MARKERS OF BONE METABOLISM IN PROTEIN ENERGY MALNUTRITION

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Abstract: This study was designed to assess the effect of protein energy malnutrition (PEM) disease on bone mineralisation. About 20 patients with PEM were enrolled and compared to a cohort of 14 healthy infants. Besides the dietetic history taking, anthropometric measurements and usual laboratory investigations, bone age estimation and measurement of serum parathyroid and osteocalcin levels were done for patients and controls. Nutritional rehabilitation was supervised for 2–4 months, and the patients were reassessed by the previous clinical, radiological and laboratory parameters. The current study demonstrated decrease in bone age and mineralisation in PEM patients with significant increase in parathyroid hormone and significant decrease in osteocalcin level, both being related to the severity of the condition, yet the bone resorption was more significant than the bone formation. Fortunately, these changes were reversible upon nutritional rehabilitation further emphasising the role of prompt and proper implication of the nutritional rehabilitation programme in PEM patients.

Keywords: bone; metabolism; osteocalcin; PTH; parathyroid hormone; PEM; protein energy malnutrition; nutritional rehabilitation.

INTRODUCTION

Protein energy malnutrition (PEM) is the most common nutritional disorder affecting children in developing countries and the third most common disease of childhood in such countries (Akuyam, 2007). It is manifested primarily by inadequate dietary intake of protein and energy. However, PEM is almost always accompanied by deficiencies of other nutrients. For this reason,

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the term severe childhood undernutrition (SCU), which more accurately describes the condition, is preferred (Heird, 2008).

Bone mineralisation is a complex process that requires adequate nutrition; protein for osteoid formation; calcium and phosphorus for calcification; weight bearing and muscle use; and modulation by thyroid, parathyroid, gonadal and pituitary hormones (Southard et al., 1991). In the year 1992, Branca et al. reported depressed bone turnover in PEM patients compared to recovered cases with no significant differences between the studied subtypes of PEM. In addition, De Schepper et al. (1991) and Bachrach (1996) enforced that nutritional as well as genetic and hormonal factors play an important role in the mineralisation of the bones during childhood and adolescence.

A key factor in the control of bone remodelling is parathyroid hormone (PTH), the principal regulator of calcium homeostasis (Poole and Reeve, 2005). Osteocalcin is synthesised by osteoblasts and its serum concentrations are believed to reflect bone formation (Loughead et al., 1990).

In the current study, we assessed the hormonal markers that reflect bone metabolism as well as the bone age in patients with PEM, aiming to identify the effect of PEM disease process with both its subtypes on bone mineralisation.

PATIENTS AND METHODS

Subjects

This study was conducted on 20 patients recruited from the inpatient department and the nutrition clinic at the Children's hospital, Ain Shams University. Enrolled cases were classified as PEM or rather SCU according to Heird (2008). Patients were further classified into two groups, edematous and non-edematous, on the basis of Z-score of weight for length and presence or absence of edema (Gernaat and Voorhoeve, 2000). The patients were 9 male and 11 female with a mean age of 11.60 ± 4.91 months. They were free form any chronic illness and were not on any medication that is likely to affect their bone metabolism.

The patients were compared to 14 clinically healthy; age and sex matched controls whose anthropometric measurements were within the normal percentiles according to Ogden et al. (2002). Those controls were recruited among those attending the hospital for minor surgical procedures and none of them was on any medication that would likely affect the bone density. They were 6 male and 8 female and their mean age was 11.57 ± 3.92 months.

Study design

After obtaining the approval of the ethical committee at Ain Shams University Children's hospital for human subject involvement, an informed consent was obtained from the parents or the care givers. The enrolled children were then subjected to detailed history taking with special emphasis on the socioeconomic standard of the family according to Park and Park (1979) and the dietetic history taking with a 24 recall of feeding.

Thorough clinical examination was done for each enrolled case with special emphasis on the anthropometric measurements. Plain X-ray was performed on the left wrist for all children to determine bone age using the Gruleich scale. Laboratory investigations were done including estimation of serum osteocalcin and PTH.

