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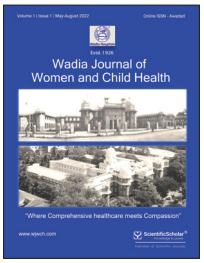
Wadia Journal of Women and Child Health



Case Report

A term neonate with intracranial bleed

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Received: 27 March 2024
Accepted: 22 May 2024
Published: 15 March 2025

DOI
10.25259/WJWCH_13_2024

ABSTRACT

A term male neonate with a birth weight of 3000 grams presented with complaints of multiple episodes of vomiting and seizures on 27th day of life. On examination, the neonate was stuporous with bulging anterior fontanelle and anisocoria. The seizures were controlled with multiple aniticonvulsants. Urgent non contrast computed tomography of brain revealed subdural hemorrhage along right cerebral convexity with mass effect and midline shift. The baby had a prolonged prothrombin time and activated partial thromboplastin time with normal platelet count suggestive of vitamin K deficiency. Other factor levels were within normal limits. The neonate required a frontoparietal craniotomy for evacuation of the subdural hematoma. Vitamin K deficiency was corrected with intramuscular vitamin K injection and the baby was discharged without any focal neurological deficits. At follow up, 1 month post discharge, the neurological examination was normal.

Keywords: Late-onset Vitamin K deficiency, Intracranial bleed, Bleeding neonate

INTRODUCTION

A bleeding neonate is a neonatal emergency and the etiological factors include abnormalities in the coagulation pathway. Postnatal intracranial bleed presents with acute neurological deterioration and requires prompt medical and surgical management. Late-onset Vitamin K deficiency occurring in an otherwise healthy neonate who has received Vitamin K at birth requires reporting.

CASE REPORT

A term male baby, born out of a non-consanguineous marriage to a 23-year-old primigravida through vaginal route in a hospital with a birth weight of 3000 g, was referred to our neonatal intensive care unit on day of life (DOL) 27 with complaints of multiple episodes of vomiting and seizures.

Antenatal sonography was normal and the mother had no medical or obstetric illness during pregnancy. The baby was vigorous at birth with APGAR scores of 9 at both 1 and 5 minutes. He was on exclusive breastfeeding and discharged from the hospital on DOL-2 and the course in the first 26 days remained unremarkable.

How to cite this article: Nair S, Jyothi S, Prashanth RR, Sharma U, Haribalakrishna A. A term neonate with intracranial bleed. Wadia J Women Child Health. 2024;3:133-6. doi: 10.25259/WJWCH_13_2024

On admission, he was moderately stuporous with a Glasgow coma scale (GCS) score of 4. Head circumference was 37 cm (between the 50th and 95th centile as per the World Health Organization growth charts) with a bulging anterior fontanelle and anisocoria. There was no facial dysmorphism and the examination of other systems was unremarkable. As seizures persisted, anti-epileptic drugs were escalated in the order of phenobarbitone, phenytoin, and continuous infusion of midazolam. Amplitude-integrated electroencephalogram confirmed status epilepticus. The neonate was mechanically ventilated for reduced spontaneous breathing efforts and following control of seizures, the vitals including blood pressure were normal. An urgent non-contrast computed tomography (NCCT) brain was done, which revealed hyperintense subdural hemorrhage along right cerebral convexity with a maximum thickness of 11 mm with mass effect and midline shift of 13.7 mm toward the left side [Figure 1a].

The details of the diagnosed workup are shown in Table 1.

Factors 8, 9, and 13, were normal. Decarboxy prothrombin (PIVKA II) level was elevated to 89020 mAU/mL (1000–3000 mAU/mL). On enquiry, there was no history of trauma. There were no fractures seen on the infantogram and the fundus examination was negative for retinal hemorrhages. The birth records were re-verified and the neonate had received Vitamin K at birth. Hence, the neonate was diagnosed as a case of late-onset Vitamin K deficiency with subdural hemorrhage.

Table 1: Lab investigations.

