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Case report

Acute arterial occlusion in the midpiece of femoral artery following total knee arthroplasty: Report of one case

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ABSTRACT

Acute arterial occlusion is a rare complication following total knee arthroplasty (TKA). The incidence as reported previously is from 0.03% to 0.17%; however, the sequelae can be disastrous because of its potential threat to limb loss. We report a case of acute arterial occlusion in the midpiece of femoral artery following TKA occurred 40 min postoperatively. The occlusion site existed at the midpiece of femoral artery is uncommon. Arterial circulation of the lower limb could not be restored by the thrombolysis and thrombectomy treatments performed within 11 h after TKA. In the end, amputation had to be carried out. In the treatment of acute arterial occlusion following TKA with a tourniquet, it is important to fully consider that arteriosclerosis may induce atheromatous plaque disruption, which might be the reason for acute arterial occlusion.

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Introduction

Acute arterial occlusion is rarely associated with total knee arthroplasty (TKA). The incidence of the complications after TKA including arterial occlusion, arteriovenous fistula, arterial aneurysm, and arterial severance is reported from 0.03% to 0.17%.^{1,2} There are approximately 35 cases that have been reported in the orthopedic literature until 2006.³ Approximately 46 cases have been reported in the orthopedic literature until 2015.^{1,2,4–18} Nonetheless, the infrequency of these complications may make the diagnosis and treatment extremely challenging to surgeons, and limb-threatening ischemia may occur subsequently.^{3,4} We report a case of acute vascular occlusion in the midpiece of femoral artery following TKA while aggressive thrombolysis and thrombectomy could not restore arterial circulation of the lower limb, and discuss the possible etiology causes.

Case report

The patient, female, 64 years old, had severe osteoarthritis for 12 years in the left knee. She had no peripheral vascular diseases or cardiovascular system diseases, no history of long-term lying in bed

and usage of glucocorticosteroid or smoking. The clinical vascular status and electrocardiogram were normal, no atrial fibrillation.

TKA was initiated with a tourniquet which was inflated to a pressure of 270 mmHg. Tourniquet time lasted for 118 min. After we released tourniquet, obvious pulsations could be palpated on the dorsalis pedis artery. 40 min postoperatively, the on-call resident found the dorsalis pedis artery pulsations turned weaker and weaker. She was guided to flex and extend her left knee continually, and no substantive hemorrhage was found. 40 ml blood volume was aspirated in the drain bottle in 2 h. Doppler examination showed no arterial signal below the midpiece of femoral artery. 6 h postoperatively, 10 mg Rivaroxaban (Bayer, Germany) was taken for routinely anticoagulant therapy. The femoral artery angiogram demonstrated the complete occlusion with abrupt cutoff of the femoral artery above the adductor magnus tendon hiatus furcation (Fig. 1), and there was no occlusion in inferior segment of the femoral artery and the popliteal artery (Fig. 2). 0.2 M international unit (IU) urokinase was injected in the same session 9 h postoperatively, but no effect. 11 h after TKA, thrombus aspiration was performed with 5-Fogarty aspiration catheter which was introduced from distal to proximal direction. After aspiration of 5 cm fresh thrombus material and injection of 0.2 M IU urokinase, the circulation was not restored. Disarticulation at the left knee was performed 4 days later due to ischemic necrosis. In the procedure, we examined the whole segment of popliteal artery, and found that there was no injury, no mural thrombosis formed in the lumens and the ectotheca of the popliteal artery was complete.

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Fig. 1. The femoral artery angiogram demonstrated complete occlusion with abrupt cutoff of the left femoral artery above the adductor magnus tendon hiatus furcation, 8 h after total knee arthroplasty in the left knee.



Fig. 2. When the catheter passed the occlusion site, angiogram showed that there was no occlusion in the inferior segment of the femoral artery and the popliteal artery; the flow of contrast medium was unobstructed.

Discussion

Acute arterial thrombosis after TKA is remarkably rare. We have reviewed the published reports associated with arterial occlusion, and summarized the features in [Table 1](#).

Approximately 46 cases have been reported in the orthopedic literature until 2015.^{1,2,4–18} Popliteal artery thrombosis is the most

frequent one. According to the report of Holmberg¹⁰ in 1996, popliteal artery thrombosis was 65.9%. In our review, it accounted for more than 80%. The case reported here is, to our knowledge, the first one in the literature that embolism segment was located at the midpiece of femoral artery, above the adductor magnus tendon hiatus furcation.

