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Unusual Complications of Rickets

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Hypocalcemia occurs when the homeostatic mechanisms responsible for the maintenance of physiological serum concentrations of ionized calcium fail. Hypocalcemia whether transient, acute or chronic, should be regarded as a sign only; therefore, an underlying pathophysiology should be sought at the same time that the treatment is initiated for all but the mildest symptoms(1).

We describe an interesting case of rickets associated hypocalcemia mani-

Received for publication: September 22,1994; *Accepted: October* 11,1994 festing as seizures and later high dose Vitamin D therapy to the child leading to coma and cardiac tetany (hungry bone phenomenon)(2). The case is presented because after extensive search of literature and to the best of our knowledge a similar presentation could not be found.

Case Report

A one-year-old female Muslim child weighing 8 kg presented with fever, cough and respiratory distress since 8 days with 6 episodes of convulsions of generalized tonic-clonic type since one day. Between convulsions, the child was irritable without impairment of consciousness. There was a past history of six episodes of generalized tonic-clonic convulsions not associated with fever since the age of seven months, the frequency of which had increased since the last one month, in which the child had four episodes. The child was receiving anticonvulsant carbamazepine in inadequate dose with no response. The developmental and natal history was normal; however, the child had been exclusively breastfed with no weaning. On examination, the child had evidence of anemia and rickets and the neurological examination was normal.

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Investigations revealed iron deficiency anemia and evidence of rickets on X-ray wrist. A provisional diagnosis of bronchopneumonia with anemia with rickets with epilepsy was made and the child put on antibiotics and carbamazepine was restarted in adequate doses. Therapeutic dose of Vitamin D in the form of injection arachitol 3 lack IU intramuscularly was given on the 3rd day of admission. The child did not show the expected improvement and on the same day developed altered sensorium. Investigations revealed: hemoglobin-6 g/dl; TLC - 8,500/cu mm, $N_{49}L_{51}$; CSF less than 5 lymphocytes, protein 30 mg/dl, sugar 40 mg/dl, chloride 117 meg/1; blood urea-33 mg/dl; and serum creatinine 1.2 mg/dl. Serum electrolytes and USG brain were normal.

The next day, child developed congestive cardiac failure and had repeated convulsions for which, injections digoxin, lasix and epsolin were given Vith no response. Since there was no improvement, a possibility of hypocalcemia was entertained and serum calcium and ECG were done. Serum calcium was 5.1 mg/dl and ECG showed prolonged QTc. Serum phosphorus was 2.7 mg/dl, serum alkaline phosphatase was 21 KA units, PTH was within normal limits, and EEG was normal. Hence a diagnosis of hypocalcemia with cardiac tetany secondary to rickets and large dose Vitamin D was made. Intravenous calcium was given and there was a dramatic improvement in the sensorium and congestive cardiac failure. X-ray wrist done 15 days later showed evidence of healing rickets. The child was discharged on oral calcium only and has been convulsion free on six months follow up.

Discussion

Tetany of Vitamin D deficiency occurs most frequently between the ages of 4 months and 3 years. This type of tetany is rare today owing to widespread prophylactic use of Vitamin D. The symptoms and signs of tetany may exist in either a latent or a clinically manifest state and rickets usually occurs concurrently. The 'manifest' tetany may have carpopedal spasm, laryngospasm and convulsions and in such a situation the serum calcium is often well under 7 mg/dl. It remains unclear why serum calcium is occasionally decreased in association with rickets; probably failure of the parathyroids to compensate for the low serum calcium levels may be a factor.

The diagnosis is based on the combined presence of rickets low serum calcium level and symptoms of tetany. The serum phosphorus level is usually low and serum alkaline phosphatase level is increased.

The prognosis is good unless treatment is delayed. Death rarely occurs, though it may result from laryngospasm and possibly from cardiac dilatation, so called cardiac tetany(3).

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