Case Reports

Subgaleal Hematoma and Seven Exchange Transfusions

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A 3 kg baby was delivered by cesarean section after prolonged labor. He had massive subgaleal hematoma. He developed anemia requiring packed cell transfusions and hyperbilirubinemia requiring a total of seven exchange transfusions and highly intensive phototherapy. There were no adverse complications of the hyperbilirubinemia or the exchange tranasfusion.

Keywords: *Exchange transfusion, Jaundice, Subgaleal hematoma*

Subgaleal hematoma (SGH) is a potentially life-threatening extra-cranial bleed that occurs most commonly in neonates after difficult instrumental deliveries(1). It can present acutely as hemorrhagic shock or anemia, and later as neonatal hyperbilirubinemia(1,2). We report a case of SGH resulting in anemia and severe hyperbilirubinemia requiring an unusually large number of exchange transfusions.

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Case Report

Baby M, a 3000 g male infant, was delivered at 38 weeks gestation by emergency cesarean section for non-progress of labor. There was no evidence of perinatal asphyxia or encephalopathy. On day 2 he was referred to our hospital for pallor and a scalp swelling. At admission, his hemodynamic and respiratory parameters were stable, his occipito-frontal circumference (OFC) was 37 cm, there was severe pallor and a SGH extending from the upper eyelids to the nape of the neck. There was no bruising or bleeding from any site.

His hemoglobin was 6.9 g/dL, platelets were adequate and there was no evidence of coagulopathy. Computed tomographic (CT) scan of the head revealed circumferential scalp swelling crossing suture boundaries suggesting of SGH. The intracranial structures were normal (*Fig . 1*).

He was treated with vitamin K injection and blood transfusions (15 ml/Kg) twice on the day of admission. On day 5 of life, he developed jaundice with total serum bilirubin (TSB) of 16 mg/dL and phototherapy was started. The blood groups of the baby and mother were both B positive. The direct Coombs test was negative and glucose-6-Phosphate dehydrogenase level was normal. The reticulocyte count was 2%. Inspite of phototherapy, the TSB progressively rose and hence he underwent a double volume exchange transfusion (DVET) on day 6. He subsequently underwent 3 more DVET's within next 36 hrs, all for TSB values above 20 mg/dL. On the 10th day the TSB (predominantly unconjugated) again rose

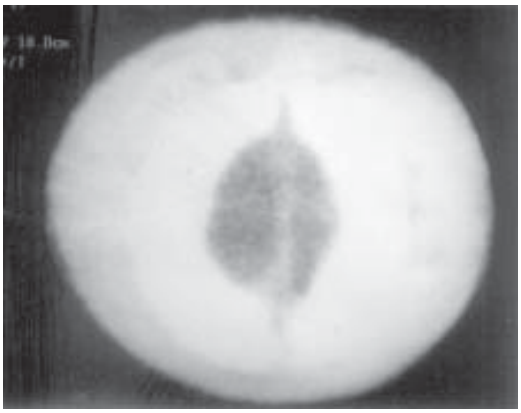

Fig. 1. NCCT scan head showing circumferential scalp swelling (patchy hypoattenuation) around skull bones (dense white).

rapidly and hence he underwent 3 more DVET's over the next 2 days. All donor blood aliquots were of B positive group, and none was more than 5 days old. There was no evidence, either clinically or hematologically, of a hemolytic reaction after any of the DVETs. It is our policy to send a sample of blood from all blood transfusion bags for G6PD assay, and we found in this case that none of the donors were G6PD deficient. The patient received highly intensive single surface phototherapy with special blue lights (Philips TL 52 with irradiance of 18 W/cm^2/nm) throughout the duration of hyperbilirubinemia. We did not drain or attempt to aspirate the SGH. No medication was used to treat the hyperbilirubinemia. At discharge he was mildly jaundiced, neurologically normal with OFC 33.5 cm. At follow up after one month the size of the swelling had decreased and brain stem evoktesponse audiometry was normal.

Discussion

This case of massive SGH is unusual for the large number of exchange transfusions required for treating the hyperbilirubinemia. The sub-aponeurotic space accommodates as much as 250 ml of blood when filled to just 1 cm in thickness(3). It is potentially life threatening because of the potential of hemorrhagic shock.

The reported incidence of SGH ranges from 1.6-3/1000 live births(2,4). The risk factors are instrumental delivery, prolonged second stage of labor; precipitate labor, coagulopathy, prematurity, large infants, fetal dystocia and severe head moulding(2,4-7). The incidence of SGH in ventouse-associated deliveries is 60 times higher than other modes of delivery(5). Male preponderance was reported(5). The index patient was a male child delivered after labour of 48 hours. There was no instrumentation or bleeding diathesis.

