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The Relationship between Bone Mineral Density and Dietary Intake in Moroccan Children with Juvenile Idiopathic Arthritis

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ABSTRACT

Background and Objective: The aim of this study was to evaluate the association between dietary intake and bone mineral density in children with juvenile idiopathic arthritis (JIA). **Methods:** A cross-sectional study carried out in Morocco between May 2010 and June 2011, covering out patients with JIA. The characteristics of patients were collected. The nutritional status was assessed by a food questionnaire including data of food intake during 7 consecutive days using 24-hour dietary recall. Food intake was quantified using the software Bilnut (Bilnut version 2.01, 1991). Bone mineral density (BMD in g/cm^2) was measured by DXA method (X-ray absorptiometry) on a Lunar Prodigy. **Results:** The study consisted of 33 patients with JIA (4 - 16 years old). The median age of patients was 10.4 ± 4.3 years. Median disease duration was 2 (1 - 4.5) years. The group of patients with low dietary intake of proteins was associated with low BMD ($p = 0.03$). Low BMD was related with low intake of magnesium ($p = 0.007$) and vitamin C ($p = 0.04$) in children aged between 4 and 9 years. Low intake of vitamin E and folate was associated with high BMD in the other range of children ($p < 0.001$). **Conclusion:** This study suggests that low intake of protein and of some micronutrients (magnesium, vitamin C, vitamin E and folate) influence bone mass in children with JIA. Prospective studies with a larger number of patients seem to be necessary in order to confirm our findings.

Keywords: Juvenile Idiopathic Arthritis; Macronutrients; Vitamins; Trace Elements; Bone Mineral Density

1. Background

Osteoporosis is currently estimated to be a major health threat [1]. It's defined by a disease characterized by loss of bone masse, accompanied by microarchitectural deterioration of bone tissue, which leads to an unacceptable increase in the risk of fracture [2]. About 90% of total adult mass is accrued by age 20, and a signification proportion of this is archived during puberty alone [3].

Juvenile idiopathic arthritis (AJI) is one of the commonest rheumatic diseases of children [4]. In one hand, several studies demonstrate reduced bone mineral density (BMD) in children with JIA [5,6]. In the other hand, JIA is often associated with poor nutritional status [7]. Nutrition is a key factor, not only for bone growth, but also for its mineralization [5]. The acquisition of adequate mineralization during childhood has proven to be a key event in the prevention of osteoporosis in adults [8]. Recent studies on various dietary components have shown that there is some correlation between their daily food intake and the genesis of osteoporosis and its fracture complications [9]. An inadequate nutrition (especially intake of macronutrients, trace elements and vitamins), can be associated with an increase in bone remodeling leading to significant loss of bone and an increase fracture risk [10]. There are few studies available in the literature assessing the relation sheep between dietary intake and bone mineral density in children. In addition, there are no studies in a Moroccan population that evaluate the same subject. The aim of this study was to assess the relationship between the dietary intake and bone mineral density in Moroccan children and adolescents with JIA.

2. Materials and Methods

2.1. Data Collection

It was a cross sectional study of children with JIA over a

*The authors declare that they have no conflicts of interest concerning this article.

period of 13 months (between May 2010 and June 2011) at the department of rheumatology of El Eyachi University hospital and department of pediatrics of university hospital of children of Rabat-Sale. Informed consent was obtained by parents from all subjects and the study was approved by ethics committee of our university hospital.

The diagnosis of JIA was based on the criteria of the International League of Association for Rheumatology (ILAR) [11]. Patients were recruited in consultation or during hospitalization. We excluded patients with any other chronic disease (endocrinal, neurological, cardiac, and renal) that affect bone metabolism. The disease and patients characteristics considered as explanatory measures were: age (year), gender, diagnosis (JIA subtype), disease duration (years), disease activity was assessed using a visual analogical scale (VAS), functional disability was determined by using the Moroccan version of Childhood Health Assessment Questionnaire (CHAQ) [12], number of tender joints, number of swollen joints and the erythrocyte sedimentation rate (ESR). Treatment with NSAIDs, corticosteroid and disease modifying antirheumatic drugs (DMARDs) was determined.

