Tibial neuropathy caused by popliteal pseudoaneurysm following total knee arthroplasty.

Moreta J, Aurrekoetxea JT, Ormaza A, Uriarte I, Martinez-de Los Mozos JL.

Department of Orthopaedic Surgery and Traumalogy, Hospital Galdakao-Ueansolo, Bizkaia, Spain.

ABSTRACT

Vascular injuries after total knee arthroplasty are uncommon but they can result in serious complications and poor clinical outcome. Early diagnosis and accurate management are important to achieve the best possible result, so postoperative vascular surveillance is advisable, specially with associated risk factors. Although the presence of neuropathy secondary to compression by an expanding pseudoaneurysm after total knee arthroplasty has been reported before, this is a unique case in which an isolated tibial neuropathy has been identified.

KEYWORDS

Total knee arthroplasty; tibial neuropathy; popliteal pseudoaneurysm; endovascular stenting.

INTRODUCTION

Neurovascular complications following total knee arthroplasty (TKA) are fortunately uncommon, but they can result in serious complications like acute ischemia, compartment syndrome and potential amputation. The most common etiology of these injuries seems to be an indirect mechanism, like aggressive joint manipulation or excessive retraction and heat injury from the exothermic reaction of the cement [1-4]. However, transection of the popliteal artery and popliteal pseudoaneurysm as a result of direct arterial trauma has also been described [5]. The risk of vascular problems is reported to be higher in patients with preexistent vascular insufficiency as exhibited by leg claudication, atherosclerosis, prior coronary bypass surgery, and decreased distal pulses [1,3].

We report a case of tibial neuropathy due to popliteal pseudoaneurysm following TKA.
CASE REPORT

A 75-year-old man with no significant medical history was referred to the author’s hospital for progressive right knee pain with diagnosis of osteoarthritis. Preoperatively, the knee was in varus mechanical alignment of 6°. A posterior-stabilized total knee arthroplasty was performed routinely using measured resection technique through a medial parapatellar approach with a thigh tourniquet inflated 100 mmHg above the systolic blood pressure (value of the tourniquet 250 mmHg), which was released before suture for careful hemostasis. Tranexamic acid or topic procoagulant agents were not used during the procedure. For soft tissue balancing, deep medial collateral ligament release was followed by posterior oblique ligament release, achieving proper ligament balancing. The total ischemia time was 75 minutes and the intraoperative blood loss was 150 ml approximately. A suction drain was applied and removed 24 hours after operation and conventional low molecular weight heparin was used postoperatively to prevent deep vein thrombosis. The postoperative blood loss was calculated by measuring the amount of blood in the drainage bottle and it was 350 ml. The drop in hemoglobin level was 2.1 g/dL (11.7 to 9.6 g/dL). His postoperative period was uneventful and he was discharged home on the 5th day of hospital stay. At ten days after surgery he presented to the emergency department because of severe pain and numbness in the sole of his right foot. On physical examination the right calf was swollen with a positive Homans’ sign and all of the extremity pulses were palpable. The neurological examination showed decreased sensation of the right plantar surface and the muscle strength testing revealed 2/5 strength for the gastrocnemius muscle and 1/5 for the flexor hallucis longus and the flexor digitorum longus muscles. With these findings the initial differential diagnosis included tibial nerve neuropathy or deep vein thrombosis (DVT). When a thorough vascular examination was performed a pulsatile mass was palpated in the popliteal fossa with no pain associated.

The duplex ultrasound showed a 37 mm diameter popliteal pseudoaneurysm with flow from the popliteal artery which had a proximal and distal diameter of 9 mm. There were no signs of arteriovenous fistula. A limb magnetic resonance angiography (MRA) confirmed a pseudoaneurysm arising from the third portion of the popliteal artery at the level of the tibial component, with posterior displacement of the artery but no stenosis (fig 1). The following day an endovascular stenting procedure was performed with a simultaneous fasciotomy in

Figure 1. Magnetic resonance angiography. Pseudoaneurysm dependent on the third portion of the popliteal artery causing tibial nerve entrapment.
order to prevent compartment syndrome. An intraoperative angiography was used to confirm the pseudoaneurysm and after endovascular stenting (fig 2A and 2B). The patient had no further complications and was discharged 1 week after vascular surgery. Nerve conduction and electromyography studies were performed 8 weeks after the TKA procedure. These tests showed fibrillation potentials in the right soleus and gastrocnemius, indicative of denervation injury.

