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Case Report

Sudden Unexplained Seizure in Healthy Term Newborn- An Exploratory Case Report

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Abstract

Cerebral infarcts are an important cause of neonatal seizures. It is an under-recognized cause of long-term neurodevelopmental disability and impaired cognitive function with significant sequel. Here we report the clinical and neuroradiologic findings of a full-term neonate who presented with right arm clonic convulsions with left postcentral gyrus stroke.

Introduction

Numerous conditions presenting with intracranial bleeding in newborns make it challenging to delineate perinatal stroke (1). Further, Infants with subtle symptoms, inappropriate neuroradiological tests, or imaging studies in the newborn can make this condition unveiled months to years later. Perinatal stroke may be described as an acute neurologic insult in vascular injury between 20-week gestation and 28 days postnatal life, diagnosed by neuroimaging or neuropathological studies. The vascular damage can be an arterial ischemic stroke, cerebral venous thrombosis, or primary intracerebral hemorrhage.

Causes of Neonatal stroke includes maternal chorioamnionitis, perinatal asphyxia, meconium exposure, intrauterine infection and neonatal vascular malformation are important causes of condition (2). During Parturition, the coagulation mechanism gets activated as an evolutionary mechanism to overcome the bleeding. Hence during the perinatal period, the odds of ischemic stroke and thromboses in non-cerebral sites are increased both in mother and child. (3)

Mechanism of neonatal stroke could be attributed to perinatal activation of coagulation mechanisms, disturbing fetal and neonatal thrombogenesis within the critical perinatal period. The possible hypotheses proposed for cerebral embolus formation in perinatal stroke are either an insult that occurred before birth (placental embolic hypothesis) or during delivery. The placenta-embolic assumption is that an embolus formed in the placenta reaches the brain through the foramen oval leading to stroke. The other theory is that during birth, a traumatic event happens to cause the issue by injury to the cervical-cerebral arteries, leading to thrombus/embolus formation. Both hypotheses are supported by a few but well-analyzed pathological or arteriographic reports. (4) Although now well recognized, its complex pathogenesis makes every case unique, which warrants the present Case report.

Case Report

A male infant was born at term to a 31-year-old mother with in significant antenatal history. Baby was delivered by cesarean section because of failure to progress, meconium-stained liquor, previous cesarean history, and maternal fever. Birth weight was 3250 grams good APGAR score. At 16 hours of age, baby was admitted to NICU for intensive phototherapy as he developed hyperbilirubinemia due to ABO incompatibility. At 20 hours of age, he created the focal clonic abnormal movement of the right hand and very subtle lip movement. These were consistent with seizures. He had no fever, good feeding no reported lethargy or apnea. He was alert and active on clinical examination, normal neonatal reflexes with no focal deficit. Systemic examination was normal. Sepsis workup was initiated by the pediatrician, and intravenous antibiotics were prescribed. Cerebral function monitor showed seizure activity. The seizures were aborted after phenobarbitone loading and maintenance.

MRI was done urgently, and it showed an area of diffusion restriction involving the left postcentral gyrus. Laboratory testing revealed a white blood cell count of 16x 10^9 with 56% neutrophils, 29% lymphocytes, hemoglobin g/dL, and platelets 401x 10 ^9. The total bilirubin was high at 143 micromole/l, consistent with indirect hyperbilirubinemia. The bone profile showed Normal, the calcium of 2.52 mmol/l, magnesium 0.67 mmol/l, phosphorus 1.9 mmol/l with an alkaline phosphatase level. Serum electrolytes, coagulation assay metabolic, including serum ammonia, lactate, and venous blood gas, were normal. Protein C assays activity levels were low at 37% (NR 70-130) anti-thrombin III was standard 69 % (Normal Range 48- 108). EEG reported frequent abnormal multifocal polymorphic discharges and bilateral, asynchronous right more than left centrotemporal with occipital field spike. Echocardiography was normal.

Discussion

Inconspicuous clinical presentation, varied electrophysiology, and clinical correlation with poor response to antiseizure drugs make "neonatal seizures" a new symptom or condition to healthcare workers every time they are faced.

