Three Cases of Hypophosphatemia Following Prolonged Seawater Immersion

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Abstract

Severe hypophosphatemia is a potentially life-threatening electrolyte disorder. There is limited information about serious electrolyte disorders in survivors of prolonged seawater immersion. We report three scuba divers who went missing in the ocean off southern Taiwan and were rescued 40 h later, about 100 km from where their dive began. They initially presented with hypothermia, severe dehydration, acute renal injury, rhabdomyolysis, severe hypophosphatemia, metabolic acidosis, and low T_3 syndrome. The possible etiologies of the hypophosphatemia include decreased phosphate intake, cellular redistribution of phosphate and increased renal excretion. Their decreased phosphate intake was a consequence of prolonged starvation. Medical treatment, which included rewarming, administration of carbohydrates, and correction of the metabolic acidosis, contributed to cellular redistribution of phosphate. The increased phosphaturia that we observed may have resulted from impaired tubular resorption of phosphate during recovery from acute renal tubular injury, aggressive extracellular fluid volume expansion, a surge of adrenal hormone, and metabolic acidosis. Aggressive managements can prevent seriously irreversible complications. Correction of life-threatening electrolyte imbalances and severe metabolic derangements can be life-saving and improve the prognosis for these patients. (J Intern Med Taiwan 2009; 20: 461-465)

Key Words：Hypophosphatemia, Seawater immersion, Rhabdomyolysis, Renal injury, Near-drowning

Background

Hypophosphatemia is a common, potentially life-threatening electrolyte disorder that is often seen in malnourished patients. We report three scuba divers who presented with hypophosphatemia after prolonged immersion in the sea.

Cases

On April 26, 2008, eight scuba divers went missing in the ocean off southern Taiwan. The powerful underwater currents and strong winds took them out of search range. During their ordeal, they endured cold saltwater (28℃ during the night),

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rough seas with three meter waves, painful sunburn, starvation, and exhaustion. They had no food or water intake, except for unavoidable ingestion of several liters of seawater. Forty hours later, they were rescued 100 km from where their dive began.

Three of the survivors, two males and one female, arrived at our hospital. They were alert, exhausted, hypothermic, and had shortness of breath, decreased skin turgor, severe sunburn, and abrasion wounds on the extremities caused by their diving suits and fins. On initial laboratory testing (performed one hour after rescue), there was mild elevation of the hematocrit (Hct, 0.409-0.504); elevated levels of blood urea nitrogen (BUN, 15-24 mg/dL) and creatinine (Cr, 0.6-1.4 mg/dL); markedly increased creatinine phosphokinase (CPK, 503-984 IU/L) with 100% as the CPK-MM fraction; decreased serum calcium (Ca^{2+}, 7.5-8.6 mg/dL) and markedly decreased serum phosphate (PO_4^{3-}, 1.0-1.7 mg/dL) (Table 1). Initially, the fractional excretion of sodium (F_ENa) was low and the fractional excretion of phosphate (F_EPO_4) was high (Table 1 and Figure 1). Measurement of arterial blood gases in room air were: pH, 7.32-7.37; $PaCO_2$, 27.9-32.7 mmHg; PaO_2, 116-117 mmHg; and HCO_3^-, 16.1-16.9 mmol/L. These readings indicate metabolic acidosis and hyperventilation (Table 1). All electrocardiograms and chest X-rays were normal. All three patients had transiently decreased total triiodothyronine (T_3) (which was normal when tested 2 weeks later) and elevated catecholamines (Table 1).

Table 1. Demographic, clinical, and laboratory data of three survivors of prolonged seawater immersion (recorded one hour after rescue)

Variables	Case 1	Case 2	Case 3	Reference Range
Age (yr) / Sex	37 / M	24 / M	44 / F	-
BMI (kg/m^2)	27.7	31.7	18.7	18.7-25.0
Temp (℃)	33.2	35.1	34.5	36.0-38.0
Hct	0.409	0.504	0.454	0.340-0.500
WBC ($\times 10^9$/L)	17.3	36.5	8.9	4.0-10.0
CPK (IU/L)	662	984	503	50-232
BUN (mg/dL)	24	15	16	8-20
Cr (mg/dL)	1.4	1.2	0.6	0.4-1.2
Na^+ (mmol/L)	149	143	142	136-144
K^+ (mmol/L)	5.8	4.4	4.3	3.5-5.1
Cl^- (mmol/L)	119	113	113	101-111
Ca^{2+} (mg/dL)	7.9	8.6	7.5	8.9-10.3
PO_4^{3-} (mg/dL)	1.6	1.7	1.0	2.7-4.5
Mg^{2+} (mg/dL)	1.8	2.1	1.7	1.8-2.5
TSH (μIU/mL)	0.24	0.12	2.13	0.27-4.20
T_3 (ng/dL)	55	58	51	80-200
Free T_4 (ng/dL)	1.07	1.06	0.53	0.93-1.70
iPTH (pg/mL)	26.7	21.4	71.4	14.00-72.00
Arterial blood gases (room air)				
pH	-	7.37	7.32	7.34-7.45
$PaCO_2$ (mmHg)	-	27.9	32.7	32.0-45.0
PaO_2 (mmHg)	-	117	116	75.0-100.0
HCO_3^- (mmol/L)	-	16.1	16.9	20.0-26.0
Fractional excretion of sodium and phosphate				
F_E Na (%)	0.5079	0.2179	0.3754	*
F_E PO4 (%)	4.5139	8.9777	7.5652	✣

