Open Journal of
Clinical & Medical
Case Reports

Volume 7 (2021)
Issue 02

ISSN: 2379-1039

Massive spontaneous subgaleal hematoma with progressive exophthalmoses and monocular evisceration

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Abstract

Background: Subgaleal hematoma (SGH) is a rare condition, most frequently observed in neonates as a complication of assisted instrumental delivery. However, beyond this age, the reported cases in literature are typically resulted from significant blunt scalp trauma, with or without associated skull fracture.

Case presentation: A 14-year-old male with no prior or present history of trauma, in the absence of any apparent underlying hematological or anatomical abnormality presented with a 2-week history of progressive head swelling and protrusion of both eyes with visual impairment. There was no history of convulsion or headaches. He denied any history of spontaneous bleeding. His international normalized ratio (1.02), clotting factor VII (94%) and clotting factor IX (65%) were normal. Brain computerized tomography scan showed massive circumferential chronic hematoma with areas of acute hematoma. No intracranial hemorrhage was observed. There were no intra or extra cranial vascular abnormalities on imaging.

Conclusion: Spontaneous SGH is uncommon in middle childhood period and underlying hematologic or vascular abnormalities may be covert. Multidisciplinary approach is the standard of care and is associated with better outcomes.

Keywords

Non-traumatic; subgaleal Hematoma; exophthalmoses; bleeding diathesis

Abbreviations

SGH: Subgaleal hematoma; CT: Computed tomography

Introduction

An injury of the emissary veins causes bleeding to the subgaleal space and is considered a cause of the formation of the subgaleal hematoma (SGH). The bleeding is normally below the galea aponeurotica; hence it can cross suture line [1]. In a newborn this space can contain up to 260 ml, which is almost all blood of a newborn (80-90 ml/kg). It explains therefore why bleeding to this space is perceived as particularly dangerous and why it can lead to death in 25% of cases [2].

Subgaleal hematoma (SGH) is usually associated with head trauma. The galea in a newborn infant can be pulled in a vertical direction and slipped in a tangential direction when exposed to an external force, like assisted labor or obstructed labor [3]. A small number of cases have been described in older children, occasionally as a result of minor head trauma such as hair braiding or hair pulling [4]. Non-traumatic or spontaneous SGH is rare, but may occur due to a ruptured aneurysm, failure of arteriovenous malformation, arteriovenous fistula of the scalp or coagulopathy [5]. This report describes a case of massive spontaneous SGH with its sequel due to delayed presentation and intervention.

Case Presentation

A 14-year-old male was referred to us from a regional referral hospital in Eastern Uganda with a 2-week history of sudden onset of head swelling that progressed hastily with no prior history of trauma to the head. One-week after the onset of the head swelling, his mother noted protrusion of the left eye followed shortly after a few days with the right eye. There was also loss of vision in both eyes but no history of convulsion or fever. His mother reported that at age of 7 years, her boy (the patient) developed spontaneous multiple nodular swellings of his limbs. These swellings were surgically excised and he had prolonged bleeding following the procedure. The child had also history of prolonged bleeding after minor injuries. The boy was the second born among six girls. The sixth born had similar occasions of bleeding after minor injuries or procedures. However, he did not report any history of spontaneous nose bleeding or bleeding into his joints or skin.

On examination, he was fully conscious and had grossly circumferentially but irregularly swollen head with blood pressure of 95/70 mmHg, which was normal for his age. Ocular examination showed both eyelids were edematous with bilateral conjunctival chemosis and the right cornea exposed and dry, the left cornea exposed and melted. The right eye had no perception to light but the left eye had perception of light only from half a meter. The right pupil was reactive to light but not the left. The exophthalmoses resulted in to exposure keratopathy and left eye conjunctival chemosis and corneal melting. Vitamin and antibiotic ointments were used to preserve the cornea of both eyes however it was futile to the left eye. There was no neck swelling. He had clear chest. His pulse rate was 98/min and his respiratory rate was 15/min. In cardiovascular examination, first and second heart sounds were clearly appreciated with no murmur or gallop appreciated. His abdomen was soft and non-tender with no palpable organomegaly. He had non-edematous extremities. He had no skin petechial, purpura or ecchymosis appreciated.

