Introduction

Severe hypophosphataemia (< 0.3 mEq/l) is rare and occurs predominantly in the setting of chronic alcoholism or total parenteral nutrition [1]. In addition to rhabdomyolysis, neurological deficits are the most striking clinical feature. Polyradiculitis with progressive paresis is frequent [2]. Confusional states, hallucinations, epileptic seizures and coma occasionally occur due to central nervous system involvement [3–5]. Reports of brain lesions in extreme hypophosphataemia are lacking. We describe a patient in whom serial CT and MRI showed reversible lesions in basal ganglia and occipital lobes associated with profound changes in serum phosphate.

Case report

A 38-year-old woman with a history of chronic alcohol abuse presented in an obtunded state following 3 weeks intermittent nausea and vomiting. Basal pneumonia, pancreatitis and alcoholic ketoacidosis were diagnosed on admission. Serum sodium was 133 mEq/l and potassium 2.8 mEq/l. After transfer to the intensive care unit, antibiotic therapy, fluid resuscitation, and total parenteral nutrition were initiated, and thiamine and cobalamin but not hypertonic saline were given.

Four days after admission the patient became sleepy and confused and developed a flaccid quadriparesis with absent deep tendon reflexes. The plantar responses were flexor. Respiratory failure necessitated intubation and assisted ventilation. CT revealed striking bilateral low density in the basal ganglia, thalamus and occipital lobes. After adequate substitution of phosphate the lesions grossly resolved and the patient recovered. This case is the first to demonstrate that profound changes of serum phosphate may be associated with reversible brain lesions.
Discussion

Central nervous system (CNS) involvement is rare in hypophosphataemia, and the pathogenesis of brain injury is not fully understood [1, 3, 5]. Phosphate depletion may lead to a fall in the brain content of high-energy compounds and to increased oxygen binding by haemoglobin [6]. It has been suggested that the decrease in oxygen availability might result in tissue hypoxia and subsequent dysfunction [2]. To the best of our knowledge, this case is the first to show reversible brain lesions associated with phosphate depletion in humans. The resolution with treatment strongly suggests a causal relation between phosphate depletion and the CNS lesions.