At 24 months of follow-up, the patient had a range of movement from 0° to 120° and he had no vascular symptoms in her limb with normal distal pulses. Plain radiograph showed correct position of the knee arthroplasty without complications (fig. 3). The patient currently suffers from residual numbness and paresthesias in the plantar aspect of his right foot with slight motor deficit (4/5 strength for the gastrocnemius, flexor hallucis longus and the flexor digitorum longus muscles).

DISCUSSION

Total knee arthroplasty is fortunately a safe procedure with good results but vascular complications can occur with serious consequences like wound healing, deep infection and even amputation. The incidence of vascular injury after TKA is uncommon and ranges from 0.03% to 0.5%, but the frequency of vascular injury may be increasing, as more elderly patients now undergoing the procedure and a higher number of revision surgeries are being performed each year [2, 3, 6-8].

The most common mechanism of arterial injury after TKA is probably arterial occlusion with thrombosis but there are other forms like arterial severance, arteriovenous fistula formation and pseudoaneurysm formation. Pseudoaneurysms develop from a disruption in the arterial wall and the most frequent mechanism is indirect trauma such as excessive retraction, aggressive manipulation and compression or thermal injury from cement [1, 3, 9, 10]. A direct trauma could produce an arterial injury during resection of the femoral condyles or the tibial plateau and when a posterior capsule release is necessary for flexion contracture [7]. Sandoval et al reported on a case of popliteal pseudoaneurysm due to a direct perforation with a surgical pin [4].

On physical examination a pseudoaneurysm can be recognised by a pulsating mass in the popliteal fossa and sometimes with an audible arterial bruit. However, this findings may be less obvious and this injury should be suspected in patients with chronic pain, swelling or local hematoma. The late presentation of pseudoaneurysm is relatively common, even months after the knee surgery and, like our case, can be confused with DVT [5, 7, 11, 12]. In our patient, the main symptoms were pain with neurological deficit in his right foot and, for this reason, the initial diagnosis was a tibial neuropathy. Compression of the tibial nerve in the popliteal fossa produces pain in the calf muscles and a variable degree of weakness in plantar flexion and adduction of the foot and toes. Sensory deficits may be seen in distribution of sural, lateral plantar and calcaneal nerves. The Duplex ultrasound showed a popliteal pseudoaneurysm and based on a magnetic resonance angiography the compressive neuropathy of the tibial nerve was confirmed. The presence of neuropathy secondary to compression by an expanding pseudoaneurysm has been reported before, but this is an unique case representing an isolated tibial neuropathy [4, 7, 12].

Several studies have suggested that patients with previous presence of vascular disease are at higher risk for vascular injuries [1, 3, 8]. Calligaro et al identified atherosclerotic disease in all patients with arterial injuries after TKA [2]. For this reason, a preoperative vascular assessment should be performed with inspection of the skin to identify changes associated with chronic ischemia. The popliteal fossa and pedal pulses should be palpated and it is also important to look for arterial calcification on radiographs. If there is any concern about the vascularization, the ankle/brachial index (ABI) should be performed. With an ABI less than 0.9 is advisable to refer the patient to a vascular surgeon before TKA. Some authors suggest that most arterial complications following TKA are associated with tourniquet use and are related to indirect vessel injury and thrombosis, especially in the previously diseased artery [6, 11, 13]. The main advantage of
tourniquet use is the bloodless field, which could facilitate the exposure and should improve the cementing technique. However, there is no evidence of this advantage and it is advisable that patients who are at risk for arterial complications be considered for TKA without the use of a tourniquet [6, 8]. In our case, we could not identify risk factors, previous vascular disease or intraoperative adverse events.

Several methods of treatment have been described to treat arterial pseudoaneurysms, depending on the size and location. If the pseudoaneurysm is small (< 5 centimeters) could be treated with doppler guided compression or percutaneous thrombin injection [15-17]. Other methods are embolization, bypass graft, endovascular stenting or excision and sewing. In the recent years, endovascular stenting has become a popular therapy and this technique has been associated with less postoperative morbidity compared with open surgery [19,20].

Although neurovascular injuries are uncommon after TKA, one should be aware of vascular complications during the procedure. There are very few reports in the literature regarding compressive neuropathy due to a popliteal pseudoaneurysm after a TKA, and to the best of our knowledge, an isolated tibial nerve palsy has not been described before. Nonetheless, popliteal artery injury should be considered in the differential diagnosis. Early diagnosis and management of the vascular injury is crucial to avoid serious consequences and we suggest careful examination of the popliteal fossa with duplex ultrasound if this complication is suspected.

REFERENCES