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Lower extremity neurological complication following routine surgical intervention

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Pneumatic tourniquets have been utilized for centuries to assist in hemostasis, resulting in faster operating times and better identification of anatomical structures. Mortality and morbidity are rare but can be associated with improper tourniquet use. This case study reports on a lower extremity neuropathy that developed after seemingly proper pneumatic tourniquet use during ankle surgery. Nerve conduction velocity (NCV) testing suggested likely etiology was from a resolved compartment syndrome.

Keywords: compartment syndrome, tourniquet, neurapraxia

The U.S. Food and Drug Administration recognizes the pneumatic tourniquet as a Class-I medical device indicating minimal harm to the patient with routine use [1]. When properly utilized, tourniquet application creates a bloodless surgical field enhancing the surgeon's ability to identify anatomic structures and reduce intraoperative blood loss [1]. Its roots can be traced back to the Roman Empire (199 BCE – 500 CE), when bronze and leather devices were donned to injured extremities to reduce bleeding during war-time amputations [1]. In 1864, Joseph Lister was the first surgeon to apply a tourniquet in the operating room [1]. Harvey Cushing introduced the pneumatic tourniquet in 1904 allowing tourniquet pressure to be manually controlled which aided in decreasing associated injuries. It has been estimated that over 15,000 surgical procedures occur daily which require the use of a pneumatic tourniquet [1]. Routine tourniquet use is not without risk of morbidity or even mortality with potential complications including compression neurapraxia, compartment syndrome, wound infection, wound hematoma, delayed recovery of muscle power, arterial hypertension, cardiorespiratory decompensation, rhabdomyolysis, and cardiorespiratory decompensation [2]. The rate of nerve injury associated with tourniquet use ranges from 0.1% - 7.7% [3]. To assist in decreasing comorbidities and mortality related to tourniquet use, numerous studies have been conducted to determine appropriate tourniquet applications, tourniquet duration, tourniquet design, and patient selection when utilizing tourniquets [1]. This Case Study reports a patient developing a common fibular neuralgia after routine use of high calf tourniquet during routine ankle surgery.

Case Report

A 48 year-old healthy female presents to the clinic complaining of chronic right ankle pain and weakness after an ankle sprain one year prior. She underwent a two-year period of conservative treatment with no resolution of her symptoms. MRI obtained showed tendinosis and possible tearing of her peroneal tendons.

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Surgical intervention was deemed necessary based on progression and chronicity of her symptoms with failure and exhaustion of conservative treatment.

The patient received a preoperative popliteal fossa and saphenous nerve block and was placed in the supine position with a pneumatic calf tourniquet set to 250 mmHg. A semi-linear incision was made along the course of the peroneal tendons. Dissection carried down to the peroneal tendons with further evaluation, identifying an intrasubstance tear of the peroneus brevis tendon. The tendon was debrided and re-tubularized in the usual manner. The foot was then everted to re-approximate the superior retinaculum. The surgical incision was closed and dressed in the usual manner. The pneumatic tourniquet was deflated at 68 minutes. Proper hyperemic response was noted. A modified Jones compression posterior splint was applied with the foot slightly everted and ankle at 90 degrees. She was discharged from PACU with all vitals stable and vascular status intact to the right lower extremity. Postoperative instructions were given to remain non-weight bearing to her right lower extremity.

The first postoperative visit occurred ten days following her procedure without any notable complications. Pain level was tolerable and no complications involving falls were noted. She was transferred into a removable CAM boot at this time, continuing non-weight bearing status to the right lower extremity. At 3 weeks post-procedure, she complained of acute onset of pain, numbness, and paresthesia in her right lower extremity, from knee to foot, which worsened at night but was constant. Based upon clinical symptoms, a diagnosis of neuralgia was made. Treatment began with oral Gabapentin 300 mg taken nightly. Physical therapy was initiated 1 week later. At 6 weeks, the patient continued to have numbness and paresthesia to the entire right foot and up to her knee. Patient denied any help from the Gabapentin medication. Physical exam revealed decreased muscle strength and a hyper-sensitivity to light touch to her right foot and lower leg. Physical therapy helped with recovery of her tendon repair but no change to her neurological symptoms was noted. Patient was sent to pain management and complex regional pain syndrome was ruled out. She tried and failed Elavil oral medication. She was intolerant to Cymbalta oral medication. Nerve conduction velocity and EMG studies were obtained approximately 14 weeks following surgery to assess for potential areas of nerve entrapment or injury. Results revealed a mild polyneuropathy affecting sensory and motor nerves without evidence of a localized neuropathy such as a tarsal tunnel syndrome or focal sensory neuropathy. The study suggested small nerve fibers may have been affected and a resolved compartment syndrome was deemed as a likely etiology of the polyneuropathy. At 5 months, the patient showed recovery from her peroneal tendon surgery but still with continued paresthesia and numbness to the right foot and ankle, up to the knee. The pain at this point is tolerable with shoe and activity modifications.

