

Vitamin K Prophylaxis

Approved – June 2009

For Review – June 2011

Preamble

Guidelines outline recommendations, informed by both the best available evidence and by midwifery philosophy, to guide midwives in specific practice situations and to support their process of informed decision-making with clients. The midwifery philosophy recognizes the client as the primary decision maker in all aspects of her care and respects the autonomy of the client (1).

The best evidence is helpful in assisting thoughtful management decisions and may be balanced by experiential knowledge and clinical judgment. It is not intended to demand unquestioning adherence to its doctrine as even the best evidence may be vulnerable to critique and interpretation.

The purpose of practice guidelines is to enhance clinical assessment and decision-making in a way that supports practitioners to offer a high standard of care. This is supported within a model of well-informed, shared decision-making with clients in order to achieve optimal clinical outcomes.

Vitamin K Deficiency Bleeding

Hemorrhagic disease in the newborn or, according to the Committee of the International Society on Thrombosis and Hemostasis, vitamin K deficiency bleeding is thought to occur due to a lack of Menaquinone producing bacteria in the intestinal tracts of babies (2).

There are three types of Vitamin K Deficiency Bleeding (VKDB): Early VKTB, Classical VKTB, and Late VKTB. Each varies in its time of onset and affected site.

Early VKTB: Onset occurs between 0-24 hours. Usually the result of a predisposing factor such as preterm intracranial hemorrhage, trauma at birth or an infection. The following sites are affected: cephalohematoma, umbilicus, intracranial, intra-abdominal, intrathoracic and gastrointestinal.

Classical VKTB: Onset occurs between days 1-7. The affected sites are: gastrointestinal tract, umbilicus, nose, needle prick sites, circumcision, and intracranial.

Late VKDB: Onset occurs between 2 weeks to 6 months. The cause at this time appears to be idiopathic. The affected sites: intracranial (30-60%), skin, nose, gastrointestinal tract, needle-prick sites, umbilicus, urogenital tract, intrathoracic. Late VKDB has a 14% fatality rate (3).

VKDB can lead to spontaneous bleeding beneath the skin, from the nose, stomach, intestines, wounds or intracranial bleeding. This bleeding can lead to permanent brain damage or death. The morbidity correlates with the severity of the vitamin K deficiency.

Vitamin K is a fat soluble vitamin of which there are two types. Phylloquinone (vitamin K1) is found in some green plants. Menaquinone (vitamin K2) is synthesized by bacteria in the human gut. This second type of vitamin K accounts for 90% of the vitamin K found in the liver which is necessary for the synthesis of prothrombin and clotting factors II, VII, IX, and X. A deficiency in vitamin K leads to increased coagulation time and potentially spontaneous bleeding (long term it can affect the ability of the body to regulate calcium and bone development).

Prevalence

The incidence of VKDB is thought to be between 5-25/100,000 with a median of 7.1/100,000 in developed countries for untreated neonates (4). However, the administration of IM vitamin K reduces this approximately 1/1 million (5).

Risk factors

- Drugs taken in pregnancy (anticonvulsants, anticoagulants, tuberculostatics, and cephalosporins)
- Marginal vitamin K in breast milk (not to be taken as an endorsement for formula)
- Inadequate milk intake
- Late onset of feeding (colostrum has higher concentrations of vitamin K than breast milk)
- Malabsorption of vitamin K (liver or bowel disease)
- More common in summer months
- More common in males
- Prematurity
- Instrumental delivery
- Perinatal asphyxia
- Surgical procedures such as circumcision

Clinical signs and symptoms

- Hematemesis (vomiting of blood)
- Prolonged jaundice
- Melena stools (difficult to differentiate from meconium)
- Failure to thrive
- Prolonged bleeding at puncture sites
- “warning bleeds” such as bleeding from the umbilicus, nose or mouth (6)

Diagnosis

The diagnosis is established by performing clotting studies. Prothrombin time (PT), usually reported as the INR, is prolonged, but partial thromboplastin time (PTT), thrombin time, platelet count, bleeding time, and levels of fibrinogen, fibrin-split products, and D-dimer are normal.

Prevention

Vitamin K is recommended as a prophylaxis for VKDB and has been administered routinely since the 1950's in North America. The risk of a baby developing VKDB can be reduced to 1/1million by the administration of exogenous Vitamin K within six hours of birth (7).

There are two routes of vitamin K administration: intramuscular and oral. Currently intramuscular is the recommended route of administration (8). However, research has shown that three oral doses of Vitamin K can produce the same indices of coagulation factors as one intramuscular dose. The effectiveness of oral Vitamin K against VKDB has not been determined largely due to the fact that the incidence of this condition is so rare (9).

Intramuscular

This route is recommended for its high efficacy rate and high compliance rate. It is given as a single dose of 1.0 mg (>1500g) or 0.5 mg (<1500g).

Oral

This may be an alternative for parents who decline the IM route out of concern for the pain it might cause. In this case the BCPHP recommends administering vitamin K in 3 doses of 2mg each: at the first feed, at 2-4 weeks, and at 6-8 weeks. This route is thought to reduce the incidence of VKDB to 4/1million (10).

Some disadvantages of Vitamin K Prophylaxis

- A retrospective British Study conducted in the early 1990s reported an association between Vitamin K administration and childhood leukemia. Further research has been conducted and the findings have not been duplicated (11).

- Oral administration poses some challenges in that babies may regurgitate some of the medication, compliance over several weeks is required, there is no oral form licensed in Canada, and the cumulative amount of oral vitamin k administered is six times the IM dose (12).

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