* F_E Na less than 1% is in favor of functional or transient acute prerenal kidney injury.

✣ F_EPO_4 should reduce to less than 5% in normal renal response to hypophosphatemia.

BMI, Body mass index; BUN, blood urea nitrogen; Cr, creatinine; CPK, creatinine phosphokinase; F_E Na, fractional excretion of sodium; F_EPO_4, fractional excretion of phosphate; Hct, hematocrit; iPTH, intact parathyroid hormone; Temp, Body temperature; TSH, Thyroid stimulating hormone; T_3, Total triiodothyronine; Free T_4, Free thyroxine; WBC, white blood cells.

Soon after arriving at our emergency department and laboratory testing, the patients were given fluids consisting of normal saline and dextrose in water. Half a liter was given in the first 2 h and 1.5 liters in the first 6 h. During the next three days, the parenteral fluids (2-4 L/day) were mixtures of sodium and potassium phosphate salts at 13-18 mmol elemental phosphorus; 300-960 mmol glucose; 200-320 mmol sodium; 51-91 mmol potassium; 1.5-2.0 mmol magnesium; and 1.4-1.8 mmol calcium. Approximately 48 h after admission, follow-up laboratory data were: CPK, 245-473 IU/L; PO_4^{3-}, 1.7-2.7 mg/dL and HCO_3^-, 19.8-26.0 mmol/L. All three patients experienced 24-48 h diuretic phase during recovery from acute renal injury, with an increase in urine output to 234-300 mL/h. This occurred within 24 h from the initiation of

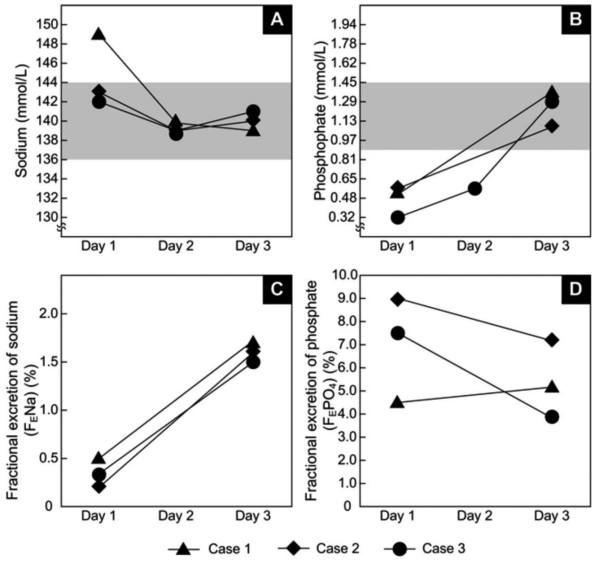

Fig.1. Changes in serum levels (A and B) and fractional excretion (C and D) of sodium and phosphate in three survivors of prolonged seawater immersion during three days following initiation of medical treatment (Gray bars indicate normal ranges).

nutritional support. After 80 h, acute renal injury, electrolyte disorders and rhabdomyolysis had remitted (Figure 1).

We controlled the infections by empirical use of broad spectrum antibiotics (amoxicillin/clavulanate at doses of 1.2 grams every 8 h and ciprofloxacin at doses of 400mg every 12 h). One patient (Case 2) experienced progression of ulceration in skin abrasions and underwent debridement and split-thickness skin graft surgery one week later. All three survivors were discharged within two weeks.

Discussion

Hypophosphatemia is prevalent in patients who are malnourished or have alcoholism, renal failure, chronic obstructive pulmonary disease, or diabetic ketoacidosis[1-3]. Scuba divers rarely experience hypophosphatemia at initial presentation following prolonged immersion in the sea.

