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Femoral Neuropathy after Reconstruction of a Defect Due to Buccal Cancer Excision Using a Thigh Flap: A casereport

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Femoral Neuropathy after Reconstruction of a Defect Due to Buccal Cancer Excision Using a Thigh Flap: A Case Report

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A case of femoral neuropathy as a complication of flap excision for reconstruction of a defect due to buccal cancer excision is presented. A 51-year-old man developed numbness over his left anteromedial thigh and medial lower leg, left quadriceps weakness, and absence of left knee jerk after a flap was obtained from his anterolateral thigh. Electromyographic examination revealed evidence of denervation in the left quadriceps. A nerve conduction study showed prolonged latency, reduced amplitude of the compound muscle action potential, decreased velocity in the left femoral nerve and prolonged latency, reduced amplitude of the sensory nerve action potential, and decreased conduction velocity in the left saphenous nerve. It is suggested that this complication was caused by traction of the femoral nerve when the free antero-lateral fasciocutaneous flap was dissected. The complication may be preventable by avoiding overstretching the femoral nerve during dissection or by application of intraoperational electrodiagnostic monitoring. (Tw J Phys Med Rehabil 2005; 33(3): 171 - 177)

Key words: femoral neuropathy, flap, buccal cancer

INTRODUCTION

The development of femoral neuropathy secondary to surgical procedures has been documented in a variety of case reports, mostly following pelvic or inguinal surgical procedures.[1-10] This lesion has rarely been found below the inguinal ligament. Femoral neuropathy due to a thigh flap excision has not been reported. We describe a case of electromyographically (EMG) documented, isolated femoral nerve injury following a thigh flap procedure for reconstruction of a defect due to buccal cancer excision. This case demonstrates the need for further clinical and electrodiagnostic evaluation when weakness in knee extension is seen in this setting.

CASE REPORT

A 51-year-old man was admitted to a medical center because of an ulcer in the left corner of his mouth. He consumed alcohol (1/2 bottle/day) for 4-5 years, betal nuts (20 grains/day) for more than 20 years, and cigarettes (1 package/day) for 30 years. He had no history of other systemic diseases. His body height was 164 cm and

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body weight was 44 kg. Biopsy of the buccal ulceration revealed squamous cell carcinoma. Under general anesthesia, he underwent wide excision of the tumor with lymph node dissection and then reconstruction with a free anterolateral thigh fasciocutaneous flap. The flap was obtained via left mid-anterior thigh longitudinal skin excision and dissection under the subfascial plane. The pedicle was supplied by the transverse branch of the lateral circumflex femoral artery. The flap size was 9.5 x 9 cm^2. The operation lasted 7 hours and 20 minutes totally. The excision with lymph node dissection took 4 hours and 5 minutes and the flap excision and reconstruction took 3 hours and 15 minutes. During the operation, the patient was in the supine position. Total blood loss was about 100 ml. According to the surgeon's report, there was no damage to the vessels supplying femoral nerve. After the operation, the patient received adjuvant radiotherapy.

Unfortunately, two months after the operation, the patient complained of left lower extremity weakness and numbness over the left anteromedial thigh and medial lower leg without involvement of lateral thigh, which is innervated by the lateral femoral cutaneous branch of the femoral nerve. He stated that the numbness had begun one week after the operation. Because his left lower extremity was rather swollen, painful, and covered with a dressing, he spent most of his time during the first two postoperative weeks lying in bed. Afterwards, his activity level improved and he could sit up and ambulate functionally. He felt left knee weakness one month after the operation when he began community ambulation training. The weakness was debilitating. According to his surgical chart, his left quadriceps muscle power was 3/5.

Thus, the patient was referred to the rehabilitation clinic for treatment of persistent left knee weakness. Although he had difficulty extending the left knee, he could stand up and walk independently. Manual muscle testing revealed 3/5 in left knee extension, and 5/5 elsewhere. Deep tendon reflex of the left knee was absent. Sensory examination revealed decreased pinprick and light touch sensations over the left anterolateral thigh and left medial lower leg. He denied any bowel or bladder dysfunction or back pain.

Electrodiagnostic testing commenced three months after the operation (Tables 1 and 2). Nerve conduction velocity (NCV) study revealed mildly prolonged distal latency in the left common peroneal nerve; other findings were within normal limits. Needle electromyogram (EMG) examination revealed mildly increased positive sharp waves (PSWs) in left vastus lateralis muscle and severely increased PSWs in left rectus femoris muscle. No motor unit action potential (MUAP) was detected in the left vastus lateralis muscle. Severely reduced MUAP amplitude and decreased motor units recruitment ratio and interference pattern were found in the left rectus femoris muscle. EMG in left tibialis anterior muscle and gastrocnemius muscle were normal. Because of inexperience, NCV study of the bilateral femoral nerves and saphenous nerves was not done at that time. Room temperature of the examination room was around 22°C, but skin temperature of the patient was not recorded.

