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Vacuum extraction, bone injury and neonatal subgaleal bleeding

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Abstract. In a population of 27 flemish newborns with subgaleal bleeding encountered within a period of 6 years, we studied the obstetrical, clinical and radiological data. In contrast with controversial findings from the available literature, there is little doubt that difficult, often elective vacuum extraction is the main cause of this neonatal emergency. Disturbances in haemostasis, when documented, were attributed to focal intrahaematoma consumption, except for one boy who presented with haemophilia and neonatal subgaleal bleeding. Conventional X-ray examination continues to be of importance for the documentation of suture diastasis, fissures and fractures. CT scan reveals both the amount of extra-osseous bleeding, the degree of bone displacement and injury as well as the type and extent of associated intracranial damage. Subgaleal haemorrhage rarely hides a growing synchrondrosal rupture.

Key words: Vacuum extraction – Subgaleal bleeding – Newborn CT scan

Introduction

Neonatal subgaleal haemorrhage is a rare but probably underreported medical emergency. Its clinical diagnosis may be difficult although the anatomical definition is unequivocal, namely, blood accumulating within a space of very loose connective tissue between the periosteum of the calvarium and the galea aponeurotica [25]. Plauché reviewed 123 patients in 1980 and concluded that instrumental vaginal delivery is the main predisposing factor [26]. We report on the diagnostic features in 27 patients. In comparing these findings with literature data, obstetric events, coagulation studies and brain CT scan results have been emphasized.

Patients

Twenty-six newborn infants referred to the neonatal intensive care unit between December 1985 and June 1991 met the two major inclusion criteria: a detailed record of obstetrical events and a correct clinical diagnosis of subgaleal bleeding. In addition one similar patient observed some years previously has been included because of the unusual aetiology of his subaponeurotic haemorrhage.

As hallmark for the clinical diagnosis of subgaleal haemorrhage served the presence of a fluctuating mass straddling cranial sutures and/or fontanelles. Inclusion required that such a mass extended beyond the margins of the natural of artificial caput succedaneum. Pitting oedema and ecchymotic discolouration of varying severity appeared after a variable interval. They spread posteriorly towards the neck and laterally around the ears, sometimes displacing the ears. Anteriorly the mass reached the orbital roof, the upper eyelids and/or the root of the nose. The maximum interval between birth and diagnosis was 4 h.

CT scan of the cranium was available in 24 patients. The scans, without contrast enhancement, were taken in unsedated infants with a third generation scanner (Somatom DRH, Siemens).

Results

Boys (16/27) outnumbered girls. Seventeen infants were firstborn. Except for 2, all were born at term. The head circumference was above the 90th percentile at birth in 8 neonates. In 15 a postnatal increase of the head circumference of at least 1 cm was noted within the first 12 h of life; in 5 the increase was at least 3 cm. Intensive care was not prolonged with neonatal death as the result in 5 newborns with serious cerebral injury. One girl died from fatal tentorial damage and a torn great cerebral vein.

For 5 of these infants the second stage of labour lasted more than 60 min (Table 1). Epidural anaesthesia was common (18/27). Fundal pressure was applied in 9 deliveries. All except one were born by vacuum extraction. In 4 caesarean section was performed because of extraction failure. In 7 the instrumental delivery was elective, i.e. because of the desire to lessen maternal discomfort of the second stage; these instrumental deliveries lacked stringent indications such as fetal distress or cephalo-pelvic disproportion. Application of the cup was done at or above Hodge's interspinal plane in at least 13 cases. For 14 inborn infants the standard procedure included the use of a 6 cm diameter metal Malmström cup and instantaneous increase of the vacuum up to 0.8 kg/cm^2.

