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HIFEMA TRAUMATIK

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***Abstrak*:** *Traumatic hyphema is an ophthalmological condition resulting from bleeding in the anterior chamber, generally caused by blunt trauma to the eyeball. This condition can cause decreased visual acuity and carries the risk of serious complications such as secondary glaucoma, corneal staining, and optic atrophy if not treated promptly and appropriately. This paper aims to provide a comprehensive understanding of traumatic hyphema, including its etiology, pathophysiology, classification, clinical manifestations, diagnosis, management, and potential complications. The method used is a descriptive study through a literature approach and clinical observation during the clerkship at the Department of Ophthalmology, M. Natsir Solok Regional General Hospital. Data were obtained from a literature review and direct observation of patients. The results indicate that hyphema is most often experienced by adolescent boys and is characterized by decreased visual acuity, pain, and blood accumulation in the anterior chamber. The diagnosis is confirmed through anamnesis, visual acuity examination, and evaluation of intraocular pressure. Management is conservative, and surgery is required in severe cases or cases accompanied by complications. Early treatment is crucial to prevent further complications. Therefore, good clinical understanding and early detection are key to improving the prognosis of patients with traumatic hyphema.*

Keywords : Traumatic hyphema; eye trauma; anterior chamber hemorrhage; visual acuity; secondary glaucoma.

INTRODUCTION

Hyphema is a medical condition characterized by bleeding in the anterior chamber of the eye, which generally occurs due to blunt trauma to the eyeball. The blood seen in the anterior chamber usually originates from tears in the iris or ciliary body blood vessels. In addition to trauma, hyphema can also occur due to intraoperative trauma, ruptured neovascularization, intraocular malignancies, or other vascular abnormalities.

According to epidemiological data in the United States, the incidence of hyphema, particularly traumatic hyphema, is estimated at approximately 12 cases per 100,000 population per year. Children and

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adolescents, particularly those aged 10–20, account for the highest proportion of hyphema cases, at 70%. Furthermore, the prevalence of hyphema is higher in males than females, with a ratio of 3:1. These epidemiological findings are further supported by Ilyas (2014) and Vaughan et al. (2019), who noted that adolescents are most susceptible due to intense physical activity and sports.

Hyphema can cause decreased visual acuity (visual acuity), depending on the degree of bleeding. In cases of complete hyphema, the patient's vision can decrease significantly to the point of only detecting light perception. Conversely, mild hyphema may be asymptomatic or cause only mild visual impairment. Although generally a direct manifestation of trauma, the presence of blood vessel abnormalities, such as those seen in intraocular tumors, diabetes mellitus, intraocular surgery, and chronic inflammation that triggers neovascularization, can also predispose to spontaneous hyphema development without a history of trauma.

From a pathophysiological perspective, traumatic hyphema can be caused by two main mechanisms: contusion that tears blood vessels in the iris or ciliary body, and a sudden increase in intraocular pressure that triggers blood vessel rupture. These mechanisms are described in detail by Kuhn and colleagues (2022) and Behbehani et al. (2020), which also discusses the role of hemostasis and fibrinolysis in the cessation and resolution of bleeding within the eyeball.

Clinically, hyphema is classified as an ophthalmological emergency requiring immediate management due to the risk of serious complications such as secondary glaucoma, corneal imbibition, and optic atrophy. If not treated appropriately, hyphema can lead to serious complications such as increased intraocular pressure (glaucoma), anterior or posterior synechiae, cataracts, corneal blood staining, secondary hemorrhage, and even permanent damage to intraocular structures. Clinical classification of hyphema according to the literature is based on the cause (traumatic vs. non-traumatic), the timing of bleeding (primary vs. secondary), the visibility of blood (micro vs. macrohyphema), and the degree of anterior chamber filling (Grade I–IV), as described by the American Academy of Ophthalmology (2021) and Carissa Aprilia et al. (2023).

Common clinical symptoms in patients with traumatic hyphema include decreased visual acuity, photophobia, and the appearance of blood in the anterior chamber. Diagnosis is established through a history of eye trauma and a physical examination supplemented by slit-lamp examination, tonometry, and ophthalmoscopy. Studies by Putri et al. (2024) and Tatsa Rizkia et al. (2022) emphasize the importance of early detection and a thorough assessment in preventing complications.

