VITAMIN K DEFICIENCY BLEEDING (VKDB) – DOES IT REALLY EXIST?

Vitamin K deficiency bleeding (VKDB) is the name given to the occurrence of haemorrhaging or bleeding in babies in the first weeks of life. It used to be known as Haemorrhagic Disease of the Newborn (HDN) but underwent a name change some time in the last decade. In order to prevent the bleeding experienced by a very small percentage of newborn babies (as well as a few not so new babies), an intramuscular injection of Vitamin K is now routinely given immediately after birth. But exactly what is Vitamin K deficiency and what is VKDB? Why do all babies have comparatively low levels of Vitamin K in their blood at birth, and why are most babies given a huge dose of the vitamin at birth?

Before tackling the issue of the whether VKDB is actually a medical condition or a medical construct, it is necessary to examine the rationale behind this routine intervention of giving almost all babies Vitamin K at birth.

Vitamin K

Vitamin K is a fat-soluble vitamin that plays a significant role in the process of blood clotting. It occurs naturally in the intestine or gut where it is produced by bacteria in the large intestine. Our bodies also get Vitamin K from the food we eat, foods such as green leafy vegetables, alfalfa, kelp, tomatoes, whole grain cereals, olive oil, and milk.

Vitamin K levels in newborn babies

The level of Vitamin K in the blood of newborn babies is between 30% to 60% of that of adults and steadily rises during the first weeks of life. Most healthy, breastfed babies will have blood levels of Vitamin K close to those of an adult within six weeks. (1)

This is where most health professionals get confused. The fact that ALL newborn babies are born with so-called “low” levels of Vitamin K has simply not registered with them as being at all significant. There appear to have been no investigations into why nature has it that babies are born with levels of Vitamin K around half that of adults. The reason for the lower levels remain unknown to this day, and over time it has become known as a “deficiency”, a flaw in human physiology that needs to be corrected.

In fact nature is so determined that babies will be born with “low” levels of Vitamin K in their blood that Vitamin K supplements the mother may decide to take prior to the birth have little or no impact on the levels of the vitamin in her newborn baby, as there is limited transfer of Vitamin K across the placenta. (1) However, some researchers have speculated that babies’ level of Vitamin K may serve to prevent the development of clotting problems during birth and in the first few weeks of life. (2) (3) Others have suggested that the comparatively low Vitamin K levels protect the unborn baby during periods of rapid cell division and rapid cell turnover. In her article on giving Vitamin K at birth, Linda Folden Palmer wrote:

“*It has recently been shown that this tight regulation of Vitamin K levels helps control the rate of rapid cell division during fetal development. Apparently, high levels of Vitamin K can allow cell division to get out of hand, leading to cancer*.” (4)

It can therefore be argued that the levels of Vitamin K in a baby’s blood at birth are not low. They are normal, and exactly what they should be. Moreover nature has gone to considerable lengths to ensure that they stay comparatively low at or for the moment of birth by ensuring that extra Vitamin K a pregnant woman may take does not easily transfer through the placenta and into the baby.

In fact it would be far more honest if health professionals stopped referring to the “low” levels of Vitamin K in newborn babies, and gave up on seeing it as a pathological condition that needs routine treatment.

As to the condition that has been invented to describe what happens to a small percentage of babies – supposedly as a result of these natural and normal levels of Vitamin K – well, it could be seen as a problem of putting the cart before the horse. Time for a short history lesson.

The history of hemorrhagic disease in infants

In 1894 Dr Townsend described 50 babies he had observed who suffered bleeding in the first two weeks of life. He named the condition “hemorrhagic disease of the newborn.” The presumed cause of this condition was not established until much later. In a very informative and fascinating article, Edmund Hey describes what happened next:

“*When it was first shown in 1939 that treatment with vitamin K could abolish symptomatic prothrombin deficiency [a lack of blood clotting ability] in the first week of life, babies were generally given menadione, a water soluble analogue. A paper in the* ***Lancet*** *in 1944 generated widespread interest. It showed a five-fold reduction in death from haemorrhage 2-8 days after birth once all babies were given 1mg of oral menadione at delivery in Goteborg, Sweden in 1940. A similar policy was soon widely adopted elsewhere even though many were unable to replicate these findings. The argument, as Ethel Dunham put it in 1948, was that “since the vitamin does no harm and may do good, it is probably best to give it to all premature infants immediately after birth.”*

