REVIEW ARTICLE

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Hyphema – from the effect to the cause

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Abstract: *The purpose of this review is to describe the etiology (traumatic, iatrogenic, spontaneous causes), clinical and paraclinical findings and the management of hyphemas.*
The clinical appearance of hyphema is variable and is influenced by the volume of blood and the amount of time erythrocytes are present in the anterior chamber. When hyphema is evident, a complete history should be obtained and a thorough physical examination performed to direct the initial selection of diagnostic tests. Secondary complications of hyphema include glaucoma, synechiae, cataract formation, blood-staining of the cornea, and blindness.
Early diagnosis and treatment can minimize or prevent the installation of secondary complications and improve the prognosis.

INTRODUCTION

Hyphema is blood collection in the anterior chamber of the eye (delimited anteriorly by the cornea, posteriorly by the lens and iris and circumferentially by the angle between iris and cornea).[1]

Depending on the blood volume present in the anterior chamber, hyphema is classified as (Figure 1):
• Grade 0: circulating erythrocytes (microhyphema)
• Grade 1: <1/3 anterior chamber volume
• Grade 2: 1/3 -1/2 anterior chamber volume
• Grade 3: >1/2 anterior chamber volume
• Grade 4: total anterior chamber volume (”eight ball hyphema”)

The symptoms that bring the patient to the doctor may vary depending of etiology, but the most common symptoms are blurred vision, pain and photophobia.

The most important thing in finding the etiology of hyphema is a detailed medical history of the patient. It’s important to ask the patient if he suffered a trauma, what was the mechanism and if an object was involved, because hyphema in some cases may be accompanied by the presence of an intraocular foreign body. Other questions will be related to certain diseases such as diabetes, sickle cell anemia or other blood disorders and treatment with antiplatelet agents or anticoagulants.

After a complete medical history, physical exam will include: biomicroscopy, FO exam, gonioscopy, ocular ultrasonography, ultrasound biomicroscopy, radiographic examination or CT (when there is severe orbit damage or the doctor suspects the presence of an intraocular

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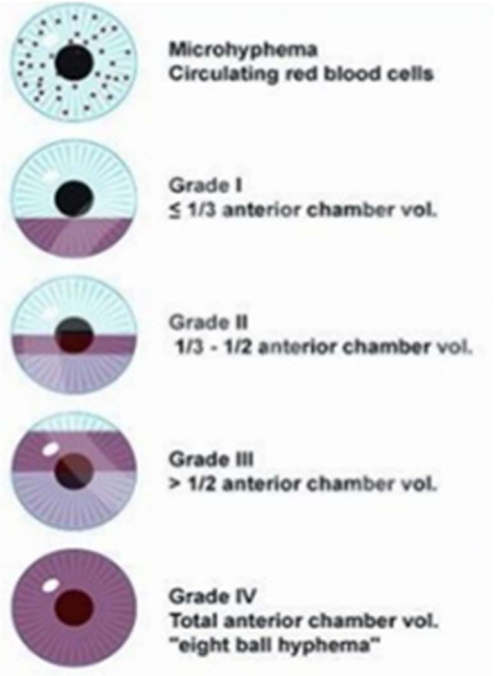
foreign body).[3]

Figure 1: Hyphema grading system
(American Academy of Ophthalmology)[2]



ETIOLOGY

Most frequently hyphema is caused by trauma, followed by intraocular surgery and spontaneous hyphema: abnormal growth of blood vessels – neovascularization (diabetes, ischemia), intraocular tumors (iris melanoma, juvenile xanthogranuloma, retinoblastoma), infections (herpetic keratouveitis), blood dyscrasias (sickle cell anemia, leukemia), anticoagulant therapy.[4]

Traumatic hyphema is most common in young males. Ocular trauma may be blunt or lacerating. The mechanisms of developing traumatic hyphema are:

- In blunt trauma occurs a rapidly anteroposterior compression of the eye with equatorial expansion and increased intraocular pressure, which leads to shearing forces that tear the vessels of the iris and ciliary body with accumulation of red blood cells within the anterior chamber.

- In lacerating trauma, traumatic agent causes direct damage of the vessels

Studies have shown that more than 50% of the cases, hyphema occupies less than 1/3 of the anterior chamber volume and less than 10% of the cases hyphema occupies the total volume of the anterior chamber.

In the case of a trauma, hyphema may be accompanied by other injuries such as angle recession (seen in 85% of traumatic hyphemas and can be associated with the development of glaucoma), traumatic iritis (presence of inflammatory cells in the anterior chamber and presence of pigment on the anterior capsule – Vossius ring – Figure 2), fixed dilated pupil in approximately 10 % of the cases, iridodialysis (disinsertion of the iris from the scleral spur), cyclodialysis cleft (separation of the ciliary body from the scleral spur, creating a direct connection between the anterior chamber and the suprachoroidal space, leading to ocular hypotony), corneal changes (from minor abrasion to corneal endothelium damage), cataract, lens subluxation, posterior segment injuries (vitreous hemorrhage, retinal edema/hemorrhages/ holes/tears, choroidal rupture etc.).[1]

Hyphema caused by ocular surgery can occur both intraoperatively and postoperatively. Intraoperatively, has been described in cataract surgery by direct damage of iris vessels, in peripheral iridectomy, cyclodialysis, and intervention on the posterior pole. Amsler sign is the presence of blood in the anterior chamber in patients with Fuchs heterochromic cyclitis, following rapid pressure change after anterior chamber paracentesis.

