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Nutritional rickets in childhood: retrospective assessment of clinical data of forty five cases seen in a Nigerian tertiary healthcare institution.

Alphonsus N. Onyiriuka, Phillip O. Abiodun, Fidelis U. Onyiriuka

Department of Child Health, University of Benin Teaching Hospital, PMB 1111, Benin City, Nigeria.
*School of Medicine, University of Benin, Benin City, Nigeria.

*Correspondence author: Dr A.N. Onyiriuka

Abstract

Nutritional rickets is common in Nigeria despite abundant tropical sunshine and its re-emergence has been documented in both developed and developing countries. To document our observation on nutritional rickets in childhood in Benin City. A retrospective chart audit was performed, covering a 12-year period on 45 children with nutritional rickets, diagnosed by clinical, biochemical and radiologic parameters. The peak age group at presentation was 2-3 years with a mean duration of symptoms before presentation of 19.0 ±11.8 months (95% Confidence Interval, CI=15.56-22.44). Male-to-female ration was 2.2:1. The commonest mode of presentation was lower limb deformity (82.2% of all cases). No case presented with hypocalcaemic seizures. The mean duration of symptoms before presentation was 20.3±9.7 months (95% CI=17.47-23.13). The mean serum calcium, inorganic phosphorus and alkaline phosphatase were 2.0±0.3 mmol/L (95% CI=1.88-2.12), 1.2±04 mmol/L (95% CI=1.03-1.42), and alkaline phosphatase 276.4±38.6 IU/L(95% CI= 258.6-294.2) respectively. Normal values in UBTH were 2.15-2.55 mmol/L, 0.8-1.48 mmol/L and 30-90 IU/L for calcium, inorganic phosphorus and alkaline phosphatase respectively. The mean serum albumin was 3.9±0.6 mg/dl (95% CI=3.56-4.24). Nutritional rickets commonly presents after the age of 2 years with lower limb deformity and a male preponderance.

Key words: Nutritional rickets, childhood, Nigeria.

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Introduction

Nutritional rickets is due to deficiency of vitamin D or calcium or both [1,2]. In developed countries, nutritional rickets is uncommon because of the public health measures that provided vitamin D supplementation to children who were at risk [3]. Nutritional rickets is a common disease among Nigerian children despite abundant tropical sunshine [4,5]. In Nigeria , it has been reported that calcium deficiency plays a prominent role as a causative factor in nutritional rickets [2,6,7]. A dark skinned infant breastfed by a vitamin D-deficient mother who remain covered for cultural reasons are particularly at risk [8]. Other risk factors documented in Nigerian studies include late introduction of cereal to infant diet (age above 7 months) and a working-class mother [4]. Some community-based and general-hospital-based surveys among African children indicated that the prevalence of rickets exceed 10% [3]. In a community-based study in Nigeria, Akpede et al [4] reported a prevalence of 2.4% for overt rickets and 14.9% for abnormalities suggestive of rickets among children below five years of age. A hospital-based study in Qatar reported a prevalence of nutritional rickets as high as 23.9% among children below five years of age [9]. In fact, there are reports indicating that rickets is re-emerging in both developing and developed countries [10-14]. This reemergence has been attributed to complacency in food fortification, changing lifestyles where children spend a greater part of their time indoors watching television and/or video movies and immigration of people from one geographic location to another [13-16].

Rickets is a systemic disorder characterized by growth failure, skeletal deformity (most striking clinical feature), hypotonia, seizure and delayed motor development [8]. The age of the patient and the underlying disorder influence its clinical presentation [17].. The diagnosis is based on clinical signs, biochemical and radiological findings [1,4]. The classical biochemical changes are increased alkaline phosphatase level, low or low normal plasma calcium and inorganic phosphorous values. A typical radiograph of rickets show cupping and fraying of distal

metaphyses of long bones [17]. The bone changes of rickets are the same, irrespective of underlying cause. They vary only in severity depending on the age (the younger the child, the more florid), and the type as well as the severity of the metabolic disorder [3]. Lack of awareness concerning rickets have been reported among clinicians. For instance, four children with florid signs of rickets receiving regular paediatric supervision at the University of Connecticut Health Centre, Newington were missed and inappropriately referred to Specialty Clinics and even there, the diagnosis was not suspected on clinical grounds [18]. One may, therefore, surmise that similar lack of awareness occurs in other health facilities around the world including developing countries.

The purpose of the present study was to document our observations on nutritional rickets among patients seen in the Department of Child Health of the University of Benin Teaching Hospital.

