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ISSN: 2161-0509
Journal of Nutritional Disorders & Therapy

Opinion Article

Vitamin K Prophylaxis in Newborn Infants and their Perinatal Risk Factors

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DESCRIPTION

Coagulation factors II (prothrombin), VII, IX, and X are the most well-known proteins that must be changed and activated in the presence of vitamin K. Protein C, the crucial coagulation inhibitor, as well as other proteins with particular roles, all depend on vitamin K. Important duties for the pediatric healthcare professional include assessing and addressing parenteral problems about vitamin K. Parents must be aware of the importance of vitamin K and have a fundamental understanding of its role in order to make an informed decision regarding their child. The Centers for Disease Control and Prevention offer a fantastic fact sheet about vitamin K.

The study of full-term infants is delivered by means of non-instrumental delivery, between injectable vitamin K prophylaxis to the newborns and childhood cancer which could not be confirmed. The low levels of vitamin K detected in newborns are due to minimal amounts of vitamin K being transported over the placenta to the fetus. Many newborn infants prefer breast milk as their primary source of nutrition, but because it contains relatively little vitamin K, infants who are solely breastfed are at an increased risk of developing VKDB. From 6 weeks to 6 months after birth, infants who are exclusively breastfed frequently have vitamin K plasma concentrations below adult norms due to their low baseline levels and poor intake.

Despite receiving IM vitamin K at birth, this drop in plasma vitamin K still occurs. Breastfeeding was recognized as a crucial component in VKDB more than 50 years ago, and it is still a problem today. VKDB was primarily restricted to infants who were exclusively breastfed and who did not receive any vitamin K at birth in surveillance. A late dosage of oral vitamin K is advised at 4 to 12 weeks old in the majority of nations with oral vitamin K supplementation policy to prevent late-onset VKDB in these newborns. Increasing the amount of vitamin K in breast milk with maternal supplementation has been tested in a small number of with varying in degrees.

Due to physiologically low vitamin K concentrations that result in low concentrations of vitamin K-dependent clotting factors, healthy neonates and babies are at risk of experiencing severe haemorrhage, especially cerebral haemorrhage. Hence, prevention of Vitamin K Deficiency Bleeding (VKDB) is crucial. It functions as a crucial cofactor for the transformation of certain glutamate residues are attached to the peptides into-carboxyglutamate residues. PIVKA (Proteins Induced by Vitamin K Absence) is the collective acronym for these under-carboxylated molecules, which includes, for instance, PIVKA-II, the glutamate precursor of prothrombin (factor II). Other vitamin K-dependent proteins include osteocalcin and matrix GLA protein, whose functions are poorly understood. The main vitamin K circulating form is phylloquinone; however menaquinone also exist in trace levels (vitamin K2). Although circulating menaquinone may be produced from the diet, intestinal flora, or both, phylloquinone in blood must have come entirely from diet. Menaquinone typically make up 90% of the reserves in human livers and phylloquinone make up 10%.

CONCLUSION

Menaquinone appear to be less significant functionally because dietary phylloquinone deficit results in subclinical vitamin K deficient symptoms without altering hepatic menaquinone storage. Microsomal y-glutamyl carboxylase may not be able to access hepatic menaquinone reserves. At one year of age, hepatic menaquinone storage is still much smaller than those of adults, and there is no menaquinone detectable in neonates. Different dose and frequency guidelines for oral vitamin K prophylaxis appear to provide nearly perfect protection from both early and classic bleeding. Nonetheless, it has consistently been noted that the associated incidence of late VKDB is greater when compared to a single 1 mg IM dose at birth.

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Received: 19-Jan-2023, Manuscript No. JNDT-23-20211; **Editor assigned:** 23-Jan-2023, PreQC No. JNDT-23-20211 (PQ); **Reviewed:** 13-Feb-2023, QC No. JNDT-23-20211; **Revised:** 20-Feb-2023, Manuscript No. JNDT-23-20211 (R); **Published:** 27-Feb-2023, DOI: 10.35248/2161-0509.23.13.225.

Citation: Nicholas N (2023) Vitamin K Prophylaxis in Newborn Infants and their Perinatal Risk Factors. J Nutr Disord Ther. 13:225.