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Case Report

Common Peroneal Nerve Palsy Associated with Intraoperative Hypotension Following Total Thyroidectomy

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Abstract

Perioperative peroneal neuropathy is an uncommon complication following surgeries performed with patients positioned supine. It may be caused by various factors aside from intraoperative compression. The authors report a case of common peroneal nerve palsy in a patient who underwent total thyroidectomy with central and bilateral selective neck dissection. The patient's body mass index was 31.3 kg/m2. She was positioned supine and the operative time was 7-h. During surgery, her mean arterial pressure intermittently dropped to 50-60 mmHg for 55 min and 61-70 mmHg for 195 min. She developed common peroneal nerve palsy on postoperative day 1. Nevertheless, the patient fully recovered without any complications within 3 weeks.

Keywords: Common peroneal nerve palsy; Hypotension; Supine position; Total thyroidectomy

Introduction

Common peroneal nerve palsy that occurs in the perioperative period is usually caused by compression, traction, or ischemia. Thus, peroneal neuropathy is an uncommon complication in patients who undergo surgery in the supine position. In the rare instances it does occur, most involve patients who spend extended periods in the intensive care unit following procedures such as liver transplantation or cardiac surgery, and/or those with risk factors for neuropathy.

In the case described here, the patient experienced common peroneal nerve palsy following total thyroidectomy with central and bilateral selective neck dissection. We believe that common peroneal nerve palsy is associated with prolonged surgery times, obesity, and lower extremity hypoperfusion due to intraoperative hypotension.

Case Report

A 51-year-old woman (height, 159 cm; weight, 80 kg) with thyroid cancer was admitted to hospital to undergo total thyroidectomy with central and bilateral selective neck dissection. Her Body Mass Index (BMI) was 31.3 kg/m2 and she had a 5-year history of primary hypertension, which was medicated with angiotensin receptor antagonist and thiazide diuretics. Other preoperative investigations were unremarkable.

After administration of intravenous glycopyrrolate (0.3 mg; Robinul, AH Robins Co, Richmond, VA, USA), the patient was transferred to the operating room. Pre-anesthetic blood pressure was 130/85 mmHg, heart rate was 79 beats/min, and oxygen saturation was 97%.

For induction of anesthesia, thiopental sodium 350 mg and rocuronium 70 mg were administered intravenously, and nerve integrity monitoring (Medtronic Xomed, Jacksonville, FL, USA) standard Electromyography (EMG) with a reinforced endotracheal tube was performed. Anesthesia was maintained using oxygen 2 L/min, nitrous oxide 2 L/min, and sevoflurane 1-2 vol%. For EMG monitoring, additional muscle relaxant was not used other than for induction.

The patient was placed in the reverse Trendelenburg position and tilted 10 degrees. Twenty-five minutes after initiation of surgery, the patient's blood pressure dropped to 70/40 mmHg, which recovered to 119/80 mmHg after injection of 10 mg ephedrine. The patient's Mean Arterial Pressure (MAP) intermittently dropped to < 60 mmHg for 55 min and, during this period, she was administered 3 injections of 10 mg ephedrine and 3 injections of 100 μg phenylephrine. The patient's MAP measured 60-70 mmHg for approximately 195 min, and was otherwise maintained at 70-90 mmHg. The total operation time was 7-h 10 min; estimated blood loss was 200 ml, urine output 350 ml and 2200 ml of fluid was administered to the patient.

On Postoperative Day (POD) 1, the patient complained of weakness in the left leg and pain in the ankle area, and left foot drop was clearly observed. Although there was no sensory loss-verified by neurological examination on POD 2-dorsi-flexion and eversion of the left ankle revealed Medical Research Council grade 4 each. A sensory and motor nerve conduction study was performed on POD 3. Results of the sensory nerve conduction study were normal, whereas the amplitude of the action potential in the motor nerve conduction study was decreased. The patient gradually recovered from POD 3 onward, and was discharged from hospital on POD 8. Three weeks after the

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operation, the patient visited the neurology department with drop foot, although fully recovered from pain. A scheduled nerve conduction study was not performed.

