

# Treatment of Severely Malnourished Children Hospitalized According to Results the Renal Metabolic Study

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## Abstract

Metabolic acidosis is common in severe malnourished pediatric patients.

**Objective:** The objective of this study was to treat severe malnourished children according to results of renal metabolic study.

**Method:** The study was prospective, clinical trial (therapeutic). Overload test with sodium bicarbonate at 5% was performed to 30 severe malnutrition: marasmus, kwashiorkor or mixed, from 6 months to 5 years, primary etiology, hemodynamically stable, metabolic acidosis, hyperchloremia and anion gap urinary positive, which in turn they divided into two groups: 15 were treated according to the results of renal metabolic study (study group) and 15 receiving usual care (control group). The calcium / creatinine and uric / creatinine ratio was determined. Test of statistical significance were used: Student t Test, chi square Test or Fisher exact probability. Z Test the percentage difference. Cramer's V Test. Statistical significance ( $p < 0.05$ ).

**Results and Discussion:** In 12 (40.0%) patients distal renal tubular acidosis was observed. There was a significant difference ( $p < 0.013$ ) between the dry weight and weight at discharge in both the study and control group. There was a significant difference ( $p < 0.05$ ) between the mean value of serum albumin entry and exit in the study group and not in the control group. It is important to treat metabolic acidosis in severe malnutrition to clinical improvement (weight) and biochemical outcomes (serum albumin), breaking the vicious circle in which these patients are frequently involved.

## Introduction

Renal Tubular Acidosis (RTA) includes a group of clinical entities in which hyperchloremic metabolic acidosis occurs, that is with the normal serum anion gap. They are characterized by alterations of bicarbonate reabsorption at the proximal tubule of the nephron (ATR proximal or type 2) or defect secretion of hydrogen ions at the distal tubules of the nephron (distal ATR or type 1) and hyperkalemic ATR (or type 4). Previously, the category of ATR type 3 was used to define children with type 1 distal ATR, who at breastfeeding age also have proximal loss of bicarbonate in the urine; however, because this loss is temporary, it has been eliminated this category [1,2].

Malnourished patients can have a pH and normal bicarbonate concentration, but a load of acids can lead to metabolic acidosis faster than a patient eutrophic.

Malnutrition decreased ability for excreting acids, even for presence of hypokalemia which is usually accompanied by an increase in the acid excretion [3].

Low protein diets are associated with decreased ammonia production and activity of glutamate dehydrogenase in the presence of an overload acid [4].

The activity of  $\text{Na}^+ / \text{H}^+$  level of the renal tubules is decreased for experimental animals with low protein diets [5], the decreased activity of the sodium-potassium pump ATPase observed in malnutrition may explain declining activity of the  $\text{Na}^+ / \text{H}^+$ .

It has been suggested that protons pump might play a role on decreased ability excreting an acids load of malnourished patient, however its activity has not been measured for malnutrition.

Therefore it is believed that the reduced ability to eliminate a load of acids in malnutrition is due to several factors: proximal tubule level decrease  $\text{Na}^+ / \text{H}^+$ , at the distal tubule decreased activity Protons pump and titratable acidity decreased as a result of hipofosfatúria.

Study de 85 hospitalized severely malnourished patients have reported metabolic acidosis in 35.29%; when testing overload with bicarbonate 5% in 30 patients was evident in 12 of them (40%) a distal renal tubular acidosis [6], which confirms the findings by other researchers [3,5].

Metabolic acidosis might cause side effects such as growth retardation, increased muscle breakdown with muscle wasting, reduced albumin synthesis predisposed to hypoalbuminemia, insulin resistance and inflammation stimulation [7].

In children with proximal renal tubular acidosis and normal renal function, bone histomorphometric studies revealed low bone formation and mineralization [8]. Other studies in epileptic children treated with ketogenic diets have also reported decreased bone mineral density [9].