Management of hyphema includes conservative approaches such as bed rest, corticosteroids and intraocular pressure-lowering medications, and surgical intervention if indicated, such as uncontrolled glaucoma or recurrent bleeding. The use of aminocaproic acid is also recommended to prevent secondary bleeding, as outlined by Gragg et al. (2021) and Kuhn (2018).

Long-term complications of traumatic hyphema, such as secondary glaucoma, corneal hemosiderosis, posterior synechiae, and optic atrophy, have been widely discussed in the literature. Wahyu Saputra (2020) noted the incidence of these complications in hyphema patients at a regional hospital, providing local clinical data that reinforces global findings.

Given the urgency and clinical impact, a comprehensive understanding of the diagnosis and management of hyphema, particularly traumatic hyphema, is crucial for medical personnel, particularly

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young doctors undergoing clinical clerkships in Ophthalmology. The existing literature provides a comprehensive understanding of traumatic hyphema, although standardization of management is still needed, particularly in healthcare facilities with limited resources. Therefore, this paper aims to explore the etiology, pathophysiology, diagnosis, management, and potential complications of traumatic hyphema, as a contribution to improving the quality of care and education in ophthalmology by combining theoretical review and clinical observations during the clerkship period, emphasizing the importance of education and early treatment to improve patient outcomes.

METODOLOGI

This paper uses a descriptive method with a clinical clerkship study approach. The aim is to systematically describe cases of traumatic hyphema based on a review of theory and scientific literature, as well as clinical observations during the clerkship in the Ophthalmology Department at M. Natsir Solok Hospital. Data sources were obtained through literature review of relevant national and international scientific journals and books, as well as direct observation of hyphema patients treated at the hospital. This paper does not involve collecting primary data directly from patients; rather, it is educational and documentary in nature, aimed at deepening students' understanding of the pathophysiology, classification, diagnosis, and management of traumatic hyphema. Data analysis was conducted by comparing theories from various scientific sources with findings or observations obtained during clinical practice. This method is expected to provide a comprehensive overview of the clinical approach to managing traumatic hyphema cases and serve as a learning resource for medical students.

RESULTS AND DISCUSSION

Etiology and Risk Factors

Hyphema is usually caused by blunt trauma to the eye, such as being hit by a ball, stone, or air rifle bullet. Additionally, hyphema can occur due to incorrect eye surgery procedures. Other conditions that can cause hyphema, but are rare, include eye tumors (e.g., retinoblastoma) and blood vessel abnormalities (e.g., juvenile xanthogranuloma).[4]

Hyphema caused by blunt trauma to the eye can result from damage to the inner tissues of the eyeball, such as tears in the iris, ciliary body, and choroid. These tissues contain numerous blood vessels, which can cause bleeding. The resulting bleeding can originate from the main arteries and branches of the ciliary body, the choroidal arteries, the ciliary body veins, and the iris vessels on the side of the pupil. Bleeding within the anterior chamber of the eyeball is visible from the outside. This blood pooling is concentrated in the lowest part due to gravity.[4,6]

Pathophysiology

There are two mechanisms suspected of causing hyphema. The first mechanism is where the force of the trauma causes a contusion, tearing the blood vessels in the iris and ciliary body, which are vulnerable to damage. The second mechanism is where the trauma causes an acute increase in intraocular pressure, leading to rupture of blood vessels in the iris and ciliary body.[4,5,6]

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Figure 1. Pathogenesis and Pathophysiology of Hyphema

Severe inflammation of the iris, abnormal blood cells, and cancer may also cause bleeding in the anterior chamber. Blunt trauma can tear the iris or ciliary body blood vessels. Contusive forces tear the iris blood vessels and damage the angle of the anterior chamber. However, it can also occur spontaneously or in ocular vascular pathology. This blood can move within the anterior chamber, contaminating the inner surface of the cornea.[4,5,6]

