In: Advances in Medicine and Biology. Volume 89 ISBN: 978-1-63483-478-0
Editor: Leon V. Berhardt

Chapter 5

TRAUMATIC RETINAL INJURIES

Steven S. Saraf*, Robert A. Prinzi, Daniel D. Kim, Ankit Desai, Nitin Kumar and Uday R. Desai
Henry Ford Hospital, Department of Ophthalmology,
Detroit, Michigan, US

ABSTRACT

We review common retinal injuries, their incidence, prognosis, and management. Macular hole formation and choroidal rupture are discussed, which result from anteroposterior compression of the globe from a blunt force. Advances in surgical techniques have shown good outcomes in addressing traumatic macular holes that do not close spontaneously. Commotio retinae and macular hemorrhages are other common injuries. The mainstay of management for commotio retinae is observation. New advances in surgical techniques have allowed recovery of vision in patients with certain types of macular hemorrhages. Retinal detachments and dialyses require prompt surgical intervention. Additional topics include retinal pigment epithelium tears, retinal contusion, retinal concussion, retinitis sclopetaria and whiplash retinopathy.

INTRODUCTION

The mechanisms of ocular trauma play a key role in understanding the pathophysiology of retinal injuries. The relative rigidity of ocular tissues, relationship of the vitreoretinal interface, and severity of trauma all contribute to the types of injuries seen in the posterior segment. Factors such as the ability of retinal tissues to recover, scar formation, and violation of ocular compartments dictate the need for acute intervention versus careful monitoring for secondary complications. A systematic review of common retinal injuries and their management is provided.

* E-mail: SSARAF1@hfhs.org; Phone: (607) 342-8705.

BLUNT TRAUMA

Blunt trauma to the globe leads to a number of retinal injuries that result from the relative rigidity of retinal tissues in comparison to the sclera. The layers of the posterior segment that are less compliant to expansion during blunt trauma include the thin foveal center, the retinal pigment epithelium (RPE), Bruch's membrane, and choroid. When there is rapid anteroposterior compression of the globe without rupture, the impact force leads to an increase in the equatorial diameter of the globe (Figure 1). The sclera easily conforms and absorbs the force of the trauma, whereas these other tissues have a tendency to rupture when stretched. When the force of blunt trauma is severe enough to cause globe rupture, the incidence of these types of injuries is less as the contents of the eye have a tendency to extrude rather than maintain their volume during compression. Therefore, injuries such as macular hole formation and choroidal rupture are suspected primarily in instances of low to moderate blunt force impact [1].

TRAUMATIC MACULAR HOLES

Traumatic macular holes (TMH) are rare in comparison to idiopathic holes, which makes it difficult to understand their incidence and prospectively study their management.

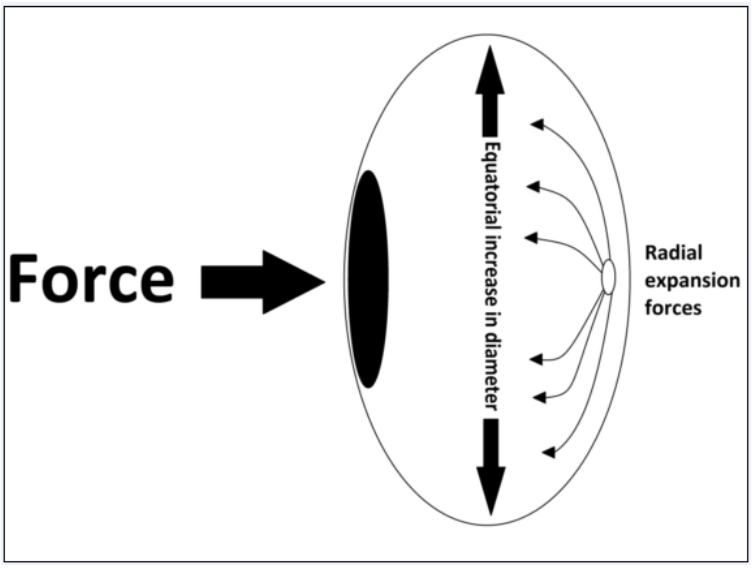


Figure 1. The equatorial expansion hypothesis for traumatic retinal injuries. When a blunt force is exerted to the globe, the equatorial diameter expands leading to radially oriented forces in the posterior pole. The sclera is compliant and conforms easily to the force, while other ocular tissues such as the fovea, internal-limiting membrane, Bruch's membrane, and choroid are less compliant and more apt to rupture.

Kuhn et al. reported that macular holes occur in 1.4% of closed globe injuries and 0.15% of open globe injuries [3]. TMH often occur in the setting of other traumatic retinal injuries such as commotio retinae, retinal hemorrhages, choroidal ruptures, or retinal breaks.

It is therefore important to thoroughly examine the trauma patient for a macular hole, especially if central vision is diminished.

TMH more commonly occurs in young males. In a large retrospective case series, the mean age of the patients was 27.1 years with 86% being men. None of the patients in the study were noted to have a PVD [4]. In the setting of blunt trauma, macular holes are thought to form acutely from the mechanical forces acting at the fovea during equatorial expansion of the eye. Lack of PVD with strong adhesions of the vitreous to the macula is thought to also contribute to its formation [1]. Alternatively, there have been published reports of macular holes forming up to 4 weeks after the initial injury (Figure 2) [5, 6]. The mechanism of hole formation in these cases is not thoroughly understood, but is thought to occur due to ruptures in the internal limiting membrane (ILM). These breaks allow the accumulation of vitreous fluid intraretinally with subsequent hole formation [4]. Intraoperative indocyanine green staining has confirmed the existence of breaks the ILM in the setting of acute blunt trauma [7]. A number of retrospective studies have examined the management of TMH, including observation versus pars plana vitrectomy with ILM peeling.

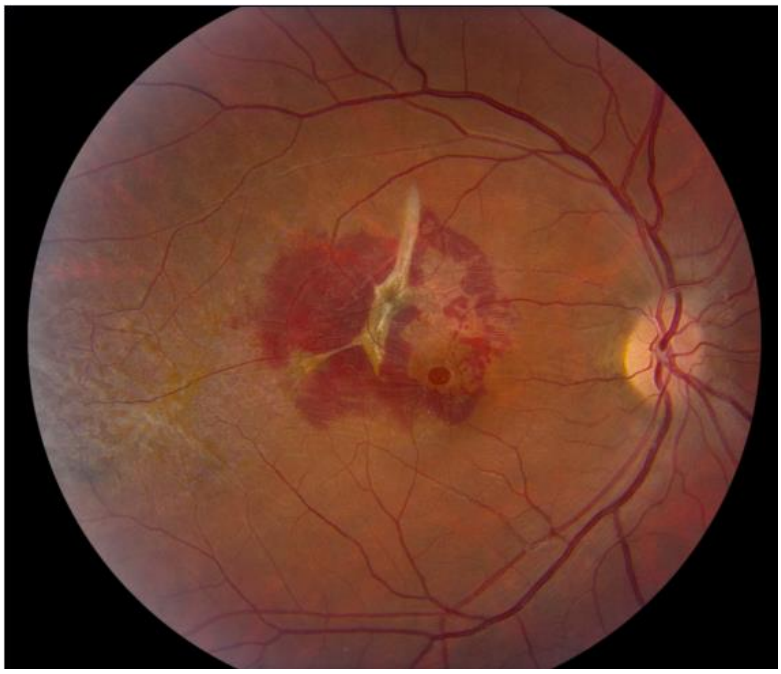


Figure 2. Blunt traumatic injury has led to a submacular hemorrhage. After observation for 4 weeks, the hemorrhage has cleared revealing a choroidal rupture. A macular hole has formed in a delayed fashion after resorption of the hemorrhage. The patient was observed and the hole was persistent. Final visual acuity was 20/160.

One study included 18 patients who were observed after TMH formation. Of the 18 eyes, 8 macular holes closed spontaneously. The average age of the patients with spontaneous closure was 14.6 years with earliest closure occurring within 1 week of diagnosis and the latest within 4 months. This study suggests that greater than 50% of observed cases may not close spontaneously and older patients may have a lower incidence of spontaneous resolution [6].

Five studies have retrospectively examined the success of pars plana vitrectomy in the closure of TMH [8–12]. The overall success rate of the combined studies is 83%, though a higher rate may be achievable with more modern techniques. The largest and most recent study included 25 patients who had pars plana vitrectomy with ILM peel. The authors reported a 96% closure rate with 84% of cases resulting in at least a 2 line improvement in vision. Sixty-four percent of cases achieved 20/50 vision or better [8]. Overall, the reviews suggest that the closure rate of TMH may approach that of idiopathic macular holes, which has been reported to be >90% when vitrectomy is paired with ILM peel [13]. In addition, statistical analyses have shown that a delay in macular hole repair does not result in significant vision loss. This suggests that a period of observation followed by pars plana vitrectomy with ILM peel is a reasonable approach [8].

CHOROIDAL RUPTURE

Choroidal ruptures typically appear as curved lines concentric to the optic nerve after rapid anteroposterior compression of the globe (Figure 2). They occur more frequently in the posterior pole or in the mid-periphery. Histologically, there is discontinuity in the RPE, Bruch's membrane, and choroid resulting from the equatorial expansion of the globe. In the subsequent weeks after trauma, there is fibrovascular proliferation in the intervening space, resulting in a dense fibrous scar with RPE hyperplasia [14]. Visual prognosis is typically poor if the central macula is involved and there is no known treatment for the rupture. The mainstay of management is careful observation for choroidal neovascularization, which results from the ingrowth of choroidal vessels and subsequent macular edema, leading to further visual morbidity. Fluorescein angiography will show a window defect at the area of rupture with subsequent staining of the fibrovascular scar. There will be leakage in the presence of a choroidal neovascular membrane [15].

