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CLINICAL REPORT

Subclinical neuropathy in diabetic patients: a risk factor for bilateral lower limb neurological deficit following spinal anesthesia?

Darshan S. Angadi · Ajit Garde

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Abstract Total knee arthroplasty performed under spinal or general anesthesia is a common successful orthopedic procedure. Nonetheless, in patients with diabetes mellitus this procedure can present unique challenges to orthopedic surgeon and anesthesiologist alike. We describe a case of an elderly male diabetic patient who developed bilaterally symmetrical lower limb neurological deficit following an uneventful total knee arthroplasty performed under spinal anesthesia. Postoperative nerve conduction study with electromyography confirmed symmetrical extensive denervation of lower limb muscles, including low-voltage fibrillation potentials and positive sharp waves. These findings were consistent with a preexisting neuropathy, thereby suggesting a subclinical neuropathy as a potential risk factor for this neurological complication. Our case highlights the fact that patients with longstanding comorbidities, namely peripheral vascular disease and diabetes mellitus, may be at an increased risk of neurological injury following regional anesthesia. Hence, we believe that preoperative evaluation of diabetic patients should include neurophysiological studies to identify subclinical neuropathy and minimize the risk of neurological injury.

Keywords Diabetes · Neuropathy · Regional · Anesthesia · Subclinical

D. S. Angadi (✉)
Department of Trauma and Orthopedics,
Northampton General Hospital,
1, Cottesbrooke Corridor,
Northampton NN1 5BD, UK
e-mail: docdarshan2002@yahoo.com

A. Garde
Department of Trauma and Orthopedics,
Airedale General Hospital, Keighley, UK

Introduction

Lower limb neurological deficit after total knee arthroplasty can be attributed to a multitude of patient-, surgery-, and anesthesia-related causes [1, 2]. The common causes include inappropriate surgical instrumentation, and prolonged application of tourniquet and tight dressings, leading to an ipsilateral neurological deficit. Investigations such as magnetic resonance imaging (MRI) and computed tomography (CT) scan of the spine are used to identify space-occupying lesions such as hematoma, abscess, and prolapsed disk. However, in the absence of any pathological findings on these neuroimaging studies, establishing an accurate etiology in some of these cases can be fraught with difficulties. In such patients, neurophysiological studies may be a useful aid in the diagnosis of a preexistent subclinical neuropathy [3]. A recent study [4] in diabetic rodent models has highlighted the risk of nerve fiber damage following nerve block with local anesthetic drugs.

We describe a case of an elderly diabetic male patient who developed bilateral symmetrical lower limb neurological deficit following an uneventful spinal anesthesia for total knee arthroplasty.

Case report

An 84-year-old man (ASA PS 2) was scheduled to undergo a total knee arthroplasty under spinal anesthesia. He had longstanding ischemic heart disease and type 2 diabetes mellitus. His routine medications included aspirin, metformin, and gliclazide. He had undergone bilateral total hip arthroplasty under general anesthesia 5 years previously. Preoperatively, his clinical assessment was unremarkable. All laboratory results were in normal range and confirmed

good glycemic control, with blood glucose level of 84 mg/dl and HbA1c level of 5.5%. Preoperative tests including a chest radiograph and ECG revealed no abnormalities.

In the preanesthesia room, he was monitored and venous access was established. He received no premedication. Spinal anesthesia was performed in the sitting position with a 25-G pencil-point needle at the L4–L5 interspace. Insertion was uneventful, followed by flow of clear cerebrospinal fluid. Hyperbaric bupivacaine (0.5%, 2.5 ml) was then injected producing a satisfactory block. A medial parapatellar approach was used, and a cemented knee arthroplasty was performed. The intraoperative period and the immediate postoperative period were uneventful. On the first postoperative day he developed paresthesia in both feet, and the second postoperative day it was noted that he was unable to move his toes. He had no symptoms of backache, and the site of spinal needle insertion was unremarkable.

Subsequent neurological assessment showed bilaterally symmetrical neurological deficit in the lower limbs (Table 1).

On vascular examination of the lower limbs, peripheral pulses of posterior tibial and dorsalis pedis arteries were present bilaterally with equal volume. Hence, angiography was not performed. Postoperative hematological parameters were within normal limits. Plain radiographs of the knee showed a cemented prosthesis with no evidence of periprosthetic fracture. Ultrasound Doppler scan showed no evidence of deep vein thrombosis. CT scan was initially performed because MRI was contraindicated by the metal clips used for skin closure of knee arthroplasty wound. CT showed features of an old, stable compression fracture of the T12 vertebra with no evidence of cord compression. MRI of the thoracolumbar spine 6 weeks later showed multilevel disk degenerative changes. However, there was no evidence of impingement of nerve roots or an epidural space-occupying lesion.