Hemorrhage in the anterior chamber activates the hemostasis and fibrinolysis mechanisms. Increased intraocular pressure, blood vessel spasm, and fibrin formation are blood clotting mechanisms that stop the bleeding. This blood clot can spread from the anterior chamber to the posterior chamber. This blood clot typically persists for 4-7 days. After that, fibrinolysis occurs. After a blood clot forms in the anterior chamber, plasminogen is converted to plasmin by coagulation cascade activators. Plasmin breaks down fibrin, resulting in the dissolution of the clot. Blood clot degradation products, along with red blood cells and inflammatory debris, leak from the anterior chamber into the trabecular meshwork and uveascleral drainage.[4,5,6]

Bleeding can occur immediately after trauma, known as primary bleeding. Primary bleeding can be mild or severe. Secondary bleeding usually occurs on the fifth day after trauma. It is usually more severe than primary bleeding. Therefore, a person with hyphema must be hospitalized for at least five days. This secondary bleeding is said to occur because the resorption of the blood clot occurs too quickly, preventing the blood vessels from regenerating.[4,5,6]

Healing blood in the hyphema is released from the AOC in the form of red blood cells through the AOC angle into the canal of Schlem, while the remainder is absorbed through the iris surface. This absorption in the iris is accelerated by the presence of fibrinolytic enzymes in this area. Some of the hyphema is excreted after decomposing in the form of hemosiderin. If hemosiderin accumulates, it can penetrate the cornea, causing a yellowing of the cornea, a condition called hemosiderosis or corneal imbibition, which can only be corrected with keratoplasty. Corneal imbibition can be accelerated by a full hyphema accompanied by glaucoma. [4,5,6,7]

The presence of blood in the anterior chamber has several associated clinical findings. Angle recession can be found after blunt ocular trauma. This indicates separation of the longitudinal and circular fibers of the ciliary muscle. Angle recession can occur in 85% of patients with hyphema and is associated with the later development of secondary glaucoma. Traumatic iritis, with inflammatory cells in the anterior chamber, can be found in patients with hyphema. In this condition, changes in iris pigmentation occur even

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after blood has been removed. Corneal changes can range from corneal endothelial abrasion to limbal rupture. Pupillary abnormalities such as miosis and mydriasis can be found in 10% of cases. Other signs that may be found include cyclodialysis, iridodialysis, pupillary dislocation, lens subluxation, and rupture of the zonules of Zinn. Posterior segment abnormalities may include vitreous hemorrhage, retinal injury (edema, hemorrhage, and tears), and choroidal rupture. Papillary atrophy may occur due to increased intraocular pressure.[4,5,6]

Clinical Classification

1. Based on the cause, hyphema is divided into: 1, 3, 4
    a. Traumatic hyphema is bleeding in the anterior chamber caused by rupture of blood vessels in the iris and ciliary body due to trauma to the anterior segment of the eye.
    b. Hyphema due to medical procedures (e.g., incorrect eye surgery).
    c. Hyphema due to inflammation that affects the iris and ciliary body, resulting in ruptured blood vessels.
    d. Hyphema due to blood cell or blood vessel abnormalities.
    e. Hyphema due to neoplasms (e.g., retinoblastoma).
2. Based on the onset of bleeding, hyphema is divided into:
    a. Primary hyphema, which occurs immediately up to 2 days after eye trauma.
    b. Secondary hyphema, which occurs 2-5 days after eye trauma.
3. Based on the visible blood, hyphema is classified into:
    a. Macrohyphema, bleeding visible to the naked eye.
    b. Microhyphema, bleeding visible under a microscope.
4. Based on the blood content in the anterior chamber (COA), hyphema can be divided into:
    a. Grade 1, blood fills less than 1/3 of the anterior chamber
    b. Grade 2, blood fills 1/3-1/2 of the anterior chamber of the eye
    c. Grade 3, blood fills 1/2 – less than the entire anterior chamber
    d. Grade 4, blood fills the entire anterior chamber, known as total hyphema, blackball or 8-ball hyphema

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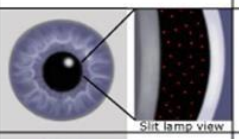
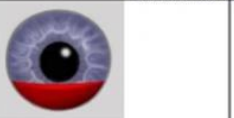
Grade	Anterior chamber filling	Diagram	Best prognosis for 20/50 vision or better
Microhyphema	Circulating red blood cells by slit lamp exam only		90 percent
I	<33 percent		90 percent
II	33-50 percent		70 percent
III	>50 percent		50 percent
IV	100 percent		50 percent

