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NUTRITIONAL RICKETS IN YOUNG NIGERIAN CHILDREN IN THE SAHEL SAVANNA*

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ABSTRACT

Objectives: **To determine the prevalence of clinical and biochemical rickets in an under-five out-patient population, relate the prevalence of biochemical rickets (BR) to the socio-cultural characteristics of families and determine the response of nutritional rickets to vitamin D therapy.**

Design: **Prospective cross-sectional and retrospective case-series surveys.**

Setting: **Paediatric general out-patient and consultant clinics.**

Subjects: **One hundred and ninety eight out-patients and twenty two patients aged >1 to 60 months treated for nutritional rickets.**

Interventions: **Clinical examination, interview with mothers and determination of biochemical abnormalities of under-fives and management of patients with rickets using stosstherapy.**

Main outcome measures: **Prevalence of BR and response to stosstherapy.**

Results: **Eight (4%) patients in the survey had clinical and biochemical rickets while 33 (17%) had biochemical rickets only; 92 (47%) other patients had isolated hypocalcaemia and/or hypophosphataemia. The prevalence of BR was higher in males ($p < 0.05$), and increased with age ($p < 0.001$). The prevalence was lower in families who were indigenous to the area ($p < 0.05$), children of Moslem families ($p < 0.05$) and children whose mothers were full-time housewives, unskilled or traders ($p < 0.01$), and who lacked any formal western education ($p = 0.157$). Three of the seven evaluable patients who received stosstherapy responded late.**

Conclusion: **The results support the hypothesis that deficiency or reduced availability of dietary calcium may be of at least equal importance with vitamin D deficiency in the aetiology of nutritional rickets in the Sahel savanna.**

INTRODUCTION

Nutritional rickets (NR) is, unexpectedly, common in tropical Africa(3,4). This paradox has been ascribed mainly to socio-cultural and religious practices which prevent adequate exposure to sunlight(3,5). This explanation, however, overlooks the possibility of other factors(6-8). Aside from this, the reports(3-5) are based on case-series of children with rickets, a limitation which makes generalisation from the results questionable.

The recent report(9) of a survey of rural and semi-urban communities in Nigeria has further confirmed the paradox of NR in the tropics. The report also suggested that the prevalence of NR in the Sahel savanna belt may be particularly high. However, biochemical confirmation of clinically diagnosed NR was not included in the survey nor was the biochemical status of children with non-clinically obvious rickets evaluated(9). These methodological problems limited the inferences which could be drawn from the study regarding the prevalence and aetiology of NR in the area.

Aside from the observations made in the survey(9), our experience (unpublished data) of the response of NR to vitamin D in the area contrasts with reports from other areas in tropical Africa(3,4,10), including the coastal and rain forest belts of Nigeria(3,5). Rather, it suggests a limited role for vitamin D deficiency, which agrees with reports from rural South Africa(6,7).

To complement the results of the survey(9), a socio-cultural, and clinical-biochemical survey of urban under-fives in the same geographic area was done. The aims were to determine the prevalence of clinical and biochemical rickets in an unselected population, relate this to the socio-cultural characteristics of the families and aspects of the histories of the under-fives, and determine the response of rickets to vitamin D. The results are used to formulate a hypothesis for the high prevalence of NR.

MATERIALS AND METHODS

The studies were conducted at the University of Maiduguri Teaching Hospital (UMTH), Nigeria. Maiduguri, in north eastern Nigeria, is in the Sahel savanna. The climate is characterised by

a long dry, dusty season of harmattan and heat periods and a short rainy season. The population is multi-ethnic, and a mix of Christians and Moslems but with the latter in the majority.

Patients and methods in the cross-sectional survey of out-patients: The patients were recruited at the Paediatric General Out-patient Clinic of the UMTH and included all consecutive under-fives, excluding neonates, who were attended to by one of the authors (I.J.), a Senior Registrar in Paediatrics, between the hours of 10 am and 1 pm, Mondays to Fridays, excluding public holidays, from 16/5/95 to 30/6/95.

