with ICG due to less leakage of the dye from the choriocapillaris. ICG generally shows hypocyanesence at the rupture site throughout all frames and may even identify ruptures that are initially not seen clinically or on FA [Patel, *et al.,* 2013]. Lastly,

optical coherence tomography-angiography (OCT-A) is a newer, alternative imaging modality that is both quick and non-invasive. It can be used to detect CNV earlier in the disease course and to follow its progression. On OCT-A, choroidal rupture typically appears as a hypointense break in the choriocapillaris plexus [Lorusso *et al.,* 2019]. If CNV forms, OCT-A may demonstrate a well-circumscribed lesion with a clear hyperintense signal characterized by numerous surrounding anastomotic vessels in the avascular layer of the retina [Lorusso *et al.,* 2019]



[Figure 1] The Fundus photograph of left eye shows The white-yellow crescent shaped lesions surrounding just nasal to the fovea consistent with choroidal ruptures. There were pre-retinal hemorrhages scattered in the midperiphery and between the macula and optic disc.

Conclusion

Traumatic choroidal rupture which was complicated by the secondary choroidal neovascularization.

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