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Femoral Nerve Injury Following Transfemoral Angiography: A Case Report

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Femoral nerve injury is a rare complication after femoral angiography. Symptoms including quadriceps weakness and atrophy, absent or reduced knee jerk and sensory impairment along the anterior thigh and medial lower leg have been found. This leads to both muscular weakness and sensory deficit, which impairs walking and interferes with quality of life. Early identification and management of femoral nerve injury could result in better prognosis.

We report on the history, diagnosis and rehabilitative outcome of a 54-year-old woman with a medical history of peripheral arterial occlusion disease. She developed significant femoral neuropathy one month after angiography. Severe neurological deficits were noted in this patient, including quadriceps paralysis and loss of sensation at the anterior aspect of the thigh. Subsequent above-knee amputation was performed due to failed conservative treatment for arterial occlusion. After about six months of intensive rehabilitation, the patient achieved clinical improvements in her functional locomotion activities. We also discuss the femoral nerve anatomy, incidence of iatrogenic femoral nerve injury, puncture-site complications in transfemoral angiography, evaluation and treatment of femoral nerve injury and our rehabilitation experience with femoral neuropathy combined with contralateral above-knee amputation. (Tw J Phys Med Rehabil 2008; 36(4): 227 - 234)

Key Words: femoral nerve, angiography, rehabilitation

INTRODUCTION

Femoral nerve injury has been reported with different conditions, including hip replacement, femoral vessel catheterization, obstetric and gynecological procedures, general and urological surgery explorations, ilioinguinal nerve blocks, and hematological or neoplastic conditions. ^[1-5] Among these conditions, femoral vessel catheterization is a commonly performed procedure for both diagnostic and therapeutic interventions, including the angiography. Femoral nerve injury is a rare complication after femoral angiography. However, it leads to both muscular weakness and sensory impairment, which

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impairs walking and interferes with quality of life. Early identification and management of femoral nerve injury could result in better prognosis.

We report on a 54-year-old woman who received angiography via the left femoral artery owing to peripheral arterial disease over the right leg and subsequently received right above-knee amputation. However, left femoral nerve palsy was noted after the amputation. Combined above-knee amputation in one leg and femoral nerve palsy in the contralateral leg is not common and it may complicate the ambulation training and further rehabilitation process.

CASE REPORT

This 54-year-old woman had a history of diabetes and hypertension under medical control for 9 years. She could walk independently without leg weakness. Due to peripheral arterial occlusion disease (PAOD), she rceived surgical intervention with artery-bypass for right lower leg (femoral to popliteal artery and popliteal to dorsal pedis and posterior tibial artery) on July 28, 2005. Owing to breast cancer, she received surgical intervention in June 2006. She also had a history of smoking for more than 3 years and quit 2 months previous.

On July 28, 2006, she was brought to the ER because of severe, persistent right leg pain that had been growing for two days. On physical examination, she presented weak pulsation of the right popliteal artery and the dorsal pedis artery but no swelling. Digital subtraction angiography (DSA) via the left femoral artery revealed occlusion of the right superficial femoral artery (SFA), multiple segmental narrowing in the left SFA, near occlusion of the right tibial artery, and thrombotic formation in the graft (Figure 1). Peripheral arterial occlusion disease with re-stenosis was diagnosed. Thrombectomy was performed on the same day. After the thrombectomy, a high dose of heparin was given. Poor wound healing and severe swelling below the right knee were noted. Subsequently, cyanosis and swelling were noted over the right lower leg. Right above-knee amputation was done on August 4, 2006. One week after the amputation, she accidentally found her left knee weak on extension with loss of sensation in the anterior aspect of the left thigh when getting up for toileting. She was bedridden after the

admission. The accurate date at which these symptoms began is unknown. The surgeon performed no further management with only observation.