Follow-up electrodiagnostic testing was done six months after the operation. NCV of the bilateral femoral nerve and saphenous nerve was determined (Table 3). The compound muscle action potentials of bilateral femoral nerves were recorded from the vastus medialis muscles (surface pickup), and stimulation with surface electrodes was applied just above the inguinal ligament, lateral to the femoral artery, and at Hunter's canal. The study found mildly decreased nerve conduction velocity, markedly reduced amplitudes of compound muscle action potentials, and prolonged latencies above Hunter's canal on the left side. The sensory nerve action potentials of the bilateral saphenous nerves were recorded from the ankle, between the medial malleolus and the tibialis anterior tendon (surface pickup). Stimulation with surface electrodes was applied 14 cm proximal to the recording electrode, between the medial belly of the gastrocnemius and the tibia. The study revealed markedly decreased nerve conduction velocity, markedly reduced amplitudes of sensory nerve action potentials, and prolonged distal latency in left saphenous nerve.

EMG examination (Table 4) demonstrated 2+ PSWs, increased duration of MUAPs, increased percentage of polyphasia, decreased motor units recruitment ratio, and decreased interference pattern in left rectus femoris muscle. 1+ PSWs were found in left vastus lateralis with no MUAP detected. Normal EMG findings were observed in left tibialis anterior, tensor fascia lata, iliopsoas, adductor magnus, paraspinal, and left biceps femoris muscles.

The patient did not receive physical therapies be-

cause of lack of time, but an active strengthening program was conducted at home. However, prolonged weakness and disuse led to left quadriceps atrophy and left knee pain.

DISCUSSION

The femoral nerve, which is formed by the L2-L4 roots, penetrates the psoas muscle and exits the pelvis by passing beneath the medial inguinal ligament to enter the femoral triangle just lateral to the femoral artery and vein. Approximately 4 cm proximal to the inguinal ligament, the femoral nerve is covered by a tight fascia at the iliopsoas groove. The femoral nerve provides the motor supply to the quadriceps femoris, pectineus and sartorius muscles. It also supplies sensory innervation to the anterior aspect and part of the medial aspect of the thigh, and the skin over the tibial surface of the leg down to the medial malleolus.

Postoperative femoral neuropathy has been reported after various surgical procedures (such as abdominal hysterectomy,[1] vaginal hysterectomy,[2] renal transplantation,[3,4] microsurgical tuboplasty,[5] aortic surgery,[6] and inguinal herniorrhaphy[7]), iliopsoas hematoma,[8-10] and tourniquet use.[11,12] Most cases were considered to be caused by direct compression of the femoral nerve by surgical instruments, such as self-retaining retractors and lateral blades.[1,3-7] Other possible causes of compression include iliopsoas hematoma,[8-10] tourniquet use[11,12] and sequestrum of bone cement.[13] Femoral neuropathy, either unilateral or bilateral, can also occur when an operation is performed in a lithotomy position.[2,14,15] The most likely cause is entrapment of the femoral nerve at the inguinal ligament. This position may allow exaggerated flexion, abduction, and external rotation of the hip joints and could lead to microvascular and local mechanical injury. In all the cases mentioned above, femoral neuropathy was mainly caused by focal compression, localized above or under the inguinal ligament. Most reported cases were based on clinical symptoms/signs and not electromyographic data. However, neurapraxia and axonotmesis were the most likely pathologies. Persistent paresis occurred in only a few of the cases.[11]

Anterolateral thigh flap for head and neck reconstruction had been widely used until recently.[16]In the dissection of the flap pedicle and the cutaneous perforator, a longitudinal skin incision is made directly above the rectus femoris muscle. The dissection is carried out medially about 1 to 2 cm in a subcutaneous plane. The deep fascia is then incised and secured to the dermis by transfixation sutures to preserve the prefascial plexus. The thigh skin is then raised and retracted laterally by a subfsscial plane dissection, which will expose the septocutaneous or musculocutaneous perforators. The motor branches of the femoral nerve innervating the vastus lateralis muscle, which is always accompanied by the descending branch of the lateral circumflex femoral artery, is well preserved. The lateral femoral cutaneous nerve is also preserved where possible (Figure 1).[17] However, during the operation, which takes an average of 1–2 hours, rectangle retractors are used to increase the operation field. The positions of the retractors were not fixed during the operation but were moved along the thigh to increase the surgical field.

