CHOROID PLEXUS HEMORRHAGE CAUSING DELIVERY ROOM SEIZURE IN A TERM NEWBORN

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SUMMARY *: Although intracranial and intraventricular hemorrages are rare in term neonates, they occur at a frequency of 3.5%, 1.1 % of them being in the choroid plexus. A neonate with choroid plexus hemorrhage and intractable seizures which started in the delivery room is presented.*

Key Words : *Intractable Seizures, Choroid Plexus.*

INTRODUCTION

Although intracranial or intraventricular hemorrhages (ICH, IVH) are considered to be rare in term infants, they occur at a frequency of 3.5% in healthy newborns, 1.1 % of them being in the choroid plexus (CP) (1). Trauma and perinatal asphyxia are held responsible for most of them; however, 25% of ICH occur with no identifiable cause (1). We report a term newborn infant presenting with seizures in the delivery room, due to choroid plexus hemorrhage.

CASE REPORT

Baby boy O was born to a 27 years old white female by spontaneous vaginal delivery at 39 weeks gestation. Prenatal history was remarkable for continuous fetal movements starting 3 days prior to delivery. The baby with an APGAR of 3 was noted to have clonic seizures in the delivery room, and required intubation and positive pressure ventilation, after which he was transferred to the neonatal intensive care unit where he was noted to have intractable generalized clonic seizures. Physical examination revealed a well-developed, poorly perfused term infant with birth weight of 3500 grams. He was flaccid, unconscious with no respiratory effort and no response to painful stimuli. Pupils were dilated , nonreactive to light, and deep tendon and newborn reflexes were absent. The rest of the physical examination was unremarkable. The baby was mechanically ventilated and phenobarbital loading was given for seizures along with supportive treatment required for perinatal asphyxia. Hypoglycemia and hypocalcemia were corrected. As seizures continued, diphenylhidatoin, diazepam and midazolam infusions were also given which all failed to stop seizure activity. High dose pyridoxine, lidocaine, and finally clonazepame was tried , which helped to decrease the frequency of seizures.

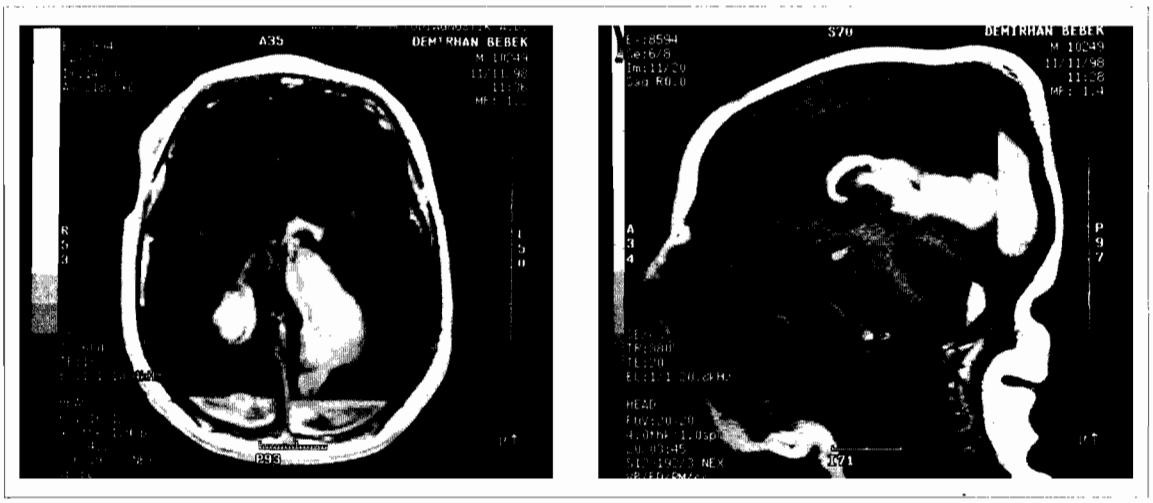

Fig. 1: (a) T1 weighted (TR/TE/NEX=380/20/3) axial image revealed the enlargement of the ventricles. The fluid level due to subacute subarachnoidal bleeding was observed in the occipital horns. (b) T1 weighted (TR/TE/NEX=380/20/3) axial image revealed the enlargement of the ventricles. Abnormally swollen choroid plexus has also a homogeneous high signal consistent with an intraventricular organised hematoma.

Laboratory investigations: Serum glucose, electrolytes, ammonia, lactate, pyruvate, magnesium, calcium and phosphorus levels were normal. His Hb: was 11gr/dl, CSF analysis on the 7th day, including cell count, protein, glucose, lactate, pyruvate and culture results revealed no abnormality. Serum and urine organic acids, TORCH infections screening were all unremarkable. MRI of the brain obtained on the 9th day of life (not before due to his poor clinical condition) revealed massive choroid plexus hemorrhage and hydrocephalus (figure 1). There were no other cerebral malformations observed. Coagulation tests including protein C, S, AT3 levels, Factor V Leiden were also within normal limits.

DISCUSSION

This case represents one of the worst examples of ICH in a term infant with intractable seizures. From the history and clinical course we concluded that the baby had started to have seizures in utero when the mother had noted continuous fetal movements. Earlier seizure occurrence particularly during the first several hours of life after delivery may suggest an antepartum component of an encephalopathic state depending on the length and stresfull events during the intrapartum period (2). Other causes of seizures such as metabolic disturbances, infections, tuberous sclerosis, or other cerebral malformations were (3-5) excluded by the normal metabolic and biochemical screening, MRI findings and normal CSF culture results. Of identified causes of CP hemorrhage cryptic hemangioma and arteriovenous malformations of CP have previously been defined (6,7). Infants with CP hemorrage can either be clinically normal full term neonates without signs of perinatal distress (8), or present with the most common symptom of 'seizure' with an onset ranging from one to 35 days of age (9), and may cause future morbidity and mortality. An overview of 2515 consecutive autopsies performed on newborns who died during the neonatal period between 1970-1989 had revealed that death had increased in term babies as a result of higher frequency of hypoxic-ischemic neuronal necrosis and CP hemorrage (10). We were not able to show a coagulation defect, trauma or vascular malformation to account for the choroid plexus hemorrhage. It might be of unidentifiable cause, or due to profound asphyxia occurring prior to labor. Neonatal seizures are mostly attributed to perinatal asphyxia, however intracranial hemorrhages concerning CP should be considered particularly during very early and intractable seizures. As happened in our patient, these two entities sometimes go hand in hand. If so; with a very good antenatal follow up and serial fetal ultrasonography we must heighten our awareness of probable CP hemorrage and any other brain lesions with an antepartum onset especially in neonates with intractable seizures

starting so early.

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