Lab parameter	Result	Normal range
Hb	14.4 g/dL	13–16 g/dL
Platelet	2.4 lakhs	>1.5 lakhs
PT	37.5 s	Control 13.6 s
APTT	61.7 s	Control 30.9 s
INR	2.97	1–1.5

Hb: Hemoglobin, PT: Prothrombin time, APTT: Activated partial thromboplastin time, INR: International normalized ratio

The neonate was started on measures to reduce intracranial pressure including head end elevation, and 3% saline infusion. After correction of coagulation abnormality with fresh frozen plasma transfusion and Vitamin K injection (1 mg intramuscular injection in the anterolateral thigh), he was taken for emergency craniotomy for hematoma evacuation. A right frontoparietal craniotomy was done, with the removal of around 300 mL of blood clot.

The neonate had good spontaneous breathing efforts post-surgery and was extubated 24 hours later. The sensorium also showed marked improvement compared to the pre-surgery state, with the patient attaining a pediatric GCS of 14 at 38–40 hours post-surgery. Anti-epileptic drugs were gradually tapered off and repeat coagulation profile including a thromboelastogram was normal and repeat PIVKA levels were normal. Repeat computed tomography (CT) brain done before discharge showed resolution of the bleed with residual cerebral edema and occipital cortical necrosis [Figure 1b]. Detailed neurological examination at discharge and 1-month post discharge revealed no neurological abnormality and is currently on regular follow-up with normal growth and neurodevelopment [Figure 2].

DISCUSSION

Vitamin K deficiency bleeding (VKDB) refers to spontaneous bruising, bleeding, or intracranial hemorrhage associated

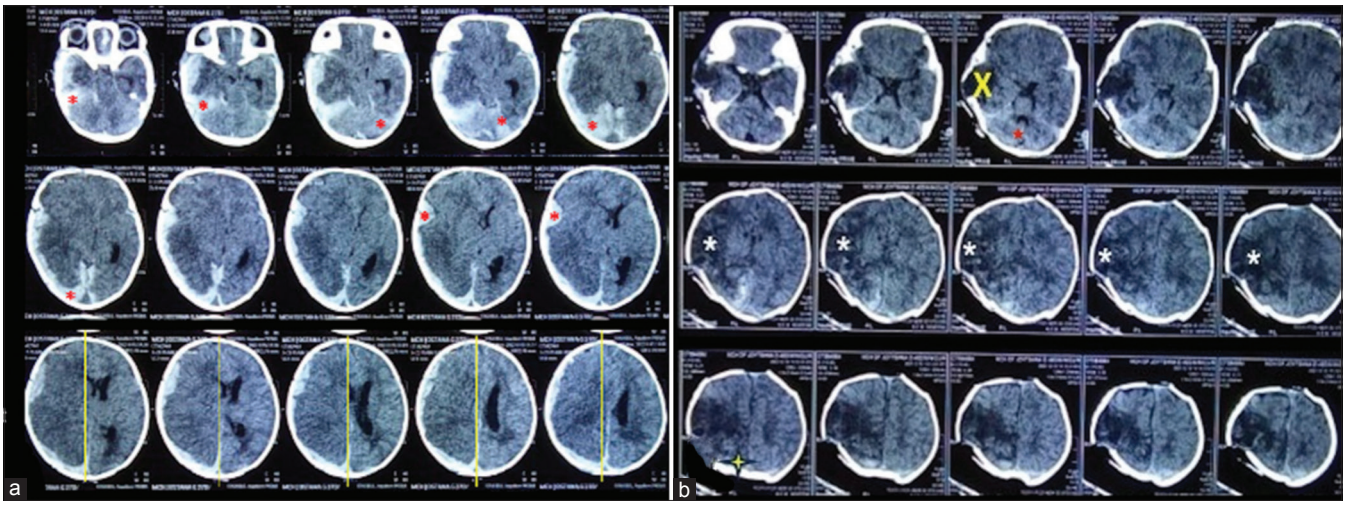

Figure 1: (a) Non-contrast computed tomography (NCCT) brain before surgery showing subdural hemorrhage with mass effect and midline shift of 13 mm as indicated by red asterisk. (b) NCCT brain post-surgery day of life 50. The yellow cross mark represents the status post decompressive craniotomy with pneumocephalus. White asterisk *represents diffuse cerebral edema persisting. *A red asterisk marks the occipital cerebral necrosis.

with the prolonged clotting time that is not due to disseminated intravascular coagulation or an inherited coagulopathy.[1] Neonates are at increased risk of developing VKDB due to multiple reasons such as low transplacental transmission of Vitamin K, decreased Vitamin K in breast milk, lack of intestinal flora for Vitamin K synthesis, and immaturity of the liver.

