Hypophosphatemia in postoperative patients with total parenteral nutrition: influence of nutritional support teams

M. J. Martínez*, M. A. Martínez**, M. Montero*, E. Campelo*, I. Castro* y M. T. Inaraja*


Abstract

Purpose, setting and subjects: We conducted a prospective, descriptive study of postoperative patients under total parenteral nutrition controlled by a Multidisciplinary Nutritional Support Team in a tertiary care hospital. Between January 2002 and November 2003. Data of nutritional status, nutritional support, hypophosphatemia, electrolyte and metabolic complications were reviewed.

Results: 215 postoperative patients (63.3% male, 68 ± 13.9 years old, 47.4% neoplasia), were included. Nutritional support according nutritional needs was made during fasting 14.2 ± 18.4 days. Mild-moderate initial malnutrition was present in 58% of patients. 18.1% developed postoperative hypophosphatemia 96 hours after starting total parenteral nutrition containing phosphate. 37.7% patients showed moderated and 6.5% severe hypophosphatemia. Nutritional intervention corrected hypophosphatemia (p < 0.001). Factors related to hypophosphatemia were hypokalemia, hypomagnesemia, hypercalcemia, female sex, neoplasia, 96-hour postoperative period and duration of nutrition.

Conclusions: Prevalence of hypophosphatemia in postoperative patients with total parenteral nutrition is high and needs timely monitoring. The intervention of Multidisciplinary Nutritional Support Team is effective detecting and correcting postoperative hypophosphatemia.

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Key words: Hypophosphatemia. Multidisciplinary nutritional support team. Electrolytic complications. Parenteral nutrition.

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Resumen

Propósito, contexto y sujetos: Realizamos un estudio descriptivo, prospectivo, en pacientes con nutrición parenteral total controlados por un Equipo Multidisciplinar de Soporte Nutricional de un hospital terciario, entre enero de 2002 y noviembre de 2003. Se revisaron los datos de estado nutricional, soporte nutricional, hipofosfatemia, y de complicaciones electrolíticas y metabólicas. Resultados: Se incluyó a 215 pacientes posoperados (63.3% varones, edad 68 ± 13,9 años, 47,4% de neoplasias). Se realizó un soporte nutricional de acuerdo con las necesidades nutritivas durante un periodo de ayuno de 14,2 ± 18,4 días. Había malnutrición de base en un 58% de los pacientes. El 18,1% de los pacientes desarrolló hipofosfatemia posoperatoria 96 horas después de iniciar la nutrición parenteral total que contenía fosfato. El 37,7% de los pacientes tuvo hipofosfatemia moderada y 6,5% grave. La intervención nutricional corrigió la hipofosfatemia (p < 0,001). Los factores relacionados con la hipofosfatemia fueron hipopotasemia, hipomagnesemia, hipercalemia, sexo femenino, neoplasia, periodo post-operatorio de 96 horas y duración de la nutrición.

Conclusiones: La prevalencia de hipofosfatemia en pacientes posoperados con nutrición parenteral total es alta y requiere una vigilancia estrecha. La intervención del Equipo Multidisciplinar de Soporte Nutricional es eficaz para la detección y corrección de la hipofosfatemia posoperatoria.

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Introduction

Postoperative (PO) patients are prone to hypophosphatemia (HP) and other metabolic or electrolytic disorders. Some reports have shown 20-50% HP in severe PO patients. Only severe HP (< 1.0 mg/dL) is symptomatic: generalised muscle weakness, seizures, coma and respiratory or cardiac failure. In order to prevent HP in patients under total parenteral nutrition (TPN), 10-20 mMol of phosphorus/1,000 carbohydrate kcal (CHkcal) is recommended. Severely malnourished patients may require more phosphorus initially. High HP variability in PO patients, mainly related to refeeding syndrome, requires serum levels monitoring and resolution.

The aim of this study is to evaluate the effectiveness of a Multidisciplinary Nutritional Support Team (MNST) to detect and correct HP in PO patients with TPN as well as other electrolyte abnormalities.