Discussion

The common peroneal nerve is an easily damaged structure in the lower extremity of the human body [1]. Common peroneal nerve palsy is caused by trauma, external compression of the knee or hip, and/or underlying neuropathy. In rare instances, peroneal neuropathy may manifest with no apparent causes [2]. Common peroneal nerve palsy during the perioperative period is usually caused by ischemia following compression or traction [1].

Descending the back of the fibula head, the common peroneal nerve surrounds the lateral side of fibula neck and branches to the superficial and deep peroneal nerve on the two heads of the peroneal longus muscle, being covered by skin and fascia, with only a 4 cm2 surface over the fibula head and neck. This surface is largely vulnerable to compression damage. The common peroneal nerve passes through the tunnel topped by the origin of peroneal longus muscle and intermuscular septum, of which the upper portion is the tibial head, and the lower portion is the upper lateral side of the tibial shaft. By limitation of movement along the tunnel, the common peroneal nerve can easily be damaged by extension forces [3].

For these anatomical reasons, the common peroneal nerve can be easily damaged by the pressure caused by a patient's position during the intraoperative period. In particular, 1-1.5% of patients positioned in the lithotomy position experience damage to the common peroneal nerve, with lateral and sitting positions also causing nerve damage [3,4].

Warner et al, reported that maintaining the lithotomy position for > 2-h is the most crucial factor contributing to perioperative common peroneal nerve palsy [3]. The authors further reported that other methods-aside from shortening the operation time-would not have a meaningful effect on the risk of peroneal neuropathy. In our case, the 7 h operation time was a major contributor to nerve palsy.

Ashish et al, conducted a retrospective investigation involving 132 patients who underwent liver transplantation; 5.3% of all patients experienced common peroneal nerve palsy in the postoperative period, a higher figure than that reported by Warner et al., [3,5]. This result demonstrates that, other than patient position during surgery, individual characteristics are also a risk factor for nerve palsy. These individual risk factors include older age, BMI < 20 kg/m2, obesity, diabetes, alcohol abuse, smoking, peripheral vascular disease, hypotension, underlying peripheral neuropathy and poor nutrition, among others [1,3-8].

Patients with a thin body type and BMI < 20 kg/m2 are susceptible to damage or pressure on the nerve because subcutaneous fat that could act as a cushion is diminished [6]. In contrast, obese patients can experience neuropathy because body metabolism has changed due to hyperinsulinemia and low insulin sensitivity. Those who are obese without diabetes can experience subclinical peripheral neuropathy caused by increased insulin resistance [7].

Yi et al, reported a case involving a patient who experienced common peroneal nerve palsy, after undergoing anterior cervical vertebrae surgery performed in the supine position lasting 3.5-h, even though the patient had no pressure or traction delivered to the nerve, nor risk factors for neuropathy [9]. Thus, the authors concluded that prolonged immobilization during surgery could also be a cause of nerve palsy.

Teeples et al, reported a case of peroneal nerve palsy and lower extremity compartment syndrome following maxillofacial surgery [10]. The authors reported that the most likely cause was a combination of arterial hypotension in a leg that likely sustained some type of presurgical injury with slightly elevated intracompartment pressure. The lower limit for cerebral blood flow autoregulation occurs at a MAP of 60-70 mmHg. However, this also applies to patients with normal blood pressure. For hypertensive patients, the cerebral blood flow autoregulation curve shifts to the right. In our case, MAP was measured to be < 60 mmHg for a total of 55 min and 61-70 mmHg for a total of 195 min. Considering that the patient was hypertensive, we conclude that the hypoperfusion state lasted for an extended period.

Overall, we conclude that various factors, including a 7-h surgery, immobility, obesity, and poor perfusion of the lower extremity due to hypotension, collectively contributed to common peroneal nerve palsy in the present case.

In conclusion, common peroneal nerve palsy in the perioperative period may occur regardless of operative position and/or type; however, proper positioning, changing body position regularly, and applying padding to the compressed area are crucial measures in preventing nerve damage. In particular, when prolonged surgery is anticipated, patients with risk factors for neuropathy, such as thin body type, obesity, diabetes, underlying neuropathy, peripheral vascular disease, alcoholism, smoking history and hypotension, should be given a sufficient explanation from the medical staff and provide informed consent before surgery.

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