Postoperatively, the Uveitis Glaucoma Hyphema syndrome may occur after cataract surgery, caused by mechanic trauma of the iris by the IOL, more common in anterior chamber IOL.[5]

Spontaneous hyphema may occur secondary to iris neovascularization (proliferative diabetic retinopathy, ocular ischemic syndrome, retinal vein occlusion, chronic choroidal detachment), chronic uveitis (herpes zoster), tumors (uveal melanoma, retinoblastoma, lymphoma, leukemia, metastasis etc.), drugs (both anticoagulant and antiplatelet agents), blood dyscrasias (hemophilia, thrombophilia, von Willebrand disease, immune thrombocytopenic purpura).[6,7]

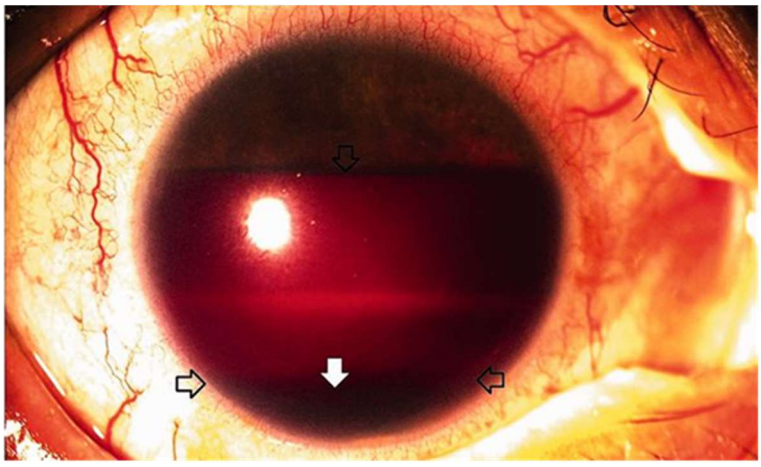
Figure 2: Vossius ring [11]

COMPLICATIONS

Obstruction of the trabecular meshwork with increased intraocular pressure is one of the complications that can result from untreated or improperly treated hyphema. This occurs in about 1/3 of hyphema cases. Studies have shown that 25% of patients develop intraocular pressure >25 mmHg and 10% of patients develop intraocular pressure > 10 mmHg. Usually, patients with a larger volume of blood in the anterior chamber have an increased risk in developing elevated intraocular pressure. Another risk factor would be the appearance of the recurrent bleeding. Intraocular pressure should be monitored daily. An untreated intraocular pressure > 35 mmHg for more than 5-7 days can lead to irreversible optic nerve damage.[8]

Recurrent bleeding or secondary hemorrhage (Figure 3) is another complication that can occur. It is described as increase of blood volume or appearance of a new layer of fresh blood in the anterior chamber. Most commonly occurs 3-5 days after the initial injury as a result of blood clot lysis. Recurrent bleeding increase the risk of other complications such as: increased intraocular pressure, corneal blood staining, optic atrophy, and peripheral anterior synechia. This complication is often associated with a weak prognosis or need for surgery.[9]

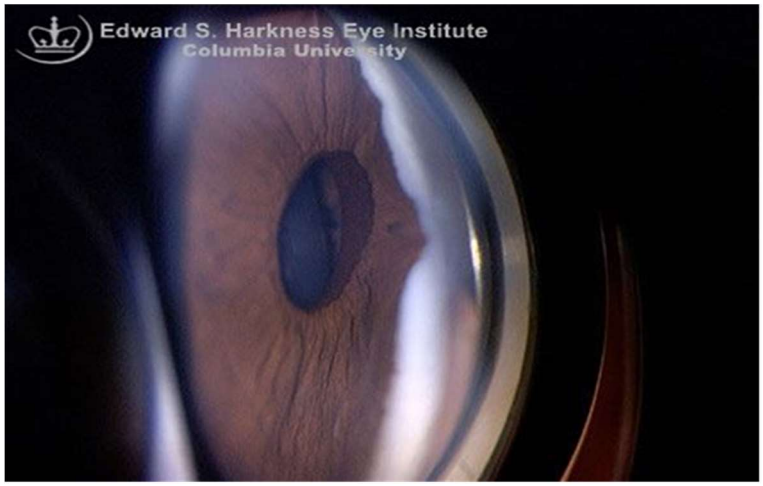
Figure 3: Recurrent bleeding [12]



Glaucoma followed by optic atrophy occurs in 10-20 % of patients with traumatic hyphema due to trabecular meshwork damage or due to peripheral anterior synechia which leads to secondary open angle glaucoma.

