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CASE REPORT

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Craniocerebral birth trauma caused by vacuum extraction: a case of growing skull fracture as a perinatal complication

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Abstract A case of growing skull fracture following birth trauma and caused by vacuum extraction is reported in order to emphasize the incidence of this peculiar head injury at the beginning of extrauterine life and to point out its relation to possible neuropsychological disturbances that may appear later in childhood. Delivery by vacuum extraction increases the incidence of perinatal injuries and consequently the incidence of neurological deficits in children. Neurosurgical repair is advocated as the appropriate treatment, with the aim not only of cosmetically correcting the lesion's typical subgaleal protuberance with cranioplasty, but also of performing a water-tight closure of the dura, enabling the cerebral cortex to "fill in" the intracerebral lesion. The surgical technique and gross pathology of the lesion are described together with radiological findings before and after surgery. Reports by other authors are reviewed in an attempt to identify the conditioning factors and pathological features of this traumatic injury to skull and brain in neonates and infants. The literature on cranial fractures associated with intracerebral lesions at this age shows a significant difference in recovery and outcome from that after similar lesions in older children.

Key words Perinatal head trauma · Growing skull fracture · Head injury in infancy and childhood · Vacuum extraction

Introduction

Head injury in neonates and infants is known to be associated with higher morbidity and mortality than similar traumatic lesions in children or adults [9, 15]. Cranial fractures following perinatal complications, whether they are progressive or not, can be associated with brain lesions that may become symptomatic later in childhood. A review of publications on the growing skull fracture as a complication following head injury in infancy reveals a number of different terms like "meningocele spuria," "leptomeningeal cyst," or "traumatic porencephalic cyst," all given to the same entity. This variety in the terminology indicates the puzzling pathomechanism of the progressive damage, involved and summarizes its pathoanatomical changes to a syndrome.

The enlarging bone defect usually occurs in neonates and infants up to the age of 24 months, tending more to appear during the 1st year of life. This fact indicates one of the conditioning factors of the lesion's development: the rapid growth of the brain during this period, and the pulsation of its tissue [15, 16]. Other traumatic alterations that have to coincide for a skull fracture to grow are laceration or rupture of the dura, free communication of the subarachnoid space through the bone gap, and interposition of the pericranium and the meninges between the diastatic edges of the fracture [3, 19]. The most common site of a growing fracture is reported [6, 18] to be the parieto-temporal area (38% of cases), followed by the parietal (25%) and