In a retrospective study of 79 eyes, a group of eyes with choroidal rupture and neovascularization were compared to eyes with choroidal rupture alone. Eyes with neovascularization had statistically larger ruptures and tended to be closer to the fovea. These findings suggest closer observation may be required for patients with large, centrally located choroidal ruptures [16]. Neovascularization can be subsequently treated with laser, and case reports have shown good prognosis with anti-VEGF therapy [17].

RETINAL PIGMENT EPITHELIUM TEAR

The development of tears of the RPE has classically been described in wet age-related macular degeneration (wet AMD) in association with areas of choroidal neovascularization [18, 19]. Trauma is considered to be a rare cause of RPE tears. When there is significant injury to the eye, blunt forces will typically be strong enough to result in breaks of both the RPE and Bruch's membrane, resulting in choroidal rupture. More rarely, an intermediate force may be strong enough to tear the slightly weaker RPE, but not strong enough to break Bruch's membrane. This maintains separation between the retina and the choroid, but the underlying RPE becomes torn and scrolled [18–20]. Diagnostically, RPE tears can be

confirmed with fluorescein angiography. The images show a large window defect from where the torn RPE has been displaced with areas of blockage where the loose RPE has become scrolled [20]. Prognosis is typically poor if the fovea is involved as the overlying retina is poorly nourished without the function of the RPE cells. The typical management of RPE tears is observation. Although more recently, patients with RPE tears and wet AMD have been treated with 360 degree retinotomy with macular translocation. This has resulted vision improvement in a number of cases, but the application of this method in the setting of retinal trauma remains to be seen [21].

COMMOTIO RETINAE, RETINAL CONCUSSION, AND RETINAL CONTUSION

Commotio retinae, or retinal contusion, is an injury that results from blunt anterior trauma that leads to contrecoup injury to the posterior retina. Initially the vision may be reduced with normal fundus appearance, but within minutes to hours, the retina opacifies to a creamy color with poorly defined borders. If the macula is spared, the vision may be normal. When the macula is involved (Berlin's edema), the vision can be greatly reduced.

Although the term "Berlin's edema" is used, the pathophysiology does not reflect the presence of extracellular edema. The opacification noted on exam is a result of intracellular fluid accumulation in the photoreceptors and RPE along with photoreceptor outer-segment disorganization.

Therefore, fluorescein angiography shows blockage of the underlying choroidal circulation at the affected areas [22].

The prognosis of commotio retina is typically excellent, unless other complicating injuries such as choroidal rupture or subfoveal hemorrhage occur. The retinal opacification generally clears in the weeks after the injury with vision commonly returning to baseline. Occasionally, a permanent decrease in visual acuity occurs, especially in the presence of other injuries such as macular holes, subfoveal hemorrhage, or choroidal rupture [22, 23].

Retinal concussion is the mild form of commotio retinae (Figure 3). The retinal opacification is less dramatic and decreased vision is usually better than 20/200 when it involves the macula. The retinal whitening improves and clears within few days with observation [2]. The vision changes recover over time with minimal deficit [2]. Fluorescein angiogram shows blockage of the choroid and some staining of the RPE in the affected area [23].

Retinal contusion is a term applied to a severe case of commotio retinae (Figure 4). The retinal opacification is dramatic and can be associated with hemorrhages [24]. The decrease in vision is variable, ranging from mild to severe and does not correlate with degree of retinal whitening [22, 23].

Given its severity, the retina may not recover entirely. There is usually permanent vision loss, especially with macular involvement [23]. The retinal whitening takes longer to recover than a retinal concussion but will eventually resolve. The RPE can also be concurrently damaged and in the long term and result in pigmented scars (Figure 5).

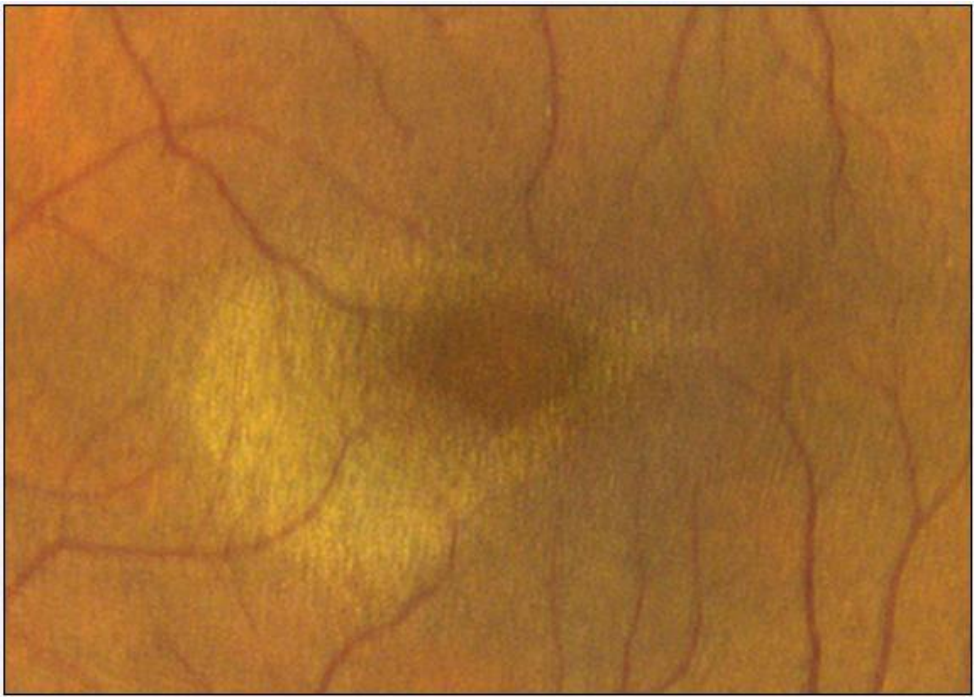


Figure 3. Retinal concussion describes mild commotio retinae. When involving the fovea, such as in this case, it is also known as Berlin's edema. There is mild retinal opacification. The opacification is due to intracellular edema and disorganization. There is no leakage on fluorescein angiogram.

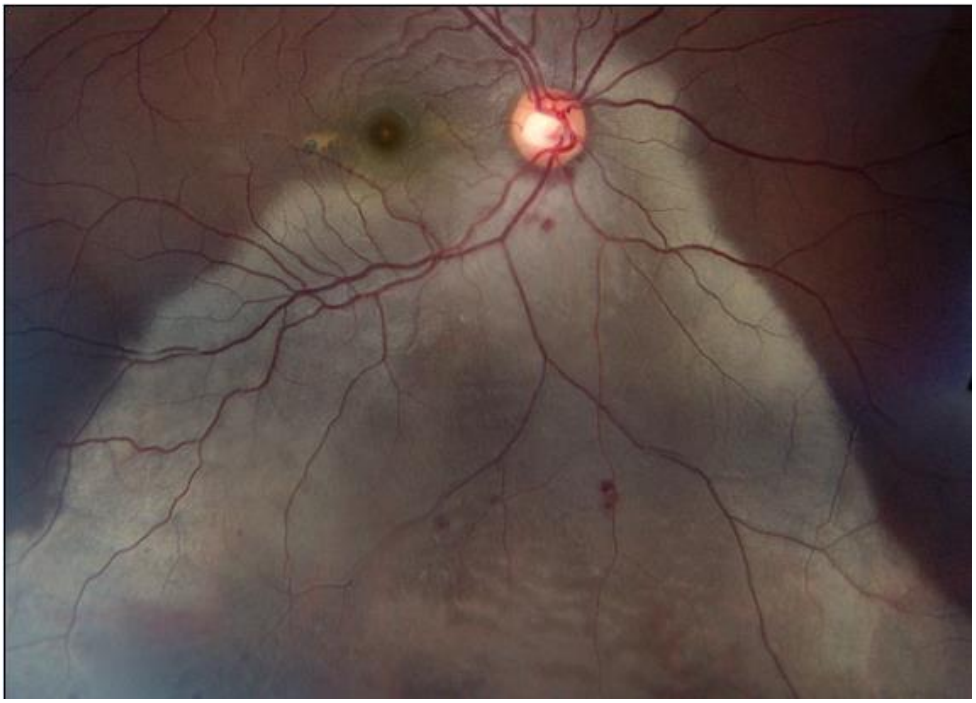


Figure 4. Retinal contusion describes dense commotio retinae associated with intraretinal hemorrhage.

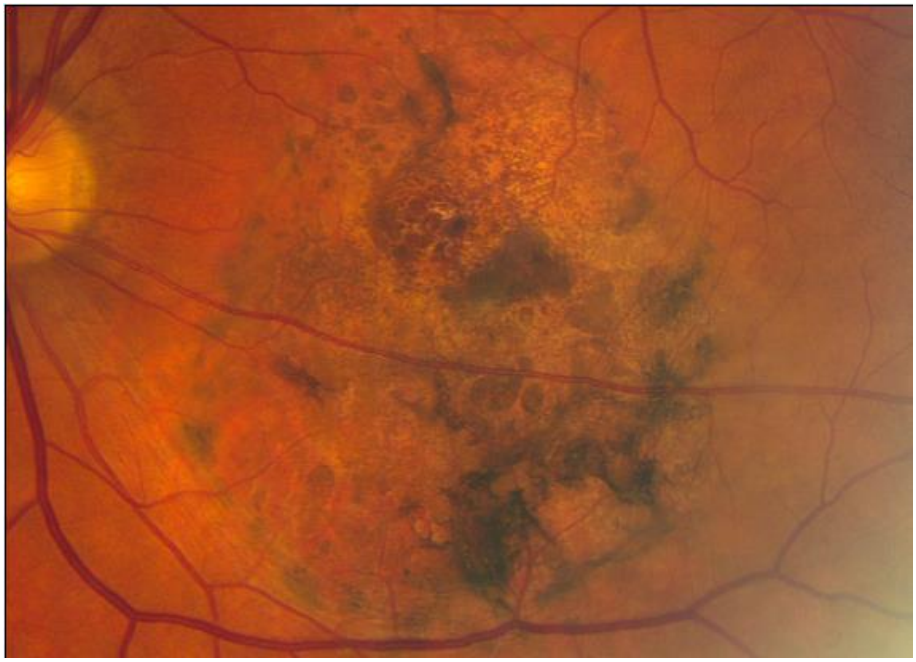


Figure 5. Retinal contusion may be severe enough to lead to RPE injury and scarring. Visual prognosis is poor when the macula is involved.