Depending on the age at presentation, VKDB can be of three types which include early, classical, and late VKDB, each with its own specific mechanism. Early Vitamin K deficiency presents within 24 h of life and occurs as a result of placental transfer of drugs that inhibit Vitamin K. Classical form usually presents within the 2nd–7th DOL and occurs as a result of poor concentration. This is a result of poor transplacental transfer of Vitamin K from the mother to the fetus resulting in low Vitamin K levels in the neonate. Late VKDB can present from 2nd week to 6 months of life and is predisposed by hepatobiliary/intestinal disease, or inadequate supplementation due to low Vitamin K levels in breast milk which we postulate as the reason for late VKDB in this neonate. Bleeding manifestations are comparatively severe with intracranial bleeding being the most common manifestation.

John *et al.* reported a similar case of a 4-month-old neonate, despite having received an intramuscular Vitamin K injection at birth, presented with seizures and vomiting and on evaluation was found to have bilateral acute on chronic subdural hemorrhage.[2] Another rare presentation of the case was reported by Palau *et al.* where a 1-month-old infant had presented with anterior mediastinal mass and scrotal bruising. Anterior mediastinal mass on evaluation was found to be due to spontaneous thymic hemorrhage. Here, the patient did not receive the recommended Vitamin K prophylaxis at birth.[3]

Figure 2: (a) Infant at discharge: Neurologically normal. (b) At 1 month post-discharge.

The incidence of late VKDB in the absence of Vitamin K prophylaxis ranges from 10.5 to 80/100,000 births. In patients who have received intramuscular Vitamin K prophylaxis, the incidences decreased to 0.24–3.2 cases/100,000 live births.[4] Our patient falls in this category where despite having received a dose of intramuscular Vitamin K, he developed bilateral subdural hemorrhage on DOL 27.

There is a lack of validated tools for the assessment of consciousness in neonates; hence, we chose to use the pediatric GCS and management of raised intracranial pressure using 3% saline also lacks well-conducted trials in neonates.[5] The decision to perform surgery in such patients depends on the clinical assessment where a sudden worsening of neurologic status always warrants immediate surgery. Urgent NCCT brain and decompressive craniotomy form the cornerstone in the management of these neonates and in those with neurologic manifestations particularly those aged 2 or younger, urgent NCCT is recommended.[6]

An acute subdural hematoma (SDH) usually appears hyperdense on the CT scan, which is secondary to the hyperintensity of coagulated blood (density of 60–80 Hounsfield units). However, in the hyperacute stage, the SDH may appear heterogeneous with hyperdense and isodense images. This is due to the presence of non-coagulated blood in connection with bleeding disorders or active bleeding. The index neonate also had a hyperdense lesion on CT scan.[7]

The role of craniotomy versus craniectomy is still debatable in neonates. However, in studies among older children and adults with traumatic acute SDH who underwent craniotomy or decompressive craniectomy as a primary management, disability and quality-of-life outcomes were similar to the two approaches.[8] The most favorable outcomes are usually seen in subdural hemorrhage, with 80% of patients having no cognitive or motor deficit.[8] The worst outcomes are seen in patients with subarachnoid hemorrhage or when multi-compartment involvement is noted.

CONCLUSION

Healthy term neonates can present with intracranial bleeding secondary to late onset vitamin K deficiency despite receiving vitamin K prophylaxis at birth, even in the absence of recognizable risk factors. A single dose of injection vitamin K, correction of coagulopathy and management of raised ICT are the key aspects of medical management. Urgent craniotomy and evacuation of intracranial hemorrhage lead to early recovery and prognosis.

Ethical approval

Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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