Case Report

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Late onset Vitamin K Deficiency Bleeding: A Preventable Disease

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Abstract

Vitamin K is an essential vitamin required for the activation of coagulation factors II, VII, IX, X and protein C and S. The deficiency of vitamin K leads to coagulopathy leading to the development of vitamin K deficiency bleeding (VKDB). We present a case of a 2-month-old male child who presented with tonic posturing of all 4 limbs. At birth vitamin k was not administered. Neonatal period was uneventful, and the child was exclusively breastfed. On further evaluation subdural hematoma was detected. A diagnosis of late onset vitamin k deficiency was suspected. Treatment with intravenous vitamin K was started with complete recovery. The diagnosis of late onset VKDB should be suspected when an intracranial hemorrhage in a previously healthy newborn is found specially when there is no history of Vitamin K administration after birth.

Keywords: Coagulation factor; Intracranial hemorrhage; Preventable Disease.

Introduction

Vitamin K is an essential cofactor for the γ-carboxylation process of factors 2,7,9,10 and protein C and protein S. Neonates are at risk for vitamin K deficient bleeding due to insufficient bacterial colonization at birth, low nutritional intake in exclusively breastfed babies, and poor placental transfer (VKDB). VKDB is of 3 types – early (<24 hours), classic (1-7 days) and late (> 2 weeks). The ingestion of oral anticoagulants, anticonvulsants, and antituberculosis agents by the mother increases the risk of early VKDB. These substances pass through the placenta and disrupt vitamin K metabolism. VKDB is caused by a physiologic vitamin K deficit at birth, which is compounded by an exclusive breast milk diet or insufficient feeding. Late VKDB appears in a newborn that is either primarily breastfed who obtains an insufficient dosage of vitamin K (none or one oral dose) or has an underlying illness condition that interferes with the absorption or supply of vitamin K. In the absence of vitamin K prophylaxis, the incidence of late VKDB is 4 to 10 per 100,000 births. When vitamin K prophylaxis is given intramuscularly (IM), the incidence of late VKDB drops to 0.24 to 3.2 per 100,000 live births.[1] Coagulation is caused by a lack of vitamin K-dependent procoagulant factors II, VII, IX, and X.

Protein Induced in Vitamin K Absence (PIVKA) is overproduced in cases of vitamin K deficiency, and its quantification is particularly useful in diagnosing the condition even after therapy has begun. If VKDB is suspected, vitamin K should be administered intravenously, intramuscularly, or subcutaneously as soon as possible. The diagnosis is confirmed when the PT and APTT improve 2

to 6 hours after receiving parenteral vitamin K. If a patient with VKDB has severe bleeding, further treatment with fresh frozen plasma (FFP) should be started. All newborn babies require vitamin K prophylaxis.

Oral route is effective like parenteral route but require higher and more doses. Except for babies with significant intestinal malabsorption, such as cystic fibrosis and cholestatic jaundice, intramuscular Vitamin K treatment potentially avoids the development of Late VKDB. To prevent late vitamin K deficiency bleeding, all breastfed newborns with diarrhoea and malabsorption require another dose of vitamin K in the post-neonatal period.

Case Report

1 month old male child presented with complaints of 2 episodes of tonic posturing of all 4 limbs and increased drowsiness.

He was born from unrelated parents at 39 weeks of gestation by normal vaginal delivery with no complications with birth weight of 2.8 kg and a 5 min APGAR of 10. Mother reported no use of drug during pregnancy. There was no history of vitamin K administration after birth. Neonatal period was uneventful, and the child was exclusively breastfed.

On admission the baby was afebrile with heart rate of 150/min and respiratory rate of 32 CPM, peripheral pulses were well felt, blood pressure was 86/62 mm hg (between 50th to 90th centile) and SpO2 of 95% on room air. He was lethargic with GCS of 12/15, pupils were bilaterally equally reactive and deep tendon reflex were brisk in both upper and lower limbs. After securing IV access patient was started on IV phenytoin and maintenance fluids.

Blood test showed anemia, normal platelet count (Table 1). Serum electrolytes were normal. Coagulation profile showed deranged PT and aPTT and normal levels o fibrinogen.

MRI Brain was done which showed early subacute subdural hematoma in the right fronto-temporo-parietal region causing midline shift (Figure 1 and 2).

Based on the history and laboratory findings a diagnosis of Late onset Vitamin K deficiency bleeding was suspected. Levels of PIVKA was sent to confirm the diagnosis which came elevated (35,000).

IV Vitamin K 10 mg was administered and 1 FFP was transfused. Considering low hemoglobin values, he was transfused 1 PCV without any adverse reaction. Repeat coagulation study showed normal PT and aPTT values. Burr hole evacuation was done to drain the hematoma in which 25 ml of blood was drained.