Fluorescein angiography is similar to retinal concussion with the retinal whitening blocking the underlying choroid. There is more intense staining and/or leakage at the level of the RPE in the affected area [23].

RETINAL HEMORRHAGES

Retinal hemorrhages are a common finding in retinal trauma. Depending on the type of injury, retinal hemorrhages can be located at the sub-RPE level in association with choroidal rupture (Figure 6-7). They can also be subretinal, intraretinal, sub-internal limiting membrane (ILM), sub-hyloid, or any combination of the above (Figure 8). Hemorrhage can also occur into the vitreous space in heavy traumatic injuries. Vision decrease is variable depending on the area involved and the amount of hemorrhage. Hemorrhages involving the macula or dense vitreous hemorrhage more commonly lead to vision loss.

Retinal hemorrhages over time becomes dehemoglobinized and may cause damage to the retina. The dehemoglobinized blood expresses free iron, which then catalyzes the formation of reactive oxygen species. Reactive oxygen species lead to direct oxidative damage to the neurosensory retina and RPE [25]. Iron has also been shown to migrate from hemorrhages in the vitreous or inner retina to the outer retina and RPE, resulting in oxidative damage to the photoreceptors and RPE [26].

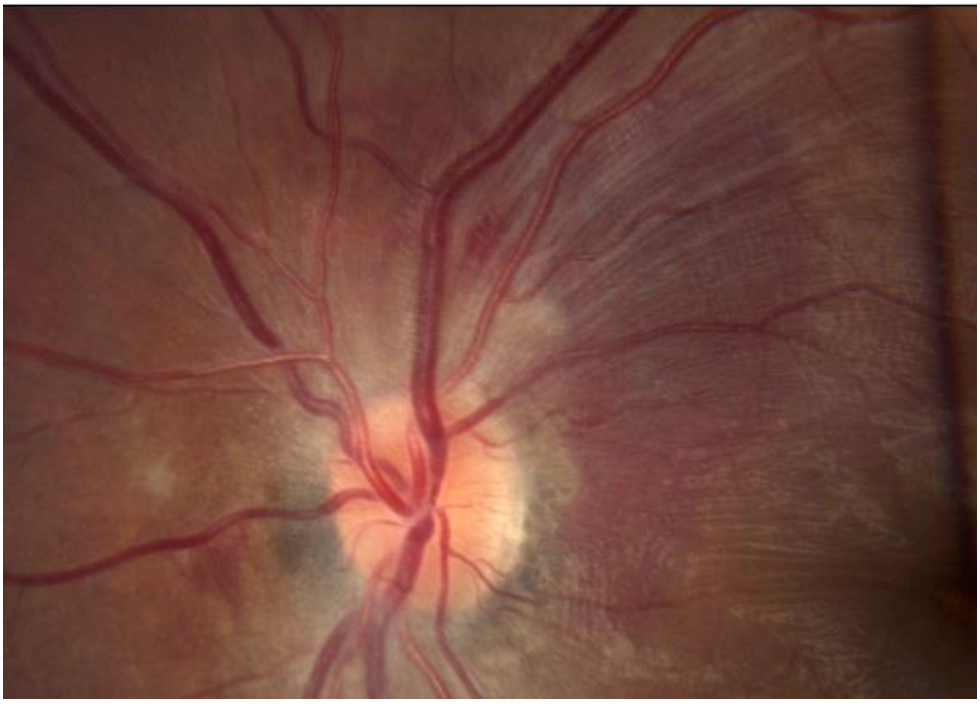


Figure 6. A sub-RPE and subretinal hemorrhage is show adjacent to the disc. There is resolving Berlin's edema.

Figure 7. A traumatic multilayered retinal hemorrhage is shown with subretinal and intraretinal components. It is associated with a choroidal rupture.

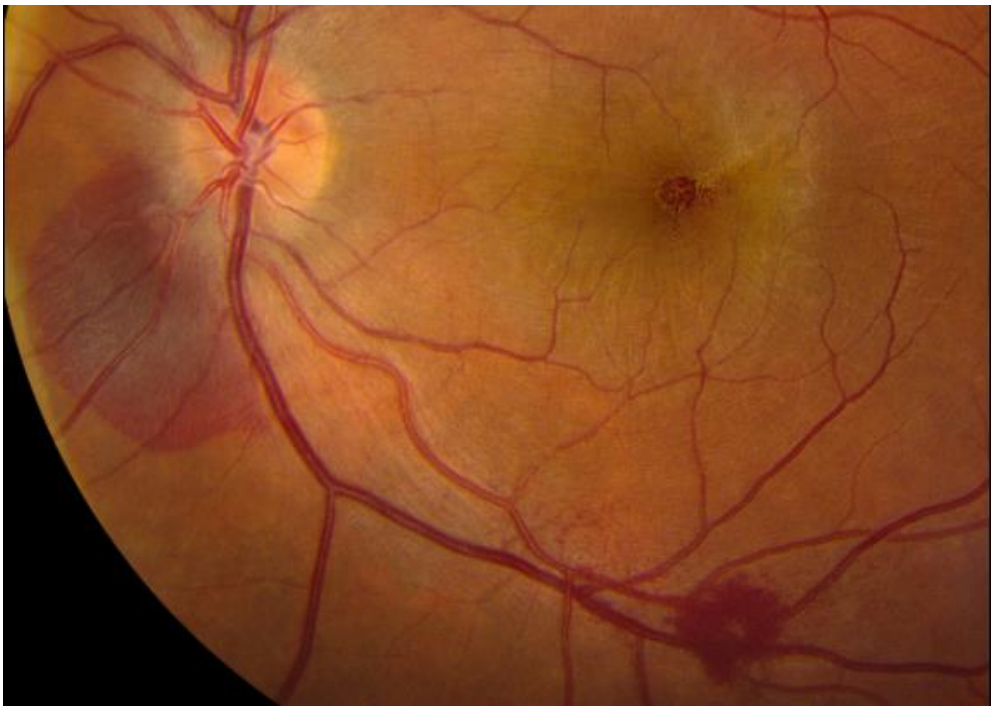


Figure 8. A multilayered hemorrhage is shown with subretinal, intraretinal, and preretinal components. It is associated with a traumatic macular hole.

The damage to the retina and RPE can stimulate cellular migration and proliferation of fibrovascular tissues. Resultant subretinal membranes can lead to separation of the retina from the RPE with poor visual outcome [27]. Animal studies show retinal damage can occur from subretinal hemorrhages as early as day one, suggesting early evacuation of hemorrhages involving the macula may lead to better prognosis [28].

Treatment of retinal hemorrhages range from observation, Nd:YAG membranotomy, to surgical displacement with or without tissue plasminogen (tPA) use [29]. For sub-RPE, intraretinal, and vitreous hemorrhages, typical management is observation as much of the blood will be absorbed over time. It is thought that hemoglobin-blinding protein hemopexin, allows the RPE to internalize the heme-hemopexin complex and clear the sub/intraretinal hemorrhage [30].

Surgical evacuation of vitreous hemorrhage is an option should the hemorrhage not resolve with time or if there are other concurrent retinal problems requiring surgery. For sub-ILM hemorrhage seen in Valsalva retinopathy, a Nd:YAG membranotomy can be used to displace the hemorrhage to the sub-hyloid and vitreous space (Figure 9) [31]. For subretinal bleeds that are at the sub-photoreceptor level or located under the fovea, pneumatic displacement has been shown to be of benefit [32].

Moving the hemorrhage away from the macula center through the use of a bubble and/or subretinal tPA, limits exposure of the fovea to the hemorrhage, giving the best chance for visual recovery.

Also, early displacement of hemorrhage has resulted in improved outcomes [33]. Some case reports suggest that sub-RPE hemorrhage may not be best treated with a surgical pneumatic displacement but rather with observation [34].

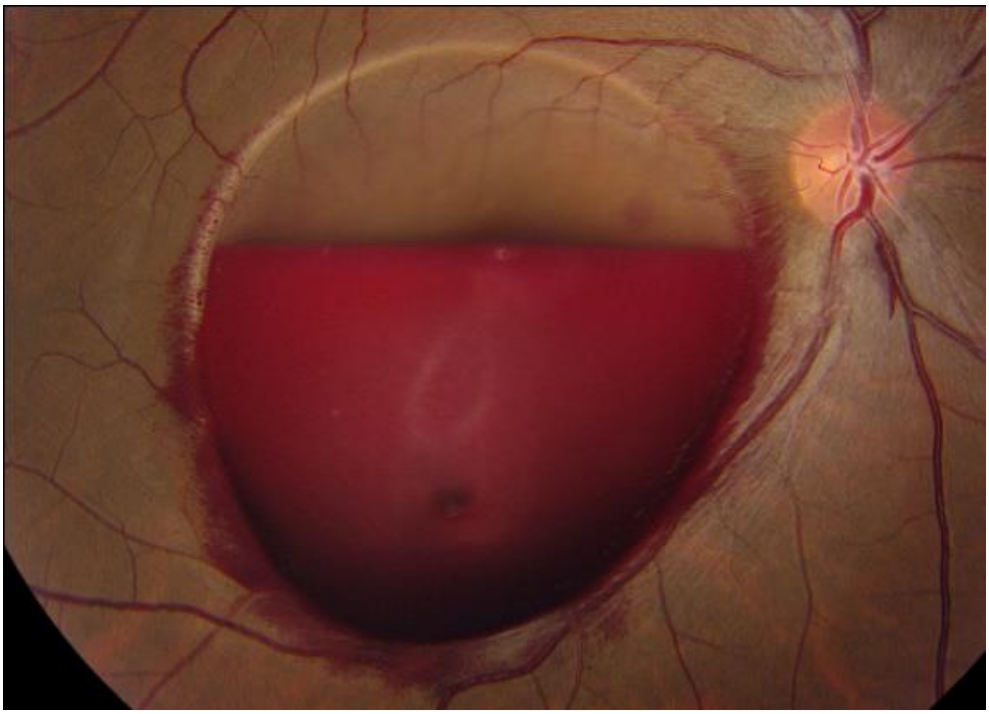


Figure 9. A sub-internal limiting membrane hemorrhage leads to a pre-retinal pocket of blood that may lead to profound vision loss. Vision can be dramatically improved with early intervention with Nd:YAG membranotomy or vitrectomy. Once the macula is re-exposed, the visual prognosis is typically good. Delayed treatment may lead to fibrosis and scarring within the pocket with poor visual recovery.

