Science Publications

Global Journal of Medical Case Reports, 2022, 2, 483
www.scipublications.org/journal/index.php/gjmcr
DOI: 10.31586/gjmcr.2022.483

Case Report

Traumatic Hyphaema: A Case Report

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Abstract: Hyphaema is the presence of blood in the anterior chamber (AC) of the eye and is commonly caused by a blunt ocular trauma. In this case report, we discuss a case of a 29-years-old African man presented to the emergency department with severe right ocular pain and blurry vision following a blunt injury. This is a teaching case report aimed to discuss the management options, potential complications and visual prognosis of this particular case.

Keywords: Traumatic hyphaema, Intraocular pressure, Sickle cell haemoglobinopathies

1. Introduction

Traumatic hyphaema is a build-up of red blood cells (RBCs) in the anterior chamber (AC), the space between the cornea and the iris, due to blunt or penetrating trauma[1], with blunt trauma being the most common cause of hyphaema of all types.[2] In this case report, we discuss a case of a 29 years old African male with a medical history of sickle cell trait (SCT) who presented with right eye traumatic hyphaema.

2. Case Report

A 29-years-old male of African origin, with a medical history of SCT, presented to A&E complaining of pain and blurry vision in his right eye after he was hit by a squash ball. Upon presentation, unaided visual acuity (VA) was 6/12 in the right eye (OD) and 6/4 in the left eye (OS). The best corrected visual acuity (BCVA) was 6/6 OD and 6/4 OS. There was no relative afferent pupillary defect (RAPD) and Ishihara was 17/17 bilaterally. On inspection there were bruised eyelids and conjunctival injection OD. Slit lamp examination revealed clear corneas bilaterally. The AC the left eye was clear, well-formed and quiet. The right AC revealed +RBCs and a hyphaema, occupying <33% of AC (Figure 1). Goldman tonometry revealed pressures of 46 mmHg OD and 16 mmHg OS. No clear view of the right fundus was observed. Fundus examination OS was normal. The patient was investigated for high IOP OD and was diagnosed with traumatic hyphaema OD. He was treated as an emergency case aiming to lower IOP medically by administrating atropinise and topical steroid with consideration of AC washout. A follow-up appointment was arranged after 2 hours.

How to cite this paper:
Solima, N. (2022). Traumatic Hyphaema: A Case Report. *Global Journal of Medical Case Reports*, 2(1), 25–31. Retrieved from https://www.scipublications.com/journal/index.php/gjmcr/article/view/483

Received: September 16, 2022
Accepted: October 25, 2022
Published: October 28, 2022

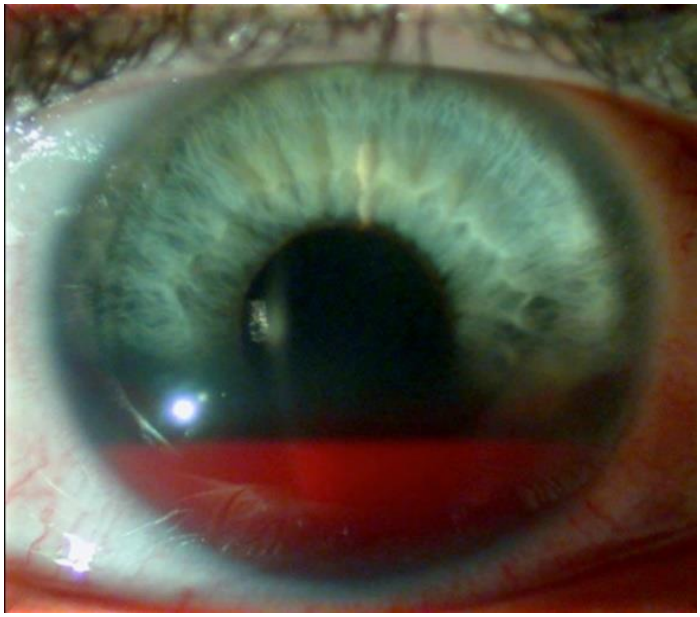

Figure 1. Colour photography of the right eye showing a hyphaema, which occupied less than 33% of the anterior chamber.

