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Case Report

Extension of tardive subgaleal hematoma into the orbit causing blindness of both eyes: a case report and literature review

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Abstract: Subgaleal hematomas (SGHs) are not uncommon. Because the subgaleal space has no anatomical boundaries, SGHs usually involve a large space; however, SGHs are typically limited to the parietal region. Cases of SGHs breaking through the fascia at the eyebrow and entering the orbit are relatively rare, especially with respect to those involving the bilateral orbits. This study reported a rare case of tardive orbital hematoma after SGH. A male patient, aged 16, experienced SGH, exophthalmos, and decreased vision within four days after head trauma, and vision was lost after seven days. Although SGH drainage surgery and orbital hematoma aspiration were performed, the patient's vision was not restored. After reviewing the relevant literature, we believe that although SGH is difficult to enter the orbit, there are opportunities for SGHs to break through the orbit and cause blindness. Thus, close observation during the follow-up period is critical. When an SGH involves the orbit and the orbital involvement leads to ophthalmoplegia and/or optic nerve damage, surgical decompression is necessary.

Keywords: Subgaleal hematoma, orbit, extend, blindness

Introduction

Subgaleal hematoma (SGH) is a common clinical disease, largely because the subgaleal space is relatively loosely defined. SGH is most commonly caused by hemorrhage from the rupture of the vein traveling in the subgaleal space [1, 2]. The vast majority of SGHs are gradually absorbed. For cases exhibiting difficulties in absorption, puncture with aspiration or incision followed by drainage can achieve satisfactory outcomes [3]. However, in rare cases, the SGH develops in the direction of the orbit and enters the orbit, causing vision disorders [4, 5]. To date, cases of SGH extending into orbit have been poorly understood, primarily because few relevant reports are available. Herein, one case of a rare SGH is reported, with the SGH breaking through into the fascia at the eyebrow and entering the orbit on the 4th day after the development of the SGH, causing visual impairment and resulting in blindness on the 7th day. The present study reports the experiences of and lessons learned from the treatment of this case as well as a review of the relevant literature.

Case report

A male patient, aged 16, suffered a head impact injury that caused extensive SGH with no other injuries and symptoms. Head computed tomography (CT) revealed no obvious axial intracranial abnormality at the eye level but revealed swelling of the frontotemporal parietooccipital scalp, which was diagnosed as an extensive SGH (**Figure 1**). Conservative symptomatic treatment was provided in a local hospital. On the 4th day after the injury, the patient gradually experienced blurred vision and eye proptosis, and vision was completely lost by the 7th day, with the eyeballs fixed in position. The patient was then transferred to the Daqing Oil Field General Hospital for treatment. The physical examination revealed the following: both eye balls were protrusive and fixed, with conjunctival edema and hyperemia; the bilateral pupils had a diameter of approximately 5.5 mm; and direct and indirect light reflexes were absent (**Figure 2A**). Extensive swelling was observed in the scalp of the occipital, parietal, temporal, and frontal regions; pulse movement was felt to

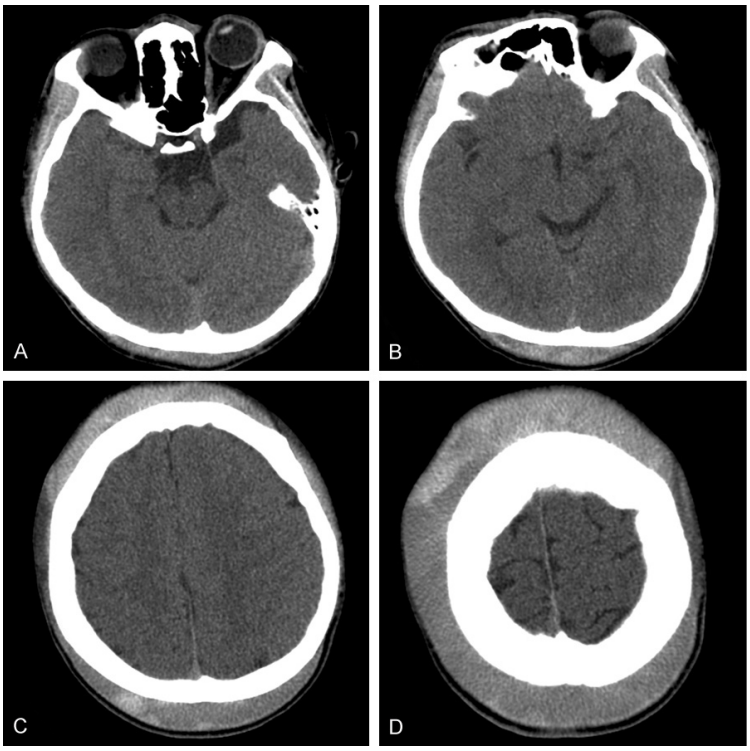

Figure 1. Head CT imaging at the time of injury. A, B: The head CT revealed no obvious axial intracranial abnormality at the eye level. C, D: Swelling of the frontotemporal parietooccipital scalp was observed with an extensive SGH.