RETINAL DETACHMENT

The mechanism of a retinal break in the setting of trauma is due to a propagated shockwave. The anterior segment recoils before the posterior segment and the vitreous immediately flows forward. This induces negative pressure in the vitreous which results in a retinal break when the negative pressure exceeds the retinal adhesion strength [35].

Trauma can cause a retinal break at the site of impact or indirectly due to anteroposterior compression immediately followed by elongation; this can cause peripheral tears/dialyses immediately (Figure 10) or delayed due to premature vitreous separation [2].

Demographically, patients tend to be young males [36]. Myopic patients are also at higher risk for traumatic detachments [37]. Traumatic detachments account for 10–20% of retinal detachments [38]. Rhegmatogenous retinal detachment is the most common type of retinal detachment associated with trauma with horseshoe tears, retinal dialyses, and giant retinal tears among the subtypes [39].

Retinal dialysis is the most common type of retinal break in the setting of trauma [40]. It is a disinsertion of the retina at the ora serrata [41]. Traumatic dialyses are typically larger (2.4 clock hours) compared to non-traumatic (1.5 clock hours) [42]. A giant retinal tear (GRT) is a retinal break extending for three or more clock hours in the presence of a posteriorly detached vitreous [43].

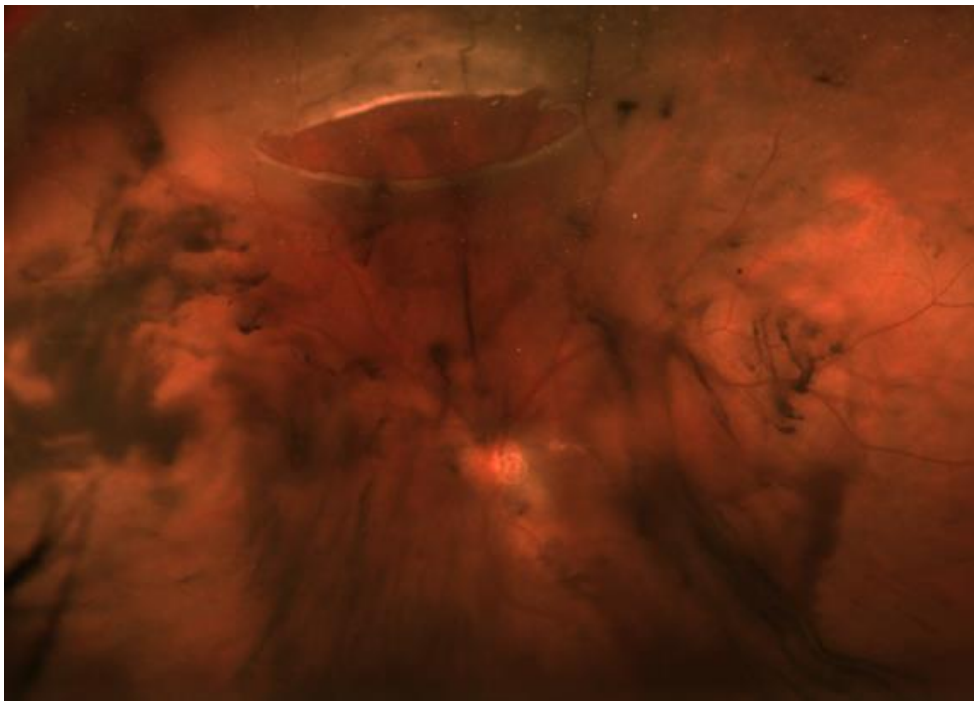


Figure 10. Widefield retinal image of a traumatic retinal tear extending approximately 2 clock hours in the superior peripheral retina. There is also a large vitreous hemorrhage. The mechanism of the injury was blunt force from an elbow during an altercation. The patient developed a retinal detachment requiring pars-plana vitrectomy. After evacuation of the vitreous hemorrhage, multiple tears were revealed, requiring instillation of silicone oil.

Retinal dialyses and GRTs may appear very similar clinically, but the distinguishing characteristic is a posterior vitreous detachment (PVD) associated with GRT [44]. Since there is a PVD in a GRT, the vitreous is adherent to the anterior flap and the posterior flap has a tendency to flip over. In contrast, in a retinal dialysis the posterior flap does not tend to fold over since it is still attached to the vitreous [45].

Traumatic retinal dialyses are typically found in either the inferotemporal or superonasal quadrant. [46] This is contrasted to non-traumatic retinal dialyses which are almost exclusively found inferotemporally [42, 47]. Trauma-associated giant retinal tears also are more commonly located in the inferotemporal and superonasal quadrant [48]. Theories exist for the increased susceptibility of these areas. The temporal peripheral retina is the last to develop embryologically and is the least vascular portion of the retina which may predispose to disinsertion. Also, the temporal retina is the least protected by the orbital bones [41]. The nasal peripheral retina has a relatively narrow vitreous base, which may increase its susceptibility to traumatic dialysis [49].

Traumatic retinal detachments typically have a delayed onset compared with other varieties of detachment [50]. This is attributable to a slow progression of the detachment due to an absence of a posterior vitreous detachment in young patients. Horseshoe and giant retinal tears typically present within 6 weeks of injury [39]. One study showed 41% of traumatic retinal detachments were diagnosed more than a year after the injury [51]. Other studies have shown that only 30-40% of post-traumatic retinal detachments were diagnosed

within one month of injury [40, 51]. This delayed presentation can be further exaggerated by retinal dialysis which has a mean presentation time of eight months after trauma. Dialyses are more likely to be associated with a delayed accumulation of subretinal fluid [39].

In regards to proliferative vitreoretinopathy (PVR), retinal dialysis has a low rate compared to other forms of detachment [52, 53]. This is due to the fact that the vitreous is still attached to the retina and prevents dispersion of the retinal pigment epithelium (RPE) [52]. In contrast, with a GRT, a large area of RPE is released and encourages the development of PVR [54, 55].

When managing a patient with a traumatic retinal detachment, it is important to perform a thorough exam. A study of 196 retinal dialysis patients found that 12% had ocular hypertension and 16% had angle recession [56]. Patients presenting with signs anterior segment trauma such as hyphema require a thorough posterior segment exam.

For the treatment of retinal dialysis, there are multiple options depending on presentation. If there are signs of a chronic detachment, then close observation can be employed [50]. Scleral buckling procedures have produced great results with primary success rates greater than 90% and low rates of proliferative vitreoretinopathy (PVR) [39, 50, 51, 57]. Alternatively, pneumatic retinopexy has been found effective in cases of superior retinal dialysis [58].

The repair of retinal detachments associated with giant retinal tears has vastly improved with the advent of pars plana vitrectomy. The use of scleral buckles, pars plana vitrecomy with silicone oil or gas tamponade, and retinopexy have all been investigated with varying levels of success in both traumatic and nontraumatic giant retinal tears [45]. Currently, in non-traumatic giant retinal tears, most are treated with pars plana vitrectomy without scleral buckle [59]. However, scleral buckles are suggested to counteract any residual vitreous base traction [60], so there may be benefit. Aylward et al published a case series of purely traumatic giant retinal tears treated and found vitrectomy with fluid-silicone oil exchange to have similar results to that of spontaneous giant retinal tears [48]. As mentioned before, giant retinal tears are associated with the highest rates of proliferative vitreoretinopathy of the subtypes of rhegmatogenous retinal detachment which leads to high redetachment rates [61].

A unique challenge in retinal detachment repair is the incarceration of retina in a scleral wound. The preferred management when the retinal incarceration is mild is the placement of a scleral buckle. However, extensive incarceration must be managed by relaxing retinotomy in order to allow the retina to be released from the wound and flattened for re-attachment. It is important to spare as much of the retina as possible while remaining far enough from the scleral wound in order to avoid cutting any granulation tissue. Cutting granulation tissue at the wound may result in severe intraoperative bleeding which may require endodiathermy. Endodiathermy is best avoided as it may result in a staphyloma or weakening of the sclera, leading to instability of the globe [62]. Some authors advocate that outcomes are improved in eyes with retinal incarceration or severe trauma when retinotomy is implemented [63, 64].

TRAUMATIC CHORIORETINAL RUPTURE (RETINITIS SCLOPETARIA)

Retinitis sclopetaria is a simultaneous break in the retina and choroid resulting from a high-velocity missile passing adjacent to but not penetrating the globe [65]. It was described

in 1901 by Goldzieher by the term chorioretinitis plastica sclopetaria [66]. Other names used in the literature for this entity include chorioretinitis proliferans, retinitis proliferans, traumatic proliferative chorioretinitis of lagrange, and acute retinal necrosis [67]. Terms such as chorioretinitis or retinitis incorrectly indicate that the scar may be a result of an inflammatory process, so the names traumatic chorioretinal rupture (TCR) or sclopetaria are more acceptable [65, 67].

