Severe, self-limiting lactic acidosis and rhabdomyolysis accompanying convulsions

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Summary: A 26 year old man with no previous history of convulsions presented in status epilepticus and severe lactic acidosis. He regained consciousness and the acidosis resolved after several hours of conservative management without intravenous bicarbonate, but he developed severe myalgia associated with marked elevation of creatine kinase and moderate raised plasma creatinine levels which resolved spontaneously after 3 days.

Severe lactic acidosis and rhabdomyolysis may accompany status epilepticus, although they appear to be self-limiting without important sequelae.

Introduction

Lactic acidosis is generally regarded as a serious disorder, resulting from a variety of causes. We report on a man who developed severe lactic acidosis and rhabdomyolysis following convulsions, in whom there was a spontaneous recovery.

Case report

A 26 year old man, who had fractured his skull a year previously, presented in status epilepticus for 30 minutes, requiring treatment with intravenous diazepam. He had allegedly consumed his habitual 12 units of alcohol the previous night. On initial assessment, he was deeply comatose and his pupils were dilated but fully responsive to light. Apart from a tachycardia, vital signs were stable, and there was no focal neurological deficit. Arterial blood gases on air showed a marked metabolic acidosis (pH 6.8, PO_2 90 mmHg, PCO_2 42 mmHg, bicarbonate 7 mmol/l, base excess –28 mmol/l), and the anion gap was 38 mmol/l (sodium 149 mmol/l, potassium 3.4 mmol/l, chloride 107 mmol/l). Transient elevations in blood glucose (11.2 mmol/l) and white cell count ($21.6 \times 10^9/l$) were recorded. Plasma lactate was 23.0 mmol/l (reference range 0.4–1.4 mmol/l) and creatinine was elevated at 200 μ mol/l (reference range 60–120 μ mol/l). No alcohol, salicylate, or other toxins were detectable in serum or urine, and a computed tomographic scan of the head revealed atrophy of the frontal lobes.

He was managed conservatively with oxygen and intravenous saline and recovered consciousness spontaneously 5 hours later, with an accompanying improvement in biochemistry (Figure 1). Thereafter he complained of myalgia and muscle weakness, particularly in his lower limbs, which lasted for 48 hours. Creatine kinase levels were markedly elevated at 29,430 IU/l (reference range 24–195 IU/l), but fell the following day to 2000 IU/l, when lactate dehydrogenase levels were slightly elevated at 650 IU/l (reference range 100–225 IU/l). Serum creatinine levels remained elevated at this stage (214 μ mol/l), but subsequently fell to within the normal range (Figure 1). After 3 days the patient was free of symptoms and discharged. A subsequent electroencephalogram did not reveal any diagnostic features, and 6 months later, he had not experienced any further seizures. A diagnosis was made of post-traumatic epilepsy associated with lactic acidosis and rhabdomyolysis.

Discussion

Lactic acidosis and rhabdomyolysis are reported, although underdiagnosed, accompaniments of generalized convulsions.^{1,2} The degree of acidosis and muscle damage in the current report, however, is the greatest yet recorded in this situation and the former could have been interpreted as life threatening.¹ This might have led to inappropriate therapy with bicarbonate,³ since the acidosis resolved following supportive therapy with oxygen, intravenous saline and anticonvulsants. The lack of respiratory compensa-

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Figure 1 Serial changes in lactic acid, bicarbonate, pH and creatinine following admission with convulsions and severe lactic acidosis.

tion for the metabolic acidosis in the present case was also reported by Orringer *et al.*¹ following a single grand-mal seizure of 30 to 60 seconds duration, and might be the consequence of suppression of the respiratory centre after the convulsion, although the use of diazepam in our patient could have compounded this effect. The low level of serum potassium in the current case is an important hallmark of metabolic acidosis secondary to lactic acidosis. Alcohol intoxication was suspected in this case but not proven, although such severe lactic acidosis is not in fact expected with ethanol abuse.⁴ On the other hand prior alcohol consumption may have been implicated in the rhabdomyolysis, despite the absence of circulating ethanol at the time of admission.⁵

The mechanism of lactic acidosis following seizure is thought to be the consequence of local muscle hypoxia and increased production of pyruvic acid, which is normally in equilibrium with lactic acid. Resolution is thought to occur predominantly due to aerobic metabolism leading to reduced production of lactic acid and enhanced gluconeogenesis, although renal clearance may also be a factor.³ In this regard, the elevation of plasma creatinine and creatine kinase may reflect transient rhabdomyolysis-induced renal impairment,^{5,6} which could have delayed the return of serum lactic acid levels to normal for more than 2 days.

It would appear that whilst severe lactic acidosis and extensive muscle damage can accompany status epilepticus, these disturbances may be self-limiting and do not require specific corrective therapy.

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