Two months after the amputation procedure, she was transferred to our ward for post-amputation rehabilitation. Compared with the acute stage, the symptoms persisted without deterioration or improvement. Upon physical examination, weakness occurred on left knee extension, absent left knee jerk with loss of sensation over the anterior aspect of the left thigh were noted. Light touch and pinprick tests showed impairment over the left anterior and medial thigh. Manual muscle testing of her left knee extension was only trace grade (Table 1). Electrophysiological examinations, including nerve conduction study (NCS) and electromyography (EMG) were performed on October 24, 2006 for her persistent weakness and impaired sensation. Markedly increased spontaneous activities with decreased recruitment were detected at the left rectus femoris muscle, but sparing the iliopsoas muscle (Table 2 & 3). No electrophysiologic activity was obtained at the left vastus medialis because of severe musculature atrophy. Mildly delayed conduction velocity was noted at both the tibial and peroneal nerves compatible with late stage diabetes. Therefore, new onset of left femoral nerve mononeuropathy was concluded. The lesion might be located below the inguinal ligament due to iliopsoas muscle sparing. On assessment, we concluded the chief problem as: (1) lower extremity weakness, (2) phantom pain occurrence over the right lower limb, (3) inability to transfer, stand and ambulate, (4) absence of right lower limb below the knee after the amputation, (5) left knee extension with genu recurvatum. The goals were to strengthen the muscle, relieve the phantom pain, retrieve the ability to ambulate and improve the quality of daily activity. For the lower extremity weakness, we added a strengthening program of gentle resistance exercises to muscles with MMT grades above F+ with monitoring for overwork fatigue and modality, IFC and electric stimulation, to muscles with manual muscle testing (MMT) grades below fair. For her phantom pain, desensitization techniques such as massaging, tapping, slapping, wrapping and friction rubbing were prescribed. For the inability to transfer, the training procedure begins with the easiest transfer and progresses to the more difficult transfer. Instructions for transfers begin on level

surfaces and progress to uneven surfaces as individual strength and skill allow. Prosthesis fabrication with pre-walking training was also performed. We spent much time improving her standing balance and ambulation ability. A gaiter was given in order to avoid genu recurvatum. After rehabilitation for about 3 months, the strength of left knee extension increased to achieve antigravity activity. She could walk with a right side above-the-knee prosthesis, using a walker under supervision. Independent wheelchair skill was also noted. A follow-up electrophysiological examination was performed on February 6, 2007. The result was similar to that obtained in the previous study. The clinical improvement was believed due to the accumulative result of functional training.

Muscles		MRC scale
Left hip	flexors	4
	extensors	4
	abductors	4
	adductors	4
Left knee	flexors	5
	extensors	1
Left ankle	dorsiflexors	5
	plantar flexors	5
Left long toe extensors		5

Table 1. Muscle strength of left lower extremity on admission

Abbreviation: MRC scale, Medical Research Council scale



Figure 1. Angiographic images showing (A) occlusion of right superficial femoral artery (arrowhead) and (B) segmental narrowing on the left superficial femoral artery (arrow).

Motor nerve conductions						
Nerves Record	Stimulation site	Latency (ms)	Amplitude (mV)	Conduction velocity (m/s)		
L Peroneal to EDB	Ankle	5.0	1.0	42.4		
	Fibular head	12.55	1.0			
L Tibial to AH	Ankle	4.95	3.9	38.7		
	Popliteal fossa	14.00	4.6			
L Femoral to VM	Inguinal ligament	Absent (seve	ere atrophy)			

Table 2. Resu	ult of nerve	conduction	study
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Abbreviation: L, left; EDB, extensor digitorum brevis; AH, abductor hallucis; VM, vastus medialis.

Electromyography							
Muscle	Sponta	aneous	Motor units action potential				
	Fib	PSW	Amp	Dur	Poly	Recruit	
L Vastus medialis	Absent		Absent				
L Rectus femoris	2+	3+	Decreased	Small	None	Reduced	
L Gluteus medius	0	0	Normal	Normal	Normal	Full	
L Iliopsoas	0	0	Normal	Normal	Normal	Full	

Table 3. Result of electromyography

Abbreviation: L, left; Fib, fibrillation; PSW, positive sharp wave; Amp, amplitude; Dur, duration; Poly, polyphasic motor unit potentials; Recruit, recruitment.

DISCUSSION

Angiography has been an important and commonly prescribed diagnostic approach for vascular lesions. The transfemoral approach is the most common route for percutaneous arterial access for angiography and has the lowest complication rate. The overall complication rate for the transfemoral approach is 1.73%, including cardiac, systemic, neurologic, puncture-site, and wire-catheter complications.^[6] Among these causes, puncture-site complications occurring with an incidence of 0.47% were the most common, including hemorrhage, arterial obstruction, pseudoaneurysm, arteriovenous fistula and limb amputation. Hemorrhage was the leading cause of puncture-site complication.^[6] Hemostasis at the puncture-site was commonly achieved by manual compression followed by a period of recumbency. This is especially important in anticoagulated patients to prevent hematoma formation and further complications.

The femoral nerve arrives from the lumbar plexus and derives from the L2-L4 spinal nerve roots. It passes through the psoas major muscle, descending between the iliacus and psoas muscle deep into the iliac fascia to reach the middle of the inguinal ligament in the pelvis. It then goes laterally into the femoral sheath in the thigh. It innervates the iliacus, sartorius and quadriceps muscles. Its terminal cutaneous branches innervate the anterior thigh sensation (via the anterior femoral cutaneous nerve) and medial distal leg (via the saphenous nerve).^[7] The iliolumbar artery, a branch of the internal iliac artery, supplies the femoral nerve in the pelvis. The lateral and medial circumflex femoral arteries contribute to the blood supply for the femoral nerve in the thigh. The blood supply to the right femoral nerve is stronger than that on the left side. This may explain why there is a high incidence of ischemic injury in the left femoral artery.

