

## Case Study

# Delayed Diagnosis of Ischemia after Popliteal Artery Injury during total Knee Arthroplasty

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## Introduction

Over 600,000 TKA are performed annually in the USA. Injuries to the popliteal artery are unusual, estimated to occur in less than 0.2% of cases [1-3]. Three mechanisms of injury have been reported, the use of a pneumatic tourniquet in patients with advanced atherosclerosis [4], sharp, direct trauma to the vessels and stretching or kinking of the artery when the knee is manipulated [5,6]. It is generally believed that popliteal artery occlusion rapidly causes acute ischemia after popliteal artery occlusion because of the paucity of collaterals. Cases of acute ischemia after TKA that slowly evolve and that have been diagnosed and treated on a delayed basis have been rarely reported [3]. This may be because delays in diagnosis frequently result in major morbidity and litigation.

Since popliteal occlusion during TKA is unusual many vascular and orthopedic surgeons may only encounter it once or twice during their careers, if ever. Surveys of vascular surgeons about their experience with these injuries have not had good response rates with only a 6.8% response rate in one series [5]. In this retrospective review of 28 cases that were collected from completed litigation we analyzed the factors that led to the delay in diagnosis and propose changes to improve clinical practice, improve results and reduce the incidence of malpractice claims.

## Patients & Methods

The records of 28 patients from 28 different institutions who were diagnosed with popliteal artery injury during TKA and after leaving the PACU that occurred between 2007 and 2018 and resulted in a medical malpractice claim that either settled or resulted in a jury verdict were reviewed. Two patients were excluded. One thrombosed a PTFE bypass and one occluded an SFA stent that had tourniquets

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applied directly over them. All of the cases were reviewed for litigation by one author (PC). The cases are part of the public record and consent to use the anonymous data was obtained from the patient's lawyers.

## Results

There were 16 men and 12 women aged 48 to 79 years. Three were diabetic, thirteen had hypertension, seventeen were on cholesterol lowering medication and seven were active smokers. None had known pre-existing atherosclerosis or significant arterial calcification on plain x-rays. None of the patients reported claudication pre-operatively. There was no significant atherosclerosis found on the subsequent imaging or during the corrective vascular surgery. The diagnosis of ischemia was made between 12 hours and five days post-operatively.

All patients had normal pulses documented preoperatively. Eighteen patients had Jones dressings that included their foot so an accurate pulse examination was not possible until the dressing had been removed. Nineteen patients were first noted to have a change in their pulse status at the time the diagnosis of ischemia was made. Nine patients had their pedal pulses recorded by either the nurse or PA as normal at the time the diagnosis of ischemia was made and six patients still had normal pulses recorded in the records after the diagnosis of popliteal artery injury had been established. All 28 patients had their CRT recorded as normal and their "perfusion" listed as normal up until the diagnosis of popliteal artery occlusion was made.

Pain that was more severe than expected was reported by nineteen patients. The physicians or physician extenders did not document the exact location or quality of the pain that these patients were experiencing in the medical records. There was either no examination or only a cursory examination of the foot documented in the medical records. The pain in these 19 patients was attributed to their operation by their treating physicians or physician extenders in the medical records and all had their pain medication increased. Retrospective analysis of the nursing notes over the course of multiple shifts clearly demonstrated that all of these patients had pain out of proportion to their examination in an area, usually the foot, where pain would not normally be expected. The other nine patients also had pain of varying severity outside of the expected post-operative area documented by multiple nurses. The presence of a regional anesthetic or narcotic use may have masked the severity of the pain in these 8 patients. Retrospectively, the distribution of the pain correlated with the distribution of pain that would be expected from distal ischemia.

