Electrolyte and Acid-Base Balance (Sodium, Potassium, and pH) during Severe Acute Malnutrition in Children under 5 Years Old in the Democratic Republic of the Congo

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Introduction

Severe Acute Malnutrition (SAM) is a serious public health problem responsible for millions of deaths annually worldwide. Nutritional deficiencies, whether quantitative or qualitative, are a very common cause leading to a state of malnutrition [1-3]. Directly or indirectly, it is the first cause of acquired immunodeficiency facilitating a large number of serious microbial infections that can lead to death [4-7]. The severe acute malnutrition is an advanced stage of malnutrition defined by the following criteria: W/T (weight/height) <-3 scores, or brachial perimeter (MUAC) <115 mm and/or bilateral presence of edema associated with medical complications or lack of appetite [8]. A weight-height index of less than -3 scores is a highly defined criterion of SAM [2,9]. Children with a weight/height greater than -2 but less than -1 score have a lower mortality risk than children with a weight-for-height index of less than -3 scores but those with a weight/height greater than -1 have an even lower risk of death [10].

In developing countries, poverty and inadequate health services are responsible for the death of millions of people yearly, particularly children due to malnutrition, and its interaction between malnutrition with micro-nutritional disorders, frequent parasite infections, diarrhea and various malabsorption-related disorders [11,12]. Some electrolyte disturbances that accompany malnutrition are even aggravated by inadequate nutrition [5]. The consequences can be dramatic and lead to a syndrome of multi-organ failure or even death. It is, therefore, useful to know, the physiopathological aspects of this syndrome in order to prevent its onset soon as possible and thus reducing resultant catastrophic risk involved [13]. Osmoregulation in the body is a vital physiological process where water moves against a gradient (that is, from the compartment of low osmolality to the compartments of the strongest osmolality). This explains the mechanism of water reabsorption caused by the reabsorption of Na+ ions in the proximal convoluted tubes of the kidneys [13,14]. The main cation of extracellular fluids is Na⁺ and their main anion is Cl-. However, the plasma contains a little less than the interstitial fluid since it is electrically neutral and its non-diffusing proteins are normally in the form of anion [15]. The K⁺ ion is the main intracellular cation. It is necessary for the functioning of nerve and muscle cells as well as several essential metabolic activities including protein synthesis [13]. The slightest variation in the concentration of K⁺ ions on the membrane has the effects on neurons and muscle fibers [16]. All biochemical reactions in the body are influenced by the pH of the environment in which they take place [17]. The acid-base balance of body fluids is therefore essential for homeostasis and its regulation is extremely precise.

This study contributes to improving the management of severe acute malnutrition. Thus, providing insight and knowledge of the electrolyte and acid-base disorders observed during severe acute malnutrition in children under 5 years old resulting in better management of the condition in the latter future.

Materials and Methods

This study was conducted in the Nutritional and Intensive Therapeutic Unit (NITU) of the General Reference Hospital Jason Sendwe located Lubumbashi, DR (Congo). NITU is a unit specializing...
in the management of cases of severe acute malnutrition (SAM); it is coordinated by a qualified nutritionist, assisted by nurses, all under the supervision of the Chief Medical Officer of Pediatrics. Acute severe malnourished children were admitted to pediatrics and referred to NITU according to their nutritional status.

The collaboration with the head of pediatrics made it possible to be informed of the new admissions on which the blood samples were made according to the criteria described below. Severe acute malnutrition was confirmed clinically after evaluation of anthropometric parameters which are: W/T, MUAC, and presence of edema. The antecedents of the malnourished were also considered, this allowed excluding the old cases or cases admitted for other pathologies for which the malnutrition occurred during the hospitalization.

