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Case Report

Complete tibial nerve lesion secondary to postoperative popliteal pseudoaneurysm following anterior cruciate ligament arthroscopic reconstruction: A series of two patients

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ABSTRACT

Background: Complications following arthroscopic anterior cruciate ligament reconstruction (ACLR) are rare, but injuries to the popliteal artery can occur. Popliteal pseudoaneurysms are a potential complication and can cause significant morbidity if not diagnosed and treated promptly.

Cases Description: We describe the cases of two patients who developed nerve injuries following arthroscopic ACLR, with subsequent diagnosis of a popliteal pseudoaneurysm. The peroneal nerve recovered spontaneously in both cases, while the tibial nerve was reconstructed using autologous nerve grafting. Satisfying, functional recoveries were observed 24 months postoperatively.

Conclusion: Prompt diagnosis and effective treatment of popliteal pseudoaneurysms are crucial to prevent further complications. However, timely diagnosis can be challenging due to inconsistent clinical presentations and a low index of suspicion. This case report highlights the need for increased awareness of this uncommon complication and provides insights into its pathophysiological mechanisms.

Keywords: Anterior cruciate ligament reconstruction, Autologous nerve grafting, Peripheral nerve injury, Popliteal pseudoaneurysm, Postoperative complication, Tibial nerve lesion

INTRODUCTION

Anterior cruciate ligament reconstruction (ACLR) is one of the most common orthopedic procedures, with approximately 100,000 patients undergoing reconstructive surgery in the US annually.[16] Although arthroscopic surgeries are minimally invasive with relatively low morbidity, they are performed near neurovascular structures, and complications are reported.[13] In ACLR procedures, the incidence of symptomatic deep vein thrombosis (DVT), arterial injuries, and pulmonary embolism is 2.1%, 0.3%, and 0.1%, respectively.[8] Perioperative, arterial pseudoaneurysms are uncommon, with an incidence of 0.3%.[10]

Pseudoaneurysms of the popliteal artery are usually present with repeated hemarthrosis and a pulsatile mass within days to weeks after ACLR.[8] Complications of pseudoaneurysms include hemorrhage, pain, thrombosis, and space-occupying effects such as neurological or venous compression.[17] According to Roganović *et al.*, a continuous nerve compression of more than 3–4 days after the neurological symptom onset was critical for causing permanent nerve damage.[21] Since popliteal pseudoaneurysms are an infrequent complication following ACLR, a high index of suspicion is necessary to guarantee a timely diagnosis.[8]

In this article, we present two unique cases featuring neurotmesis of the tibial nerve and axonotmeses of the peroneal nerve caused by a postoperative, popliteal pseudoaneurysm following ACLR.

CASE DESCRIPTION

Case 1

A healthy, 34-year-old male patient had sustained a valgus trauma to his left knee, which resulted in a complete ACL rupture. A delayed arthroscopic reconstruction was scheduled 1 year after the injury. Immediately after surgery, the patient complained about severe pain and paresthesia, which irradiated to the popliteal fossa, his left calf, and foot. Some days later, a complete loss of dorsiflexion and plantar flexion of the ankle and foot manifested. First, a DVT was excluded by Doppler ultrasonography. A compartment syndrome was then suspected, and an emergency fasciotomy of the left leg was performed. Subsequently, the patient was discharged on the 10th postoperative day in an improved general condition, yet significantly restricted ankle and foot movement (overall M1 – British Medical Research Council [BMRC] scale).

On the 18th postoperative day, his pain worsened again, and complete paralysis of the dorsiflexion and plantar flexion of the foot arose. Moreover, a tingling sensation and numbness on the dorsal left leg and the plantar surface of the left foot persisted. An iatrogenic pseudoaneurysm of the popliteal artery was confirmed by angiography. An endovascular repair of the popliteal pseudoaneurysm was successfully performed with seven covered stents. The patient was discharged from the hospital without pain, yet no visible movement of his ankle or foot.