Data were obtained using a proforma questionnaire. The questions were administered by the Senior Registrar (I.J.) who was fluent in both English and Hausa, the lingua franca in the area. Historical information sought for each patient included socio-demographic- and bio-data of both the mother and child, duration of breastfeeding, age at introduction of cereals to the infant's diet, use of multivitamin preparations or cod liver oil, past history of convulsions or pneumonia (cough associated with fever and difficult/rapid breathing), history of bowing of the legs in the child's siblings and age at walking. Delayed motor milestones was defined as failure to walk without support by 14 months of age.

Physical examination involved seeking for signs of rickets and determination of nutritional status. Patients with a clinical diagnosis of rickets had plain radiographs of the wrists to confirm the diagnosis. Nutritional status was determined by measurement of the mid-upper arm circumference (MAC) and estimation of weight-for-age (WFA) ratios. Nutritional status was classified based on MAC values into normal (MAC >16 cm), subnormal (MAC 13.6 - 15.9 cm), and malnourished (MAC <13.5 cm). Nutritional status was also classified using the Welcome Trust International Working Party criteria based on WFA ratios plus the presence or absence of oedema.

Following clinical assessment, 5 ml of free-flowing venous blood was collected into a plain plastic container from a vein in the antecubital fossa without the application of a torniquet. The serum was separated and stored at -20°C until analysed by (E.A.S) for alkaline phosphatase, calcium, inorganic phosphorus, albumin and urea levels. The analysis was done using standard methods(14,17).

The sample size for the study was calculated using the formular: $n = p(100 - p)/e^2$, with p = 17.3% (determined from the proportion of children with clinically obvious rickets, 2.4% plus the proportion of children with clinical signs suggestive of rickets, 14.9% in the community survey(9)). The minimum sample size of 57 was multiplied by a factor of three to allow for the comparisons expected to be made.

Criteria for the diagnosis of rickets and other abnormalities: Clinical rickets (CR) was defined as the presence of at least three signs of rickets. These signs included widened wrists, bow legs or knock knees/genu valgum intermalleolar or interpatellar distance of at least 2.5 cm, wind-swept deformity of the lower limbs, and beading of the costochondrial junctions. Biochemical rickets (BR) was defined as hyperphosphatasia alone or in association with hypocalcaemia or hypophosphataemia. Isolated hypocalcaemia or hypophosphataemia (isolated HC/HP) was defined as hypocalcaemia or hypophosphataemia alone or in association with each other but without accompanying hyperphosphatasia. Hyperphosphatasia was defined as serum alkaline phosphatase of about twice the upper limit of normal, hypocalcaemia as serum calcium <2.2 mmol/l and hypophosphataemia as serum inorganic phosphorus <1.25 mmol/l(20). Hyperphosphatasia was further classified into mild-moderate (>2 but <4 times upper limit of normal) and severe (>4 times upper limit of normal) and hypocalcaemia into mild-moderate (≥1.88 but <2.2 mmol/l) and severe (<1.88 mmol/l(20).

Data analysis: The patients were classified into groups based on the presence of clinical and biochemical abnormalities. WFA ratios were transformed to Z scores, with Z score = x/sd, where sd is the standard deviation(21). Frequencies were compared using Yates-corrected $\chi 2$-test or Fisher's exact test as appropriate. Means were compared using analysis of variance. Two-tailed p-values <0.05 were taken as significant. Statistical analysis was done using Epi Info Version 6(22).

Retrospective case-series study: The case-series included patients with rickets seen and followed up by one of the authors (GOA) at the Consultant's General Out-Patient Clinic held weekly over a period of two and a half years, January 1993 to June 1995. Clinical diagnosis of rickets was confirmed biochemically and radiologically.

The diagnosis of NR was based on the following criteria: presence of biochemical abnormalities of hyperphosphatasia and hypophosphataemia plus/minus hypocalcaemia, absence of a family history of hereditary forms of rickets, and absence of clinical features of renal or hepatic disease and malabsorption. Renal disease was further excluded by urinalysis and determination of serum urea and electrolytes.