Methods

All PO patients under TPN from January 2002 to November 2003 in a tertiary care hospital (420 beds) were prospectively included. MNST included a physician, a pharmacist, a nurse and a dietician. TPN started progressively, (≤ 20 Kcal/Kg/d initially). Patients without renal impairment received 10-15 mMol of Phosphorus/800-1,000 CHkcal at the beginning of TPN. Physician and pharmacist carry on daily monitoring, setting analytical tests and requirements adjusting. Data of Nutritional Status (NS), hypophosphatemia, nutritional support, nutritional and clinical evolution, electrolytic and metabolic complications were recorded by MNST every day with Nutridata® program. Plasma electrolytes monitoring were performed at least once a week in ward patients and twice a week in recovery units. Complications were defined as values above or below normal laboratory range. Team effectiveness was assessed through the difference between initial abnormal serum levels (complication onset) and final serum levels (at the end of TPN). Consideration was taken about that each patient could have more than one electrolyte abnormality, even with some electrolytic disorder repeated.

Categorical variables were analysed with Chi-square test and quantitative variables by Student-t test and Wilcoxon’s test. Bivariate correlations were used to compare quantitative and qualitative variables. Multivariate analysis was performed by using all the baseline variables to determine the risk factors for HP. A statistical SPSS v 8.0 package was used; statistical significance was considered at p < 0.05.

Results

215 PO patients (63.3% male, age 68 ± 13.9 years) were included (34.4% gut resection, 14.4% gastrectomy, 6% bilipancreatic surgery, 10.2% peritonitis). Neoplasia as primary diagnosis was present in 47.7%.

During 14.2 ± 18.4 fasting days PO patients received TPN adjusted to nutritional needs (Harris-Benedict formula): 30.4 ± 6.0 Kcal/Kg/d, 13.9 ± 2.7 g N/d, 0.20 ± 0.052 g N/Kg/d, 150 ± 26.9 non-protein Kcal/g N. Median surgical hospital stay was 20 days [95% CI (25.04-32.54)]. Initial NS was classified as 34.0% normal, 34.4% mild, 24.2% moderate and 7.4% severe malnutrition. Final NS improved in 50.7% patients and no changed in 22.5%. Mortality rate was 25.6% (55 patients).

Table I shows several electrolytic abnormalities. The incidence of HP is indicated in table II.

<table>
<thead>
<tr>
<th>Electrolytic complications (215 patients)</th>
<th>Incidence</th>
<th>Correction of complication by intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean (initial-final) (P)</td>
</tr>
<tr>
<td>Hypokalemia</td>
<td>41.9</td>
<td>3.1-4.0 mEq/l (p &lt; 0.001)</td>
</tr>
<tr>
<td>Hyponatremia</td>
<td>39.1</td>
<td>131-136 mEq/l (p &lt; 0.001)</td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>22.3</td>
<td>1.54-1.77 mg/dl (p &lt; 0.001)</td>
</tr>
<tr>
<td>Hyperphosphatemia</td>
<td>14.9</td>
<td>5.4-4.5 mg/dl (p &lt; 0.001)</td>
</tr>
<tr>
<td>Hyperkalemia</td>
<td>10.2</td>
<td>5.6-4.6 mEq/l (p &lt; 0.001)</td>
</tr>
<tr>
<td>Hypernatremia</td>
<td>12.6</td>
<td>149-142 mEq/l (p &lt; 0.001)</td>
</tr>
<tr>
<td>Hypermagnesemia</td>
<td>10.7</td>
<td>2.72-2.27 mg/dl (p &lt; 0.05)</td>
</tr>
<tr>
<td>Hypercalcemia</td>
<td>17.7</td>
<td>11.4-10.9 mg/dl (p &lt; 0.001)</td>
</tr>
</tbody>
</table>

Table I

Incidence of electrolytic abnormalities

Categorical variables were analysed with Chi-square test and quantitative variables by Student-t test and Wilcoxon’s test. Bivariate correlations were used to compare quantitative and qualitative variables. Multivariate analysis was performed by using all the baseline variables to determine the risk factors for HP. A statistical SPSS v 8.0 package was used; statistical significance was considered at p < 0.05.

