Case report

Superficial femoral artery pseudoaneurysm, compartment syndrome, and deep vein thrombosis after total knee arthroplasty

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Abstract

Vascular injuries after total knee arthroplasty are highly infrequent, especially in the femoral artery. These lesions can cause severe damage. Early diagnosis is important to prevent catastrophic complications (such as loss of limb) and to offer adequate treatment. This study reports a patient with femoral artery injury of unknown etiology after total knee arthroplasty. Progressive and insidious symptoms from deep vein thrombosis to compartment syndrome made management even more challenging, requiring amputation of the extremity.

Introduction

Total knee arthroplasty (TKA) has been one of the most successful procedures in orthopedic history during the last 50 years [1], but there have been reports on complications and vascular lesions in literature leading to devastating conditions [2]. Available literature reports a low incidence of vascular complications, although the incidence is unknown given that many of the reported studies are isolated case reports.

Incidence ranges between 0.03% and 0.17%, as reported by Calligaro et al. in 2003 [3] in his series. More recently, He et al. in 2016 [4] and Zhao et al. in 2017 [5] proposed that vascular lesions can range from an active hemorrhage to the development of pseudoaneurysms or false popliteal aneurysms even up to 5 months after surgery [2]. They also explain the generation of diverse symptoms that may or may not lead to an early and adequate diagnosis that could allow for appropriate treatment. Similarly, vascular lesions can also present with compartment syndrome, which, by itself, is highly rare after TKA and even rarer in association with vascular trauma, leading to increased morbidity [6-9]. This study presents a case with no clear etiology for vascular injury after TKA and how it was managed.

Case history

A 73-year-old woman with a past medical history of hypertension and TKA 15 days before admission presented to the emergency department complaining of pain in the right leg associated with edema and progressive movement impairment since the surgery. At admission, she brought a venous Doppler report with a diagnosis of deep vein thrombosis compromising gastrocnemius, popliteal, distal third femoral and posterior tibial veins. Physical examination revealed her surgical wound was without signs of infection; however, she had significant leg edema, decreased distal pulses, skin color changes, and prolonged distal latency longer than 4 seconds with weak pulses in her right leg, suggesting critical ischemia and associated compartment syndrome. Arterial ultrasound revealed a pseudoaneurysm at the level of the superficial femoral and the popliteal arteries.

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At 25 hours after admission, vascular surgery considered clinical features of irreversible critical limb ischemia classified as Rutherford grade III. Computed tomography angiography confirmed occlusion of a pseudoaneurysm located approximately 10 cm proximal to the knee with reconstitution of blood flow in the posterior tibial, peroneal, and anterior tibial arteries toward the ankle (Fig. 1). Surgical exploration was considered to evaluate whether she was a candidate for revascularization.

During surgery, using a medial approach to the thigh, in the muscular plane of the adductors, a pseudoaneurysm of $6 \times 6$ cm was found. It was resected and repaired with a femoral-popliteal bridge of approximately 10 cm (Fig. 2). Deep popliteal vein thrombosis was also evident despite the use of thromboprophylaxis.

After confirmation of distal pedal pulse, fasciotomies of the 4 leg compartments were performed through anterolateral and posteromedial approaches. However, after fasciotomies, muscles were still pale and unresponsive to electrical or mechanical stimuli. Vacuum-assisted closure was used for fasciotomy management and future closure.

During follow-up, the patient had a significant improvement in total creatine kinase values after the surgical procedure as well as in her general clinical condition. She required 3 more surgical procedures for debridement of necrotic tissue, evaluation of distal tissue vitality, and vacuum-assisted closure change with no further complications. Unfortunately, at 16 days after hospital admission, tissue vitality was not restored, and the presence of blisters, venous congestion, cutaneous necrosis (Figs. 3 and 4), as well as an increase in creatine kinase and C-reactive protein confirmed the need of transfemoral amputation.

Before the procedure, clinical and surgical options were discussed with the patient and with a panel of specialists, including psychiatry and psychology.

One week after the procedure, the patient was in adequate clinical condition. She continued evaluations with internal medicine, psychiatry, psychology, and nutrition to ensure rehabilitation.

Figure 1. CT scan of the right inferior limb. Flow is interrupted by the presence of a pseudoaneurysm in the superficial femoral artery with limited distal recanalization (arrows).

Figure 2. Femoral-popliteal bridge done by a vascular surgeon.

Discussion

The development of the case presented in this study deviated from the normal evolution of a knee arthroplasty. Clearly, there were many inquiries during the treatment period of the patient, requiring a detailed literature research as follows.