Anthropometric measurements

- Weight was estimated by an electronic scale with minimal clothing.
- Length was measured to the nearest 0.1 cm with portable measuring board.
- Skull circumference.
- Mid arm circumference.
- z score was calculated according to Gernaat and Voorhoeve (2000).
- Weight, height and skull circumference were plotted against the percentiles to obtain the percent from the median for age according to Ogden et al. (2002).

Blood collection

About 5 ml of venous blood samples were collected from each child under complete aseptic conditions. Blood was divided into two tubes one on EDTA for complete blood picture and the second was left to clot. Serum from the second tube was separated by centrifugation at 2,500 rpm for 5 min and was stored in three airtight capped containers at –20°C for estimation of the other laboratory parameters.

Biochemical analyses

1 Complete blood picture using the Coulter T660, Miami, USA.

2 Serum albumin using autoanalyser, synchron Cx4/Cx5 (Beckman, USA).

3 Serum Osteocalcin level was assessed by solid phase Enzyme Amplified Sensitivity Immunoassay using the kit supplied by BioSource Europe SA (Nivelles, Belgium) according to Power and Fottrell (1991).

4 Serum PTH level was assessed by solid phase Enzyme Amplified Sensitivity Immunoassay using the kit supplied by BioSource Europe SA (Nivelles, Belgium) according to Bouillon et al. (1990).

Nutritional rehabilitation

Nutritional rehabilitation of PEM patients was done according to the WHO (1999). Initially, management of life threatening and emergency conditions was done in the first week then the start of feeding was supervised. Caloric intake was 80–100 Kcal kg day^{-1} with the continuity of breast-feeding in cases of breast fed infants. The diet given was low in protein, fat and sodium, high in carbohydrates as almost all severely malnourished infants have infections, impaired liver and intestinal functions and problems related to electrolyte imbalance.

Rehabilitation stage followed with the return of the infant's appetite. The caloric intake increased to 150–200 Kcal Kg day^{-1} with increase in the amounts and decrease in the frequency. High protein diet was given and vitamins and minerals (potassium, magnesium and zinc) were continued in increased amounts. Iron was given during this stage to treat the anaemia present. The infant remained in the hospital for the first part of this rehabilitation phase (at least three weeks after admission), and then followed up in the nutritional rehabilitation outpatient clinic.

Follow up

Patients were reevaluated after 2–4 months of the nutritional rehabilitation programme using the previously mentioned clinical and laboratory parameters.

Statistical analysis

Standard computer program SPSS for Windows, release 10.0 (SPSS Inc, USA) was used for data entry and analysis.

All numeric variables were expressed as mean ± standard deviation (SD) for normally distributed variables and as median (interquartile range) for non-parametric variables. Comparison of different variables in various groups was done using Student's *t*-test (*t*) and Mann–Whitney test (*z*) for normal and non-parametric variables, respectively. Comparison of different variables before and after nutritional rehabilitation was done using paired *t*-test (*t*) and Wilcoxon matched pairs test (*z*) for normal and non-parametric variables, respectively. Pearson's correlation test was used for parametric data, and Spearman's correlation test was used for correlating non-parametric variables. For all tests a probability (*p*) less than 0.05 was considered significant. Graphic presentation of the results was also done (Daniel, 1995).

Table I Comparison between anthropometric measurements of protein energy malnutrition patients before nutritional rehabilitation and that of controls