Figure 2. Hyphema Classification

Clinical Manifestations

The clinical manifestations of traumatic hyphema are generally characterized by decreased visual acuity, which varies depending on the degree of bleeding in the anterior chamber. Intraocular pressure (IOP) in patients may be within the normal range or elevated, especially if the angle of the anterior chamber is obstructed by a blood clot. On internal ophthalmological examination, the pupil may appear normal, mydriasis, or oval due to trauma to the iris. Furthermore, dilation of the pericorneal blood vessels is often found as an inflammatory response. One characteristic sign of hyphema is a visible accumulation of blood in the lower part of the anterior chamber when the patient is in a sitting position. If the bleeding persists or recurs, blood staining of the cornea can occur, which can permanently impair corneal clarity and vision.

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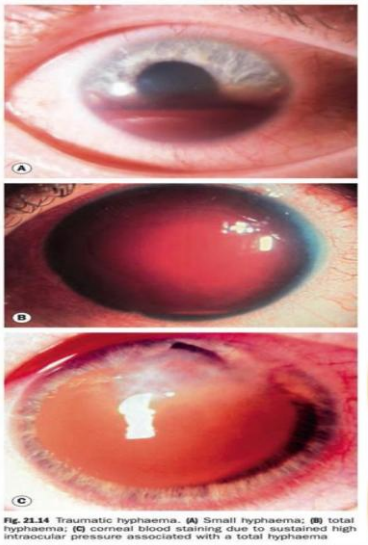

Figure 3. Clinical Manifestations of Hyphema

Diagnosis

The clinical diagnosis of traumatic hyphema generally begins with a history of eye trauma, such as blunt force trauma or direct injury. Physical examination reveals hemorrhage in the anterior chamber (AC), visible with a flashlight, accompanied by visual impairment and signs of irritation of the conjunctiva and pericorneal blood vessels. Patients often complain of photophobia (sensitivity to light), double vision, and blepharospasm due to eye pain and discomfort. Palpebral edema (swollen eyelids) and mydriasis may also accompany the complaint, in addition to difficulty seeing at close range. In some cases, systemic symptoms such as lethargy, disorientation, or somnolence are also present. Patients typically complain of sharp pain in the eye accompanied by watery eyes. When the patient is sitting, blood may be seen pooling in the lower ACAC and, in severe cases, can fill the entire anterior chamber. In addition, complications such as blood staining of the cornea and anisocoria (difference in pupil size) can also be found, which are serious indicators that require immediate evaluation and treatment.[8,9]

A direct consequence of hyphema is decreased vision because blood interferes with the refractive media. Blood filling the anterior chamber can directly increase intraocular pressure due to the increased blood volume in the anterior chamber. This increase in intraocular pressure is called secondary glaucoma. Secondary glaucoma can also occur due to a mass of blood blocking the trabecular meshwork, which drains aqueous humor from the anterior chamber. Furthermore, prolonged blood in the anterior chamber can cause blood staining of the corneal wall and damage to corneal tissue.[8,9]

In addition, supporting examinations are performed in cases of traumatic hyphema to assess the severity and detect potential complications. Visual acuity (visual acuity) is examined using a Snellen chart, where decreased visual acuity can indicate damage to the cornea, aqueous humor, iris, or retina. Visual field evaluation is also important, as decreased visual acuity can be caused by ocular vascular pathology or secondary glaucoma. Intraocular pressure (IOP) is measured using tonography to determine whether there

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is increased pressure due to obstruction of aqueous humor outflow by blood. Slit-lamp biomicroscopy helps assess anterior chamber (ACF) depth, iris-corneal contact (iridocorneal), aqueous flare (cloudiness of the ocular fluid), and the presence of posterior synechiae. Ophthalmoscopy is used to evaluate internal ocular structures, such as the retina and optic nerve. Furthermore, provocative tests can be performed if IOP is within normal limits or only slightly elevated to detect occult glaucoma. Examining pupillary reactions is also important for assessing the integrity of the oculomotor nerve. A simple penlight examination can help detect blood in the anterior chamber and quickly assess pupillary light reactions in clinical practice. All of these tests complement each other in establishing a diagnosis and determining appropriate management steps for patients with traumatic hyphema.

