Review Article

Subgaleal Haematoma Extending into the Orbit Following Blunt Head Trauma as a Cause of Permanent Blindness: A Case Illustrated Review

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Abstract

Concurrent occurrence of subgaleal haematoma (SGH) and orbital subperiosteal haematoma (OSPH) is an unusual post-traumatic phenomenon. It is especially seen in cases of massive SGH. This report is a review, illustrated with a personal clinical experience, of this clinical disease association. Extensive literature review using the PubMed search engine for all cases of blindness from SGH and OSPH in the English language was conducted for this report. Vision-threatening complications of massive traumatic SGH that is associated with OSPH are rare but well described. The resulting orbital bleed most commonly involves the extraconal orbital roof. It is essentially an orbital epidural haematoma. Its aetiopathogenesis is that of an orbital space occupying lesion (SOL) causing clinical evidence of raised intraorbital pressure (IOP) including chemosis, proptosis, ophthalmoplegia, exposure keratopathy and orbital cellulitis and blindness. The visual impairment is usually temporary and responds well to prompt intraorbital decompression. However, permanent blindness ensues if suboptimal treatment including missed diagnosis and or delayed treatment is offered.

Keywords: Blindness, orbital subperiosteal haematoma, review article, subgaleal haematoma

Introduction

Subgaleal haematoma (SGH) is an extracranial haemorrhage into the soft tissue of the scalp between the galea aponeurotica and the periosteum of the skull—the epicranium. This space is occupied by loose connective tissues which as a continuum make up the traditional fourth of the five layers of the scalp, the loose areolar tissue layer. In addition, and significantly so in the formation of SGH, the space also contains small blood vessels, mainly venous, which drain the scalp.[1,2] Some of these venous channels are actually emissary veins connecting the intracranial dural sinus with superficial scalp veins. SGH results from shearing forces acting on these vessels during trauma.[2-4]

Orbital subperiosteal haematoma (OSPH) is an intraorbital extraconal bleed in the space between the orbital bone and the orbital periosteum.[5] It is most commonly seen in the orbital surface of the frontal bone.[6] It is rare following trauma and results from either rupture of the subperiosteal blood vessels or as an extension of cranial convexital SGH.[2,7]

In the setting of massive post-traumatic SGH, OSPH is usually a high tension lesion causing intraorbital space occupying effects.[8] These include clinical evidence of raised intraorbital pressure (IOP) like proptosis, chemosis and exposure keratopathy. Such cases can and do cause blindness which is permanent sometimes. Hence, although life-threatening and vision-threatening occurrences are unusual complications of SGH, they have been described in the global literature. Hence if orbital involvement, OSPH, in such cases leads to ophthalmoplegia or optic nerve (ON) compromise, prompt surgical decompression should be done.[9,10]

Sometime in the past, we had the benefit of managing such a case of SGH and OSPH causing permanent blindness in a young schoolboy. In this report we present a review of the subject of the aetiopathogenesis of SGH, OSPH, and blindness as a result of OSPH associated with massive SGH. We illustrated this

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review with the report of the clinical course of the index case managed by us.

Blindness from Massive SGH Associated with OSPH

Aclinical case illustration

A 13-year-old schoolboy was admitted into our service via the emergency unit with a 2-week history of progressive diffuse scalp swelling, bilateral proptosis (worse on the right), as well as right-sided blindness of 6 days' duration. He had suffered a trivial fall at home a few days before onset of symptoms. There was a background history suggestive of chronic sinusitis but none of bleeding disorder. He had no fever, seizures, limb weakness or vomiting

He was fully conscious with normal mental status. He had bilateral proptosis worse on the right with severe conjunctival chemosis, bilateral lid oedema and right pan-ophthalmitis [Figure 1]. The left pupil was 4 mm in size and reacted sluggishly to light; the right pupil could not be assessed in view of complicating severe exposure keratopathy. He had no light perception in the right eye but the visual acuity (VA) was 6/9 in the left. There was generalized hypertonia and hyperreflexia with bilateral extensor plantar responses but the muscle power was normal in all the limbs. There were no signs of meningeal irritation. The scalp swelling was diffuse, fluctuant, tender and associated with bilateral parietal bossing [Figure 1]. Test aspiration of the subgaleal collection yielded altered blood.[11] Cranial computed tomography (CT) scanning showed extensive mixed-density subgaleal collection. Same-density collection was noted in the orbits and frontal sinuses [Figure 2].