TCR is caused by a high-velocity object passing adjacent to the globe which causes rapid deformation of the globe by a shock wave with a subsequent increase in stress on the sclera, choroid, retina, and posterior vitreous cortex (Figure 11). The choroid and retina have been shown histologically to simultaneously rupture and retract at the site of the break revealing bare sclera [68, 69]. Histopathological studies have also shown an intact sclera along with loose and dense fibrous connective tissue with some areas of hyperplastic retinal pigment epithelium [69].

There is a proliferation of fibrous tissue after the initial injury which results in the retina and choroid becoming firmly adherent to the sclera [69]. Ultrasonography has confirmed that despite thickening at the edges of the rupture, there is no separation of the choroidal-retinal complex from the sclera [68]. There have been no reports of choroidal neovascularization in the setting of TCR suggesting that the fibrinous material at the site of the injury may act as a deterrent [67].

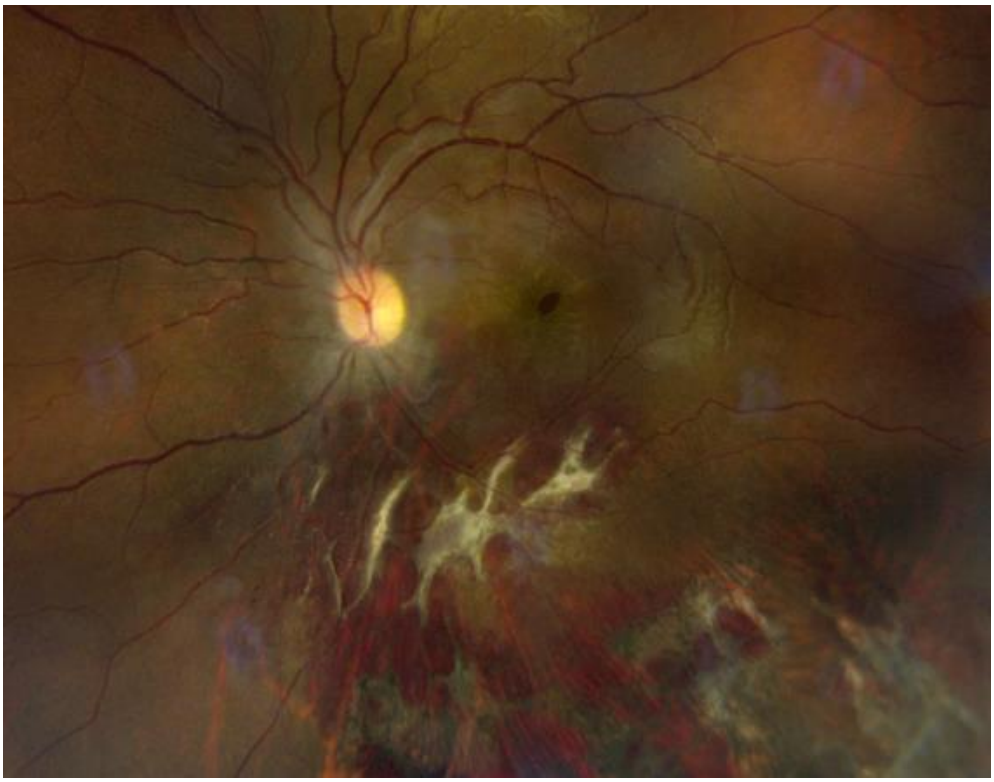


Figure 11. Fundus photograph montage demonstrating traumatic chorioretinal rupture (sclopetaria) in a 19 year-old man one month after a gunshot wound to the left orbit. There is fibrous tissue proliferation and bare sclera inferiorly. A traumatic macular hole is also present.

The rate of deformation due to trauma appears to contribute as well. Experimental studies involving rabbits have shown that rapid rates of deformation inducing detachments between the choroid and sclera, while slow rates of deformation resulted in detachments between the retina and choroid [70].

There is a low incidence of retinal detachment in TCR. Most trauma occurs in younger individuals with an intact posterior hyaloid over the region of the rupture, which prevents liquid vitreous from entering the subretinal space. The retina and choroid remain firmly attached and thus preventing fluid access to the subretinal space [71].

On fundus exam, there is a large retinal and choroidal break with surrounding retinal whitening and hemorrhage. The sclera may also be visible within the break.

Observation is the mainstay of treatment as retinal detachment is uncommon. The largest series of TCR cases was published by Ahmadabadi et al which included 13 eyes of 13 patients [72]. During the follow-up period (mean of 37 months), the retina remained attached in all eyes. All the eyes involved in the study had vitreous hemorrhage and some degree of hyphema. In another study of 8 eyes of 7 patients with TCR, the retina remained attached in all eyes at 6 months [68]. One eye was treated initially with a scleral buckle, which the authors stated was retrospectively unnecessary given the low incidence of retinal detachment. However, late retinal detachment occurred more than one year after the initial injury because of retinal breaks at sites distant from the original TCR, so close observation and thorough peripheral examination is advised [68]. TCR generally carries a poor prognosis. Extension of the damage to the posterior pole either with the initial injury or due to fibrovascular proliferation subsequently developing can cause a loss of macular function [73]. In the case series mentioned earlier, final visual acuity ranged from no light perception to 20/1200 [72].

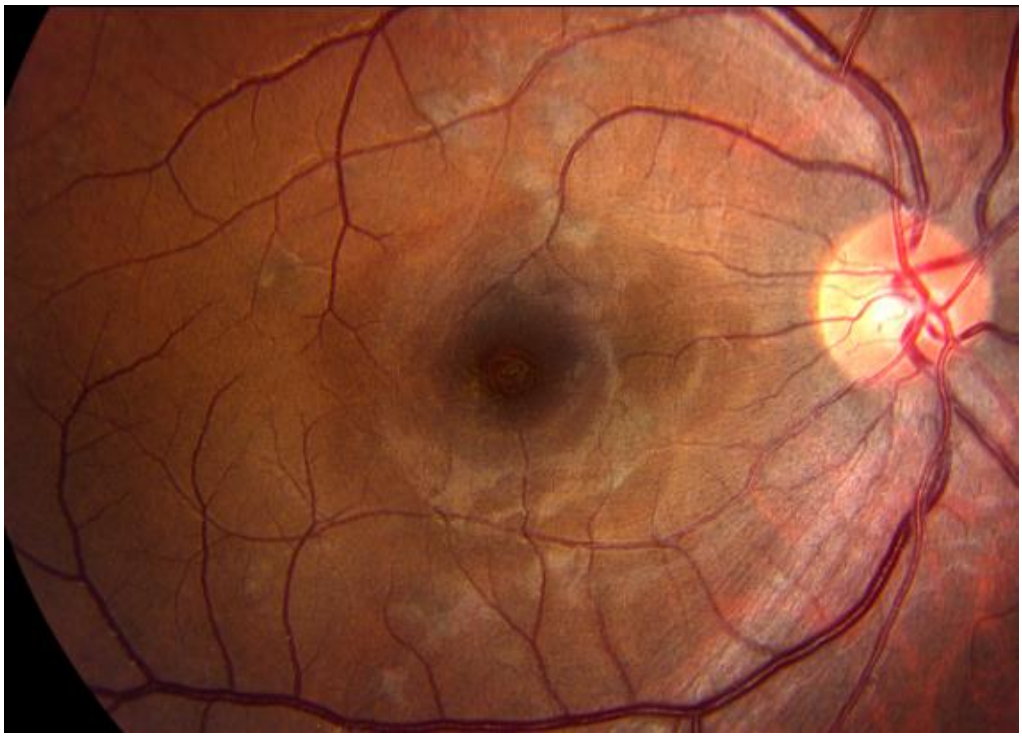
WHIPLASH RETINOPATHY

A whiplash injury occurs when the head is forced backwards due to a posterior pushing force. This is commonly seen after “rear-end” vehicle accidents with an estimated derived annual incidence of 4:1000 in the United States [74]. A whiplash injury may involve the eyes in several ways including the onset of Horner’s syndrome, nerve palsies, and accommodation deficits related to central trauma [75, 76].

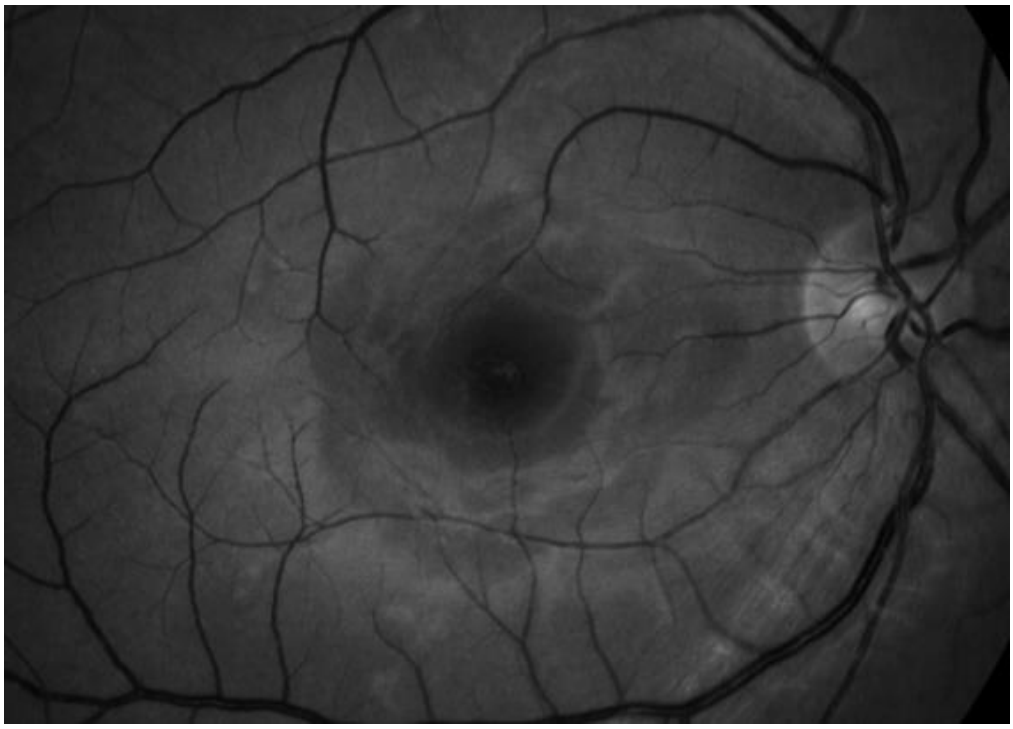
In regards to the retina, a whiplash retinopathy or maculopathy has been described (Figure 12). This typically occurs after a flexion-extension injury without direct eye trauma. The injury results in an acute mild reduction in visual acuity rarely worse than 20/30 in one or usually both eyes. Vision improves to 20/20 within a few days. The peripheral retina becomes thickened and can become opacified. A foveolar depression of approximately 50-100um in diameter is seen and a faint grey haze can be appreciated in the fovea. There may be a slight vitreous detachment.

