Case report

Traumatic anterior dislocation of a prosthetic knee, from trauma to delayed onset of vascular injury

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A B S T R A C T
Anterior dislocation of a prosthetic knee is a rare event. Only few episodes have been described in the past and have never been linked with neurovascular injury. This could lead orthopaedic surgeons to focus on the implant instability factors and underrate other complications. The authors report a case of a patient who experienced popliteal artery dissection 80 hours after traumatic anterior knee dislocation of a previously well-functioning total knee arthroplasty. Given that there is a lack of clinical cases that have established guidelines for management of this problem, the authors have focused on the importance of performing computed tomography angiography in the management of an anterior traumatic knee dislocation of a prosthetic knee.

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Introduction

The anterior dislocation of total knee arthroplasty (TKA) is a rare and insidious event that could conceal many complications. Despite literature having never reported vascular complication, the authors describe a case of a patient with a well-functioning TKA that experienced traumatic anterior dislocation, a delayed onset of popliteal artery dissection, and revascularization syndrome.

Case history

A 72-year-old female who had undergone a right cruciate-retaining TKA 3 years earlier reported to the emergency department after knee rotational trauma. She presented with complete right limb functional impotence, knee pain, and swelling without any apparent nerve involvement. Standard radiograph views showed anterior dislocation of the prosthetic knee (Fig. 1a). The orthopaedic surgeon on duty reduced the dislocation under conscious sedation within 2 hours after trauma. Axial traction combined with valgus and intrarotational forces were applied. A knee-ankle-foot orthosis was applied. The knee was maintained at 20° of flexion to relax the neurovascular bundles. Postreduction radiograph did not show any fractures or signs of prosthesis components loosening (Figs. 1b and 2). Before applying a cast, a Doppler ultrasound evaluation of popliteal vessels (comparing with the opposite leg) was performed (Linear probe, 7.5 to 15 kHz; device: MyLab 40; Esaote, Florence, Italy). The radiologist reported "no alteration of the flow distal to the third part of femoral artery or signs of thrombosis involving popliteal artery." The condition of the patient remained stable for 2 days. Moderate knee pain in popliteal area (NRS 2-3) was well controlled with nonsteroidal anti-inflammatory drugs. During the third day, 80 hours after trauma, the patient reported foot numbness and dysesthesia. Under assessment, the limb showed pallor and poikilothermia at the distal third of the leg and foot. Arteries showed a palpable low flow. Neurologic evaluation showed progressive development of palsy and hyposensitivity distally to the knee. At first, the neurologic assessments using Medical Research Council (MRC) Scale for Muscle Strength [1] showed extensor hallucis longus, extensor digitorum (longus and brevis) and fibularis muscles (longus and brevis) strength 0-5; tibialis anterior, 2-5; flexor hallucis...
longus, 2-5; flexor digitorum longus, 3-5; and gastrocnemius and soleus, 3-5. Assessment of sensitivity showed hyposensitivity and numbness on the whole right leg. Suspecting vascular involvement, computed tomography (CT) angiography was urgently performed demonstrating thrombotic occlusion of popliteal artery due to vessel dissection. Subsequent angiography confirmed the diagnosis (Fig. 3a). Fibrinolysis (5000 UI of heparin and 100,000 UI of urokinase) was obtained immediately after angiography, but it reached only partial reflow of the artery. Complete revascularization (Fig. 3b) was obtained 44 hours after the beginning of the revascularization procedure by means of 2 popliteal stents (Viabahn 5 × 50 mm and 6 × 50 mm) and prolonged fibrinolysis (5000 UI of heparin and 100,000 UI of urokinase).

No nerve functions were regained after revascularization procedure, and no sensitivity was detected below the knee after that. The functional score of tibialis anterior, flexor hallucis longus, flexor digitorum longus, gastrocnemius and soleus decreased to 0-5 in 2 hours from the onset of symptoms. Twelve hours after the revascularization procedure, the patient developed a compartment syndrome due to revascularization syndrome. Four-compartment fasciotomy was performed by medial and lateral approaches, and vacuum therapy was applied (no major bleeding was detected). After surgery, the swelling of soft tissues did not allow surgeons to close the skin. Anterior and lateral muscle compartments appeared poorly vascularized and sick and developed massive liquefactive necrosis. Multiple regular surgical debridements were performed until the skin healed by second intention in 3 months. One month after trauma, the patient was able to walk with crutches and articulated-knee-ankle-foot orthosis. After 5 months, the patient did not recover any nerve functions distal to the knee.

