THE USE OF MAGNESIUM IN SUSPECTED LEVOBUPIVACAINE TOXICITY: A CASE REPORT

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A 60-year-old kyphoscoliotic lady weighing 40 kg was scheduled for a left total hip replacement. Of note in her history were 1) slow atrial fibrillation of unkown etiology and not treated, 2) a history of gastroesophageal reflux for which she was taking a proton pump inhibitor, and 3) limitation of exercise probably due to her severe arthritis.

Physical Examination

A thin, pleasant, well-adjusted lady. The only abnormal findings were an irregular pulse (rate 80 BPM) and painful limitation of leg movement. The BP was 120/90. ECG revealed artrial fibrillation with a slow ventricular response (80 BPM).

Anaesthesia

Anaesthesia was induced with 100 mg of propofol followed by 60 mg of succinylcholine. Intubation was performed as part of a rapid sequence induction and an oral endotracheal tube lubricated with 4% ligmocaine was passed without difficulty. On recovery from the effects of the succinylcholine she was allowed to breathe spontaneously, turned on her side and Anaesthesia maintained with nitrous oxide (60%), oxygen (39%), and isoflurane (1-1.5%). The anaesthetic was entirely uneventful and the operation was completed in 1 1/2 hours. Blood loss was approximately 850 cc which was replaced by 1500 ml of lactated ringers and 500 ml of a gelatin solution. At the end of the procedure, before the patient was awake, it was decided to perform a psoas block to facilitate postoperative analgesia. The nerve plexus in the psoas compartment was located using a nerve stimulator and on obtaining the appropriate response 35 cc of 0.4% levobupivacaine was injected. There were no immediate complications. The patients was extubated, and allowed to awaken in the OR. Analgesia was obtained and the patient returned to the PACU in satisfactory condition.

At his stage all vital signs were within normal limits. Within 15 minutes the anaesthesiologist was called urgently to the PACU where the patient was found to be disoriented, restless, hypertensive, and tachycardic. The heart rate was approximately 160 bpm, (the exact nature of the rhythm could not be determined). The blood pressure was 180/110. A tentative diagnosis of L-bupivacaine toxicity was made and the following treatment was instituted.

Lignocaine 50 mg was given intravenously with no effect. Ten mg of esmolol was injected which also failed to produce any improvement. One gram of magnesium was then administered with an immediate effect on both the cerebral status and the cardiovascular parameters. Blood pressure decreased to 125/85, the heart rate returned to 110/per minute, and the patient's state of consciousness was normalised. The patient's subsequent recovery was uneventful. Analgesia lasted approximately 12 hours.

Discussion

The anaesthetic technique employed was simple and uncomplicated. The dose of levobupivacaine administered was at the upper level of the accepted dose i.e. 140 mg. For this patient, a thin 40 kg kyphoscoliotic lady, the dose was probably excessive. The reaction

was likely due to vascular absorption - although some drug entering intravenously could not be excluded. The diagnosis of levobupivaciane toxicity was based upon work currently being done and submitted for publication in the Anaesthesia laboratory at University of Cape Town where comparative studies on the toxicity on bupivacaine vs levobupivacaine were being performed (using rates). In these animals the response was found to be hypertension, tachycardia, and disorientation of the animal. Magnesium ameliorated this reaction. In the absence of being able to make a definitive diagnosis of the nature of the tachycardia it was decided initially to use lidocaine. Some authorities had advocated its use in this situation on the basis of competitive inhibition in the Fast channels. This is no longer believed to be appropriate. Esmolo was used empirically in the event that the tachycardia was ventricular in origin. Magnesium was administered on the basis of the lab findings described above when it was found that the drug was effective in reversing L-bupivaciane toxicity. Magnesium has been used in a multiplicity of other clinical situations. This may be yet another indication for its use.

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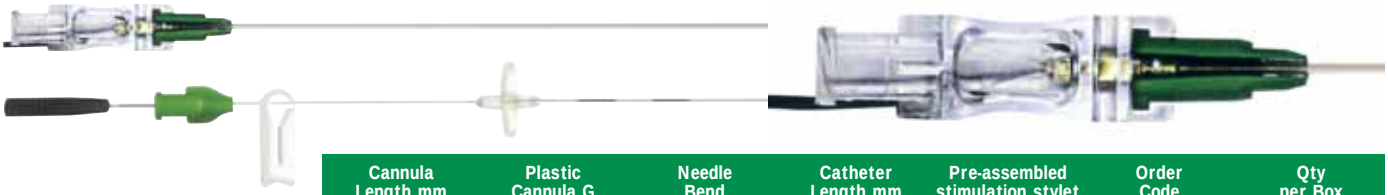

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