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Major review

Controversies in the pathophysiology and management of hyphema

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ABSTRACT

Traumatic hyphemas present dilemmas to physicians. There are numerous controversies pertaining to the optimal approach to traumatic hyphema and no standardized guidelines for its management. We address some of these controversies and present a pragmatic approach. We discuss various medical agents and surgical techniques available for treatment, along with the indications for their use. We address the complications associated with hyphema and how to diagnose and manage them and consider the management of hyphema in special situations such as in children and sickle-cell anemia and in rare clinical syndromes such as recurrent hyphema after placement of anterior chamber intraocular lenses.

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1. Introduction

For hyphema, the accumulation of blood in the anterior chamber, the most common cause is ocular trauma (blunt or penetrating)[33,62]; however, it can also be seen after intraocular surgery or spontaneously in conditions such as rubeosis iridis, juvenile xanthogranuloma, retinoblastoma, metastatic tumors, iris melanoma, myotonic dystrophy, keratouveitis, leukemia, hemophilia, thrombocytopenia, and Von Willebrand disease.[2,6,9,53,60,63,64,74,94] Hyphema can be a herald sign of major intraocular trauma and can itself cause complications such as secondary hemorrhage and glaucoma.[103] Even small hyphemas may be associated with significant damage to intraocular tissue.

Despite being a common condition, the management protocols for hyphema are still unclear. Conservative management

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options include bed rest, head elevation, an eye shield, and the use of pharmacologic agents (topical or systemic steroids, antifibrinolytics, cycloplegics, miotics, aspirin, traditional Chinese medicine, and conjugated estrogen).[34] Aside from the use of antifibrinolytics to prevent secondary hemorrhage, however, there is no evidence of benefit from the use of these conservative measures.[34] Furthermore, there is a lack of consensus regarding a treatment and follow-up strategy targeted at preventing delayed visual loss from complications of hyphema, as well as the management of certain special situations such as concurrent sickle-cell anemia.

We aim to address the controversies in the pathophysiology, evaluation, and management of hyphema. Because trauma is the commonest cause, we focus our discussion on closed-globe traumatic hyphema; however, we also analyze special situations such as uveitis, pediatric hyphema, cataract surgery, refractive surgery, and hyphema in patients with sickle-cell anemia.

2. Pathophysiology of hyphema

The mean annual incidence rate of traumatic hyphema is estimated as 17/100,000 population in individuals less than 18 years of age[1] and 20.7/100,000 population in individuals less than 20 years of age.[50] Direct orbital injury resulting in traumatic hyphema usually consists of a high-energy blow to the orbit (61%–66%), impact from a projectile (30.2%–36%), or injury secondary to an explosion (2.4%–3%).[50,97] Athletic injuries have become a major cause of traumatic hyphema, whereas accidents at work have become relatively less frequent. Kearns reported that athletic injuries accounted for 39.2% of 314 cases of traumatic hyphema, whereas accidents at work were responsible for 9.9% of the cases.[49]

The commonest source of blood in hyphema is a tear in the anterior face of the ciliary body.[107] A direct blow to the eye can rupture the blood vessels at the root of the iris. The most frequently ruptured vessels are the major arterial circle of the iris and its branches, the recurrent choroidal arteries, and the veins crossing the suprachoroidal space between the ciliary body and episcleral venous plexus.[95,107]

Blunt injury is also associated with anteroposterior compression of the globe and simultaneous equatorial globe expansion. Equatorial expansion induces stress on anterior chamber angle structures, which may lead to rupture of iris stromal and/or ciliary body vessels with subsequent hemorrhage.[22,95] Another possible source of initial hemorrhage is a rapid increase in intraocular pressure (IOP) immediately after the contusive trauma. This eventually leads to rupture of the fragile vasculature of the iris from the pupillary sphincter and/or angle.[22]

Lacerating injury may be associated with direct damage to blood vessels and hypotony, both of which can precipitate hyphema.[107] There is no consensus regarding the predominant source of bleeding (angle vessels or iris sphincter vessels) and their respective risks of rebleeding; however, current opinion is that fragile angle vessels have the higher risk of bleeding as a result of their proximity to the major arterial circle of the iris.[22]

Delayed hyphema after intraocular surgery may be the result of granulation tissue at the wound margin or caused by damaged uveal vessels (e.g. from surgical trauma or from intraocular lens-induced uveal trauma).[63,94] This mechanism should be considered in patients with a history of ocular surgery who present with spontaneous hyphema.

In the pediatric age group (less than 18 years of age) hyphemas in the absence of predisposing ocular or systemic disease or medication should arouse the suspicion of nonaccidental injury.[59] A significant but rare cause of spontaneous hyphema in children is juvenile xanthogranuloma. Juvenile xanthogranuloma is a predominantly dermatological disorder most commonly presenting in children less than 2 years old characterized by a raised, orange skin lesions occurring either singly or in crops that will regress spontaneously. The most common ocular finding is diffuse or discrete iris nodules that are often quite vascular and may bleed spontaneously. Occasionally, the lesions may present in other areas such as ciliary body, anterior choroid, cornea, lids, and orbit.[47] Complications include uveitis and glaucoma, with resulting visual loss and phthisis. Biopsy of skin lesions helps to confirm the diagnosis. The lesions classically contain an infiltrate of lipid-laden histiocytes, lymphocytes, eosinophils, and Touton giant cells.

Histologic examination of hyphemas reveals an erythrocyte aggregate enveloped by a pseudocapsule of fibrin-plated coagulum.[47] Clot absorption takes place by breakdown of fibrin by fibrinolytic agents and escape of red blood cells through the trabecular meshwork and Schlemm canal.[47] Agents that open the trabecular meshwork thus accelerate clot absorption.

3. Clinical features and examination

The importance of a detailed history and a thorough ocular and systemic evaluation cannot be stressed enough. The nature of the injury points to the likely type of damage sustained and therefore the prognosis. The priority in trauma is always to stabilize airway, breathing, and circulation, and make an assessment for threats to life. This is followed by an ophthalmic evaluation that includes inspection for gross ocular injury, evaluation of the adnexae, visual acuity, pupillary function, ocular motility, and the position of the globes.

Extensive conjunctival chemosis and hemorrhage may indicate an occult scleral rupture (Fig. 1). Proptosis may be secondary to a retrobulbar hematoma, and restriction in ocular motility may suggest an orbital blowout fracture or a contusive head injury. Every attempt should be made to examine the adnexal region carefully and rule out any associated orbital or head trauma. Hyphema can be associated with open-globe (Fig. 2A) or closed-globe injuries. In patients with open-globe injury, primary wound closure is the priority (Fig. 2B). One should not attempt surgical washout of hyphema in open-globe injuries as the blind approach could lead to adverse consequences. Surgical washout can be considered in patients with nonresolving hyphema or sickle-cell trait because of the higher risk of secondary glaucoma and permanent visual loss.[34] We shall focus on hyphema after closed-globe trauma.

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