NOT ALL SUBDURALS ARE TRAUMATIC

John Wahba*  Swarnlata Saroey  Ewa Lichtarowicz-Krynska  Colin Michie

ABSTRACT

Late Vitamin K Deficiency Bleeding (VKDB) may develop in rare situations between 1 and 6 months of age. A case is presented in which the infant did not receive Vitamin K at birth and developed an intracranial bleed at 4 weeks of age. Despite presenting with a fixed dilated pupil this infant recovered well. The case illustrates a problem of the persistence of misinformation relating to optimal infant care.

THE CASE

A four-week old exclusively breast-fed male infant was presented to the emergency department with a two day history of inconsolable crying and vomiting. The baby was born at term at home by spontaneous vaginal delivery with an unremarkable antenatal and postnatal course. Vitamin K was not given at birth following parental refusal. On examination the baby had a tense, bulging anterior fontanelle, with a right fixed and dilated pupil which was unresponsive to light. Careful collection of the history and examination of the infant showed no signs of non-accidental injury. Initial investigations revealed a significantly prolonged APTT of 172.4 seconds and PT could not be estimated as the sample had not clotted. A CT scan of the skull showed an extensive subdural haemorrhage with a 1.2cm right to left midline shift, left lateral ventricle dilatation and an impending tonsillar herniation (Figure 1). There were no signs of skull fracture, bony trauma or parenchymal bleed. The baby was given intravenous Vitamin K and fresh frozen plasma (FFP). The clotting profile after the Vitamin K showed an APTT of 42.7 seconds and a PT of 15.4 seconds.

The baby was intubated and transferred to a neurosurgical unit as an emergency. There he underwent a right decompressive craniotomy, evacuation of the subdural haematoma, and insertion of intracranial pressure bolt. He was neuroprotected for 4 days following this. A screen for metabolic anomalies including glutaric acidemia defects, a skeletal survey, ophthalmology review and a background check for any child protection issues

* Corresponding author: John Wahba, Department of Paediatrics, Ealing Hospital NHS Trust, Uxbridge Road, London, UB1 3HW, United Kingdom
were unrevealing. An EEG showed moderate wave abnormalities in the left temporal region and the baby was started on a prophylactic dose of phenytoin. He was then transferred back to our care on intravenous phenytoin and Vitamin K. He showed complete correction of his clotting profile with an APTT of 35.9 seconds and PT 10.2 seconds. His neurology normalised too, with normal eye movements and pupillary reactions. Six months after this episode has normal growth and development with a mild hearing loss on the right side.

DISCUSSION

At birth, Vitamin K levels are less than the detectable level of 0.02 ng/ml but most babies achieve normal haemostasis at this stage and detectable levels are usually found by 12 hours of age, reaching adult levels by day 4[1]. It has been postulated that low levels of Vitamin K in the neonatal period may protect against toxic and mutagenic metabolites in the newborn.[2] It has been shown that formula fed infants have 10 times as much Vitamin K as breast fed infants as it is deficient in breast milk.[3] It is therefore the current recommendation that all newborns should receive Vitamin K prophylaxis either in three oral doses or as a single intramuscular injection.[4, 5] Both routes have been shown to be equally safe and effective in a Cochrane review published in 2000.[6]

Vitamin K Deficiency Bleeding (VKDB), previously referred to as Haemorrhagic Disease of the Newborn (HDN) is defined as the inadequate activity of Vitamin K dependant factors which is corrected after administration of Vitamin K.[7] It is classified into early, classical and late VKDB. The late subtype occurs at 1-6 months of age and has an incidence of 4.4-7.2 per 100,000 live births,[8,9] occurring mainly in breast-fed babies.[10]

It is associated with conditions that lead to Vitamin K malabsorption in the gut including cystic fibrosis, biliary atresia, α1 antitrypsin deficiency, and αβ lipoprotein deficiency.[11] It is diagnosed by a prolonged APTT and PT, normal fibrinogen and platelets, and evidence of haemorrhage. Following Vitamin K administration by the intravenous or subcutaneous routes (the intramuscular route should be avoided) there is biochemical normalization of the clotting profile. In a severe bleed, as seen in our case, further blood products may be needed such as FFP or clotting factor concentrates. Furthermore, imaging studies should be performed if abnormal neurology is encountered.

As in our case and as identified in the British Paediatric Surveillance Unit surveys,[12] parental refusal is found to be the most frequent explanation for not giving vitamin K at birth. There is a variety of cultural and religious reasons given by parents, but many attribute it to a concern relating to an increased risk of childhood cancers. This information relates to work by Jean Golding on the British ALSPAC cohort, reported in 1990. Despite more recent studies from Denmark, Sweden and the USA that have failed to
replicate Golding’s findings [13,14] these concerns remain. Persistence of health misinformation has been found to be strongly supported by internet ‘crowdsourcing’. [15] The authors identified several readily accessible sites on the first page of an internet search that suggested the use of vitamin K was associated with a risk of malignancy and autism.

In summary parenteral Vitamin K should always be offered to the newborn and its use encouraged by midwives and doctors as it can prevent this rare but life threatening disorder. The risks of vitamin K deficiency must continue to be included in the education of prospective parents and midwives.

Figure 1- Acute Subdural Haemorrhage. Axial CT scan showing an extensive subdural haemorrhage with a right to left midline shift and left lateral ventricular dilatation.

REFERENCES

[2] IsraelS LG, Friesen E, Jansen AH, IsraelS ED. Vitamin K1 increases sister