Across the globe incidence of neonatal seizure varies between 1.0-39.5 per 1000 live births. (5). Multiple shreds of evidence suggested a heterogenic etiological profile of neonatal seizures with hypoxic-ischemia, stroke, or infection is the most common cause. (6) Further variation in prenatal/ perinatal care and diagnostics may be the possible reason for different rates in the detection of this condition, warranting

further evidence to get a comprehensive view of this condition. This ignited up this present piece of the audit.

Neonatal seizures has multifactorial risk factors with varied maternal, fetal, and neonatal characteristics have been implicated. Giraud, in his work, identified the significant factors were male gender, obstetrical determinants (first pregnancy, cesarean section), and two perinatal complications ie, peripartum hypoxia, fetal or neonatal inflammatory state (7)

Time of onset

In term infants onset of neonatal seizures occurs within the first week of life. (4,7,8,9) Similarly, the neonate was presented with an episode within 14 hours of delivery in the present work. Over 35 times are being observed for developing emboli during the due term and especially after caesarian section delivery of the baby (10) this is reported to be a natural mechanism to handle hemostasis by activating the coagulation mechanism. (12-13)

Cerebral infarct

Cerebral infarct increases the risk of neurological disability later in life. (9). Focal arterial infarct contributed more to cerebral palsy than hypoxia or developmental anomaly. (10)

Yvonne Wu et al. reported that among the multiple factors predisposing, prothrombotic disorder, CHD, meningitis, and systemic infections occupy a prime position in causing neonatal seizures. In our study, too, ABO incompatibility coupled with indirect hyperbilirubinemia may be the predisposing factor responsible for unexplained episodes reported in an otherwise normal neonate. Hence cautious note about bilirubin levels and blood incompatibility among neonates is needed.

Side of involvement

A recent meta-analysis revealed that Left-sided strokes, cortical involvement, and the involvement of the parietal and temporal lobes increased epilepsy risk. (14) In the present study, there was a bilateral cortical involvement; hence the ward should be monitored prospectively.

A clear picture of infarcts distribution can be made if MRI can be repeated after a few days, giving a clue that can lead to good prognostic values. The size and location of the infarct are better circumscribed if the MRI is postponed or repeated after a few days and has critical predictive values. Most newborns show an acute infarct within the Middle cerebral artery territory (15). In the present study, the infarct is located in this domain.

Management

In most cases, only supportive therapy is recommended until there is any underlying emergency condition. Management protocol includes the following steps first, to assess EEG for if there are any subclinical seizures. Secondly, to start with antiepileptic drugs if needed for a shorter duration, as evidence suggested potential neurotoxicity of antiepileptic medications on the immature brain if continued for a longer-term. (16, 17).

The placenta being a reliable source to search for any newborn-mother pathology, should be preserved for later examination whenever feasible (18). Evidence suggested frequent clubbing of epileptic episodes with cerebral palsy, compromised academic performance, and cognitive abilities. Hence regular monitoring to identify any impairments and multidisciplinary management should go hand in hand to overcome this debilitating disease impact. Although severe developmental disorders are rare, more than two-thirds of the children inflicted by this postnatal stroke exhibited mild neurodevelopmental disabilities unidentified by their family members and friends. Hence synergistic environmental and psychosocial determinants of this condition can comprise the individual's quality of life.

Mode of Delivery

Literature supports newborns delivered by Caesarian reported the highest prevalence of perinatal stroke. Though surgery increases the risk of thrombosis, whether it is an individual risk parameter is still questionable (19). In the present work, Caesarian delivery could have been a triggering factor. However, further studies are needed to substantiate.

Clinical manifestations

Wu, in his work, revealed: "signs of encephalopathy did not accompany the majority of infants diagnosed with perinatal stroke in the newborn period, and they were normal between seizures". Authors in this work also reported similar clinical presentations.

In our case, neither Fetal distress nor low Apgar scores were reported, contradictory to many issues. (20). Hence keen observation and correct diagnosis can help identify this disorder which otherwise can lead to morbidity in the later stage.

Conclusion

The ward was regular at birth except for hyperbilirubinemia in the present case. Any abnormal biochemical parameter detected in newborns should kindle lateral thinking, and ruling out neonatal stroke is recommended wherever feasible.

A variety of risk factors and clinical presentations of this neonatal seizures /stroke make it an unresolved puzzle. Further, experts' opinion about this disorder is finding its roots in a different direction, but still, we cannot get the whole picture. Hence every new case with various clinical manifestations should be documented to build the chain of evidence.

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