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LETTER TO EDITOR (VIEWERS CHOICE)

**HYPOVITAMINOSIS D IN AN INFANT WITH HYPOCALCEMIC SEIZURES SECONDARY TO MATERNAL VITAMIN D DEFICIENCY**

*Shalini Tripathi\*, Sunil Taneja\*\*, Samarth Vohra\*\**

**Key words:** hypocalcemia, neonate, seizure, vitamin D.

A 2 months old boy was admitted to the intensive care unit of our hospital with convulsions. This child was born of normal vaginal delivery and had a birth weight of 2.9 kg. He was exclusively breast fed. There was no fever or trauma or birth asphyxia. He was admitted to another hospital with generalized convulsions at

the age of 1 1/2 months. After treatment for 10 days there, the seizures did not improve and thus the child was referred to our hospital. On admission, the child had generalized tonic clonic convulsions. The child was treated with intravenous phenobarbitone followed by intravenous phenytoin but as the seizures continued and so midazolam infusion was started. On midazolam infusion, seizure episodes were partially controlled.

Blood glucose levels were normal on admission. CSF examination was normal. CT scan head was normal. Serum calcium (ionized) was low (2.36 mg/dl). Despite calcium supplementation the repeat calcium levels were also low. On reviewing the antenatal history, mother was not taking any antiepileptic medication and was not on any calcium and multivitamin supplementation. Further investigations in the child showed serum magnesium of 2.6 mg/dl (Normal= 1.2-2.6 mg/dl), serum alkaline phosphatase of 1072 IU/L, serum parathyroid hormone (PTH) level of 696 pg/ml (15-68 pg/ml), serum 25 hydroxy vitamin D of 14.2 nmol/l (Normal = 75-250 nmol/l). X ray wrist showed diffuse rarefaction however there was no cupping at the epiphyseal end. Thus the child was diagnosed to have vitamin D deficiency. Patient was started with oral 1, 25- hydroxy calcitriol with intravenous calcium. After 24 hrs of this treatment seizures were controlled, serum calcium was repeated and became normal on 3rd day after treatment. Mother's serum calcium level was 2.27 mmol/L (normal= 2.19 mmol/L to 2.60 mmol/L), serum 25-hydroxy vitamin D was 13 nmol/l (Normal = 17 nmol/L to 95 nmol/l), serum PTH level was 88 pg/ml (normal=10 - 65 pg/ml). Thus, she was also diagnosed to have vitamin D deficiency.

Vitamin D deficiency in the neonate secondary to maternal deficiency is an important cause of seizures in the newborn period. The appropriate daily allowance of vitamin D during pregnancy and lactation is unknown, although it appears to be greater than the current dietary reference intake of 200-400 IU/day. (1) Maternal diet low in vitamin D with no extra supplementation is an important risk factor for neonatal vitamin D deficiency. Exclusive breast-feeding is recommended up to 6 months of age with all its beneficial effects on child survival. Several studies have concluded that adequate intake of vitamin D cannot be met with human milk as the sole source of vitamin D. (1) Most of the vitamin D in a neonate is acquired from maternal transfer, so vitamin D deficiency in mothers is likely to have adverse consequences for their infants. (2-4) The supply of vitamin D in utero, rather than from milk, is the major determinant of vitamin D status in early neonatal life in mammals. (5) Shulman et al published two neonatal cases with hypocalcemic seizures. Following this, the Canadian Paediatric Society guidelines recommend vitamin D supplements for all breastfed babies (400 U/day) to begin at birth and continue until the infant is 12 months of age. For those residing above the 55th parallel, vitamin D intake should be increased to 800 U/day between October and April. (6) Erdeve et al reported a neonate presenting with severe hypocalcemic seizures on day 1 of life due to maternal vitamin D deficiency. (7) Hatun et al analyzed the characteristics of young infants (<3 mo) diagnosed with vitamin D deficiency in Turkey. A total of 42 infants were diagnosed with vitamin D deficiency. The majority (78.7%) presented with seizures. (8) Teaema et al have done a retrospective study on 19 newborns in Qatar with symptomatic hypocalcemia. Vitamin D deficiency with or without relative hypoparathyroidism was the attributed cause. (9)

At present, vitamin D supplementation is not a part of antenatal care/Integrated Management of Neonatal & Childhood Illness (IMNCI) programmes in India. Vitamin D supplementation is done only for low birth babies. Evaluation of vitamin D status should be included into the workup of hypocalcemia in early infancy. Emphasis should be given to prevent vitamin D deficiency by supplementing pregnant women and large studies are needed for vitamin D supplementation of infants who are exclusively breast-fed.

