Bilateral severe pain at L3–4 after spinal anaesthesia with hyperbaric 5 % lignocaine

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Summary

We describe a case of transient pain in both legs after spinal anaesthesia with a hyperbaric solution of 5 % lignocaine delivered in a single bolus through a 22-gauge Quincke needle. The patient remained in a supine decubitus position throughout surgery and 31 h later pain was referred bilaterally to the L3 and L4 dermatomes. (Br. J. Anaesth. 1996; 76: 328–329)

Key words
Anaesthetic techniques, subarachnoid. Complications, neurological. Anaesthetics local, lignocaine.

Lignocaine 5 % in hyperbaric solution has long been used for subarachnoid anaesthesia and was considered safe [1] until reports of permanent neurological complications began to appear in 1991, after the introduction of continuous spinal anaesthesia through microcatheters [2, 3]. Microcatheters were blamed mainly at first, until cases of severe transient pain in both lower extremities were reported later after spinal anaesthesia with 5 % lignocaine in hyperbaric solution delivered in a single dose [4, 5]. We describe a case involving a patient who developed severe pain in both legs the second day after spinal anaesthesia with 5 % lignocaine.

Case report

A 66-yr-old man, 159-cm tall and weighing 80 kg, was scheduled for removal of a penile implant that had caused skin ulceration. The patient had chronic bronchitis but there was no history of neurological problems. Blood clotting and chemistry were normal. Prior history included repair of an umbilical hernia, haemorrhoidectomy and implantation of a penile prosthesis on two occasions. Anaesthesia for all four operations comprised spinal injection of 0.5 % bupivacaine. On this occasion, the patient was seated and the lumbar area washed with povidone. A spinal needle with a 22-gauge Quincke point was inserted easily at the L3–4 interspace. Correct placement was confirmed by free flow of CSF. We then administered 5 % lignocaine 90 mg in a 7.5 % glucose solution for subarachnoid anaesthesia (Laboratorios Palex SA, Barcelona, Spain) over a period of 10 s. The patient was awake during the spinal anaesthetic. There were no signs of pain or paresthesiae on insertion of the needle or during injection of the local anaesthetic and the patient was placed immediately in the supine decubitus position. Anaesthesia reached the T8 level and surgery lasted 35 min with no complications. Seventy minutes after injection of the anaesthetic, after signs of sensory and motor recovery had been observed, the patient was transferred to the ward. Thirty-one hours after surgery he complained of continuous burning pain perceived symmetrically on both sides from the hips to the popliteal fossa (L3–4). The pain was unaffected by change in position and no loss of strength or sensation was evident. There was no neck stiffness and no fever. The pain was not relieved by non-steroidal anti-inflammatory drugs (NSAID), heat pads or benzodiazepine compounds. The pain clinic was consulted and bilateral neuropathic pain, possibly related to subarachnoid anaesthesia, was diagnosed. Treatment with carbamazepine and metamizole, and a dose of amitriptyline at night, produced obvious improvement. This regimen was replaced by clonazepam and metamizole 24 h later, when dysarthria appeared. After 2 days on this treatment, and 4 days after anaesthesia, the pain disappeared completely and the patient was discharged 6 days after surgery with no sequelae.

Discussion

Neurological complications of subarachnoid anaesthesia are rare but potentially serious and at times difficult to diagnose. There are various causes, including direct neural damage by puncture, infections, haematomas, ischaemia caused by hypotension or other factors such as vasoconstriction or root distension [6]. The usual local anaesthetics and doses used for spinal anaesthesia are considered safe and have rarely been blamed for complications. Major neurological problems are rare; in 65 000 operations with intradural anaesthesia, Kane [7] found only 16 such events, ranging from cases of mononeuropathy to lesions of the lumbar plexus. However, in another prospective study of 10 098 cases of spinal anaesthesia in which the authors recorded minor neurological complications [8], pain identical to that seen after general anaesthesia in the lower extremities was found in 1 % of cases.

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The safety of hyperbaric 5% lignocaine began to be questioned by Schneider and colleagues [4], who reported four cases of pain in the lower extremities (L5–S1) after spinal anaesthesia with single doses through a needle. The authors emphasized the possibility that the lithotomy position may have played a role, given that in dissections in that position the L5–S1 roots were lower and distended. A similar case was reported recently by Sjöström and Bläss [5].

Our case differed from the previous reported cases of transient lumbosacral neuropathy in that the patient was placed in the supine decubitus position, the affected roots were L3 and L4, and the pain appeared later and was relieved by antidepressants and anticonvulsants. The symmetrical nature of the pain and the lack of pain or paraesthesiae during the procedure suggests that no damage was caused by the needle. There was no hypotension or hypoxia that might have led to medullary ischaemia and the anaesthetic did not contain adrenaline. Chemical contamination was highly unlikely as we used disposable equipment and strict aseptic procedures.

A single injection of hyperbaric 5% lignocaine was administered at the recommended dose for subarachnoid anaesthesia [6]. Moreover, a 22-gauge needle was used, and therefore overdose or poor distribution because of slow injection seems unlikely to have caused the complication. Our patient’s pain was at the L3 and L4 dermatomes but, in all previously reported cases, patients were placed in the lithotomy position and L5 and S1 were always affected. We do not know what role the supine decubitus position or other factors may have played in producing the highly unusual location of our patient’s lesion.

In nine of 11 cases of cauda equina syndrome described in the literature and in six cases of transient lumbosacral neuropathy, including the one described here, the only agent clearly implicated is hyperbaric 5% lignocaine, although these complications have been observed in animal studies after subarachnoid anaesthesia with 2-chloroprocaine and bupivacaine in clinical concentrations [9, 10]. In another study in rats, sensory deficit lasted longer in the group receiving 5% lignocaine in 7.5% glucose than in groups receiving 0.75% bupivacaine in 8.25% glucose, 5% amethocaine with 5% glucose or saline solution [11]. Similarly, in the same experimental model, it was shown that the presence of 7.5% glucose did not affect the ability of 5% lignocaine to produce sensory deficits [12].

The possibility of developing minor, transient neuropathy after subarachnoid anaesthesia should not be underestimated, given that the responsibility for follow-up and treatment of pain belongs to the anaesthetist. Pain therapy was different in all cases; three of the patients were relieved by NSAID, another by pethidine and one patient refused to take analgesics [4, 5]. Our patient also received NSAID but he was not relieved of pain until antidepressants and anticonvulsants were added. We believe that the clinical picture described here occurs more often than the literature suggests, but, as the condition unfolds 24 h after surgery, it often remains undiagnosed.

We believe that the use of hyperbaric 5% lignocaine for subarachnoid anaesthesia is not risk-free when given in a single dose through a large-calibre needle or when a position other than lithotomy is used. We conclude that its clinical use should be questioned and further studies should be performed.

References