Clinical Reports

Bilateral leg pain following lidocaine spinal anaesthesia

Gail R. Pinczower MD, H.S. Chadwick MD,
Robin Woodland MD, Mark Lowmiller MD

Spinal anaesthesia is considered to be a safe and effective method of providing anaesthesia for a variety of surgical procedures. Recently, observations have been made that associate the use of hyperbaric lidocaine with bilateral leg pain. We report nine patients who developed strikingly similar neurological symptoms following routine spinal anaesthesia using hyperbaric lidocaine 5% solutions. All patients had their anaesthesia and surgery in the ambulatory or "short stay" care setting. In each patient, moderate to severe, bilateral, posterior, leg pain developed within 24 hr of the anaesthetic administration. The pain was described as either sharp or cramping with or without associated back pain. None of the patients demonstrated objective neurological deficits. In all cases the symptoms resolved fully within one week. The dose of lidocaine administered in these nine patients ranged from 40 to 100 mg. Although the aetiology of the symptoms is not clear the local anaesthetic or its formulation may have been responsible.

La rachianesthésie est considérée comme une méthode sûre et efficace de produire l'anesthésie pour une grande variété d'interventions. Récemment, on a rapporté des douleurs bilatérales au membre inférieur associées à l'utilisation de lidocaïne hyperbare. Nous rapportons ici les cas de neuf patients qui ont développé des symptômes neurologiques identiques après une rachianesthésie à la lidocaïne hyperbare à 5%. Tous les patients avaient été anesthésiés et opérés dans un cadre ambulatoire ou de court séjour. Chez tous ces patients, des douleurs au membre inférieur, bilatérales, postérieures, de modérées à graves sont apparues dans les 24 h consécutives à l'anesthésie. La douleur était décrite comme aiguë et spasmodique associée ou non à de la lombalgie. Aucun des patients n'a présenté de déficit neurologique. Dans tous les cas, les symptômes sont disparus en deçà d'une semaine. La dose de lidocaïne administrée à ces patients était de 40 à 100 mg. Bien que l'étiologie de ces symptômes soit incertaine, l'anesthésique local ou sa formulation semble être en cause.

Key words
ANAESTHETIC TECHNIQUES: spinal;
ANAESTHETICS, LOCAL: lidocaine;
COMPLICATIONS: NEUROLOGICAL.

From the Departments of Anesthesiology, University of Washington, Seattle, Washington and Swedish Medical Center, Seattle, Washington.

Address correspondence to: Dr. H.S. Chadwick, Department of Anesthesiology, RN-10, University of Washington Medical Center, Seattle, Washington 98195.

Accepted for publication 22nd October, 1994.

In 1992, we provided care for three patients who reported back and posterior leg pain after receiving spinal anaesthesia. The patients' intraoperative anaesthetic courses were uneventful, and they were discharged from the hospital without unusual symptoms on the day of surgery. All three patients described onset of lower back and bilateral posterior leg pain within 24 hr of their procedure. In reviewing the literature, at that time, we were unable to find this type of pain reported as a complication of spinal anaesthesia. Inquiries with other colleagues were made to determine if similar cases had been observed. As a result of our inquiries, we collected 17 cases in addition to our original three. All 17 cases involved the use of either lidocaine 2% or hyperbaric lidocaine 5% as the anaesthetic agent. Most involved a variety of postoperative symptoms unlike the ones we had observed; however, some shared strikingly similar features. We chose five specific criteria which were common to the cases with which we were familiar.

1 Bilateral radicular-like leg pain with or without back pain
2 Moderate or severe pain
3 Onset of pain within 24 hr of surgery
4 Duration of pain >24 hr
5 No previous history of severe back or leg pain

After reviewing all available medical information from the 17 additional cases that were collected, we were left with nine (including our own) in which all of the inclusion conditions were present. All involved spinal anaesthesia

TABLE Patient data

Age	*Sex*	*Procedure*	*Month/ Year of surgery*	*Dose, lidocaine*	*Needle type*	*Surgical position*
42	F	Cystoscopy	2/92	50 mg + epi 0.2 mg	Sprotte #24	Lithotomy
58	M	Achilles tendon repair	10/89	65 mg + epi 0.2 mg	Quincke #26	Prone
36	F	Varicose vein ligation	1/92	70 mg	Whitacre #27	Supine
72	F	Cystoscopy	3/92	50 mg + epi 0.2 mg	Sprotte #24	Lithotomy
56	M	Knee arthroscopy	5/91	65 mg + epi 0.1 mg	Quincke #26	Supine
34	F	Cervical cerclage	6/86	40 mg	Quincke #26	Lithotomy
43	M	Cystoscopy	5/88	60 mg	Quincke #26	Lithotomy
71	M	TURP	5/86	50 mg	Whitacre #27	Lithotomy
51	M	Knee arthroscopy	1/92	60 mg + epi <0.1 mg repeat 40 mg	Quincke #25	Supine

