

# Classical vitamin K deficiency bleeding in newborn with mother on antitubercular drugs

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## Abstract

Early vitamin K deficiency bleeding in neonate born to a mother on antitubercular drugs is a known entity. We report a case of a full term, small for gestation age baby born per vaginally to a mother on antitubercular drugs. The baby developed classical vitamin K deficiency bleeding in the form of intracranial bleed on third day of life in spite of receiving vitamin K at birth.

**Key words:** New born, haemorrhagic disease, vitamin K, Antitubercular drugs

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## Introduction

Vitamin K deficiency bleeding (VKDB) in infancy is an acquired coagulopathy secondary to reduction of vitamin K (VK)-dependent coagulation factors below hemostatic levels [1].

The clinical forms of hemorrhagic disease due to Vitamin K (VK) deficiency are classified into early, classic and late on the basis of age of onset and etiology.

Early VKDB (onset less than 24 hours of age) is almost exclusively due to placental transfer of maternal drugs like anticonvulsants, antitubercular drugs and VK antagonists which inhibit vitamin K (VK) activity in the baby. Classical VKDB (onset between 2 to 7 days) occurs primarily in exclusively breast fed infants due to delayed or inadequate feeding and who have received no or inadequate neonatal VK prophylaxis. Late hemorrhagic disease (onset between 8 days to 12 weeks) occurs in exclusively breast fed infants who have not received VK prophylaxis at birth or have suffered from chronic diarrhea or cholestatic liver disease [2]. We report a case where a neonate born to a mother on antitubercular drugs developed classical VKDB in spite of receiving Vitamin K prophylaxis.

## Case Report

A full term, small for gestation, male baby with birth weight 2.2 kg was born to a second gravida mother per vaginally. The baby did not require any resuscitation, received vitamin K prophylaxis and was exclusively breast fed. The baby was admitted in neonatal intensive care unit at the age of 56 hours with complaints of refusal to feed and irregular breathing. On examination baby had severe pallor, cyanosis, tachycardia, shallow respiration, prolonged capillary refill time and feeble peripheral pulses. He had a bulging and tense anterior fontanelle of size 3x3 cm. He also had a haematoma on the left thigh at the site where vitamin K injection was given and was bleeding from all pricking sites. His central nervous system (CNS) examination showed hypertonia and dilated but weakly reacting pupils. His investigations revealed haemoglobin of 7 gm/dl, normocytic normochromic anemia, wbc count 20,000/cumm, platelet count 2,20,000, prolonged prothrombin time (PT) 120 seconds, partial thromboplastin time 100sec and normal liver function tests (LFTS). Ultrasonography of skull showed large intraparenchymal and intraventricular bleed. Initial septic workup was normal. The baby was resuscitated, put on mechanical ventilator. He received parenteral vitamin K, fresh frozen plasma and packed cell transfusion, but died on eighth day of hospitalization due to ventilator associated pneumonia and coagulase negative staphylococcus sepsis. Maternal history

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revealed intake of isoniazid and rifampicin by mother since 5 months of gestation with normal LFTS. Based upon the age, clinical presentation, abnormal PT and maternal history a diagnosis of classical VKDB was made.

## Discussion

Antenatal use of antitubercular drugs usually leads to early VKDB where as classical VKDB occurs in exclusively breastfed babies due to delayed or inadequate feeding. V<sub>k</sub> prophylaxis at birth helps in preventing classical VKDB [1,3,4]. Our case had atypical features. Firstly, the baby born to a mother on antitubercular drugs had bleeding on the third day of life and hence the presentation was in classical form. Secondly classical VKDB commonly presents with bleeding from umbilicus, circumcision site and gastrointestinal tract, but our case had intracranial bleed which is a rare manifestation of classical VKDB. Late HDN commonly presents with intracranial bleed as is suggested by various studies and case reports [5,6,7].

Thirdly, our case developed VKDB inspite of receiving prophylactic vitamin K at birth. Now the question arises, is there any way by which this could have been prevented? It has been proved in various studies that early VKDB can be prevented by prophylactic use of vitamin K antenatally in mother on antiepileptics [4,8,9,10].

But whether similar prophylaxis will also help in preventing VKDB in neonates with mother on antitubercular drugs, more studies need to be done in this regard.

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## Conclusion

VKDB in neonate born to a mother on antitubercular drugs can occur inspite of VK prophylaxis at birth and present in classical form also. Hence the role of antenatal vitamin K prophylaxis in such mothers need to be studied.

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