Management

Hyphema that occupies more than 5% of the anterior chamber should be rested. Steroid eye drops should be started immediately. Aspirin and non-steroidal anti-inflammatory drugs should be avoided. Pupillary dilation can increase the risk of rebleeding and should therefore be delayed until the hyphema resolves spontaneously. Therefore, early evaluation to check for posterior segment damage may require an ultrasound examination. The eye should be examined periodically for secondary hemorrhage, glaucoma, or corneal blood spots caused by iron pigment. Rebleeding occurs in 16-20% of cases within 2-3 days. This complication carries a high risk of glaucoma and corneal staining. Several studies suggest that oral aminocaproic acid (100 mg/kg body weight every 4 hours to a maximum of 30 g/day for 5 days) can stabilize blood clot formation, thus reducing the risk of rebleeding. Glaucoma management includes topical therapy with beta-blockers (e.g., timolol 0.25% twice daily), prostaglandin analogs (e.g., latanoprost 0.005% at night), dorzolamide 2% two or three times daily, or apraclonidine 0.5% three times daily. Oral therapy with acetazolamide 250 mg orally four times daily and hyperosmotic agents (mannitol, glycerol, and sorbitol) may also be used if topical therapy is ineffective. Glaucoma drainage surgery may be necessary in very severe cases.[4,11]

Hyphemas should be surgically evacuated if the intraocular pressure remains elevated (>35 mmHg for 7 days or 50 mmHg for 5 days) to avoid optic nerve damage and corneal staining, but there is a risk of rebleeding. If the patient has a hemoglobinopathy, glaucomatous optic atrophy is more likely, and surgical removal of the clot should be considered early. Vitrectomy instruments are used to remove the central clot and lavage the anterior chamber. An irrigation device and mechanical probe are inserted anterior to the limbus through the clear cornea to avoid damaging the iris and lens. Do not attempt to remove a clot that is in the angle of the anterior chamber or within the iris. Here, a peripheral iridectomy is performed. Another method for clearing the anterior chamber is viscoelastic evacuation. A small incision is made at the limbus to inject the viscoelastic, and a larger incision 180 degrees away (from the first incision) to allow the hyphema to be pushed out.[4,11]

Late-onset glaucoma can develop over several months or years, especially if the anterior chamber angle is narrowed in more than one quadrant. In rare cases, the corneal blood spots gradually disappear over a period of up to a year.4 In principle, it is divided into two main groups: conservative/non-surgical treatment and surgical treatment.

1. Conservative/Non-Surgical Treatment
    a. Bed rest
    b. Medications: coagulants, mydriatics, ocular hypotensives, corticosteroids, and antibiotics.[4]
2. Surgical Treatment

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Performed if there is secondary glaucoma, signs of corneal imbibition or corneal hemosiderosis, and no reduction in hyphema height with non-surgical treatment for 3-5 days. To prevent optic nerve atrophy, surgery is performed if the maximum intraocular pressure is >50 mmHg for 5 days or the maximum intraocular pressure is >35 mmHg for 7 days. To prevent corneal imbibition, surgery is performed if the average intraocular pressure is >25 mmHg for 6 days or if signs of corneal imbibition are found.4,6,9 Surgical intervention is performed to prevent peripheral anterior synechiae if the total hyphema persists for 5 days or the diffuse hyphema persists for 9 days. Surgical intervention is usually indicated on or after 4 days. The overall indications are as follows:

a. Four days after the onset of total hyphema
b. Microscopic corneal bloodstaining (any time)
c. Total hyphema with an IOP of 50 mmHg or more for 4 days (to prevent optic atrophy)
d. Total hyphema or hyphema filling more than 3/4 of the anterior chamber for 6 days with a pressure of 25 mmHg (to prevent corneal bloodstaining)
e. Hyphema filling more than 1/2 of the anterior chamber that persists for more than 8-9 days (to prevent peripheral anterior synechiae)
f. In patients with sickle cell disease with a hyphema of any size with an IOP of more than 35 mmHg for more than 24 hours. If the IOP remains elevated at 50 mmHg or more for 4 days, surgery should not be delayed. One study noted optic atrophy in 50 percent of patients with total hyphema when surgery was delayed. Corneal bloodstaining occurs in 43% of patients. Patients with sickle cell hemoglobinopathy require surgery if intraocular pressure is not controlled within 24 hours.[4,9]