2.2. BMD Assessment

All BMD measurements were obtained with the same DXA instrument (Lunar Prodigy; GE Lunar, Madison, WI). BMD (g/cm²) was measured in the lumbar spine (L1-L4) and total body. The lumbar spine and the total body BMD values were transformed into Z scores by comparing them with age- and sex-specific reference values for this equipment [13,14]. According to the International Society for Clinical Densitometry recommendations osteoporosis was defined as a Z-score less than 2 with a fracture history. Low BMD was defined as a Z-score less than 2 without a significant fracture history [15].

2.3. Dietary Evaluation

Nutrient intake was determined using the 24 hour diet recall during 7 consecutive days [16]. The food questionnaire had two parts; the first identified all foods consumed during the day previous to the interview; the second part; specified food frequency to appreciate food eating habits. Two nutritionists analyzed the food dietary to quantify the food consumed from the recorded information. Nutrient intake was analyzed by software bilnut (Bilnut version 2.01, 1991), validated and standardized. The dietary intake of macro and micronutrients were assessed against the recommended dietary allowances (RDA) [17]. The analysis of micronutrients was made according to the age (group between 4 years and 9 years and group between 10 years to 16 years). We considered that 50% to 60% as the appropriate percentage of calories from carbohydrates, between 10% and 15% the percentage related to proteins and between 25% to 30% the percentage of lipids [7].

2.4. Anthropometric Measures

Weight (kg) and height (m) were measured according to the recommendation of the World Health Organization (WHO). The results of the BMI (Kg/m^2) were compared with reference values of Hammer *et al.* [18,19].

2.5. Statistics

Analysis was carried out using the statistical package for the social sciences (SPSS) version 16.0. Data for patients were presented as mean ± standard deviation or median (IQ) for continuous variables and as frequencies and percentage for categorical variables. For dietary intake of macronutrients, we are divided patients on 3 groups, low, normal and high dietary intake, and we used one way Anova test to compare values of BMD (g/cm^2) between the 3 groups. As regards micronutrient intake, Student's t-test for independent samples was used to compare values of BMD (g/cm^2) between two groups: with low and with normal dietary intake of micronutrients. Significance level was p value less than 0.05.

3. Results

Thirty three patients where included in this study. The mean age of our patients was 10.4 ± 4.35. 54.5% of our patients were males. The median disease duration was equal to 2 (1 - 4.5) years. Eleven patients (33.3%) had a low BMD in lumbar spine, and nine (27%) in total body, and no patient had an osteoporosis. Demographic and clinical characteristics of patients are described in **Table 1**.

We found that patients with JIA had an excessive intake of proteins, carbohydrates and lipids in 30.3%, 63.6% and 54.5% of the cases respectively. Moreover all patients had a low consumption of micronutrients.

Low intake of proteins was associated with a low BMD ($p = 0.03$) (**Table 2**). No difference was observed between dietary intake of glucids and lipids, and BMD (**Table 2**).

Daily mean intake of micronutrients show that low dietary intake of vitamin C was associated with an increase on BMD ($p = 0.04$) and low dietary intake of magnesium was associated with decreased BMD ($p < 0.0001$) in children aged between 4 and 9 years (**Table 3**). Low intake of vitamin E and folate was associated with increased BMD in children between 10 and 16 years ($p < 0.001$) (**Table 4**).

4. Discussion

In our study, we show that low dietary intake of proteins was associated to reduce bone density. It has been suggested that dietary protein intake may be a risk factor for osteoporosis, especially in childhood, and high-protein diets are associated with increased bone loss [5]. Two mechanisms are discussed. The protein metabolism is accompanied by a significant production of amino acids that can promote osteoclast function and bone resumption. Also, protein intake may be involved indirectly in the genesis of osteoporosis by altering the metabolism of insulin like growth factor (IGF)-I [20,21].