There is alteration of longitudinal growth in the presence of metabolic acidosis [10]. Both cartilage production and bone formation are decreased resulting in short stature. The effect of metabolic acidosis on growth can be due to decreased secretion of growth hormone or its effects on peripheral tissues [11]. Low serum concentrations of insulin-like growth factor 1 (IGF-1) and low hepatic levels of growth hormone receptor and IGF-1 receptor mRNAs have been reported in rats with metabolic acidosis [12].

Experimentally induced metabolic acidosis in normal human for at least seven days produces serum albumin reduction [13,14]. Reduction of protein synthesis, increased protein breakdown and impaired oxidation of amino acids have been suggested as factors contributing to a decrease of serum albumin by presence of metabolic acidosis [15-17].

Rat studies have shown that the metabolic acidosis is associated with impaired glucose tolerance and insulin resistance [18,19].

For above reasons it was decided to perform a study of treatment of hospitalized severe malnourished patients according to the results of a study about renal tubular function.

## Method

It is a prospective research, clinical trial (therapeutic), whose purpose was to treat thirty severely malnourished pediatrics patients according results a study of renal tubular function, through the load test with sodium bicarbonate 5 %, hospitalized at the Pediatric Department attached to the Hospital "Dr. Enrique Tejera", located at Valencia, Venezuela. Over the study period, eighty five severe malnourished children were evaluated, thirty of them who met the inclusion criteria of hyperchloremic metabolic acidosis and anion gap urinary positive, were split into two groups of fifteen after being randomized. One group was treated according to the results of renal metabolic study (case group) and the other was given usual treatment of the Gastroenterology and Nutrition (control group).

### Inclusion criteria

Severe malnourished patients between 6 months and 5 years old with severe malnutrition primary etiology of both sexes, hemodynamically stable (without diarrhea or signs of dehydration). All clinical forms of severe malnutrition (Marasmus, Kwashiorkor mixed) and malnourished patients with severe metabolic acidosis, hyperchloremia and positive urinary anion gap were included; blood  $\text{pH} \leq 7.35$ , serum bicarbonate  $\leq 22 \text{ mEq/L}$ , serum chloride  $\geq 108 \text{ mEq/L}$  and positive urinary anion gap (sodium + potassium - chlorine).

### Exclusion criteria

Secondary severe malnourisheds patients, infectious,

cardiovascular disease, neurological, neoplasia, cystic fibrosis. Denial of informed parental consent. Bacteriuria in urinalysis.

### Collection of information

For information collect, was developed a protocol in which the following variables were included:

**Age:** chronological age in years and months (EC) given as a range for each year of age, between zero and eleven months was used.

**Nutritional diagnosis:** Definition of serious malnutrition in patients without edema was based on the indicator weight for height expressed in z value (Normalized Standard Deviation). The reference is the study of the National Center for Health Statistics (NCHS), recommended by the World Health Organization (WHO) [20] using as cutoff -3 SD. In patients with any degree of edema the anthropometry was performed when melted or disappear edema (dry weight). The type of severe malnutrition (marasmus, kwashiorkor or mixed) was determined by the score of McLaren, et al. [21]. Severe malnutrition is classified as acute if growth in size it was normal, or chronic when it was down. The reference height / age indicator comes from Fundacredesa Charts, Proyecto Venezuela, 1993. The cutoff points used were 3 and 97 percentile [22].

### Renal metabolic study

Each patient metabolically stable (after missing diarrhea, dehydration, edema, with improvement of the infectious process) was taken 6 mL venous blood sample without anticoagulant to obtain serum and perform biochemical determinations (urea, creatinine, calcium, phosphorus, uric acid, total and fractional protein). Arterial blood sample (1 mL) heparinized was taken for determination arterial blood gases and serum electrolytes. Urine sample was obtained for urinalysis and urinary electrolytes.