The surgical procedures performed are:

1. Paracentesis

Paracentesis is a surgical procedure to remove fluid/blood from the anterior chamber of the eye using the following technique: a 2 mm corneal incision is made from the limbus toward the cornea, parallel to the iris. Usually, pressure on the edge of the wound will release coagulum from the anterior chamber. If the blood does not completely drain, the anterior chamber is rinsed with physiological saline. Usually, the corneal incision for paracentesis does not require suturing. Paracentesis is performed if the intraocular pressure does not decrease with diamox or if blood remains in the COA on days 5-9.

2. Irrigate the anterior chamber with physiological saline.
3. Using a similar procedure to cataract extraction, the corneosclera is opened 120 degrees.[4]

Complications

1. Secondary Hemorrhage

This bleeding often occurs on days 3 to 6, and its incidence varies widely, ranging from 10 to 40%. This secondary hemorrhage occurs due to iris irritation caused by trauma or as a continuation of the primary hemorrhage.[11]

2. Secondary Glaucoma

Secondary glaucoma in traumatic hyphema is caused by blockage of the trabecular meshwork by blood droplets/clots. The presence of blood in the anterior chamber can impede the outflow of aqueous humor because blood elements block the angle of the anterior chamber and trabeculae, leading to glaucoma.

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Secondary glaucoma can also occur due to contusion of the ciliary body, resulting in a recess in the angle of the anterior chamber, which disrupts aqueous humor outflow.[1,4]

3. Corneal Hemosiderosis

This hemosiderosis occurs when secondary hemorrhage is accompanied by increased intraocular pressure. Visual impairment due to hemosiderosis is not always permanent, but can sometimes return to clarity over a long period (2 years). The incidence is approximately 10%.3 Iron in the eyeball can cause siderosis bulbi, which, if left untreated, can lead to phthisis bulbi and blindness.[6]

4. Posterior synechiae

Posterior synechiae can occur in patients with traumatic hyphema. This complication is the result of iritis or iridocyclitis. This complication is rare in patients receiving medical therapy and is more common in patients undergoing surgical evacuation of the hyphema.[8]

5. Optic atrophy

Caused by increased intraocular pressure.[1]

Prognosis

The prognosis depends on the amount of blood accumulated in the anterior chamber. Hyphema with little blood and without glaucoma has a good prognosis (bonam) because the blood will be reabsorbed and disappear completely within a few days.[4,12]

For hyphema that has developed glaucoma, the prognosis depends on the extent to which the glaucoma causes a visual acuity defect. If visual acuity has reached 1/60 or lower, the prognosis for the patient is poor (night) because it can cause blindness.[4,12].

CONCLUSIONS

Traumatic hyphema is an ophthalmological emergency characterized by bleeding in the anterior chamber of the eye due to blunt trauma, typically involving tears in the iris or ciliary body blood vessels. This condition most commonly occurs in adolescent boys and can cause serious visual impairment, even blindness, if not treated appropriately. Diagnosis of hyphema requires a careful history of trauma, visual acuity examination, and evaluation of intraocular pressure. Additional examinations such as slit-lamp examination, tonometry, and ophthalmoscopy are helpful in determining the extent of bleeding and detecting potential complications.

Treatment of hyphema consists of conservative approaches, such as bed rest, administration of corticosteroids and intraocular pressure-lowering medications, and patient education. Surgical intervention is necessary in cases with uncontrolled intraocular pressure, persistent bleeding, or the development of complications such as secondary glaucoma and corneal hemosiderosis. Appropriate and prompt treatment is crucial to prevent further complications, such as secondary bleeding, anterior synechiae, and optic atrophy. Therefore, early recognition, close monitoring, and clinical education for healthcare workers, especially medical students, are important steps in improving the prognosis of patients with traumatic hyphema.

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