Group / Studied parameter	Non-edematous Group (I) (n = 10) Mean ± SD [Median (IQR)]	Edematous Group (II) (n = 10) Mean ± SD [Median (IQR)]	Control Group (III) (n = 14) Mean ± SD [Median (IQR)]	Group I Vs. III t/z* (p)	Group II Vs. III t/z* (p)	Group I Vs. II t/z* (p)
z score	–1.09 ± 1.30	–1.95 ± 1.52	1.08 ± 0.98	– 4.67	– 3.81	– 0.36
	[–1.50 (2.00)]	[–0.75 (2.00)]	[1.29 (2.00)]	(p < 0.001)	(p < 0.001)	(p > 0.05)
Weight	4.79 ± 0.96	6.43 ± 2.80	9.07 ± 1.58	– 3.88*	– 2.24*	– 0.91*
	[4.60 (1.30)]	[4.80 (4.7)]	[9.50 (2.00)]	(p < 0.001)	(p < 0.001)	(p > 0.05)
Weight % from mean for age	50.18 ± 3.28	68.30 ± 25.20	94.59 ± 6.41	– 4.11*	– 2.47*	– 1.06*
	[50.00 (4.70)]	[66.60 (43.4)]	[94.70 (9.10)]	(p < 0.001)	(p < 0.01)	(p > 0.05)
Length	59.50 ± 2.36	65.00 ± 7.83	69.29 ± 4.43	– 3.10*	– 1.18*	– 1.37*
	[60.00 (4.00)]	[64.00 (16)]	[70.00 (8.00)]	(p < 0.001)	(p > 0.05)	(p > 0.05)
Length % from mean for age	80.96 ± 5.73	89.86 ± 7.15	94.74 ± 1.75	– 3.10*	– 1.18*	– 2.58*
	[80.50 (10.00)]	[89.00 (13.00)]	[94.50 (3.00)]	(p < 0.001)	(p > 0.05)	(p < 0.01)
Skull circumference	41.00 ± 2.43	41.60 ± 2.37	44.36 ± 1.90	– 2.95*	– 2.72*	– 0.30*
	[41.00 (3.30)]	[42.00 (5.00)]	[44.50 (3.00)]	(p < 0.01)	(p < 0.01)	(p > 0.05)
Skull circumference % from mean for age	90.94 ± 2.01	92.62 ± 3.09	98.20 ± 2.04	– 8.66	– 5.35	– 1.44
	[90.50 (3.60)]	[92.40 (5.50)]	[97.00 (3.60)]	(p < 0.001)	(p < 0.001)	(p > 0.05)
Mid arm circumference	8.94 ± 1.45	9.90 ± 1.58	11.47 ± 1.10	– 4.87	– 2.89	– 1.42
	[9.50 (1.90)]	[9.50 (2.50)]	[11.50 (1.50)]	(p < 0.001)	(p < 0.01)	(p > 0.05)

*Non-parametric data detected by Shapiro–Wilk test and presented as median (interquartile range).
The test of significance used here is Mann–Whitney test.
$p < 0.05$ is significant, $p < 0.01$ is highly significant, $p < 0.001$ is very highly significant and
$p > 0.05$ is non-significant.

RESULTS

The results of the present study reveal lower anthropometric measurements in non-edematous as well as edematous PEM patients compared to those of the controls (Table 1). These measurements show significant improvement after nutritional rehabilitation (yet not reaching the control values in most of them) (Table 2).

Tables 3 and 4 show that both serum albumin and haemoglobin levels are significantly lower in both groups of PEM compared to the controls, and their values show significant improvement after nutritional rehabilitation yet not reaching the control values. In addition, TLC and alkaline phosphatase values are significantly higher in both groups of PEM patients compared to the controls and decrease after nutritional rehabilitation. Regarding serum calcium, it is only significantly lower in edematous group with significant improvement after nutritional rehabilitation. On the other hand, serum phosphorus does not differ from controls neither before nor after nutritional rehabilitation in both studied groups.