Approximately 3 months later, the patient was referred to our clinic due to ongoing neurologic symptoms. Clinical assessment revealed M0 ankle plantar flexion, M1 ankle eversion, dorsiflexion, and inversion (BMRC). In addition, the absence of foot sensation (S0) and a positive Tinel's sign in the popliteal fossa were noted. Electrodiagnostic tests confirmed absent sensory potentials (superficial peroneal and sural nerve) and loss of motor function (peroneal and tibial nerve). These findings strongly indicated severe axonal injury, specifically of the neurotmesis variant, affecting the left tibial and common peroneal nerves at the juncture of the popliteal fossa. Consequently, a surgical nerve revision was scheduled.

Intraoperatively, we confirmed the continuity of the peroneal and tibial nerves. However, a segment of the tibial nerve in the popliteal fossa was necrotic [Figure 1], which was adequately resected. The subsequent pathological analysis revealed extensive coagulation necrosis affecting nerve components, fibrovascular tissue neoformation, granulation tissue areas with mononuclear cells, and scattered giant histiocyte granulomas. The created 11 cm long defect of the tibial nerve was grafted by four strands of the ipsi- and contralateral harvested sural nerves [Figure 1]. 8.0 monofilament sutures secured the grafts. The peroneal nerve was neurolyzed.

At the final follow-up, 24-month post nerve reconstruction, sensory improvements within the tibial nerve area were noted (S1). However, he also experienced dysesthesia while walking

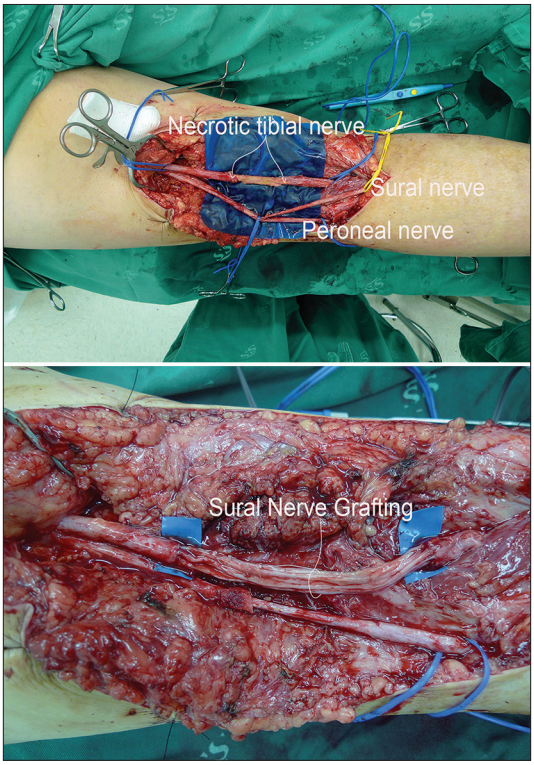

Figure 1: Intraoperative pictures of case 1. First, the division of the sciatic nerve into its three branches (tibial, peroneal, and ultimately sural nerve) was dissected, and the nerves neurolyzed within the scar tissue of the popliteal fossa. The necrotic segment of the tibial nerve (upper panel) was resected until an intact fascicular structure was observed. The lower panel shows nerve repair using four, 11 cm strands of sural nerve grafts.

barefoot. Motor function exhibited recovery in ankle plantar flexion to M4 (BMRC), through toe plantar flexion remained absent. Dorsiflexion of both the foot and toes had improved to M4 and M2 (BMRC), respectively. Furthermore, visual inspection revealed persistent atrophy of the calf muscles.

