A Case Report: Complicated Traumatic Hyphaema

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ABSTRACT

A 72-year-old male presented with a left traumatic hyphaema due to blunt trauma. Clinical examination found a 60% hyphaema and a mild increase in intraocular pressure. The hyphaema was slowly resolving with the patient suffering a secondary haemorrhage and a reduction in visual acuity. The patient was taking anticoagulant medication, which predisposed him to developing a secondary haemorrhage. The secondary haemorrhage was surgically cleared but an improvement in visual acuity was not seen. A traumatic cataract was found upon slit lamp examination and was surgically removed resulting in an improvement in vision.

Keywords: traumatic hyphaema, blunt trauma, angle recession, traumatic cataract, anti-coagulants, intraocular pressure

INTRODUCTION

A hyphaema is an accumulation of red blood cells within the anterior chamber.[1] Hyphaemas are generally caused by blunt or penetrating ocular trauma, but can also occur after surgery, spontaneously, or in patients who take anticoagulant medications that alter platelet and thrombin function such as aspirin and warfarin.[1,2]

Patients with hyphaemas often present with reduced vision or pain in the eye and a history of trauma.[3] Complications that can occur in hyphaema patients include elevated intraocular pressure, and secondary haemorrhage.[2] Elevated intraocular pressure is the most common complication of traumatic hyphaema and if left untreated it may result in clinical consequences such as glaucomatous optic neuropathy.[4,5] Thus, in order to prevent such consequences, accurate intraocular pressure readings are imperative.

A secondary haemorrhage is the second most common complication of traumatic hyphaema.[4] Predisposition to a secondary haemorrhage is linked to several factors with one of them being the use of anticoagulant medication, which prevent platelet aggregation and plug formation.[6]

Potential long-term complications include, late onset angle-recession glaucoma, late-onset traumatic cataract, and/or retinal detachment.[4] Studies have found that the extent of angle recession is directly correlated to an increase in risk of development of angle-recession glaucoma in the future.[2,5,7] Therefore, in cases of traumatic hyphaemas caused by blunt trauma measurement of the extent of angle recession is helpful in determining potential risk.

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CASE REPORT

A 72-year-old male presented to the emergency department with a left hyphaema following blunt trauma to the eye. The right eye was unaffected and normal in appearance. The patient complained of reduced vision in the left eye with no pain or photopsia.

The patient had no significant past ocular history and was slightly myopic. His medical history revealed lung cancer and heart disease, including coronary stent surgery four years earlier. Hence it was noted that the patient's medications list included two anticoagulants acetylsalicyclic acid (Aspirin) and clopidogrel bisulfate (Plavix).

Upon initial ocular examination no orbital bruising was seen around the left eye. Left visual acuity (VA) without correction was 6/24, improving to 6/18 with pinhole and right VA was 6/5. Slit lamp examination showed the left anterior chamber had a 60% hyphaema with 4+ red blood cells, the pupil was round and no epithelial defects were seen. TonoPen intraocular pressure (IOP) was 18mmHg in the right eye and 26mmHg in the left. Examination of the left fundus was not possible due to the presence of the hyphaema hence a B-scan was performed. There was no evidence of peripheral vascular disease, retinal detachment or vitreous haemorrhage.

The patient's diagnosis was a left hyphaema, caused by blunt trauma with a mild increase in IOP. Management

included bed-rest with the patient's head elevated at 30 degrees. Prescription of gutte prednisolone acetate (Prednefrin Forte), gutte apraclonidine (Iopidine), gutte isopto homatropine (Homatropine) and acetazolamide (Diamox) tablets was instructed. In consultation with the patient's cardiologist, cessation of both anticoagulants was also advised. Review of the patient was to be the following day. However the patient did not return for review until five days after initial presentation.

