Ropivacaine-induced seizure after extradural anaesthesia

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Summary

Ropivacaine is considered safer than bupivacaine, on the basis of experimental and human data that indicate a lower systemic toxicity. Here we report the occurrence of a single seizure after i.v. administration of ropivacaine 120 mg intended for extradural block in a patient having postpartum tubal ligation. The only prodromal symptom was nervousness, and the only cardiovascular manifestation was sinus tachycardia. Systemic toxicity, although less than that expected with bupivacaine, can occur with ropivacaine. (Br. J. Anaesth. 1998; 80: 843–844)

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Ropivacaine is an amide-type local anaesthetic agent that is prepared as an S-isomer. Previous studies showed the relative neuro-and cardiovascular safety of ropivacaine compared with bupivacaine.1,2 Here we report a case of systemic toxicity after extradural anaesthesia with ropivacaine. To our knowledge, this is the first reported case of systemic toxicity after attempted extradural block using ropivacaine.

Case report

On April 21, 1997, a 23-year-old woman (G4 P3, 59 kg, ASA I) was to undergo postpartum tubal ligation. On the evening before the procedure, a lumbar extradural catheter with a closed end and two side holes was placed for labour pain. Aspiration was negative for cerebrospinal fluid and blood. Examination revealed full cervical dilatation, so no local anaesthetic was injected. After a normal vaginal delivery, the extradural catheter was left in place in anticipation of the tubal ligation.

Next morning, anaesthesia for postpartum tubal ligation was discussed with and accepted by the patient. Monitoring included pulse oximetry, non-invasive arterial pressure and electrocardiogram (ECG). After negative aspiration for blood and cerebrospinal fluid, 0.75% ropivacaine was injected extradurally in fractionated doses. Initially, a 2-ml bolus was given (15 mg). Three min later, there were no signs of subarachnoid block; ropivacaine 4 ml (30 mg) was therefore injected. No changes in the patient’s status were detected and another 4 ml (30 mg) was injected, combined with fentanyl 100 μg. After a second negative aspiration, another 4 ml (30 ml) was given. There were no signs or symptoms suggestive of intravascular injection, such as altered consciousness, blurred vision, ringing in the ears or metallic taste. Administration of an additional 4 ml was then attempted, but after injection of only 2 ml (15 mg), the patient closed her eyes and stated that she felt nervous; no further drug was given. The total dose of ropivacaine was 120 mg injected in increments over a period of 11 min. Within a few seconds of injection of the last increment, the patient experienced a generalized tonic–clonic seizure lasting approximately 1 min. During this episode the patient maintained her airway and, apart from sinus tachycardia of 120 beats min^-1 (as proven by ECC), there were no other arrhythmias. Arterial pressure was 115/70 mm Hg, and oxygen saturation 100%. The patient spontaneously recovered before medical intervention but was lethargic. Apart from minor injury to the edges of the tongue, there were no injuries nor neurological, respiratory or cardiovascular sequelae. No sensory or motor effects developed subsequent to the local anaesthetic injection. The procedure was cancelled. Inspection of the extradural catheter after removing the opaque adhesive tape revealed blood in the distal portion.

The next day the patient was discharged home. She was readmitted a few days later, had tubal ligation under spinal anaesthesia, and made an uneventful recovery.

Discussion

Ropivacaine is a relatively new amide local anaesthetic agent that is commercially prepared as a pure S-enantiomer.1–4 Experimental data have shown that ropivacaine is less cardio- and neurotoxic than racemic bupivacaine.1,6 Central nervous system toxicity after accidental i.v. administration of local anaesthetics has been reported before.7,8 However, only one case of convulsion was reported with ropivacaine after transarterial brachial plexus block.9 Various techniques have been advocated to reduce the unintentional i.v. injection of local anaesthetics during extradural analgesia; none has been shown to be infallible.10–12

CASE REPORTS

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In the case reported here, the serum drug concentration was not determined. However, the absence of a history of any illness, especially epilepsy or pre-eclampsia, the nonrecurrence of the convulsions, the absence of motor and sensory block, and the timing of the convulsion in relation to the local anaesthetic injection were suggestive of a toxic reaction to the drug.

In studies using intravenous ropivacaine and bupivacaine on human volunteers, there was a clear difference between the two drugs with regard to their ability to induce signs and symptoms of central nervous system (CNS) toxicity. Although the early symptoms of CNS toxicity were similar with both drugs, they appeared more often, occurred earlier, at a lower dose and at a lower arterial and venous plasma concentration with bupivacaine. I.v. infusion of ropivacaine was well tolerated and without prodromal CNS symptoms in seven of 12 volunteers, in spite of the intravenous infusion of 150 mg in 15 min.1 Our patient developed a convulsion with only one prodromal symptom, namely nervousness, at a dose of 120 mg injected intermittently over 11 min.

The absence of classic symptoms of CNS toxicity in spite a large dose of ropivacaine may indicate a better tolerance of ropivacaine, but it takes away the warning signs of imminent convulsions. It may therefore be prudent to add a reliable agent that acts as a marker for unintentional intravenous administration, such as epinephrine. Another alternative may be to use as a marker lidocaine, which readily elicits prodromal symptoms without adverse cardiovascular effects. In addition, fractionation of the total dose, although it did not alert us to the position of the extradural catheter, is important because it limits the systemic toxicity of local anaesthetics.

There are various possible reasons for failure to detect the intravascular position of the catheter. The catheter could have been against a blood vessel wall; a blood clot in the terminal part of the catheter might have acted as a one-way valve; and the opaque tape could have prevented the detection of blood in the distal portion of the catheter.

In conclusion, this case report demonstrates the relative absence of prodromal symptoms of CNS toxicity after i.v. injection of a relatively large dose of ropivacaine and the absence of serious manifestations of cardiovascular toxicity. It emphasizes the importance of fractionating the total dose of a local anaesthetic, as this prevents a major reaction in the event of i.v. injection. It also highlights the value of using a marker to detect intravascular injection.

References