

## **An atypical case of two instances of mepivacaine toxicity**

Sir,

We report an interesting case of two atypical instances of mepivacaine toxicity in the same patient, characterized by a pronounced cardiovascular system excitement that caused, in the first instance, an ischemic stroke that obscured the diagnosis of local anesthetic intoxication leading us to repeat the error.

A 76-year-old, 70 kg woman, ASA physical status III, affected by vascular cognitive impairment and chronic hypertension was referred to our Emergency Department after an accidental fall. A knee X-ray showed a left patella fracture; her blood pressure was 195/105 mmHg. The patient was hospitalized; 100 mg metoprolol and 0.4 ml nadroparin calcium were added to the therapy. Four days later the patient underwent patella synthesis. Double peripheral nerve blockade of the sciatic and femoral nerves by 600 mg (8.5 mg/kg) of plain mepivacaine was placed. After 20 min, the heart rate and blood pressure suddenly increased to 220/130 mmHg. The patient became unresponsive to verbal commands; no tonic-clonic activity was noted but physical examination revealed right hemiparesis.

An urgent brain computed tomography (CT) revealed no intracranial hemorrhage so, a diagnosis of acute ischemic stroke was made and intravenous thrombolysis was instituted. At 7 h after the attack, the patient became alert and oriented. Physical examination revealed right hemiparesis regression; a repeated brain CT scan revealed signs of ischemic edema localized to the left frontoparietal apical regions. The patient was re-scheduled for surgery six days later. As in the first instance, femoral and sciatic nerve block was induced with 600 mg of plain mepivacaine. Approximately 20 min later, the heart rate suddenly increased to 120 bpm and the blood pressure to 220/120 mm Hg. The patient became confused, with slurred speech and slow response to simple verbal commands, but remained conscious. Neither tonic-clonic activity nor major neurologic deficits were noted. Cardiovascular parameters and neurological signs normalized within 1 h after the incident.

According to available data, plain mepivacaine seems quite safe even at doses exceeding the maximum recommended.<sup>[1]</sup> Although the close temporal relationship between the onset of stroke signs and symptoms and the anesthetic procedure had prompted suspicions, we initially ascribed the stroke to a perioperative event and we attributed them to the unconsciousness and the highly elevated blood pressure.<sup>[2]</sup> It was only after the second instance that we realized we faced two episodes of mepivacaine toxicity. In retrospect, we speculate that the ischemic stroke was a consequence of a hypertensive crisis due to systemic absorption of a potential mepivacaine toxic dose.<sup>[3]</sup>

Local anesthetic intoxication, mepivacaine in particular, can increase both, blood pressure and heart rate as consequence of sympathetic nervous system activation.<sup>[4]</sup> In this setting, metoprolol could have behaved as a non-selective  $\beta$ -adrenergic blockade resulting in unopposed  $\alpha$ -adrenergic activity producing vasoconstriction and increase in systemic vascular resistances.<sup>[5]</sup> Pre-existing uncontrolled hypertension could have acted as a superimposed factor and all mechanisms together might explain the rapid onset of exceptionally high blood pressure. Unfortunately, serum mepivacaine concentrations, which could have provided clearer answers about the possible mepivacaine overdosage, were not measured in our patient.

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