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Case report

LATE ONSET HEMORRHAGIC DISEASE OF NEWBORN DUE TO CMV HEPATITIS PRESENTING AS SCALP HEMATOMA

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ABSTRACT

Vitamin K deficiency bleeding (VKDB) according to recent studies is the preferred term for hemorrhagic disease of the newborn (HDN). This is due to deficiency of clotting factors as a result of vitamin K deficiency. VKDB was first described over a hundred years ago but its relationship to vitamin K was not released until 40 years later. Vitamin K is required for the production of clotting factors II, VII, IX and X. It is involved in the normal clotting of blood, is present in some plants and is also synthesized by some *E. coli* in the gut. Due to low levels of vitamin K all newborn infants are at risk of developing hemorrhagic disease of the newborn. The body has very limited ability to store the vitamin. We present an unusual case of Neonatal Hepatitis due to CMV as a rare cause of late onset vitamin k-deficiency bleeding.

Keywords: Cholestatic liver disease, Vitamin K, Vitamin K deficiency bleeding.

INTRODUCTION

Hemorrhagic disease of the newborn (HDN) is a coagulation disturbance in newborns due to Vitamin K deficiency. As a consequence of vitamin K deficiency there is an impaired production of coagulation factors II, VII, IX, X by the liver.[1,2] Newborns are relatively vitamin K deficient for a variety of reasons. They have low vitamin K stores at birth, Vitamin K passes the placenta poorly, the levels of vitamin K in breast milk are low and the gut flora have not yet been developed (Vitamin K is normally produced by bacteria in the intestines). HDN causes an increased risk of bleeding. The common sites of bleeding are the gastrointestinal tract, Mucous membranes, umbilicus, injection sites and circumcision.[3]

A number of review articles provide useful overviews of vitamin K and its importance to human beings. Bleeding disorders in newly born infants were first described over 100 years ago when Townsend reported 50 cases in 1894.[1] Vitamin K, however, was only discussed about 40 years later, by Dam, in a study of bleeding disorder in chickens.[1]

CASE REPORT

A term male baby appropriate for gestational age (AGA) with birth weight 2400grms was

delivered vaginally to a primigravida mother at a private hospital without any antenatal or postnatal complications, baby was discharged on breast feeding on 3rd day of life. The baby was asymptomatic till 1month 5days and was on exclusive breast feeding brought to our hospital with c/o convulsion and swelling over scalp on left side (7 x 8 cm), there being no h/o drug intake during pregnancy.

On physical examination baby had severe pallor, icterus, hepatomegaly, one oval shaped (7x8cm) swelling present over left fronto-temporo-parietal region, admission weight was 3400grms, head circumference was 34.5cm.

Investigations revealed Hb 5.9 %, TLC 18400/cumm, DLC (N 56%, L40%,M4%), platelet count 4.9 Lacks/cumm, prothrombin time(PT)>2 min, partial thromboplastintime (APTT)>2 min, LFT (Total Bilirubin 9.3gm/dl, Direct Bilirubin 5.6gm/dl, SGOT 290IU/L, SGPT235IU/L) , CSF study reaveled 2 cells, all lymphocytes with normal protein and sugar, CT scan suggestive of hemorrhagic contusions in left temporal-parietal region, generalized cerebral oedema, scalp hematoma ,USG abdomen showed Liver parenchymal disease. Torch study of baby CMV (cytomegalovirus) IgG & IgM Both positive also mother CMV reports shows IgG +ve, IgM -ve

Fig 1: Plain CT scan of brain

Respectively showing hemorrhagic contusion in left temero-perital region, generalized cerebral oedema, scalp hematoma

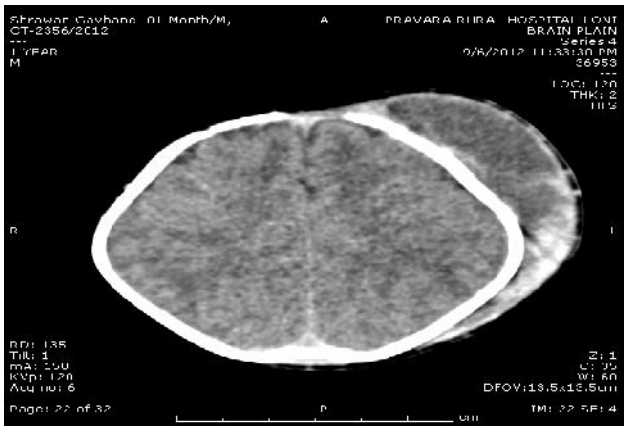

Fig 2: Contrast CT of brain

In treatment IV vit k 1mg, inj phenobarbitone, inj Gancyclovir (6mg/kg/day, 12 hrly) for 21 days with monitoring complete blood count every alternative day. Patient recovered completely.

On follow up patient showed complete recovery with normal hematological parameter and with normal growth and development.

DISCUSSION

In 1894, Townsend described a self-limited bleeding condition that usually occurs 1-5 days after birth in patients with nonclassic hemophilia.[1,4,5]The term vitamin K originated from coagulations-vitamin in German.[5] Henrik Dam and Edward Doisy won the 1943 Nobel Prize for the discovery and functions of vitamin K. Subsequent research has provided significant contributions to current knowledge of vitamin K and its association with coagulation factors, namely the vitamin K–dependent coagulation factors VII, IX, and X.[6]

- **Early VKDB** occurs within 24 hours of birth.
- **Classic VKDB** happens between day 1 and day 7 of life.
- **Late VKDB** occurs between week 2 and week 12 of life.

Late VKDB can result in significant morbidity and mortality due to intracranial hemorrhage and has resulted in most developed countries having in place a protocol for giving supplemental vitamin K to all newborn babies.

Late VKDB most commonly occurs at 2-12 week of age, although cases can occur up to 6 months after birth. All cases are in breast-fed infants due to low vitamin k content of breast milk. An additional risk factors are occult malabsorption of vitamin k (cystic fibrosis), cholestatic liver disease, pancreatic disease, intestinal disorders (celiac sprue, inflammatory bowel disease, short bowel syndrome). Cholestatasis in newborns can be due to infectious, genetic, metabolic or abnormalities giving rise to mechanical obstruction of bile flow or to functional impairment of hepatic excretion function and bile secretion[3]

Human cytomegalovirus (CMV) is widely distributed. Most CMV infections inapparent, but virus can cause a variety of clinical illness that range from mild to fatal. The incidence of congenital CMV infection ranges from 0.2% to 2.2% (average 1%) of all live births, with the higher rates among populations with a lower economic standard of living. The risk for fetal infection is greatest with maternal primary CMV infection (30%) and much less likely with recurrent infection (<1%).In infants and young children, primary CMV infection occasionally causes pneumonitis, hepatomegaly, hepatitis (cholestatic liver disease) and petechial rashes.[3]

In our case, patient had swelling over scalp with convulsion due to intra-cerebral contusion which was present with late VKDB due to unusual causes of hepatitis due to CMV infection.A randomized controlled study with ganciclovir (6 mg/kg/dose every 12 hrly for 2-4 weeks) concluded as a treatment for hepatitis due to CMV infection.

CONCLUSION

We suspected late VKDB due to its presentation at age of 4 weeks, PT/APTT had been improved after giving single dose vitamin k, reduction in swelling over scalp and also recovered from neonatal cholestasis(hepatitis) after giving inj Gancyclovir. CMV induced hepatitis is an unusual presentation of late VKDB. On follow up patient showed complete recovery with normal hematological parameter and with normal growth and development.

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