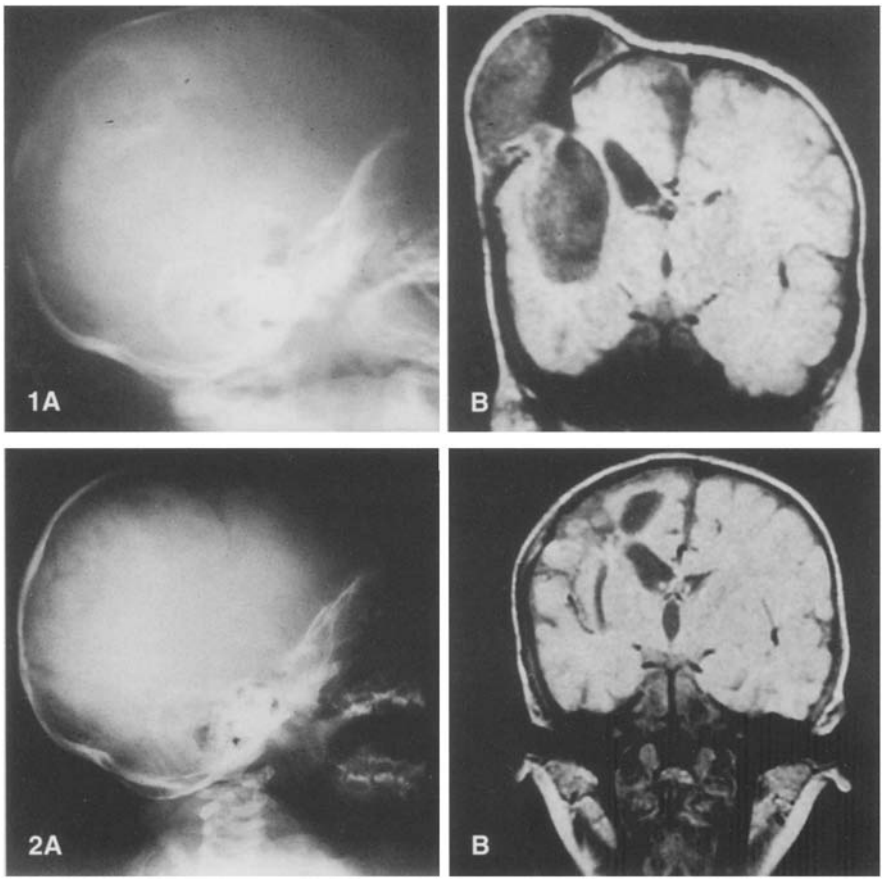

Fig. 1 A, B About 5 months after birth trauma: plain X-ray and MRI findings show skull and brain damage in the right parietal area with a typical growing skull fracture. A tense scalp pouch over the bone defect is filled with CSF and communicates through a cortical defect with an intracerebral cyst. The right ventricle is partly dilated and has shifted towards the lesion

Fig. 2 A, B One year after surgery, radiological follow-up reveal good healing of the cranioplasty and closure of the cortical defect

frontal (12%) regions. Although patients may be of school age or even grown up at the time of hospital admission and detection of the fracture [12, 14, 19], the first 24 months of life are recognized as the age at which this traumatic condition occurs. There have been no reports of real *growing* skull fractures in adults or appearance of the lesion following any kind of head injury (including osteoclastic craniotomy with dural damage) if one of the conditioning factors mentioned above was missing.

If not associated with other lesions, diastatic cranial fractures less than 3 mm in length in newborns and infants, like epidural or subdural hematomas, are usually given no special treatment. However, they carry a high risk of generating progressive brain damage, making X-ray follow-up and CT examination essential for early detection and treatment.

The case we report of growing skull fracture caused by vacuum extraction and associated with severe cerebral damage adds another argument against this method of assisted delivery. The brain damage beneath the fracture can be overlooked or underestimated, since neurological symptoms may be absent initially, developing after months or years in the form of behavioral disturbances, epileptic seizures, and contralateral weakness. Epidemiological observations on head injuries in young patients have mostly failed to differentiate between infants and young children.

Case report

Our 5-month-old female patient had sustained head trauma at birth after an uncomplicated pregnancy by being delivered via vacuum extraction. The first sign of the lesion was a small floating scalp pouch noticed in the right parietal area, which was misinterpreteted as caput succedaneum. However, the soft mass would not disappear during the following weeks, and the baby, who appeared neurologically normal, underwent a series of plain X-ray examinations over the following months that showed evidence of an enlarging skull lesion.

Referred to our department in December 1990, the child appeared to be developing normally without obvious neurological symptoms. CT and MR images demonstrated typical pathomorphological features of a growing skull fracture, i.e., a hypodense, mainly cystic lesion located in the right parietal region which apparently communicated with an extracranial pouch through a dural and bone defect filled with CSF (Fig. 1). In addition, the pouch seemed to contain some hypodense tissue, suspected to be herniated brain. The anterior horn of the ipsilateral ventricle was dilated, showing a shift towards the cyst without communication.

Upon surgery, a scalp flap was formed over the bone defect and the protruded pouch was carefully tapped of clear CSF. After collapse and removal of the pouch, a cone-shaped brain tissue defect

was disclosed beneath the torn dural and arachnoidal layers, resembling a porencephalic cyst. Inspection of the cortex revealed no further pathological changes. The extracranial extension of the cyst, i.e., the resected pouch itself, contained only particles of a jelly-like pulp, histologically representing residuals of necrotic tissue. Repair and watertight sealing of the 2×3 cm dural defect was consequently carried out using a graft of pouch tissue. Finally, cranioplasty was performed by rearranging bone grafts taken from the fracture's edges and placed mainly over the porencephalic cyst.