Data retrieved for analysis included biodata, presenting signs and symptoms, results of biochemical and radiological investigations and treatment. Treatment included stosstherapy administered as a single or divided large dose of vitamin D given intramuscularly in the out-patients clinic to ensure compliance. The patients were also given prescriptions for 900-1000 mg of calcium lactate or Sandoz daily and their parents advised on the need for a balanced diet including dairy products.

Response to treatment was determined from a review of changes in the clinical, biochemical and radiological profile. Patients with decreases in alkaline phosphatase and increases in serum calcium and inorganic phosphate, and with radiological signs of mineralisation, were considered to have responded. Response was classified into early (within three months) and delayed (after three months). Biochemical changes on follow-up were computed as a percentage change from the values at diagnosis.

Ethical considerations: Approval for the study was given by the Ethics and Research Committee of the UMTH. Verbal informed consent was obtained from the parents/guardians of the children. The results were made available to the parents/guardians of the children involved in the survey during follow up attendance. This occasion was also used to offer treatment to those children with abnormal results.

RESULTS

Prevalence of clinical and biochemical indices of abnormal calcium metabolism in out-patients: A total of 206 patients were evaluated clinically but only 198 gave consent for venesection and were therefore included in analysis. Eight (4%) patients had CR; the diagnosis of rickets was confirmed radiologically and biochemically in all eight. Four (2%) patients had two clinical signs of rickets. Three of the four had BR while the fourth had only hypocalcaemia. Ten (5%) other patients had single signs. Two of the ten had wind swept deformities. One of the two patients had mild BR while the other had only hypocalcaemia and radiological examination did not show active rickets in either patient. None of the other eight patients with single clinical signs had BR but six of the eight were hypocalcaemic, this being severe in two. The biochemical status of the patients is shown in Table 1 in relation to the presence of clinical signs of rickets.

Table 1

Biochemical status versus number of clinical signs of rickets in under-five out-patients

Biochemical status	No. with clinical signs of rickets*				Total	
	>3 (n =8)	2 (n = 4)	1 (n = 9)	None (n = 176)	No. (n = 198)	% (100)
Hyperphosphatasia only:						
Mild-moderate	–	–	1	9	10	5.0
Severe	–	–	–	2	2	1.0
Hyperphosphatasia plus HC/HP:						
Mild-moderate	3	–	–	14	17	8.6
	(1)			(3)	(4)	
Severe	5	3	–	4	12	6.1
	(4)	(1)		(1)	(6)	
HC/HP only	–	1	7	84	92	46.5
			(2)	(26)	(28)	
Normal	–	–	2	63	65	32.8

Abbreviations: HC/HP = hypocalcaemia or hypophosphataemia

*Figures in parentheses are n with severe hypocalcaemia (serum calcium <1.88 mmol/l)

Table 2

Biochemical status in relation to gender, nutritional status, and renal function status of under-five out-patients

Feature	Biochemical status			p*
	BR (n = 41)	Isolated HC/HP group (n = 92)	Normal (n = 65)	
No. (%) male sex	29 (71)	44 (48)	39 (60)	0.038
Nutritional status:				
n/N (%) with MAC ≤13.5 cm	9/25 (36)	33/44 (75)	16/33 (49)	0.004
No. (%) with WFA ratio ≤80%	9 (22)	40 (41)	18/64 (28)	0.026
No.,	41	92	64	0.002
Mean,	90.12	82.47	89.94	
sd WFA ratio in %	13.92	14.30	15.43	
No.,	41	92	64	0.001
Mean,	6.03	5.50	5.99	
sd WFA z scores@	0.91	0.95	1.03	
No.,	41	92	65	0.022
Mean,	37.77	35.86	37.49	
sd serum albumin in g/l	4.52	4.59	4.07	
Renal function status:				
No.,	35	72	65	0.008
Mean,	3.01	2.07	2.46	
sd serum urea in mmol/l	1.79	1.30	1.37	

Abbreviations: BR = biochemical rickets, Isolated HC/HP = isolated hypocalcaemia/hypophosphataemia, MAC = mid arm circumference, WFA = weight for age ratio, sd = standard deviation

* Group χ^2 test for difference in frequencies; analysis of variance for difference between means.

