PEER REVIEWED

Commotio Retinae

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Abstract

Commotio retinae is an acute traumatic retinopathy due to direct and/or indirect blunt trauma to areas in and around the eye. Most often presenting with retinal whitening, commotio retinae involves disruption of the outer retina, namely the photoreceptors and the retinal pigment epithelium. Risk factors include high impact sports, violence, motor vehicle accidents, and other activities associated with blunt trauma to the head and orbits. This case report reviews the epidemiology, pathophysiology, differential diagnosis, and treatment options for commotio retinae.

Key Words: *commotio retinae, optical coherence tomography, trauma, Berlin's edema, retinal whitening*

Introduction

Commotio retinae is acute retinopathy resulting from blunt trauma to the globe. Commonly seen as whitening of the deep sensory retina, it has been recognized histologically as a disruption of the photoreceptor outer segment and damage to the retinal pigment epithelium (RPE).[1,2] Commotio retinae has also been referred to as "Berlin's edema" as it was originally theorized that the discoloration was caused by extracellular edema. However, since that time many studies have disproven this hypothesis.[2,3] Differentials include other traumatic retinal conditions such as choroidal rupture, Purtscher retinopathy, traumatic macular hole, chorioretinitis sclopetaria, and retinal detachment. Non-traumatic differentials include conditions that present with a similar whitening as seen in ischemic retinal conditions such as retinal artery occlusions (RAOs).

Patients typically present with chief complaints of blurry vision, visual field loss, and/or metamorphopsia following trauma. Standard of care when diagnosing commotio retinae includes a thorough eye exam including dilation and scleral depression to aid in diagnosing any accompanying retinal breaks or detachments.[4] Furthermore, additional ocular injuries associated with blunt trauma, such as open globe penetrations, orbital fractures, lens subluxation, and macular holes, must be ruled out upon initial assessment.

With optical coherence tomography (OCT), clinicians can obtain in vivo images of the specific retinal layers affected by commotio retinae. The affected layers can then be monitored for resolution as the condition currently has no treatment and usually resolves without any visual sequelae.[5] This case report aids clinicians in utilizing and interpreting spectral-domain OCT (SD-OCT) findings in their management of commotio retinae.

Case Description

A 21-year-old Caucasian male presented to the eye clinic 8 hours after being hit in the right eye with a soccer ball. He had a chief complaint of decreased vision in the right eye since that time. He denied having pain and any flashes or floaters in his vision. His ocular history was significant for myopia but otherwise unremarkable. The patient's medical history was also unremarkable. He denied taking

medications or having any drug or non-drug allergies. There was no family history of ocular disease.

His best-corrected visual acuity was 20/40 in the right eye and 20/20 in the left eye. Testing with pinhole showed no improvement in acuity of the right eye. No significant findings were noted on external examination. The patient's pupils were equal, round, reactive, without afferent pupillary defect; confrontation visual fields were full in both eyes; ocular motilities demonstrated a full range of motion in both eyes; slit lamp exam was unremarkable. Intraocular pressure measured with Goldmann applanation tonometry was 10 mmHg in each eye. Dilated fundus examination with accompanying scleral depression revealed no significant retinal findings in the right eye **(Figure 1)**. The optic nerve was flat and distinct with a cup-to-disc ratio of 0.4 round. Examination of the left eye was unremarkable. Despite the normal retinal appearance in the right eye, a macula OCT scan was performed due to the reduced visual acuity. The scan was reliable based on signal strength and lack of artifacts. Results revealed disruption of the foveal inner segment/outer segment (IS/OS) junction **(Figure 2)**. Imaging of the left eye was unremarkable. Based on the patient's history of trauma and OCT findings, he was diagnosed with grade 1 commotio retinae in the right eye.[6]

No treatment was indicated at that time, and the patient was scheduled for a follow-up exam in 4 weeks. He was asked to return to the clinic if he noted any changes in vision. At the follow-up visit, the patient noted a subjective improvement in his vision. The visual acuity in the right eye had improved to 20/20. All other findings, including with dilation, were stable to the previous examination. Scleral depression was unremarkable. A repeat OCT revealed complete resolution of the previous IS/OS disruption in the right eye **(Figure 3)**. The patient was asked to return for a follow-up visit in 4 weeks for repeat dilated fundus examination and to rule out traumatic angle recession with gonioscopy. The patient failed to return and was lost to follow-up.