Zhang Q. *et al.*, found that higher protein intake, especially from animal foods, appeared to have a negative effect on bone mass accrual in pubertal girls, which is different with our data [22]. In the study of Vatanparast H, they found that protein intake has a beneficial effect on bone mass of young adult females when calcium intake is adequate; protein, in the absence of sufficient calcium, does not confer as much benefit to bone [23]. In the longitudinal study including 560 women aged between 14 and 40 years, they suggest that a higher protein intake does not have an adverse effect on bone, and low intake on vegetal protein is associated with a less bone mass, [24].

Moreover, many scientists have examined the relationship between types of protein and urinary calcium excretion, and found that animal protein was associated with increased urinary calcium excretion, soy protein was not. There is sufficient evidence suggesting soy isoflavones may have potential benefits for bone, but a relationship has not been established between the consumption of ipriflavone and maintenance of bone mineral density [25].

These studies were conducted in healthy subjects, while our population is made of children with chronic inflammatory arthritis and who take corticosteroids which could lower their bone mineral density and subsequent can may explain our results.

In our study, we found that dietary intake of glucids and lipids were not associated with bone mass. Epidemi-

Table 1. Clinical characteristics of patients.

Characteristics AJI	(n = 33)
Age (year)[1]	10.45 ± 4.35
Sex males[2]	18 (54.5)
DAS28 ESR[1]	5.30 ± 1.10
Disease duration (year)[3]	2 [1 - 4.5]
Visual analogical scale (0 - 10)[3]	20 [10 - 50]
CHAQ score (0 - 3)[3]	0.5 [0 - 1.6]
JIA clinical subtypes[2]	
Oligoarticular	9 [27 - 3]
Polyarticular	16 [48 - 5]
Systemic	8 [24 - 2]
Nutritional status[2]	
Underweight	9 (27.3)
Normal	16 (48.5)
Obesity	8 (24.2)
BMD lumber spine (g/cm^2)[3]	0.6 [0.1 - 1]
BMD total body (g/cm^2)[3]	0.7 [0.3 - 1.1]
Z score lumber spine < –2[2]	11 (33.3)
Z score total body < –2[2]	9 (27.3)
NSAID[2] (yes)	26 (78.7)
DMARDs[2] (yes)	17 (51.6)
Oral corticosteroid[2] (yes)	14 (42.4)

DAS28 = disease activity score; CHAQ = Childhood Health Assessment Questionnaire; JIA = juvenile idiopathic arthritis; BMD = bone mineral density; NSAID = non-steroidal inflammatory drugs; DMARDs: Disease-Modifying Anti-Rheumatic Drugs. [1]Mean ± S.D.; [2]Number and percentage; [3]Median and IQR.

Table 2. Association between daily macronutrients intake and BMD in children with JIA.

BMD (g/cm^2)		Lumber spine	p	Total body	p
Proteins (% of energy)	Low	0.510 ± 0.155	**0.03**	0.735 ± 0.049	0.2
	Normal	0.833 ± 0.192		0.840 ± 0.134	
	High	0.627 ± 0.230		0.246 ± 0.205	
Glucids (% of energy)	Low	0.619 ± 0.260	0.4	0.726 ± 0.179	0.4
	Normal	0.714 ± 0.242		0.785 ± 0.239	
	High	0.733 ± 0.193		0.826 ± 0.112	
Lipids (% of energy)	Low	0.723 ± 0.180	0.7	0.752 ± 0.179	0.2
	Normal	0.710 ± 0.323		0.908 ± 0.202	
	High	0.652 ± 0.245		0.749± 0.147	

BMD: bone mineral density; JIA: juvenile idiopathic arthritis. Values are the mean ± SD; p, descriptive level of one way Anova. Low dietary intake of proteins was associated with low BMD in lumber spine.

Table 3. Association between daily micronutrients intake and BMD in children with JIA aged between 4 and 9 years.