The retinal opacity and foveal haze disappears over time but leaves a border that becomes more distinct. The foveolar depression persists over the long term. Fluorescein angiogram is usually normal but can show a focal area of hyperfluorescence [77, 78].

The differential diagnosis of whiplash maculopathy includes mild macular retinitis, toxic maculopathies, solar retinopathy, shaken-baby syndrome, and congenital variations of the macula.

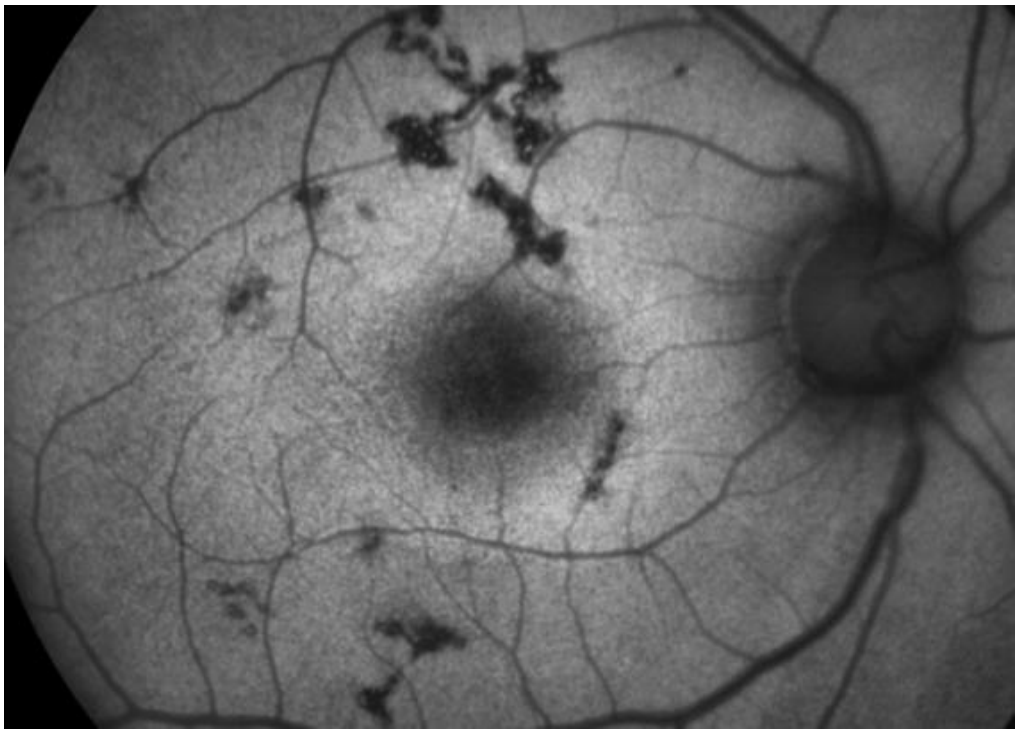


A

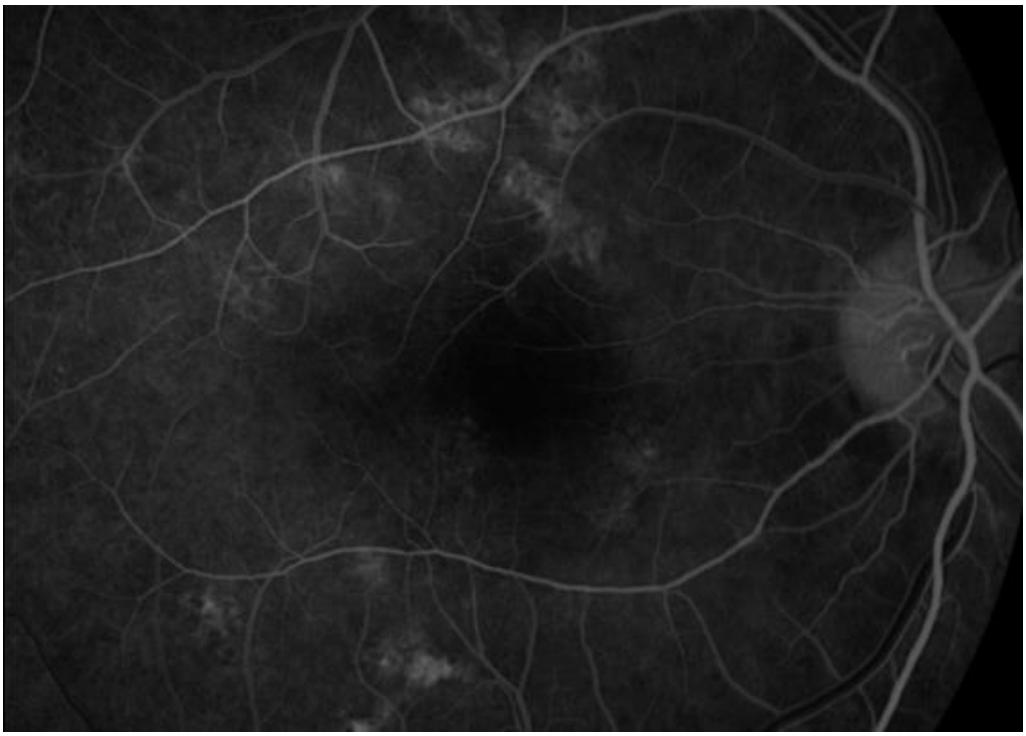


B

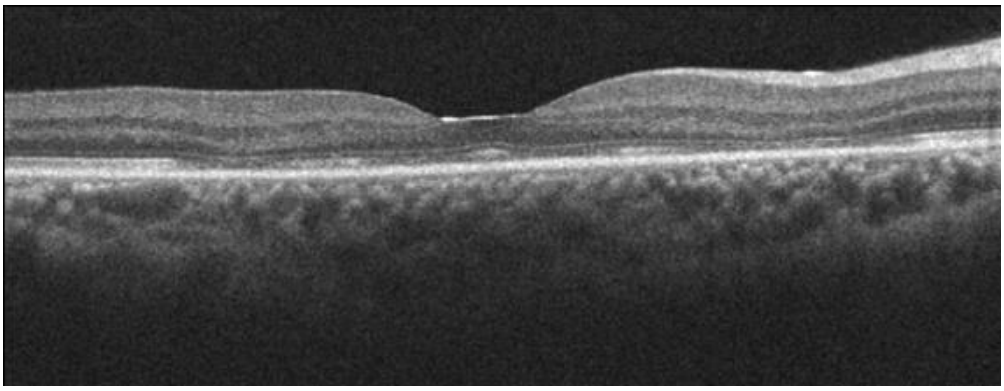
Figure 12. (Continued).



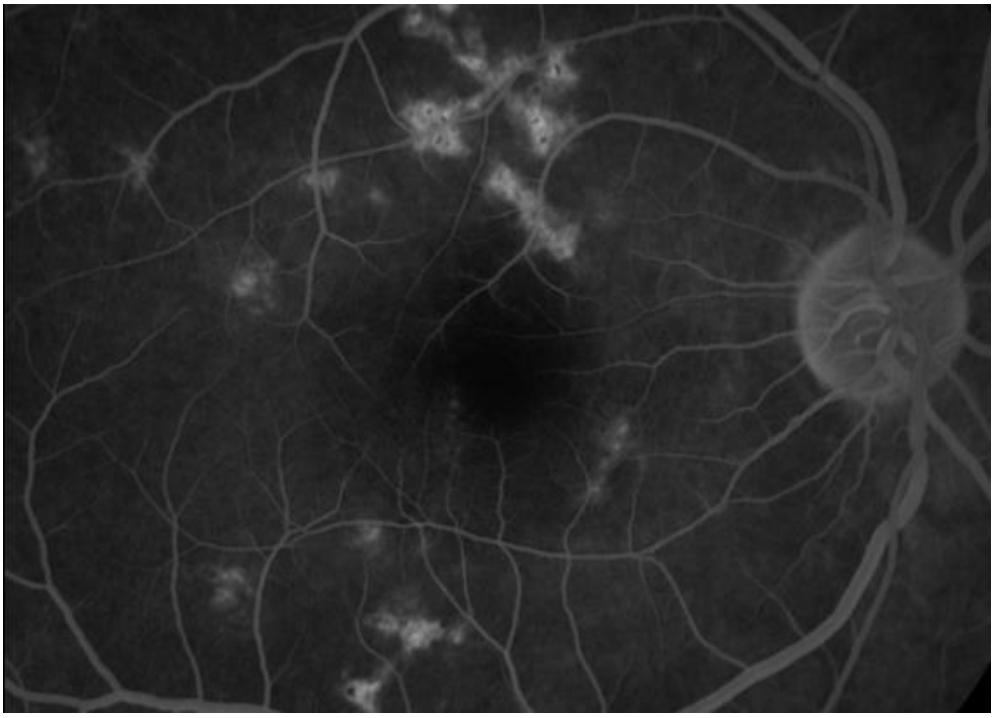
C



D



E



F

Figure 12. Whiplash retinopathy. The early appearance of whiplash retinopathy in color (A), red-free (B), and fundus auto-fluorescence (C). The fovea is slightly opacified in the color and red free images. There is also disruption of the RPE superiorly and inferiorly to the fovea with perifoveal stippling. Fluorescein angiogram shows staining of the disruptions in the RPE in the early laminar venous (D) and late phases (F). Ocular coherence tomography of the macula shows disruptions in the perifoveal The hallmark of whiplash retinopathy is a history of a whiplash injury with mild vision decrease that improves rapidly. Careful slit lamp examination and red-free fundus photography should highlight the foveal depression and haze.