When this did not stop some babies from developing a bleeding tendency, or dying with an intraventricular haemorrhage, physicians started using larger and larger doses. Prothrombin levels are always relatively low at birth by adult standards, and remain so for some time, and it was (wrongly) thought that the low level seen at birth, and not just the further post-delivery drop, must be due to relative fetal vitamin K deficiency. By 1953 came a first report that high dose use could cause haemolytic anaemia, and by 1956 it has been established that this could, in turn, cause severe jaundice and even death from kernicterus after unbound bilirubin entered the brain. The dose administered was cut back after that but, within five years, the water soluble product was starting to be replaced by the natural, fat soluble, plant form of Vitamin K….

Routine prophylaxis soon became the norm for every baby (not just every preterm baby) in some countries. Intramuscular prophylaxis also became the route universally adopted, mainly because manufacturers never got round to licensing a product for oral use. It also became routine to give a 1mg injection, even though this was a thousand times more than the dose of menadione needed each day, and ten times the dose used in the only controlled trial of clinical efficacy ever conducted” (5)

An oral form of Vitamin K was finally developed in the late 1990s and is now available for parents who prefer to give Vitamin K to their baby orally. The product available in New Zealand is called Konakion MM. Its effectiveness has now been established with some precision. (6)

HDN becomes VKDB

Now to return to the decision to change the name of the condition from Haemorrhagic Disease of the Newborn to VKDB. There were some good reasons behind the change of name for this condition. One reason is that deficiency bleeding is not only seen in the first week of life. Another is that much bleeding that occurs in newborn babies is **not** caused by Vitamin K deficiency. (5) (9) What researchers have now discovered is that there are a number of other factors involved in the bleeding that occurs in a few babies during the first few weeks of life.

VKDB is typically separated into three categories:

- Early VKDB – occurs in the first 48 hours after birth
- Classic VKDB – occurs in first week of life
- Late VKDB – occurs in babies between 2-12 weeks of life.

Early VKDB is rare and is **not** caused by the naturally "low" levels of Vitamin K found in all newborn babies. It almost exclusively occurs in babies whose mothers are on anticonvulsants (especially phenobarbitone and phenytoin or maternal coumarin products used to prevent seizures), or are on anti-tuberculous therapy during pregnancy. (1) (7) (8). It can be prevented by the administration of Vitamin K at birth.

Classic VKDB is the most common form and occurs in the first week of life in 0.25 – 1.5% of newborn babies according to some papers (1) (9) and in 1-2 per 1000 babies according to others (5). It is associated with inadequate intake of Vitamin K following birth probably as a result of a delay in feeding or an inadequate volume of breastmilk. (1) (9)

Late VKDB is very, very rare and very, very serious. It occurs in infants between 2-12 weeks of age in 4.4 – 7.2 per 100,000 babies. Most of these babies are found to have cholestatic liver disease or cystic fibrosis. Others have severe malabsorption syndromes. The bleeding is a symptom of the underlying disease, **not** of vitamin K deficiency. The rate of VKDB can be reduced to 1.4 – 6.4 per 100,000 babies by giving the newborn an injection of Vitamin K which will prevent the development of late VKDB – but not in those with severe malabsorption syndromes. (10) Almost half of the babies with late VKDB will suffer permanent brain damage or death.

So is VKDB the problem?
A strong case can be made that both early and late VKDB have almost nothing to do with a so-called deficiency of Vitamin K in newborn babies but are either the result of medications taken by the mother that impact negatively on her baby (early VKDB), or the result of a pre-existing medical condition in the baby (late VKDB).

