Repeated transient neurological symptoms after spinal anaesthesia with hyperbaric 5% lidocaine

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Summary
We report a case of repeated delayed pain after cystoscopy under spinal lidocaine anaesthesia, which may be caused by transient radicular irritation. The possible aetiology of the symptoms is discussed. (Br. J. Anaesth. 1998; 81: 471–472).

Keywords: anaesthetics local, lidocaine; anaesthetic techniques, subarachnoid; complications, transient radicular irritation

Lidocaine (lignocaine) has been used extensively in spinal anaesthesia since 1949. However, following the description of cauda equina syndrome in 1991 after continuous spinal anaesthesia via microcatheters, lidocaine has been associated with transient neurological symptoms when used as a single injection for spinal anaesthesia. The first cases of transient radicular irritation (TRI) with lidocaine were reported in 1993. We present a case of repeated pain after spinal anaesthesia with lidocaine.

Case report
A 74-yr-old man with a superficial bladder tumour was scheduled for cystoscopy. His past history included transurethral resection of his bladder tumour 3 months earlier under spinal anaesthesia. History, physical examination and laboratory results were otherwise unremarkable. Spinal anaesthesia was performed using a sterile technique in the sitting position with a 24-gauge Sprotte needle (Pajunk, Germany) in the L3–4 interspace using the midline approach. After confirming free flow of clear cerebrospinal fluid, 5% hyperbaric lidocaine 75 mg was injected with no dilution. There was no pain or paraesthesia during insertion of the needle or injection of drug. Analgesia to T10 was achieved with no hypotension, and without the use of vasopressors.

Surgery was performed in the lithotomy position and lasted 30 min. Motor function in the legs returned 110 min after spinal puncture and the patient was moved to the ward with instructions to mobilize 6 h later. The patient had no complaints on the following morning but before discharge he asked if he was going to have the same discomfort he had felt after previous surgery. He explained that on the day after his previous surgery, he felt pain in the hips, buttocks and legs, radiating to the toes, which increased on the second postoperative night (36 h after the procedure). The pain was worse at rest and he thought it was a result of anxiety. He did not recall any weakness, headache or fever. The pain disappeared after 24 h without the use of analgesics.

Review of the past anaesthetic record revealed spinal anaesthesia with a 24-gauge Sprotte needle, 5% hyperbaric lidocaine 75 mg, no hypotension, a 45-min surgical procedure in the lithotomy position, motor function returning 100 min after injection of lidocaine and mobilization 6 h after anaesthesia. We delayed discharge, and that afternoon, 30 h after spinal puncture, the patient complained of similar symptoms. Pain was characterized as dull, involving the hips, buttocks and legs, radiating to the toes. There was no headache, fever, or sensory, motor or muscle tendon reflex abnormalities. There were no signs of bladder or bowel dysfunction. Acetaminophen 500 mg orally every 6 h partially improved his symptoms. The pain never reached the intensity of the first episode and disappeared completely after 18 h. There were no sequelae and the patient was discharged. Interestingly, 6 months later, two 30-min cystoscopy procedures performed under general anaesthesia in the lithotomy position were not associated with these symptoms.

Discussion
We have presented a case of repeated pain associated with the use of spinal lidocaine. The symptoms were similar to those described in the literature but interestingly, the patient did not complain of problems after the previous surgery. The syndrome may have been missed as many patients are discharged before symptoms occur. We believe that it is necessary to ask all patients who have received previous spinal anaesthesia specifically about this complication.

Even though the first description was with 5% hyperbaric lignocaine, TRI has been described with lower concentrations and osmolarities. Prospective studies with spinal lidocaine have shown an incidence of TRI of 10–37%. Lidocaine can produce rapid neurological damage in animal models with no recovery of normal function. In contrast, in TRI, there is delayed onset associated with complete recovery. Therefore, TRI may not be related to drug...
toxicity (defined as persistent biochemical, functional or structural effects) but to a delayed irritative process of unclear aetiology.

Whether the pain is radicular or myofascial is not clear. Bilateral pain is not typical of a myofascial aetiology, and neither is relief with movement. Although TRI has been described in all positions, its incidence is higher in the lithotomy position, perhaps because of stretching of the lumbosacral nerve with possible compromise of neural perfusion and increased vulnerability to the effects of lidocaine. Another explanation of TRI may be nerve or spinal cord ischaemia, related to lidocaine-induced reduction in nerve blood flow, the position of the patient and concomitant use of non-steroidal anti-inflammatory drugs (NSAID).

In contrast with our case, some patients with TRI present with central pain (burning or electrical quality with hypersensitivity or decreased sensation) which improves with anticonvulsant and antidepressant treatment. The only clinical finding in our patient was the delayed onset after a possible injury. It is improbable that TRI has been unrecognized for almost 50 yr (after approximately 50 million administrations of spinal lidocaine) and therefore other factors need to be considered. Recent changes in clinical practice that could be associated with an increased incidence of TRI include use of pencil-point needles, early mobilization of patients after spinal anaesthesia and use of NSAID. Pencil-point needles are less traumatic to the dural membrane, but slow injection through a side-port needle directed sacrally can lead to high peak concentrations of injected drug, and its blunt tip might dislodge the arachnoid membrane from the dural sheath producing bleeding (with nerve root irritation). Three prospective studies published to date have been performed with different needles and positions, and none has addressed the question of early mobilization or NSAID use.

Regardless of the cause, the clinical course of our patient was similar to that reported previously with lidocaine, implying a common mechanism. Additional studies are needed to determine the aetiology of these symptoms.

References