### Overload test bicarbonate

It was performed the test overload bicarbonate according to Edelman modified technique [23] to patients with metabolic acidosis of renal origin (hyperchloremia and positive urinary anion gap). Arterial blood gas analysis was done on the morning of the test overload. Dose of sodium bicarbonate at 5% was calculated for correction of metabolic acidosis by the formula (ideal-actual bicarbonate)  $\times$  weight  $\times$  0.6. The figure of 22 mEq / L bicarbonate was used as an ideal. The dose of sodium bicarbonate to the overload was 3 mEq / kg body weight. It was administered intravenously diluted with the same amount of 5% glucose solution. The total infusion time was two hours. When after loading it proceeded to take samples of arterial blood, venous and urine. The first were taken without applying tourniquet preferably in the radial or brachial artery with previously heparinized syringes. Then extraction undocked the syringe sealing the end to ensure anaerobiosis. Venous withdrawals were made in the veins of the elbow crease with minimal compression tourniquet, preserving them in tightly capped tubes. Urine samples were collected by spontaneous voiding or bladder catheterization and transported under refrigeration to the laboratory, ensuring that for urinary gases kept their anaerobic conditions. Once the samples of arterial blood, venous blood and urine were taken, venous blood and urine gases, serum electrolytes and urine, serum creatinine and urine were determined.

## Index calculation

With the results we proceeded to calculate the indices: fractional excretions bicarbonate, sodium and potassium by the formulas:

$$\frac{O/PHCO_3}{O/PCr} \times 100; \quad \frac{O/PNa}{O/PCr} \times 100; \quad \frac{O/PK}{O/PCr} \times 100;$$

O represents the calculated concentration of the substance in urine; P represents the concentration in plasma; and Cr, creatinine concentration. It was considered as a normal fractional excretion of bicarbonate <15%, otherwise it is classified as a bearer of proximal renal tubular acidosis (ATRP).

The fractional excretion of sodium 3% and fractional excretion of potassium 20% was considered normal. PCO<sub>2</sub> difference between urine (U) and blood (B): (UB) PCO<sub>2</sub>, after loading, was considered normal > 20 mmHg; contrary result was considered as a carrier of Distal Renal Tubular Acidosis (DRTA).

To evaluate hypercalciuria calcium / creatinine ratio was used. Patients over two years with values 0.20 were considered positive, and those patients under two years of age with values 0.30 were considered positive [24]. To evaluate hyperuricosuria uric / creatinine ratio acid was used considering positive value 0.50 [25].

## Treatment of patients according to the metabolic study Renal

Those patients in afterload presented fractional excretion sodium bicarbonate >15% were classified as proximal renal tubular acidosis and treatment was with sodium bicarbonate at a dose of 5 mEq / kg / day (divided into three doses). Patients in afterload had a difference of PCO<sub>2</sub> urine-plasma (<20 mm Hg) were listed as distal tubular acidosis and treated with citrate K at doses of 2 mEq / kg / day orally in three doses.

**Table 1:** Distribution of patients according to the characteristics: age, gender, growth in size and clinical form.

CHARACTERISTICS	GROUPS	
	TREATED BY METABOLIC STUDY F (%) <sup>*</sup>	CONVENTIONAL TREATMENT F (%) <sup>**</sup>
<b>AGE MONTHS</b>		
1 - 11	6 (40.0)	8 (53.2)
12 - 23	7 (46.7)	3 (20.0)
24 - 48	2 (13.3)	2 (13.3)
more de 48	0 (0.0)	2 (13.3)
<b>GENDER</b>		
Female	8 (53.3)	9 (60.0)
Male	7 (46.7)	6 (40.0)
<b>GROWTH IN SIZE</b> (Indicator T/E)		
Percentile T/E ≤ 3	15 (100)	15 (100)
Percentile T/E > 3	0 (0.0)	0 (0.0)
<b>CLINIC FORM</b>		
Kwashiorkor	6 (40.0)	10 (66.7)
Mixed	4 (26.7)	2 (13.3)
Marasmus	5 (33.3)	3 (20.0)

<sup>\*</sup>Percentages based on 15 patients

<sup>\*\*</sup>Percentages based on 15 patients

Natriuresis patients treated with prostaglandin inhibitors (Indocid) 1mg / kg / day. Hypercalciuria patients treated with diuretics (Dihydroclorotiazida) at doses of 2 mgs / /kg/day.