Table 1 Findings of neurological assessment

Factor	Lower limb	
	Right	Left
Sensory		
Light touch	Absent—sole, lateral aspect of foot and lateral aspect of leg	Absent—sole, lateral aspect of foot and lateral aspect of leg
Temperature	Normal	Normal
Proprioception	Absent—toes and ankle	Absent—toes and ankle
Motor (power)		
Hip	Normal (5/5)	Normal (5/5)
Knee	Normal (5/5)	Normal (5/5)
Ankle	Plantar flexion (1/5)	Plantar flexion (1/5)
	Dorsiflexion (0/5)	Dorsiflexion (0/5)
Reflex		
Knee	Normal	Normal
Ankle	Absent	Absent
Plantar	Absent	Absent

The paucity of diagnostic information from the aforementioned imaging modalities prompted a neurophysiological evaluation. Nerve conduction study with electromyography was performed on the 10th postoperative day. Nerve conduction studies were carried out in the lower limbs bilaterally and in the right upper limb to include sensory responses for right sural, median, and ulnar nerves. The proximal motor studies included the right median, ulnar, left peroneal, and for the right common peroneal both to the extensor digitorum brevis, abductor hallucis, and tibialis anterior muscles. Stimulating the peroneal nerve while recording from the tibialis anterior muscle produced normal conduction velocities, but a marked drop in amplitude of motor responses was noted. This finding showed that the neurological deficit was not caused by a focal abnormality of the common peroneal nerve. In both lower limbs there was no measurable motor response from either extensor digitorum brevis or abductor hallucis muscle. This result demonstrated severe axonal loss with signs of extensive bilateral denervation of distal lower limb muscles, including low-voltage fibrillation potentials and positive sharp waves. These findings were consistent with a preexisting neuropathy as it would usually take 3–4 weeks after nerve damage for these signs to be noted [5]. This finding confirmed the diagnosis of subclinical neuropathy.

A subsequent neurophysiological evaluation performed at the 6-month postoperative follow-up visit showed no functional improvement with findings of complete denervation in the tibialis anterior muscles bilaterally. However, there was relatively well preserved motor unit recruitment in the quadriceps. A conservative management approach was adopted, and the patient was subsequently referred for neurorehabilitation.

Discussion

Total knee arthroplasty is a common orthopedic procedure performed under spinal anesthesia [6]. However, in patients with diabetes mellitus this procedure can present unique challenges to the orthopedic surgeon and the anesthesiologist alike [7, 8]. Furthermore, subclinical neuropathy in these patients has a reported incidence of higher than 50% [9, 10]. Regional anesthesia has become a popular anesthetic option in the perioperative management of patients with diabetes mellitus [8].

Neurological injury may be caused by ischemic, mechanical, or chemical factors, either alone or in

combination. These factors include prolonged and severe arterial hypotension compromising blood supply to the cord [11], a spinal hematoma from coagulation abnormality [12], and mechanical trauma by the needle bevel contributing to neuropathy [13]. None of these concerns was noted in our case. Patients with longstanding comorbidities such as peripheral vascular disease or diabetes mellitus may be at an increased risk of further neurological injury from a secondary insult at another site. This double-crush phenomenon has been attributed to factors such as mechanical (needle trauma), ischemic (epinephrine-induced vasoconstriction), and toxic (local anesthetic neurotoxicity) [14]. Neurological complications may also result from a direct neurotoxic effect of local anesthetic agents that is concentration- and dose dependent [15]. Some neuraxial components may theoretically be more susceptible, such as cauda equina, because it is partially unmyelinated and has a large surface area. The spinal roots contained within the dural root cuffs lack the protective mechanical and metabolic milieu noted in peripheral nerves [16]. Rodent model studies have clearly demonstrated an increased risk of nerve fiber damage in diabetic rats, albeit with nerve blocks [4, 17]. Research in this vital area to provide definitive answers is still ongoing [8, 18].

Our patient developed a new-onset, bilateral, symmetrical neurological deficit in the lower limbs in the early postoperative period. The distribution of neurological deficit converged over L5 and S1 dermatomes and the posterior column in particular. This finding pointed toward a lesion causing bilateral L5 and S1 radiculopathy rather than a more distal unilateral peripheral nerve lesion related directly to the surgery or tourniquet application. The spinal anesthetic procedure was noted to be uneventful as the patient reported no symptoms (pain/paresthesia) while undergoing surgery. Neuroimaging with CT and MRI scans did not show any evidence of spinal cord damage or compression from a hematoma. Hence, neurophysiological evaluation was used to obtain diagnostic information. The neurophysiological tests (nerve conduction and electromyography) provide dual advantages in cases such as our patient. First, they help to establish a diagnosis. Second, they also help distinguish neurological damage related to medical procedures and preexisting asymptomatic neuropathy when new neurological symptoms appear after a medical procedure.

Neurological complications following 'quality of life'-improving procedures such as knee arthroplasty have far-reaching implications for the physicians, but more so for the patient. Establishing the definitive role of causative/risk factors in these cases can help minimize or potentially eliminate such complications altogether. Subclinical neuropathy in diabetic patients is a well-defined condition. However, there is a paucity of literature on the exact triggers that allow this condition to lead to profound neurological symptoms as occurred in our patient. Therefore, adequate preoperative counseling of diabetic patients undergoing elective surgical procedures under regional anesthesia regarding subclinical neuropathy is imperative and a crucial component of safe clinical practice. Our case highlights the fact that patients with longstanding comorbidities, namely peripheral vascular disease and diabetes mellitus, may be at an increased risk of neurological injury. Hence, we believe that preoperative evaluation of diabetic patients should include neurophysiological studies to identify subclinical neuropathy and minimize the risk of neurological injury.

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