@ z scores calculated using the sd for the entire sample of 198 patients (mean (sd) WFA ratio = 86.39 (15.00)%)

Sixty five (33%) patients were biochemically normal. Two of the 65 had a single clinical sign of rickets. Ninety two (47%) patients had isolated HC/HP. One of the 92 had two clinical signs of rickets, while seven had single signs and 84 had no sign. Hypocalcaemia was severe in 28 (30%) of the 92 patients with isolated HC/HP.

Forty one patients (21%) had BR alone (6%) or in association with hypocalcaemia or hypophosphataemia (15%). BR was severe in 14 (34%) of the 41 patients. Eight (20%) of the 41 patients with BR also had CR, three (7%) had two clinical signs of rickets, and one (2%) a single sign (windswept deformity) while 29 (21%) had no sign. The difference in the prevalence of BR in patients with at least two clinical signs versus those with <1 sign was highly significant (relative risk (95% CI) = 42.1 (5.6, 316.9), p <0.0001).

The prevalence of severe hypocalcaemia was 5/8 (63%) in patients with CR, 3/14 (21%) in those with one to two clinical signs of rickets and 30/176 (17%) in those with no sign (χ^2 = 10.24, df = 2, p = 0.006).

Prevalence of biochemical abnormalities in relation to gender, age, nutritional status, and clinical diagnosis: This is shown in Table 2. Males were more frequent among patients with BR (p = 0.038).

Based on the WFA ratio, four (2%) patients were severely malnourished(13). Three had marasmus and one marasmic-kwashiorkor. Two of the severely malnourished patients had isolated HC/HP and two were normal biochemically. Based on the MAC, 58 (57%) of the 102 eligible patients (age >12 months) were malnourished.

Biochemical status was significantly related to nutritional status (p=0.026 for the frequency of malnutrition using WFA ratio, p=0.004 for the frequency of malnutrition using MAC, p = 0.002 for the variance of WFA ratio, p = 0.001 for the variance of Z scores of WFA ratio, and p = 0.022 for the variance of serum albumin). The variance of serum urea was also significantly (p = 0.008) related to biochemical status. Generally, patients with BR were comparable to those with normal biochemical status while those with isolated HC/HP were different (Table 2).

The relationship between age and biochemical status is shown in Table 3. There was a significant linear trend ($\chi 2$ for linearity = 15.09, p <0.001, df = 2) of increasing prevalence of BR with age after the first half of infancy. There was no significant relationship between the prevalence of isolated HC/HP and age ($\chi 2$ for linearity = 3.40, p = 0.183, df = 2).

The influence of the family's religion on the biochemical status within the different age groups is also shown in Table 3. Overall, 70 (35%) were Christians. The frequency of BR varied with age among patients in both religions, p = 0.02 for the Christians and p = 0.093 for the Moslems.

Prevalence of BR and isolated HC/HP in relation to clinical diagnosis: This is shown in Table 4. The prevalence of BR was significantly higher in patients with a diagnosis of rickets while the miscellaneous diagnoses were significantly more frequent in those with a normal biochemical status.