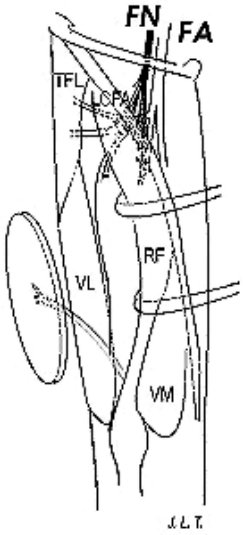

Figure 1. Illustrations of the distally based anterolateral thigh flap with different perforators. FA, femoral artery; FN, femoral nerve; LCFA, lateral circumflex femoral artery; VM, vastus medialis muscle; VL, vastus lateralis muscle; TFL, tensor fascia lata muscle; RF, rectus femoris muscle. Modified from Plast Reconstr Surg. 2004; 114:1768-75[20]

Peripheral nerve trunks have viscoelastic properties. Haftek et al reported the relationship between load and elongation during gradual stretching of the rabbit tibial nerve to the point of complete rupture.[18] The first structure to rupture is the epineurium, when the elastic limit is reached. The diameter of the fascicles decrease with obliteration of the endoneurial spaces, axonal narrowing, and myelin disruption. The next events are rupture of the perineurial sheaths, interruption of nerve fibers within the fascicles, and finally complete severance of the nerve. The endoneurium never becomes disrupted before the perineurium, and the perineurium never before the epineurium. The longitudinal extent of damage is considerable and is not confined to a single localized lesion. The pattern of damage is essentially the same for gradual and sudden stretching. Milder degrees of traction injury may produce no more than a reversible conduction block. More severe injury may involve a considerable length of the nerve.[19]

In the second electrodiagnostic examination of our patient, the distal latency of the left femoral nerve was prolonged relative to the normal side when stimulation was above and below the inguinal ligament, but the latency was the same when stimulation was at Hunter's canal. Compound muscle action potential amplitude was decreased to one third that of the normal side. Motor NCV was normal in the segment spanning the inguinal ligament, but was three quarters that of the normal side in the segment distal to the inguinal ligament and proximal to Hunter's canal (Table 3). Distal latency of saphenous nerve was prolonged, sensory nerve action potential amplitude decreased up to a quarter, and sensory NCV decreased to one-half on the deficient side relative to the normal side (Table 3). From the NCV data, it was concluded that the slow conduction segment was located between the inguinal ligament and Hunter's canal. EMG showed that active denervation was in progress in the left vastus lateralis and rectus femoris muscles. Decreased MUAP amplitude associated with decreased motor unit recruitment ratio was found in left rectus femoris muscle. Moreover, no MUAP amplitude in left vastus lateralis muscle was detected by the time we examined the patient (Table 4). On the basis of the electrodiagnostic data, compression injury was less likely because (1) compression of nerves usually results in selective segmental demyelination of larger fibers without severe axonal damage unless the compression is chronic, and because (2) decreased NCV indicates the lesion should be more localized. In our patient, the lesion was large (extending from above the inguinal ligament to Hunter's canal), and severe axonopathy was present. Ischemic injury was also less likely because blood loss was minimal and, according to the surgeon's report, the vessels supplying femoral nerve were not damaged during the operation. On the basis of the electrodiagnostic data, the femoral neuropathy in our case was most likely due to traction injury by the retractor. The complication may be preventable by avoiding overstretching the femoral nerve during surgery or by application of intraoperational electrodiagnostic monitoring.

Table 1. Motor NCS three months after the operation

Nerve	Sites	Recording site	Lat. (ms)	Amp. (mV)	Segments	Lat. diff. (ms)	Dist. (cm)	Vel. (m/s)
Left common peroneal	1.Ankle	EDB	5.15	5.2	1-0	5.15		
	2. Fibular head		11.50	5.1	2-0	6.35	28	44.1
Left tibial	1. Ankle	AH	3.75	13.0	1-0	3.75		
	2. Popliteal fossa		11.45	8.1	2-0	7.70	34	44.2

EDB: extensor digitorum brevis, Lat: latency, Amp: amplitude, Lat Diff: latency difference, Dist: distance, Vel: velocity, and AH: abductor hallucis.

