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INTRACEREBRAL BLEEDING IN LATE HEMORRHAGIC DISEASE OF THE NEWBORN

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ABSTRACT Late onset HDN can present as bleeding from any site of the body but more commonly from intracranial vessels. It is diagnosed if bleeding occurs after 7th day of life with normal platelet count, prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), associated with stopping of bleeding and PT/PTT returning to normal after giving vitamin K. Among the types of intracranial bleeding in late hemorrhagic disease of newborn, intracerebral parenchymal bleeding is least common.

KEYWORDS : Late HDN, intracranial bleed, young infant

INTRODUCTION

Hemorrhagic disease of the newborn (HDN) is one of the most common causes of acquired hemostatic disorder in early infancy[1]. It is categorized as early, classical or late depending on the time of onset. Late HDN usually occurs between 2-8 weeks but can occur anytime in the first year. Incidence of Late-HDN in the eastern world is 25-80/100,000 births which is higher than that in the western world (4-25/100,000 births[2]. Newborns have only 20-50% of adult coagulation activity. Lack of vitamin K administration at birth, exclusive breast feeding, chronic diarrhea and prolonged use of antibiotics make them more prone to vitamin K deficiency bleeding[1]. Almost 2/3rd of the babies with late HDN present with serious intracranial bleeds leading to high morbidity and subsequent mortality. Routine vitamin K prophylaxis at birth has brought down the incidence of late HDN.

CASE REPORT

A 2 month male child, born out of non-consanguineous marriage was admitted in our unit with complaints of fever since 3 days and abnormal body movements of all 4 limbs since the same duration. The perinatal history revealed term singleton pregnancy without any complication in mother and the baby was born by normal vaginal delivery without any postnatal complications. He did not receive vitamin K at birth. The baby was on exclusive breastfeeding. There was no history of trauma, no family history of seizure disorder and no history of any blood related disorder in the family. On admission, vitals were stable, there was pallor, and there were no other remarkable findings on general examination. On systemic examination, there was loss of power in right half of body with increased tone and spasticity, and reflexes were exaggerated, suggestive of right sided hemiparesis.

Routine hematological investigation showed: Hb-6.1 g%, platelet count- 1.2 lakhs/cm^3, WBC count-9000/cm^3 (70% neutrophils, 25%lymphocytes), CRP 65.4mg/dl, total bilirubin 1.1mg/dl (direct bilirubin 0.5mg) SGOT/SGPT/AlkPO4 were 96/63/3911U respectively. Renal functions and electrolytes were within normal limits. Blood and urine cultures were negative. Lumbar puncture was not done as there was negative consent from parents. Bleeding time was 1 min 45 seconds and clotting time 4 mins. Serum calcium was 6.2 mg/dL and after correction, it increased to 9 mg/dL. The coagulation profile showed: prothombin time (PT): 18.0 s, activated partial thomboplastin time (aPTT): 38.0 s and International Normalized Ratio (INR): 1.05.

Transfontanelle ultrasonography revealed large well defined hypoechoic lesion measuring 2.2cm by1.9cm in left periventricular region, compressing left ventricle. MR angiography of head and neck (Figure 1) revealed huge intracerebral hemorrhage in the left cerebral hemisphere (parietal) with midline shift. CECT brain (Figure 2) was also done, which showed acute intraparenchymal hematoma in left parietal region with 7.0mm midline shift