Weakness of extension in the affected knee from quadriceps denervation, quadriceps wasting, absent or reduced knee jerk and loss of sensation in the leg and thigh could be found in patients with femoral nerve palsy.^[8] The incidence of femoral nerve palsy has been reported as 2.2% of cases after renal transplantation, 3% of cases after total hip arthroplasty, 7% to 12% of cases after abdominal hysterectomy, 3.4% after aortic aneurismal repair and 0.21% of cases after cardiac catheterization.^[1,9,10] Many reasons including compression, stretching, ischemia and iliopsoas hematoma could cause femoral nerve injury. Because of the anatomical location, the most common etiology of femoral neuropathy is a mass lesion, mostly a hematoma, located in the iliopsoas muscle causing either direct compression or local ischemia of the femoral nerve.^[11] Hematomas are occasionally noted after femoral catheterization. Although most of these hematomas are small and asymptomatic, moderate or pronounced hematomas have been reported in up to 4% of hospitalized patients after femoral catheterization, and delayed bleeding has been reported in 1.2%.^[12] It was also observed that in 0.15% patients who underwent a femoral artery cannulation with retroperitoneal hematoma had ipsilateral femoral nerve palsy. There was increasing risk in these patients of simultaneously acquiring severe

coagulopathy or systemic anticoagulation.^[13] Complications with psoas muscle hematoma have been reported occurring in 7% of patients treated with anticoagulants.^[7] In our case, she received femoral artery angiography via the left femoral artery in the inguinal area. After the vessel procedure, she received a systemic anticoagulant agent with a high dose of heparin. Iliopsoas hematoma occurrence after the angiography with femoral nerve compression is highly suspected. According to our electrodiagnostic studies, left femoral nerve mononeuropathy with iliopsoas muscle sparing was noted. This may indicate that the lesion site was below the inguinal ligament, which is compatible with the femoral angiography puncture site.

One may think that her femoral neuropathy may be the presentation of diabetic neuropathy, peripheral arterial occlusion disease, or the complication of amputation. First, although she had diabetes mellitus for 9 years and mildly delayed conduction velocity was noted at both the tibial and peroneal nerves in our nerve conduction study, she had neither subjective sensory complaint nor weakness over the upper limbs during the whole course. Diabetic neuropathy symptoms often occurred progressively, but her femoral neuropathy was noted suddenly. Second, the femoral nerve received blood supply from iliolumbar artery in the pelvis and from lateral and medial circumflex femoral arteries in the thigh. According to our digital subtraction angiography, there was no stenosis over these arteries. For the above reasons, we do not prefer diabetic neuropathy or peripheral arterial occlusion disease as the cause of her femoral neuropathy. According to Celebrezze JP et al, postoperative femoral neuropathy has received little attention in the general surgical literature.^[3] The complication, however, has been documented in both the urological and gynecological literature, most commonly after abdominal hysterectomy and other pelvic surgeries. Seldom have reports argued that postoperative femoral neuropathy resulted from amputation surgery in the present. Therefore, in our case, the possibility of postoperative femoral neuropathy is not zero but low.

If the femoral nerve palsy resulted from hematoma, pseudoaneurysm, or extrusion of prosthetic elements, radiographic studies including X-ray, ultrasound and computed tomography scanning were suggested. Electrodiagnostic studies were suggested to be performed 6 weeks and 3 and 6 months after the comment on treatment.^[10] Because the presenting symptoms were subtle in our case, CT scan was not arranged during the acute stage.