At the first review appointment, the patient complained of intermittent discomfort in the left eye. He felt that the vision in the left eye had been improving, only to decrease the day before review, upon recommencing the anticoagulants. Left VA with correction was 6/60, with improvement on pinhole to 6/36. Left IOP obtained via iCare and Goldmann applanation was 30mmHg and 28mmHg respectively. Slit lamp examination of the left eye, showed a reduction in hyphaema size to 50%. The anterior chamber appeared deep and contained 4+ red blood cells.

The hyphaema at this visit was slowly improving. Management included continuing with the current eye drop regime with the addition of gutte brimonidine tartrate (Combigan). Cessation of the anticoagulants was also advised and the patient's cardiologist was contacted in regards to this issue. A review appointment was booked for the next day.

At the second review appointment (6 days post presentation) the patient's vision had decreased further, correcting to hand movements with no improvement on pinhole. Goldmann applanation IOP in the left eye was 27mmHg. Slit lamp examination showed that the hyphaema consisted of 20% free-floating red blood cells and 40% coagulated blood. Management included continuation of the current medication regime and cessation of the anticoagulants.

At the third review appointment (8 days post presentation) there was no change in vision. Left IOP via iCare and Goldmann applanation was 31mmHg and 32mmHg, respectively. Slit lamp examination showed a 60% deep hyphaema with 40% red blood cells, and the presence of microcysts within the anterior chamber. The hyphaema was not clearing but the IOP remained stable. The current medication regime was continued.

At the fourth review appointment (9 days post presentation) visual acuity remained unchanged. Left IOP via iCare and Goldmann applanation was 27mmHg and 29mmHg, respectively. Slit lamp examination showed superior iris debris, mild stromal staining and the presence of microcysts. As the hyphaema showed no signs of spontaneous resolution and the IOP remained elevated, an anterior chamber washout procedure was planned.

However, two days later the patient presented to the emergency department complaining of pain and loss of vision in left eye overnight. Left IOP via TonoPen was 48mmHg and slit lamp examination showed that the hyphaema covered the entire pupil. There was ++corneal staining and 4+ red blood cells were seen in the anterior chamber. The patient suffered a secondary haemorrhage with an increase in IOP and an urgent anterior chamber washout procedure was done.

One day post anterior chamber washout, left VA remained unchanged and IOP via Goldmann applanation was 18mmHg. Slit lamp examination showed signs of corneal staining and the anterior chamber in the left eye was deeper than the right, containing 4+ inflammatory cells. The presence of a cataract was observed in the left eye as well as possible recession of the angle due to the trauma sustained.

One week after the procedure (18 days post presentation), left VA with correction was 6/60, improving with pinhole to 6/18. Left IOP via iCare was 5mmHg. Slit lamp examination showed the presence of corneal staining, and an anterior and posterior subcapsular cataract, with a grading of 2+ and 1+ respectively. No cyclodialysis cleft was observed and the optic disc was normal. The anterior chamber contained 2-3+ inflammatory cells. As improvement was evident isopto homatropine and brimonidine tartrate were ceased. Prednisolone acetate was continued but at a reduced frequency and the anticoagulants were also recommenced.

Two weeks after the procedure (25 days post presentation) left VA with correction was 6/36, improving with pinhole to 6/9. Left IOP via both iCare and Goldmann applanation was 13mmHg. Slit lamp examination showed a deep anterior chamber with 1+ inflammatory cells and no abnormalities were seen at the optic disc. Management included reducing prednisolone acetate and review was booked for one month's time.

At the next review appointment (55 days post presentation) it was decided that cataract surgery was needed to improve left VA. Pre-cataract operation, left VA with correction was 3/36, which improved with pinhole to 6/18. Post-cataract operation, left VA without correction was 6/18, which improved on pinhole to 6/9. The patient was to be reviewed in 6 months.