Figure 1: A patient with massive SGH and OSPH. Note the marked scalp swelling (the biparietal bossing), and bilateral proptosis, worse on the right. The right eye has severe chemosis, and severe exposure keratopathy

He was optimized for surgery, and had joint neurosurgery and otorhinolaryngology operative interventions. Via bilateral linear frontal and parietal stab incisions, about 250 ml of altered acute/subacute subgaleal blood was evacuated. Sub-brow incisions were also made bilaterally to access the orbits. Ten and five millilitres of blood clots were evacuated from the right and left orbital extraconal regions respectively [Figure 3]. The right sub-brow incision was extended inferomedially to allow access for an ipsilateral external fronto-ethmoidectomy [Figure 3b]. Blood clots were also found in the frontal sinuses. A fronto-nasal catheter was left in place for 2 weeks to allow for development of a new fronto-nasal drainage track.

He had no abnormal intra- or post-operative event. There was progressive resolution of the symptoms but the right-sided blindness persisted. Three weeks after admission, he was discharged from the hospital with good cosmetic outcome and improvements of the inflammatory changes involving the right eye [Figure 4].

Discussion

The subgaleal space is a loose tissue space in the scalp, especially in children beyond childhood and in adolescents. It contains loose connective tissue and small blood vessels, the latter mainly venous. Some of these are emissary veins crossing the space to connect the dural sinuses with the superficial scalp

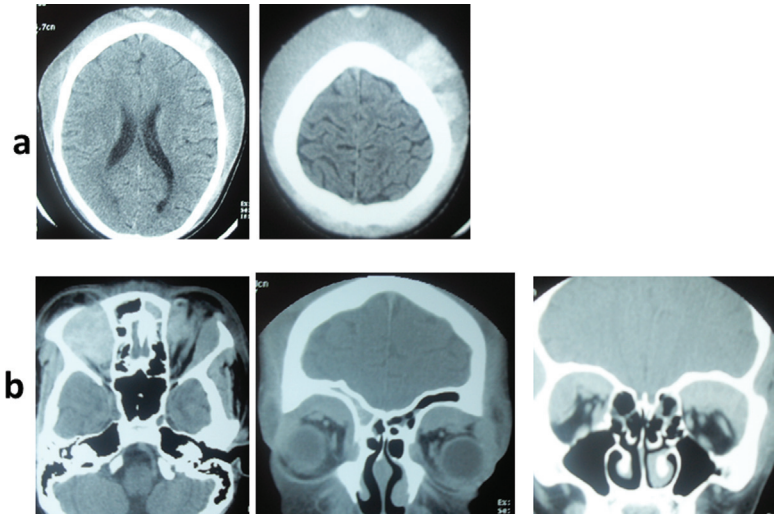

Figure 2: Cranial CT images of the massive SGH and OSPH: (a) axial CT image showing the massive mixed-density subgaleal collection; (b) axial and coronal images showing the intraorbital bleed. The lesions are extraconal and involve the frontal orbital periosteal spaces. There is associated involvement of the right frontal sinus

veins.[1,12,13] The subgaleal space is a very extensive area spanning all of the cranial convexity. It is delimited circumferentially by the attachments of the galea aponeurotica: anteriorly to the bony orbital ridge bilaterally; laterally to the zygomatic arch and the auricular muscles on each side; and posteriorly to the nuchal line.[14]

Subgaleal haematoma

SGH occurs when shearing forces applied to the scalp violate the blood vessels, mainly venous, criss-crossing this capacious subgaleal space. The causes of this type of bleed include trauma. This includes a few usually minor incidents such as falls from the bed, especially in infants, or occasionally more severe trauma as in road crashes, either motor-vehicular, motor-cycle or even bicycle.[7,11] Other causative incidents include hair combing/pulling and hair braiding. There are also spontaneous cases from coagulopathies such as in the von Williebrand's disease, the sickle cell disease and even connective tissue disorders.[5,7,14,15] The resulting SGH is usually localized; hence it is hardly a subject of scientific write-ups. However, it can become massive enough to come to clinical attention, causing problems such as compromise of the blood volume in the younger infants.[1] Extending beyond the galeal attachment laterally at the zygomatic arch, it has also been known to involve the head-and-neck soft tissue space causing airway obstruction and skin necrosis.[16] The largest clinical series of post-traumatic SGH was reported in Ibadan by Adeloye and Odeku.[17] Most followed head trauma but appeared to be largely asymptomatic. They were therefore managed, mainly, with 'masterly inactivity'.