However, it is still controversial to know what is the best treatment for femoral nerve palsy due to iliacus hematoma. Holscher reported on a good nerve injury progression following percutaneous drainage in their cases.^[14] However, a young man has been reported to have had full recovery after conservative treatment for 6 weeks after a large hematoma caused incomplete femoral nerve palsy.^[15] In another study, 31 patients with various etiologies for femoral neuropathy received axonal loss estimates.^[16] The investigator presented that patients with axonal loss of less than 50% have improved within 1 year. Fewer than half of the patients with axonal loss greater than 50% improved with conservative management alone. They concluded that irrespective of the cause of femoral mononeuropathy, functional improvement could be achieved in 2 out of 3 patients within 2 years of onset, but no later than this and that the axonal loss estimate is the only factor influencing prognosis. Ducic et al^[10] considered that iatrogenic and idiopathic femoral nerve lesions should be followed clinically and electrodiagnostically for 3 to 6 months after a vigorous physical therapy. If there is no improvement on physical examination or electrodiagnostic study after 3 to 6 months, surgical intervention was recommended. In another study, Tamai et al^[17] reported on the outcome for 24 patients with femoral neuropathy. Fourteen of 19 patients who received surgery had a complete neurological recovery. With conservative treatment, 3 of 5 patients had a complete neurological recovery. Both groups had a similar outcome. In our case, she only received rehabilitation for 3 months. Although the follow-up electrophysiological examination revealed no neuroregeneration of the femoral nerve injury, manual muscle testing of knee extension improved from trace to poor grade. Further recovery of the femoral nerve injury could be expected.

Generally, post-amputation management includes wound care, edema control postoperative pain issue and functional rehabilitation. Our patient had an adequate wound condition and no edema in the residual limb. She complained of phantom pain, especially in the morning. The pain subsided with the use of desensitization tech-

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niques such as massaging, tapping, slapping, wrapping and friction rubbing. To prevent hip flexion from contracture, proper positioning was performed. Groah et al considered that for femoral nerve injury ROM exercises, strengthening exercises, gait and balance retraining, use of orthoses and focused modalities facilitate functional recovery.^[18] In our patient, we offered the same rehabilitation program. She obtained improvement in the aspects of standing balance, transferring ability and ambulation.

However, a problem that we faced was how to perform ambulation training in patients with both one leg amputation and contralateral femoral nerve injury. The quadriceps muscle plays an important role in the mid-stance phase and swing phase during the gait cycle. In this patient, genu recurvatum in the left leg was noted in the mid-stance phase due to her weak left knee extension. We therefore applied a knee gaiter to avoid genu recurvatum. In contrast, there was no obvious problem in swing phase during the gait cycle. It is possible that the iliopsoas muscle may compensate for the weakness in the quadriceps muscle. To achieve a better gait pattern, we arranged muscle-strengthening exercises for the bilateral hip flexor, abductor, adductor, extensor and left knee extensor and flexor. We also prescribed a right aboveknee prosthesis and a walker for her. With these devices, she could walk step by step under supervision. She could also use wheelchair well after the wheelchair activity training. This could compensate for the high energy cost in long-distance ambulation. Adequate rehabilitation indeed improved her functional status, although she did not receive any surgical intervention.

CONCLUSION

Femoral nerve injury secondary to surgery and iatrogenic procedure is an infrequent disease and often missed because of its insidious presentation. We reported this case to emphasize that femoral neuropathy can result as a complication after an iatrogenic procedure. Amputation combined with contralateral weak knee extensor seldom occurred. We also reported our experiences in performing rehabilitation for a patient with both amputation and femoral nerve injury.

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血管攝影術後併發股神經損傷:病例報告

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因各項治療或診斷性手術操作而導致股神經損傷,可能源自不同情形:包括髖關節置換術、婦產科 手術、泌尿系統手術、鼠蹊部神經阻斷術、腫瘤術後,以及血管攝影術後等。由於血管攝影術是一項臨 床上經常執行的必要診斷或治療步驟,然而造成股神經損傷併發症的病例卻很罕見,引起的症狀包括股 四頭肌萎縮無力、膝部深部肌腱反射減弱、大腿前側及小腿内側感覺缺損等。早期診斷與治療,有助於 患者的復原。

我們報告一名 54 歲女性患者,有糖尿病、高血壓及乳癌術後的病史。一年前右側小腿血管罹患阻塞 性病變,接受過動脈繞道(bypass)手術治療,此次因為右小腿疼痛就醫,理學檢查發現右側膝膕動脈及足 背動脈脈動微弱,因此安排血管攝影,經由左側股動脈進行攝影檢查。由於嚴重血管阻塞,經過内膜切 除術及肝素投予治療後無效,小腿逐漸壞死,因而一週後進行右側下肢膝上截肢。術後傷口復原良好, 但是左下肢膝部無力伸直且大腿前側感覺缺失。術後兩個月接受神經肌肉電學檢查,顯示左側鼠蹊韌帶 下方的股神經損傷,推斷與血管攝影術後的併發症相關。

藉此病例報告,我們將探討醫源性股神經損傷的發生、經股動脈血管攝影術的相關併發症,與股神經損傷的評估與治療,並討論股神經損傷與對側膝上部位截肢兩症並存時,患者的復健療程與預後。(台 灣復健醫誌 2008;36(4):227-234)

關鍵字:股神經(femoral nerve),血管攝影術(angiography),復健(rehabilitation)