## Conventional treatment (Gastroenterology and Nutrition, Department of Pediatrics):

Feeding soy formula sucrose free was started; the water requirements were 80 cc / kg in kwashiorkor and 100 cc / kg in the marasmus. Caloric requirements were 80 calories / kg in patients with kwashiorkor and 100 calories / kg in the marasmus. Once the diarrhea disappeared, concentrate chicken at lunch and dinner was introduced according to age, then cornmeal muffin with cheese, butter at breakfast and finally was given compotes of antidiarrheal fruits; as the increased calorie and protein requirements dextrin-maltose, corn oil and protein module was used.

## Statistical Analysis

Data were analyzed using SPSS number 19. Statistical significance tests were applied to analysis of mean differences for independent samples (t Student), chi-square (Goodness of Fit Test) and the Freeman-Halton extension of Fisher Exact Test, and the Z Test of percentage difference based on absence zero and also Cramer's V test for categorized variables. The criterion of error of less than 5% (p<0.05) was used for analysis of statistical significance.

## Results

When evaluating 30 patients with metabolic acidosis that resulted, hyperchloremia and positive urinary anion gap was observed:

In the group of 15 patients who were subsequently treated according to test results overload sodium bicarbonate 5% (case group) age that predominated was between 1 and 23 months, 86.7% (n = 13) followed by those between 24 and 48 months 13.3% (n = 2). The female had a slight predominance 53.3% (n = 8) on the male 46.7% (n = 7). All patients had an increase in size < 3 percentile. In 66.7% (n = 10) the kwashiorkor and mixed clinical forms was observed. The marasmus in 33.3% (n = 5) (Table 1).

In the group of 15 patients who were subsequently treated according to usual protocol Nutrition Service (control group), the age that predominated was between 1 and 23 months, 73.2% (n = 11), with similar distribution to groups between 24 and 48 months 13.3% (n = 2) and 48 months 13.3% (n = 2). Female gender predominated 60% (n = 9) on the male 40% (n = 6). All patients had an increase in size the < 3 percentile. In 80% (n = 12) the kwashiorkor and mixed clinical forms was observed. The marasmus in 20% (n = 3) (Table 1).

A significant association between age group and type of treatment as a result of the V Cramer was not found, as it was of 0.360 for a probability of more than 5% (P>0.05).

A significant association between gender distribution of patients by type of treatment was not found; with Chi<sup>2</sup>= 0; GL=1; P=1.00 (P>0.05).

A significant association between forms of severe malnutrition and groups according to type of treatment according to the V Cramer was not found because it was 0.268; P = 0.33 (P >0.05).

Since no statistically significant differences for the studied characteristics among groups that were subsequently treated and

**Table 2:** Mean values (x) and standard deviation (s) of arterial blood gases, Serum electrolytes and urinary electrolytes.

PARAMETERS	GROUPS	
	TREATED BY METABOLIC STUDY* X ± S	CONVENTIONAL TREATMENT** X ± S
<b>ARTERIAL BLOOD GASES</b>		
pH sanguine	7.4 ± 0.09	7.3 ± 0.06
HCO <sub>3</sub>	19.1 ± 2.4	19.7 ± 2.6
PO <sub>2</sub>	107.2 ± 30.3	110.9 ± 28.5
PCO <sub>2</sub>	26.0 ± 11.1	27.0 ± 6.4
<b>SERUM ELECTROLYTES</b>		
Na <sup>+</sup>	134.2 ± 4.8	137.8 ± 4.6
K <sup>+</sup>	4.5 ± 0.7	4.1 ± 0.6
Cl <sup>-</sup>	109.0 ± 3.2	107.0 ± 3.0
<b>URINARY ELECTROLYTES</b>		
Na <sup>+</sup>	40.3 ± 26.2	84.4 ± 85.5
K <sup>+</sup>	20.4 ± 14.1	30.6 ± 19.7
Cl <sup>-</sup>	53.4 ± 33.4	87.5 ± 50.5

\*Values based on 15 patients treated according to metabolic study

\*\*Values based on 15 patients with conventional treatment

untreated according to the results of renal metabolic study (load test with bicarbonate 5%), evidence the similarity of the patients in both groups and therefore reliability differences subsequently found.