There may be a relationship between the severity of the whiplash injury and ocular involvement. Parons et al. describes a case report where a single whiplash injury resulted in the death of a young patient. They noted optic nerve sheath hemorrhages, posterior pole hemorrhages, retinoschisis, and serous RPE detachments. They postulate that the shearing

stress of the whiplash injury results in vitreous traction, which may cause retinal splitting between the inner and outer segments of the retina as well as the RPE detachments. Some aspects of the severe whiplash injury is similar to shaken baby syndrome where the mechanism is essentially repeated flexion-extension injury [77]. Conversely, isolated retinal hemorrhages may be on the other end of the spectrum, where fine blood vessels are broken without retinal splitting.

The mainstay of treatment of whiplash retinopathy is careful observation. Most patients with whiplash retinopathy will recover within a few days with no residual disability [77, 78]. It can be postulated that with more severe whiplash injuries where retinal hemorrhages result, prognosis can range between good to poor depending on extent of the hemorrhage, time until absorption, and its location.

CONCLUSION

Ocular trauma leads to an array of retinal pathology. Retinal detachments and retinal dialyses require prompt surgical management to prevent loss of vision. Acute submacular or pre-retinal hemorrhages may also have better outcomes with prompt treatment to evacuate the blood. Delayed surgery is indicated in traumatic macular holes if spontaneous closure does not occur. Most other injuries require careful observation for secondary sequelae such as choroidal neovascularization or delayed retinal detachment.

REFERENCES

[1] Miller J. B., Yonekawa Y., Eliott D., Vavvas D. G. A review of traumatic macular hole: diagnosis and treatment. *Int. Ophthalmol. Clin.,* 2013; 53(4):59-67. doi:10.1097/IIO.0b013e3182a26efe

[2] Youssri A. I., Young L. H. Y. Closed-globe contusion injuries of the posterior segment. *Int. Ophthalmol. Clin.,* 2002; 42(3):79-86.

[3] Kuhn F., Morris R., Witherspoon C. D., Mann L. Epidemiology of blinding trauma in the United States Eye Injury Registry. *Ophthalmic Epidemiol.,* 2006; 13(3):209-216. doi:10.1080/09286580600665886

[4] Huang J., Liu X., Wu Z., et al. Classification of full-thickness traumatic macular holes by optical coherence tomography. *Retina Phila Pa.,* 2009; 29(3):340-348. doi:10.1097/IAE.0b013e31819241d0

[5] Yamada H., Sakai A., Yamada E., Nishimura T., Matsumura M. Spontaneous closure of traumatic macular hole. *Am. J. Ophthalmol.,* 2002; 134(3):340-347.

[6] Yamashita T., Uemara A., Uchino E., Doi N., Ohba N. Spontaneous closure of traumatic macular hole. *Am. J. Ophthalmol.,* 2002; 133(2):230-235.

[7] Hirata A., Tanihara H. Ruptured internal limiting membrane associated with blunt trauma revealed by indocyanine green staining. *Graefes Arch. Clin. Exp. Ophthalmol. Albrecht Von Graefes Arch. Für Klin. Exp. Ophthalmol.,* 2004; 242(6):527-530. doi:10.1007/s00417-004-0875-1

[8] Johnson R. N., McDonald H. R., Lewis H., et al. Traumatic macular hole: observations, pathogenesis, and results of vitrectomy surgery. *Ophthalmology*, 2001; 108(5):853-857.
[9] Rubin J. S., Glaser B. M., Thompson J. T., Sjaarda R. N., Pappas S. S., Murphy R. P. Vitrectomy, fluid-gas exchange and transforming growth factor--beta-2 for the treatment of traumatic macular holes. *Ophthalmology*, 1995; 102(12):1840-1845.
[10] García-Arumí J., Corcostegui B., Cavero L., Sararols L. The role of vitreoretinal surgery in the treatment of posttraumatic macular hole. *Retina Phila Pa.*, 1997; 17(5):372-377.
[11] Amari F., Ogino N., Matsumura M., Negi A., Yoshimura N. Vitreous surgery for traumatic macular holes. *Retina Phila Pa*., 1999; 19(5):410-413.
[12] Chow D. R., Williams G. A., Trese M. T., Margherio R. R., Ruby A. J., Ferrone P. J. Successful closure of traumatic macular holes. *Retina Phila Pa.*, 1999; 19(5):405-409.
[13] Christensen U. C. Value of internal limiting membrane peeling in surgery for idiopathic macular hole and the correlation between function and retinal morphology. *Acta Ophthalmol.*, (Copenh.). 2009; 87 Thesis 2:1-23. doi:10.1111/j.1755-3768.2009. 01777.x
[14] Aguilar J. P., Green W. R. Choroidal rupture. A histopathologic study of 47 cases. *Retina Phila Pa.*, 1984; 4(4):269-275.
[15] Chee S.-P., Jap A., Cheung C. M. G. The prognostic value of angiography in Vogt-Koyanagi-Harada disease. *Am. J. Ophthalmol.*, 2010; 150(6):888-893. doi:10.1016/ j.ajo.2010.06.029
[16] Secrétan M., Sickenberg M., Zografos L., Piguet B. Morphometric characteristics of traumatic choroidal ruptures associated with neovascularization. *Retina Phila Pa.*, 1998; 18(1):62-66.
[17] Chanana B., Azad R. V., Kumar N. Intravitreal bevacizumab for subfoveal choroidal neovascularization secondary to traumatic choroidal rupture. *Eye Lond. Engl.*, 2009; 23(11):2125-2126. doi:10.1038/ eye.2008.434
[18] Gass J. D. Pathogenesis of tears of the retinal pigment epithelium. *Br. J. Ophthalmol.*, 1984; 68(8):513-519.
[19] Krishan N. R., Chandra S. R., Stevens T. S. Diagnosis and pathogenesis of retinal pigment epithelial tears. *Am. J. Ophthalmol.*, 1985; 100(5): 698-707.
[20] Levin L. A., Seddon J. M., Topping T. Retinal pigment epithelial tears associated with trauma. *Am. J. Ophthalmol*., 1991; 112(4):396-400.
[21] Polito A., Cereda M., Romanelli F., Pertile G. Macular translocation with 360 degrees retinotomy for management of retinal pigment epithelial tear: long-term results. *Br. J. Ophthalmol.*, 2011; 95(1):74-78. doi:10.1136/bjo.2009.170381
[22] Lam A., Benson W. E., Jeffers J. B. Blunt trauma. In: Duane's Clinical Ophthalmology. Vol 3. 2006 Edition. Lippincott Williams & Wilkins.
[23] Williams D. F., Mieler W. F., Williams G. A. Posterior segment manifestations of ocular trauma. *Retina Phila Pa.*, 1990; 10 Suppl. 1:S35-S44.
[24] Pulido J. S., Blair N. P. The blood-retinal barrier in Berlin's edema. *Retina Phila Pa.*, 1987; 7(4):233-236.
[25] He X., Hahn P., Iacovelli J., et al. Iron homeostasis and toxicity in retinal degeneration. *Prog. Retin. Eye Res.*, 2007; 26(6):649-673. doi:10.1016/j.preteyeres.2007.07.004
[26] Doly M., Bonhomme B., Vennat J. C. Experimental study of the retinal toxicity of hemoglobinic iron. *Ophthalmic. Res*., 1986; 18(1):21-27.

[27] Gillies A., Lahav M. Absorption of retinal and subretinal hemorrhages. *Ann. Ophthalmol.*, 1983; 15(11):1068-1074.

[28] Glatt H., Machemer R. Experimental subretinal hemorrhage in rabbits. *Am. J. Ophthalmol.*, 1982; 94(6):762-773.

[29] Tennant M. T. S., Borrillo J. L., Regillo C. D. Management of submacular hemorrhage. *Ophthalmol. Clin. N. Am.*, 2002; 15(4):445-452, vi

[30] Hunt R. C., Hunt D. M., Gaur N., Smith A. Hemopexin in the human retina: protection of the retina against heme-mediated toxicity. *J. Cell Physiol.*, 1996; 168(1):71-80. doi:10.1002/(SICI)1097-4652(199607)168:1<71::AID-JCP9>3.0.CO;2-5

[31] Kuruvilla O., Munie M., Shah M., Desai U., Miller J. A., Ober M. D. Nd:YAG membranotomy for preretinal hemorrhage secondary to valsalva retinopathy. *Saudi J. Ophthalmol. Off. J. Saudi Ophthalmol. Soc.*, 2014; 28(2):145-151. doi:10.1016/ j.sjopt.2014.02.006

[32] Goldman D. R., Vora R. A., Reichel E. Traumatic choroidal rupture with submacular hemorrhage treated with pneumatic displacement. *Retina Phila Pa.*, 2014; 34(6):1258-1260. doi:10.1097/ IAE.0000000000000019

[33] Sobolewska B., Utebey E., Bartz-Schmidt K. U., Tatar O. Long-term visual outcome and its predictive factors following treatment of acute submacular hemorrhage with intravitreous injection of tissue plasminogen factor and gas. *J. Ocul. Pharmacol. Ther. Off. J. Assoc. Ocul. Pharmacol. Ther.*, 2014; 30(7):567-572. doi:10.1089/ jop. 2013.0135.

