Therapeutic magnesium for eclampsia: An unusual cause for antepartum flaccid paralysis

Sir,
We describe a patient who developed profound transient weakness with parenteral magnesium therapy for eclampsia.

This 23-year-old second gravida with amenorrhea of five months duration, presented with headache and vomiting for four days followed by diplopia, visual impairment, altered sensorium and generalized tonic-clonic seizures. She was noted to have hypertension (240/130 mmHg) and was initially given nifedipine. Later, Pritchard regime was initiated for eclampsia and pregnancy was terminated using prostaglandin E2. She developed rapidly progressive quadriaparesis and was referred to us. She had pallor, pedal edema and BP of 120/70 mmHg. She had hypotonia, mild neck flexor weakness and profound weakness of all the four limbs (MRC grades 0 to 1/5, proximally and distally). Oculomotor and facial muscles were spared and nonfatigable. All muscle stretch reflexes were absent and plantar response was flexor. Sensory functions were normal.

She had hypermagnesemia: 11.6 meq/L (normal - 2.5 meq/L) and renal failure: blood urea - 64 mg/dl (normal - 45 mg/dl),
serum creatinine - 4.6 mg/dl (normal - 0.9 mg/dl). Other biochemical parameters and electrocardiogram were normal. CT brain showed bilateral parieto-occipital hypodensities.

Nerve conduction studies (NCS) revealed reduced amplitude of compound muscle action potentials (CMAPs) from stimulation of the right median, ulnar and common peroneal nerves [Table 1]. However, sensory conductions were normal. On repetitive nerve stimulation (RNS) at 2Hz, a decremental response was obtained from orbicularis oculi (13%), trapezius (30%) and abductor digitii minimi (23%). There was no incremental response at 20Hz stimulation.

A diagnosis of acute flaccid paralysis due to neuromuscular junction defect, secondary to use of magnesium sulfate for eclampsia was considered. She made rapid recovery following dialysis over the next one week and later became ambulant and independent. Nifedipine was used to control BP subsequently. After six months, her renal functions and NCS were normal [Table 1] and RNS test did not reveal any decrement.

Our patient received Pritchard’s regime. Magnesium toxicity is rare and is closely linked to its plasma concentration; hypoactive muscle stretch reflexes: 4-7 meq/L, ECG changes (prolonged PQ interval and widening of QRS): 5-10 meq/L, absent muscle stretch reflexes: 8-10 meq/L, respiratory paralysis: 10-15 meq/L and cardiac arrest: 25 meq/L. Careful monitoring of muscle stretch reflexes, respiratory rate and urine output and serum magnesium could identify early toxicity.

Magnesium-induced “pharmacological” myasthenic syndrome is attributed to neuromuscular abnormality, decreased motor endplate sensitivity to acetylcholine and reduced excitability of the muscle fiber membrane. Our patient had reversible low-amplitude CMAPs and decremental response suggesting involvement of both the pre- and postsynaptic region. Electrophysiological features resolved pari passu with clinical improvement. The patient did not manifest myasthenic symptoms in the past as well as during follow-up. Magnesium toxicity is usually observed following parenteral magnesium in patients with renal failure. Our patient incidentally also had reversible visual impairment and bilateral parieto-occipital hypodensities in the CT scan.

This case illustrates the need for close monitoring of patients receiving parenteral magnesium, especially those with renal impairment. Hypermagnesemia should be considered in the differential diagnosis of acute flaccid paralysis, particularly in the setting of management of eclampsia.

Table 1: Amplitude of compound muscle action potentials - during serial nerve conduction studies

<table>
<thead>
<tr>
<th></th>
<th>Median (normal: &gt;6.0 mV)</th>
<th>Ulnar (normal: &gt;6.0 mV)</th>
<th>Common peroneal (normal: &gt;3.0 mV)</th>
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<tr>
<td>May 2004</td>
<td>0.57</td>
<td>0.24</td>
<td>0.22</td>
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<td>November 2004</td>
<td>12.2</td>
<td>9.5</td>
<td>4.92</td>
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</tbody>
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References