Lidocaine Toxicity Treated with Low-dose Propofol

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LOCAL anesthetics cause concentration-dependent central nervous system (CNS) toxicity characterized by excitation and convulsions.1,2 Treating this CNS excitation with anticonvulsants while maintaining a responsive patient presents conflicting goals, because most drugs used to treat the CNS effects of local anesthetics also cause marked sedation. Whether the short-acting anesthetic propofol increases or decreases the seizure threshold and, in subhypnotic doses, treats local anesthetic toxicity is unclear.3,4 We report a patient undergoing carotid endarterectomy who developed restlessness and uncontrollable muscle twitching after receiving lidocaine and who was treated successfully with small doses of propofol.

Case Report

A 74-year-old man, weighing 70 kg, presented with 90% occlusion of the left internal carotid artery for endarterectomy. The surgeon requested monitored anesthesia care. The patient had experienced several episodes of amaurosis fugax of the left eye but was otherwise normal neurologically. He had a long history of hypertension, gout, peptic ulcer disease, and atrial fibrillation. All were well controlled with his medications, which included nifedipine, lisinopril, and sucralfate.

The patient arrived in the operating room alert and oriented. A catheter was inserted into the right radial artery. The blood pressure was 178/80 and the pulse rate 80 and irregular. Midazolam (1 mg) and fentanyl (50 µg) were administered intravenously. The surgeons injected approximately 10 ml 1% lidocaine without epinephrine subcutaneously before incision.

After 30 min, the surgeons had injected a total of 500 mg lidocaine into the wound and around the left carotid artery, which was now fully exposed but not clamped. At this time, the patient became agitated, spoke incoherently, and developed profound shaking of both lower extremities. An additional 1 mg midazolam and 150 µg fentanyl were given intravenously, but they failed to reverse the patient's agitation or shaking. His blood pressure increased to 200/90 and his pulse to 100 bpm, while oxygen saturation remained normal. Two 30-µg boluses of esmolol decreased his blood pressure to 150/60 and pulse rate to 70 but did not affect his agitation, confusion, or muscle tremors.

We suspected lidocaine toxicity and drew a blood sample from the radial artery catheter. Propofol was immediately available and a 20-µg bolus (0.28 mg/kg) was administered intravenously. The patient rapidly ceased the gross shaking of his lower extremities and became responsive to verbal questions. He lay quietly on the surgical table with his eyes closed. After a few minutes, the restlessness and tremors returned but were reversed with an additional 20-µg bolus of propofol. An infusion of propofol was begun at 100 µg·kg\(^{-1}\)·min\(^{-1}\) and gradually reduced to 75 µg·kg\(^{-1}\)·min\(^{-1}\) for the remaining 60 min of the surgical operation. The patient's vital signs remained at his baseline; he lay quietly, sedated but responsive, as the surgery proceeded uneventfully. Carotid stump pressure was 34/27 mmHg. Upon arrival in the recovery room, serum electrolytes and glucose were normal and the patient moved all extremities without asymmetry. The serum lidocaine concentration was reported as 3.5 µg/ml. The patient's postoperative course was uneventful, and he was discharged from the hospital in improved condition.

Discussion

Lidocaine can selectively depress central inhibitory centers, allowing excitatory activities.5 Lidocaine toxicity can present as lightheadedness and dizziness followed by tinnitus, shivering, and muscle twitching.6 As the concentration increases, lidocaine toxicity progresses to generalized convulsions and coma. Pharmacologic treatment of the CNS effects of lidocaine includes barbiturates and benzodiazepines.6 Midazolam is especially effective, whereas ketamine and fentanyl with droperidol are not.7,8 Although anesthesiologists frequently administer lidocaine and propofol together, the effects of propofol on lidocaine toxicity are not clear.

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Propofol produces anesthesia at a dosage of 2.5 mg/kg, whereas smaller doses provide sedation. Whether propofol in clinical dosages reduces epileptogenic activity is not known. Propofol will shorten the duration of electroconvulsive seizures, but may cause opisthotonos and seizures in some patients. At doses causing mild sedation, propofol protects mice against convulsive effects of pentylentetrazol. A recent study showed 1 mg/kg propofol and 2 mg/kg thiopental administered intravenously were equally effective in stopping bupivacaine-induced seizures.

This patient exhibited many signs of CNS toxicity of local anesthetics. Embolic encephalopathy was unlikely due to the bilateral symptoms, and electrolyte, oxygenation, and glucose abnormalities were ruled out by normal measurements. The concentration of lidocaine in the peripheral blood of our patient was less than that usually associated with toxicity (greater than 5 μg/ml), but lidocaine injections beside or into arteries in the neck are known to cause excessive concentrations in the brain while systemic concentrations remain below toxic threshold. A single bolus of lidocaine injected directly into the carotid artery of our patient would have peaked and washed out rapidly from the brain, perhaps as we administered propofol. No intrar arterial injection was apparent in this case, and we treated lidocaine toxicity over the longer duration, which would result from perivascular absorption of drug.

The surgeon in this case had just fully exposed the carotid artery when uncontrollable muscle twitching developed; he wanted the patient to remain awake and communicative, to assess the effects of carotid artery clamping. Small doses of midazolam, fentanyl, and esmolol did not stop the patient’s restlessness and muscle tremors, but one 20-mg dose of propofol (0.28 mg/kg) was effective. This small dose of propofol did not cause sedation and improved the level of consciousness. The relief from shaking lasted only a few minutes, after which it returned and was relieved again by propofol. The CNS toxicity of lidocaine may result from depression of subcortical inhibitory control by α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (GABA) over normally occurring excitatory activity. Propofol may specifically reverse this deficiency by acting as a GABA-mimetic drug. Although clinical experience with and research documentation for treating lidocaine toxicity with benzodiazepines is well established, propofol should undergo further study as an alternative treatment.

References