the touch; and the temperature of the scalp was slightly elevated. Routine blood tests, routine coagulation tests, and routine clotting factor tests were all normal. The eye CT after admission revealed hemorrhage above both orbit as well as the inner and outer spaces of the muscle cone; the hemorrhage was most serious in the outer upper region, resulting in a curved inward proptosis and shifting of the extraocular muscle under the pressure. The SGH was connected to the orbital hematoma, and the eyeballs were displaced forward under the pressure; no obvious intracranial abnormality was observed (**Figure 2B-D**). Head-enhanced MRI revealed that the equal signal of the hemorrhage in the orbit behind the eyeballs occupied most of the space of the orbit and pushed the eyeballs forward; the extensive SGH in the frontotemporal parietooccipital region exhibited equal signals, and the hemorrhage extended into the orbit behind the eyeballs (**Figure 3**). The diagnosis after admission was extensive SGH in the frontotemporal parietal occipital region and bilateral orbital hematoma. Considering the patient's severe ocular symptoms, emergency puncture for bilateral orbital hematoma was performed and approximately 10 ml of blood with no coagulation was drawn. After the hematoma was extracted, the bilateral eyeballs were depressed, reducing the tension. Subsequently, SGH subcutaneous drainage was performed, but the patient's vision did not recover after the treatment. The follow-up head CT after one month revealed that the SGH and orbital hematoma had been gradually absorbed, but the vision never recovered. The follow-up after three months did not reveal any restoration of vision.

Discussion

The subgaleal space is located between the periosteum and the epicranial galea and comprises loose connective tissue; vessels connecting the scalp vein and skull diploe vein as well as the intracranial venous sinus are located within this space. External shear force during trauma may result in the rupture of the vessels, causing a large amount of blood to flow into the subgaleal space to form an SGH. The subgaleal space is not bounded by suture lines; therefore, an SGH can involve massive swelling of the entire scalp [6, 7]. Most SGHs do not require any treatment and can be spontaneously absorbed or cured after aspiration. Spontaneous resolution may occur within 1-3 weeks, or needle aspiration and compressive dressing can be applied to relieve local pressure and to promote absorption [8]. Because the subgaleal space is bounded by the attachment of the galea to the supraorbital ridges, zygomatic arches, auricular muscles, tissues of the posterior triangle, and the nuchal ridge, although SGHs can involve a large space, they are generally limited to the parietal bone of the head due to the difficulty in breaking through the attachment of the eyebrow fascia. However, in extremely rare cases, SGHs can also break the attachment of the fascia and eyebrow, thus entering the orbit to cause eye disorders. Even if SGHs involve the eyes, they are typically uni-

