Case Raport

Vacuum delivery: 2 cases of subgaleal haemorrhage

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Abstract

Subgaleal haemorrhage (SGH) is a rare complication of vacuum delivery. Although SGH can rapidly be life-threatening, its symptoms are initially confused with both common and benign cephalohematoma or caput succedaneum. Cardiovascular compromise lead to systemic difference in presentation: alterations in colour, heart rate and general condition. Vital parameters are important clues to improve diagnosis after instrumental delivery.

The diagnosis of SGH remains clinical: important swelling of the scalp that accumulates in the declive areas, pallor, tachycardia, irritability, convulsions or hypotonia are the main signs. No further investigation is necessary before urgent therapy. Management consists of rapid correction of hypovolemia with saline and/or red blood cell transfusions.

Early initiation of treatment is associated with better outcomes but further follow-up studies are needed to better describe the uncertain prognosis.

We report two cases of subgaleal haemorrhages following vacuum-assisted deliveries. In both cases, the initial presentation was severe, with hypovolemic shock and coagulopathy. Early diagnosis and intensive treatment resulted in a favourable outcome.

Introduction

Subgaleal haemorrhage (SGH) leads to the accumulation of blood in the unrestricted space between the subgaleal fascia and the skull. It can be a serious, life-threatening complication of childbirth. Its associations with instrumental delivery or coagulopathy is well known. However, some cases occur during delivery without instruments and without coagulopathy. The severity is variable and can range from a small swelling of the scalp to a massive haemorrhage leading to hypovolemic shock and disseminated intravascular coagulopathy.

We report two cases of SGH associated with vacuum-assisted deliveries. In both cases, the initial presentation was severe but early diagnosis and intensive treatment resulted in a favourable outcome.

The aim of this article is to highlight the clinical features leading to the diagnosis and suggest guidelines for management. The importance of early recognition and the need for careful monitoring in instrumented deliveries will be emphasized.

Clinical cases

We report 2 similar clinical cases. Both neonates were male, born at term at 40 weeks with respective weights of 3515 g and 3600 g.

In the first baby, delivery was induced after prolonged rupture of the amniotic membranes. Vacuum instrumentation was indicated for foetal malrotation and was complicated by several instrument disengagements. For the second baby, labour was spontaneous. After several unsuccessful pushing efforts, vacuum instrumentation was performed without success (4 pop-offs). The baby was finally born by emergency caesarean section for an altered foetal heart rate.

In both cases, the babies were born pale, hypotonic, apnoeic and bradycardic. Mask ventilation allowed an increase in heart rate and recovery of spontaneous breathing.

However, patients remained pale, hypotonic and became tachycardic (>180/min) with increasing head circumference and increasingly prominent front. SGH was rapidly suspected in both patients. Their management was similar but with a longer timing in the 2nd baby who was outborn. Both received NaCl 0.9%. for volume expansion and vitamin K for prevention of haemorrhagic disease of the newborn.

In the 1st case, , blood analysis at 30 minutes of life showed lactic acidosis (pH 7.24 – pCO_2 24 mmHg – EB -15 mEq/l - lactates 103 mg/dL), haemoglobin 15 g/dl (N 16.5-20), platelets 162000/mm3 (N 175000-500000) and a coagulation disorder (Quick 34% (N 70-100%) - aPTT ratio 1.3 (N <1.2) - fibrinogen 1.4 g/L (N 1.0-1.2)). There was no clinical evidence of hypoxic ischemic encephalopathy (HIE).The head circumference continued to increase due to persistent bleeding. The disseminated intravascular coagulation prompted the administration of a single dose (50 IU/kg) of human prothrombin complex (Cofact©), a combination of factors II, VII, IX, X and proteins C and S. This was associated with clinical improvement and a normalisation of coagulation factors. The cerebral ultrasound was normal, with no sign of intracranial haemorrhage. The infant was discharged from hospital at 7 days of age and his neurological evolution was satisfactory.