Subjects and Methods

In this retrospective descriptive study, conducted at the University of Benin Teaching Hospital (UBTH), Benin City, Nigeria, the case notes of 45 children diagnosed as having nutritional rickets between 2000 and 2011 were retrieved. This number excluded children whose case notes could not be traced. The diagnosis in each case was based on clinical, biochemical and radiologic parameters. The clinical parameters included deformity of the lower limbs, enlargement of the wrists/ankles, rickety rosary, bossing of the skull. The biochemical criteria included low serum calcium and inorganic phosphorus and elevated alkaline phosphatase values. The radiologic criteria were osteoporosis, widening of the epiphyseal ends, cupping and fraying of the metaphyses, thinning of the cortices and deformities of the shaft of the long bones. Renal rickets and other forms of hereditary diseases presenting with rickets were excluded based on negative family history, normal plasma urea and electrolyte profile and normal urinalysis results. Information extracted from each case note included age, gender, weight, family religion, occupation of the parents, educational attainment of the parents, the presenting complaints, physical signs of rickets present, X-ray reports, the results of laboratory tests, mode of treatment, outcome of treatment. The socioeconomic status of the family was determined using the criteria suggested by Ogunlesi et al [19]. This was analyzed via combining the highest educational attainment, occupation and income of the parents (based on the mean income of each educational qualification and occupation). In this Socioeconomic Classification System, Groups I and II represent high socioeconomic class, Group III represents middle socioeconomic class while Groups IV and V represent low socioeconomic class. In this way, the subjects were categorized into high, middle and low socioeconomic classes. The statistical analysis involved calculation of percentages, means, confidence intervals. Chi-square test and Students' t test were used in assessing the significance of the differences with p value set at < 0.05.

Results

The peak age group at diagnosis was 2-3 years. The mean duration of symptoms before presentation was 19.0±11.8 months (95% CI=15.56-22.44). Among the 45 cases, 31(68.9%) were males and 14 (31.1%) were females, giving a male-to-female ratio of 2.2:1 (Table 1). Among the 45 cases, 41(91.1%), 3(6.7%) and 1(2.2%) were from Christian, Muslim and Traditional religion families respectively. Duration of breast feeding was less than 6 months in 7(15.6%); between 6 to 12 months in 28(62.2%); and above 12 months in 11(22.2%) cases. The mean serum calcium, inorganic phosphorus and alkaline phosphatase values are displayed in Table 2. The serum albumin values were available in 12 cases and the mean was 3.9±0.6mg/dl (95% CI=3.56-4.24). As shown in Figure 1, the commonest mode of presentation was lower limb deformity (82.2%). Among the 37 cases who presented with lower limb deformity, 21(53.3%) had bowing of the legs (Genu varum), 12(33.3%) had knock knee (Genu valgum) and the remaining 4(13.4%) had windswept deformity. Twenty seven (60.0%) cases presented with bossing of the skull, comprising frontal bossing 15(55.6%), bipatietal bossing 6(22.2%) and fronto-occipital bossing 3(11.1%). Other forms of bossing of the skull found included 2(7.4%) with a combination of frontal and biparietal bossing and one (3.7%) with a combination of biparietal and occipital bossing. The frequency of physical findings in children with nutritional rickets is displayed in Figure 2. No case presented with hypocalcaemic seizures. The mean duration of symptoms before presentation was 20.3±9.7 months (95% CI=17.47-23.13). The distribution of the socioeconomic status (SES) of the families of the patients showed that 12.3%, 52.6%, and 35.1% belonged to high, middle and low SES respectively.

The patients were managed on out-patient basis and the treatment consisted largely of administration of vitamin D 5,000 Units daily for 8 weeks with calcium supplement; followed by maintenance vitamin D 400 units daily. The duration of treatment varied from three to 12 months with an average of five months. Healing was observed between three to five months of therapy depending on the severity of the case. In mild cases, the enlargement of the wrists resolved between two to three months of treatment. One of the major challenges of management was the high rate of default to follow up. As a consequence, their ultimate fate is usually not known.

