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Convulsions as primary manifestation of nutritional rickets

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Abstract: *Introduction:* Rickets is diagnosed based on classical clinical features like craniotabes, rachitic rosary, widening of wrist joints, pot belly, hypotonia, bowed legs and supported by the laboratory evidence of hypocalcemia, hypophosphatemia, and raised alkaline phosphatase. Hypocalcemic convulsions are a rare manifestation of nutritional rickets. Decline in the magnitude of the rickets requires high index of suspicion to identify this treatable condition. Herewith presenting retrospective study of twelve cases of rickets who presented with hypocalcemic seizures as primary manifestation. *Materials and methods:* A detailed retrospective analysis of diagnosed cases of rickets presenting with convulsions was done. Children who were admitted with hypocalcemic convulsion and subsequently diagnosed to have rickets were included in the study. Children who did not fulfill the criteria for diagnosis of rickets either clinically or biochemically or radiologically were excluded from the study. Details including age at presentation, weight, sex, gestational age and other associated diseases were collected and analyzed. Rickets was diagnosed on the basis of clinical features, biochemical parameters (serum calcium, phosphorous, alkaline phosphatase) and radiological findings. *Results:* A Total of 12 children; 8 male and 4 females constituted the study subjects. Mean age of presentation was 6 months. All children had presented with preceding convulsions or active convulsions. One child who presented with active convulsions was treated with parenteral phenobarbitone. All 12 of them were evaluated for the cause and found to have hypocalcemia without any other cause for convulsions. Further clinical examination revealed features of rickets and were subjected to radiological and biochemical investigations. The mean calcium value was 6.3mg/dl, phosphorus -5.35mg/dl, alkaline phosphatase-890.13 units. All the cases had radiological features of rickets. All 12 were treated with parenteral vitamin-D and they responded well to the treatment. *Conclusions:* Convulsion is a rare manifestation of rickets and as a primary presentation is even rarer. Current observational study indicates that rickets could be a cause of hypocalcemic convulsion in young infants and children. Screening infants and young children presenting with convulsion for rickets can help in early diagnosis and institution of specific therapy.

Keywords; Rickets, Convulsion, Primary manifestation

Introduction

Rickets is diagnosed based on classical clinical features like craniotabes, rachitic rosary, widening of wrist joints, pot belly, hypotonia, bowed legs and supported by the laboratory evidence of hypocalcemia, hypophosphatemia, and raised alkaline phosphatase. Hypocalcemic convulsions are a rare manifestation of nutritional rickets. Decline in the magnitude of the rickets requires high index of suspicion to identify this treatable condition.

Material and Methods

This is an hospital based retrospective study from December 2003-2010. All the children presenting with history of convulsions were worked out for the cause of convulsion and those children who were found to have hypocalcaemic convulsion were later worked up for rickets. The diagnosis of rickets was based on clinical features, biochemical parameters (serum calcium, serum phosphorus & alkaline phosphatase) and radiological features. Clinical features includes asymmetric counter of the head, craniotabes, widening of sutures, patent and wide anterior and posterior fontanel, macrocephaly with flattening of vertex(caput quadratum or box head), bossing of the skull (frontal and parietal bones) rachitic rosary, pigeon chest, harrison groove or sulcus, potbelly ,widening of wrist, knee and ankle joints. Deformities of the spine, pelvis and legs result in rachitic dwarfism and bowed legs. Non-rickets causes of hypocalcemic convulsion were excluded from

the study by doing appropriate metabolic and radiological workup. Case files of these children's were analyzed for age at presentation, sex,weight, clinical features, biochemical parameters (serum calcium, serum phosphorus & alkaline phosphatase) and radiological features. Past history, treatment given were correlated.

Results

A total of 12 children met the inclusion criteria and were analyzed. 8 of them were male and 4 were female. The mean age of presentation was 06 months. All the 12 children presented with history of convulsions. One child presented with active convulsion to the emergency department and required parental phenobarbitone. On clinical examination all the 12 children had clinical features of rickets. Biochemical parameters were consistent with the diagnosis of rickets with mean calcium of 6.3 mg/dl, phosphorus 5.35 mg/dl and alkaline phosphatase of 890.13 units. X-ray wrist of the non-dominant hand showed features of rickets in the all children. No other causes of hypocalcaemia other than nutritional rickets were found in these cases. All of them were treated with intramuscular vitamin-D. On further follow up, biochemical parameters came back to normal range within a week and check x-ray done 4 weeks later showed features of healing.