Subgaleal hematoma is associated with hypoxic ischemic encephalopathy, head trauma such as intracranial hemorrhage, skull fracture and cerebral edema in various studies(8). In a study by Govaret *et al*.(9), 14 of 27 babies with SGH revealed various abnormalities of parietal bones on CT scan. Three mechanisms of bleeding were suggested: linear skull fracture, suture diastases and fragmentation of the superior margin of parietal bone. In our patient there was no skull fracture, sutural diastases or intracranial bleed on cranial CT scan.

The clinical presentation varies from insidious onset of anemia to hypovolemic shock(1,2). In a study of 101 subjects over a period of 30 months, the clinical presentation of SGH was hyperbilirubinemia in 57 patients (56.4%), anemia requiring blood transfusion in 32.7% and shock in 3 babies. Four patients developed severe unconjugated hyperbilirubinemia, which required exchange transfusions(2). Our patient presented with anemia requiring blood transfusions at admission and later developed severe hyperbilirubinemia

requiring seven DVET's. To the best of our knowledge this is the maximum number of DVET's a patient with SGH has undergone so far in published medical literature.

Phototherapy appears to have been ineffective in our patient, despite adequate light intensity and wavelength. This is somewhat contrary to the experience of Tan *et al.*(10) who found phototherapy to be equally effective in the cephalhematoma. The patient was started on prophylactic antibiotics after the third exchange, and did not develop sepsis during the hospital stay.

An important issue is whether we should have aspirated the blood at the outset to prevent repeated DVETs. The literature is ambiguous about this. There is a sizeable risk of introducing infection during aspiration of an otherwise sterile hematoma, and hence aspiration has generally been recommended only when one wishes to exclude the presence of an infected cephalhematoma(11).

The literature is also silent about the possible use of glucoronyl transferase inducers such as phenobarbitone, or the use of heme oxygenase inhibitors, such as metalloporphyrins, in the presence of large hematomas to prevent the occurrence of hyperbilirubinemia. One animal study has shown that single dose tin protoporphyrin does not reduce bilirubin formation following artificially created hematomas(12).

The mortality rate following SGH is 14-17%(4,5). The most important risk factors associated with death following SGH include severe anemia requiring urgent blood transfusion within first 12 hours of birth and significant birth asphyxia(5). After the acute phase, SGH resolves over 2 to 3 weeks. Newborns with isolated SGH who survive the acute episode show no evidence of subsequent long-term major neurological deficit or developmental delay(4,8).

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Intramuscular Hemangioma Complicated by a Volkmann's Like Contracture of the Forearm Muscles

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Intramuscular hemagiomas are rare tumors constituting less than 1% of all hemangiomas. The clinical picture is usually unlike a conventional vascular tumor. Pre-operative diagnosis is very difficult and most often, the condition is recognized only during surgery or after histopathological examination. This is a report of one such rare tumor, which presented as a painful mass in the Flexor Digitorum Superficialis. It was accompanied by the hitherto unreported complication of a Volkmann's like contracture of the deeper forearm muscles. The peculiar feature of this tumor are highlighted and are discussed with a review of relevant literature.

Keywords: *Hemangioma, Volkmann's contracture.*

Intramuscular hemangiomas are rare tumors constituting a mere 0.8% of all hemangiomas(1,2). They deserve attention not only because of their rarity but also because of their invariably confusing clinical presentation as well as intriguing etiopathogenesis.

Case Report

A 12-year-old girl presented with a painful swelling in the left forearm. There was history of significant trauma to the left forearm, at the age of 6 years. Following the injury, she was initially taken to a traditional bonesetter who splinted and immobilized the limb for 4 weeks. The child was asymptomatic and had no movement deficits on splint removal. Over the next few years, she gradually developed a painful swelling in the left forearm and began finding it increasingly difficult to use the affected hand. The swelling was diffuse and slow growing and was not associated with any constitutional symptoms like fever or weight loss.

Local examination suggested a diffuse intramuscular mass in the flexor compartment. It was warm, tender and boggy. The swelling was neither compressible nor pulsatile and no bruit could be heard on auscultation. The wrist and fingers were held in flexion and passive extension was grossly limited and very painful. Active flexion of the fingers and wrist was also restricted and painful. Passive flexion of the wrist permitted some extension of the fingers at the interphalangeal joints (*i.e.*, a positive Volkmann's sign). However, as the patient

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