CASE IN POINT

PEER REVIEWED

Traumatic Hyphema After Minor Ocular Trauma in a Child with Sickle Cell Trait: A Case Report

Kamian Buggage, BS, Ayobami Olanrewaju, MBBS, MPH, FAAP

Volume 66 - Issue 2 - February 2026

Introduction. We report the case of a 12-year-old boy with sickle cell trait (SCT) who developed traumatic hyphema and subsequently experienced acute visual decline. Despite multiple emergency department visits, appropriate intervention was delayed. The patient did not respond to medical therapy, including topical timolol and latanoprost and oral acetazolamide, and ultimately required anterior chamber washout for persistently elevated intraocular pressure (IOP). Notably, carbonic anhydrase inhibitors, such as acetazolamide, are relatively contraindicated in patients with sickle hemoglobinopathies since they may promote intraocular acidosis and red blood cell sickling. This case underscores the importance of early recognition and prompt intervention for hyphema in patients with SCT. As an ophthalmologic emergency, this case also highlights the need for improved clinician awareness and patient education regarding potential complications associated with SCT.

Case Description. A 12-year-old boy presented to the emergency department with worsening right eye pain, redness, photophobia, eyelid swelling, and blurred vision. His symptoms began 3 days after being struck in the right eye with a toy gun that shoots small, water-absorbent polymer beads at a high rate of speed. He had previously been evaluated at 3 different emergency departments, where evaluation was limited, and he was prescribed prednisolone eye drops, timolol, latanoprost, and oral acetazolamide without symptom relief.

Upon presentation, the patient's intraocular pressure (IOP) was markedly elevated at 49 mmHg in the right eye and 27 mmHg in the left (normal range for both eyes, 10-20 mmHg). The patient's pupillary examination revealed a non-reactive, dilated right pupil with a relative afferent pupillary defect. A slit-lamp examination demonstrated 4+ pigmented cells, 2+ flare, microcystic corneal edema, and a hazy anterior chamber in the right eye. Funduscopic examination could not be assessed due to photophobia.

Given the persistently elevated IOP and anterior chamber findings, ophthalmology was urgently consulted and recommended anterior chamber washout, which was scheduled for the following day.

A subsequent sickle cell screening revealed hemoglobin A, 57.2% (normal range, 96-98%) and hemoglobin S, 39.2% (normal range, 0%), consistent with sickle cell trait (SCT).

The patient underwent anterior chamber washout without complications. Surgical intervention was indicated because of refractory IOP elevation in the setting of traumatic hyphema and underlying SCT, given the increased risk of vision-threatening complications.

At 3 days postoperatively, visual acuity in the right eye was 20/70-2, consistent with moderate visual impairment, while the left eye remained 20/15-2 (no impairment). The patient was initially followed up with ophthalmology for ongoing monitoring and evaluation for potential long-term complications, such as angle recession glaucoma. Unfortunately, as of this writing, the patient has been lost to follow-up with ophthalmology and pediatric hematology.

Discussion. Sickle cell trait, the heterozygous state of sickle cell disease, is often perceived as clinically benign. However, under specific physiological stressors—such as hypoxia, dehydration, and acidosis—red blood cells in individuals with SCT may undergo sickling, leading to significant morbidity. A particularly dangerous condition in this patient population is traumatic hyphema, as sickled red blood cells can obstruct the trabecular meshwork, causing acute increases in IOP and potentially irreversible vision loss if not promptly treated.[1,2]

Given this risk, screening for sickle hemoglobinopathies is warranted in patients with traumatic hyphema who have risk factors for SCT or sickle cell disease, including those of African descent, as well as individuals from malaria-endemic regions, including South-East Asia and India. Even a small or low-grade hyphema can significantly impair aqueous outflow in sickle cell disease and SCT, resulting in uncontrolled IOP elevation and subsequent optic neuropathy, thereby posing a serious threat to vision.[1,3]

Another vital teaching point in this case is that this patient was given oral acetazolamide as part of the initial medical treatment for his elevated IOP. Carbonic anhydrase inhibitors are relatively contraindicated in sickle cell disease because they can cause acidosis in the aqueous humor, which in turn promotes red blood cell sickling. These sickled cells in the eye can then block the trabecular meshwork, worsening IOP.[4,5]

A meta-analysis of 30 trials (23 randomized and 7 quasi-randomized) involving 2,969 participants assessed multiple medical interventions for traumatic hyphema, including antifibrinolytic agents, corticosteroids, cycloplegics, miotics, aspirin, conjugated estrogens, traditional Chinese medicine, eye patching, head elevation, and bed rest. The review demonstrated no significant effect of any intervention on visual acuity at either short-term (≤ 2 weeks) or longer-term follow-up. The authors concluded that none of the evaluated strategies improved visual outcomes.[6]