Figure 1 demonstrates that serum osteocalcin levels are significantly lower in both groups of PEM patients in comparison to the controls with no differences on comparing the two groups together. After nutritional

Table 2 Comparison between anthropometric measurements of protein energy malnutrition patients before and after nutritional rehabilitation

Group / Studied parameter	Non-edematous patients (n = 10) Mean ± SD [Median (IQR)]			Edematous patients (n = 10) Mean ± SD [Median (IQR)]		
	Before	After	t/z* (p)	Before	After	t/z* (p)
z score	–1.09 ± 1.30 [–1.50 (2.00)]	–1.18 ± 0.67 [0.88 (1.26)]	– 6.09 (p < 0.001)	–1.95 ± 1.52 [-0.75 (2)]	1.61 ± 1.46 [1.25 (2.55)]	– 3.79 (p < 0.01)
Weight	4.79 ± 0.96 [4.60 (1.30)]	7.20 ± 0.5 [7.00 (0.90)]	– 2.81* (p < 0.01)	6.43 ± 2.80 [4.80 (4.70)]	8.84 ± 2.12 [8.50 (3.30)]	– 2.83* (p < 0. 01)
Weight % from mean for age	50.18 ± 3.28 [50.00 (4.70)]	70.02 ± 6.20 [70.00 (6.80)]	– 2.81* (p < 0.01)	68.30 ± 25.20 [66.60 (43.40)]	83.44 ± 11.32 [79.70 (16.10)]	– 2.51* (p < 0.01)
Length	59.50 ± 2.36 [60.00 (4.00)]	62.94 ± 2.08 [63.00 (3.00)]	– 2.83* (p < 0.01)	65.00 ± 7.83 [64.00 (16.00)]	67.80 ± 7.41 [67.00 (15.00)]	– 2.83* (p < 0.01)
Length % from mean for age	80.96 ± 5.73 [80.50 (10.00)]	81.38 ± 5.01 [83.80 (7.30)]	– 0.27* (p>0.05)	87.22 ± 5.27 [88.50 (10.10)]	89.86 ± 7.15 [89.00 (13.00)]	– 2.81* (p<0.01)
Skull circumference	41.00 ± 2.43 [41.00 (3.30)]	43.32 ± 1.62 [43.50 (2.30)]	– 2.83* (p < 0.01)	41.60 ± 2.37 [42.00 (5.00)]	43.90 ± 2.55 [45.00 (5.10)]	– 2.87* (p < 0.01)
Skull circumference% from mean for age	90.94 ± 2.01 [90.50 (3.60)]	93.86 ± 1.06 [93.60 (1.90)]	– 4.09 (p < 0.01)	92.62 ± 3.09 [92.40 (5.50)]	95.02 ± 3.16 [95.70 (5.00)]	– 5.18 (p < 0.01)
Mid arm circumference	8.94 ± 1.45 [9.50 (1.90)]	11.30 ± 1.14 [11.00 (2.00)]	– 3.39 (p < 0.01)	9.90 ± 1.58 [9.50 (2.50)]	12.24 ± 1.47 [12.50 (2.00)]	– 4.26 (p < 0.01)

*Non-parametric data are detected by *Shapiro–Wilk* test and presented as median (interquartile range). The test of significance used here is *Wilcoxon matched pairs test*.

$p < 0.01$ is highly significant, $p < 0.001$ is very highly significant and $p > 0.05$ is non-significant.

Table 3 Comparison between laboratory data of protein energy malnutrition patients before nutritional rehabilitation and that of controls