Laboratory investigations and brain computed tomography (CT) scan was done. The initial hemoglobin was 10 g/dl (reference range: 11.2-16 g/dl) and platelets 232 X 10^3/ul (reference range: 150-450 X 10^3/ul). All his renal function, liver function test and electrolytes were normal. Even, his coagulation profile was normal consistently. The bleeding time was 2 mins and 26 secs (reference range: 2-5 mins) by Duke's method. The clotting time was 4 mins and 13 secs (reference range: 5-10 mins) by Lee and white method. The prothrombin time was 13 secs (normal range 11-16 sec), activated partial thromboplastin time was 30 secs (36-40 sec) and international normalized ratio was 1.02. Clotting Factor VIII was 94% (normal range 55-145%) and Factor IX was 65% (normal range 70-140%). The hematologists were consulted and it was deduced that hemophilia A and B was unlikely in the boy. Blood coagulation factors V, VII, X, XI, and XII were not done due to lack of resource in our setting. Aspiration of the fluid was done for histopathology and cytopathology exams. The result showed hemorrhage with no cell seen but cytomorphologic features of hematoma.

Contrasted brain CT with axial cuts and coronal reconstruction showed massive fluid collection involving the entire scalp with multiple non-enhancing hyper dense (75HU) contents within it. The swelling extended to bilateral orbits and with resultant bilateral proptosis. There was no fracture of the calvarium and the cerebral hemisphere, brain stem and cerebellum demonstrated normal attenuation without any focal abnormality. The basal ganglia, internal capsule, corpus callosum and thalamus appear normal. There was no abnormal parenchymal or dural enhancement. The cerebral ventricles were normal sized and symmetrically arranged with no sign of increased intracranial pressure. The interhemispheric fissure was centered midline and the cisternal space appeared normal. The sellar, the pituitary and the parasellar structures were normal. The cerebellopontine angle areas were normal (Figure 1).

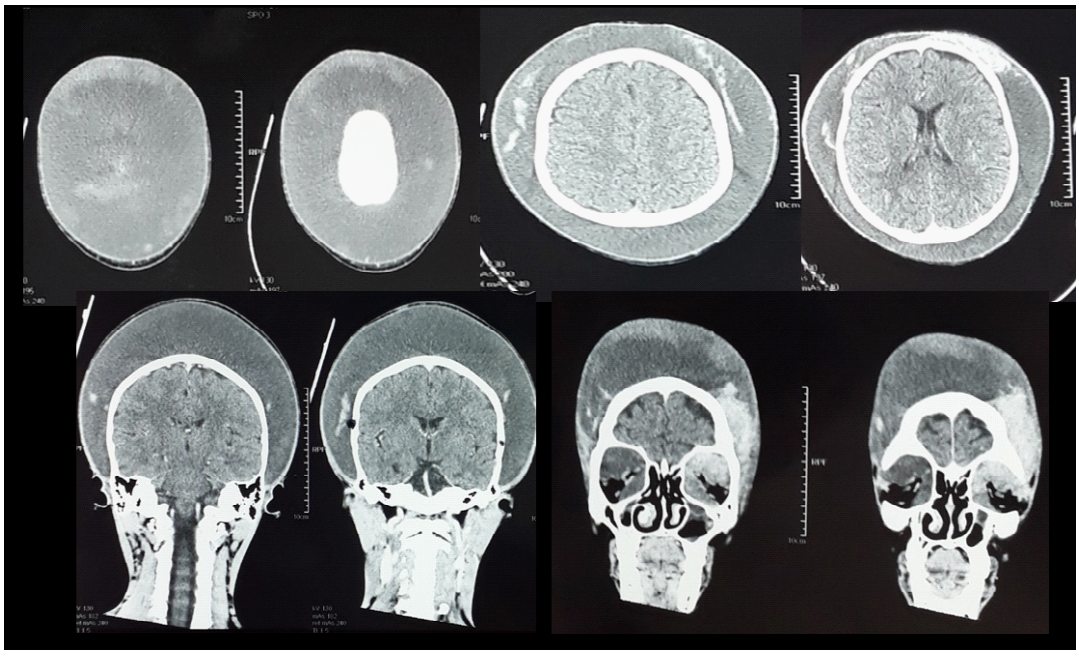

Figure 1: Enhanced CT scan of the brain on axial (first row) and coronal (second row) planes demonstrates massive subgaleal fluid in the entire scalp with multiple hyper dense masses (chronic clots), extending to bilateral orbits with resultant bilateral proptosis.