Discussion

Complications arising after tourniquet use during lower extremity surgical procedures are rare but still occur. A questionnaire survey in Norway estimated neurological complications associated with lower extremity tourniquet use occurred in one per 3752 applications [4]. Our case study reports neurological complication occurring secondary to a possible compartment syndrome that occurred 3 weeks after the surgical procedure. Compartment syndrome is a potentially serious complication which can occur once interstitial pressure in a closed fascial compartment increases to a level which impedes vascular flow resulting in myoneuronal function impairment and soft tissue necrosis [5]. Normal compartment pressures allowing capillary perfusion are described ranging from 0 to 8 mmHg [6]. Once interstitial pressure increases above this range, blood flow is impaired leading to the associated complications [6]. Previous case studies have reported compartment syndrome occurring after tourniquet use [5,7,8]. but in our case study the clinical presentation of compartment syndrome was not present directly following surgery. Classic presentation of compartment syndrome has been described as pain out of proportion, pain on passive stretching of the affected compartment with associated clinical symptoms of pallor, pulselessness, and paresthesia of the affected extremity [5]. Compartment syndrome resulting after lower extremity tourniquet application has been reported to occur after prolonged ischemia time with reperfusion edema, direct muscle trauma secondary to repeated inflations of the tourniquet and improper positioning [9]. As described per the surgical report, the patient was appropriately

positioned on the operating table, with proper application, location, and duration of a pneumatic tourniquet and without repeated inflations. This patient did obtain regional anesthesia via a popliteal fossa and saphenous nerve block which some suggest can delay the diagnosis of compartment syndrome [10].

Pain is a cardinal feature of compartment syndrome which theoretically can be altered by analgesia. Our patient did not begin to experience pain until approximately 3 weeks following her surgical procedure. Mar, et al., reported 32 of 35 patients who received epidural analgesia had "classic signs" of compartment syndrome which included pain out of proportion. Their conclusion stated there was no convincing evidence regional analgesia delays the diagnosis of compartment syndrome [10].

Peripheral nerves are composed and organized into connective tissue structures forming a framework to provide protection and function to nerve fibers. These connective tissue structures include the endoneurium, perineurium, and epineurium. Individual nerve fibers are surrounded by the endoneurium. Fascicles, groups of endoneurium, are enveloped by the perineurium. Epineurium encases bundles of fascicles [11]. Vessels in the epineurium are more vulnerable to compression trauma resulting in permeability changes compared to endoneurium vessels. Vessel permeability changes occurring secondary to trauma lead to associated edema formation and accumulation. Endoneurium edema is prevented from draining into adjacent areas due to a blood-nerve-barrier and a lack of lymphatic channels. Perineurium edema is prevented from draining into adjacent areas due to a selective diffusion barrier. Past studies have suggested edema accumulation inside nerve fascicles create a "miniature compartment syndrome" which could alter nerve function [12]. A miniature compartment syndrome may affect or impair nerve function through a sustained increase in fascicle pressure, altering endoneurium fluid electrolyte composition or reducing blood flow to nerve segments.

Ochoa, et al., demonstrated nerves directly beneath and near the tourniquet cuff edge were subjected to injury due to external compression. This direct pressure has been shown to cause displacement of Nodes of Ranvier and myelin sheath invagination which disrupts nerve conduction [13]. The resulting damage associated with displacement of Nodes of Ranvier and myelin sheath invagination is associated with partial or complete local conduction block which is usually reversible within weeks or months. Nodes of Ranvier are essential components of nerve function and are located along peripheral nerve axons to increase conduction velocities [14]. Compression from tourniquet application has been shown to displace Nodes of Ranvier up to 300 nanometers from their original site [13]. Tourniquet induced compression can affect larger nerve fibers responsible for motor function or smaller nerve fibers responsible for pain, temperature and autonomic function.

Our case study reports a polyneuropathy affecting motor and sensory nerves with a likely etiology of a resolved compartment syndrome. Clinical presentation of classic compartment syndrome was not present during this patient's immediate postoperative period. Nerve injury resulting in the polyneuropathy most likely was secondary to nerve injury sustained from external compression via a pneumatic tourniquet. As discussed, nerve function can be altered from an increase in fascicle pressure secondary to edema accumulation or displacement of essential nerve components required for normal nerve function.

If the patient experiences nerve related injuries after surgery, proper evaluation and a thorough work-up is warranted to determine the severity of injury. To determine the severity of the lesion, a nerve conduction study can be utilized to confirm the lesion grade. Seddon, et al., classified nerve injuries into three grades, neuropraxia, axonotmesis, and neurotmesis based on the severity of lesion [16]. Sunderland later expanded this classification into five different nerve injury patterns. Neurapraxia, Grade 1, is the mildest injury and produces a local nerve conduction block at the site of injury with normal nerve conduction proximal and distal to injury. There is no associated injury to the surrounding nerve tissues. Axonotmesis, Grade 2, is seen when demyelination occurs at the injured site leading to Wallerian degeneration distal to the demyelinated segment [17]. Nerve regeneration is possible due to the preserved endoneurium and perineurium. If full functional recovery of the nerve occurs within 3 months after the injury it is classified as a neurapraxia but if recovery occurs at a rate of one inch per month

the injury is classified as axonotmesis. Fibrillations and denervation potentials can be seen distal to the site 3 weeks following the injury. Recovery is spontaneous and complete with axonotmesis injuries but can take weeks to years [18]. Damage to the endoneurium without damage to the epineurium is seen in grade 3 injuries. Damage to the myelin, endoneurium, perineurium, and axon indicates a Grade 4 injury. Grade 5 injuries are seen with complete transection of the nerve [17].

Nerve-related injuries during surgery can create a complex postoperative course. If questionable nerve symptoms do occur, proper work up is warranted to determine diagnosis and severity of the damage. Treatments range from oral and topical medications to surgical neurolysis. This case study shows our patient developing polyneuropathy 3 weeks after seemingly proper surgical use of calf tourniquet, likely from a resolving compartment syndrome after surgical use of tourniquet. If a patient displays the appropriate symptoms, a high suspicion for compartment syndrome is warranted.

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