The postoperative course was uneventful and the baby was taken home by her parents 6 days after surgery. The follow-up plain X-ray and MR controls revealed satisfactory healing of the bone defect and „closure" of the cortical damage (Fig. 2). The traumatic porencephalic cyst itself and the dilated ipsilateral ventricle show no further evidence of progress. More than 3 years after operation, the little girl has now developed a left-sided spastic hemiparesis, indicating significant neurological impairment due to vacuum extraction.

Discussion

Craniocerebral trauma in infancy and childhood belongs to the most frequent causes of death in Europe and the United States. However, only few epidemiological studies have focused on the influence of age on head injuries in early life [5–7, 10], while most investigators fail to make a clear distinction between infants and young children. Raimondi and Hirschauer [13] point out that the higher incidence of skull fracture in children with open sutures and fontanels is associated with higher morbidity and mortality. It is concluded that infants are more vulnerable to blunt head trauma and suffer more neurological deficit than young children. In addition, physiological differences between infants and young children, e.g., the incompletely myelinated brain of the very young, have significant influence upon the survival and course of these two groups after injury. Head injuries among 0- to 4-year-olds result in a rather poor clinical outcome and significantly higher mortality (62%) than head injuries to children aged between 5 and 10 years, who show a good recovery in two-thirds of cases [15].

Cranial fractures in neonates and infants, although not always easy to detect, are a frequent radiological finding after injury and have an incidence of up to 26.8% during the first 6 months of age [6]. They are divided into two main subtypes, *linear* and *depressed* fractures, and are usually caused at birth by a narrow pelvic passage or pressure against the last lumbar vertebra, the sacral promontory, forceps, or vacuum extractors. Both diastatic linear and depressed skull fractures can give rise to the development of a growing skull fracture [13, 15]. Association with intracerebral lesions and neurological deficits that may appear later in childhood is common. However, the significance of skull fractures among newborns and infants is often underestimated, since the majority of these lesions demand no special treatment and heal spontaneously within weeks or months. Latent neurological disturbances resulting from associated cerebral damage can remain silent for months or years, giving no cause for further investigations. Additional lesions causing neurological symptoms, such as epidural hematomas, which otherwise occur at a high incidence in association with skull fractures, are less common in newborns and infants because of the adherence of the dura to the inner table of the skull.

The evolution of the CNS during the first few years of life and the resultant rapid changes in psychomotor performance make the Glasgow Coma Scale (GCS) for adults an unreliable instrument in cases of pediatric brain trauma. A modified Glasgow Coma Scale for the assessment of children under the age of 3 years is recommended and appears useful [13, 15]. Physical disabilities which appear of minor concern initially, cognitive deficits, and epileptogenic signs, if unrecognized in infants, can increase over the years to become insurmountable handicaps. Therefore, prediction of outcome should not be exclusively related to the time of the infants' discharge from hospital. Intracranial injury is the second most frequent cause of seizures in neonates and children, with an incidence of 10% [5, 15]. CT, EEG, and psychological tests are recommended in cases of suspected craniocerebral trauma. Radiological follow-up of brain lesions using MRI is preferable to CT in detecting residual defects and revealing early evidence of increasing intracerebral alterations [9].

In his analysis of traumatic birth injuries, Hovind [10] points out that unexplained death in infants is often associated with a perinatal trauma – a possibility that should be always taken into consideration and might be of help in cases of medicolegal problems. Di Rocco and Velardi [7] reported on statistical investigations which showed up to 5% mortality following craniocerebral trauma at birth. Hemiparesis, epileptic seizures, and cognitive disorders of unknown origin in children should likewise be tracked back for possible head injuries at birth or later.

Early surgery of growing skull fractures and early rehabilitation care to overcome associated deficits are suggested as appropriate management. Prevention of neurological impairment at birth will be furthered by the abandonment of delivery-assisting methods like vacuum extraction. Vacuum extractors have been repeatedly blamed for major head injuries ranging from life-threatening subgaleal bleeding [4] to intracranial hemorrhage [2]. Our report of a traumatic porencephalic cyst caused by this means reveals another dangerous complication due to the force of the suction instrument on the insufficiently protected, immature brain at birth.

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