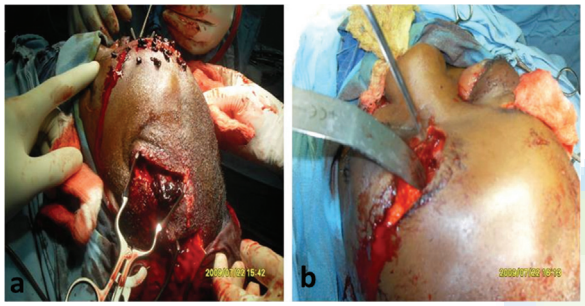

Figure 3: Surgical evacuation of the SGH/OSPH. (a) Stab wounds in the scalp to deliver the SGH, and sub-brow incisions to evacuate the OSPH. The two haematoma are seen to be clotted blood, would not be deliverable in the orbital subperiosteal space with mere needle aspiration. (b) The right sub-brow incision was extended downward for a frontoethmoidectomy to deliver the associated clots in the frontal sinus

Figure 4: Blindness from massive SGH and OSPH. The before (a) and after (b) clinical photographs of the patient. There is good cosmetic outcome, cranial and orbital. The proptosis was much ameliorated on both sides

Orbital subperiosteal haematoma

Orbital haematomas are classified as intraorbital (intraconal) or subperiosteal (extraconal).[3,18] The former follows ophthalmic procedures in the main. They occasionally form in the setting of systemic coagulation disorders. OSPH on the other hand can be traumatic, congestive, systemic and spontaneous in origin.[2,5] Again, the most common cause of OSPH can be termed as a complication of ophthalmic procedures. They are rare following trauma and can follow minor accidents; or they can be associated with brain injuries like extradural haematomas of the frontobasal region.[3,7,19-23] More interestingly, OSPH can follow a post-traumatic SGH.[10,13] The orbital portion of the frontal bone is the most frequent site of OSPH. This is because it provides the greatest surface area of the orbit.[6] Also, it is concave and has loose attachments of the orbital periosteum unlike the other surfaces of the orbital cavity. In these other orbital surfaces apart from the frontal bone portion, the periosteum is not easily peeled off the bone due to its attachment at the orbital bony suture lines.[13,24] We are aware of only one case in the literature where the post-traumatic OSPH originated from the medial orbital wall towards the apex and not from the orbital roof.[25]

Combined subgaleal haematoma and orbital subperiosteal haematoma

Combination of SGH and OSPH is a rare occurrence following craniofacial trauma. Each entity is more wont to occur singly. Pope-Pegram and Hamill reported one combined case in the year 1986. A comprehensive literature review by them found nine other cases of isolated OSPH and only one of SGH and OSPH antedating their own report. OSPH can form from rupture of the subperiosteal blood vessels, during trauma, but the orbital subperiosteal space is largely avascular. Hence, when associated with SGH, OSPH is usually deemed to be due to the leakage caused by high pressure of massive subgaleal bleed into the orbital periosteal space through the galeal attachment to the orbital ridge.[1,15,26,27]

SGH plus OSPH plus blindness

Although not so common, vision-threatening complications of SGH are well described.[10] Visual impairment from SGH and OSPH arises from two main causes. One is compressive ON neuropathy from raised IOP when significant volume of

bleed occupies the orbital cavity.[28] The other is from exposure keratopathy, or orbital cellulitis, complicating prolonged, unmitigated proptosis and ophthalmoplegia that sometimes accompany this condition.[7,10,15,29,30] The risk of compressive neuropathy of ON is particularly higher in bilateral OSPH in the setting of massive SGH, as in the case illustration here.[7] It can also be due to vascular compromise to the central retinal artery and vein.[14]

Although the visual impairment recovers in many cases following prompt evacuation of the OSPH, thereby bringing down the raised IOP,[4,10,15] many not-so-fortunate cases suffer permanent blindness. The case illustrated here is one of many such cases.[7,12,13,31] Herein lies the need for great clinical vigilance to make precise diagnosis and institute prompt restorative treatment in cases of vision-threatening SGH and OSPH. In fact, many cases in the literature suffer from the initial non-aggressive diagnosis and treatment of their condition by unwary physicians. Some were seen, and discharged home by the physicians on many occasions as insignificant, minor complications of head trauma before increasing proptosis and impending visual decline forced a change in the line of management.[12,29,32]

There are several differential diagnoses of OSPH. These include carotico-cavernous fistula, cavernous sinus throm bosis, orbital subperiosteal abscess, paranasal sinus mucoc oeles, orbital roof fractures, bleeding disorders, orbital pseudotumours or such tumours as rhabdomyosarcoma and lymphangioma.[2,4,30,33] Most can, however, be excluded based on a detailed clinical history, physicals and appropriate laboratory and radiological studies.

