Case Report

A chronic glue sniffer with hyperchloraemia metabolic acidosis, rhabdomyolysis, irreversible quadriplegia, central pontine myelinolysis, and hypothyroidism

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Key words: central pontine myelinolysis; hypothyroidism; hyperchloraemic metabolic acidosis; toluene

Introduction

Toluene is an aromatic hydrocarbon with widespread industrial use as an organic solvent. Chronic or acute exposure is known to cause acid–base and electrolyte disorders [1], and be toxic to the nervous system, and the haematopoietic system [2-5]. Three major forms of clinical presentation can be encountered [5]: neuropsychiatric, gastrointestinal, and muscular disorders. In most patients, these manifestations tend to resolve within a few days after abstinence from sniffing and correction of electrolyte abnormalities. But toluene intoxication may also cause severe and persistent neurological damage.

Case report

A 29-year-old unemployed Chinese man was admitted to the hospital for severe muscular weakness. The patient was conscious and lucid. He admitted being a glue sniffer for at least 8 years, but denied abuse of any other drugs or medications. In fact, 5 months earlier he had been admitted for the same problem. He had then recovered without sequelae from an episode of hypokalaemic paralysis and tubular acidosis. He was lost of follow-up but continued glue sniffing.

On admission the physical examination revealed an acute ill-looking man in respiratory distress. His temperature was 36°C, the pulse rate 106/min, the blood pressure was 86/50 mmHg, and the respiratory rate 34/min. The cranial nerves were grossly intact. The muscle strength of his four limbs was severely reduced. The deep tendon reflexes were normal except in the right leg. The Babinski sign was negative on both sides. The arterial blood pH was 7.064, PaCO2 30.7 mmHg, HCO3 8.3 mEq/L, PaO2 76.7 mmHg when the patient was breathing room air. The serum creatinine was 1.5, urea 15, calcium 8.8 and phosphate 0.5 mg/dl, sodium 132, potassium 2, and chloride 113 mEq/L. The urine pH was 6.0. The creatinine kinase was 27 890 U/L (100% MM isof orm). The diagnosis of hyperchloraemic metabolic acidosis and hypokalaemic paresis with rhabdomyolysis was made and the patient was immediately supplemented with potassium chloride and alkalinizing salts. Renal function, serum potassium and phosphate concentration, and the acid–base status normalized within 5 days, however, without amelioration of muscle strength, whereas during this period his mental status deteriorated. After another 4 days the patient regained consciousness, but bilateral cutaneous anaesthesia was noted at all levels below C3 and quadriplegia persisted.

Computerized tomography of the brain revealed hypodensity lesions of the pontine area bilaterally (Figure 1). A brain stem auditory evoked potential study performed 5 weeks after admission was indicative of bilateral abnormalities at the level of the pons, whereas the somatosensory evoked potential study revealed abnormalities below the cervical level. Motor and sensory nerve velocities as well as the results of classic electromyography revealed severe neuropathy with active denervation.

Initially hypothyroidism was present: T₄ 2.81 mg/dl, T₃ < 10 mg/dl, free T₄ 0.31 mg/dl, TSH 0.70 mU/l. Two weeks later, however, the thyroid function tests were normal, with a normal TRH stimulating test.

After 12 months of follow-up quadriplegia is still present. Communication with other persons is possible only with eye blinking and head shaking.

Discussion

This patient presents some distinctive clinical manifestations of inhalational toluene intoxication.
Hyperchloraemic metabolic acidosis, hypokalaemia and acute renal insufficiency due to extracellular volume were salient presenting features. It has been argued that the presence of hyperchloraemic metabolic acidosis in toluene intoxication is indicative of distal tubular acidosis [1]. However, as reviewed by Carlisle et al. [6] overproduction of hippuric acid, a major metabolite of toluene, plays an important role in the genesis of metabolic acidosis. As tubular secretion of the hippurate anion result in rapid excretion in the urine, this metabolic acidosis will not be accompanied by an elevated anion gap in the plasma unless advanced renal failure is present. Furthermore, reviewing the literature these authors concluded that in most patients urinary excretion of ammonium is not low [6]. Low urinary ammonium excretion is a prerequisite for the diagnosis of distal tubular acidosis. However, as part of the hippurate anion in the urine is not matched by ammonium, urinary loss of sodium and potassium may be enhanced. As in our patient this may have resulted in hypokalaemia and rhabdomyolysis as well as in sodium loss, extracellular volume contraction and impaired renal function. Normally these disturbances improve rapidly with repletion of potassium and extracellular volume.

The first patient with permanent neurological sequelae from long-term solvent sniffing was reported by Grabski [7]. Toluene abuse has been linked to cerebellar degeneration, corticobulbar and corticospinal damage, cerebral and brainstem atrophy with abnormalities of the EEG and of brainstem auditory evoked potentials [8]. It is also associated with encephalopathy, peripheral and optic neuropathies, sensorineural hearing loss, status epilepticus, myopathy, choreothetotic movement disorders, myoclonus and schizophrenia-like psychosis [8]. However, intoxication by toluene or other aromatic hydrocarbons seems seldom to be the cause of peripheral neuropathy [7]. In our patient a combination of a bilateral pontine lesions and a myelopathy with sensory deficit below the level of C3 was present. As no other aetiologies could be detected, we believe that toluene toxicity was the cause of this distinct clinical picture, which to our knowledge has not been described previously.

Initially the patient presented with low T3, T4, free T4 and TSH level. Although, due to the critical status of the patient, the TRH stimulating test was not performed, the data are in agreement with a secondary form of hypothyroidism due to a lesion of either pituitary gland or the hypothalamus. This lesion was apparently transient, as repeated tests done 3 weeks later, after admission, showed normal results.

References


Received for publication: 29.8.95
Accepted in revised form: 6.5.96