**Samples collection**

This study was done for a period of one month, each day NITU admits at least 1 case of malnutrition. For the first week, the blood was taken on 8 children, the second week on 6 children the third week on 5 children and fourth week the blood was taken on 10 children. The data was collected through this approach; 1 ml of blood was taken from the venipuncture at the elbow crease on children already confirmed malnourished at admission and on normal children. The experimental procedures were carried out in accordance with instructions provided by Lubumbashi Provincial Laboratory Medical Ethics committee.

**Ionograms analysis**

This analysis was held in a suitable laboratory in Lubumbashi. Then Blood ionograms were performed on 30 children from 6 to 60 months; 24 of them were admitted to the NITU and 6 children were normal. The tube in which the samples were placed contained an anticoagulant and the collected blood was rapidly centrifuged at 1000 rpm for 1 minute. Blood gas analyzer ABL800 FLEX was used for the analysis.

**Data analysis**

The experiments were performed in sample size calculation using a proportional method that is 1 ml=1 child. The data was calculated using percentage.

<table>
<thead>
<tr>
<th>Natraemia</th>
<th>With edema n1=12</th>
<th>Without edema n2=12</th>
<th>Control n3=6</th>
<th>Malnourished Total n=24</th>
<th>Total children n=30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normonatraemia (133-143mmol/l)</td>
<td>0</td>
<td>3 (33.3%)</td>
<td>6 (100%)</td>
<td>3 (12.5%)</td>
<td>9 (30%)</td>
</tr>
<tr>
<td>Hyponatraemia (&lt;133mmol/l)</td>
<td>10 (83.3%)</td>
<td>9 (66.7%)</td>
<td>0</td>
<td>19(79.2%)</td>
<td>19(63.3%)</td>
</tr>
<tr>
<td>Hypernatraemia ( &gt;150mmol/l)</td>
<td>2(16.7%)</td>
<td>0</td>
<td>0</td>
<td>2(8.3%)</td>
<td>2(6.7%)</td>
</tr>
<tr>
<td>Total (%)</td>
<td>12(100%)</td>
<td>12(100%)</td>
<td>6(100%)</td>
<td>24(100%)</td>
<td>30(100)</td>
</tr>
<tr>
<td>Kalaemia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normokalaemia (3.5-4.5mmol/l)</td>
<td>0</td>
<td>0</td>
<td>6 (100%)</td>
<td>0</td>
<td>6 (20%)</td>
</tr>
<tr>
<td>Hypokalaemia (&lt;3mmol/l)</td>
<td>9 (75%)</td>
<td>7 (58.3%)</td>
<td>0</td>
<td>16(66.7%)</td>
<td>16 (53.3%)</td>
</tr>
<tr>
<td>Hyperkalaemia (&gt;5mmol/l)</td>
<td>3 (25%)</td>
<td>5 (41.7%)</td>
<td>0</td>
<td>8(33.3%)</td>
<td>8 (26.7%)</td>
</tr>
<tr>
<td>Total (%)</td>
<td>12(100%)</td>
<td>12(100%)</td>
<td>6(100%)</td>
<td>24(100%)</td>
<td>30(100%)</td>
</tr>
</tbody>
</table>

**Results**

**Patient characteristics**

This study was based on 30 children aged 6-60 months. 45.8% (n=11) of severe acute malnutrition was aged 6 to 24 months, which is a weaning period, Table 1.

<table>
<thead>
<tr>
<th>Sex</th>
<th>Ni</th>
<th>%</th>
<th>Age (months)</th>
<th>Ni</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>16</td>
<td>53.3</td>
<td>6 – 24</td>
<td>11</td>
<td>45.8</td>
</tr>
<tr>
<td>Female</td>
<td>14</td>
<td>46.7</td>
<td>24 – 42</td>
<td>10</td>
<td>37.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>42 – 60</td>
<td>9</td>
<td>16.7</td>
</tr>
<tr>
<td>Total</td>
<td>30</td>
<td>100</td>
<td></td>
<td>30</td>
<td>100</td>
</tr>
</tbody>
</table>

**Table 1:** Distribution of malnourished by gender and age.

Shows that 66.7% (n=16) of the severely acute malnourished were male. 50% (n=12) of the malnourished patients, 40% of children had edema. 20% (n=6) of these children were normal.

