

Intracranial hemorrhage of a newborn due to a congenital defect on the roof of the cavernous sinus

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ABSTRACT

Spontaneous early perinatal bleeding associated with intracranial hemorrhage results from brain anomalies or systemic reasons. A congenital dural defect of the cavernous sinus may be another cause of nontraumatic intracranial hemorrhage in the newborn.

A female newborn died one hour following the birth. She had a pale skin. Predominantly left sided subdural hemorrhage causing brain compression was found on examination. There was no congenital abnormality on investigations but a dural defect of 2 mm in diameter on the wall of the left side of the cavernous sinus.

Presence of dural defect on the wall of the cavernous sinus may result in early perinatal death associated with intracranial hemorrhage. *Neuroanatomy; 2007; 6: 56–57.*

Key words [cavernous sinus] [dural defect] [intracranial hemorrhage]

Introduction

Anomalies related to cardiovascular system are the most commonly seen congenital anomalies that are the causes of death during the newborn period. Central nervous systems anomalies such as; microcephaly, anencephaly, hydrocephaly, encephalosele, spina bifida are also well defined as the frequent causes of death [1].

The aim of this study is to present an infantile postpartum death case, caused by a congenital defect that occurred on the roof of the cavernous sinus, which was not stated in any literature.

Case Report

This case concerns about girl baby, who died 1 hour after her birth. She experienced a difficult and prolonged birth due to being the first baby of the family. Although her prenatal examinations were done irregularly, birth was normal and successful in the beginning, however her general health status and apgar score went down during her first life hour, and forensic autopsy has not been considered after her death. However, pathological autopsy performed to reveal reason of spontaneous death. The physical examination showed normal development of a term baby. The umbilical cord was properly cut and clamped. The newborn had a pale hypovolemic skin tone. When the head was opened, no ecchymosis was detected under the hairy scalp and in both temporal muscle groups. Anterior and posterior fontanelles were open. The bones of the skull were intact; however, subdural hematoma that

caused a serious cerebral compression was detected all over the brain surface, more prominently on the left side. It was not accompanied by subarachnoidal hemorrhage. No aneurysmal dilatation was noted during evaluation of intracranial vessels. The brain was non-edematous, but parahippocampal gyrus was herniated. On the passage of the left internal carotid artery through the cavernous sinus (CS) roof, a dural defect of 2 mm in diameter was detected (Figure 1). Upon internal jugular vein compression on the neck, it was observed that venous blood within the cavernous sinus appeared to flow out of this point into the subdural space. Through the defect, a fibrous trabecular band belonging to cavernous sinus was seen. The defect was not connected to any of the veins on the brain.

When the neck, chest, and abdomen were exposed, no findings of trauma or congenital anomaly were detected in the organs of these spaces. However, all the organs had a hypovolemic appearance. Histopathological evaluation results for the brain, heart, liver, retina, and kidneys indicated no pathology.

She was donated as a cadaver to Ankara University School of Medicine, Department of Anatomy.

Discussion

Trauma associated intracranial hemorrhage in the newborn may be due to obstetric trauma, shaken baby syndrome, motor vehicle accident, and falls [2]. Particularly, when there is no eyewitness, in as high as 95% of the cases

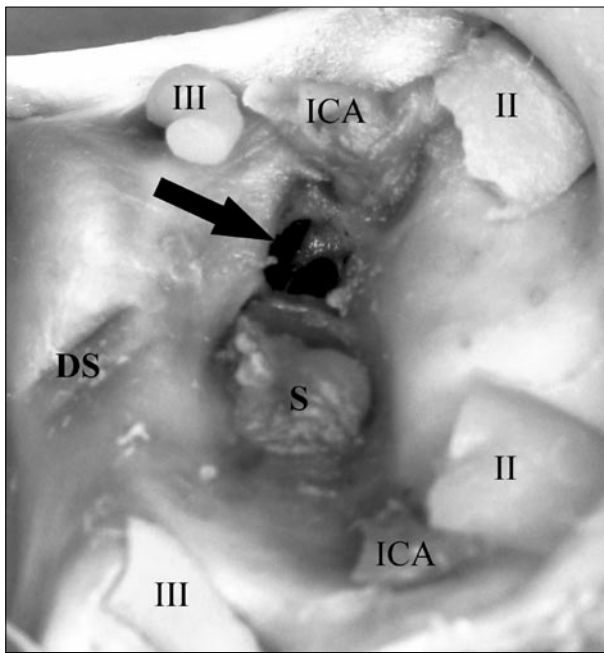


Figure 1. The dural defect (*arrow*) located on the posterior of the left optic nerve. (*II*: optic nerve; *III*: oculomotor nerve; *ICA*: internal carotid artery; *DS*: dorsum sella; *S*: pituitary stalk; *arrow*: dural defect)

presenting with subdural hematoma, child abuse may be suspected [2]. In our case, no trauma associated finding was detected during physical examination, and no asphyxia was noted. Thus, shaken baby syndrome was ruled out. In histopathological evaluation, except for a new hemorrhage under the dura, no other pathologies were found.

The etiology of spontaneous hemorrhages in children may be classified as structural, nonstructural, and hemorrhage of unknown etiology [2]. Structural etiology is directly associated with anomalies of the brain. These may be bleeding into the infarct, vascular malformation, vasculopathy, dural sinus thrombosis, brain tumor, and intracranial infections [2]. Nonstructural etiology causes hemorrhage indirectly by affecting the coagulation

and cerebral blood flow. This group includes systemic conditions such as platelet diseases, coagulation defects, hypertension, cancer and cardiac anomaly. In hemorrhages of unknown etiology, primary thalamic hemorrhage of the term infant and germinal matrix hemorrhage can be listed [2].

Cavernous sinus embryologically develops between Carnegie stages 10 and 23. In Carnegie stages 10-13, mesoderm tissue that will form CS emerges. In stages 14-16, ICA progenitors located in CS is seen. In this period, the neighboring mesenchyma on the ventral of mesencephalon and metencephalon forms a net. Later, leptomeninges layers form out of this net. The mesenchyma surrounding this net is divided into two layers, one forming the bones and the other, the dura [3]. In our case, the dural defect may be due to unclosed mesenchyma that forms the dura in stages 14-16. The normal development of fetus until birth is related to low venous blood pressure in the cavernous sinus. In our case, the arachnoid membrane covering the defect and the left optical nerve had blocked the blood escape out of the defect into the intracranial space. Besides, during fetal life, venous blood that leaks out of the defect may have been thrombosed upon encountering the arachnoid membrane, thus forming a plug.

The obstruction of blood flow to placenta in the newborn increases the resistance by two folds. During the first respiration of the newborn, intrathoracic pressure decreases to -60 cm H₂O and then increases to $+40$ cm H₂O to deflate the lungs [4]. At that time, the difference of nearly 100 cm H₂O pressure that develops within the thorax is reflected upon intracranial sinuses by superior vena cava and internal jugular vein. In our case, the change in the physiological pressure of the venous system during delivery that caused blood escape from the defect in the cavernous sinus and brain herniation was the reason of death. A newborn has nearly 300-375 ml blood [4]. In addition to cerebral compression and herniation, hypovolemia can be another reason of death in our case. Such a congenital defect may be regarded as a considerably important case among forensic medicine practice.

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