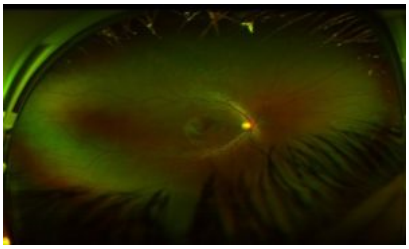

Figure 1. Fundus photograph of the right eye demonstrating no visible retinal or macular abnormality. Click to enlarge

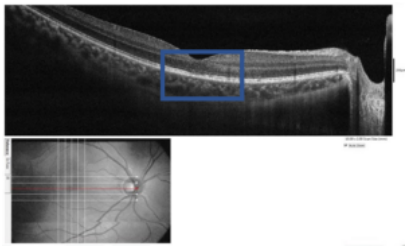

Figure 2. OCT scan of the right eye 8 hours post-trauma. Disruption of the IS/OS junction is present representing grade 1 commotio retinae (blue box). Click to enlarge

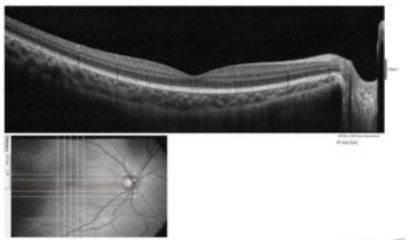

Figure 3. OCT scan of the right eye 4 weeks post-trauma. Disruption of the IS/OS junction had resolved. Click to enlarge

Education Guidelines

Key concepts

1. The basic anatomy, physiology, and function of the retina and its response to trauma
2. Commotio retinae may have minimal or no clinical signs, which makes additional testing important for accurate diagnosis
3. Clinical signs and symptoms to help differentiate commotio retinae from similar conditions
4. Recognition of OCT findings and long-term implications in patients with commotio retinae
5. Education on proper eye protection in work and sports to prevent ocular trauma

Learning objectives

1. Recognize the clinical presentation of commotio retinae including signs and symptoms

2. List the potential differential diagnoses of commotio retinae
3. Generate an appropriate management plan based on the results of multi-modal testing such as OCT

Discussion questions

1. Knowledge, understanding, and facts about the clinical case and condition presentation

a. describe the typical appearance and presentation of commotio retinae
b. discuss complications of trauma that can accompany commotio retinae

2. Differential diagnosis

a. what other condition(s) should be considered as differential diagnoses for commotio retinae and how can a clinician differentiate between them?

3. Patient management and role of the optometrist

a. what treatment is indicated for commotio retinae?
b. what is the prognosis for commotio retinae patients with macular involvement?
c. what is the prognosis of commotio retinae patients with no macular involvement?
d. what type of patient education is required for patients who have experienced traumatic injuries to the eye?

4. Critical-thinking concepts

a. what is the value of OCT in managing patients with commotio retinae, especially macula-involving?
b. what additional testing should be performed in patients who are diagnosed with commotio retinae?

Assessment of learning objectives

Foundational knowledge of retinal anatomy is vital in understanding the pathophysiology of commotio retinae. Therefore, retinal OCT serves as a perfect teaching tool for educators. Educators could present normal retinal OCT scans and assign the students to properly identify and label anatomical landmarks. Once completed, this could be compared to retinal OCTs of patients with commotio retinae. Students would be asked to compare and contrast the two OCT images and try to arrive at how disruption or damage to specific retinal structures leads to corresponding patient signs and symptoms. This assignment could be done in small or large-group settings such as in a laboratory or clinic conference room. This could be presented to optometry students in all class years.

Higher-level concepts can also be assessed. Case studies can be presented to optometry students in their third and fourth year either in a formal or online classroom. Students can be initially presented the case details and then can be tasked with arriving at a proper diagnosis and management plan based on the presented findings and ancillary testing. Understanding and knowledge can be evaluated through open-ended questioning to the class or through formal testing with multiple-choice questions using a platfrom such as TurningPoint.

In an online format, the case can be presented along with appropriate adjunct testing and results. Students can then be tested, through a multiple-choice format, on concepts involving pathophysiology, signs and symptoms, differentials, interpreting testing, and treatment and management. Before students can move on to the next question in the series, they would be required to answer the questions correctly. Feedback can be provided after the question is answered correctly. This can allow students to learn from their mistakes and therefore strengthen their knowledge base.