BMD (g/cm²)	Lumber spine	p	Total body	p
Calcium (mg)				
Low	0.552 ± 0.209	0.7	0.720 ± 0.068	0.2
Normal	0.600 ± 0.001		0.650 ± 0.070	
Phosphorus (mg)				
Low	0.574 ± 0.223	0.6	0.713 ± 0.085	0.8
Normal	0.522 ± 0.093		0.705 ± 0.010	
Magnesium (mg)				
Low	0.100 ± 0.000	**0.007**	0.700 ± 0.000	0.8
Normal	0.594 ± 0.146		0.711 ± 0.073	
Fer (mg)				
Low	0.563 ± 0.234	0.9	0.725 ± 0.079	0.3
Normal	0.552 ± 0.104		0.684 ± 0.047	
Zinc (mg)				
Low	0.587 ± 0.261	0.6	0.727 ± 0.091	0.4
Normal	0.531 ± 0.103		0.694 ± 0.044	
Vitamin B1 (mg)				
Low	0.574 ± 0.223	0.6	0.723 ± 0.075	0.3
Normal	0.522 ± 0.093		0.680 ± 0.054	
Vitamin C (mg)				
Low	0.562 ± 0.234	0.9	0.738 ± 0.064	**0.04**
Normal	0.530 ± 0.100		0.634 ± 0.052	
Vitamin E (mg)				
Low	0.563 ± 0.200	0.7	0.711 ± 0.073	0.8
Normal	0.500 ± 0.001		0.700 ± 0.001	
Folate (µg)				
Low	0.560 ± 0.208	0.9	0.720 ± 0.068	0.2
Normal	0.550 ± 0.070		0.650 ± 0.070	

BMD: bone mineral density; JIA: juvenile idiopathic arthritis. Values are the mean ± SD; p, descriptive level of Student's t-test. Low intake of magnesium and vitamin C was associated with decreased BMD in lumber spine, and increased BMD in total body respectively.

Table 4. Association between daily micronutrients intake and BMD in children with JIA aged between 10 and 16 years.

BMD (g/cm²)	Lumber spine	p	Total body	p
Calcium (mg)				
Low	0.805 ± 0.250	0.9	0.761 ± 0.229	0.5
Normal	0.800 ± 0.001		0.900 ± 0.001	
Phosphorus (mg)				
Low	0.805 ± 0.250	0.9	0.761 ± 0.229	0.5
Normal	0.800 ± 0.001		0.900 ± 0.001	
Magnesium (mg)				
Low	0.773 ± 0.245	0.2	0.753 ± 0.241	0.4
Normal	0.973 ± 0.180		0.876 ± 0.040	
Fer (mg)				
Low	0.784 ± 0.241	0.3	0.758 ± 0.234	0.4
Normal	0.980 ± 0.254		0.900 ± 0.001	
Zinc (mg)				
Low	0.773 ± 0.245	0.2	0.753 ± 0.241	0.4
Normal	0.973±0.180		0.876 ± 0.040	
Vitamin B1 (mg)				
Low	0.805 ± 0.250	0.6	0.766 ± 0.229	0.8
Normal	0.800 ± 0.001		0.900 ± 0.001	
Vitamin C (mg)				
Low	0.829 ± 0.222	0.2	0.789 ± 0.228	0.3
Normal	0.600 ± 0.424		0.630 ± 0.190	
Vitamin E (mg)				
Low	0.805 ± 0.243	**<0.001**	0.761 ± 0.229	**<0.001**
Normal	0.500 ± 0.001		0.600 ± 0.001	
Folate (µg)				
Low	0.805 ± 0.243	**<0.001**	0.789 ± 0.228	**<0.001**
Normal	0.500 ± 0.001		0.600 ± 0.001	

BMD: bone mineral density; JIA: juvenile idiopathic arthritis. Values are the mean ± SD; p, descriptive level of Student's t-test. Low dietary intake of vitamin E and folats was associated with high BMD in both lumber spine and total body.

ological data indicate that high-fat diets, especially those rich in saturated fatty acids, may contribute to reduced bone density and increased fracture risk, in older as well as younger people [26]. One other study that assessed the relation of dietary fat to hip bone mineral density (BMD) in men and women indicated that BMD is negatively associated with saturated fat intake [27]. Several studies have shown the importance of individual fatty acids in enterocyte membrane dynamics, Vitamin D3 activity, and prostaglandin formation, which can have important effects on intestinal calcium absorption as well as urinary calcium excretion [28]. Also, dietary lipids can influence GH and osteoblast formation [21].