In the second case, pH at 30 minutes of life revealed a severe acidosis (pH 6.83 – pCO_2 103 mmHg – EB -19,2 mmHg - lactates 144 mg/dl). The child also had a coagulopathy which was rapidly corrected after a transfusion of fresh frozen plasma. Neurological examination revealed a moderate encephalopathy (Thompson score of 8), confirmed by a discontinuous normal voltage pattern on amplitude-integrated electroencephalography. Therapeutic hypothermia was initiated at 5 hours of life, once the haemorrhage and coagulopathy were controlled. Cerebral MRI at 1 week of age demonstrated a collection of subgaleal blood without intracranial haemorrhage or anoxic-ischemic lesions. The early evolution was good with normalization of both clinical neurological examination and electroencephalogram.

Discussion

Pathophysiology: The scalp is composed of five layers: the skin; the dense connective tissue; the epicranial fascia, a tough fibrous layer; the loose connective tissue and finally the dense periosteum. The subgaleal space lies be-

Table 1 : *Comparison of the different swelling on the head in neonatal period*

	Subgaleal hematoma	Cephalhematoma	Caput succedaneum
Physiopathology and anatomy	Blood accumulation from rupture of emissary veins in unlimited space between epicranial aponeurosis and periosteum	Blood accumulation from rupture of diploic veins in limited space between the skull and the periosteum	Collection of serosanguinous fluid between epicranial aponeurosis and skin.
Location	Diffuse and extended (nuchal ridge, orbital margins, beside the ears)	Usually over parietal bones	At point of contact Subcutaneous
Extension	Posteriorly to nuchal ridge (decline), laterally to temporal fascia, and anteriorly to the orbital margins, ACROSS sutures lines	Limited by the margins of bones and does NOT cross the suture lines	Can extend ACROSS sutures lines
Clinical signs	- Diffuse and boggy swelling - Hypovolemic and acidosis signs: pallor, respiratory distress, tachycardia, seizure	- Circle swelling with palpable and limited contour	- Diffuse and boggy swelling - Pitting edema, shift with gravity
Volume	Not limited – accumulation up to 260 ml	Rarely severe	20-40ml
Timing	Rapid onset, at birth, but growth mean 1-6h after birth but sometimes symptoms after 1 day.	Growth during 12-24h after birth	Maximal at birth
Management	1) Hemodynamic troubles: - NaCl 0,9% (10-20cc/kg) - Red blood cells transfusion 2) Correction of coagulopathy and DIC. 3) Global support according to related organ diseases. Resolution over 2-3 weeks	Spontaneous resolution over 2 to 3 weeks	Spontaneous rapid resolution in 48-72h
Complications and associations	Acute complications: - Shock - Disseminated intravascular coagulation - Multiple organ failure - Death Medium term complications: - Jaundice - Anemia Long term complications - Cerebral Palsy - epilepsy	- Jaundice - Anemia	- Jaundice - Anemia

tween the periosteum and the epicranial fascia and is bounded from front to back only by the frontal muscle and the posterior nuchal lines and laterally by the temporal muscles (1,2). These features reduce the possibility of anatomical tamponade and allow for an accumulation of blood up to 260 ml in the case of SGH (1,3). SGH is life-threatening and so, early differential diagnosis with cephalohematoma or caput succedaneum is therefore crucial. Table 1 summarizes the characteristics of those three types of cephalic collections. With SGH, each increase of one centimetre in head circumference seems to indicate a 40 ml haemorrhage (1,2,8). This explains the high prevalence of hypovolemic shock, as the circulating blood volume is approximately 80-90 ml/kg in newborns.

Epidemiology and risk factors: SGH is a rare postnatal complication. The incidence is approximately 1/2500 live births, with a male predominance (2,4,5). The mortality rate is as high as 15% in some studies[3].