Discussion

A retinal condition due to ocular trauma, commotio retina was first described by Berlin in 1873 as Berlin's edema as he thought extracellular edema was the cause of the presenting retinal findings.[1,3] Although the term Berlin's edema is still used at times today to describe commotio retinae at the macula, studies have shown no evidence of extracellular edema but rather damage to the photoreceptors and the RPE.[2,5,6] As trauma is the direct cause, many risk factors can lead to commotio retinae either in a contrecoup or coup fashion. Risk factors for commotio retinae include trauma from high-impact sports, namely ball sports, but can also include blunt trauma to the face and orbits from violent encounters, car accidents, and falls. One study noted the presence of commotio retinae in 30% of patients presenting to a hospital for traumatic eye injuries.[7] Young males make up the majority of patients affected by commotio retinae with studies noting the average age of patients to be 20-30 years.[2,7] Exact epidemiological numbers are likely lacking as many patients tend not to present to eyecare or other providers when symptoms are lacking, which can be the case with commotio retinae.

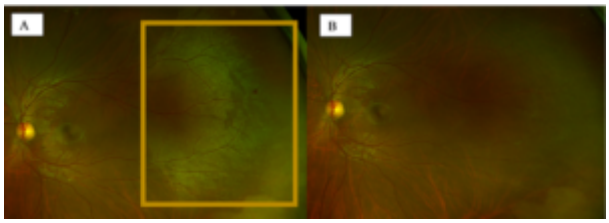

Figure 4. (A) An unrelated patient with temporal peripheral commotio retinae on initial presentation (yellow box). **(B)** Repeat imaging one month later showed complete resolution of the conditon.
Click to enlarge

Patient presentation can vary depending on the extent of trauma and the location of commotio retinae. Commotio retinae usually presents as a gray-white opacification of the retina with possible adjacent intraretinal hemorrhaging and retinal pigment disruption. If located at the fovea, patients can present with vision loss accompanied by field loss. However, some patients may be asymptomatic if the condition is located beyond the macula.[5,8] Patients can also present with vision loss without identifiable retinal whitening. In this case OCT is key in detecting photoreceptor damage.[9] The location of commotio retinae in the retina according to one study favored the temporal retina (inferior temporal, temporal, or superior temporal) **(Figure 4)**. This aligns with trauma affecting the overlying sclera as the nasal sclera is somewhat protected by the normal orbital anatomy.[7] The extent of vision loss varies and is dependent on the level and amount of photoreceptor disruption. If the condition is limited to the mid-periphery or periphery of the retina, visual acuity and visual field loss are unlikely to occur.[2,6] As the condition is traumatic in nature, patients can present with associated findings and complications. These can include angle recession, traumatic macular hole, retinal tear and detachment, corneal abrasion, choroidal rupture, lens subluxation, hyphema, orbital fractures, cataract, and traumatic uveitis.[1,5,8]

As previously mentioned, Berlin hypothesized that the retinal findings in commotio retinae represented intracellular edema; however, no intracellular edema is present.[3,6] Other proposed mechanisms of action have included glial cell proliferation and fragmentation of photoreceptor outer segments. However, in their study, Mansour et al. revealed the retinal findings were due to traumatic disruption to the outer retina. Specifically, it is thought that concussive forces from trauma damage the outer retinal layers due to their inelastic structure when compared to the elastic sclera or the elastic layers of the inner retina. This leads to disruption of the photoreceptor outer segments along with deposition of debris in the subretinal space and RPE disturbances.[1] With the advent of OCT, further investigations into the pathophysiology of commotio retinae have shown IS and OS hyper-reflectivity indicating disruption from the trauma while also correlating to areas of involvement noted on fundus examination.[1,10]

TABLE 1
Four-Step Grading Scale for Commotio Retinae

Grade	Morphological Features on SD-OCT
1	Increase in inner segment/outer segment (IS-OS) junction reflectivity
2	Loss of reflection in cone outer segment tips (COST)
3	COST and IS/OS junction defects
4	COST, IS/OS junction, and external limiting membrane (ELM) defects

SD-OCT = spectral-domain OCT

Table 1. Four-step grading system for morphological features of commotio retinae seen on SD-OCT as determined by Ahn et al.[6] Click to enlarge

Ahn et al. further classified the degree of photoreceptor and RPE involvement with a four-step grading scale for commotio retinae, which allowed for predictions in visual and anatomic outcome **(Table 1)**.[6] Four specific bands were identified in the outer retina: the external limiting membrane (ELM), the photoreceptor IS/OS junction, the cone outer segment tips (COST), and the RPE. Using these landmarks, grade 1 represents an increase in IS/OS reflectivity with disappearance of thin hypo-reflective optical space. Grade 2 represents COST defects. Grade 3 represents COST and IS/OS junction defects with grade 4 representing COST defects, IS/OS junction, and ELM defects.[6,11] It was concluded based on this study that eyes with a higher grade, and therefore more retinal layer involvement, at baseline scan correlated to worse visual and anatomical outcomes.[6]

With advances in technology, such as those seen with OCT angiography (OCT-A) and enhanced depth imaging OCT (EDI-OCT), additional pathophysiological components of commotio retinae have been suggested.