In their study, Rubinacci A. *et al.*, show the presence of positive relationships between bone mineral content (BMC) and lipid intakes in the population of women in early menopause, but they have no association between glucids intakes and BMC [29]. In animal study, it has been demonstrated in a group of rats with a diet rich on fructo-oligosaccharides decreased content bone on calcium and phosphorus and resistance bone, compared to a control group [30]. But these results are contradictory with data of other studies.

Regarding the dietary intake of micronutrients, our results show that low intake of magnesium is related with a low bone mineral density. Rude *et al*. tested the effects of deficient diets on bone tissue in rats [31]. The results showed the histology decreased trabecular bone volume, increased osteoclast activity without activation of osteoblasts and biologically hypercalcemia, a decrease in serum parathyroid hormone (PTH) and 1, 25 (OH) vitamin D. Thus, the Mg depletion would lead to bone resorption uncoupled could exert inhibitory effect on PTH. In the data of the literature, small epidemiologic studies suggest that an excessive magnesium intake was associated with higher BMD in elderly men and women [32,33]. In clinical trials of magnesium supplementation, there is a little evidence that magnesium is essential to prevent osteoporosis in the general population [34,35]. Also, one recent study from the WHI suggested that higher intakes of magnesium were associated with a risk of wrist fracture [36].

Vitamin C is an essential cofactor for collagen formation and synthesis of hydroxyproline and hydroxylysine. A several studies show a positive association between vitamin C and bone mass. Low intakes of vitamin C are associated with loss of BMD [37,38]. Celia J. Prynne *et al.*, explored the association between bone mineral status and fruit and vegetable intakes in adolescent boys and girls (aged 16 - 18 y), young women (aged 23 - 37 y), and older men and women (aged 60 - 83 y). In boys, significant positive associations were found between dietary vitamin C and BMC and BMD. No significant univariate association was found in the girls, and a significant negative association with BMD was found in the older women [39]. One study found that higher intake on vitamin C was associated with fewer fractures; however, there are no randomized clinical trials, [40]. In our study, we found a significant association between low intake of vitamin C and increased bone mineral density, which is contradictory with the literature data.

In our data, we showed a negative association between intake of vitamin E and BMD. In a Japanese study including 441 women aged 20 to 35 years, they found that increased vitamin E intake was associated with greater total spine BMD [41]. Farrell V. A. *et al.* showed that dietary vitamin E intake did not have any similar BMD association [42].

Folate, vitamin B2 (riboflavin), and vitamin B12 may affect bone directly or through an effect on plasma homocysteine levels [43]. In the current study, decreased intake of folate was associated with high BMD. In a study of Rejnmark L. *et al.*, they found that high dietary intake of folate exerts positive effects on BMD [31]. Also, Rivas A. *et al.* showed that the BMD was signifycantly associated with the intake of folate [44].

There was no association between BMD and the other micronutrients.

Our study is limited by its cross-sectional design, sample size and non-controlled design, but identification of relationship between nutrition and bone status it so important and especially in children with rheumatoid arthritis like juvenile idiopathic arthritis. Thus, more research on the role of diet on bone health is required. In addition, more emphasis should be placed on understanding the role of diet and nutrition on bone health during childhood and adolescence.

5. Conclusion

This study showed that in children with JIA, adequate dietary intake of proteins and magnesium can have a beneficial effect on the bone mass; low dietary intake of vitamin C, vitamin E and folate exerts positive effects on BMD; but further studies are needed to confirm this association.

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