The average value of blood pH for patients who were subsequently treated according to test results overload with sodium bicarbonate 5%, was 7.4 with deviation of 0.09, while in the patients treated conventionally the average value was 7.3 with deviation of 0.06 (Table 2).

Mean value of HCO<sub>3</sub> of patients who were treated according to test results overload with sodium bicarbonate 5% was 19.1 with deviation of 2.4, whereas those treated conventionally it was 19.7 with deviation 2.6 (Table 2)

PO<sub>2</sub> average of the treated according to test results overload with sodium bicarbonate 5% was 107.2 with deviation of 30.3, while in the other group was 110.9 with deviation 28.5 (Table 2).

For PCO<sub>2</sub>, the mean in the group treated in accordance to test results overload with sodium bicarbonate 5% was 26.0 with deviation of 11.1, while in the group treated conventionally was 27.0 with deviation 6.4 (Table 2).

The Na<sup>+</sup> blood average for the case group was 134.2 with deviation of 4.8, whereas in the control group the mean was 137.8 with deviation of 4.6 (Table 2).

K<sup>+</sup> blood result related to the mean values in the group of overload with sodium bicarbonate 5% was 4.5 with deviation of 0.7, while patients who were subsequently treated conventionally was 4.1 with deviation of 0.6 (Table 2).

Chlorine in blood in the group of fifteen patients who were subsequently treated according to test results with bicarbonate overload 5% sodium was 109.0 with deviation 3.2, while in the control group treated conventionally was 107.0 with deviation of 3.0 (Table 2).

**Table 3:** Minimum, maximum Values, average(X) and standard deviation (S) initial serum bicarbonate, afterload, serum chlorine and urinary anion gap.

PARAMETERS	MINIMUM*	MAXIMUM*	X ± S*
HCO <sub>3</sub> INITIAL	13.4	23.5	19.4 ± 2.5
HCO <sub>3</sub> AFTERLOAD	19.7	34.0	25.5 ± 3.0
Cl SERUM	104.0	116.0	107.9 ± 3.2
ANIÓN GAP URINARY	-63.6	218.0	17.4 ± 48.6

\*Values based on 30 patients with metabolic acidosis who underwent the overload test with bicarbonate 5%.

**Table 4:** Presence of alterations renal metabolism.

ALTERATIONS RENAL METABOLISM	F (%)
Distal renal tubular Acidosis (Δ PCO <sub>2</sub> ) <sup>*</sup>	12 (40.0) <sup>***</sup>
Hypercalciuria <sup>**</sup> (>2years ≥ 0.20; <2years ≥ 0.30)	7 (33.3) <sup>***</sup>
Hyperuricosuria <sup>***</sup>	12 (57.1) <sup>***</sup>

\*Percentages based on 30 patients with metabolic acidosis + overload test with sodium bicarbonate 5%

\*\*Percentages based on 21 patients

\*\*\* (P &lt; 0.001)

Δ PCO<sub>2</sub> = Differential OCO<sub>2</sub> - PCO<sub>2</sub>

By jointly compute initial values for parameters HCO<sub>3</sub>, and afterload, and serum chlorine, urinary anion GAP was determined that the minimum values were respectively 13.4, 19.7, and 104.0, -63.6, while the maximum values in the same order were 23.5, 34.0, 116.0 and 218.0. Meanwhile, the average values for initial HCO<sub>3</sub> were 19.4 and standard deviation of 2.5. HCO<sub>3</sub> afterload result equal a 25.5 and standard deviation 3.0. Serum chlorine average was 107.9 and standard deviation of 3.2. Urinary GAP anion average 17.4 and 48.6 standard deviation (Table 3).

In the evaluated patients, it was found in 40.0% distal renal tubular acidosis, being significantly different effect (P < 0.001) compared to 0%. As hypercalciuria incidence was 33.3%, also significant with respect to the absence. Also the incidence of hyperuricosuria (57.1%) resulted differed of 0 (P < 0.001 (Table 4).