IV Vitamin K 10 mg was continued for 5 days followed by oral vitamin K twice weekly. During hospitalization, he did not have any further bleeding or any other symptom. Coagulation parameters were normal at discharge and hemoglobin was increased. He was sent home with instructions to take 10 mg of vitamin K twice a week for the next month.

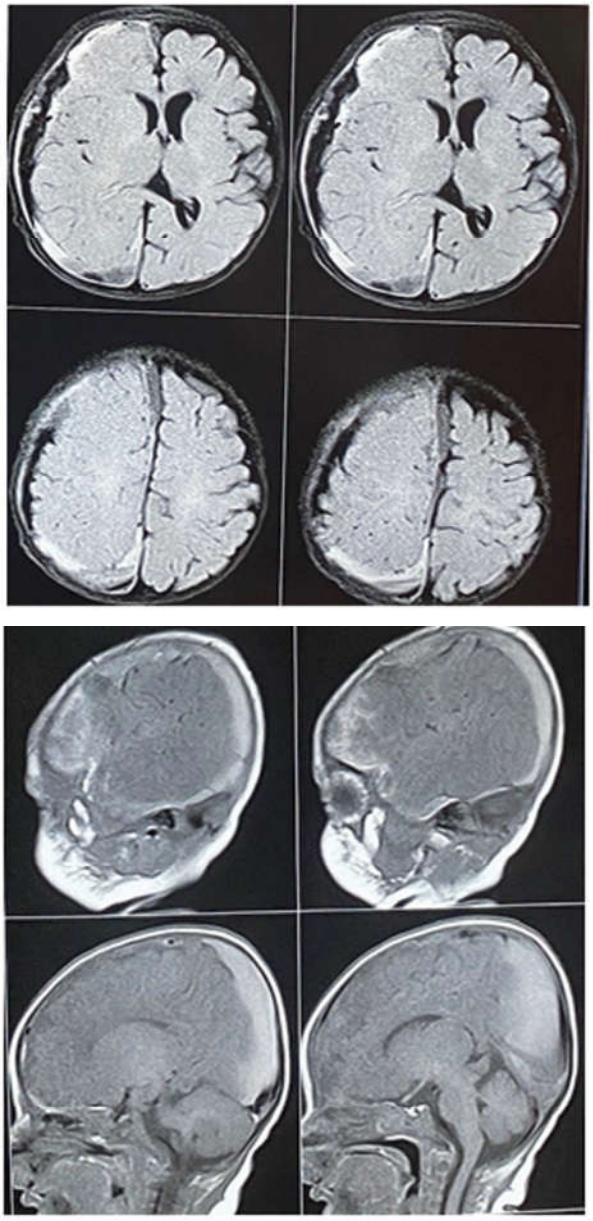

Fig. 1: Showing subdural hematoma in the right fronto-temporo-parietal region.

Test	Result	
Hb	At Admission 6.6	At Discharge 8.8
PCV	19.2	26.8
Platelet	2,44,000	2,51,000
PT	60	11
INR	4.8	1.12
aPTT	82	24
Fibrinogen	322	312
AST	26	28
ALT	24	22
PIVKA	78,000	

Discussion

Administration of vitamin K 1 mg intramuscularly is recommended to prevent VKDB. This advice was based on findings that intramuscular treatment was more clinically efficacious than oral administration, and that multiple oral doses were needed to provide sufficient protection against late VKDB onset in breastfeeding children.

Due to the shorter duration of action of oral vitamin K compared to intramuscular administration, a single vitamin K oral dosage at birth is effective in preventing classic VKDB but is ineffective in preventing late VKDB.

Golding et al. reported a case control study in 1992, concluding that vitamin K administered intramuscularly at birth increased the incidence of cancer and leukaemia in future children when compared to oral or no treatment. However, further research found no link between parenteral vitamin K prophylaxis at birth and the development of malignancies in children.[2] Late VKDB should be suspected when an intracranial hemorrhage is found in a previously healthy newborn. Central nervous bleeding is the first clinical manifestation of late VKBW in about 50 to 60% of the cases.

The 1-month-old had no history of prolonged jaundice, bowel or liver diseases. No history of trauma was noted, and adequate post-natal growth was present which ruled out malabsorption. Since the baby was delivered at home, he did not receive vitamin K injection after birth which raised the possibility of late onset vitamin K deficiency bleeding. Raised PT, aPTT, a normal platelet and fibrinogen levels and a raised levels of PIVKA confirmed the diagnosis of late vitamin K deficiency bleeding. Estimation of the levels of PIVKA is extremely helpful in confirming the diagnosis and also very cost effective.

Conclusion

Late Vitamin K deficiency bleeding is a preventable cause of intracranial hemorrhage in a newborn. All newborns should receive IM Vitamin K1 mg after birth. LHDN should be suspected when an Intracranial hemorrhage in a previously healthy newborn is found specially when there is no history of Vitamin K administration after birth. Timely diagnosis is very crucial in preventing the mortality and morbidity associated with it.

References

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