In all cases the local anaesthetic was lidocaine 5% with dextrose 7.5%; "epi" indicates that epinephrine was added to the local anaesthetic solution. In the last case a repeat spinal anaesthetic was injected ten minutes after the first injection because of incomplete anaesthesia following the initial injection.

for outpatient or short stay procedures in which hyperbaric lidocaine 5% was the anaesthetic agent. For the sake of brevity we present three of these cases in detail. All nine cases are summarized in the Table.

Case reports

Case 1

A 42-yr-old woman with a history of recurrent urinary tract infections underwent cystoscopy as an outpatient under spinal anaesthesia. She had a history of ovarian cancer that had been treated surgically with radiation therapy 19 yr previously. The postoperative course was complicated by bowel obstruction necessitating an ileostomy, as well as recurrent urinary tract infections which required multiple courses of antibiotic therapy. Her current medication included nitrofurantoin, oestrogen, and amitriptyline. A spinal anaesthetic was performed at the L_3–L_4 interspace with 50 mg lidocaine 5% in dextrose 7.5% using a #24-gauge Sprotte needle. No paraesthesia were elicited. The patient was placed in lithotomy position for the procedure which proceeded uneventfully. She was discharged from hospital the same day in good condition. Later that evening she noted the onset of sharp pains radiating from her "tailbone" down both buttocks and posterior thighs. She also complained of a persistent headache that did not have the characteristics of a postdural puncture headache. There were no associated signs of fever, chills, bowel or bladder dysfunction, focal weakness, numbness or paraesthesia. After three days of bed rest and oral analgesics her symptoms resolved.

Case 2

A 58-yr-old man with Achilles tendonitis underwent an outpatient procedure for tendon debridement with spinal anaesthesia. His only other medical problem was hypercholesterolaemia for which he was taking lovastatin. Spinal anaesthesia was induced with a 26-gauge Quincke tip needle placed atraumatically in the midline at the L_3–L_4 interspace. Following injection of 65 mg lidocaine 5% in dextrose 7.5% with 0.2 mg epinephrine, the patient was turned prone for uneventful surgery. That evening he awoke with severe "crampy" pain involving the lower back and radiating to the posterior thigh and calf region bilaterally. He denied any associated systemic symptoms, sensory, or motor impairment. The pain persisted with no relief from hot baths, movement, or acetaminophen with codeine. His symptoms did diminish somewhat following medication with ibuprofen. Complete resolution of the pain occurred after two days.

Case 3

A 36-yr-old healthy woman was scheduled for right saphenous vein ligation and division. An autraumatic spinal anaesthetic was performed with a 27-gauge Whitacre needle using a paramedian approach at L_3–L_4 interspace. A total of 70 mg lidocaine 5% without epinephrine was injected after dilution with 1 ml CSF. The following morning she was discharged from the hospital with a prescription for acetaminophen with codeine for bilateral "leg stiffness." That evening, however, her leg pain worsened and was described as a severe shooting pain extending from the buttocks down the back of both legs. The pain, which persisted over the next three days, was noted to be worse at night and less bothersome while walking during the day. There were no other neurological symptoms such as weakness, paraesthesia, bladder or bowel dysfunction. An evaluation performed by a neurologist four days postoperatively revealed no neurological deficit. The patient's symptoms gradually subsided over the next several days without further treatment. She remained asymptomatic four months postoperatively.

Discussion

The anaesthesia literature is replete with articles that describe neurological sequelae related to spinal anaesthesia.[1-4] Complications vary from transient backache to paraesthesia to permanent paralysis and sacral autonomic dysfunction. The incidence of back pain in the ambulatory care setting is reported to be 10–20% even when modern small gauge needles are used.[5,6] Recently, Schneider *et al.* reported four patients having gynaecological procedures, who developed back and bilateral leg pain following spinal anaesthesia with hyperbaric 5% lidocaine.[7] Another recently published case involving a 63-yr-old man undergoing transurethral resection of a bladder tumour was reported by Sjöström and Bläss.[8] The association of bilateral leg pain without neurological deficits following spinal anaesthesia seems to be a new observation in the anaesthesia literature.