3. Epidemiology

The incidence of traumatic hyphaema is 12 per 100,000, with 70% happening in children.[3] Most commonly it occurs in males aged 10-20 years old and often happen due to recreational or sport injuries.[4] One study reported that sport injuries account for 60% of traumatic hyphaema cases.[1]

4. Pathophysiology

Traumatic hyphaema may be caused by blunt or penetrating trauma.[2] Hyphaema due to blunt trauma is frequently caused by tears of the major vessels of the iris or ciliary body.[2] Blunt force triggers antero-posterior compression of the closed globe, resulting in immediate equatorial expansion which generates a shearing force along the iris and ciliary body (Figure 2[5]).[6]

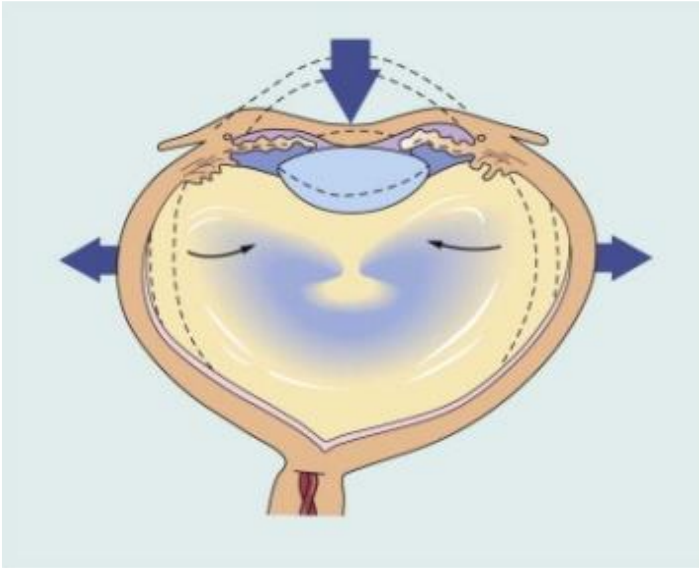

Figure 2. The effect of blunt trauma on the eye.[5]

Seven rings injury patterns have been described due to blunt trauma to the eye (Figure 3), including retinal dialysis, zonular dehiscence, trabecular meshwork tear, cyclodialysis, angle recession, iridodialysis and radial tears of the pupillary sphincter.[7]