Group / Studied parameter	Non-edematous Group (I) (n = 10) Mean ± SD [Median (IQR)]	Edematous Group (II) (n = 10) Mean ± SD [Median (IQR)]	Control Group (III) (n = 14) Mean ± SD [Median (IQR)]	Group I Vs. III t/z* (p)	Group II Vs. III t/z* (p)	Group I Vs. II t/z* (p)
Total leucocytic count (cells mm^{-3})	12.86 ± 4.34 [11.70 (7.20)]	9.70 ± 3.07 [9.00 (5.50)]	7.03 ± 0.80 [7.00 (1.20)]	– 4.11* (p < 0.001)	– 2.23* (p < 0.05)	– 1.67* (p > 0.05)
Serum Hgb (gm dL^{-1})	8.74 ± 1.30 [8.80 (1.90)]	8.46 ± 1.09 [7.80 (2.00)]	10.61 ± 0.64 [10.70 (1.40)]	– 3.17* (p < 0.01)	– 3.64* (p < 0.001)	– 0.30* (p > 0.05)
Serum albumin (gm dL^{-1})	3.76 ± 0.32 [3.90 (0.40)]	2.28 ± 0.65 [2.40 (0.70)]	4.26 ± 0.36 [4.20 (1.10)]	– 2.83* (p < 0.01)	– 4.12* (p < 0.001)	– 3.81* (p < 0.001)
Calcium (mg dL^{-1})	9.76 ± 0.73 [9.50 (1.10)]	9.20 ± 0.24 [9.10 (0.40)]	9.76 ± 0.51 [9.80 (1.10)]	– 0.24* (p > 0.05)	– 2.85* (p < 0.01)	– 2.14* (p < 0.05)
Phosphorus (mg dL^{-1})	4.24 ± 1.01 [4.50 (1.60)]	4.34 ± 0.51 [4.20 (0.80)]	4.41 ± 0.86 [4.70 (1.60)]	– 0.46 (p > 0.05)	– 0.24* (p > 0.05)	– 0.28 (p > 0.05)
Alkaline phosphatase (IU L^{-1})	135.00 ± 16.22 [136.00 (23.00)]	168.80 ± 27.69 [170.00 (40.00)]	112.71 ± 23.21 [110.00 (29.00)]	2.61 (p < 0.05)	5.39 (p < 0.001)	– 3.33 (p < 0.05)

*Non-parametric data detected by Shapiro–Wilk test and presented as median (interquartile range). The test of significance used here is Mann–Whitney test.
$p < 0.05$ is significant, $p < 0.01$ is highly significant, $p < 0.001$ is very highly significant and $p > 0.05$ is non-significant.

Table 4 Comparison between laboratory data of both studied groups of protein energy malnutrition patients before and after nutritional rehabilitation

Group / Studied parameter	Non-edematous patients (n = 10) Mean ± SD [Median (IQR)]			Edematous patients (n = 10) Mean ± SD [Median (IQR)]		
	Before	After	t/z* (p)	Before	After	t/z* (p)
Total leucocytic count (cells mm-3)	12.86 ± 4.34 [11.70 (7.20)]	9.76 ± 1.16 [9.40 (2.30)]	– 2.10* (p < 0.05)	9.70 ± 3.07 [9.00 (5.50)]	9.24 ± 1.96 [10.20 (3.80)]	– 0.15* (p > 0.05)
Serum Hgb (gm dL-1)	8.74 ± 1.30 [8.80 (1.90)]	10.24 ± 0.54 [10.30 (0.80)]	– 2.81* (p < 0.01)	8.46 ± 1.09 [7.80 (2.00)]	9.88 ± 1.54 [9.30 (2.90)]	– 2.81* (p < 0.01)
Serum albumin (gm dL-1)	3.76 ± 0.32 [3.90 (0.40)]	4.22 ± 0.32 [4.30 (0.50)]	– 2.10* (p < 0.05)	2.28 ± 0.65 [2.40 (0.70)]	3.90 ± 0.41 [3.90 (0.70)]	– 2.83* (p < 0.01)
Calcium (mg dL-1)	9.76 ± 0.73 [9.50 (1.1)]	9.72 ± 0.45 [9.80 (0.80)]	0.00* (p > 0.05)	9.20 ± 0.24 [9.10 (0.40)]	10.14 ± 0.80 [10.50 (1.40)]	– 2.51* (p < 0.01)
Phosphorus (mg dL-1)	4.24 ± 1.01 [4.50 (1.60)]	4.74 ± 0.90 [4.30 (2.00)]	– 1.09 (p > 0.05)	4.34 ± 0.51 [4.20 (0.80)]	4.66 ± 0.97 [4.80 (1.00)]	– 1.03 (p > 0.05)
Alkaline phosphatase(IU L-1)	135.00 ± 16.22 [136.00 (23)]	121.40 ± 22.83 [115.00 (44.00)]	1.23 (p > 0.05)	168.80 ± 27.69 [170.00 (40.00)]	131.80 ± 21.81 [130.00 (32.00)]	4.31 (p < 0.01)

*Non-parametric data are detected by *Shapiro–Wilk test* and presented as median (interquartile range). The test of significance used here is *Wilcoxon matched pairs test*.
$p < 0.05$ is significant, $p < 0.01$ is highly significant and $p > 0.05$ is non-significant.