Case 2

A 19-year-old female and healthy patient had sustained a valgus mechanism trauma on her left knee during skiing. As she was suffering from constant knee instability, the patient received an ACLR 3 years after the injury. Two days postoperatively, the patient started experiencing continuous popliteal pain radiating to the left leg and foot. Moreover, a pulsating mass and blister were noted at the popliteal fossa, and marked tenderness and swelling of the leg and foot [Figure 2]. On the 4th day, the pain aggravated. Yet, an initial Doppler ultrasonography showed no abnormalities. The patient underwent an MR angiography [Figure 3] of the left leg on the 8th day. In conjunction with a second Doppler sonography, an iatrogenic pseudoaneurysm of the popliteal artery was diagnosed. An endovascular repair of popliteal pseudoaneurysm was performed, whereby the acute symptoms were handled. On the 13th postoperative day, a second surgery was performed to drain the suspected hematoma due to increased swelling in the popliteal fossa.

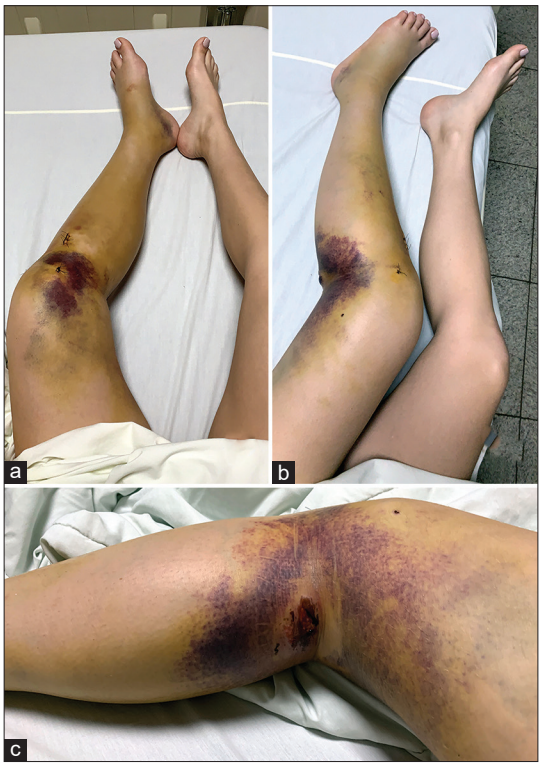

Figure 2: (a-c) Clinical presentation of case 2, 7 days after the anterior cruciate ligament reconstruction. The left leg showed significant swelling, a pulsatile mass in the popliteal fossa, and a posterior hematoma. At this time, the patient already complained about pain and beginning paresthesia.

Because of persistent paralysis, electrodiagnostic studies were performed 3 and 9 months postoperatively. Twelve months after injury, the peroneal nerve recovered spontaneously (dorsiflexion of the foot and toes M3, S3), but paralysis of the tibial nerve persisted. Atrophy of the gastrocnemius muscles was evident, and the plantar flexion remained M0 (BMRC). Hence, an operative repair was scheduled. Intraoperatively, a complete tibial nerve rupture was verified and reconstructed with four strands of 6 cm sural nerve grafts harvested from the contralateral side [Figure 4]. Both gastrocnemius motor branches were identified proximally and distally to the lesion and repaired with contralateral sural nerve grafts of 5 and 4 cm in length [Figure 4]. Grafts were secured in place by 8-0 monofilament sutures and fibrin glue (Tissucol, Austria).

At the last follow-up 24 months after the nerve reconstruction, the patient reported significant improvements in sensory disturbances related to the tibial nerve area. She could perceive tactile sensations, with heightened unpleasant sensations when encountering certain stimuli like uneven surfaces (S2). Her motor recovery demonstrated improvements in plantar and dorsi flexion to M4 (BMRC), while toe flexion was still absent. Notably, inspection revealed the persistence of calf muscle atrophy.