After admission, proptosis of the left eye worsened and the cornea completely melted then the whole content of the orbital cavity protruded and evisceration of the left eye done by ophthalmologist. He was managed conservatively and his hematoma resolved progressively over weeks and the right eye's corneal erosion gradually disappeared and visual acuity was restored. Thirty-two days after admission, the patient was discharged without any neurological deficits and was referred to hematology clinic for further work up and follows up.

Discussion

Spontaneous SGH is a very rare entity. Sometimes causative associations have been proposed, for instance, aneurysms of the superficial temporal artery, scalp arteriovenous fistula, and coagulation disorders [1,6]. None of the above proposed anatomic and functional causes could be determined from the patient.

The pathogenesis of the extensive hematoma in the present case was puzzling, especially in the absence of trauma. It was originally thought that some form of bleeding diathesis might be responsible, but this theory was challenged when the preliminary screening for the patient's hemostatic status were done. The functional tests and blood coagulation factors V, VII, X, XI, and XII were missing. Moreover, the mother's report was compelling. SGH is sometimes associated with coagulation disorders. One group reported a von Willebrand disease as a cause of SGH [7]. Therefore, coagulation disorders are not completely ruled out for the development of SGH in this. It had been reported in children with vitamin K deficiency [8], hemophilia [9], factor XIII deficiency [7] and von Willebrand disease [10] which can be treated by adequate replacement therapy. The child didn't bleed much during evisceration of his left eye and his blood pressure was consistently normal.

The resolution of SGHs does not generally require intervention, and aspiration of the hematoma if its extensive as in this case, alleviates symptoms briefly, but does not reduce the recovery time [11,12]. No trial of intervention was done for fear of excessive bleeding since no apparent cause could be established.

Our patient may raise awareness of unexpected complications of delayed exophthalmos and subsequent corneal ulceration. So far, there have been only a few reports of delayed exophthalmos following SGH and these cases resulted from blunt forehead trauma [13,14]. In one case, permanent blindness of the involved eye was observed 6 days after the injury [14]. Our patient developed bilateral blindness after two weeks of the onset of the head swelling. And a week later the left eye's orbital contents protruded and the ophthalmologist decided to remove the whole eye. Another report showed that the frontal bone under the eyebrow is one of the weakest points of the attachment of the galea to the base of the calvarium [15]. In the present case, it is suspected that the high pressure in the subgaleal space caused by the massive SGH tore both periorbital tissues, and since the hematoma was liquefied due to the occurrence of secondary fibrinolysis, the hematoma was able to spread through the subperiorbital space. Prakash and colleagues reported a 10-year-old with bilateral proptosis from a SGH in whom the proptosis was reduced immediately after aspiration of the SGH [16]. In our patient, no aspiration was attempted for fear of continuous bleeding. Thus, his proptosis worsened and his left eye was removed.

Conclusion

Detection of the cause of subgaleal hematoma is very important in its management. Imaging is the only way to detect the extent and the nature of the fluid collection. Functional and coagulation factors are very important in ascertaining the possible cause of the spontaneous bleeding. Delayed intervention in such cases would result in loss of an organ.

Declaration

Ethics approval and consent to participate: The parents of the child provided a written informed consent to participate in the study.

Consent for publication: The parents of the child provided an informed written consent for this case to be published in a peer-reviewed journal.

Availability of data and materials: The information used and/or analyzed during this case report is available from the corresponding author on reasonable request.

Competing interests: The authors declare that they have no conflict of interests.

Funding: No funding

Authors' contribution: SG, AO, VM, FB participated in the data collection, data follow-up, data analysis and case report preparation. SG participated in data collection. All authors read and approved final case report.

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Manuscript Information: Received: Nov 24, 2020; Accepted: Feb 16, 2021; Published: Feb 19, 2021

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Citation: Sereke SG, Sahal AO, Mboizi V, Bongomin F. Massive spontaneous subgaleal hematoma with progressive exophthalmoses and monocular evisceration. Open J Clin Med Case Rep. 2021; 1726.