Table 3

Variation of biochemical status with age in under-five out-patients

Biochemical status	Age in months*				Total	
	>1-6 (n = 42) [18]	7-12 (n = 52) [11]	13-24 (n = 59) [21]	25-60 (n = 45) [19]	No. (n = 198)	%
Normal	13 [7]	24 [6]	18 [4]	10 [3]	65	32.8
Biochemical rickets	12 [7]	4 [0]	8 [4]	17 [9]	41	20.7
%	28.6	7.7	13.6	37.8		
Isolated hypocalcaemia/hypophosphataemia	17 [4]	24 [5]	33 [14]	18 [7]	92	46.5

*Figures in parentheses are n patients from Christian families

Statistics: χ^2 for distribution of biochemical status with age = 19.66, df = 6, p = o 003; χ^2 for variation of biochemical rickets with age = 16.77, df = 3, p <0.001; and χ^2 for variation of isolated hypocalcaemia/hypophosphataemia with age = 3.49, df = 3, p = 0.322.

Table 4

Relationship between biochemical abnormalities and clinical diagnosis among under-five out-patients in the survey

Clinical diagnosis	No. (%) of patients (n = 198)	No. with BR (n=41)	HC/HP (n = 92)	p*
Non-respiratory bacterial infections**	23 (11.6)	8	11	0.114
Acute respiratory infections	34 (17.2)	8	16	0.858
Malaria	47 (23.7)	7	28	0.593
Acute respiratory infections plus malaria	7 (3.5)	–	3	0.243
plus acute watery diarrhoea	7 (3.5)	–	4	0.386
Diarrhoea:				
acute watery	4 (2.0)	–	2	Not valid
persistent	5 (2.5)	–	4	0.278
acute dysenteric	11 (5.6)	–	6	0.208
Rickets	15 (7.6)	12	1	<<0.001
Severe protein energy malnutrition	4 (2.0)	–	3	Not valid
Miscellaneous@	41 (20.7)	6	14	0.002

*Group χ^2 for difference in frequencies.

**Pyoderma, conjunctivitis, abscesses, septicaemia, etc.

@Non-specific skin rash, unexplained weight loss, aphthous ulcer, helminthic infections, etc; one child with biochemical rickets presented with twitching, and one with normal biochemical status had recurrent convulsions.

Table 5

Prevalence of biochemical abnormalities in relation to selected maternal socio-cultural characteristics, sib ship history, and under-five characteristics in the survey

		No. of patients	No. with		p*	
		(n = 198)	BR (n = 41)	HC/HP (n = 92)	1	2
Socio-cultural features:						
Ethnic group:						
	Kanuris	40	4	25	0.044	0.04
	Hausa-Fulanis	44	6	20		
	Other northerners+	53	12	20		
	Southerners++	61	19	27		
Western educational:						
	None	94	14	46	0.431	0.157
	Primary	26	7	11		
	Post-primary	78	20	35		
Occupation:	Full-time housewives	151	24	73	0.016	0.005
	Unskilled/trading	16	4	6		
	Skilled/professionals@	31	13	13		
Religion:	Islam	129	21	62	0.1	0.036
	Christianity	69	20	30		
Sibship history of bow legs:						
	Yes	20	12	6	<0.001	<0.001
	No	178	29	86		
Under-five characteristics:						
Delayed walking:	Yes	10	5	2	0.076	0.065
	No	98	20	49		
Past history of:						
pneumonia:	Yes	28	11	11	0.029	0.009
	No	170	30	81		
convulsions:	Yes	19	8	7	0.053	0.027
	No	179	33	84		

Abbreviations: BR = biochemical rickets, HC/HP = isolated hypocalcaemia/hypophosphataemia
*p group χ^2 test: 1 = overall, 2 = prevalence of BR; +Mandara, Shuwa Arab, etc;
++Ibos, Yorubas, etc;
@skilled workers = seamstresses, etc; professionals = teachers, nurses, etc.

Overall, the prevalence of a presumed infective illness (bacterial, viral or malarial) was significantly higher in patients with isolated HC/HP (74/92 versus 23/41 in patients with BR and 41/65 in normal patients; p = 0.007 for group with 2 df). The other associations were not significant.

Prevalence of BR in relation to selected child-raising and socio-cultural characteristics of mothers and histories of under-fives: There was no significant relationship between the prevalence of BR or isolated HC/HP and the use or non-use of cod liver oil or multivitamin preparations, duration of breastfeeding or age at introduction of cereals to the infant's diet.

