A Possible role of Antenatal Vitamin K Administration in Preventing Epiphyseal Stippling in Fetal Hydantoin Syndrome

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Abstract

The effect of conventional anti-convulsant, Phenytoin, on bone health is described in the literature. There is increasing evidence on the role of Vitamin K in bone health and in prevention of osteoporotic fractures (4) We report a case of a newborn with fetal hydantoin syndrome associated with epiphyseal stippling and consider a possible protective role of antenatal administration of vitamin K to mothers on phenytoin to prevent vitamin K induced epiphyseal stippling in the neonate.

Case Report(s)

Twenty five years old primigravida, a known epileptic since 12 yrs of age, was on anticonvulsants. She conceived while on phenytoin. She delivered a term IUGR baby with a birth weight of 1.56 Kgs and characteristic dysmorphic features of wide anterior fontanel, depressed nasal bridge, metopic ridging, hypoplasia of the hand and toe nails, stiff tapered fingers, overriding of the 5th toe bilaterally suggestive of fetal hydantoin syndrome (1) Radiological features were suggestive of epiphyseal stippling of bilateral femoral heads, proximal and distal foot bilaterally. Upper limb radiographs were essentially normal.

Discussion

Teratogenic effects of phenytoin were attributed to arene oxide metabolites which covalently bind to cell macromolecules resulting in cell death mutation, tumors and birth defects. Maternal ingestion of phenytoin induces the fetal microsomal enzymes with resultant increased oxidative degradation of vitamin K which gives rise to vitamin K deficiency. Vitamin K deficiency contributes to poor bone health owing to undercarboxylation of osteocalcin. (5) Vitamin K is a cofactor of gamma-carboxylase, which converts the glutamic acid (Glu) residue in osteocalcin molecules to gamma-carboxyglutamic acid (Gla), and is therefore essential for gamma-carboxylation of osteocalcin. Vitamin K also enhances osteocalcin accumulation in the extra cellular matrix of osteoblasts. Gla-containing osteocalcin promotes normal bone mineralization. The role of osteocalcin in bone mineralization remains unclear; it probably regulates the growth of hydroxyapatite crystals. Vitamin K may not only stimulate bone formation but also suppresses bone resorption. Vitamin K deficiency primarily decreases the bone mineralization and subsequently a decrease in bone mass thus resulting in an increased susceptibility to bony deformities (5) Facial abnormalities causing abnormal development of cartilaginous nasal septum is attributed to anticonvulsant induced vitamin K deficiency. This anticonvulsant induced vitamin K deficiency can result in neonatal epiphyseal stippling in fetal hydantoin syndrome which has shown to have long term complications of avascular necrosis of femoral heads, coxavara, limb length discrepancies as well as residual arthritis and could possibly be prevented by antenatal administration of vitamin K to mothers on antenatal phenytoin therapy to prevent bony deformities and epiphyseal stippling and subsequently its long term complications in the newborn. This therapeutic consideration would possibly prove to be extremely beneficial for those known epileptic pregnant women who are in need of phenytoin antenatally.

References


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