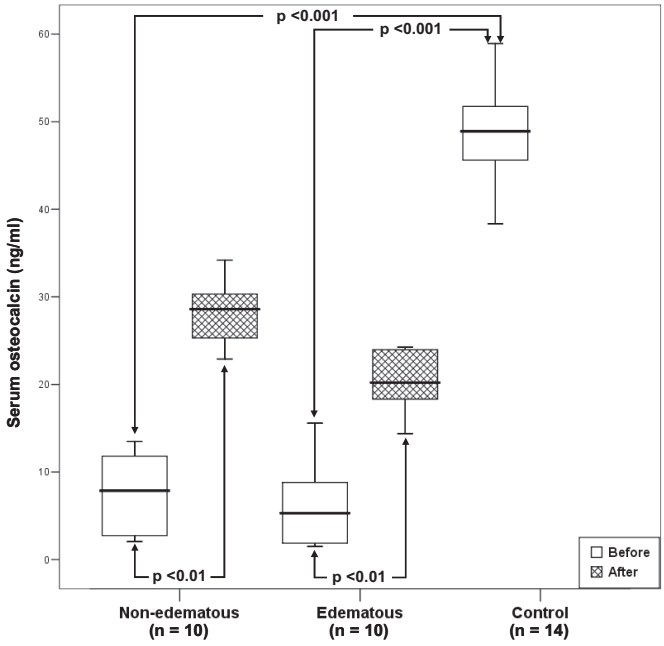

Figure 1 Serum Osteocalcin levels in edematous and non-edematous PEM patients before and after nutritional rehabilitation in comparison to the controls

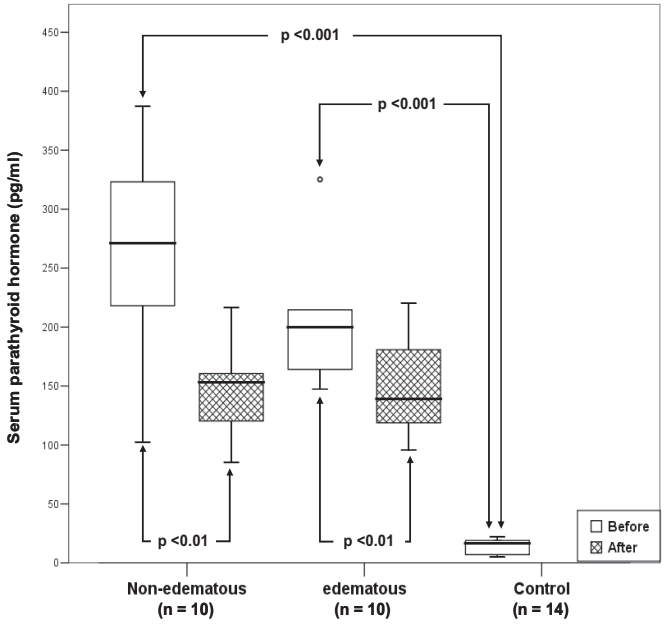

Figure 2 Serum parathyroid levels in edematous and non-edematous PEM patients before and after nutritional rehabilitation in comparison to the controls