Table 1. *Distribution of age at presentation and gender of 45 children with nutritional rickets.*

Age (years)	Males No(%)	Females No(%)	Total
Below 2	6(20.0)	3(20.0)	9(20.0)
2 to 3	11(36.7)	7(46.7)	18(40.0)
>3 to 4	7(23.3)	18(40.0)	11(24.4)
Above 4	6(20.0)	6(20.0)	7(15.6)
Total	30(100.0)	30(100.0)	7(15.6)

Table 2*. Mean serum calcium, phosphorus and alkaline phosphatase values in 45 children with nutritional rickets.*

Biochemical Parameter	*Normal Serum values in UBTH*	*Mean Serum values*	*95% CI*
Calcium (mmol/L)	2.15-2.55	2.0±0.3	1.88-2.12
Phosphorus (mmol/L)	0.8-1.48	1.2±0.4	1.03-1.40
Alkaline phosphatase (IU/L)	30-90	276.4±38.6	258.6-294.2

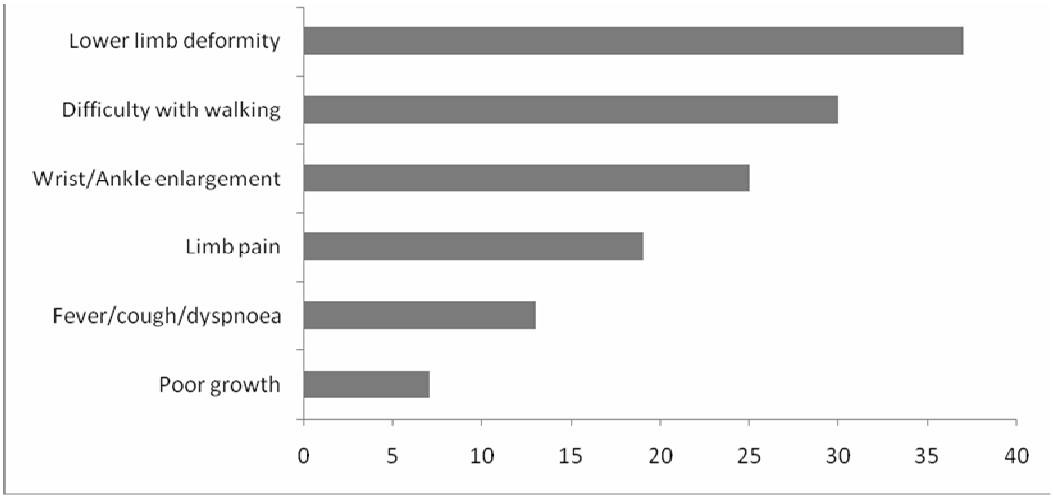

Figure 1. *Frequency of presenting symptoms*

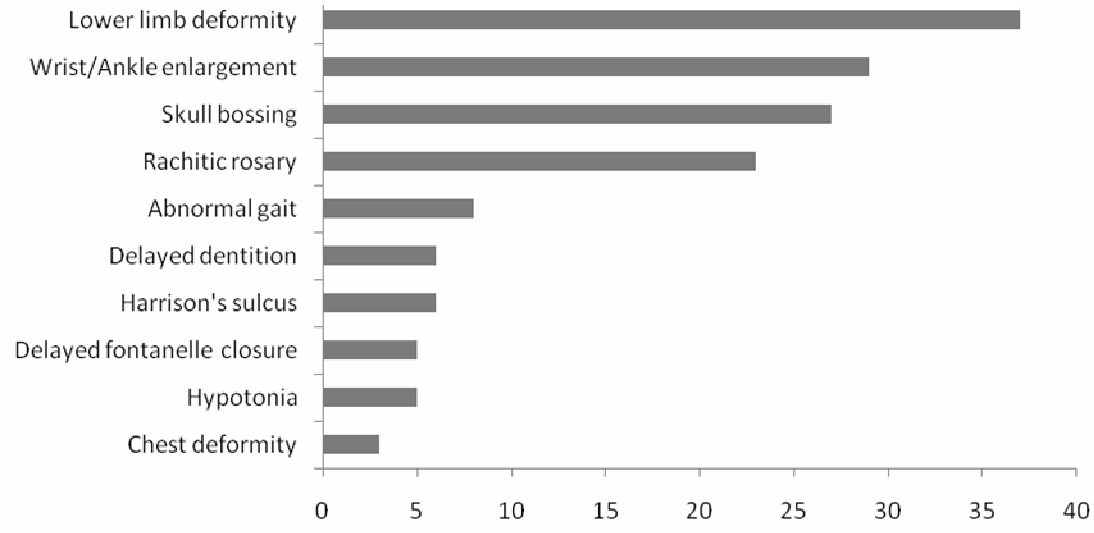

Figure 2. *Frequency of clinical signs*

Discussion

In the present study, the peak age at presentation was 2-3 years. Other Nigerian studies have reported varying peak ages at presentation. For instance, 13-24 months was reported from Port Harcourt [20] and 3-4 years was reported from Ibadan [21]. The peak age reported in the present study corresponds largely to the period when children in Nigeria are weaned to maize-based milk-free complementary foods low in calcium content. This is in keeping with the current evidence which has linked nutritional rickets in Nigeria to calcium deficiency [2,6,7]. This view is reinforced by the report of Thatcher et al [2] which stated that calcium deficiency rickets occurs after the age of two years whereas rickets due to pure vitamin D deficiency occur below the age of 18 months. A similar view has been documented in the literature. For instance, Greenbaum [3] stated that calcium deficiency occurs after the cessation of breastfeeding and so, tends to occur later than the nutritional vitamin D deficiency that is associated with breastfeeding. The mean serum albumin of 3.9 mg/dl observed in the present study suggests that the nutritional status of the patients was fair. Similar observation has been reported in previous studies in Nigeria [20,21].

As in previous studies, [11,20-23] male preponderance was observed in the present study but with different male-to-female ratios. The reason for the striking male preponderance is not clear. One may postulate that if there is a gene locus on the X-chromosome which is related to sensitivity of vitamin D, then a gene dosage effect with the male child being more sensitive to lack of vitamin D, thereby developing rickets in a setting of low calcium intake. In this context, Fisher et al [24] have postulated that vitamin D receptor (VDR) polymorphism might relate to the susceptibility of some Nigerian children to develop rickets when faced with calcium deficiency. They concluded that VDR variant (F allele) was associated with increased risk of developing rickets in the given environment and nutrition. A study in two countries (Turkey and Egypt) reached a similar conclusion [25]. They stated that in the environment of calcium insufficiency combined with vitamin D deficiency, VDR genotypes might predispose to rickets by increased frequency of the F allele. The F allele is believed to confer a transcriptionally more efficient VDR [26].