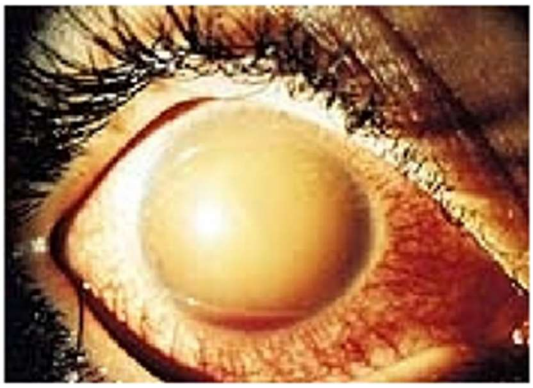
Another complication that can occur in an untreated hyphema is synechiae formation (Figure 4), both posterior synechia and peripheral anterior synechia. Peripheral anterior synechia are more common and occur after inflammation or persistent blood clot. Patients with hyphema dating more than 7 days have a higher risk of developing synechiae.[10]

Figure 4: Peripheral Anterior Synechiae [13]



Corneal blood staining (Figure 5) is the corneal impregnation with intra and extracellular hemoglobin and hemosiderin particles. This occurs in 2-11% of cases, particularly in cases of total hyphemas, increased intraocular pressure, recurrent bleeding, long persistent of a blood clot, corneal endothelial cell dysfunction. The first sign of corneal damage is the presence of fine, deep, yellow line in the stroma, especially central, followed by extension in 24 -36 hours. After the resolution of hyphema, cornea gradually clarify, in months or even years, from the periphery to the center.[15]

Figure 5: Corneal blood staining [14]



MANAGEMENT

The goals of the treatment are:

- clearing the hyphema
- reducing the bleeding rate
- treating the associated tissue lesions
- minimizing the long-term sequelae

Hyphema, unaccompanied by any complications, should be treated conservatively with a protective patch, limited activity, elevate the head of the bed at an angle of 30-45 degrees, so that the hyphema can settle out inferiorly and avoid obstruction of vision and limit the exposure of cornea to blood. The patient should be closely monitored during the first days when the risk of bleeding recurrence and increased intraocular pressure is higher.

Hospitalization is recommended in cases of noncompliant patients, patients with blood dyscrasias, with severe ocular and orbital damage, and patient with an increased intraocular pressure and sickle cell anemia.[16]

The medical treatment consists of administration of topical corticosteroids or, in severe cases, systemic corticosteroids to decrease inflammation associated with hyphema, topical cycloplegics (atropine 1% for 5 days: 1 drop/day in children, 1 drop x 3/ day in adults)

to stabilize the blood-aqueous barrier, to decrease pain by relaxing the ciliary spasm and prevent the formation of posterior synechiae, topical aqueous suppressants (beta-blockers and alpha-agonists) or systemic carbonic anhydrase inhibitors and hyperosmotic agents (acetazolamide or mannitol – 1,5 mg/body weight) if topical management fails to control the pressure, antifibrinolytic agents (aminocaproic acid – 50-100 mg/body weight every 4 hours, up to a maximum of 30 g/day, for 5 days, tranexamic acid) to reduce the risk of recurrent bleeding.[4,15,17]

Surgical intervention is required in approximately 5% of cases with the following indications: IOP elevation > 50 mmHg for 5 days, IOP elevation > 35 mmHg for 7 days to avoid optic nerve damage, IOP elevation > 25 mmHg for 5 days in cases of total hyphema to prevent corneal blood staining, IOP elevation > 25 mmHg for more than 24 hours at patients with sickle cell anemia, large stagnant clots persisting for more than 10 days to prevent peripheral anterior synechia formation.[16]

Surgical techniques are paracentesis/ AC washout for liquid blood – is the safest and simplest method able to evacuate circulating red blood cells, can be repeated, avoid affecting the conjunctiva in case of a possible future filtration surgery, provide control of intraoperative bleeding and fast reduction of intraocular pressure, and bimanual cutting/aspiration for clotted hyphema, using the vitrectomy probe. Other surgical interventions that may become necessary include: peripheral iridectomy and trabeculectomy for glaucoma, cyclodiathermy and ultrasonic emulsification and aspiration.[10]

PROGNOSIS

Prognosis for visual recovery is related to the following factors: amount of associated damage to other ocular structures (choroidal rupture, retinal detachment, posttraumatic macular edema, lens opacification, vitreous hemorrhage, and angle-recession glaucoma), whether secondary hemorrhage or complications such as glaucoma, corneal blood staining or optic atrophy occur.

Studies have demonstrated that more than 75% of patients have a good prognosis, so approximately 80% of patient with hyphema <1/3 AC regain visual acuity of 20/40 (6/12) or better, 60% of patient with hyphema >1/2 AC but less than total of AC regain visual acuity of 20/40 (6/12) or better, while only 35% of those with total hyphema have good visual results. Poor visual outcome in traumatic hyphema can be directly attributed to the hyphema in 11% of patients; the poor visual outcome is usually the result of secondary hemorrhage associated with optic atrophy or corneal bloodstaining.[10,16]

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