HYPOPHOSPHATAEMIA IN BURNS

Castana O., Rempelos G., Faflia C., Apostolou C., Theodori A., Romana C., Alexakis D.

Department of Plastic and Reconstructive Surgery, Evangelismos General Hospital, Athens, Greece

SUMMARY. Hypophosphataemia is defined as a phosphate level of less than 2.5 mg/dl (0.8 mmol/l). Many of the pathophysiological changes and therapeutic interventions in the first week post-burn affect serum phosphorus concentration. A retrospective study reviewed the medical records of nine severely burned patients, and a special reference of decreased phosphate serum level was noted. Serum phosphorus levels declined, with a nadir between days 2 and 6 post-burn. The severe hypophosphataemia that often follows major burns returns to normal phosphorus levels on days 18 to 20 post-burn. Weight-based phosphorus dosing is safe to use in critically burned patients receiving nutritional support. Moderate doses effectively increase serum phosphorus concentrations.

Introduction

Hypophosphataemia is defined as a serum phosphate level of less than 2.5 mg/dl (0.8 mmol/l). Normal phosphorus (P) levels in adults are about 1% of total body weight. It is present in every cell of the body, although 85% of the body’s P is found in the bones and teeth. Normal plasma concentrations range from 0.8 to 1.6 mmol/l, or 2.5 to 5 mg/dl (0.032 mmol phosphate = 1 mg). P is absorbed more efficiently than calcium. Nearly 70% of the element is absorbed via the intestines, the rate depending on the levels of calcium and vitamin D and on the activity of the parathyroid hormone (which regulates the metabolism of P and calcium).

Patients with serious burns may lose phosphate, and replacement may be necessary since P plays major roles in intracellular energy metabolism. In the form of phosphate esters, it is a constituent of purine nucleotides, an important source of intracellular energy, and in the form of phospholipids it is a major structural component of cell membranes.

In the regulation of P, the intestine plays no significant role in phosphorus absorption - about 80% of dietary phosphate is absorbed. The kidney is the important organ for the homeostatic control of P so that the renal excretory rate of P primarily regulates the serum P concentration. The regulatory mechanisms are unknown and independent of the parathyroid hormone.

Critically ill patients receiving intravenous feeding often have low phosphate levels. Phosphate levels should be closely monitored in such patients, particularly if kidney function is impaired. Inorganic phosphates avoid compatibility with calcium in total parenteral nutrition (TPN) or enteral nutrition (EN) solutions. The addition of phosphate to TPN or EN solutions should be performed under the supervision of a licensed nutritionist.

The refeeding syndrome and associated electrolyte abnormalities occur in such patients when additional doses of P are not administered. The syndrome embraces a constellation of metabolic disturbances that occur as a result of the reinstitution of nutrition in patients who are starved or severely malnourished or have metabolic disturbances due to their critical condition. Patients can develop fluid and electrolyte disorders, especially hypophosphataemia, along with neurological, pulmonary, cardiac, neuromuscular, and haematological complications. For the prevention and treatment of the refeeding syndrome, the most important steps are the identification of patients at risk of developing the syndrome, the cautious institution of nutrition support, and the correction and supplementation of electrolyte and vitamin deficiencies.

Many of the pathophysiological changes and therapeutic interventions in the first week post-burn affect serum phosphorus concentration. A burn brings about a precipitous decrease in serum phosphate concentration that reaches its maximum between days 2 and 5 post-burn. Despite aggressive phosphorus supplementation, normal phosphorus levels in the serum are rarely seen before day 10 post-burn. Exogenous administration of epinephrine leads to hypophosphataemia. Burns, and the profound catecholamine release with which they are associated, may be the cause of the early decrease in serum phosphorus. In the early post-burn period the metabolic response occurs with increased plasma glucose, catecholamines, glucagons, and cortisol.

Possible explanations for post-burn hypophosphataemia include:

1. Intracellular accumulation of phosphate, inadequate phosphorus intake, excessive phosphate loss into the extravascular fluid, and hyperbolic urinary phosphate excretion.
2. The large doses of Ringer’s lactate given for initial burn resuscitation may decrease serum P by means of several mechanisms: a) metabolic alkalosis induced by lactate infusion may lead to an increase of glycolysis, which promotes the transfer of P to the intracellular space; b) lactate is converted into glycose in the liver, a process that requires high energy phosphate availability.