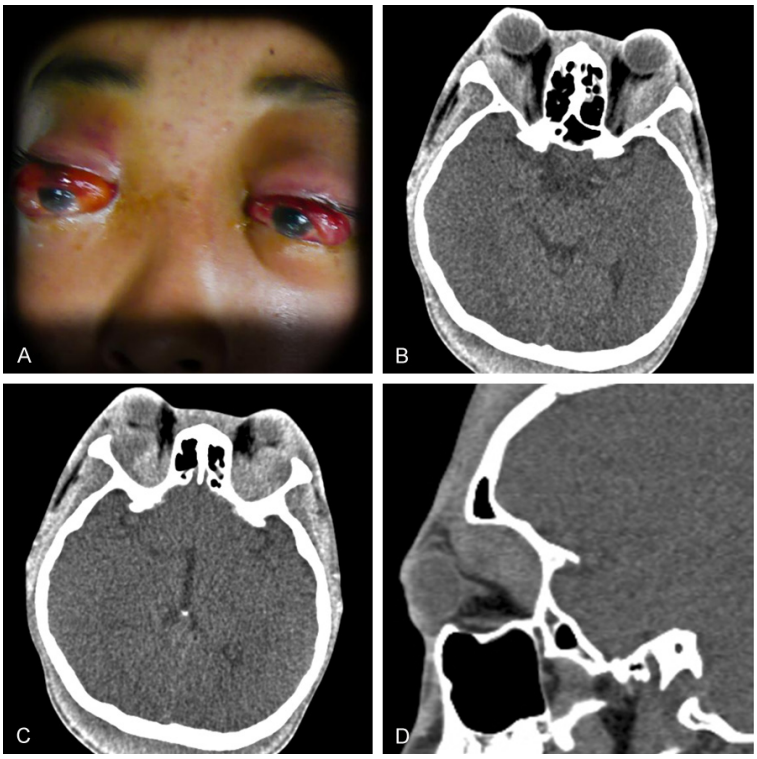

Figure 2. The image on the 4th day after the injury. A: The image demonstrates that both eyeballs were protrusive and fixed, with conjunctival edema and hyperemia. B, C: Hemorrhage above both orbits as well as within the inner and outer spaces of the muscle cone was observed; hemorrhage was most serious in the outer upper region, resulting in a curved inward proptosis, and the extraocular muscle was shifted under the pressure. D: The SGH was connected to the orbital hematoma, and the eyeballs were displaced forward under the pressure.

lateral; cases involving both eyes are extremely rare [9]. This study reported a rare case of SGH after head trauma. The SGH was initially limited to the parietal bone of the head. After four days, the SGH entered the bilateral orbits, causing vision loss. Although aggressive treatment was provided, vision could not be restored. The present study reported the experiences of and lesson learned from the treatment of this case.

Generally, the orbital septa can protect against the extension of an SGH into the orbit. However, in the lateral canthal region, there is a potential gap between the orbital septa and the lateral horn of the levator palpebrae aponeurosis. This gap can allow the orbital extension of the SGH under an abnormally high pressure [10]. Once the hematoma enters the orbit, it can more easily spread in the orbit, largely because most of the superior orbital wall is constituted by the frontal bone, with no suture separation; thus, it is easy for a hematoma to diffuse under the periosteum [11, 12]. The cases of hemorrhage diffusion into the orbit after SGH that have been reported in the literature typically occurred in children and adolescents [13]. In adolescents, the fascia tissue attached to eyebrow is immature; when the pressure of the SGH hematoma cavity is high, the blood clots can more easily separate the fascia tissue at this region to enter the orbit [14]. Literature reports also suggest that if an SGH is complicated by subperiosteal hematomas, the SGH can easily enter the orbit because the hematoma can easily spread along the periosteum [15]. Another reason for the higher likelihood of hemorrhage diffusion after SGH into the orbit in children and adolescents is that these cases are likely to be complicated by coagulopathy; thus, minor head injury could cause severe SGH and potential spread into the orbit [16]. For example, Natarajan et al reported one case of a 6-year-old boy with factor XIII deficiencies who developed severe SGH after minor trauma involving the orbit; fortunately, his vision was not affected. After the application of supplemental coagulation factors and scalp drainage surgery, satisfactory recovery was achieved [17]. In some adults, long-term oral administration of anti-platelet aggregation drugs may also induce the SGH to spread toward the rear of the orbit. For example, in 2007, Chotirmall et al reported one case of posttraumatic SGH complicated by hematoma orbital extension in a 78-year-old female with a long-term medication history of oral clopidogrel [15]. For the SGH case reported in the present study, the hemorrhage was very severe, with bilateral orbital involvement. The patient had no history of oral anti-platelet drugs, and the examinations of coagulation function and coagulation factors revealed no abnormalities. Nevertheless, this study recommends examinations of coagulation function in young patients.