Patient position, type of procedure, and needle type do not appear to be factors in the development of symptoms, as these variables showed no pattern in our nine patients (Table). Back pain was an important complaint in four, although in three it was not. Abnormal stretching of the ligaments and muscles in the back (especially in the lithotomy position) has been implicated as a cause of back pain following spinal anaesthesia.[6,9] All of the previously reported cases involved surgery in the lithotomy position.[7,8] Schneider *et al.* speculated that the lithotomy position flattens the lumbar lordosis, stretching the cauda equina, and places the L_5 and S_1 roots in the most dependent position. The authors suggest that this puts a subset of nerve roots at jeopardy due to reduced blood flow and exposure to relatively high concentrations of hyperbaric local anaesthetic.[7] Five of our nine patients also had surgery in the lithotomy position which may have contributed to their symptomatology. However, four of our patients had surgery in the supine or prone positions which indicates that surgical position is not a necessary factor in this phenomenon.

Our case series consists of uncomplicated lidocaine spinal anaesthesia for routine ambulatory care or "short stay" procedures. None of the cases that we identified involved other local anaesthetics which suggests that the lidocaine or its 7.5% dextrose formulation may have played a role. Spinal lidocaine manufactured by both Astra and Abbott was used in these cases. Both pharmaceutical companies were questioned by the authors regarding any recent changes in formulation, of which there were none. This makes it unlikely that a change in pharmaceutical preparation was a factor.

In five of the nine cases we report, epinephrine was added to the anaesthetic solution. In a long-term follow up of spinal anaesthetics, the role of epinephrine did not appear to be a factor contributing to neurotoxicity.[1] However, spinal cord blood flow may be vulnerable to the vasoconstrictive effects of epinephrine. It has been suggested that anterior spinal artery blood flow may be jeopardized especially in the presence of systemic hypotension or arteriosclerosis.[10] The symptoms in our patients differed markedly from those seen with anterior spinal artery syndrome and it seems unlikely that they were related to spinal cord ischaemia.

Although concentrations of local anaesthetics currently available for intrathecal use are considered to be free of neurotoxicity, some studies suggest this may not be true. The neurotoxic potential of intrathecally administered lidocaine has been studied in rabbits by Ready *et al.*[11] Evidence of dose-dependent permanent neurological deficits of the hind extremities and/or histological damage to the spinal cord was seen with intrathecally administered hyperbaric lidocaine concentrations ranging from 4–32%. A recent animal study found evidence of neurological injury with subarachnoid lidocaine 5% but not with bupivacaine 0.75% or with tetracaine 0.5%. All three local anaesthetics were rendered hyperbaric with dextrose 5–8.5%.[12] This suggests that there are inherent differences in the potential of these concentrations of local anaesthetics to cause neurological sequelae.

Cauda equina syndrome has been reported in a number of patients following the use of hyperbaric lidocaine 5% in continuous spinal anaesthesia.[13,14] Injection of hyperbaric lidocaine 5% into models of the spinal canal can result in maldistribution and potentially toxic concentrations of local anaesthetic in dependent regions within the CSF.[15-17] The cause of the leg pain in the cases we report may be due to direct effects of concentrated local anaesthetic solution. Alternatively, the hypertonic nature of the injected solution (lidocaine 5% in dextrose 7.5%) may cause osmotic-induced injury to exposed nerve tissue.[18] The dorsal nerve roots or adjacent meninges in the lumbo-sacral area may become irritated or injured by exposure to relatively undiluted anaesthetic solutions resulting in back pain radiating into both the legs.

Because all nine cases occurred in association with outpatient or "short stay" procedures, the question arises as to whether or not ambulatory surgical patients are at greater risk of developing post-anaesthetic complications following spinal anaesthesia? We perform many lidocaine spinal anaesthetics in obstetric patients for tubal ligation, saddle block, and Caesarean section, but have not observed these symptoms in that population. The possibility must be considered that some factor associated with ambulatory procedures predisposes patients to complications of this sort. Perhaps the greater utilization of ambulatory care facilities has resulted in increased use of lidocaine for spinal anaesthesia.[19] We may only now be seeing this

phenomenon as a result of closer observation and heightened concern about patient satisfaction. Alternatively, the lack of early ambulation and the greater use of opioid analgesia may mask the symptoms of back pain in hospitalized surgery patients, as has been suggested by others.[20]

The similar symptomatology among the cases presented suggests that we are observing a syndrome resulting from a specific causal mechanism, possibly the result of a mild form of neurotoxicity. It is difficult to calculate the incidence of this bilateral "sciatica-like" syndrome. Many patients are discharged from hospital shortly after their procedure, and post-anaesthetic follow up may be less than optimal. Although we do not know the aetiology of the back and leg pain in these cases, we believe that anaesthetists should know that such cases can occur. Similar sentiments were recently expressed in an editorial by de Jong.[21] It should be stressed that none of these patients demonstrated objective neurological findings. Although the experience was distressing for both patients and anaesthetists, the pain resolved within a few days without any permanent sequelae. The distressing, albeit temporary nature of the symptoms may lead some to question the advisability of using hyperbaric spinal lidocaine in the ambulatory care setting. Some of us continue to use lidocaine for short duration spinal anaesthesia because of the rare nature of these problems while others prefer to use procaine which also has a short duration of action.