Mechanisms

Primary sources of information consist of case reports, showing that vascular trauma has specific etiologies that can be directly related to surgical procedures, and even to anesthetic procedures (postsurgical pain management). Surgical procedures in the series reported are more frequently described in the popliteal artery [3-5,10]. There are also reports on the use of tourniquet producing proximal artery injury, especially in the femoral artery [4,11], as well as on adductor canal blockage with posterior hematomas and pseudoaneurysms during endovascular procedures [12-14].
Literature has also reported cases in which an etiology has not been found [4,6,15]. In such cases, the theories proposed to explain the development of a vascular lesion include the following: 1. The use of tourniquet in patients with atheromatous disease, causing plaque separation and emboli. 2. The use of tourniquet might produce lesions in the intimal layer of blood vessels associated with hyperviscosity, and slow blood flow favors the formation of thrombi and secondary ischemia. 3. After the correction of flexion deformities, the intimal layer of blood vessels gets injured activating an inflammation cascade reaction and the formation of thrombi. 4. Lesions from punctures during a surgical procedure [2]. All the mechanisms described previously correlate with the mechanism proposed by Butt et al. in 2010, which includes the use of tourniquets and puncture lesions associated with surgical procedures. In our case, there is no clear etiology for the pseudoaneurysm.

Diagnosis

Most complications in TKA are diagnosed in the first 24 to 48 hours, allowing for early treatment and avoiding the need of amputation, contrary to what happened in our case [2-5,7,8,10,11,15,16]. Nonetheless, despite optimal management of complications following early diagnosis, there can be loss of functionality, such as foot drop and articular rigidity, or requirement of external aids for mobility, and the quality of life of patients can get compromised [6,7,9,17].

Although clinical suspicion is based on an adequate physical examination and is the first step for treatment, the use of diagnostic aids is fundamental to identify and recognize the degree of vascular compromise [5,8-11,18].

The use of arterial Doppler is quite frequent, much like the use of CT contrast scans. In compartment syndromes, measuring compartment pressures can also be helpful. In our case, the use of these tools associated with physical findings allowed for quick decision making regarding the need for surgical revision.

The primary location of vascular trauma complications related to TKA is usually in the popliteal artery [2-4,7,10,16], probably associated with surgical manipulation. Reported incidence on popliteal vascular injury in some studies ranges between 0.003% and 0.19% [19-22]. Nevertheless, the incidence of vascular injuries different from popliteal artery is not clear despite available literature on vascular injury at different levels (either proximal and distal femoral and anterior tibial arteries) [4,5,11-15,20,23]. Apart from this, anesthetic or analgesic procedures have also been reported as causative for vascular injury [14]. In many of the reports, however, no clear etiology has been identified for the vascular trauma or the origin of compartment syndrome despite diagnostic aids and surgical exploration [5,6,9,15,24]; in addition, poor prognosis factors related to delayed diagnosis or treatment (longer 6 to 8 hours) result in a functional compromise or even amputation of the limb [2,4,7,8,16].

Treatment

Diagnostic methods, treatment protocols, and approaches to complications derived from vascular trauma secondary to surgery have changed during the past years. A confirmed diagnosis of vascular trauma allows surgeons to use an endovascular procedure or noninvasive procedures to minimize the risk of complications. In the case of pseudoaneurysm, ultrasound-guided compression repair is the gold standard of treatment, especially when the lesion is in the proximal femoral artery [12]. Nonetheless, treatment options with endovascular devices that can treat occlusions or even thrombi might not provide satisfactory results [4,11-14]. Some of these lesions can still be treated by open surgery with exploration and repair, mainly when the lesion produces ischemic changes or compartment syndrome, and fasciotomies are required [7-9,15,16,18,24].

Despite the need for amputation in our patient, studies report a low incidence of amputations as definitive management for these patients, and it must come as an option after multiple surgical procedures that include debridement of necrotic tissue, failed fasciotomy closure, failed revascularization, among others [4,7,11].

Summary

The incidence of vascular complications that can lead to the development of compartment syndromes in the context of TKA is quite low. Delayed clinical suspicion could produce limb loss or even mortality for patients. Though the use of diagnostic aids is fundamental to identify and repair vascular lesions, clinical examination and medical proficiency remain the key for identifying signs and
symptoms of limb ischemia or compartment syndrome. Early diagnosis could allow for less morbid treatment and better results, especially if done within 6–8 hours after the onset of initial symptoms. Aggressive treatment to maintain adequate tissue perfusion may require multiple procedures that can increase morbidity and risk of infection. In general, amputation is not commonly used as definitive management and is undertaken only in cases in which ischemia threatens the life of the patient. Finally, this case report considers that, for our patient, the main negative prognostic factor was delayed diagnosis and no clear identification of the etiology of the vascular lesion.

Conflict of interest

The authors declare there are no conflicts of interest.

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