With EDI-OCT, evidence of choroidal involvement has been studied. Burke et al. noted an increase in subfoveal choroidal thickness and increase in choroidal areas in eyes with commotio retinae compared with the normal fellow eye. This also correlated with a decrease in visual acuity in the involved eye. Although the exact pathophysiology was unclear to the authors, they suggested the choroidal findings could be dilation of choroidal blood vessels in response to the trauma.[12] However, a more recent case series noted opposite findings. In this study, 70% of patients had a thinning in central choroidal thickness of the involved eye when compared with the fellow non-involved eye. This was attributed to decreased choroidal blood flow secondary to the trauma.[13] It can be concluded that additional studies are needed to clarify the role of the choroid in commotio retinae.

With OCT-A, researchers have attempted to elucidate possible retinal vascular involvement. Results have varied. Mansour and Shields as well as Wangsathaporn and Tsui did not note any changes in the superficial and deep capillary plexus or the choriocapillaris.[14,15] However, another more recent study suggested that vascular changes are present, as patients had enlargement of the foveal avascular zone along with decreased vessel density.[16] The authors suggested that their study differs from the previous two in that their patients had more severe blunt injuries resulting in the changes observed. They also stated that their findings correlated to previous studies in which laser speckle flowgraphy showed reduced choroidal blood flow and indocyanine green angiography showed choroidal vascular damage.[17,18] Although the studies showed possible associations of a hemodynamic nature, one should note that they were all case reports with very small sample sizes. Again, large case-controlled studies are needed to further validate a possible vascular component in commotio retinae.

Novel OCT findings have also been discovered in recent years. One group of researchers noted an original finding they termed micro-elevation of the ellipsoid zone in patients with commotio retinae. Although they noted the finding, they could not pinpoint an exact pathogenesis. Fortunately, the finding was temporary.[19] Another novel finding was a non-reflective dark space in the sclero-choroidal interphase of some patients with commotio retinae, which the study authors speculated may have

represented a fluid-filled space secondary to trauma. This finding was also temporary and it resolved during the clinical course of the condition.[13] These findings were not noted in our patient.

Differentials diagnosis

It is important to differentiate commotio retinae from other conditions that may present similarly, namely those of a traumatic nature. Common differential diagnoses include retinal detachment, choroidal rupture, Purtscher retinopathy, traumatic macular hole, and chorioretinitis sclopetaria. One should note that these differentials can present concurrently with commotio retinae due to their similar traumatic etiology. Other differentials can include conditions that can present with a white/chalky retina, namely, ischemic retinal conditions.

- The impact from blunt trauma to the eye may cause abnormal vitreous traction resulting in retinal tears, which can lead to rhegmatogenous retinal detachments (RRD).[20,21] Patients report an increase in floaters and flashes of light and a decrease in vision. When viewed clinically, a RRD typically has an elevated bullous appearance.[22]
- Traumatic macular hole is a full-thickness defect of the neurosensory retina. Due to this finding, entering visual acuity tends to be much worse than in cases with macula-involving commotio retinae. It may be present after ocular injuries and can be found concurrently with commotio retinae. Traumatic macular holes tend to occur in younger males and are also often associated with sports and work-related accidents.[23]
- Purtscher retinopathy, a rare, occlusive micro-vasculopathy, is characterized by multiple white areas surrounding the optic disc and fovea. Additional features include retinal hemorrhages, cotton wool spots, and Purtscher flecken (polygonal patches of intraretinal whitening in the posterior pole). These features are not typically seen with commotio retinae. Patients commonly present with sudden painless bilateral vision decrease within 48 hours of trauma, which can include direct head trauma, injuries or compression of the chest, and long bone fractures.[24,25] History, in addition to retinal findings, is key because commotio retinae tends to be a result of head trauma only.
- Trauma to the globe can result in a break in the RPE, Bruch's membrane, and choriocapillaris, and is termed choroidal rupture.[26,27] Choroidal rupture appears as white-yellow curvilinear or crescent moon-shaped lesions that eventually lead to retinal scarring. In direct globe injuries, these ruptures are located anteriorly and parallel to the ora serrata, whereas indirect injuries produce ruptures concentric to the optic disc.[27] This appearance is in stark contrast to the overall and transitory whitening seen with commotio retinae.
- Chorioretinitis sclopetaria, also known as traumatic chorioretinal rupture, is rupture to the choroid and overlying retina caused by forces from a high-velocity projectile (e.g., gunshot, BB gun) to or near the globe. Histopathology shows photoreceptor loss, damage in Bruch's membrane and the choroid, hyperplasia of the RPE, and multi-layered retinal hemorrhaging. The affected areas of the retina and choroid are subsequently replaced with fibrous tissue, giving the appearance of retinal whitening. This condition often has a poor visual prognosis, especially if the macula is involved.[28-30]
- RAOs also present with a white chalky retina; however, they can be differentiated based on retinal anatomy and etiology. Artery occlusions tend to affect older patients with underlying systemic conditions in an acute fashion, while commotio retinae patients tend to be young males with a history of trauma.[31] Artery occlusions also result in more severe vision loss if the fovea is affected compared with commotio retinae.[31] Clinicians can also use OCT to differentiate the conditions if needed, as commotio retinae affects the outer retina while RAOs affect the inner retina.[32]