**Malnourished depending on the natraemia**

These 30 children were subdivided into 3 groups of which 12 had edema 12 others had no edema and 6 were normal. The results show that among the 24 severely malnourished children, 12 of them had edema. 10 (83.3%) of these 12 had hyponatraemia (70-90) mmol/L and 2 (16.7%) children only had hypernatraemia (155-162) mmol/L. The other 12 without edema, 3 (33.3%) among them had a normal rate of natraemia (133-145) mmol/L compared to normal children. Then 9 (66.7%) of these 12 presented the hypernatraemia and the control are presented in (Table 2).

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Malnourished depending on pH

The potassium analysis approved that 9 (75%) malnourished patients had Hypokalaemia (1.9-2.3) mmol/L and 3 (25%) of them had Hyperkalaemia (5.8-6.1) mmol/L in the group of children with edema. In the group without edema, 7 (58.3%) had Hypokalaemia (2.5-2.3) mmol/L (P < 0.05) and 5 (41.7%) had hyperkalaemia (6-6.8) mmol/L. None of these children had shown normokalaemia compared to normal children (Table 2).

Malnourished depending on the kalaezia

The potassium analysis approved that 9 (75%) malnourished patients had Hypokalaemia (1.9-2.3) mmol/L and 3 (25%) of them had Hyperkalaemia (5.8-6.1) mmol/L in the group of children with edema. In the group without edema, 7 (58.3%) had Hypokalaemia (2.5-2.3) mmol/L (P < 0.05) and 5 (41.7%) had hyperkalaemia (6-6.8) mmol/L. None of these children had shown normokalaemia compared to normal children (Table 2).

Discussion

Ionic salts provide essential minerals for neuromuscular excitability, secretory activity, membrane permeability, and several other cellular functions. Therefore, ions are important factors in the regulation of water movement. Edema, as a fluid distribution disorder, can increase electrolyte disturbances [18]. Thus, the group with edema manifested the existence of electrolyte disorders that follow. Hyponatraemia was the most observed disorder; 79.2%, the malnourished had manifested it with a clear dominance in a group with edema among 24 malnourished children (Table 2). In case of edema, there was a relative excess of water with respect to salts hence the extracellular hyperhydration. This situation seemed to reinforce the importance of edema sequestering water and sodium in the extravascular environment [19]. In the group of malnourished children without edema, hyponatraemia was probably caused by the digestive loss of Na⁺ due to diarrhea and vomiting [18,20]. However, children who had no diarrhea and vomiting or one of those two had normal Na⁺ corresponding to the control group (Table 2). In this case, the blood sodium concentration decreased by extravascular leakage or dilution due to water retention, especially in cases of edema. Hyponatraemia is a low dilution and plasma osmolarity (POsm). Consumption of water exceeding the renal excretion capacity results in hyponatraemia [21,22]. It is also related to the presence of other solutes such as glucose or mannitol, which cause a call to water, dilution of the extracellular medium and intracellular dehydration [16,23]. In addition to losses, cases of hyponatraemia were observed (8.3%) among malnourished patients with edema. There was an excessive release of free water by edema and this resulted in an exceptional increase of sodium in the blood plasma [24]. Hyponatraemia is usually due to water loss, which means a lack of drink or sodium gain, because of that the cells shrink [25,26]. This situation raises the risk of inappropriate nutrition in a world where nutrition therapy has become automatic. Maintaining the balance between gains and losses of Na⁺ ion is one of the main functions of the kidneys [14]. The Na salts (NaHCO₃, and NaCl) in their ionized form constitute 95% of the solutes present in the extracellular fluid [27]. Regulation of the equilibrium of water and Na⁺ ions are inextricably linked to arterial pressure and blood volume. The regulation of sodium ion balance involves the nervous and hormonal mechanisms [21]. The disorders of the natraemia are associated with the disorders of the volemia: dehydration or hyperhydration.