**Table 5:** Means values (x) and Standard Deviation (s) of evolution of the weight values.

GROUPS AND WEIGHT (grs)	X ± S	STATISTICAL SIGNIFICANCE
<b>TREATED BY METABOLIC STUDY</b>		
Weight income (n=14) Weight at discharge (n=14)	6107.1 ± 1681.3 5882.8 ± 1702.3	t = -1.69; G.L.= 13 P < 0.113 (n.s.)
Dry weight (n=12) Weight at discharge (n=12)	5671.6 ± 1754.8 5882.8 ± 1702.3	t = +2.95; G.L.= 11 P < 0.013
Dry weight (n=8) Hereinafter weight (n=8)	5671.6 ± 1754.8 7325.0 ± 1704.1	t = +3.95; G.L.= 7 P < 0.006
<b>CONVENTIONAL TREATMENT</b>		
Weight income (n=15) Weight at discharge (n=15)	7620.1 ± 2347.0 7242.8 ± 2291.9	t = -1.83; G.L.= 14 P < 0.08 (n.s.)
Dry weight (n=14) Weight at discharge (n=14)	7065.0 ± 1994.6 7437.1 ± 2246.3	t = +3.08; G.L.= 13 P < 0.009

(n.s.) No significant difference.

**Table 6:** Mean values(x) and Standard Deviation (s) of total protein and serum albumin at admission and discharge.

BLOOD CHEMISTRY	GROUPS		STATISTICAL SIGNIFICANCE
	TREATED BY METABOLIC STUDY X ± S	CONVENTIONAL TREATMENT X ± S	
Total protein	5.4 ± 1.5	4.8 ± 0.9	t = ± 1.24; G.L. = 27 P< 0.224 (n.s.)
Albumin (admission)	2.2 ± 0.8	2.0 ± 0.9	t = ± 0.54; G.L. = 27 P< 0.571 (n.s.)
Albumin(discharge)	3.0 ± 0.6	2.1 ± 0.1	t = ± 3.67; G.L. = 12 P< 0.003

(n.s.) = No significant difference.

In the group of patients who were treated according to test results overload with sodium bicarbonate 5%, weight (grs) average income was 6107.1 and standard deviation 1681.3, with the exit of 5882.8 and 1702.3 respectively ( $P > 0.05$ ). As the dry weight mean value and standard deviation, the figures were  $5671.6 \pm 1754.8$ , there was significant difference ( $P < 0.013$ ) to egress weight. With regard to the successive average weight (measured after discharge of the patient), the mean value was 7325.0 and 1704.1 standard deviation, establishing a significant difference ( $P < 0.006$ ) when compared with the average dry weight (Table 5).

Looking at the results for the group of patients who were treated conventionally weight (grs) average income was 7620.1 and standard deviation 2347.0, at the exit 7242.8 and 2291.9, without significant difference ( $P > 0.05$ ) between mean values. For dry weight mean value and standard deviation, the figures were  $7065.0 \pm 1994.6$ , having a significant difference ( $P < 0.009$ ) relative to weight of discharge (Table 5).

The average value of the total protein in the group treated as test results overload with sodium bicarbonate 5%, was 5.4 with deviation of 1.5, while in the control treated without considering the results of metabolic testing was 4.8 with deviation of 0.9, no significant difference ( $P < 0.05$ ) between both mean values (Table 6).

The average value of the blood albumin at admission in the case group was  $2.2 \pm 0.8$ , while in the control group was  $2.0 \pm 0.9$ , no finding significant difference ( $P > 0.05$ ) between the mean values.

The average value of albumin in the blood to exit (group case) of fifteen patients, of which information was obtained in six of them, who were treated according to the results of metabolic testing was  $3.0 \pm 0.6$ , whereas in the control group, from fifteen patients, in eight of them treated without considering the results of metabolic testing was  $2.1 \pm 0.1$ , being significant ( $P < 0.05$ ) between both mean values (Table 6).