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## LETTER TO EDITOR (VIEWERS CHOICE)

**METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS MENINGITIS WITH BELL'S PALSY***Shalini Avasthi\*, O P Bhatt\*\*, Sunil Taneja\*\*, Samarth Vohra\*\**

**Keywords:** community acquired MRSA, CA-MRSA, pyogenic meningitis

A 4 month old girl presented with right sided Bell's palsy. There was history of fever for 1 week for which she had received intravenous antibiotics. There was no history of ear discharge, head trauma and birth asphyxia. All the developmental milestones were normal as per the age. On examination she had right sided lower motor neuron type facial nerve palsy. Other systems were normal. Investigations showed hemoglobin of 9gm%, white cell count of 24,200/cumm (83% polymorphs, 14% lymphocytes, 1% eosinophils, 2% monocytes), platelet count of 65,000/cumm. Cerebrospinal fluid (CSF) showed 640 cells/cumm (60% polymorphs, 40% lymphocytes), proteins 53 mg% and sugar 37 mg%. CT scan brain was normal. Diagnosis of partially treated pyogenic meningitis was made. She was treated with intravenous ceftriaxone and amikacin. After 72 hours on this treatment, child continued to have fever and suddenly developed bilateral lateral rectus palsy. Repeat CSF examination showed 1100 cells/cumm (52% polymorphs, 48% lymphocytes), protein 58gm% and sugar 27mg%. At the same time the report of the first CSF culture showed growth of MRSA sensitive to Vancomycin. MRI brain was done which showed meningitis. Child was started on Vancomycin at 60 mg/kg/day. After Vancomycin, patient improved clinically, was afebrile, she was able to close right eye and right nasolabial fold appeared. CSF examination, after 21 days of intravenous Vancomycin was normal and the repeat culture was sterile. However bilateral lateral rectus palsy persisted.

Traditionally Methicillin resistant Staphylococcus Aureus (MRSA) infections have been acquired almost exclusively in hospitals, long-term care facilities, or similar institutional settings. (1) Risk factors for MRSA colonization or infection in the hospital include prior antibiotic exposure, neurosurgery, admission to an intensive care unit, surgery, and exposure to an MRSA-colonized patient. (2) Community acquired MRSA infection (CA-MRSA) is associated with a history of recent hospitalization, close contact with a person who has been hospitalized, or other risk factors, such as previous antimicrobial drug therapy. CA-MRSA infections in the absence of identified risk factors have been reported infrequently. A study done at Chicago demonstrated that that the prevalence of CA-MRSA among children without identified risk factors is increasing. (3) Pyogenic meningitis due to MRSA has been rarely reported. One case of MRSA pyogenic meningitis in an adult has been reported in which also the MRSA meningitis was spontaneous however it was not associated with any focal neurological deficits. (4) Bell's palsy as a presentation of pyogenic meningitis without predisposing factors is also rare. In a case report, authors have reported an unusual case of bilateral simultaneous facial palsy occurring during the course of a persistent otitis media in a 7 month-old child. (5) Similarly there is a case report of

a patient with Streptococcus pneumoniae meningitis, multiple cranial nerve involvement, and cerebellar signs suggestive of basilar meningitis. (6) Two cases with Bell's palsy due to Varicella Zoster virus in children are reported. These children were also had pyogenic meningitis. (7)

Thus MRSA infections in children can occur without predisposing factors and can lead to pyogenic meningitis with focal neurological deficits.

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