As for Classic VKDB – babies who are not fed at birth and those who do not get unrestricted access to the breast in the first few weeks of life are certainly at increased risk of developing Classic VKDB. This is because both colostrum and hind milk are rich in Vitamin K, and until bacterial activity in a baby's gut starts to produce a secondary source, breastmilk is the baby's only source of this essential vitamin. It has been suggested that restrictive breastfeeding practices were responsible for the reportedly low levels of Vitamin K received by babies during the first week or two after birth, rather than there being anything unnatural or not normal with either the baby or the breastmilk. (2)

How common is VKDB?
It seems that there has never been any reliable research into the actual prevalence of VKDB. In his article on the issue Edmund Hey stated:

"*Recent studies, using a standardised definition, seem to show that the condition is not now very common, even in communities where prophylaxis is not yet available. It is certainly not nearly as common as some authoritative reports claim. Two studies in Japan before the introduction of routine prophylaxis had suggested that one in every 6000 breastfed babies might sustain a late bleed when more than two weeks old. The true risk of bleeding in the first week of life (the "classic" presentation) remains less clearly defined.*" (5)

This raises another set of questions about where the figures quoted as to the incidence of HDN/VKDB came from and how they were reached, but this is beyond the scope of this article.

Vitamin K in breastmilk
An article on this issue would also not be complete without at least a passing reference to what health professionals like to also see as a deficiency – the so-called "low" levels of Vitamin K in breastmilk. Because breastmilk has lower levels of Vitamin K than cow's milk and artificial formulas, the "low" label has been applied to breastmilk in the same way and by the same people as it has to the levels found in newborn babies.

This raises the question of whether you believe that breastmilk is a wondrous substance which provides all that a baby needs or whether it is a poor alternative to cow's milk.

Conclusion

So what do we really know about the significance of the levels of Vitamin K in babies' blood during pregnancy and in the first weeks following birth? Not very much it would seem. While it is true that newborns have lower levels of Vitamin K at birth than adults, this is not a pathological condition needing treatment. It also very unlikely to be a problem unless mothers are taking certain medications or unless the baby has an as yet unrecognised medical condition such as cholestatic liver disease or cystic fibrosis. It is therefore misleading to attribute the bleeding that can occur in these circumstances to a deficiency in Vitamin K. It can instead be argued that these babies have a need for extra Vitamin K that the vast majority of healthy babies born to healthy mothers simply do not need.

The problem that remains is that it is not possible to know exactly which babies will develop bleeding. Although as has been demonstrated, some factors that put babies at risk have been identified. It is therefore important that parents are given accurate information and permitted to make their own decisions.

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References:

(1) Marcia L. Buck. "Vitamin K for the Prevention of Bleeding in Newborns." *Pediatric Pharmacotherapy*. Vol 7 No 10. October 2001.
(2) Sara Wickham. "Vitamin K – An Alternative Perspective." *AIMS Journal* Vol 12. No 2 Summer 2001.
(3) LG Israels, ED Israels. "Observation on Vitamin K deficiency in the fetus and newborn: has nature made a mistake?" *Semin Throm Hemost* 1995:21:357-63.
(4) Linda Folden Palmer. "Vitamin K at Birth: To Inject or Not."
(5) E Hey. "Vitamin K – what, why and when." *Archives of Disease in Childhood Fetal and Neonatal Edition* 2003; 88:F80.
(6) R von Kries et al. "Oral mixed micellar vitamin K for prevention of late vitamin K deficiency bleeding." *Archives of Disease in Childhood and Neonatal Edition* 2003; 88:F109-12.
(7) Lachlan Smith. "Intramuscular Vitamin K in the newborn and childhood cancer – a literature review of evidence for best practice." *NZMSJ*. March 2004;20-23.
(8) National Women's Newborn Services Clinical Guideline – Vitamin K. www.adhb.govt.nz/newborn/Guidelines/Blood/VitaminK.htm. Accessed 2/2/06
(9) National Health and Medical Research Council. "Joint Statement and recommendations on Vitamin K administration to newborn infants to prevent Vitamin K deficiency bleeding in infancy." October 2000.
(10) Committee on Fetus and Newborn's Policy Statement of the American Academy of Pediatrics. "Controversies Concerning Vitamin K and the Newborn." *Pediatrics.* Vol 112. No 1 July 2003.

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