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 ketoadidosis 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 and particularly the thalamus. The cortical and subcortical regions of both occipital lobes were also affected (Fig. 1). The lesions did not enhance with contrast medium. Volume loss, particularly of infratentorial structures, was noted. Cerebrospinal fluid examination was unremarkable. The respiratory weakness and severe flaccid quadriparesis were interpreted as consistent with acute polyradiculitis or neuropathy due to hypophosphataemia. The serum showed extremely low phosphate (0.02 mEq/l; normal range 0.9–1.2 mEq/l), slight hyponatraemia and hypokalaemia (132 and 4.1 mEq/l, respectively). Under substitution therapy, serum phosphate rose steadily and was within the normal range on day 11. Concurrently the patient’s state of consciousness became normal and she gradually regained the power in her arms and legs. On day 12 she could be extubated.

CT on the 8th and the 13th day of admission revealed marked decrease in the size of the lesions (Fig. 2). The low density in the occipital lobes had completely resolved.

On day 24 MRI on a 1.5-T unit still showed several small high-signal lesions in the rostral thalamus on T2-weighted images (Fig. 3). In addition, a large, round lesion in the basis pontis was detected, with high signal on T2- and slightly low signal on T1-weighted images. The abnormal foci did not enhance with contrast medium. On day 34 MRI did not show any change. At this time the tetraparesis had resolved completely, but mild ataxia and dysarthria were present.
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.

Fig. 1 Initial CT (4th hospital day) shows bilateral low density in basal ganglia and occipital lobes. At this time the patient’s serum phosphate was extremely low (0.02 mEq/l)

Fig. 2 CT (8th hospital day) demonstrating partial resolution of basal ganglia and occipital lesions following phosphate substitution