## Discussion

All of the patients (100%) with metabolic acidosis were with alteration of growth in height, which agrees with those reported by other authors that have drawn attention to the impact of metabolic acidosis on linear growth [26-29]. Both the formation of cartilage and bone are decreased resulting in short stature. Low serum growth factor linked to insulin (IGF-1) levels and low hepatic receptor and growth hormone receptor mRNAs of IGF-1 has been reported in rats with metabolic acidosis [12]. The metabolic acidosis is also associated with reduced Triiodothyronine (T3) and Thyroxine (T4) and elevated TSH [30,31], also determining growth hormones.

The metabolic alterations found were distal tubular dysfunction, hypercalciuria, hyperuricosuria. In hypercalciuria of the evaluated patients could be intervening several factors such as distal renal tubular dysfunction, metabolic acidosis which is accompanied by release of bone calcium ions to neutralize the excess hydrogen ions, the positive balance of sodium found in the severe malnutrition that causes a volume expansion extracellular secondarily inhibits tubular reabsorption of calcium, interleukin I and other cytokines and prostaglandins monocytic origin that would be released in infectious processes in these patients with increased secondary calcitriol and increased bone reabsorption [32]. Hyperuricosuria may be the result of protein hypercatabolism associated with metabolic acidosis [33] and concurrent infections.

When comparing the dry weight (after the disappearance of edema) and weight at discharge of patients in the study, it was noted in all patients treated and not treated according to renal metabolic study, that had a weight gain, not observed statistical significance when comparing the groups together. In analyzing the mean values of total protein and albumin ingress and egress a statistically significant difference ( $P < 0.05$ ) was obtained between the average value of albumin at discharge of treated patients ( $3.0 \mu\text{g} / \text{dl}$ ) and untreated ( $2.1 \mu\text{g} / \text{dl}$ ). The results show that patients treated according to the metabolic study achieved clinical improvement (weight) and biochemical (albumin) that will make them less vulnerable to infection, the vicious circle malnutrition and disease, which leads to frequent readmissions and chronic alterations of its ponder-statural growth.

These results agree with other studies which show that experimentally induced metabolic acidosis in normal human for at least seven days reducing serum albumin [13,14]. The reduction of protein synthesis, increased protein breakdown and impaired oxidation of amino acids, have been suggested as factors contributing to a reduction of serum albumin in the presence of metabolic acidosis [15,17].

## Conclusions and Recommendations

If possible, in dehydrated patients, oral Rehydration Solution for Malnourished patients (ReSoMal), which contains less sodium, increased potassium intake and trace elements (zinc and copper) are given.

Once the hemodynamically stable patient, the treatment of infectious focus are given with attention the nephrotoxicity of antibiotics in patients with low albumin levels.

It is feeding formulas adapted to gastrointestinal disorders that often these patients will begin. Water requirements will be 20% low in those edematous and 100 ml / kg in marasmus. Once the patient melts edema progressively increase fluid intake. Caloric requirements are 80 calories / Kg in patients with edema and 100 calories/ Kg in children with marasmus. Hyperprotein diets are avoided to reduce the risk of hypercalciuria.

All severe malnourished patients, once hemodynamically stable are determined the following variables: Arterial blood gases, serum creatinine, serum and urinary electrolytes, urinalysis, serum calcium and phosphorus.

In patients presenting one of the following changes: serum bicarbonate  $< 22 \text{ mEq} / \text{l}$ , blood pH  $< 7.35$ , urinary pH  $> 5.5$ , urine

density <1020, positive urinary anion gap, is considered a carrier metabolic acidosis distal renal tubular dysfunction. As these potentially trainers patients urinary calculi by hypercalciuria, we recommend avoiding dietary an excessive load of sodium chloride, used as alkalizing potassium citrate better than bicarbonate sodium, which reduces the formation of urinary calculi and allows potassium intake in these patients.

This operation is expected to reduce hospital stay and watch the good progress of the patient from the clinical point of view, improved weight and biochemical results as serum albumin, breaking the vicious circle in which these patients are frequently involved.

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