Traumatic hyphema carries significant long-term risks, most notably angle recession glaucoma, which may develop months to years after the initial injury due to damage to the trabecular meshwork. Angle

recession is commonly observed following blunt ocular trauma, and while only a subset of patients develop glaucoma, the risk persists indefinitely. Other sequelae include peripheral anterior synechiae, corneal blood staining, and optic atrophy from prior IOP elevation. Accordingly, long-term follow-up with periodic gonioscopy and IOP monitoring is essential.[7]

Mir and colleagues at the Wilmer Eye Institute[1] examined the clinical features and outcomes of hyphema in patients with SCT. Although the study was limited by sample size, they found that more than half of the participants (57%) required surgical intervention for IOP control. Importantly, although their study was limited in size, none of the patients experienced serious IOP-related complications, such as glaucomatous optic neuropathy, corneal bloodstaining, or retinal vascular occlusion, nor did they develop significant postoperative complications, like rebleeding or lens injury. These findings suggest that the advantages of early surgical IOP management may outweigh potential risks in selected patients with SCT. Overall, the authors concluded that patients with SCT are particularly prone to IOP elevation following hyphema, with the majority needing 1 or more surgical procedures to achieve adequate control.[1]

Conclusion. Clinicians should not assume that SCT is benign in all cases. Traumatic hyphema in patients with SCT is an ophthalmologic emergency that warrants prompt evaluation and, in many cases among patients with SCT, early surgical intervention to prevent irreversible vision loss. Topical carbonic anhydrase inhibitors should be avoided due to their potential to exacerbate intraocular acidosis and red blood cell sickling. Patient and caregiver education, together with heightened clinical vigilance, are critical to ensuring timely diagnosis and effective management. In addition, long-term ophthalmology follow-up is essential for monitoring late complications, such as angle recession glaucoma and other sequelae of ocular trauma.

AUTHORS

Kamian Buggage, BS[1] • Ayobami Olanrewaju, MBBS, MPH, MTropPaed, FAAP[2]

AFFILIATIONS

[1] Louisiana State University Health, Shreveport, Shreveport, LA

[2] St. Jude Shreveport Affiliate Clinic Pediatric Hematology and Oncology, and Louisiana State University Health, Shreveport, Shreveport, LA

CITATION

Buggage K, Olanrewaju A. Traumatic hyphema after minor ocular trauma in a child with sickle cell trait: a case report. *Consultant*. Published January 2, 2026. DOI: 10.25270/con.2026.01.000003

Received: September 1, 2025. Accepted: October 2, 2025.

DISCLOSURES

The authors report no relevant financial relationships.

ACKNOWLEDGMENTS

None.

CORRESPONDENCE
Ayobami Olanrewaju, MBBS, MPH, MTropPaed, FAAP, Louisiana State University Health, Shreveport, 1501 Kings Hwy, Shreveport, LA 71103 (email: ayobami.olanrewaju@lsuhs.edu)

References

1. Mir T, Iftikhar M, Seidel N, Trang M, Goldberg MF, Woreta FA. Clinical characteristics and outcomes of hyphema in patients with sickle cell trait: 10-year experience at the Wilmer Eye Institute. *Clin Ophthalmol Auckl NZ*. 2020;14:4165-4172. doi:10.2147/OPTH.S281875

2. Bansal S, Gunasekeran DV, Ang B, et al. Controversies in the pathophysiology and management of hyphema. *Surv Ophthalmol*. 2016;61(3):297-308. doi:10.1016/j.survophthal.2015.11.005

3. Gragg J, Blair K, Baker MB. Hyphema. In: StatPearls. StatPearls Publishing; 2025. Accessed September 30, 2025. http://www.ncbi.nlm.nih.gov/books/NBK507802/.

4. Ebied AM, Gracey S. Acetazolamide-induced sickle cell crisis. *Ann Pharmacother*. Published online June 12, 2018. doi:10.1177/1060028018782479

5. Stoner A, Harris A, Oddone F, et al. Topical carbonic anhydrase inhibitors and glaucoma in 2021: where do we stand? *Br J Ophthalmol*. 2022;106(10):1332-1337. doi:10.1136/bjophthalmol-2021-319530

6. Woreta FA, Lindsley KB, Gharaibeh A, Ng SM, Scherer RW, Goldberg MF. Medical interventions for traumatic hyphema. *Cochrane Database Syst Rev.* 2023;3(3):CD005431. doi:10.1002/14651858.CD005431.pub5

7. Ng DSC, Ching RHY, Chan CWN. Angle-recession glaucoma: long-term clinical outcomes over a 10-year period in traumatic microhyphema. *Int Ophthalmol.* 2015;35(1):107-113. doi:10.1007/s10792-014-0027-5

HMP Education

HMP Omnimedia

HMP Europe