Majority of the children with rickets, in the present study, were from Christian homes. Similar observation was reported in two previous studies in southern Nigeria (Benin City and Port Harcourt) [20,22]. In contrast, a study from Zaria (Northern Nigeria) reported that majority of the children were from Muslim homes [27]. The opposite finding suggests that the frequency of the type of religion practiced by the family depends on which part of the country the study was conducted. In Nigeria, the Muslims predominate in the north whereas the Christians predominate in the south. It is unlikely that religion had any significant bearing on the occurrence of nutritional rickets in these children. The socioeconomic status (SES) of the families of these children was similar to that reported in the general population of Nigeria,[19] suggesting that SES did not play an important role in the occurrence of rickets in the present study. A similar observation has been reported in previous studies [4].

In the present study, the commonest presenting feature was deformity of the lower limbs. Other studies have observed a similar finding [19,23]. One possible explanation is the easy with which such deformity can be identified by both the parents and the physicians. In contrast, a study at the University College Hospital, Ibadan reported that inability to walk since birth was the commonest presenting complaint [21]. This difference might reflect the documented variability in the chief complaint in children with rickets [3]. In this series, the mean duration of limb deformity before presentation was 20 months. This is not surprising as previous Nigerian studies have reported a similar finding [20,22]. Some of the parents had hoped the lower limb deformity would disappear with time, accounting for delayed presentation. They eventually present when the deformities fail to resolve or worsen. Other possible explanation for the late presentation in Nigeria include lack of easy access to medical facilities affecting majority of the population and wrong beliefs that certain diseases cannot be cured by orthodox medicine.

The general poor socioeconomic status of the parents discourage them from seeking medical attention for their children, particularly if they have to travel long distances and pay from their pockets for health care. It is also possible that because the presenting features of nutritional rickets are not acute and alarming to prompt seeking medical attention. This in contrast to the finding in Sydney where hypocalcaemic seizure is a common presenting complaint [11]. Similarly, hypocalcaemic seizure was reported as presenting complaint in 19% of cases in a Canadian study [16]. In the present study, there was no case presenting with hypocalcaemic seizure.

Some limitations need to be considered. The retrospective design of the study based on data from case notes, limited the analysis not only to those whose case notes could be traced, but also, to the data recorded in such case notes. In conclusion, the clinical, biochemical and radiologic features of severe rickets as seen in Nigerian children are largely similar to its occurrence elsewhere. The unique features among Nigerian children included late presentation, peak age incidence of between 2-3 years and high rate of loss to follow-up.

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