Table 2. Needle EMG of the left lower extremity 3 months after the operation

	Spontaneous activity					Motor unit action potential				
	IA	Fib	PSW	CRD	Fasc	Amp	Dur.	Poly	MUR	IP
Rectus femoris	1+	0	3+*	0	0	500uv	N	<15%	Discrete	3-
Vastus lateralis	1+	0	1+*	0	0	0	0	0	0	0
Tibialis anterior	N	0	0	0	0	N	N	<15%	N	>80%
Gastrocnemius	N	0	0	0	0	N	N	<15%	N	>80%

IA: insertional activity, Fib: fibrillation, PSW: positive sharp wave, CRD: complex partial discharge, Fasc: fasciculation, Amp: amplitude, Dur: duration, poly: polyphasia, MUR: motor unit recruitment, IP: interference pattern, and MUAP, motor unit action potential.
1+: mildly increased, 2+: moderately increased, and 3+: severely increased.
1-: mildly decreased, 2-: moderately decreased, and 3-: severely decreased.
*: RECTUS FEMORIS: PSW: 400uv, MUAP amplitude was 500uv.
*: VAST LATERALIS: PSW: 100uv, no MUAP was detected.

Table 3. Motor and sensory NCS six months after the operation

Nerve	Sites	Recording site	Lat. (ms)	Amp. (mV)	Segments	Lat. diff. (ms)	Dist. (cm)	Vel. (m/s)
Left femoral	1.A. Ing.Canal	V. M.	7.25	1.4	1-0	7.25		
	2. B. Ing.Canal		6.40	1.3	2-0	6.40		
	3. H. Canal		4.30	1.4	3-1	-2.95	13.5	45.8
Right femoral	1. A. Ing.Canal	V. M.	6.30	5.3	1-0	6.30		
	2. B. Ing.Canal		5.25	5.6	2-0	5.25		
	3. H. Canal		4.20	5.2	3-1	-2.10	12.5	59.5
Left saphenous	Medial leg	Ankle	(peak) 3.95	(peak) 1.4		3.95	14	35.4
Right saphenous	Medial leg	Ankle	(peak) 2.20	(peak) 6.3		2.20	14	63.6

A: above, B: below, H: Hunter's, V. M.: vastus medialis, Lat: latency, Amp: amplitude, Lat diff: latency difference, Dist: distance, and Vel: velocity.

Table 4. Needle EMG of left lower extremity six months after the operation

	Spontaneous activity					Motor unit action potential				
	IA	Fib	PSW	CRD	Fasc	Amp	Dur.	Poly	MUR	IP
Rectus femoris	N	0	2+*	0	0	3mv	2+	3+	1-	2-
Vastus lateralis	N	0	1+**	0	0	0	0	0	0	0
Iliopsoas	N	0	0	0	0	N	N	<15%	N	>80%
Tibialis anterior	N	0	0	0	0	N	N	<15%	N	>80%
Tensor fasciae latae	N	0	0	0	0	N	N	<15%	N	>80%
Adductor magnus	N	0	0	0	0	N	N	<15%	N	>80%
Lumbar paraspinal	N	0	0	0	0	N	N	<15%	N	>80%
Biceps femoris	N	0	0	0	0	N	N	<15%	N	>80%

IA: insertional activity, Fib: fibrillation, PSW: positive sharp wave, CRD: complex partial discharge, Fasc: fasciculation, Amp: amplitude, Dur: duration, poly: polyphasia, MUR: motor unit recruitment, and IP: interference pattern.
1+: mildly increased, 2+: moderately increased, and 3+: severely increased.
1-: mildly decreased, and 2-: moderately decreased.
*: 50uv.
**: 100-200uv, no MUAP was detected.

CONCLUSION

This report presents a case of femoral neuropathy after thigh flap excision. Postoperative femoral neuropathy is not uncommon, however, it is rarely localized below the inguinal ligament. In operations involving the thigh area, surgeons must pay attention to the possibility of overstretching surrounding tissues. If patients have quadriceps weakness and sensory complaints, femoral neuropathy should be considered even if the operation is not performed in the lithotomy position or without the use of retractors. Electrodiagnostic study is indicated when postoperative neuropathy is suspected. It is suggested that this complication was caused by traction of the femoral nerve during the dissection of the free antero-lateral fasciocutaneous flap.

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大腿皮瓣重建口腔癌術後之股神經病變：病例報告

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一位口腔癌病人接受局部腫瘤切除合併大腿皮瓣重建術後，併發股神經病變。出現的症狀是取皮瓣側肢體的麻木感及膝蓋伸展無力。理學檢查顯示肌力下降及深層肌腱反射消失。電學檢查顯示患肢股神經的感覺及運動神經傳導速度下降，且股神經支配的肌群有急性去神經化現象產生。此種現象可能是由於在取皮瓣的過程中拉扯到股神經所致。預防的方法是避免在取皮瓣時對股神經做長時間的壓迫。尤其病人體型瘦小時更要注意。（台灣復健醫誌 2005; 33(3): 171 - 177）

關鍵詞：股神經病變(femoral neuropathy)，皮瓣(flap)，口腔癌(buccal cancer)

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