In summary, we report a series of nine patients who developed bilateral posterior leg pain following routine hyperbaric lidocaine spinal anaesthesia in the ambulatory care setting. These symptoms may represent a mild form of neurotoxicity from lidocaine or from the hypertonic nature of the injected solution. The purpose of this report is to alert others of this potential problem and to encourage further study of this phenomenon.

References

1 *Vandam LD, Dripps RD* A long-term follow-up of 10,098 spinal anesthetics. II. Incidence and analysis of minor sensory neurological defects. Surgery 1955; 38: 463–9.

2 *Steen PA, Michenfelder JD.* Neurotoxicity of anesthetics. Anesthesiology 1979; 50: 437–53.

3 *Kane RE.* Neurologic deficits following epidural or spinal anesthesia. Anesth Analg 1981; 60: 150–61.

4 *Greene NM.* Neurological sequelae of spinal anesthesia. Anesthesiology 1961; 22: 682–98.

5 *Quaynor H, Corbey M, Berg P.* Spinal anaesthesia in day-care surgery with a 26-gauge needle. Br J Anaesth 1990; 65: 766–9.

6 *Kang SB, Goodnough DE, Lee YK, et al.* Comparison of 26- and 27-G needles for spinal anesthesia for ambulatory surgery patients. Anesthesiology 1992; 76: 734–8.

7 *Schneider M, Ettlin T, Kaufmann M, et al.* Transient neurologic toxicity after hyperbaric subarachnoid anesthesia with 5% lidocaine. Anesth Analg 1993; 76: 1154–7.

8 *Sjöström S, Bläss J.* Severe pain in both legs after spinal anaesthesia with hyperbaric 5% lignocaine solution. Anaesthesia 1994; 49: 700–2.

9 *Bridenbough P, Greene N.* Spinal (subarachnoid) neural blockade. *In*: Cousins M, Bridenbaugh P (Eds.). Neural Blockade in Clinical Anesthesia and Management of Pain. Philadelphia: JB Lippincott, 1988; 246.

10 *Wetchler BV.* Anesthesia for Ambulatory Surgery. Philadelphia: JB Lippincott, 1991; 344.

11 *Ready LB, Plumer MH, Haschke RH, Austin E, Sumi SM.* Neurotoxicity of intrathecal local anesthetics in rabbits. Anesthesiology 1985; 63: 364–70.

12 *Drasner K, Sakura S, Chan VWS, Bollen AW, Ciriales R.* Persistent sacral sensory deficit induced by intrathecal local anesthetic infusion in the rat. Anesthesiology 1994; 80: 847–52.

13 *Rigler ML, Drasner K, Krejcie TC, et al.* Cauda equina syndrome after continuous spinal anesthesia. Anesth Analg 1991; 72: 275–81.

14 *Schell RM, Brauer FS, Cole DJ, Applegate RL II.* Persistent sacral nerve root deficits after continuous spinal anaesthesia. Can J Anaesth 1991; 38: 908–11.

15 *Rigler MR, Drasner K.* Distribution of catheter-injected local anesthetic in a model of the subarachnoid space. Anesthesiology 1991; 75: 684–92.

16 *Lambert DH, Hurley RJ.* Cauda equina syndrome and continuous spinal anesthesia. Anesth Analg 1991; 72: 817–9.

17 *Ross BK, Coda B, Heath CH.* Local anesthetic distribution in a spinal model: a possible mechanism of neurologic injury after continuous spinal anesthesia. Reg Anesth 1992; 17: 69–77.

18 *Wildsmith J.* Catheter spinal anesthesia and cauda equina syndrome: an alternative view (Letter). Anesth Analg 1991; 73: 368–9.

19 *Wetchler BV.* Anesthesia for Ambulatory Surgery. Philadelphia: JB Lippincott, 1991; 350.

20 *Fibuch EE, Opper SE.* Back pain following epidurally administered Nesacaine-MPF. Anesth Analg 1989; 69: 113–5.

21 *de Jong RH.* Last round for a "heavyweight"? (Editorial). Anesth Analg 1994; 78: 3–4.