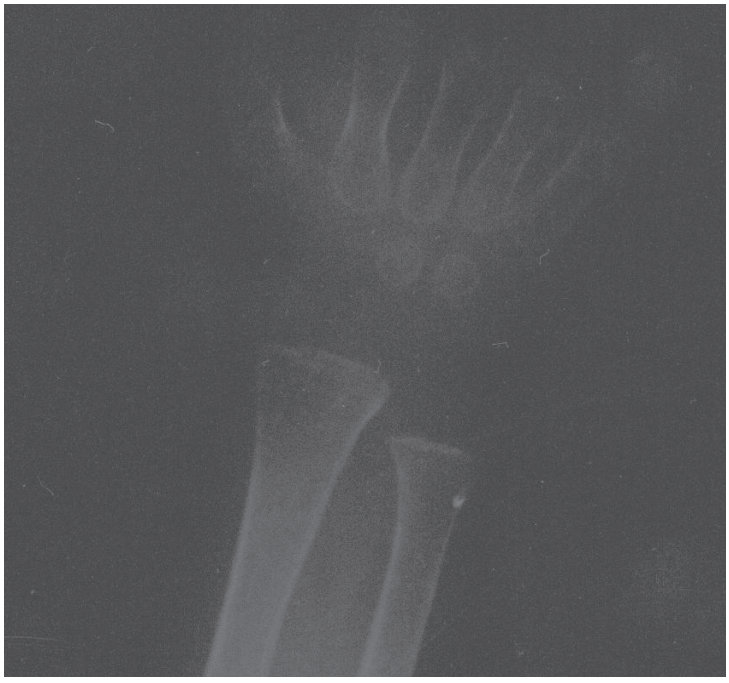

Figure 3 X-ray left wrist of a 24 months old PEM patient showing delayed bone age (15 months) with evidence of bone demineralisation, cortical thinning and early rickets

rehabilitation, these levels increase significantly in both studied groups, yet they are still significantly lower than the controls.

As regards PTH levels, they are represented in Figure 2. Serum parathyroid is significantly higher in both groups of PEM patients in comparison to the controls with no differences on comparing the two groups together. After nutritional rehabilitation, these levels decrease significantly in both studied groups, yet they are still significantly higher than the controls.

The correlation studies reveal significant negative correlation between PTH levels and the *z* score in all studied patients before nutritional rehabilitation (*r* value is -0.73 and *p* value is < 0.001). In addition, there is positive but non-significant correlation between serum osteocalcin levels and the *z* score in all studied patients before nutritional rehabilitation (*r* value is 0.07 and *p* value is >0.05).

Bone age determination for PEM patients on admission reveal delayed bone age in 80% of the patients with evidence of bone demineralisation, cortical thinning, evidence of early rickets in 10% and healed rickets in 20%. Figure 3 demonstrates X-ray of the left wrist of a 24 months old PEM patient showing delayed bone age (15 months) with evidence of bone demineralisation, cortical thinning and early rickets.

DISCUSSION

The results of the current study revealed statistically lower values of the studied anthropometric measurements in both studied groups of PEM initially in comparison

to the controls with significant improvement after nutritional rehabilitation. The decreased initial values of weight and length agree with Wellcome's classification of PEM (Wellcome, 1970) and are fulfilling one of the constant features of malnutrition, namely, growth retardation in weight and height (Jelliffe and Jelliffe, 1992). The lower skull circumference measurement agrees with Needlman (2001) who stated that severe undernutrition depresses head growth, and this is considered as ominous predictor of later cognitive disability. As regards the low measures of mid arm circumference, it is reinforced by Castiglia (1996) who stated that mid arm circumference reflects the state of muscle and subcutaneous fat, so it is affected in PEM especially the non-edematous.

Increased leucocytic count in both studied groups of PEM initially in comparison to the controls could be attributed to the high incidence of infections found in malnourished patients as reported by Chandra (1997). The initially detected low haemoglobin levels in both studied groups come in agreement with el-Nawawy et al. (2002) and Borelli et al. (2007) who reported anaemia in PEM and suggested that it is a result of ineffective erythropoiesis. In addition, the initial decrease in albumin in our series of PEM patients agrees with Rahman and Begum (2005).

Calcium and phosphorus levels were within the normal reference ranges in non-edematous and edematous groups of PEM patients (Pesce, 2008). Although the initial value of alkaline phosphatase was statistically higher in both studied groups compared to the controls, this enzyme was within normal range for age and sex according to Pesce (2008). Similarly, Adejuwon et al. (1994) previously reported normal alkaline phosphatase in edematous PEM. On the other hand, Kumari et al. (1993) reported decreased levels of this enzyme in PEM patients.