DISCUSSION

Blunt trauma to the eye causes globe compression, which leads to an equatorial expansion of the globe.[8] This expansion places stress on anterior chamber structures, and a subsequent haemorrhage can result due to the rupturing of iris and/or ciliary body vessels.[2] Hyphaema absorption occurs via the trabecular meshwork pathway, where the erythrocytes leave the anterior chamber as intact and undamaged cells. Uncomplicated hyphaemas generally resolve within one week from initial presentation.[2] However, in this case the patient's hyphaema did not resolve

However, in this case the patient's hyphaema did not resolve within one week and a secondary haemorrhage occurred.

The most common resultant complication of traumatic hyphaema is elevated intraocular pressure (IOP).[4] The term intraocular pressure can be described as the amount of tension utilised by the aqueous humour within the intraocular tissues in response to the balance between its production and drainage.[9] Elevated IOP was seen at initial presentation and continued to be elevated at each review appointment. The increase in IOP occurs as a result of obstruction of the trabecular meshwork from red blood cells, fibrin and platelets or from direct damage to the outflow pathways.[10] If elevated IOP is left untreated it may further result in clinical consequences such as glaucomatous optic neuropathy, acute retinal arteriolar occlusion or corneal bloodstaining.[5]

To prevent these clinical consequences, accurate IOP measurement is imperative. The most common commercially available tonometers used for IOP measurement are based on corneal indentation/applanation principles.[9] The tonometry devices utilised in this case were the iCare, TonoPen and Goldmann Applanation Tonometer (GAT). The GAT device is the current gold standard for IOP measurements because it has been shown to have less variation in repeated IOP measurements.[11,12] The accuracy of GAT however, is influenced by corneal thickness, curvature and biomechanical factors, with these factors being highly variable among individuals.[13] In patients with thin corneas (<525μm) GAT underestimates IOP and in thicker corneas (>555μm) GAT overestimates IOP.[9]

Studies have found that iCare IOP measurements are also influenced by central corneal thickness (CCT).[14] When compared to GAT, iCare overestimated IOP at higher CCTs and underestimated at lower CCTs.[14] Overall, iCare would be practical for screening purposes but if used clinically, CCT should always be considered in addition to the measurement.[9] When compared to GAT, the TonoPen was similar to GAT in that the device showed a weak statistically significant correlation with CCT values.[15] This device also underestimated IOP in thinner corneas and overestimated in thicker corneas.[15] Therefore, TonoPen measurements should be interpreted cautiously, especially in eyes with thick corneas.[9]

Among all the commercially available tonometers, no device has been found to give accurate IOP measurement regardless of CCT.[9] Therefore, rather than using multiple devices on a patient as within this case report, clinicians should choose the tonometer best suited to the clinical indication and use it consistently during clinical follow up.[9] Consistent use of the same tonometer is just as important as the choice of tonometer itself.[16]

After elevated IOP, the second most common complication of traumatic hyphaema is a secondary haemorrhage.[4] As previously mentioned, the patient's hyphaema did not resolve within one week and a secondary haemorrhage occurred. A secondary haemorrhage often occurs within the first five days of the initial bleed and occurs as a result of retraction and lysis of the clot, which had occluded the initial damaged vessel.[1,6] In this case, the patient suffered a secondary haemorrhage approximately 13 days after initial presentation with an associated increase in IOP. Factors which predispose a patient to a secondary haemorrhage include a presenting VA of 6/60 or worse, an initial hyphaema volume of more than one-third of the anterior chamber, the use of salicylates or other anticoagulating drugs, positive sickle cell anaemia and delayed seeking of medical attention (greater than 24 hours).[6]

Aspirin (acetylsalicyclic acid) is an anticoagulant, which has an inhibitory effect on the blood clotting mechanism of the body.[17] It prevents platelet aggregation and plug formation, which results in a prolonged bleeding time.[17] A study conducted by Crawford et al[17] concluded that the incidence of secondary haemorrhages increased with the administration of aspirin. Thus this drug would be contraindicated in conditions where clot formation is needed to enable resolution, such as the presence of a traumatic hyphaema. Recchia et al[8] recommended restriction of these drugs for two weeks after a traumatic hyphaema unless necessary for a medical condition. The patient was taking both salicylates and antiplatelet drugs for management of his cardiac condition, so it was critical to cease these to prevent a secondary haemorrhage. However, as it is important not to impact upon the patient's cardiovascular health, the cardiologist is best involved in the management process.

