2006: 20 (4): 327 - 329
e-mail: http://www.fusabil.org

OLGU SUNUMU

Intracranial Hemorrhage Due To Vitamin K Deficiency In An Infant Despite Prophylaxis At Birth

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Vitamin K deficiency bleeding is rare in infants who received vitamin K prophylaxis at birth. We present a case of a 7-week-old infant with intracranial hemorrhage due to vitamin K deficiency bleeding, who received intramuscular vitamin K prophylaxis at birth. In his history, he had used a parenteral antibiotic four days ago. We noticed on that the use of an antibiotic may play a role in late vitamin K deficiency bleeding and it may be responsible for insufficiency of single dose vitamin K, administered at birth.

Key Words:. *Infant, Intracranial hemorrhage, Prophylaxis, Vitamin K.*

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Geliş Tarihi : 28.12. 2005
Kabul Tarihi : 21. 08. 2006

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Doğumdaki Proflaksiye Rağmen Bir Bebekte K Vitamini Eksikliğine Bağlı Kafa İçi Kanama

K vitamini eksikliğine bağlı kanama doğumda K vitamini proflaksisi alan bebeklerde nadirdir. Bu yazıda doğumda intramusküler K vitamini proflaksisi almasına rağmen K vitamini eksikliğine bağlı kafa içi kanaması olan yedi haftalık bir bebeği sunduk. Bebeğin hikayesinde dört gün once parenteral bir antibiyotik kullanımı mevcuttu. Geç K vitamini eksikliğine bağlı kanamada antibiyotik kullanımının önemli bir rol oynayabileceğine ve doğumdaki tek doz K vitamini uygulamasının yetersiz kalabileceğine dikkat çekildi.

Anahtar Kelimeler: *Bebek, K vitamini, Kafa içi kanama, Proflaksi.*

Introduction

Vitamin K deficiency bleeding (VKDB) is manifested by two patterns, according to American Academy of Pediatrics Committee on Fetus and Newborn (1), early (from birth to 2 weeks of age) and late (onset from 2 weeks to 12 weeks of age) VKDB.

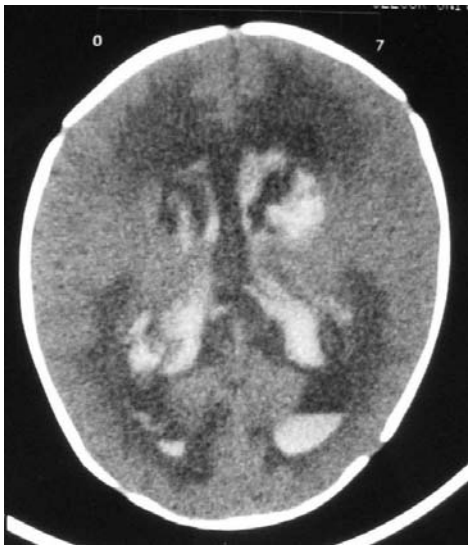
Late VKDB is observed almost exclusively in breastfed infants who did not receive vitamin K at birth (2). The rate of late VKDB (often manifesting as sudden central nervous system hemorrhage) ranges from 4.4 to 7.2 per 100 000 births (1). When a single dose of oral vitamin K has been used for neonatal prophylaxis, the rate has decreased to 1.4 to 6.4 per 100 000 births (1). However, VKDB has been reported in infants who received vitamin K at birth (2-6). Vitamin K prophylaxis at birth in our country is routinely administered by a single injection of 1 mg intramuscular vitamin K.

We report a case with intracranial hemorrhage (ICH) due to VKDB, who received intramuscular vitamin K prophylaxis at birth.

Case Report

A male infant aged seven weeks was referred to Department of Pediatrics, Selçuk University, Meram Medical Faculty because of coma, fever, convulsions and ICH, detected by cranial computed tomography (CT), from State Hospital. A parenteral antibiotic (sulbactam-ampicilline) treatment had been begun four days ago because of a respiratory tract infection. His sucking had impaired gradually. In the last day, he had suffered convulsion. In the history, his delivery was uneventful and his development was normal. He was breastfed, born at term of healthy mother, in hospital. Intramuscular 1 mg vitamin K had been administered at birth. The family history was negative for any bleeding disorder. At the admission, he was in coma. Physical examination was revealed anterior fontanel bulging, weak pupil reaction, anisocoria, increased muscle tonus, decreased deep tendon reflexes, absence of neonatal reflexes. There were no ecchymoses and petechiae.

Laboratory investigations revealed coagulopathy as follows: protrombin time (PT) was >70 s (normal range: 11-13.5 s), partial thromboplastin time (PTT) >120 s (normal range: 28-36 s), fibrinogen 168 mg/dl (normal range: 125-300 mg/dl), hemoglobin 7.2 g/dL, platelets $230{,}000\times10^9$/L, leukocytes 8380×10^9/L, SGOT 36 IU/L, SGPT 18 IU/L, total bilirubin 0.5 mg/dl, direct bilirubin 0.2 mg/dl, urea 10 mg/dL, sodium 138 mmol/L, potassium 5.2 mmol/L, calcium 10.3 mg/dL, glucose 187 mg/dL. Microscopic examinations of urine and stool were normal. Lumbar puncture did not indicate meningitis but revealed hemorrhagic fluid. Cultures of blood, urine, stool and cerebrospinal fluid were normal. One day after the vitamin K administration (2 mg intravenously), PT was 12.7 s and PTT 30.6 s. Cranial CT showed intraventricular hemorrhage (Figure 1).



Ventricular tap, mannitol, phenobarbital, and packed red blood cells were administered. Nevertheless, hydrocephaly was developed. The patient was referred to neurosurgery department because of ventriculoperitoneal shunt surgery. At the follow-up, the patient was experienced epilepsy, developmental delay and blindness.

Discussion

In a bleeding infant, a prolonged PT without any finding considering other bleeding disorders is almost diagnostic of VKDB. Rapid correction of PT and/or cessation of bleeding after vitamin K administration are confirmative the diagnosis (7). In the present case, the marked prolongation of the values of PT and PTT had improved in a short time after the administration of vitamin K and any finding considering other bleeding disorder was not identified.

Late VKDB is often secondary to diarrhea, malabsorption, neonatal hepatitis, or prolonged antibiotic therapy. Many infants with late VKDB had acute ICHs which can be the first manifestation of the disease (2, 8). The localizations of the ICHs were reported as follows: parenchymal (31.3-91%), subarachnoid (27.3-90.6%), subdural (0-37.5%) and intraventricular (12.5-27.3%) (9-11).

In the present case, there was no evidence of diarrhea, malabsorption, liver dysfunction, disseminated intravascular coagulation. The possible risk factors of the present case despite prophylaxis at birth are a low level of vitamin K in the mother's milk, use of a parenteral antibiotic. Latini et al. (3) and Solves et al. (4) reported that two cases with ICH due to VKDB despite vitamin K prophylaxis at birth did not have any secondary cause. Suzuki et al. (5) reported that a case with ICH due to VKDB despite prophylaxis had a history of use of oral antibiotic given two days before the onset of bleeding. Loughnan et al. (6) reported two premature infants who developed late VKDB despite intravenous vitamin K prophylaxis. One of them had hepatitis and the other did not have any secondary cause.

We speculate that the use of a broad spectrum antibiotic may play a role in late VKDB and it may be responsible for insufficiency of single dose vitamin K administered at birth for prophylaxis. Therefore, additional vitamin K supplementation may be considered to prevent late VKDB in the use of a broad spectrum antibiotic.

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