Hypothermia is the most common in patients after prolonged cold water immersion[4-7]. In our three patients, hypothermia-related hypoperfusion of muscle tissue, strenuous exertion to remain afloat, and severe hypophosphatemia likely contributed to rhabdomyolysis[1-3,7,8]. Their severe dehydration, rhabdomyolysis, and unavoidable ingestion of saltwater contributed to acute renal injury[1-3,7-11]. The initial low fractional excretion of sodium that we observed indicated prerenal kidney injury[12]. The presence of low T_3 syndrome suggested that this was probably a transient response[13,14].

The three survivors that we treated were susceptible to hypophosphatemia because of their decreased phosphate intake, cellular redistribution of phosphate and increased renal excretion. Their decreased phosphate intake was a consequence of prolonged starvation. Factors that contributed to cellular redistribution of phosphate included rewarming, administration of carbohydrates, and correction of metabolic acidosis following rescue[1-3,15]. Extracelluar hypophosphatemia can result from increased cellular uptake of phosphate and increased insulin secretion during refeeding[1-3]. During rewarming and correction of metabolic acidosis, glycolysis and accelerated metabolic processes can cause phosphorus to move from the extracellular fluid to intracellular spaces, especially in the liver and muscle tissue[3,15].

The increased fractional excretion of phosphate (F_EPO_4) that we observed indicated impaired renal phosphate reabsorption during early recovery from acute renal tubular injury[9-11,16]. Hyperphosphaturia may also have resulted from aggressive extracellular fluid volume expansion, a surge of adrenal hormone, or chronic metabolic acidosis[17-20].

Severe hypophosphatemia can contribute to respiratory distress, rhabdomyolysis, hemolysis, left ventricular dysfunction, or critical dysrythmia [1-3]. At serum phosphate levels of 0.5-0.8 mmol/L (1.5-2.5 mg/dL), respiratory failure and cardiac dysfunction

may develop because of impaired function of smooth and skeletal muscle tissue[1-3]. At serum phosphate level of less than 0.25 mmol/L (< 0.8 mg/dL), seizure, coma, and paralysis can result[1-3]. Acute progressive hypophosphatemia [< 0.7 mmol/L (< 2 mg/dL)] can also contribute to rhabdomyolysis[1-3].

Severe hypophosphatemia [< 0.7 mmol/L (< 2 mg/dL)] is a critical electrolyte abnormality that requires aggressive correction[1-3]. Treatment typically involves administration of intravenous phosphate with neutral mixtures of sodium and potassium phosphate salts at initial doses of 0.2-0.8 mmol/kg (10-50 mmol) of elemental phosphorus over the course of 6 h[1-3]. Less severe hypophosphatemia [0.5-0.8 mmol/L (1.5-2.5 mg/dL)] can be treated with 750-2,000 mg/day oral phosphate, with doses divided to avoid gastrointestinal side effects[1-3].

Conclusion

These three cases demonstrate the occurrence of severe hypophosphatemia in scuba divers after prolonged immersion in the sea and their recovery following medical support. Aggressive managements can prevent seriously irreversible complications. Correction of life-threatening electrolyte imbalances and severe metabolic derangements can be life-saving and improve the prognosis for these patients.

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長期浸泡海水之後的三個低血磷症病例

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摘　要

嚴重的低血磷症是一個有潛在性致命危險的電解質不平衡疾病。很少有文獻發表關於長時間在大海中浸泡後的倖存者發生的嚴重電解質不平衡疾病。我們發表了三位在南台灣的大海中迷失的潛水者。在經歷了四十小時的掙扎，他們在距離本來的潛水地點約一百公里外的海上獲救。低體溫、橫紋肌溶解症、嚴重的脫水、急性腎損傷、嚴重的低血磷症、代謝性酸中毒及低三碘甲素症是最初的疾病表現。可能造成低血磷症的原因包括了磷的攝取不足、細胞內外的重新分佈及腎臟排出磷的增加。長時間的飢餓造成了磷的攝取不足。醫療因素包括碳水化合物的補充、低體溫的回溫及代謝性酸中毒的矯正等造成了細胞內外磷的重新分佈。增加腎臟排出磷的原因包括有急性腎損傷的恢復期的腎小管對於磷的再吸收功能不足，積極的補充體液治療造成細胞外液容積擴張，壓力性荷爾蒙的大量分泌和病人本身的代謝性酸中毒等。積極的治療可以避免嚴重不可逆的併發症。合併矯正危及生命的電解質不平衡和嚴重的代謝異常是保護生命的措施，並且可以改善這些病患的預後。