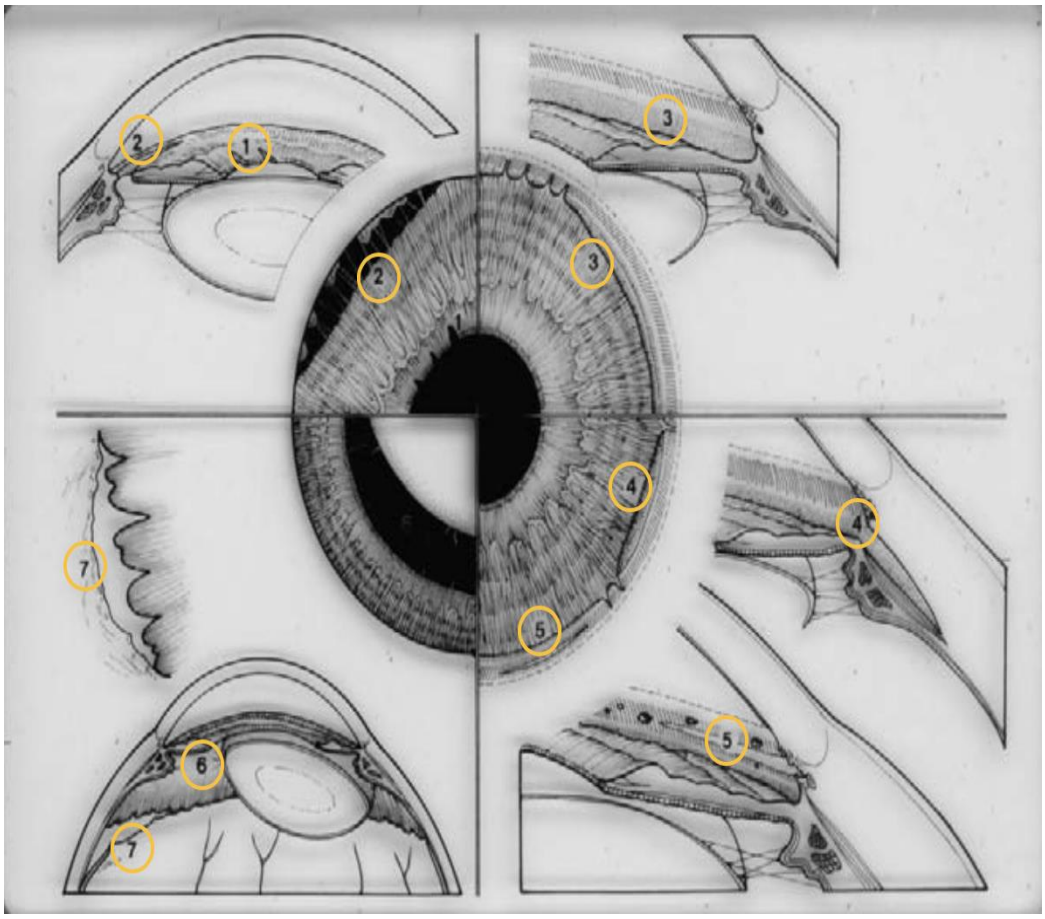

Figure 3. The seven rings in blunt eye trauma: (1) sphincter tear, (2) iridodialysis, (3) angle recession, (4) cyclodialysis, (5) TMW tear, (6) zonular tears and (7) retinal dialysis.[7]

Blunt ocular trauma may also cause chorioretinal injury, cataract, traumatic iritis, traumatic uveitis and commotion retinae to name a few.[5] Hyphaema due to penetrating injury can also be linked with direct blood vessels injury and low IOP.[6] In patients with sickle cell haemoglobinopathies, the RBCs become sickled in the aqueous humour, causing passing via trabecular meshwork (TMW) challenging,[8] leading to constant IOP elevation even with small hyphaemas.[6]

5. Complications

Traumatic hyphaema can result in elevated IOP for various reasons.[6] It may occur acutely due to TMW blockage due to RBCs, inflammatory cells or clot debris or because of pupillary block due to a clot affecting both AC and PC.[6] In eyes with a history of traumatic hyphaema, the late-onset glaucoma incidence is 0–20%.[6, 9, 10] Blunt trauma with a significant IOP elevation or optic nerve contusion may lead to optic nerve damage and, in turn, permanent worsening of vision.[6] Corneal bloodstaining is another complication, with an incidence of 2-11% following traumatic hyphaema.[6] Re-bleeding (secondary haemorrhage) is seen in approximately 30% of cases[1] and may increase the risk of permanent visual loss.[4] Other complications of traumatic hyphaema include peripheral anterior synechiae (PAS), and accommodative impairment.[6]

Patients with sickle cell disease (SCD) or SCT, like our patient, tend to have high IOPs and respond poorly to medical treatment. They also have a higher risk of secondary haemorrhages and, even with moderate IOP rise, they are more likely to develop glaucomatous nerve damage.[11] RVO has been also reported in patients with SCD/SCT as a complication of traumatic hyphaema and glaucoma.[12, 13]

6. History and Clinical Examination

History must consist of questions related to the trauma itself including the mechanism of injury, eye pain, change in vision, photophobia, vomiting, nausea and any history of bleeding disorders especially SCT/SCD.[4] Our patient was originally from Sierra Leone, a country in West Africa with a medical history of SCT.[14] This case emphasises the necessity to obtain a thorough history of ethnic origin and blood disorders from all patients with hyphaemas. A thorough evaluation can be undertaken when ruptured globe[4] and orbital fractures[15] have been eliminated.[4] Initially, the lashes, eyelids, lacrimal apparatus and cornea clarity should be inspected. Later, VA, direct and indirect pupillary responses, RAPD, confrontational visual fields and extra-ocular muscles should be evaluated.[4] Ishihara chart is performed to test optic nerve function.