External forces during delivery, particularly traction and rotation, can cause rupture of the outflow veins in the subgaleal space. This explain why instrumental deliveries are associated with an increased incidence of SGH up to 1/250, with vacuum extraction being a greater risk than forceps (1,2,4,6). The most important risk factors for vacuum failure are malrotation of the foetus, higher birth weight, small maternal size, nulliparity and induction of labour (7,8). Other risk factors for SGH are controversial: the number of vacuum disengagements, vacuum use for more than 20 minutes, vacuum application over the sagittal sutures or too close to the anterior fontanel, and prolongation of the second stage of labour (duration of active pushing from full dilation) above 120 minutes (4,8,9).

Clinical presentations of SGH range from limited scalp swelling to severe blood loss complicated by hypovolemic shock and coagulopathy (1). SGH in uninstrumented vaginal delivery appears to be less severe and requires a lower rate of blood transfusion (10).

Diagnosis and further investigations: The diagnosis is based primarily on clinical presentation. An early diagnosis allows urgent and appropriate management.

In babies born without instrumentation and presenting with SGH, coagulation disorders such as vitamin K deficiency or haemophilia should be investigated (3,5).

In the case of instrumental delivery, careful observation of the newborn should focus on head circumference, heart rate, tone and colour (1,2,6,12,13). Shock can appear in the minutes after birth. The average onset of symptoms is 1 to 6 hours, but they can still appear up to 2 days after birth (1). In units with early screening, the mortality rate has decreased from 15% to 2.8% (8).

Treatment: The management is based on circulatory stabilisation through restoration of blood volume and correction of coagulopathy. Ventilatory, inotropic or vasopressor support should be used if necessary. Correction of hypovolemia is the mainstay of treatment, starting with boluses of normal saline and/or red blood cells. Early blood transfusion may stop the progression of bleeding (8,14). Head banding increases intracerebral pressure, leading to head trauma or cerebral oedema, and is not recommended. Surgery has been performed previously but does not appear to be helpful (1,4,10,15,16).

Aggressive and rapid correction of the coagulopathy (INR threshold 1.5) with fresh frozen plasma (10-20 ml/kg) in the first instance is considered important to avoid disseminated intravascular coagulation (8,17). Without a clear correlation between the severity of thrombocytopenia and the risk of bleeding, the thresholds for platelet transfusion remain unclear (17). Despite its

high efficacy the administration of recombinant activated factor (especially VII), as in our first patient, is controversial given a potential increase in thromboembolic risk (4,16,17).

Finally, after stabilisation, attention should be paid to hyperbilirubinemia resulting from hematoma resorption. Prolonged and intense phototherapy can be necessary (1).

Therapeutic hypothermia in active haemorrhage remains controversial as it can be associated with thrombocytopenia and hypotension and therefore could potentially increase the risk of intracranial haemorrhage (ICH) (18,19). These potential side effects must be weighed against the demonstrated benefits of hypothermia in HIE in reducing deaths and improving neurological outcomes in survivors.

Early diagnosis and prompt initiation of treatment have been associated with better outcomes, as in our patients.

SGH is a severe disease, with a high incidence of mortality (between 5 and 14%) and adverse outcomes (1-3,5,10,14). Neurological sequelae resulting from cerebral ischemia include seizures, neurodevelopmental delay and cerebral palsy (1,5,20). Factors associated with poor outcome include anaemia, coagulopathy, metabolic acidosis, renal failure, hypotension and seizures (15).

Conclusion

SGH is a rare but severe cause of neonatal morbidity and mortality. Haemorrhage into an unrestricted space can rapidly lead to hypovolemic shock . The management of SGH includes volume expansion and hemodynamic support, correction of coagulation disorders and management of neurological and other organ involvements. Identification of risk factors, early diagnosis, close observation and prompt treatment are all important to avoid rapid deterioration. Instrumental delivery should not be considered routine and warrants careful monitoring of the newborn.

Conflict of interest

No potential conflicts of interest in relation to this article have been reported.

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