3. Refeeding syndrome.

Whether hypophosphataemia contributes significantly to the early post-burn hypo function of multiple organs is not known. Most patients who had the complication of hypophosphataemia also had some severe illness.\textsuperscript{1-4}

**Materials and methods**

A retrospective study of the records of nine severely burned patients with a special reference to the serum phosphate levels was conducted. The lowest mean value of serum phosphate in this group was 0.59 ± 0.2 mmol/l (normal range, 0.8-1.5 mmol/l), occurring on days 5 to 6 post-burn. Serum phosphate returned to normal levels in five of the patients except two who died. The patients who died showed the lowest serum P value. In the two other patients intravenous administration of P was performed, regulating the dose proportionally to body weight (Figs. 1-3).

**Results**

Serum phosphate levels declined, reaching a nadir between days 2 and 6 post-burn. The severe hypophosphataemia that often follows major burns returned to normal serum phosphorus levels on day 18 to 20 post-burn day.

When P levels drop below 2.0 mg/dl, intravenous therapy is indicated. When P varies between 2.0 and 3.0 mg/dl, \textit{per os} therapy is required.

**Conclusions**

Weight-based phosphorus dosing is safe for use in critically burned patients who receive nutritional support. Moderate doses effectively increase serum phosphorus concentrations.
RÉSUMÉ. L’hypophosphatémie est définie comme une condition où le niveau de phosphates est inférieur à 2,5 mg/dl (0,8 mmol/l). Beaucoup des modifications pathophysiologiques et des interventions thérapeutiques dans la première semaine post-brûlure exercent des effets sur la concentration du phosphate sérique. Nous avons effectué une étude rétrospective des fichiers cliniques de neuf patients grands brûlés, et nous avons noté une situation particulière d’un niveau de phosphate sérique diminué. Les niveaux du phosphate sérique diminuaient et touchaient des valeurs minimales entre deux et six jours après la brûlure. L’hypophosphatémie sévère qui se produit après les brûlures importantes retourne aux niveaux de phosphate normaux 18/20 jours après la brûlure. L’administration de phosphate en doses qui dépendent du poids du patient est une méthode sûre et utilisable aussi pour les patients dont les conditions sont critiques et qui reçoivent un support nutritif. Les doses modérées augmentent en manière efficace les concentrations du phosphate sérique.

BIBLIOGRAPHY


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Address correspondence to: Prof. Ourania Castana, Assimacopoulou 17 st, 15342 Agia Paraskevi, Athens, Greece. Tel.: 0030 6932104870, 0030 2106012270; e-mail: castana_ourania@yahoo.gr.

G. WHITAKER INTERNATIONAL BURNS PRIZE-PALERMO (Italy)
Under the patronage of the Authorities of the Sicilian Region for 2011

By law n.57 of June 14th 1983 the Sicilian Regional Assembly authorized the President of the Region to grant the “Giuseppe Whitaker Foundation”, a non-profit-making organization under the patronage of the Accademia dei Lincei with seat in Palermo, a contribution for the establishment of the annual G. Whitaker International Burns Prize aimed at recognizing the activity of the most qualified experts from all countries in the field of burns pathology and treatment. Law n° 23 of December 2002 establishes that the prize becomes biannual. The next prize will be awarded in 2011 in Palermo at the seat of G. Whitaker Foundation. The amount of the prize is fixed at Euro 20,660.00. The Adjudicating Committee is composed of the President of the Foundation, the President of the Sicilian Region, the Representative of the National Lincei Academy within the G. Whitaker Foundation, the Dean of the Faculty of Medicine and Surgery of Palermo University or his nominee, a Representative of the Italian Society of Plastic Surgery, three experts in the field of prevention, pathology, therapy and functional recovery of burns, the winner of the prize awarded in the previous year and a legal expert nominated in agreement with the President of the Region as a guarantee of the respect for the scientific purpose which the legislators intended to achieve when establishing the prize. Anyone who considers himself to be qualified to compete for the award may send by January 31st 2011 his detailed curriculum vitae to: Michele Masellis M.D., Secretary-Member of the Scientific Committee G. Whitaker Foundation, Via Dante 167, 90141 Palermo, Italy.
fondazionegwhitaker@virgilio.it