[34] Sampangi R., Chandrakumar H. V., Somashekar S. E., Joshi G. R., Ganesh S. SD-OCT to differentiate traumatic submacular hemorrhage types using automatic three-dimensional segmentation analysis. *Ophthalmic Surg. Lasers Imaging Off. J. Int. Soc. Imaging Eye*, 2011;42 Online:e32-e36. doi:10.3928/15428877-20110224-04

[35] Liu X., Wang L., Wang C., Sun G., Liu S., Fan Y. Mechanism of traumatic retinal detachment in blunt impact: a finite element study. *J. Biomech.*, 2013; 46(7):1321-1327. doi:10.1016/j.jbiomech.2013.02.006

[36] Vote B. J., Casswell A. G. Retinal dialysis: are we missing diagnostic opportunities? *Eye Lond. Engl.*, 2004; 18(7):709-713. doi:10.1038/ sj.eye.6701310

[37] Goffstein R., Burton T. C. Differentiating traumatic from nontraumatic retinal detachment. *Ophthalmology*, 1982; 89(4):361-368.

[38] Hilton G. F., McLean E. B., Brinton D. A. Retinal Detachment: Principles and Practice. Vol. 2nd ed. San Francisco, CA: American Academy of Ophthalmology; 1995.

[39] Johnston P. B. Traumatic retinal detachment. *Br. J. Ophthalmol.*, 1991; 75(1):18-21.

[40] Cox M. S. Retinal breaks caused by blunt nonperforating trauma at the point of impact. *Trans. Am. Ophthalmol. Soc.*, 1980; 78:414-466.

[41] Smiddy W. E., Green W. R. Retinal dialysis: pathology and pathogenesis. *Retina Phila Pa.*, 1982; 2(2):94-116.

[42] Hollander D. A., Irvine A. R., Poothullil A. M., Bhisitkul R. B. Distinguishing features of nontraumatic and traumatic retinal dialyses. *Retina Phila Pa.*, 2004; 24(5):669-675.

[43] Schepens C. L., Freeman H. M. Current management of giant retinal breaks. *Trans. - Am. Acad. Ophthalmol. Otolaryngol. Am. Acad. Ophthalmol. Otolaryngol.*, 1967; 71(3):474-487.

[44] Shunmugam M., Ang G. S., Lois N. Giant retinal tears. *Surv. Ophthalmol.*, 2014; 59(2):192-216. doi:10.1016/ j.survophthal.2013.03.006

[45] Gopal L., Sharma T., Lois N. Giant retinal tears. In: Retina. Vol. 3. 5th ed. London: Saunders/Elsevier; :852.
[46] Delori F., Pomerantzeff O., Cox M. S. Deformation of the globe under high-speed impact: it relation to contusion injuries. *Invest. Ophthalmol.,* 1969; 8(3):290-301.
[47] Hagler W. S., North A. W. Retinal dialyses and retinal detachment. *Arch. Ophthalmol. Chic. Ill,* 1960. 1968;79(4):376-388.
[48] Aylward G. W., Cooling R. J., Leaver P. K. Trauma-induced retinal detachment associated with giant retinal tears. *Retina Phila Pa.,* 1993; 13(2):136-141.
[49] Weidenthal D. T., Schepens C. L. Peripheral fundus changes associated with ocular contusion. *Am. J. Ophthalmol.,* 1966; 62(3):465-477.
[50] Kennedy C. J., Parker C. E., McAllister I. L. Retinal detachment caused by retinal dialysis. *Aust. N Z J. Ophthalmol.,* 1997; 25(1):25-30.
[51] Ross W. H. Traumatic retinal dialyses. *Arch. Ophthalmol. Chic. Ill,* 1960. 1981; 99(8):1371-1374.
[52] Qiang Kwong T., Shunmugam M., Williamson T. H. Characteristics of rhegmatogenous retinal detachments secondary to retinal dialyses. *Can. J. Ophthalmol. J. Can. Ophtalmol.,* 2014; 49(2):196-199. doi:10.1016/ j.jcjo.2013.12.013
[53] Mitry D., Singh J., Yorston D., et al. The predisposing pathology and clinical characteristics in the Scottish retinal detachment study. *Ophthalmology,* 2011; 118(7):1429-1434. doi:10.1016/ j.ophtha.2010.11.031
[54] Leaver P. K., Lean J. S. Management of giant retinal tears using vitrectomy and silicone oil/fluid exchange. A preliminary report. *Trans. Ophthalmol. Soc. U K,* 1981; 101(1):189-191.
[55] Yoshino Y., Ideta H., Nagasaki H., Uemura A. Comparative study of clinical factors predisposing patients to proliferative vitreoretinopathy. *Retina Phila Pa.,* 1989; 9(2):97-100.
[56] Zion V. M., Burton T. C. Retinal dialysis. *Arch. Ophthalmol. Chic. Ill 1960,* 1980; 98(11):1971-1974.
[57] Bonnet M., Moyenin P., Pecoldowa C., Grange J. D. [Retinal detachment caused by a tear at the ora serrata]. *J. Fr. Ophtalmol.,* 1986; 9(3):231-242.
[58] Melgen S. E., Michels M. Pneumatic retinopexy for the treatment of giant retinal dialyses. *Am. J. Ophthalmol.,* 1994; 118(6):762-765.
[59] Ang G. S., Townend J., Lois N. Epidemiology of giant retinal tears in the United Kingdom: the British Giant Retinal Tear Epidemiology Eye Study (BGEES). *Invest. Ophthalmol. Vis. Sci.,* 2010; 51(9):4781-4787. doi:10.1167/iovs.09-5036
[60] Ie D., Glaser B. M., Sjaarda R. N., Thompson J. T., Steinberg L. E., Gordon L. W. The use of perfluoro-octane in the management of giant retinal tears without proliferative vitreoretinopathy. *Retina Phila Pa.,* 1994; 14(4):323-328.
[61] Malbran E., Dodds R. A., Hulsbus R., Charles D. E., Buonsanti J. L., Adrogué E. Retinal break type and proliferative vitreoretinopathy in nontraumatic retinal detachment. *Graefes Arch. Clin. Exp. Ophthalmol. Albrecht Von Graefes Arch. Für Klin. Exp. Ophthalmol.,* 1990; 228(5):423-425.
[62] Banaee T. Retinotomy/Retinectomy. *In: Vitrectomy. Vol. InTech.;* 2012: 39-54.
[63] Zhang M.-N., Jiang C.-H. 360-degree retinectomy for severe ocular rupture. *Chin. J. Traumatol. Zhonghua Chuang Shang Za Zhi Chin. Med. Assoc.,* 2005; 8(6):323-327.

[64] Zhang S., Gao R., Zhu M. [Surgical treatment of traumatic retinal detachment with incarceration]. *Zhonghua Yan Ke Za Zhi Chin. J. Ophthalmol.*, 1999;35(4):290-292.

[65] Chen Y., Zhao M., Zhou P. Traumatic Chorioretinopathies. In: Retina. Vol. 2. 5th ed. London: Saunders/Elsevier; :1564-1570.

[66] Goldzier W. A study of the pathology of orbital gunshot traumas. *Z. Augenheikd.*, 1901; (6):277.

[67] Papakostas T. D., Yonekawa Y., Skondra D., Vavvas D. G. Traumatic chorioretinal rupture (sclopetaria). *Int. Ophthalmol. Clin.*, 2013; 53(4): 119-125. doi:10. 1097/IIO.0b013e3182a26f18

[68] Martin D. F., Awh C. C., McCuen B. W., Jaffe G. J., Slott J. H., Machemer R. Treatment and pathogenesis of traumatic chorioretinal rupture (sclopetaria). *Am. J. Ophthalmol.*, 1994; 117(2):190-200.

[69] Dubovy S. R., Guyton D. L., Green W. R. Clinicopathologic correlation of chorioretinitis sclopetaria. *Retina Phila Pa.*, 1997; 17(6):510-520.

[70] DeGuillebon H., Zauberman H. Experimental retinal detachment. Biophysical aspects of retinal peeling and stretching. *Arch. Ophthalmol. Chic. Ill 1960*, 1972; 87(5):545-548.

[71] Fraser E. J., Haug S. J., McDonald H. R. Clinical presentation of chorioretinitis sclopetaria. *Retin. Cases Brief Rep*., 2014; 8(4):257-259. doi:10.1097/ ICB. 000000000 0000095

[72] Ahmadabadi M. N., Karkhaneh R., Roohipoor R., Tabatabai A., Alimardani A. Clinical presentation and outcome of chorioretinitis sclopetaria: a case series study. *Injury*, 2010; 41(1):82-85. doi:10.1016/ j.injury.2009.02.016

[73] Agarwal A., Gass J. D. M. Gass' Atlas of Macular Diseases. Vol. 5th ed. Edingburgh: Elsevier, Saunders; 2012.

[74] Barnsley L., Lord S., Bogduk N. Whiplash injury. *Pain*, 1994; 58(3): 283-307.

[75] Duke-Elder S. Mechanical Injuries System of Ophthalmology. Vol. XVI. London, UK: Henry Kimpton; 1972.

[76] Burke J. P., Orton H. P., West J., Strachan I. M., Hockey M. S., Ferguson D. G. Whiplash and its effect on the visual system. *Graefes Arch. Clin. Exp. Ophthalmol. Albrecht Von Graefes Arch. Für Klin. Exp. Ophthalmol*., 1992; 230(4):335-339.

[77] Parsons M. A., Talbot J. F., Mudhar H. S., Rutty G. N. The pathology of whiplash maculopathy and retinopathy: Illustrated observations derived from a fatal roller-coaster accident. *Forensic Sci. Med. Pathol.*, 2005; 1(1):19-25. doi:10.1385/ FSMP:1:1:019.

[78] Kelley J. S., Hoover R. E., George T. Whiplash maculopathy. *Arch. Ophthalmol. Chic. Ill 1960*, 1978; 96(5):834-835.