Cases	Serum Calcium (mg/dl)	Serun Phosphorus (mg/dl)	Alkaline Phosphatase (units)	Radiological Evidence	Age (month)	Treated With
Case 1	6.7	4.2	685	Present	06	Vit - D
Case 2	6.8	4.5	810	Present	07	Vit - D
Case 3	7.0	5.0	785	Present	08	Vit - D
Case 4	7.4	5.0	750	Present	09	Vit - D
Case 5	7.7	5.5	232	Present	11	Vit - D
Case 6	5.0	6.5	1235	Present	11	Vit - D
Case 7	6.2	4.9	900	Present	08	Vit - D
Case 8	6.0	4.7	1343	Present	03	Vit - D
Case 9	6.0	5.2	1201	Present	02	Vit - D
Case 10	5.3	7.0	602	Present	05	Vit - D
Case 11	5.6	8.5	1052	Present	01	Vit - D
Case 12	6.0	3.3	1087	Present	02	Vit - D

Discussion

Rickets is a disease of growing bone, usually manifesting in the latter half of the first year or in the second year. It is uncommon in infants under 3 months of age. The earliest manifestations are quite vague and nonspecific which include irritability, restlessness and profuse sweating. As the disease progress, characteristic clinical features will be evident which includes asymmetric contour of the head, craniotabes (it is one of the earliest sign of rickets and is due to thinning of the outer table of the skull). widening of sutures, patent and wide anterior and posterior fontanel, macrocephaly with flattening of vertex (caput quadratum or box head), bossing of skull (frontal and parietal bones) rachitic rosary (palpable enlargement of costochondraljunction), pigeon chest, Harrison groove or sulcus, potbelly (due to abdominal wall hypotonia), widening of wrist knee and ankle joint. Deformities of spine, pelvis and legs result in rachitic dwarfism and bowed legs [1-3].

The decline in the prevalence of rickets as a result of improved socio–economic status and improved nutrition have resulted in limited exposure and experience in diagnosing rickets. The rarity of florid clinical rickets as

seen in the past and fewer cases of clinical rickets has resulted in declined clinical exposure to the young clinicians. As a result, the early diagnosis has become uncommon, as one do not routinely evaluate for the evidence of rickets. In this changed scenario, the identification of rickets by minimal clinical features and rare clinical features is even more unlikely. Suspecting rickets in children presenting with seizures, pseudotumor cerebri and other uncommon features is much more difficult.

Hypocalcemic seizures are a rare manifestation of rickets and presenting as a primary manifestation is even rarer. However, there have been few published data on this issue in recent times. Cetinkaya et al [4] while analyzing the causes of seizures in children in an urban reference hospital in Turkey noted that hypocalcemia due to rickets was the leading cause of afebrile seizures, accounting for 25.6% of children out of 156 children aged 1-24 months. Ahmed et al [5] have reported 65 infants who presented with hypocalcemic seizures were subsequently found to have rickets. 46 of these infants were totally or predominantly breastfed. In the same group of 15 mothers, their infants were found to have low levels of 25 (OH) vitamin D. Bloom et al [6] reported three cases of vitamin D dependent rickets acutely presenting to the emergency department in a United States hospital with variable presentation. One had hypocalcemic seizures, another had tetany and another one had fracture due to child abuse. Similar presentation due to rickets was due to lack of poor exposure to sunlight and poor dietary intake in an 8 month old girl from Western Australia [7]. Salaria et al [8] have reported a case of rickets presenting as pseudotomor cerebri and seizures in a 4 month old male infant from North India. This particular infant had presented with bulging anterior fontanels and later found to have features of raised intra cranial pressure and rickets. The serum calcium was 5.4mg/dl and there was no other cause for seizures. Jain et al [9] have reported another one-year-old female child with rickets associated with hypocalcemic seizures and features of rickets. This child had deteriorated with cardiac tetany (hungry bone phenomenon) following administration of vitamin-D intramuscularly. Few other sporadic cases with this unusual presentation were reported [10-11].

The above studies shows that vitamin-D dependent rickets continues to occur significantly in the developing world and in the developed world the cases are seen mainly in some ethnic groups. The above studies also shows that although florid rickets is uncommon, the vitamin-D dependent rickets continues to occur albeit either as mild disease or atypical/uncommon manifestations. Hypocalcemic seizures, being a dramatic presentation brings the child to the medical attention. A conscious attempt to look for the clinical features of rickets, especially in the developing world is very helpful in the treatment and saving the child from the serious health effects of an easily treatable disease.

In the current study, all the 12 cases of rickets presented with history of convulsion or active convulsion. Out of 12 cases 1 case presented with active convulsion in emergency department and was treated with anticonvulsant. All the cases were treated with intramuscular vitamin-D. Follow-up biochemical parameters after a week showed improvement and repeat x-ray after 4 week of treatment showed features of healing.

The mean serum calcium was 6.3mg/dl and improved dramatically following administration of calcium. The cluster of 12 cases analyzed by us is one of the large number of cases reported on the seizures as primary manifestations of nutritional rickets in the recent times. Our experience highlights the importance of keeping an open mind to the possibility of rickets in infants and young children presenting with hypocalcemic seizures, especially in the developing countries. In conclusion our experience suggests that rickets could be an important cause of seizures in infants and young children with hypocalcemic seizures. Hence infants and young children should be evaluated for the evidence of rickets both clinically and biochemically. The biochemical evaluation in children with hypocalcemic rickets, even in the absence of clinical evidences helps in the early recognition and appropriate treatment.

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