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Table I shows several electrolytic abnormalities. The incidence of HP is indicated in table II.

HP significantly decreased 32.5% (28/86 patients) in 2003 versus 52.3% (67/129 patients) in 2002 (p < 0.05). MNST intervention adjusted PO HP (p < 0.001) and main abnormalities. In moderate HP, a phosphate supplementation of 20 mMol/1000 CHkcal/d corrected HP; whilst severe HP needed 20-40 mMol/1000 CHkcal in a 24-h period after detection.

HP was found to correlate with hypokalemia (p < 0.001), hypomagnesemia (p = 0.001), hypercalcemia (p < 0.001), female (p = 0.007), neoplasia (p < 0.05), length of TPN (p < 0.001) and surgical hospital stay (p < 0.001). We found bivariate correlation between initial malnutrition and 96-hour PO HP (p < 0.05) but not with HP (considering all degrees).
Discussion

In our study, moderate HP in PO patients under TPN with standard phosphate addition was high, mainly in the 96 hours PO period, in spite of an increased phosphate supplementation the first day of TPN. Moderate HP was frequent (37.7%) but incidence of severe HP was only 6.6%, although both were promptly detected and corrected without increased morbidity. The importance of grossly abnormal serum tests, if done, may not be recognised if patients are not treated by nutrition units.

Risk of calcium phosphate precipitation limits the amount of inorganic phosphate in TPN so standard addition in our case was ≤ 20 mMol of phosphate per bag. We used glicerolphosphate when a higher supplementation was required.

In the context of refeeding syndrome, we detected hypokalemia and hypomagnesemia as related factors with HP. We found bivariate correlation between initial malnutrition and 96-hour postoperative HP, but not with total HP. Acute renal failure was less related with HP, although the occurrence of HP in chronic renal failure was recently reported. We had a high incidence of neoplasias which could be associated with an increased risk of HP as reported previously. All patients received 15-20 Kcal/kg initially and by the 4th day were receiving < 40 Kcal/kg. Standard phosphate supplement may not be enough in these PO patients, especially when organic salts are used. Relation between HP and duration of TPN and surgical hospital stay could be explained by a higher severity of patients.

Early detection of this complication allows to a rapid resolution, before clinical consequences. Appropriate therapeutic monitoring permits to know the amount and degree of complications and the ability of MNST to correct them.

Quality improvement in TPN care can be achieved developing a program implemented by MSNT, including patients monitoring. According to our results, monitoring serum phosphate in the first 96 hours of TPN and in prolonged TPN is needed, and must be done on regular basis.

In conclusion, prevalence of hypophosphatemia in postoperative patients under total parenteral nutrition is high. Phosphate supplements must be higher from the beginning, mainly in patients prone to hypophosphatemia. The intervention of a MNST shows to be effective in order to detect and correct postoperative hypophosphatemia.

References


### Table II

<table>
<thead>
<tr>
<th>Phosphate abnormalities</th>
<th>Incidence (patients)</th>
<th>Initial-final Phosphate mean (mg/dl)</th>
<th>Correction of complication by MNST intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate (P &lt; 2.2 mg/dl)</td>
<td>37.7</td>
<td>1.62-3.33</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>(81)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severe (P ≤ 1.0 mg/dl)</td>
<td>6.5</td>
<td>0.80-2.87</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>(14)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>96 h PO Moderate</td>
<td>18.1</td>
<td>1.49-3.4</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>(36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>96 h PO Severe</td>
<td>1.4</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Hypophosphatemia</td>
<td>(3)</td>
<td>1.44-3.23</td>
<td>P &lt; 0.001</td>
</tr>
<tr>
<td>Total Hypophosphatemia</td>
<td>(95)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