In the setting of trauma and associated boggy, diffuse scalp swelling in keeping with SGH, visual complaints including proptosis and diplopia should raise the suspicion of vision-threatening OSPH.[13] Clinical examination showing the scalp swelling crossing the suture lines would further suggest the presence of an SGH. Ocular examination should screen for chemosis, lid retraction, proptosis, ophthalmoplegia, exposure keratopathy and also ascertain VA. Ecchymosis, the hallmark of intraorbital/retroorbital haemorrhage is usually absent in the subperiosteal bleed.[14] The proptosis is usually one of downward, outward displacement and restriction of upward gaze. In short, the initial ophthalmic examination includes an eight-point eye exam, and forced duction test. Also needed are baseline and serial VA, tonometry for IOP, indirect ophthalmoscopy and exophthalmometry. Serial evaluations are for cases being managed non-operatively. Laboratory investigations are complete blood count and differentials, and the clotting profiles, and haemoglobin electrophoresis, as the case may be.[2,10,13,14]

Supportive/confirmatory radiologic investigations include orbital echography and CT scanning. The CT should screen the cranium/brain, paranasal sinuses, as well as the orbit for associated (or predisposing) conditions. Diagnosis is usually clinched with the CT in SGH and OSPH [Figure 1]. Magnetic resonance imaging is sometimes used, but this is usually when other conditions (see differentials) are being screened for.[34]

Treatment of SGH and OSPH

Recommendations for the treatment of these disease conditions can only be on case-by-case basis. Pope-Pegram and Hamill[4] in 1986 outlined an algorithm to this end which must still be relevant today. Isolated SGH uncomplicated can be managed expectantly, as is isolated OSPH. Many resolve spontaneously on this line of masterly inactivity.[17] This is especially true of cases emanating not from trauma, or from only minor incidents.[1,24] The same treatment line is employed for many cases of SGH in association with OSPH. But these are usually the localized cases (say, frontal area only) of SGH with only minimal-volume, non-proptosis-inducing, vision-sparing OSPH. Surgical decomp ression becomes necessary in symptomatic cases, particularly in vision-threatening OSPH. As a matter of fact, the case reported by Pope-Pegram and Hamill[4] was rushed to surgery in the wake of continuing visual decline on a conservative line of care. The key to making this important therapeutic decision is in serial clinical and ancillary evaluations as detailed above. Any impending neurological or visual deterioration should alert the attending team to the need for orbital (and cranial) decompression.[10,13]

Meanwhile, in the non-operated cases, medical treatment to try to lower IOP usually involves either topical agents such as timolol maleate, or carbonic anhydrase inhibitors, such as acetazolamide, intravenously or per oram.

Surgical intervention may be no more than needle aspiration of the OSPH.[13,30] Usually some 5 ml or so of liquefying blood is all that is evacuated to bring the raised IOP down, and also cause resolution of the proptosis. This is however a blind procedure, and may not work for clotted blood. In that case, an orbitotomy, more commonly via a brow incision, is employed. Or even a frontoethmoidal exposure as employed in the case illustrated here.[7] The SGH component is treated on its own merit.[17,32] In cases like the one illustrated, there may be the need to make separate scalp stab incisions to effect its effectual drainage. More extensive dissections like the transcoronal or transfrontal, are also employed when necessary.[7]

Conclusions

Vision-threatening complications of massive post-traumatic SGH associated with OSPH are rare but well described. The resulting orbital extraconal bleed is usually a space occupying lesion (SOL) causing clinical evidence of raised IOP including chemosis, proptosis, ophthalmoplegia, exposure keratopathy and orbital cellulitis and blindness. The visual impairment is usually temporary and responds well to prompt orbital decompression. Permanent blindness, however ensues, if suboptimal treatment including missed diagnosis and or delayed treatment is offered.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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