Fetal acidosis developing during vacuum extraction was documented in 8 patients. Nineteen infants needed resuscitation at birth. Thirteen were ventilated for more

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Table 1. Neonatal subgaleal haemorrhage: clinical and biochemical data

	$n = 27$
First stage >12 h	8
Second stage >60 min	5
Maternal anaesthesia: general/epidural	2/18
Fundal pressure applied	9
Delivery by vacuum extraction	26
– number of tractions >4	15
– cup detachment(s)	7
– indication: elective/maternal	7/9
fetal distress/unknown	5/5
– at or above Hodge's interspinal plane (unknown)	13 (13)
– secondary caesarean section	4
– spontaneous vaginal delivery	1[a]
Meconium staining	5
Fetal scalp pH: unknown / <7,25	16/2
pH umb. artery: unknown / <7,25	13/10
Apgar score ≤3 at 1 min	10
Resuscitation at birth: – bag and mask	12
– endotracheal	7
$NaHCO_3$ need (day 1) >10 meq	12
>100 ml of plasma/blood expander	9
Haemoglobin nadir <12 g/dl	15
Vitamin K prophylaxis at birth	22
Coagulation data: uninformative	8
normal	10
HDNB	0
DIC	8
haemophilia	1

HDNB, Haemorrhagic disease of the newborn baby; DIC, diffuse intravascular coagulation
[a] Birth weight 1420 g, head and foot presentation

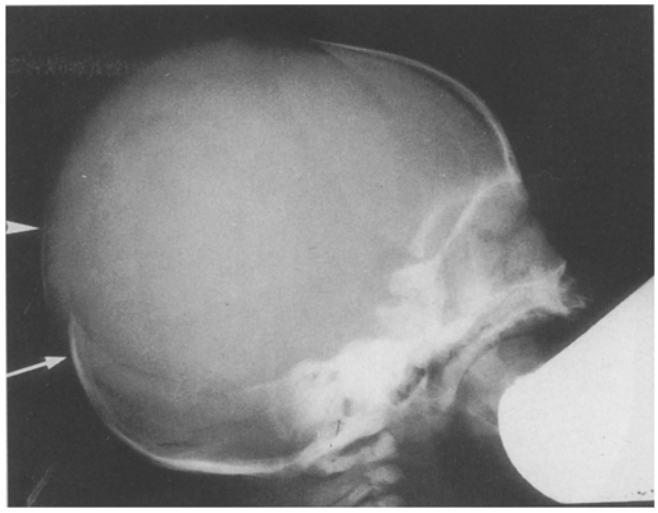

Fig. 1. Lateral skull X-ray film: obvious separation between squama occipitalis *(arrow)* and parietal bone *(arrowhead)*

Table 2. Neonatal subgaleal haemorrhage: CT data

Feature	$n = 24$
Timing of CT scan: day 0–3/after day 3	20/4
Amount of extra-osseous blood:	
– little +	1
– moderate ++	3
– plenty +++	20
Angulation of adjacent bones	14
Intracranial lesions:	
– subarachnoid bleeding (isolated)	24 (10)
– intraventricular bleeding	3
– epidural bleeding	1
– intracerebral bleeding	4
– subdural bleeding – supratentorial	6
– infratentorial	6
– occlusion of a cerebral artery	2
– superior sagittal sinus thrombosis	1
– cranio-cerebral erosion	1

than 24 h. Postnatal metabolic acidosis was present in 13 neonates. Twelve of them needed more than 10 meq of $NaHCO_3$ in the first hours of life. Although only 12 infants were actually hypotensive, 22 newborns became anaemic. Sixteen of them required transfusion of blood or a derivative for volume expansion.

All of our patients received intravenous vitamin K within a few hours from birth. Solid arguments in favour of disseminated intravascular coagulation were present in 8. One infant, with a Thrombotest of 12% (moderately low) suffered from haemophilia A (severe type). Eight coagulation profiles were uninformative, yet, apart from the single boy with haemophilia, the clinical evolution and serial coagulation studies have excluded an inborn disorder of haemostasis in each of these infants.