Treatment and management

As of now, there is no approved treatment for commotio retinae. The condition tends to resolve over the course of a few weeks to a few months. Initial restoration of the photoreceptors tends to begin at 1 week post-injury.[7]

In general, the condition has a favorable prognosis, and most cases resolve completely within 4 weeks with no additional sequelae.[7] However, in more serious cases, patients may be left with permanent macular damage resulting in vision impairment and paracentral scotomas.[4,23] As mentioned earlier, eyes with a higher grade of commotio retinae, as determined by OCT, tend to have worse visual and anatomical outcomes.[6] Blanch et al. noted that in their study of 53 patients with macula-involving commotio retinae, 26% had an end acuity of less than or equal to 20/30. According to the authors, if symptomatic paracentral defects were accounted for, the number of patients with visual impairment (acuity loss and/or field loss) would likely have been higher. They also noted the prognosis of extra-macular commotio retinae to be very favorable with almost all patients recovering pre-trauma levels of visual acuity.[7]

As with many other ocular conditions affecting the posterior segment, steroids have been considered as a treatment option. Although rarely used, this includes systemic and intravitreal steroids. Mendes et al. in their case report used off-label high-dose intravenous methylprednisolone (550 mg) for 3 days followed by oral prednisolone (30 mg) for 10 days for a patient who presented with commotio retinae and counting fingers vision that had persisted for 2 months. The vision improved to 20/200 with this treatment regimen. The patient was then given an intravitreal injection of triamcinolone with vision improving and stabilizing at 20/150.[8] In another case report, oral prednisolone (50 mg) was used for 5 days in a patient with commotio retinae.[14] The visual acuity did improve from 20/30 to 20/20 over the course of a week, but one cannot assume the steroid led to this. The improvement may have been due to natural progressive healing. These case reports are of limited sample size. Larger studies are required to validate the use of any medications, including steroids, in the treatment of commotion retinae. Due to the favorable outcomes without interventional treatment and the lack of case-controlled studies on steroid treatment, steroids are not a viable treatment option currently.

Trauma being the direct cause of commotio retinae underscores the need for preventive measures that can be taken to avoid visual complications, namely avoidance of high-risk activities and proper use of protective eyewear (polycarbonate lenses, face masks, and protective visors). More than 600,000 sports and recreation-related ocular injuries occur every year, with a small proportion leading to permanent vision loss. Up to 90% of sports-related ocular injuries are preventable by using the correct eye protection.[33] Eye protection is strongly encouraged for all participants in sports in which there is a risk of injury.[33] Monocular precautions should be mandatory for those who have lost vision as a result of any traumatic condition, including commotio retinae. As patients may present with additional traumatic conditions/complications at onset, they should be addressed and treated appropriately.[4] In addition to performing scleral depression to rule out retinal breaks and detachments, clinicians should perform gonioscopy of the angle to rule out angle recession as an additional complication. One report noted that 60% of eyes developed some degree of angle recession after non-penetrating or concussive trauma.[34] Clinicians should be aware that angle-recession glaucoma can develop months to years after the initial trauma and therefore glaucoma testing may be warranted in the future.[35,36]

Conclusion

This case report highlights the importance of SD-OCT for diagnosing and monitoring commotio retinae, especially when there is a lack of obvious retinal whitening. Despite numerous previously documented cases with classic presentations, the condition may not always present with characteristic retinal whitening, as seen in this case.[9,37]

While there is no approved treatment for commotio retinae, it is advised that patients be monitored frequently post-diagnosis to evaluate for any treatable sequalae. It is also strongly encouraged that any individual participating in sports or other high-risk activities wear proper eye protection to prevent ocular injury.

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