It is impossible to directly injure the midpiece of femoral artery in TKA, so the reason for arterial occlusion is incomprehensible. If we suppose that superficial femoral artery depressed by tourniquet resulted in thrombosis, and the thrombus flow to the distal femoral artery induced occlusion at the arterial branch, thrombolytic and the thrombectomy should be effective. But it turned out to be in vain. So we believed that the arterial thrombosis was not the real etiopathogenesis. Besides the thrombosis, acute arterial occlusion can also be caused by embolization of disrupted atheromatous plaques.^{1,11} It has been suggested that the mechanical pressure of the tourniquet may traumatize atheromatous vessels, causing fractures and dislodgment of the plaque, especially for the old patients. The vessels are calcified and lost its elasticity, and intimal tear and plaque embolization could result from the fixation of the femoral artery by the tourniquet and the stretching of the distal artery during intraoperative manipulation.^{11–13} In the procedure of artery angiogram, we saw the catheter could be introduced in close proximity to the artery tunica intima and passed smoothly through the occlusion site ([Fig. 3](#)). Angiogram showed that there was no occlusion in the inferior segment of the femoral artery or the popliteal artery. All the signs indicated that the pathologic changes such as arteriosclerosis or atheromatous plaque preexisted at the occlusion artery segment. When we released the tourniquet after surgery, the rapid changes of arterial pressure and ischemic reperfusion injury might induce an intimal injury or atheromatous plaque disruption in the femoral artery. Hemorrhage in such plaque tissue led to intimal bulging, which resulted in the arterial occlusion in the present case.

The curative effect of artery occlusion following TKA was poor, as evidenced by high rates as 70% of limb loss in previous reports.⁴ It remains controversial to determine the best therapy. Necessary surgical treatment is possible with bypass grafting and revascularization^{2,4} or thrombectomy using aspiration catheter^{7,8} including prior thrombolytic therapy.^{5,6} Thrombectomy is the most frequently described treatment, whereas the result is complete recovery or amputation. Early diagnosis and intervention are the keys to success, but the outcome varies.¹¹ We performed thrombolysis and aspiration thrombectomy without delay. But unexpected amputation had not been prevented in this case. Calligaro² reported the largest single-center experience with management of acute ischemic complications associated with THA and TKA. He claimed that this complication was best managed by an aggressive protocol including arterial bypasses and emergency revascularization. Arterial thrombectomy was successful in approximately one fourth (5 of 18, 28%), only when acute thrombosis without associated intimal damage. In our case, the femoral artery intimal injury and atheromatous plaque disruption were the most probable reasons for occlusion, and no thrombosis existed. Therefore, thrombectomy could not restore the circulation. For this patient, arterial bypasses may be the better choice. The key point of the failure was that we did not find the real etiopathogenesis for artery occlusion in time.

Arterial complications following TKA often occur in patients with an existing history of peripheral vascular disease or high risk factor diseases-related chronic arteriosclerosis such as hypertension, diabetes, rheumatoid, thromboangitis obliterans, hyperlipidemia, prior smoking and obesity. But this patient did not have any such history. It is suggested that there may be other undetermined risk factors and careful vascular examination that should be

Table 1

The features of arterial occlusion in the reported cases.

Author	Publishing time	Cases	Arteries affected	Therapy
Rand JA	1987	3/9022	Popliteal artery	
Calligaro KD	1994	7/4097	Posteriortibial artery(3/7) Popliteal artery(4/7)	Femorodistal bypasses
Ohira T	1997	1	Popliteal artery	Thrombolytic therapy with urokinase
Kobayashi S	1999	1	Popliteal artery	Thrombolytic therapy with urokinase/prostaglandin E1
Berger C	2002	1	Posterior tibial artery	Thrombectomy using aspiration catheter
Calligaro KD	2003	24/13618	Popliteal artery	Thrombectomy/Infrapoplitealbypass/Fasciotomy
Matziolis G	2004	2	Popliteal artery	Thrombectomy using aspiration catheter
Gregory PC	2006	1	Posteriortibial artery	Thrombectomy using aspiration catheter
Bayne CO	2008	1	Popliteal artery	Arterial anastomosis

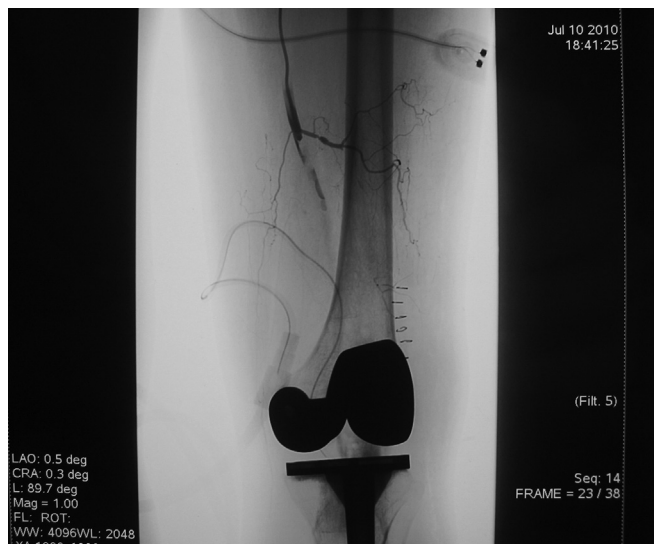


Fig. 3. The catheter could be introduced against the artery tunica intima smoothly past the occlusion site in the distal and proximal direction in the procedure of the femoral artery angiogram.

performed for all patients, not only to those with vascular disease history. To our knowledge, the only risk factor for this patient's acute arterial occlusion might be the time of using tourniquet and her age. It is important to fully consider that arteriosclerosis or atheromatous may induce intimal injury in the treatment of acute arterial occlusion with a tourniquet following TKA. For old patients, as the time of using tourniquet become shorter, the risk of artery intimal injury and atheromatous plaque disruption will decrease.

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