Conventional X-ray films of the skull documented a skull fracture in 8 infants, unusual separation between the squamous part of the occipital bone and the parietal bones in 7 (Fig. 1), suture diastasis in 9 and fragmentation of the superior margin of the parietal bone in 3. On CT scan (Table 2), extra-osseous bleeding was severe in all but 4. In the latter, the interval between birth and scanning was more than 3 days. Fractures were evident on CT in 5 neonates, but the presence of a prepontine pneumatocoele suggested the existence of a basal skull-fracture in 1 other child. Angulation between adjacent bones, especially parietal and occipital, was frequently seen (14/24) (Fig. 2). All scans revealed posterior cerebral interhemispheric densities compatible with subarachnoid haemorrhage. Eight infants had an associated subdural bleeding. In 4 the latter was severe enough in order to contribute to the fatal outcome. In this patient group, three intraventricular and four intracerebral bleedings, one epidural haematoma, two infarctions within the region of a cerebral artery (one middle, one posterior), one thrombosis of the superior sagittal sinus and one growing craniocerebral erosion following a ruptured interparietal synchondrosis were also noted.

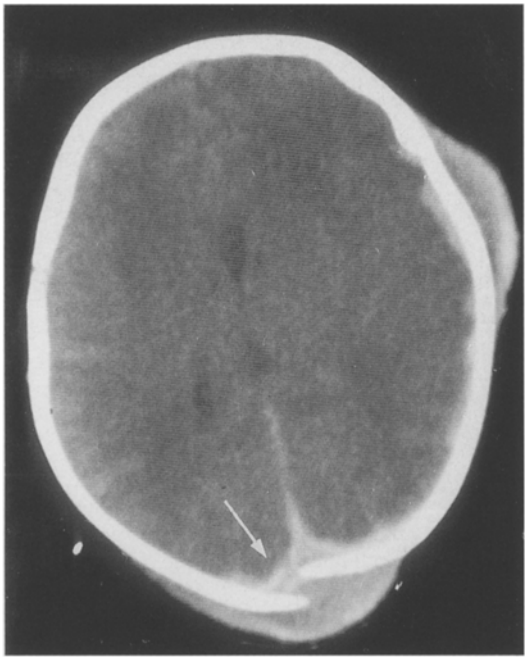

Fig. 2. Uncontrasted CT scan: interparietal dehiscence above the superior sagittal sinus *(arrow)*

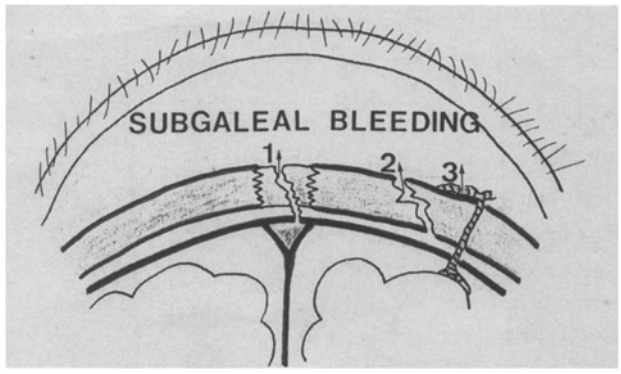

Fig. 3. Schematical drawing of the events leading to subgaleal haemorrhage: *1* synchondrosal rupture; *2* bone fracture; *3* ruptured emissary vein

Discussion

Already about 30 years ago several authors recognized that subgaleal haemorrhage is a potentially fatal but often curable neonatal emergency [6, 8, 13, 22, 23, 30, 31].

Inherent to the loosely organised connective tissue in the subgaleal space is the potential of receiving abundant quantities of extravasated blood thus leading to hypovolaemic shock. This is in contrast to the space-restricted cephalhaematoma. Different hypotheses have been put forward concerning the pathogenesis of subgaleal haemorrhage (Fig. 3). In some instances an underlying skull fracture has been documented [19]. Rupture of an interosseous synchondrosis – mainly between the parietal bones – may also cause direct mechanical injury to major intracranial veins or sinuses [22]. Distortion of or traction upon emissary veins bridging the subdural and subgaleal space have also been invoked as an explanation of its pathogenesis [3].