DISCUSSION

We present two secondary tibial nerve injuries following ACLR as a long-term complication. The delayed diagnosis of popliteal

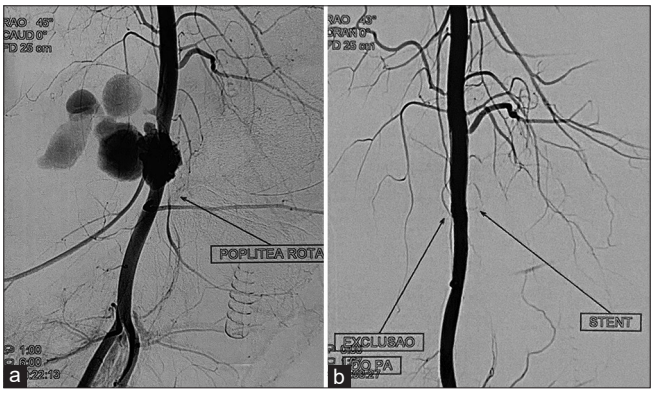

Figure 3: (a) Magnetic resonance (MR) angiography confirmed a popliteal pseudoaneurysm. A laceration of the anterolateral wall resulted in the formation of a large unencapsulated and partially thrombosed pseudoaneurysm measuring 45 by 52 mm. The postoperative pseudoaneurysm caused secondary ischemic damage to the peroneal and tibial nerves. (b) MR angiography postintervention: following endovascular stent placement, showing normal flow across the popliteal artery.

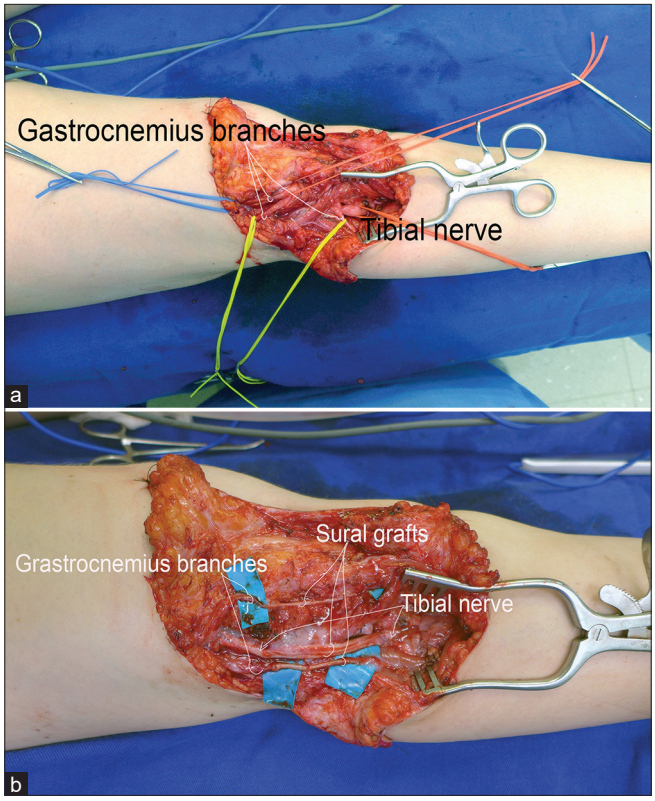

Figure 4: Intraoperative pictures of case 2. (a) Through a dorsal zigzag incision in the popliteal fossa, the tibial nerve and its branches to the gastrocnemius muscle were individually dissected. (b) The severed tibial nerve and both muscular branches were reconstructed by a sural nerve graft harvested from the contralateral leg.

pseudoaneurysms resulted in prolonged compression, which caused permanent nerve damage in the discussed cases. Hence, we especially emphasize the importance of considering this potential complication after ACLR.

Although ACLR is considered a safe procedure with low complication rates,[7] several studies[3,11,14,18,20,24] have already described iatrogenic common peroneal and tibial nerve injuries. Injuries involving direct trauma to the nerves caused by intraoperative, direct posterior capsular violations,[18,20,24] posterior trocar placement,[14] or closed traction during intra-operative knee manipulation.[3,11] However, no study was identified to describe secondary tibial nerve injuries following a postoperative pseudoaneurysm. During knee arthroscopic surgeries, vessel violations are usually related to the popliteal artery.[4,5,22] Due to a growing demand for arthroscopic ACLR, the incidence of iatrogenic popliteal pseudoaneurysm has increased in recent years.[10] Hence, several recommendations, which are reviewed elsewhere,[1,7] have been proposed to prevent vessel violations.