A full thickness laceration should be ruled out by evaluating the cornea carefully using Seidel test.[15] Followed by measuring the IOP.[4] Slit lamp examination on the initial visit should document corneal clarity and the cornea should be closely assessed for corneal bloodstaining. Moreover, the vertical height of the hyphaema, should be monitored closely for subsequent increase in size, which is an indication of recurrent haemorrhage.[15] The AC should also be carefully evaluated to recognise the presence of, and differentiate between, RBCs, white blood cells (WBCs) and ghost cells.[15] Dilated fundoscopy examination, without scleral indentation, is crucial to evaluate the posterior segment (PS) for complications related to the initial trauma.[15]

Ultrasonography (B-scan) can help in viewing the PS when there is no fundus view initially, and will help to confirm the presence of vitreous haemorrhage, retinal treads, retinal detachment,[5, 16] lens dislocation and intraocular foreign bodies.[4] High-frequency ultrasound biomicroscopy (UBM) may be beneficial in identifying regions of weak zonules cyclodialysis and angle recession.[17] Gonioscopy can be performed six weeks following the trauma to evaluate angle recession. It must not be performed prior this time as this technique can increase the risk of re-bleeding.[7]

Various grading systems are used to grade hyphaemas. They can be graded based on the amount of blood in the AC. Grade 0 (microhyphaema) occurs with suspended RBCs in the AC with no layered clot, grade I hyphaema has < 33% filling of the AC, grade II has 33%-50% filling, grade III >50% but < total AC filling and grade IV has 100% AC filling (Figure 4[18]).[11]

Following initial investigations, a full blood count (FBC) should be requested. Furthermore, a coagulation profile should be ordered, especially for those with a known history of bleeding diathesis or who are on anticoagulants.[4] Patients should be tested for sickle cell haemoglobinopathy if they have a family history of SCT or SCD or if their status is uncertain.[4, 5]

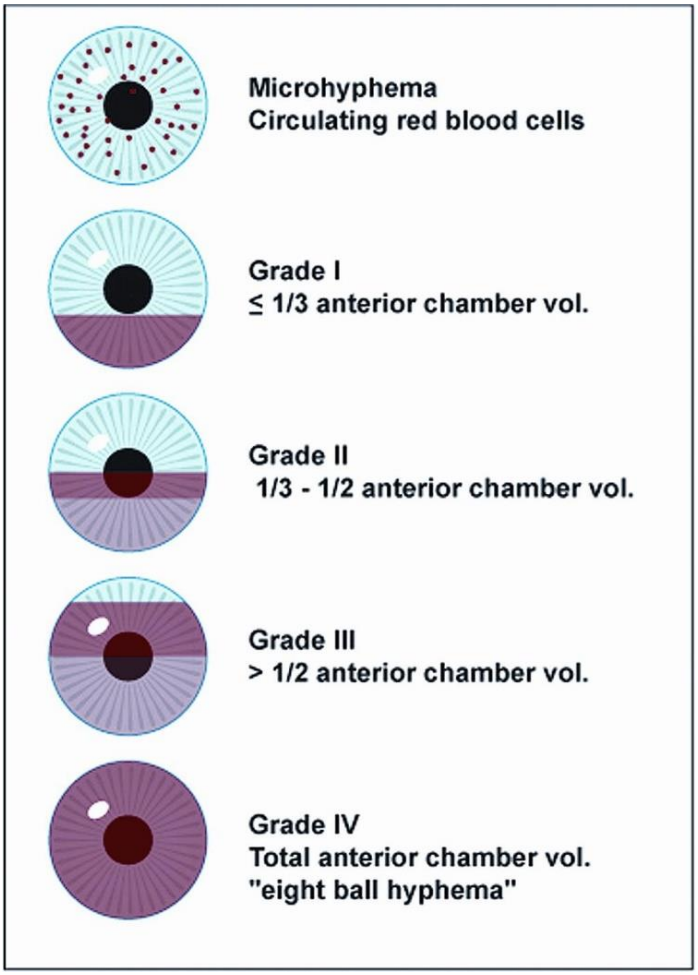

Figure 4. Hyphaema grading scale.[18]

7. Differential Diagnosis (DDX)

Traumatic hyphaema due to blunt trauma. Other DDX includes spontaneous hyphaema, especially for those who have an underlying health condition such as SCD.[4] Hyphaema secondary to intraocular operation or laser.[4] Inflammatory or infectious conditions such as herpetic uveitis. Tumours and neovascularisation.[4, 6]