Hypokalaemia was observed in 66.7% with an almost similar distribution in both groups (Table 2). The hyperkalaemia is often present as Pk <3.5 mmol/liter, this concentration causes almost no problems; the results showed that the symptoms do not always occur in patients up to Pk<2.5 mmol/L but significant muscle weakness usually occurs at Pk<2.0 mmol/liter. Diarrhea and vomiting cause hypokalaemia due to potassium leakage into the intracellular environment [28]. In hypokalaemia, an increase in aldosterone stimulation causes renal loss or an increase in Na⁺ release [25]. Compare to the control group, hyperkalaemia was observed in 33.3% with a predominance in the group of malnourished without edema (Table 2). Fe⁺ deficiency has caused anemia in malnourished, and this anemia causes hyperkalaemia when potassium is released at the time of the destruction of red blood cells during hemolysis. Hyponatraemia makes the extracellular medium hyperosmotic. this leads to the release of water with potassium from the intracellular medium to the outside of the cell and the kaliemia increases [15]. An excess of K⁺ ions in the extracellular fluid can be followed by a loss of excitability of the membranes of neurons and muscle fibers. The heart is particularly sensitive to the concentration of K⁺ ions [28], hyperkalaemia or hypokalaemia disrupts electrical conduction in the heart and leads to sudden death. K⁺ ions are also part of the body's buffer system that compensates for the rise in body fluid pH [29,30]. The movement of the hydrogen ions in the cells is compensated by the opposite movement of the K ions which maintain the equilibrium of the cations on both sides of the plasma membrane [28].

Acidosis has been observed in all malnourished with or without edema (Table 2). Acidemia is the lowering of the blood pH, it becomes an acidosis in the case that there are the processes that provoke it. Metabolic acidosis is detected as an increase in plasma anion difference (GA), but without the change in pH or plasma [HCO₃⁻] [31,32]. In a situation of pronounced acidosis, with the depletion of chemical buffers, the potassium ions are released from the cell in exchange for the H ions in an attempt to reduce the acidity of the extracellular medium and thereby increase that of the intracellular environment [22]. The pH of these malnourished varied between 1-4 compared to

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Table 2: Distribution of the malnourished according to electrolyte disturbances. The data represent a percentage. Natremia-63.3% of hyponatraemia and 6.7% of hypernatremia, Kalaemia-53.3% of hypokalaemia and 26.7% of hyperkalaemia. Concerning pH, 100% of the malnourished with or without edema have acidosis.

<table>
<thead>
<tr>
<th></th>
<th>Normal PH</th>
<th>Acidosis</th>
<th>Alkalosis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>6(100%)</td>
<td>0</td>
<td>24(100%)</td>
<td>30(100%)</td>
</tr>
<tr>
<td>6(20%)</td>
<td>12(100%)</td>
<td>12(100%)</td>
<td>0</td>
<td>24(100%)</td>
</tr>
</tbody>
</table>

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the control group in which pH of children ranged between 7-7.5. In severe acute malnutrition, two situations explain the acidosis:

- Carbohydrate deficiency induces neoglycogenesis through the use of fatty substances as a source of energy with the consequence of the accumulation of ketonic bodies which are strong acids
- Proteins being buffers, their deficiency increases the risk of acidity [17].

**Conclusion**

This study focused on the electrolyte and acid-base aspects during severe acute malnutrition. This has highlighted micro-nutritional disorders, which accompany deficiencies in protein and carbohydrates. Blood ionograms revealed variable electrolyte and acidosis disorders compared to normal children. The results indicated that hyponatraemia was the most observed disorder with a clear predominance in the group of edemas. Considering electrolyte disturbances as admission criteria will facilitate therapeutic management by reducing the mortality rate in patients suffering from malnutrition.

**Conflict of Interest**

The authors declare no conflict of interest.

**References**