Clinical presentation of popliteal pseudoaneurysms usually involves pain, paresthesia, pulsating calf edema, and/or a palpable mass in the popliteal fossa.[10] The dorsalis pedis and posterior tibial arterial pulses are affected inconsistently.[10] A Doppler ultrasonography may be performed as an initial screening tool. Still, the gold standard investigation technique is a CT angiogram, which provides a sensitivity of 95.1% and a specificity of 98.7%.[23] Still, the clinical diagnosis of popliteal pseudoaneurysm is challenging due to a low index of suspicion and a difficult clinical examination of the popliteal fossa.[9] As reported in our series of cases, misdiagnoses such as DVT or compartment syndromes may further delay appropriate treatment.[2]

Although nerves are relatively resistant to ischemia, sustained stretch and compressive forces can result in secondary ischemic damage.[6] The anatomical organization and the intraneural and microvascular blood supply of the nerve explain the susceptibility of peripheral nerves to compressive forces. The tensile strength of a nerve is determined primarily by the robust perineural layer composed of elastin and collagen. In principle, nerves can stretch approximately 10%–20% before structural damage occurs.[15] Pseudoaneurysms may cause direct mechanical nerve distortion and microvascular compression.[21] We identified three cases in which nerve damage was related to compression by a popliteal hematoma or popliteal pseudoaneurysm. Peicha *et al*.[18] and Krivić *et al*.[14] reported a complete palsy of the common peroneal nerve after a popliteal hematoma. Janssen *et al*.[10] reported a sensory loss in areas of the saphenous, medial plantar, calcaneal, and superficial peroneal nerves following a popliteal pseudoaneurysm. Similarly, in our cases, nerve injuries were provoked by prolonged, popliteal artery compression to the tibial nerve.[21] In our first case, a necrotic segment of the tibial nerve was evident during surgical exploration [Figure 1].

Injury to the peroneal nerve results in characteristic foot drop and sensory deficits of the dorsal foot. However, as in the presented cases, spontaneous recovery is observed in 76%–87% of peroneal nerve injuries within 4–6 months.[19] The tibial nerve contributes to the motor function of the posterior compartment and the intrinsic muscles of the foot and sensorily supplies the plantar surface of the foot and the caudal surface of the leg.[12] Hence, reconstruction of the tibial nerve is essential to restore proper gait and at least some protective sensation. Both patients achieved satisfactory plantar flexion of the ankle (M4). However, plantar sensation and toes flexion remained restricted. Similarly, Kim *et al*.[12] observed good plantar flexion in most tibial nerve injuries. Ankle inversion, mostly driven by the posterior tibial muscle, and toe flexion are more difficult to restore.[12] Intrinsic foot muscle function was rarely regained.[12] However, functional recovery of plantar flexion of the ankle, which is essential for "push-off" in walking, combined with at least minimal protective sensation to the sole of the foot in most patients, makes repair of the tibial division very worthwhile.[12]

Considering that both patients achieved satisfying ankle plantar flexion, whether the age difference (19 vs. 34 years) or the delay in reconstruction (3 vs. 12 months) significantly influenced regeneration remains unsolved.

CONCLUSION

Although complications following ACLR are rare, a profound understanding of the pathophysiologic processes is essential for adequate management of potential sequelae. Due to ambiguous clinical presentation, prolonged compression by the popliteal pseudoaneurysms resulted in secondary peroneal and tibial nerve damage. The correct and timely diagnosis was a major challenge in both cases. Especially the combination of vascular and neurologic symptoms might have been misleading.

Declaration of patient consent

Patients' consent not required as patients' identities were not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The author confirms that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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