Burning Leg Pain during Spinal Anesthesia in a Diabetic Patient

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Bizarre leg pain under spinal anesthesia has been reported in patients with phantom limb pain[1–8], syphilis[9,10], sciatica[1], and osteoarthritis[11], but not in patients with diabetes. We present a case of burning leg pain in a diabetic patient with no definite symptom of peripheral neuropathy. The pain was triggered by spinal anesthesia, but not by epidural anesthesia.

Case Report

A 76-yr-old, 63-kg man with a 10 year history of diabetes was scheduled for transurethral resection of the prostate under spinal anesthesia. He had been treated with sulfonylurea until 5 years previously, and since then his blood glucose level had been maintained at about 130 $mg \cdot dl^{-1}$ by dietary treatment alone. He had suffered from diabetic retinopathy and received photo-coagulation of his retinas many times in the past 8 years. He also had suffered from varicosities and necrosis of the big toe of his right foot since about the same time. Ten months previously, the patient experienced tingling pain in the left inguinal region, which subsided spontaneously after about one month. He had no other symptoms or signs suggesting diabetic neuropathy on admission.

On arrival at the operating room, his blood glucose level was 116 $mg \cdot dl^{-1}$. Spinal puncture at the L.3–4 interspace was performed without difficulty, and 2.5 ml of 0.3% dibucaine (Percamine-S™) with 0.2 mg epinephrine was injected. There was no paresthesia or bleeding throughout the procedure. After about 15 min, on moving the patient to the lithotomy position, he suddenly complained of sharp burning pain in his feet. The pain occurred at one min intervals and lasted for a few seconds each time. The pain was stronger in the right foot than the left, and was localized below the ankles with a stocking-like distribution. The level of sensation to pinprick was T10, and his feet were completely analgesic. The surgery was canceled. The pain gradually disappeared after 3 hours, but his feet remained analgesic for further 2 hours.

The surgery was rescheduled two weeks later. This time, epidural anesthesia was used with 8.5 ml of 1.5% lidocaine without epinephrine. Analgesia below the level of T9 was obtained uneventfully, and the surgery was performed.

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About one year later, the patient complained of slight numbness in both legs, stronger on the right side. Nerve conduction velocity in the right arm was found to be slowed[12]; 48.2 $m \cdot sec^{-1}$ on median nerve (normal, 51) and 45.7 $m \cdot sec^{-1}$ on ulnar nerve (normal, 50). A response could not be obtained in the right peroneal nerve, probably due to a technical problem. The patient was diagnosed as having stage 2 (symptomatic) diabetic neuropathy[13].

Discussion

Diabetic neuropathy is more prevalent than is generally thought: approximately 8% of diabetics are estimated to be affected at the time of diagnosis, and 50% of them may develop neuropathy within 25 years[14]. Tingling pain in the leg may be the earliest symptom of diabetic neuropathy[14,15], and an association between foot ulceration, retinopathy, and neuropathy has been reported[16]. Thus, it is likely that our patient had latent diabetic neuropathy. In addition, the character of the leg pain in the present case closely resembles the symptoms of symmetric distal polyneuropathy. This type of diabetic neuropathy predominantly affects small fibers and causes burning and cramp-like pain usually in the legs[14]. Paresthesia may manifest as a burning sensation, and neuropathic symptoms first appear in the lower extremities in a "stocking" distribution[14,17].

The pain in our case may be explained by the gate control theory of phantom limb pain[18,19], or as a direct effect of dibucaine and epinephrine on the spinal cord. According to the gate control theory, sensory deafferentation of the brain stem decreases the inhibitory influence on pain transmission and produces abnormal physiological activity in spinal and brain cells. The "pattern generating mechanism" is then activated and results in specific phantom limb pain. This hypothesis has been applied to explain spinal anesthesia-induced phantom limb pain[5,7,18]. In our case, it is also possible that cessation of sensory deafferentation by spinal anesthesia unmasked the symptoms of latent neuropathy.

There are few reports of exacerbation of phantom limb pain by epidural anesthesia[20–22], and no reports of bizarre pain. In the three reported cases, the pain occurred during the onset or wearing off of the epidural anesthesia, not during its period of action. From this fact, Bulder assumed a differential blockade of the nerves during the onset and the regression of the epidural anesthesia[20]. Earlier blockade of large-diameter fibers compared to small-diameter fibers may open the gate and the spontaneous activity generated by small-diameter fibers may result in the pain. In any case, epidural anesthesia-induced phantom limb pain may differ from the spinal anesthesia-induced bizarre pain.

Dibucaine seems to have a tendency to cause unusual leg pain under spinal lanesthesia. It was used in 7 of the 15 cases of phantom limb pain which have been reported during the last four decades[1–8]. Others implicated were four cases of tetracaine[3,9], three of procaine[3], and one of bupivacaine[7]. Of the four reported cases of unusual leg pain under spinal anesthesia, dibucaine was used in three cases[1,9,10], and bupivacaine in one case[11]. Lidocaine has not been reported to cause such pain under either spinal or epidural anesthesia. However, this bias may be explained by the fact that dibucaine was once widely used or that phantom limb pain during spinal anesthesia has become a well-known complication which is now infrequently reported.

The risk of spinal cord ischemia by additive vasoconstrictors has been refused in humans[23,24], and vasoconstric-

tors were used in only two cases among the reported cases of bizarre leg pain under spinal anesthesia[6]. Thus, it is unlikely that the added epinephrine caused the pain. However, even a small change in spinal blood flow by epinephrine may have been avoided, because many experimental observations have suggested the decreased oxygen delivery due to microangiopathy as a cause of diabetic neuropathy[14].

In summary, we experienced a case of burning leg pain during spinal anesthesia in a diabetic patient with no apparent symptom of neuropathy. Dibucaine with epinephrine resulted in pain, but epidural anesthesia using lidocaine alone did not. The patient developed neuropathy symptoms about one year later, and the spinal anesthesia may have unmasked the latent diabetic neuropathy.

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