Timeless controversy "characterises" the aetiology of neonatal subgaleal bleeding. The retrospective study of Plauché has singled out instrumental vaginal birth as a crucially important predisposing factor [26]. Only 28% of his infants were born after spontaneous vaginal delivery. Of the remaining, 9% occurred after secondary caesarean section, 14% after forceps and 49% following vacuum extraction. Malmström admitted that high and lengthy vacuum extraction may cause subgaleal haemorrhage [24]. This report underlines the importance of the association of neonatal subgaleal bleeding with vacuum extraction. Taking into account that 14 babies were inborn we calculated an incidence of 7/1000 vacuum extractions in our own perinatal centre, thus confirming Plauché's suggested incidence of 5–10/1000 extractions [26]. The mechanical trauma to the fetal cranium was complex in several patients because of a long first stage, persistent occiput-posterior position or fundal pressure applied during descent. In nine infants difficult vacuum extraction remained the only mechanical insult. Acidosis developed during extraction in several fetuses. An important hint for the role of mechanical injury is the fact that all bleedings were diagnosed within the first hours of life. Twelve newborns were transfused on clinical grounds. The indication was supported by a bedside rule stating that an increase of the head circumference with 1 cm roughly corresponds to a loss of 38 ml circulating blood volume [27]. The mortality rate here reported (19%) is comparable to that in the literature (20%–25%) [26]. Associated intracranial bleedings as severe as to lead to a fatal outcome, included laceration of the right hemisphere in one newborn, rupture of the superior sagittal sinus and bilateral tentorial tears in two others.

Erosion of parietal bones and dehiscence between adjacent bones were first reported shortly after the introduction of vacuum extraction [5, 8, 15, 16, 22, 29]. These phenomena have been documented both by in vivo roentgenographs and by postmortem examination. Awon, using an experimental vacuum extraction-like manoeuvre in deceased newborns, has demonstrated obvious elevation of the parietal bones underneath the cup [4]. Fractures and fissures of the parietal bones have also been reported [2, 5, 9, 15, 29]. Though standard radiographs have confirmed some of these observations, more information was obtained by the use of CT scan, a method rarely used before in this condition [11, 28]. Apart from the expected "crown" of abundant epicranial blood, parieto-occipital bone dehiscence was frequent and bone fragmentation could sometimes be obviated. Though all babies had evidence of subarachnoid bleeding only those with extensive intracranial damage have subsequently died.

We propose that three types of neonatal subgaleal haemorrhage can be distinguished. The first type is due to minor mechanical trauma superimposed on primary failure of haemostasis as is the case in haemophilia A [11, 18, 28]. The second type appears to be due to vitamin K deficiency after a normal or difficult delivery. Such haematomata mostly develop after 2 or 3 days [1,

3, 17, 31]. Routine administration of vitamin K at birth should eliminate this type of subgaleal bleeding. Any newborn, without a disorder of haemostasis, may be a victim of the third type i.e. purely traumatic neonatal subgaleal haemorrhage, typically related to difficult vacuum extraction [7, 10, 14, 20, 21]. Secondary abnormalities of the coagulation profile due to increased consumption of coagulation factors, may compound the pathogenesis.

Urgent restoration of circulating blood volume (about 40 ml per cm increase of head circumference) is the cornerstone of immediate treatment. The severity of neonatal subgaleal bleeding should be evaluated with blood pressure monitoring, serial determination of packed cell volume, coagulation studies and cranial X-ray film (including CT scan).

Routine administration of vitamin K at birth and adherence to the most stringent indications for the use of vacuum extraction are obvious prophylactic measures. On the contrary the effectiveness of compressive bandages or aspiration of the haematoma has found but anecdotical support [17, 22].

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