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Case Report

Paediatrics Section

# Subgaleal Haematoma due to Vitamin K Deficiency in an Infant: A Case Report

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## ABSTRACT

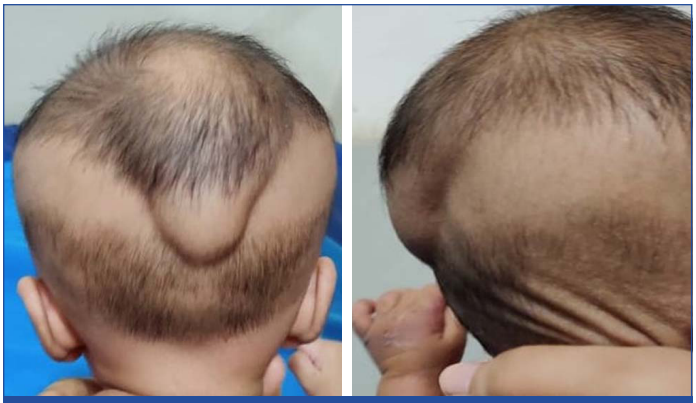
Haemorrhagic Disease of the New Born by Vitamin K Deficiency Bleeding (VKDB), occurring shortly after birth and caused by deficiency of vitamin K dependent factors (factors II, VII, IX, and X) has been well documented. Subgaleal haemorrhage is an infrequent but fatal complication of childbirth, especially if accompanied by coagulation disorders. It can be due to many causes like traumatic birth, coagulation factors deficiency, vitamin K deficiency etc. The most common cause is traumatic birth. Authors hereby report a case of subgaleal haematoma in an eight-week-old female child secondary to vitamin K deficiency. The patient responded well following administration of parenteral vitamin K.

**Keywords:** Coagulation factors, Haemorrhage, Trauma

## CASE REPORT

An eight-week-old female child was admitted in the Paediatrics Department with a chief complaint of swelling over the scalp since 10 days. The swelling was present on the parieto-occipital region, gradually progressing in size. There was no history of swelling since birth or delayed cord falling or umbilical discharge or bleeding. The guardian of the patient gave no history of fall or trauma. There was no significant past history. In the family, there were no similar complaints or history of bleeding disorder or transfusion. The baby was born of non consanguineous marriage and was a first order child via spontaneous vaginal delivery without any history of trauma, prolonged labour or any instruments used during birth. There was no record of vitamin K given after birth. The baby was exclusively breastfed. The patient was immunised according to age and there was no history of haematoma formation after vaccination. Later the baby developed swelling which gradually increased in size.

On examination, the baby was alert and playful. The baby was vitally stable and accepted feeds well orally. The swelling was present over the parieto-occipital region measuring 7x7 cm [Table/Fig-1,2], which did not have well-defined margins, was non pulsatile, soft and non tender and was not fixed to bone or skin. There was no skin changes over the swelling and the temperature was normal. It was movable and crossed the suture lines. No signs of inflammation and induration was present. It showed a positive transillumination test. On general examination, pallor was present with no oedema or lymphadenopathy. Anterior fontanelle was open, measuring 2×3 cm, non bulging with normal tension. No signs of bleeding manifestations like petechiae, purpura, or bruises were present. The ophthalmic examination and systemic examinations were normal.



**[Table/Fig-1]:** Swelling (7x7 cm), non erythematous, over parieto-occipital region.
**[Table/Fig-2]:** Lateral view of the swelling. (Images from left to right)

Blood investigations included Complete Blood Count (CBC) which revealed low haemoglobin (11 gm/dL) with normal total leucocyte count of 6100 cells per $mm^3$ and platelet count of 4,52,000/µl with deranged Prothrombin Time (PT), activated Partial Thromboplastin Time (aPTT) and international normalised ratio [Table/Fig-3]. Liver function test was normal. Ultrasonography of skull revealed a diffuse anechoic collection measuring 4×3.1 cm, noted in subcutaneous plane at the site of swelling in the parieto-occipital region of the head and with no features of intraventricular haemorrhage, suggestive of subgaleal aponeurosis.

One dose of injection vitamin K of kenadion i.e., 1 (1 mg/0.5 mL) in a dose of 0.5 mL was given, after which PT/APTT was reduced and injection vitamin K was continued for total of three days after which repeated PT/APTT was found to be normal [Table/Fig-3]. The swelling gradually decreased and the baby was vitally stable. The patient was discharged on supplements.

| Variables | PT/APTT (sec) | INR |
|---|---|---|
| On Admission | 31.46/55.23 | 2.27 |
| After 1st Dose (24 hr) | 18.6/42 | 1.3 |
| After 3rd Dose (24 hr) | 16.4/36.43 | 1.1 |
| After 2 Weeks | 16.4/37.4 | - |

**[Table/Fig-3]:** Coagulation profile on admission and after treatment.
INR: International normalised ratio, PT/aPTT: Prothrombin time/activated partial thromboplastin time

On follow-up after two weeks PT/APTT were within normal limits. Thrombin Time was 14.6 seconds, platelet aggregation test with Platelet Rich Plasma (PRP) showed: Adenosine diphosphate (ADP=10 µM): 75%, Ristocetin (1.25 mg/mL): 80%. Factor assay was normal. On further follow up of two weeks, the swelling gradually decreased in size to 6X5 cm [Table/Fig-4]. Since then, the patient did not come for further follow-up. The patient was diagnosed as late form of Vitamin K Deficiency Bleeding (VKDB), presenting as subgaleal haematoma.