8. Management

Medical management of traumatic hyphaema consists of eye protection with a protective shield, rest, elevating the head at least 30 degrees, and avoidance of aspirin, non-steroidal anti-inflammatory agents (NSAIDs) and anticoagulants.[4] To enable a full examination and for pain control, topical analgesics such as topical proparacaine can be used.[1] Nausea can be managed by intravenous ondansetron.[4] The use of antifibrinolytic agents for example tranexamic acid for traumatic hyphaema is debatable; they have potential for lowering the rate of re-bleeding but have numerous side effects.[1] Pilocarpine should be avoided in these patients as it constricts the pupil and fundus examination will be further impaired.[6]

Topical cycloplegics such as cyclopentolate can be used once intraocular hypertension and acute glaucoma have been ruled out.[4] They will paralyse the iris muscle and ciliary body resulting in dilatation of the pupil, consequently reducing pain and preventing the formation of posterior synechia.[4, 6] Corticosteroids will help to

reduce inflammation by inhibiting fibrinolysis and stabilising the blood-ocular barrier.[6] Both corticosteroids and cycloplegics can lower re-bleeding risk.[6] IF IOP is >25 mmHg, the first-line treatment is usually topical beta blockers and carbonic anhydrase inhibitors (CAIs).[7] Examples of topical beta-blockers include beta-adrenergic antagonists (such as timolol) and topical alpha-2 agonists (such as apraclonidine).[1] Examples of CAIs include topical dorzolamide, systemic methazolamide and acetazolamide.[11] Oral CAIs can be used if topical CAIs are not effective in managing IOP.[8] Even though oral CAIs are efficient in reducing IOP, they have various side effects.[8] In case of uncontrolled IOP, intravenous mannitol can be utilised. [8] In patients with sickle cell haemoglobinopathies, such as our patient, the options for medical treatment are limited. The high IOP can be managed medically by beta-adrenergic antagonists or alpha-2 adrenergic agonists.[6] Topical CAIs can be added but should be used cautiously as they can reduce the aqueous pH and enhance more sickling of the RBCs.[6] Methazolamide should be used if systemic CAIs is needed in patients with SCD as it leads to less systemic acidosis and, therefore, less RBCs sickling than acetazolamide.[6] Mannitol can induce volume contraction and acidosis in patients with sickle cell and can be used as a last resort to avoid surgery.[6] Outpatient treatment can be only considered if the patient has normal IOP, grade II or less hyphaema, no history of SCD/SCT, no coagulopathy or bleeding diathesis, and able to adhere with daily ophthalmology evaluations.[4] As our patient did not meet the previous criteria, he should be admitted to hospital for careful follow up.

Surgical management (AC washing of blood with or without Trabeculectomy) of traumatic hyphaema is indicated in certain conditions when prolonged elevated IOP causes complications, for example, optic atrophy and corneal blood staining.[4] To avoid corneal blood staining, AC washout is indicated if there is early corneal blood staining or the IOP > 25 mmHg for five days.[4] To avoid optic atrophy, AC washout is indicated if IOP >60 mmHg for two days, or >35 mmHg for seven days.[4] To avoid PAS, AC washout is indicated for a complete hyphaema lasting for five days, or any hyphaema failing to reduce to a volume of <50% by days.[4] Patients with sickle cell haemoglobinopathies are at a greater risk of constant IOP elevations due to RBCs sickling and require aggressive IOP control. Surgery is indicated in SCD or SCT patients if the IOP≥24 mmHg for 24 hours or if the IOP transiently>30 mmHg for two to four days.[4, 19]

Prognosis

Beside the complications mentioned previously, traumatic hyphaema is typically a self-limiting condition which rarely leads to permanent vision loss if there is no associated damage to the cornea, lens or optic nerve.[1] Complications and VA are worse in total hyphaema than subtotal hyphaema.[6] In one study, 35% of patients achieved a VA (20/40) following total hyphaema whereas 76% of patients achieved the same VA following subtotal hyphaema.[20]

Consent: Informed consent was obtained from the patient to publish their anonymised data.

Interest of conflict: Author declares no interest of conflict.

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