The influence of maternal socio-cultural features on the prevalence of BR is shown in Table 5. The prevalence of BR was highest in children of migrants from southern Nigeria (p <0.05), "working-class" mothers (p <0.01), Christians (p <0.05) and mothers with Western education (p = 0.157). The relationship between the prevalence of BR and sibship history and histories of the under-fives is also shown in Table 5. Only the effect of delayed motor milestones did not attain statistical significance. None of the factors shown in Table 5 had a significant association with the prevalence of isolated HC/HP.

Case-series study: Twenty four patients with rickets were seen during the period. Two patients were excluded from analysis: the first had rickets secondary to chronic liver disease, while the second had rickets secondary to prolonged prophylaxis (31 months) with diateben/ rifampicin.

The twenty two patients analysed included 13 males and nine females. The median age was 28.5 months. The youngest patient was 13 months of age and the oldest eight years, but only one was older than five years. Four patients (three males, one female) had a history of older sibs who had been "successfully" treated for rickets but no patient had a family history suggestive of hereditary rickets. Eleven patients were from Christian families. Twelve were of non-Kanuri, non-Hausa-Fulani ethnic origin.

Table 6

Summary of clinical, radiological, biochemical and therapeutic profile of patients in the case series (n=8)

Serial No.	Age, sex, religion duration of symptoms	% abnormality in basal chemistry	Total dose of vitamin D given, duration of follow-up, %change in chemistry**	Remarks
1.	36 mo, M, Christian, ill 14 mo	AP +968, ca -55, Pi-87	600,000 iu, 2 mo, AP-502, Ca +32, Pi+113	Well nourished; early response; limb pains subsided by 1 mo of treatment
2.	30 mo, F, Moslem,	AP +43, ca-50, Pi-200	1, 200, 000 iu, 3.25 mo, AP +127, Ca+41, Pi-80	Marasmic child with florid clinical and radiological signs; early response; walked by 2 mo of treatment.
3.	36 mo, M, Moslem, ill 2 mo	AP +141, Ca-82, Pi-73	1,200,000, iu, 4 mo, AP-74 Ca +33, Pi +243	Underweight; early response; radiological signs of healing by 4 mo of treatment.
4.	33 mo, M, Moslem, ill 8 mo	AP +329, CA -91, Pi-200	1, 200,000 iu, 10 mo AP -149, +36, Pi +150	Well nourished, early response, limb pains/ limping subsided by 2 mo of treatment.
5.	36 mo, F, Christian, ill 24 mo	AP +152, Ca -107, Pi -233	1, 200, 000 iu, 6 mo, Ap -68, +2, Pi +133	Underweight; delayed response, walked by 3 mo of treatment; legs straightening by 6 mo.
6.	16 mo, M, Christian, ill 4 mo	AP +682, Ca -114, Pi -237	1, 400, 000, iu, 26 mo, Ap -542, Ca +38, Pi +117	Well nourished; florid signs; delayed response; radiological signs of healing by 8 mo; residual deformities by 20 mo.
7.	36 mo, M, Christian, ill 12 mo	Ap +300, Ca -114, Pi -338	1, 800, 000, iu, 21 mo Ap -149, Ca +32 Pi +197	Underweight; delayed response; mother (a nurse) inadvertently gave twice the advised dose of vit D. at diagnosis; walked by 2 mo; radiological signs of healing by 5 mo; residual deformities by 21 mo.
8.	22 mo, M, Moslem, ill 9 mo	AP +40, Ca -82, Pi -183	900, 000, iu, 25 mo, AP +83, Ca + 21, Pi +67	Well nourished; bronchopneumonia at diagnosis and 5 mo later; clinical signs included pigeon chest; responded but ?type

*Percentage elevation or depression at diagnosis calculated from reference range of normal (lower limit for serum calcium and phosphorous and upper limit for alkaline phosphatase);

** computed as percentage change from the original.

Abbreviations: AP, Ca, Pi = serum alkaline phosphatase, calcium and phosphorus.