Worth noting here is that the nutritional rehabilitation programme also caused significant improvement in the previously mentioned laboratory parameters further proving the success of its implementation.

Regarding serum osteocalcin levels, the current study revealed significantly lower levels in both groups of PEM patients in comparison to the controls. This finding agrees with Prudhon et al. (1991) who studied osteocalcin in kwashiorkor and suggested that serum osteocalcin levels might be related to protein energy status. In addition, our results are in accordance with Ndiaye et al. (1995) who reported decreased osteocalcin levels in both marasmus and kwashiorkor patients denoting affection of bone formation in such patients. The decreased bone age and the radiological evidence of bone demineralisation as well as cortical thinning detected in 80% of our studied PEM patients further supports this point. Worth noting here is that this percentage dropped to 50% when we excluded the patients that had rachitic manifestations. Soliman et al. (2006) similarly reported demineralisation and cortical thinning without metaphyseal changes in 40% of their series of PEM patients who had no rachitic manifestations.

After nutritional rehabilitation, the osteocalcin levels showed significant increase in the studied PEM patients whether edematous or non-edematous. This improvement is exactly consistent with Prudhon et al. (1991) who also reported a fourfold increase in serum osteocalcin after rehabilitation of the malnourished patients.

The results of the current study showed that PTH levels were significantly higher in both groups of PEM patients in comparison

to the controls with no differences on comparing the two groups together. After nutritional rehabilitation, these levels decreased significantly in both studied groups. Although serum calcium levels were within normal range in our series of PEM patients, the elevated serum PTH can still be related to their calcium level based on the phenomenon of hysteresis. This phenomenon is defined as a different PTH value for the same serum calcium concentration during the induction of and recovery from hypo- and hypercalcaemia (Felsenfeld et al., 2007).

The correlation studies reveal significant negative correlation between the initial PTH levels and the *z* score in all studied patients denoting that the rise in this hormone is dependent on the severity of PEM. In addition, there is positive but non-significant correlation between the initial serum osteocalcin levels and the *z* score, which also proves that the decrease in osteocalcin is related to the severity of PEM but to a less degree.

In conclusion, the current study demonstrated decrease in bone age and mineralisation in PEM patients whether edematous or non-edematous with significant increase in PTH and significant decrease in osteocalcin level, both being related to the severity of the condition, yet the bone resorption is more significant than the bone formation. Fortunately, these changes seem to be reversible upon nutritional rehabilitation further emphasising the role of prompt and proper implication of the nutritional rehabilitation programme in PEM patients. Nevertheless, we need larger scale studies with long-term follow up to make sure that there is no long-term residual affection of bone density due to the PEM process since the results of the study did not reach the control values at the reevaluation.

BIOGRAPHY

May Fouad Nassar is a Professor of Pediatrics at the Faculty of Medicine, Ain Shams University. In the field of community service and development, she played a central role in many governmental projects aiming at welfare of Egyptian children. She is specialised in clinical nutrition with following up of clinical cases as well as teaching medical students, nurses, house officers and residents. Most recently, she has achieved international reputation and acknowledgment through several distinguished publications and has been awarded the Nation's prize for young researchers in 2006 and Ain Shams University prize for best international publication in 2007 and 2008.

Dina Ahmed Amin is a Senior Lecturer of Pediatrics Ain Shams University, Cairo, Egypt. She is concerned with managing and following nutritional and neurological cases in Ain Shams University Hospitals. She is a Member of the Egyptian Medical Syndicate, the Egyptian Society of Child Neuro-Psychiatry (ESCNP) and the Gaucher Committee, project HOPE Egypt. She fulfilled the Lecturers training course in Ain Shams University 2006. She is involved in giving tutorials to residents, house officers, undergraduates and postgraduates, also participating in preparing clinical, oral exams and OSCE for under and postgraduates. Her publications focus on nutritional and neurological diseases.

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