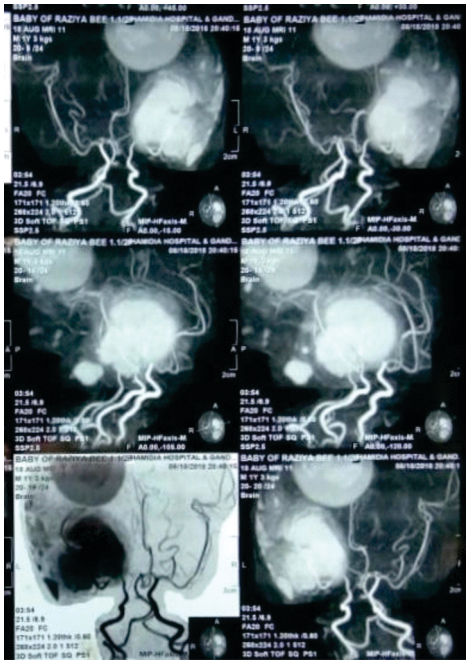
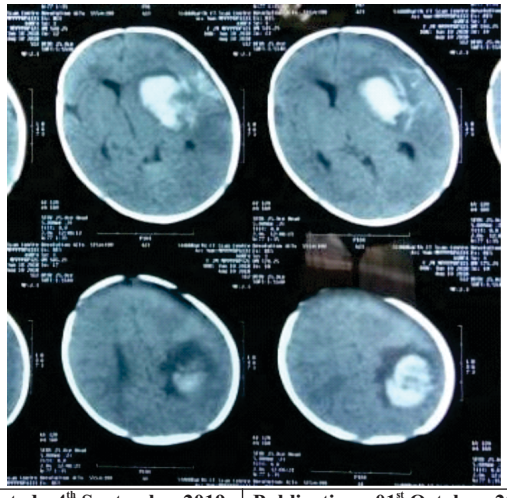


Figure 1- MR angiography showing huge parietal bleed in left hemisphere



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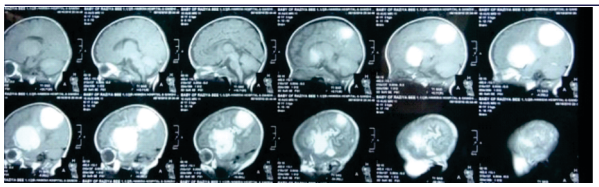

Figure 2 – CECT brain showing similar findings to MRI brain with midline shift

A diagnosis of late hemorrhagic disease of newborn was made. Baby received 2 units of fresh frozen plasma (FFP) and 2 units of packed red blood cells along with vitamin K over next 5 days. He was discharged after 1 week with minimal neurological sequelae with continued physiotherapy and follow up. Now his age is 8 months and is doing well with no neurological sequelae so far.

DISCUSSION

Late HDN is a rare disease with high mortality and morbidity[4]. Late HDN may be primary or secondary to several other disorders[5,6]. It is one of the most frequent causes of intracranial hemorrhage in infants. None of coagulation factor cross the placenta from mother to fetus ; at birth the concentration of vitamin K dependent factors (II, V, VII, IX, X) and contact factor (XI, XII) are reduced to about 50% of adult values and are further decreased in preterm infants. Lack of vitamin K administration on birth, exclusive breast feeding, chronic diarrhea and prolonged use of antibiotics make them more prone to vitamin K deficiency. Bleeding occurs because of insufficient vitamin K dependent coagulation factors activity. HDN is major probability in a bleeding infant if PT-aPTT is higher and fibrinogen level and platelet counts are normal. If bleeding stops and PT-aPTT returns to normal after vitamin K supplementation then diagnosis becomes more obvious.

Late HDN can present with convulsion, poor sucking, irritability and pallor. Hemorrhage of gastrointestinal system, mucosal membranes and skin can accompany the disease. Intracranial hemorrhage is the major cause of morbidity and mortality. Mortality is reported in 14-50% cases[7]. The intake of vitamin K in exclusively breastfed babies is less then 5 µg/L but formula fed infants intake is almost 50 µg/L. HDN is more frequent in babies who are born at home especially seen in developing countries[8].

Risk of intracranial hemorrhage in late HDN is reported in 50-80% of the cases. While subdural is the most common location for hemorrhage and subarachnoid is 2nd most common type. Zengin et al.[10] reported subdural, subarachnoid and intraparenchymal bleeding as 100%, 80% and 30%, respectively[10]. In a study fromTurkey convulsion and poor feeding were seen in 47% patient and pallor in 20%[7]. Most cases in developing countries like India of late HDN are found to be born at home and not given vitamin K prophylaxis[7]. Vitamin K prophylaxis reduces the incidence of late HDN from 5.1 cases per 100000 births by 90%. A single parenteral dose reduces the risk by a factor of 14.3[11]. Early diagnosis and supportive therapy improved the outcome in our case.The diagnosis of late HDN should be excluded as early as possible. In case of suspected intracranial bleeds due to late HDN, resorting to neuroprotective ventilation strategies along with therapy with parenteral vitamin K and plasma transfusion can decrease mortality and morbidity.

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