Liver Disease Associated Subperiosteal Hematoma in a Postoperative Cataract Surgery Patient

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Abstract

Nontraumatic subperiosteal hematoma (NTSOH) has been reported to be associated with systemic coagulopathies. We present a 52-year-old female with a history of alcohol use who has recently undergone cataract surgery that develops a postoperative NTSOH. Liver disease-associated thrombocytopenia was diagnosed after the development of the hematoma.

Keywords: Subperiosteal hematoma; Liver disease; Nontraumatic hematoma

Introduction

Subperiosteal orbital hemorrhage (SOH) is a rare entity that typically occurs in young males, most commonly due to direct facial or orbital trauma. There are reports of non-traumatic SOH due to increased intracranial pressure, systemic diseases causing bleeding diatheses, and paranasal sinusitis [1-3]. We report a case of non-traumatic subperiosteal hemorrhage in a patient who has recently undergone bilateral cataract surgery.

Case Report

A 52-year-old white woman presented to the emergency room with eye pain and difficulty opening her right eye for the past 24 hours. She denied a history of trauma and had seen multiple ophthalmologists over the past day. A diagnosis of orbital cellulitis was made by a community physician and the patient was started on amoxicillin/clavulanate. The patient was noted to have jaundice and an extensive systemic workup led to a diagnosis of alcohol-induced thrombocytopenia. Liver disease-associated thrombocytopenia was diagnosed after the development of the hematoma.

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Intraocular lenses were well-positioned in both eyes and there was mild motility deficit of the right eye in all directions of gaze but no evidence of restriction with forced ductions. Intraocular pressure (IOP) was 14 in the right eye and 11 in the left eye. Slit-lamp exam revealed 4+ periorbital ecchymosis, edema, and tightness of the right eyelids and 360 degrees of chemosis of the conjunctiva of the right eye. The patient had undergone cataract surgery postoperatively. She was not using any medications in her right eye.

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Figure 1: 52-year-old female presented with periocular ecchymosis, edema, and tightness of the right eyelids and 360 degrees of chemosis of the conjunctiva of the right eye. The patient had undergone cataract surgery in the right eye 12 days ago and in the left eye 4 days prior to presentation.

Figure 2: Computed tomography of facial bones and orbit 1-mm sections. A. Sagittal view of orbit revealing homogenous mass in the superior orbit, causing compression of the superior aspect of the right globe. The location appears to be subperiosteal and measures 3.5 x 1.5 x 2.5 cm. B. Coronal view of orbit shows mass to lie in superonasal quadrant of globe, causing distortion of right eye.

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(ALT) of 22 (normal = 5-45); and ammonia level was 55 (normal = 12-43). During her hospitalization she had mental status changes due to elevated ammonia levels and was treated with vitamin K, folate, vitamin B12, and thiamine. Surgical intervention was deferred secondary to thrombocytopenia and the increased risk of bleeding.

The following day, the patient complained of worsening vision and distortion. There was advancement of the ecchymosis to involve both eyes and VA had dropped to 20/200 in the right eye (Figure 3). Repeat DFE revealed evidence of retinal compression with folds along superior and inferior arcades and peripheral macula with a blunt foveal light reflection. There was also evidence of inferior choroidal elevation but no evidence of optic disc edema or pallor.

When the patient was clinically worsening, an orbitotomy with drainage of the hematoma was performed. She was transfused preoperatively with two units of fresh frozen plasma (FFP) and two units of platelets which decreased INR to 1.5 and increased platelets to 129,000. Surgery confirmed the location of the hematoma to be subperiosteal along the roof of the orbit. She had immediate relief of pain, with a normal DFE and VA improved to 20/30 in the right eye by one week later.

