Delayed recovery from anaesthesia due to acute phenytoin therapy

Sir,

Spontaneous recovery from neuromuscular block occurs through redistribution, buffered diffusion or metabolism of the neuromuscular blocking agent administered.[1] Delayed recovery from anaesthesia is a dilemma for the anaesthesiologist. Acute administration of phenytoin leads to augmentation of the neuromuscular blocking agents thereby prolonging recovery from anaesthesia.[2] Furthermore, greater anaesthetic depth has been reported in patients on oral phenytoin therapy for over a week.[3] On the other

hand, resistance to the neuromuscular blocking action of the nondepolarising neuromuscular blocking agents has been reported in patients chronically administered anticonvulsants including phenytoin.[4] We hereby report a case of delayed recovery from anaesthesia because of prolonged neuromuscular block in a patient on acute phenytoin therapy.

A 50-year-old male weighing 52 kg was scheduled for emergency laparotomy because of acute intestinal obstruction. The patient also had history of sudden loss of consciousness 3 days back in addition to his current problem. His computed tomography (CT) scan revealed haemorrhagic contusions in parietal area and neurosurgeon advised infusion of 8 ampoules (800 mg) of phenytoin in 100 ml of normal saline for the intraoperative period. On examination, patient was conscious and well oriented. Blood pressure was 110/70 mm Hg and pulse was 96/min regular. The haemogram, blood sugar, serum sodium and potassium, renal function tests, liver function tests, coagulation profile, chest X-ray and electrocardiography were within the normal limits. In the operating room standard monitors were attached. Rapid sequence induction was done with fentanyl 100 μg, thiopentone sodium 300 mg and rocuronium 50 mg and trachea was intubated with endotracheal tube of internal diameter 8 mm. Anaesthesia was maintained with isoflurane 1%, oxygen-nitrous oxide. After induction of anaesthesia, infusion of phenytoin (800 mg in 100 ml normal saline) was started slowly. Intraoperative course was uneventful. Surgery lasted for 2 h. Patient didn't recover from neuromuscular blockade post-operatively and he was continued on positive pressure ventilation. Spontaneous efforts were seen after 2 h of end of surgery, which was further confirmed by neuromuscular monitor. After recovery from neuromuscular block, the post-operative course was uneventful.Recovery from anaesthesia can be delayed because of various reasons such as residual neuromuscular blockade, overdosage of benzodiazepenes and opioids, dyselectrolytemia, acid-base disturbances, low serum albumin levels, hypoglycaemia, hyperglycaemia, hypothermia, low levels of pseudocholinesterase, liver disease, renal disease and uraemia.[5] Clue to the solution of this clinical problem is found in a pertinent review of the history, physical examination, and laboratory investigations. Standard dose of fentanyl was given, and benzodiazepines were not used. Serum electrolytes, blood gas analysis, serum albumin and blood sugar were within normal limits. Normothermia was maintained throughout the surgery. Liver disease, renal disease, and uraemia were ruled out on relevant investigations in the present patient. Hence, persisting neuromuscular blockade could be the only reason for delayed recovery in this patient who was confirmed by neuromuscular monitoring.

Prolonged neuromuscular blockade in this patient could be because of acute administration of phenytoin. However, we did not go for the serum phenytoin levels done in this patient, which could have further substantiated our contention. We did not use prophylactic dose of phenytoin and used loading dose of phenytoin in order to ensure therapeutic plasma levels as patient had history of loss of consciousness 3 days prior to present surgery and CT scan revealed haemorrhagic contusions in parietal area. Levati *et al*. in a study on perioperative prophylaxis with phenytoin recommended that the loading dose of phenytoin should be used in such situations.[6] Acute phenytoin therapy prolongs neuromuscular blockade by decreasing the stimulus-induced release of acetylcholine from the motor nerve terminals. Augmentation of the rocuronium induced neuromuscular block by the acutely administered phenytoin has been documented. Spacek *et al*. in a study on 15 patients receiving phenytoin during surgery in a loading dose of 10 mg/kg concluded that phenytoin acutely administered augments the neuromuscular block produced by rocuronium.[2]

One should be aware of delayed recovery from neuromuscular blockers in a patient on acute phenytoin therapy. Supportive mechanical ventilation should be continued till spontaneous efforts are seen. Further, precise assessment of neuromuscular transmission would be beneficial in such a situation.

Teena Bansal, Sarla Hooda, Rajmala Jaiswal, Manish Bansal

Department of Anaesthesiology and Critical Care, Pt. B.D. Sharma University of Health Sciences, Rohtak, Haryana, India

Address for correspondence:
Dr. Teena Bansal,
2/8 FM, Medical Campus, PGIMS,
Rohtak - 124 001, Haryana, India.
E-mail: aggarwalteenu@rediffmail.com

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