At present, the patient describes her life as unsatisfactory and she will probably need ankle-arthrodesis (tibio-astragalus and hindfoot arthrodesis) to correct the acquired varus and equinus deviation, but she is not physically and mentally ready to undergo any other surgery.

The patient expressed written consent to disclose her case in anonymous form.

Discussion

Traumatic anterior knee dislocation in patients with previously well-functioning TKA is an extremely rare and insidious event.

Dislocation of TKA was first described in 1979 when Insall et al. [2] reported 4 cases of posterior subluxation. Anterior dislocation problems of prosthetic knee were previously approached by Wang and Wang in 1997 when they reported 3 cases of anterior dislocation after cruciate-retaining implant [3]. Until 2016, just 5 cases of traumatic anterior dislocation of TKA had been described in English literature [4]. Different types of etiological factors were reported, and they were not always traumatic factors; the authors focused especially on instability factors. A greater laxity in flexion than in extension, malposition of components, extensor mechanism dysfunction [5], valgus deformity of the knee [6], and fracture of polyethylene [7] were all recognized as risk factors. Regardless of these factors, in case of trauma, the scenario could change and traumatic anterior dislocation of a TKA could happen also in a previously well-functioning implant.

As the event is so infrequent, there is no clear guideline for its management. Some authors, in case of traumatic anterior knee dislocation, suggested conservative treatment for the first 3 months. The injured leg should be placed in a brace for 6 weeks. In case of permanent instability, the prosthesis revision with a more constrained implant should be delayed by at least 3 months to reduce the risk of neurovascular bundle damage (already involved in the previous trauma) [5].

In traumatic dislocation of nonprosthetic knee, the reported incidence of vascular damage ranges from 30% to 60% [8], while, as reported by Pao et al. in 2003, vascular complications have never been described after TKA-dislocations. In this work, the authors focused on the low-energy nature of the trauma required in case of dislocation of a prosthetic knee [9], in contrast to dislocation of a native knee that is, commonly, a consequence of an high-energy trauma.

In our case, the first complication the patient experienced was exactly an acute limb ischemia (ALI) 3 days after traumatic anterior knee dislocation. ALI is defined as a sudden decrease in limb perfusion causing a potential threat to limb viability, with a risk of major limb loss. “Acute” means that the complication occurs within 2 weeks from the traumatic event [10]. When the artery injury is complete with sudden lumen obliteration, as it often happens in high-energy trauma, the clinical picture appears immediately clear. The manifestation delay occurs especially when the injury (more often in low-energy trauma) causes an intimal lesion that could develop thrombosis and vessel occlusion up to 72 hours later. In the presence of a clinical suspect of ALI, CT angiography and immediate revascularization are recognized to be the gold standard [11]. The
patient often does not complain of any symptoms during occlusion onset [12]. This is the reason why many authors declare that, after knee dislocation, the presence of distal pulses and normal distal capillary refill are not enough to rule out vascular injuries and instrumental analysis, such as angiography, should be performed anyway [13].

Basing on the rare case reports reported in literature, the authors focused on the importance of performing the CT angiography. After diagnosis of traumatic anterior knee dislocation, calling in the senior consultant on duty is advisable and relocation must be confirmed by radiographs. Then, Doppler ultrasound should be performed so as to deeply assess pulselessness or vascular damage. In case of a positive report, CT angiography and vascular treatments must be performed urgently. In case of absence of alterations detected by ultrasound and in case of other urgent problems affecting the patient, the CT angiography could be delayed. However, the ultrasound negative report does not rule out the need for CT angiography, and authors suggest performing it as soon as possible. Early diagnosis of vascular involvements prevents irreparable damage [14]. Seventy-two hours of clinical observation are advisable. These recommendations are based on our single-case experience, and on the rare case reports reported in the literature, so further investigations and much more experience would be essential to strengthen them. After the complete revascularization, the patient experienced her second major complication: local revascularization syndrome. This is not a rare complication affecting patients after revascularization procedures. An incidence rate of 20% of compartment syndrome has been reported in ALI patients undergoing reperfusion [15]. Dermatofasciotomy of all compartments of the leg should be immediately performed in the presence of a clear diagnosis of compartment syndrome [16]. Other authors reported the need of fasciotomy after development of popliteal artery occlusion in TKA [17].

Summary

Anterior traumatic dislocation of TKA is a rare and challenging event.
Before focusing on implant stability, we should be very careful about vascular and nervous impairment that could onset up to 72 hours later. Even if not supported by clear scientific literature, in the case of anterior traumatic dislocation of TKA diagnosis, CT angiography should be performed to rule out vascular lesions.

References