**[Table/Fig-4]:** Swelling on two weeks of follow-up. The size of the swelling reduced to 6×5 cm.

## DISCUSSION

Vitamin K deficiency bleeding, earlier known as haemorrhagic disease of the new born, is a type of coagulopathy in infants which occur in those who has low vitamin K stores. Vitamin K is a cofactor for the activation of coagulation factors II, VII, IX, X, and protein C and S. These proteins are produced into the bloodstream and aid in the conversion of fibrinogen to fibrin, resulting in the formation of a haemostatic thrombus, resulting in an elevated bleeding risk in these newborns [1-3].

Multiple risk factors for VKDB exist in newborns, particularly those who are exclusively breastfed, because vitamin K transfer in placental circulation is inadequate, with extremely low cord concentrations compared to adult levels [1], infant liver reserve levels are significantly lower than adult levels, low vitamin production is due to immature gut flora, and vitamin K content in breast milk is significantly low [3-5].

The VKDB comes in three varieties: early, classic, and late. Infants born from mothers who were treated during pregnancy with anticonvulsants, antitubercular agents, certain antibiotics such as cephalosporins, or vitamin K antagonists (warfarin) and who did not get vitamin K prophylaxis before birth develop early form during the first 24 hours of life [2]. Without vitamin K supplementation, the incidence in at-risk infants ranges from 6% to 12%.

The classic form occurs between 1 to 7 days of life and is generally idiopathic. Term new born who does not receive injection vitamin K at birth, presents with incidence of 0.01-0.44% [6]. This is associated with the low placental transfer of vitamin K, low concentration in breast milk, absence of gastrointestinal flora in the newborn gut.

The late form occurs between the second and sixth months of life, with a peak between three and eight weeks after delivery, and is more common in exclusively breastfed infants or neonates with malabsorption or cholestasis, as vitamin K absorption is dependent on bile availability in the intestine. The haemorrhagic manifestations mainly involve gastrointestinal tract and skin most commonly, however the central nervous system may also be affected [7,8].

In the present case, the mother was unaware about whether she received the vitamin K prophylaxis before pregnancy and the child presented at 7-8 weeks of life with no traumatic birth history, history of trauma, no neurological manifestation, exclusively breastfed and improved on vit k administration, hence was diagnosed as late VKDB with no neurological defects.

Infants with VKDB should receive 1 mg of parenteral vitamin K. For rapid correction in adolescents, parenteral dose is 2.5-10 mg. In addition to vitamin K, severe life threatening bleeding should receive FFP infusion.

Subgaleal haematoma is a lethal condition because the blood loss causes hypovolemic shock. During a six year period study it was found that 26 of 27 patients with Subgaleal Haemorrhage (SGH) had been delivered by vacuum extraction [9]. SGH is more common in vacuum and forceps assisted deliveries, but this also occur in spontaneous vaginal deliveries associated with macrosomia, foetal distress or prolonged second stage of labour and in the present case, the delivery was spontaneous and non traumatic [9]. SGH is due to rupture of the emissary veins, which are present between the dural sinuses and the scalp veins [10]. Instrumented delivery, trauma, and coagulation abnormalities are all possible causes of SGH. It's more common with vacuum and forceps deliveries, although it can sometimes happen on its own [11-14]. SGH can lead to severe hypovolemia, and approximately one-quarter of babies who require neonatal intensive care for this condition die. Detailed history, physical examination and relevant routine and specific investigations should be done. Vitamin K-Prophylaxis is very important to prevent these haemorrhagic diseases of the newborn. Both oral and intramuscular vitamin K (one dose of 1 mg) protect against classical VKDB, while intramuscular vitamin K prevents late VKDB better.

Evaluation of the severity of neonatal subgaleal bleeding can be measured through frequent blood pressure monitoring, Complete Blood Counts (CBC), and platelet counts, as well as by coagulation studies. For accurate diagnosis, computed tomography is recommended.

The Canadian Paediatric Society and the College of Family Physicians of Canada recommends the following initial stabilisation and acceptable maternal/newborn interaction, one IM dosage of vitamin K (0.5 mg for infants weighing ≤1500 gm or 1 mg for infants weighing >1500 gm) is typically administered to all newborns within six hours of birth [15].

## CONCLUSION(S)

The present case highlights the importance of administration of vitamin K at birth, as SGH can also present later in a child born via spontaneous delivery without any instrumentation or trauma, due to vitamin K deficiency.

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