Five patients had bronchopneumonia, one suppurative otitis media and one tetany on presentation. Two patients had a history of recurrent convulsions. Three patients had marasmus. Four patients had wind swept deformities only while eighteen had combination of features, including wind swept deformities in four. Eighteen patients had hypocalcaemia and ten had hypophosphataemia. The radiological features of rickets were "mild" in eight patients and "florid" in fourteen. All the 22 patients had normal serum urea and electrolytes and urinalysis profiles. The evaluation of response to vitamin D was possible in only eight patients. Eight patients did not attend for follow up or attended for <1 month while six others who attended for follow up lacked enough follow up radiological and biochemical data for assessment.

The stosstherapy, clinical, radiological and biochemical profiles and response to treatment of the eight evaluable patients are summarised in Table 6. Four patients (Nos. 1-4) were early and three (Nos. 5-7) late responders while response was difficult to judge in one patients (No. 8) who lacked early biochemical assessment on follow up. None of the patients showed clinical or biochemical evidence of hypercalcaemia during follow up.

DISCUSSION

There has not, to our knowledge, been any study of an unselected urban outpatient population to define the size of the problem of NR in West Africa. The 4.0% prevalence of CR in the present study is higher than the 2.4% in rural and sub-urban communities in the region(19) but similar to the 5.3% in urban communities in India(19). It is much lower than the 30% prevalence reported among Ethiopian out-patients some three decades ago(10). The pattern of illnesses in the survey is similar to that in ambulatory patients in most hospitals in the tropics. The higher prevalence of CR in out-patients versus the community may be because children with NR have a higher incidence of common illnesses and are thus more likely to be taken to the hospital.

Two striking observations are the high prevalence of BR, which is far in excess of that of CR, and the even higher prevalence of isolated HC/HP. The rather inevitable conclusion from these observations is that there may be widespread disorder of calcium homeostasis in the area. This supports the observation of an unusually high prevalence of NR made in the community survey(9).

The high overall prevalence of biochemical abnormalities (74% had at least one abnormality), and the limited correlation between clinical and biochemical diagnosis (only eight of the 41 patients with BR had GR), indicate that clinically inapparent rickets is an important problem in the area. Mass screening for BR may thus be worthwhile to facilitate early diagnosis and treatment. Late presentation is common in NR in the tropics(3,10) and may be associated with increased mortality(10).

The relative importance of calcium or vitamin D deficiency or reduced bioavailability of dietary calcium, factors which operate singly or together to determine the prevalence of NR(6), varies with geographical location, culture, religion, socio-economic status, and domicile(36). While earlier reports from north(4), west(3) and east(10) Africa emphasize the dominance of vitamin D deficiency, the importance of dietary calcium deficiency has been shown in South Africa(6,7,23). The pattern of biochemical derrangements, and the disparity in prevalence of CR and BR, in the present study is similar to that reported from rural South Africa(6,7,23).

Other reports also emphasise the significance of dietary calcium deficiency. Walker has commented extensively on the causes of a low intake of calcium in Africans. Maize, millet, guineacorn, groundnut and beans, common staples in the Sahel savanna, are low in calcium(25). This can be compounded by limited access to richer sources of calcium such as green leafy vegetables, poultry and dairy products. Also, oxalates and phytates in unrefined cereals can compete with dietary calcium for absorption. Dietary calcium supplementation for both pregnant women, lactating mothers, and under-fives may be worthwhile in this region.

Vitamin deficiency rickets of nutritional origin is commoner in males(3,4), unlike rickets due to calcium deficiency(6,7). Only the higher prevalence of BR in males in this study is in support of vitamin D deficiency as an aetiological factor in the prevalence of NR in the area.