Discussion

Subperiosteal hematomas forming on flat bones have been reported in the orbit, the calvarium, and the iliac wing [4]. They occur most commonly secondary to trauma with onset appearing immediately or even months later [2,3]. Seigel et al. reported 3 cases in 1982 describing young males ranging from 15-37 years old who developed these hematomas after trauma [5]. The trauma may be significant or minimal, as in a case reported by Sharma et al. in 2007 in which a child developed a large hematoma after minimal brow trauma [5,6]. Presenting symptoms may include pain, edema, ecchymosis, proptosis, diplopia, and decreased vision, especially if there is optic nerve compression. SOH most commonly presents in the superior orbit, as in our patient. These hematomas develop between the bone and separated periostium and usually occur as a result of direct rupture of subperiosteal blood vessels or as an extension of the subgaleal hematoma [5]. It is thought to occur in this location because this is the widest area in the orbit that is not interrupted by firm adhesions to the bony orbit, so there is space for expansion of the hematoma [4]. Seigel et al. felt the frequent delay in development of these hematomas supports the theory that secondary hyperemia and orbital congestion may lead to decompression and then indirectly, rupture of subperiosteal blood vessels, thus resulting in a hematoma [5]. Typically this tends to occur in younger children because with age the periosteum is tightly adherent to the calvaria thus making the separation between the bone and periosteum more difficult, although patients up to 73 years of age have also been reported in the literature [3].

Imaging will assist with diagnosis. On CT, SOH appears as a biconvex well-defined non-enhancing mass of homogenous density; the location and extent of the hematoma can be delineated and also allows excluding orbital wall fractures. The lesion can best be seen in the coronal or sagittal view. If the hematoma is seen on magnetic resonance imaging, T1-weighted imaging will demonstrated a low signal strength in the hyperacute stage and high signal strength in the acute phase, 3-7 days after the appearance of the lesion. The signal strength appears the opposite on T2-weighted imaging, while both T1 and T2 weighted imaging beyond 7 days of onset will show high signal strength until the clot organizes and the signals both shift to low strength [3].

In the absence of a history of trauma, a thorough systemic workup should be initiated to reveal underlying non-traumatic causes of SOH [5]. Moorhy et al. has reported two cases of liver disease-associated SOH, one with a history of hepatitis B not previously diagnosed, and the other with known alcoholic liver cirrhosis [9]. Our patient is the first known case reported who was diagnosed with alcohol-associated liver disease that initially presented with a NTSOH. Liver disease-associated NTSOH occurs due to depression of clotting factors II, VII, IX, and X. This results in increased PT and PTT, which our patient had. Our patient also had thrombocytopenia, which may also occur in severe cirrhotic liver disease. These changes predispose patients with liver disease to have hemorrhagic complications. Other diseases noted in patients with complicated SOH were hemophilia, disseminated intravascular coagulation, and scurvy [2,3].

The coincidental incident in our patient was the development of SOH postoperatively after cataract surgery. In other reported cases of NTSOH by Atalla et al. patients had sentinel events such as emesis, strangulation, or straining [3]. We cannot rule out that our patient had an unrecognized traumatic event given her alcoholism; however her examination and history were consistent and reliable with family reports. The development of SOH cannot be directly associated with the recent cataract surgery, it is a concern for general ophthalmologists who may see such findings postoperatively. On routine cataract surgery in otherwise healthy patients, medical clearance obtained from the primary care physician may not reveal such findings of liver disease because routine screening does not include obtaining liver function tests. The finding of compression of the globe with initial normal intraocular pressures in our patient also raises the concern that the post-cataract patient may have had hypotony or wound leak early on that may have complicated her post-operative course. The SOH presentation is similar to a retrobulbar hemorrhage, which is another catastrophic hemorrhagic event that may occur during routine ophthalmologic surgery in the compromised patient.

Treatment of NTSOH initially begins with correction of the associated condition. If there is no evidence of optic nerve compromise, such as decreased vision or changes on DFE, the patient can be observed for resolution. If there is some evidence of compromise, such as in our patient who had further decreased vision since initial presentation and development of new fundus changes, surgical drainage is recommended. There are some reports of needle aspiration of the SOH.
but the mainstay of treatment is an orbitotomy with drainage [8,9]. Previously reported cases by Moorthy et al. resolved without invasive treatment [9].

References