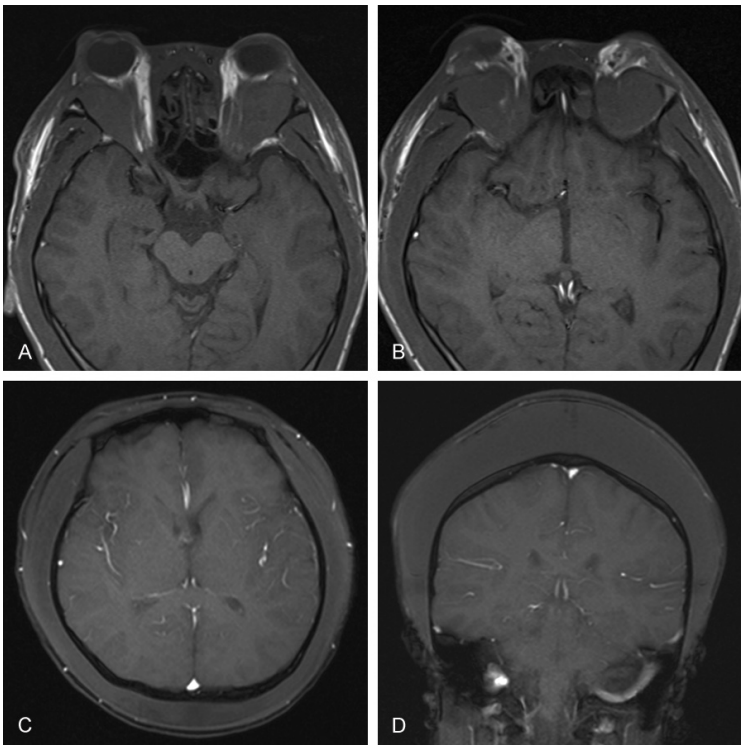

Figure 3. MRI imaging when the symptoms of eye disorder occurred. A, B: The enhanced MRI revealed that the equal signals of the hemorrhage in the orbit behind the eyeballs occupied most space of the orbit and pushed the eyeballs forward. C, D: Extensive SGH in the frontotemporal parietooccipital region was observed with equal signals.

In addition to the potential presence of primary coagulation disorders, the development of an SGH may also cause a secondary coagulation disorder, leading to the difficulty in controlling SGH; the continuously increasing hemorrhage will generate high pressure that will push the SGH into the orbit. For example, in 2005, Fujisawa et al reported a case of SGH immediately after a trauma in a 12-year-old girl characterized by marked exophthalmos and corneal ulceration in the left eye on the 8th day; drainage was performed for the SGH, and aspiration was performed for the orbital hematoma, resulting in satisfactory postoperative recovery. Meanwhile, a chemical analysis of the SGH revealed that it contained extremely low levels of fibrinogen and platelets and high levels of fibrinogen and fibrin degradation products, suggesting that secondary fibrinolysis had occurred in the subgaleal space [18].

Once the SGH involves the eyes, urgent treatment is needed to avoid visual impairment. Puncture aspiration treatment can be applied first. If the result is poor, drainage surgery is needed. For example, in 1986, Pope-Pegram et al reported a case of SGH after trauma in a 6-year-old girl. The patient's left eye was involved, leading to left exophthalmos and decreased visual acuity; an emergency subbrow incision was conducted through the periosteum to release a gush of red-brown fluid, with the immediate resolution of the proptosis and a decrease in the intraocular pressure. An SGH communicating with the orbital subperiosteum was intraoperatively observed, and drainage was placed after the surgery. Due to the timely treatment, the patient recovered well [5]. In addition to subbrow incision, because the rear of the orbital periosteum and the subgaleal space are connected, the treatment of simple SGH drainage can also be applied. For example, in 1988, Lee et al reported one case of SGH in a 13-year-old girl; the patient developed proptosis with ocular pain in one day after the development of SGH that led to vision loss. CT revealed an extensive SGH and bilateral orbital hematomas; a small incision was made in the left temporal region, and the ocular symptoms were relieved after the drainage [9].

The most important factor affecting the prognosis of SGH involving orbit is the timing of the treatment. The cases reported above obtained satisfactory outcomes because of the timely treatment. In the case in this study, the orbital hematoma complicated with visual impairment developed on the 4th day after the development of the SGH, and this orbital hematoma was tardive. This orbital hematoma was not treated soon after its occurrence. The orbital puncture was not conducted until the 7th day, on which the vision was lost; the timing was thus too late. Although the hematoma was finally absorbed, vision failed to be restored. This case thus teaches a valuable less: although tardive cases of hematoma behind the orbit after SGH are rare, attention is nonetheless needed. For example, in 2005, Fujisawa et al reported a case of SGH after the trauma in a 12-year-old

girl in whom a left orbital hematoma occurred on the 8th day after trauma [18]. In 1995, Pomeranz et al reported one case of SGH complicated with heterozygous factor VII deficiency in a 6-year-old girl; SGH developed on the 6th day after the trauma, and tardive orbital hematoma developed on the 3rd day after the occurrence of SGH. Careful follow-up of patients with SGH is believed to be necessary because the development of proptosis may be delayed; furthermore, nonsteroidal anti-inflammatory drugs (NSAIDs) should not be used because of their impact on platelet function [13].