The other results support a greater role for the deficiency or reduced availability of dietary calcium. First, is the trend in prevalence of BR with age. The peak age for vitamin D deficiency rickets is lower than that of calcium deficiency rickets, while the incidence of the former usually decreases with age. Second, there was a relatively low prevalence of CR (4%) in the presence of a high prevalence of BR(21%). This is the pattern associated with dietary calcium deficiency(7). It contrasts with the high prevalence of CR in vitamin D deficient populations(10). Third, is the relationship between the prevalence of BR and socio-cultural factors. This is unlike that expected in NR due to inadequate exposure to sunlight(3,4). Fourth, there was delayed response to vitamin D in three of seven evaluable patients, the need for repeat stosstherapy to elicit response, and rarity of toxicity from repeated stosstherapy. Only nine of 66 patients with NR in two studies(4,27) of patients given stosstherapy required further doses of vitamin D to achieve response. Although delayed response was also reported in an earlier series from Ibadan, Nigeria(3), small daily doses of vitamin D rather than stosstherapy were used and delayed response was observed only in marasmic children. Sixty per cent of the patients had marasmus in the Ibadan study(3), while most of our patients were well nourished or had only undernutrition. Response to stosstherapy in vitamin D deficiency rickets is expected within two weeks and certainly by six to twelve weeks(27), whereas response to treatment is slower in rickets due to calcium deficiency(24).

Finally, there was high prevalence of hypocalcaemia and hypophosphataemia, both in the prospective survey and case-series study. In contrast, most of the patients in the Ibadan study(3) had normal serum calcium levels. Sustained calcium deficiency leads to secondary hyperparathyroidism with consequent hyperphosphaturia and hypophosphataemia(20,24). Although dietary practices which exclude dairy products can produce combined calcium and vitamin D deficiency(27), the abundant sunshine in the Sahel savanna may be expected to compensate for dietary deficiency of vitamin D.

Other findings in this study may also be discussed. First, is the association between a past history of pneumonia and the prevalence of BR. This may be because rickets predisposes to bronchopneumonia(4,10). Second, tetany was observed in only one, and convulsions in two, respectively, of the 22 patients in the case-series study. Other authors(4,27) have also noted the rarity of tetany as a manifestation of rickets whereas the frequency of a history of convulsions is more variable, eight of forty one patients in one study(4) and one of 42 in another study(27). Our results are within these limits. The tendency to the high frequency of a past history of convulsions in those with BR in the survey can be explained by the varying degrees of associated hypocalcaemia. Thirdly, there was a positive sibship history of rickets in 18% of patients in the case-series study. This is close to the 11% in Sudanese children(4). Twenty nine per cent of the patients with BR in the survey also had a positive sibship history of bow legs. El Hag and Karrar(4) have commented that a family history of rickets in settings with a high prevalence of NR is likely to be environmentally, rather than genetically, determined. This may even be truer in the Sahel savanna, with the high likelihood of calcium deficiency. Fourthly, there was a significant relationship between nutritional status and biochemical indices of calcium metabolism. This relationship has been commented on by other authors(3,4,10). Finally, there was a high default rate from follow up in the case series study. This is worrying, but justifies the use of stosstherapy, the safety(27) of which was further confirmed in this study.

One limitation in this study was the inability, due to lack of resources, to determine the serum calcidol and parathyroid hormone levels. Perhaps, the results could have helped to determine, conclusively, the validity or otherwise of the hypothesis that vitamin D deficiency may be less important than dietary calcium deficiency as an aetiological factor in the prevalence of NR in the area. This limitation should, however, not be detrimental to the main

messages in the report but should be considered, if possible, in future studies.

In conclusion, the frequency of CR markedly underestimates that of BR in the Sahel savanna and "routine" or "mass screening" for biochemical abnormalities may be justified to facilitate early diagnosis and treatment. Although the aetiopathogenesis of the observed abnormalities can be multifactorial, the pattern of biochemical derangements and the relationships with socio-biological and religio-cultural factors suggest that deficiency or reduced availability of dietary calcium may be of at least equal importance with vitamin D deficiency.

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