Surgical management of the patient's secondary haemorrhage was an anterior chamber washout to clear the haemorrhage and reduce IOP.[6] The procedure has minimal surgical complications and involves washing out the erythrocytes from the anterior chamber clot, leaving behind the fibrin matrix.[6] Post anterior chamber washout the patient's IOP was successfully reduced but left VA remained poor.

A secondary haemorrhage is generally more severe than the initial hyphaema and is thought to be associated with a poor visual prognosis.[1,18] However a retrospective study conducted by Cho et al[10] concluded that a secondary haemorrhage does not result in a poor visual outcome. The severity of the trauma and the associated ocular injuries, especially posterior segment injuries were found to result in a poor visual outcome.

A final visual acuity of less than 6/9 has been found to be associated with macular hole, traumatic cataract and macular degeneration.[6] In this case a traumatic cataract was detected on slit lamp examination and this was deemed the cause of the reduced VA. Cataract formation may occur as an early or late complication of ocular trauma. Trauma-related lens abnormality results from either a loss of transparency

or loss of positioning.[19] The patient had left cataract surgery and post operation VA subsequently improved.

Possible angle recession was also noted on slit lamp examination. Patients with angle recession are at a higher risk of developing secondary glaucoma later on in life and often require long term monitoring to prevent irreversible optic nerve damage.[7,8] The extent of angle recession has been found to directly correlate with an increased risk of developing a secondary glaucoma, particularly if the angle is recessed more than 180 degrees.[2] One prospective study found that the early predictors of traumatic glaucoma following blunt trauma were heavy trabecular meshwork pigmentation, an elevated baseline IOP, hyphaema, angle recession and lens displacement with a cataract,[7] with most of these factors being present in this case. There was no exact measurement taken of the extent of angle recession seen in the patient, so in hindsight this information would be useful in determining the relative risk of the patient developing a secondary glaucoma in the future.

CONCLUSION

An uncomplicated traumatic hyphaema generally resolves within a week of the initial presentation. In this report, the patient's hyphaema did not spontaneously resolve and a secondary haemorrhage occurred. This was likely associated with the patient's use of salicylates and antiplatelet drugs taken for management of his cardiac condition. Therefore it is critical to cease these drugs to prevent complications such as a secondary haemorrhage. However, this results in a clinical dilemma, as cessation of the drugs may impact negatively upon the patient's cardiovascular health. Thus liaison between the ophthalmologist and cardiologist would be vital in the management of these cases of traumatic hyphaema.

In this report the secondary haemorrhage was managed via an anterior chamber washout, which did not improve vision. This reduction in vision was due to the development of a traumatic cataract, as vision improved after cataract surgery was performed.

Elevation in intraocular pressure is another complication of a traumatic hyphaema. If this is not effectively managed numerous clinical consequences may occur. In order to prevent these clinical consequences accurate IOP measurement is imperative. Therefore, rather than using multiple devices on a patient as in this case report, it is recommended that clinicians choose and consistently utilise at each review appointment, a tonometer best suited to the clinical condition. By doing so the accuracy of results would be maintained, thus ensuring that there are no contradictory measurements, ultimately ensuring appropriate patient management.

A factor that should also be taken into account in cases with blunt trauma is angle recession. This should not be overlooked because angle recession is directly correlated with an increased risk of development of a secondary glaucoma, especially if the extent of recession is more than 180 degrees. Therefore information about the extent of angle recession would be useful in determining the patient's relative risk of developing a secondary glaucoma in the future.

ACKNOWLEDGEMENTS

The authors would like to acknowledge and thank Linda Santamaria for her support and assistance.

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