Cases of SGH involving the orbit are extremely rare and have thus been largely ignored in the clinical treatment of SGH. Based on the experience of the present case, although it is difficult for SGH to enter the orbit, there remains chance for the SGH to break through the orbit and cause blindness. Therefore, close observation during follow-up is critical, and routine eye examinations during the treatment of SGH are necessary. When the SGH involves the orbit and the orbital involvement leads to ophthalmoplegia and/or optic nerve damage, surgical decompression is necessary.

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Disclosure of conflict of interest

None.

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References

[1] Vu TT, Guerrera MF, Hamburger EK and Klein BL. Subgaleal hematoma from hair braiding: case report and literature review. Pediatr Emerg Care 2004; 20: 821-823.

[2] Smith SA, Jett PL, Jacobson SL, Binder ND, Kuforiji TA, Gilhooly JT, Piatt JH Jr, Pillers DA, Reynolds JW and Benda GI. Subgaleal hematoma: the need for increased awareness of risk. J Fam Pract 1995; 41: 569-574.

[3] Strowitzki M, Eymann R, Schleifer J and Steudel WI. Vertex epidural hematoma with communicating bifrontal subgaleal hematomas treated by percutaneous needle aspiration. Pediatr Neurosurg 2001; 35: 1-4.

[4] Wolter JR, Vanderveen GJ and Wacksman RL. Posttraumatic subgaleal hematoma extending into the orbit as a cause of permanent blindness. J Pediatr Ophthalmol Strabismus 1978; 15: 151-153.

[5] Pope-Pegram LD and Hamill MB. Posttraumatic subgaleal hematoma with subperiosteal orbital extension. Surv Ophthalmol 1986; 30: 258-262.

[6] Panigrahi S, Mishra SS, Das S and Patra SK. Large subgaleal hematoma as a presentation of parahemophilia. J Neurosci Rural Pract 2013; 4: 240-242.

[7] Kichari JR and Gielkens H. Massive traumatic subgaleal haematoma. Emerg Med J 2013; 30: 344.

[8] Anton J, Pineda V, Martin C, Artigas J and Rivera J. Posttraumatic subgaleal hematoma: a case report and review of the literature. Pediatr Emerg Care 1999; 15: 347-349.

[9] Lee KS, Bae HG and Yun IG. Bilateral proptosis from a subgaleal hematoma. Case report. J Neurosurg 1988; 69: 770-771.

[10] Prakash S. Bilateral proptosis from a subgaleal hematoma. J Neurosurg 1990; 72: 835.

[11] Pasaoglu A, Orhon C, Akdemir H, Uzunoglu H, Oktem S and Yardim S. Subperiosteal intraorbital haematoma following minor head trauma. Case report. Acta Neurochir (Wien) 1989; 97: 83-85.

[12] Atalla ML, McNab AA, Sullivan TJ and Sloan B. Nontraumatic subperiosteal orbital hemorrhage. Ophthalmology 2001; 108: 183-189.

[13] Pomeranz AJ, Ruttum MS and Harris GJ. Subgaleal hematoma with delayed proptosis and corneal ulceration. Ann Emerg Med 1995; 26: 752-754.

[14] Takano I, Suzuki K, Sugiura Y, Suzuki R, Nagaishi M, Tanaka Y and Hyodo A. [A Case of Subgaleal Hematoma with Exophthalmos and Diplopia]. No Shinkei Geka 2015; 43: 727-731.

[15] Chotirmall SH, Pearson E, Saad AZ, Moore A, Kneafsey B and Donegan CF. Posttraumatic subgaleal hematoma with orbital extension associated with clopidogrel usage in an elderly patient: case report. J Am Geriatr Soc 2007; 55: 135-136.

[16] Kim D and Taragin B. Subgaleal hematoma presenting as a manifestation of Factor XIII deficiency. Pediatr Radiol 2009; 39: 622-624.

[17] Natarajan MS, Prabhu K, Braganza A and Chacko AG. Posttraumatic subgaleal and orbital hematoma due to factor XIII deficiency. J Neurosurg Pediatr 2011; 7: 213-217.

[18] Fujisawa H, Yonaha H, Oka Y, Uehara M, Nagata Y, Kajiwara K, Fujii M, Kato S, Akimura T and Suzuki M. A marked exophthalmos and corneal ulceration caused by delayed massive expansion of a subgaleal hematoma. Childs Nerv Syst 2005; 21: 489-492.