

# Severe hypophosphataemia during binge eating in anorexia nervosa

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## Abstract

**A patient presented with severe hypophosphataemia that had been precipitated during binge eating. It was corrected by restricting the binges, and by hyperalimentation through a duodenal tube together with intravenous supplementation with sodium phosphate for a short period. Phosphate concentrations should be monitored in patients with severe anorexia complicated by bulimic episodes.**

A low serum phosphate concentration is a complication that has been recorded in several conditions, including hyperalimentation by intravenous fluids deficient in phosphate,<sup>1 2</sup> alcohol withdrawal, diabetic ketoacidosis, recovery after extensive burns, and refeeding of malnourished prisoners of war. In anorexic patients hypophosphataemia has been noted during both intravenous hyperalimentation and oral refeeding (when the patients were starved but using laxatives). In some anorexic patients hypophosphataemia may have played a part in the deaths of patients who were treated with intravenous hyperalimentation without phosphate supplementation.

## Case report

A white girl aged 16 years old, 160 cm in height, had started dieting at the age of 11 years, and was admitted to hospital several times. Her admission to our adolescent's ward (weighing 26 kg) was her sixth admission. She had eaten practically nothing except for some unsweetened biscuits, white low fat cheese, and vegetables, and drunk coffee with saccharin. She considered herself fat and wished to weigh 23 kg. She also said that she was using large quantities of laxatives.

On admission she was emaciated and severely cachectic, and complained of feeling cold. Her blood pressure was 80/45 mm Hg and did not change when she stood up, her pulse rate was 50-64/minute and temperature 36°C. Her skin was dry and scaly. Initial biochemical values were within the reference range (see table). Urinalysis gave normal results.

During the third week in hospital she started binge eating of large quantities of food from every possible source. Her phosphate concentrations dropped abruptly to 0.13, 0.16, and 0.26 mmol/l consecutively. She was placed on a 7.52 MJ diet comprising three meals and three snacks. Her eagerness to binge forced us to restrain her freedom, to feed her through a duodenal tube, and give her sodium phosphate intra-

venously. Even after she had been tied to her bed, she persuaded her room mates to bring her food, and even succeeded in pulling out the nasal tube and sucking it directly. Her extreme aggression necessitated antipsychotic drug treatment. She gradually became calmer and we were able to start oral feeding. Her weight was restored and she was discharged from our ward weighing 39 kg.

Follow up consultations took place twice a week. All her biochemical values including phosphorous remain within the reference ranges. Because of her anorexia, however, we are pessimistic about her prognosis.

## Discussion

Phosphorus is essential to the body's energy and storing mechanisms,<sup>1-4</sup> and it plays a part in carbohydrate, fat, and protein metabolism. In addition, it is necessary for the structure of the phospholipids composing cell membranes and intracellular organelles of muscle and nerve tissue.<sup>5</sup>

Hypophosphataemia can produce the clinical signs of ATP deficiency or cellular hypoxia affecting the neuromuscular, cardiopulmonary, haematological, gastrointestinal, renal, endocrinological, and skeletal systems.<sup>6 7</sup> It is a rare clinical entity. The daily requirement is 800-1000 mg of elemental phosphorus. Dietary sources include eggs, milk, poultry, cheese, nuts, and vegetables. The kidney reabsorbs up to 90% of phosphorus and stool losses are small, being 100-200 mg/day.<sup>3</sup> This patient by avoiding practically all food while resorting to laxative abuse reduced her phosphate intake and increased her faecal excretion.

Serum phosphate concentrations reflect only a small proportion of total body phosphorus because 99% of phosphate is intracellular.<sup>8</sup> Although within the reference range on admission, her serum phosphate concentration apparently fell precipitously after her binges, thus showing that her total body phosphorus was depleted by her fasting and laxative abuse.

The neurons, red blood cells, and cardiac muscle are particularly vulnerable to ATP depletion.<sup>6 9</sup> Presumably the phosphate supplying these tissues from the serum was just adequate during the periods of nutritional deprivation and the catabolic state. The severe bulimic phase changed her body metabolism from a catabolic to an anabolic state, creating an abrupt demand by her liver and muscles for more phosphate for the production of the phosphorylated intermediates required by glycolysis, glycogen formation, and protein synthesis.<sup>1</sup>

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## Results of biochemical and haematological investigations

Sodium	142 mmol/l
Chloride	100 mmol/l
Potassium	4.7 mmol/l
Blood urea nitrogen	16.4 mmol/l
Creatinine	62 µmol/l
Total protein	67 g/l
Glucose	6.0 mmol/l
Aspartate aminotransferase activity	18.8 U/l
Calcium	2.37 mmol/l
Phosphate	1.49 mmol/l
Haemoglobin	127 g/l
Packed cell volume	0.40 with $12 \times 10^{-3}$ reticulocytes
Erythrocyte sedimentation rate	11 mm in first hour

Phosphate then shifted rapidly from her serum into her muscle and liver.<sup>1-4</sup> The drop in serum phosphate threatened to deprive her nervous system, red blood cells, and heart of their phosphate requirement, creating a metabolic emergency.

Hypophosphataemia is a well known cause of death in anorexic patients who are receiving phosphate deficient intravenous hyperalimentation,<sup>2</sup> and has also been reported in a patient who had been refed orally.<sup>1</sup> Normal serum concentrations do not exclude the existence of dangerous total body phosphate depletion. We suggest that all bingeing anorexic patients should have their serum phosphate concentrations monitored and adequate phosphate supplementation should be given. If serum phosphorus concentrations start to drop, the patient should be restrained in bed by all legitimate means to

stop her having binges and subsequently misusing laxatives and inducing vomiting.

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#### Measles and vitamin A

Firmly in Michael Caine's 'not many people know that' category is the fact that nearly 60 years ago a paper in the *British Medical Journal* pointed to vitamin A having a protective effect in measles.<sup>1</sup> In 1987 the World Health Organisation recommended vitamin A supplements for children with measles in areas where vitamin A deficiency is rife and where measles has a high mortality.<sup>2</sup>

A recent report from Cape Town (Hussey and Klein, *New England Journal of Medicine* 1990;323:160-4) confirms the beneficial effect of vitamin A in severe measles. Altogether 189 children entered a randomised controlled trial receiving either vitamin A 400 000 IU or placebo orally. Mortality was 10.3% in the placebo group and 2.2% in the vitamin A group. In the supplemented children the duration of pneumonia was halved and that of diarrhoea reduced by a third. Complications such as post measles croup and herpes stomatitis were significantly reduced.

Measles kills about 2 million children a year so this work is clearly of immense importance especially in developing countries, but low serum concentrations of vitamin A are known to occur during measles even in populations not known to be deficient in the vitamin and the authors therefore advocate giving the supplement to any child who has severe measles.

ARCHIVIST

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