Numbness developed in all 28 patients in distributions not specific to one nerve. In all cases there was a normal sensory examination documented at least on one occasion in the post-operative period before the diagnosis of ischemia was confirmed. In spite of the use of short acting local anesthetics the nerve blocks were stated to be the cause of some of the sensory neuropathies up until four days post-operatively in 16 patients. Numbness in the toes was attributed to femoral blocks in seven cases even though the toes are not in the distribution of the

femoral nerve. Similarly, medial foot and toe numbness was attributed to the peroneal nerve in 6 patients. The causes of these presumed peroneal nerve symptoms of delayed onset were listed as being due to intra-operative nerve stretching, use of a tourniquet or dressings that had been applied too tightly even though at least part of the numbness was outside of the area innervated. In most cases the distribution of the numbness was poorly documented by the treating physicians. Motor neuropathies occurred in 20 patients. The presumed causes of these motor neuropathies were similar to the causes listed for sensory neuropathies.

The etiology of the popliteal injury was confirmed by arteriography in 9 patients, CT angiography in 4 (limited by scatter from the knee prosthesis), Duplex scanning in 4 and intra-operatively in 13 patients. There was one popliteal AVF. This was stated to have been caused by the saw. Eight partial or total transections were believed to be caused by instruments. One of these was felt to be secondary to a drill injury because of the nature of the operative findings. In the other cases the exact instrument that caused the injury was not identified. Thirteen intimal injuries with thrombosis were listed as having been caused by operative stretching. The causes of the other seven thromboses were not identified because the injured area was bypassed and not explored. There were no pseudoaneurysms found in this series. Interestingly, there was no reported excessive bleeding in any of these cases when the tourniquet was released. Four of these injuries were directly repaired with patches after thrombectomy. The majority underwent bypass after thrombectomy failed to restore flow.

Compartment pressures were measured before the diagnosis of a popliteal artery injury was made in six patients. In all cases the compartment pressures were in the normal range. All six had evidence of compartment syndrome when they were finally operated upon. All 24 patients underwent fasciotomies at the time of popliteal exploration because of the delay in diagnosis. Eighteen patients ultimately underwent major amputations because of their advanced ischemia and muscle damage. There were 15 above knee and 3 below knee amputations. Ten patients retained their legs. They all suffer from the consequences of severe ischemic neuropathy.

During discovery surgeons were queried about the reasons for the delay in the diagnosis of ischemia. Three quarters stated that it was their belief that if the popliteal artery was disrupted it would cause immediate severe ischemia because of the lack of collaterals around the knee. These surgeons quoted six hours as the length of time that the calf could tolerate warm ischemia [7]. Six believed that regional blocks could last up to 3-4 days depending upon how the patient metabolized the local anesthetic.

## Discussion

According to the American Academy of Orthopedic Surgery the number of TKA performed annually is expected to increase to 3.48 million by 2030. Allowing for an approximate 0.2% rate of popliteal artery injury we can expect close to 7,000 of popliteal injuries to occur in 2030. Both orthopedic and vascular surgeons are aware that total ischemia causes signs and symptoms that manifest themselves quickly and dramatically [7]. How dramatically and quickly ischemia develops depends upon the number and adequacy of the geniculate collaterals. There is a 38% reported incidence of geniculate artery injury during TKA which may explain the observed variability in presentation after arterial injury during TKA [8]. As this series demonstrates these geniculate collaterals can provide some perfusion to the

lower leg causing the signs and symptoms of ischemia to come on slowly and insidiously and CRT to be normal. There are many pitfalls that surgeons fall into when signs and symptoms are not dramatic that will be discussed.

Capillary refill times are very subjective and observer dependent. They have not been subject to rigorous comparative testing to assess their accuracy. It has been reported that a normal CRT does not give appropriate information about the adequacy of perfusion and cannot be relied upon [9]. An increased CRT suggest ischemia and must be evaluated with more objective testing. As this series demonstrates CRT was totally inaccurate in diagnosing the presence of ischemia in these TKA patients with popliteal artery thrombosis. CRT should not be used when assessing post-operative TKA patients because it can provide misleading information and delay the diagnosis of ischemia.

The presence of palpable pulses in this series of patients despite the presence of ischemia was also found in prior reports by orthopedic surgeons [3]. Numerous studies have questioned the reliability of distal pulse examination in out-patient settings when the patient is in pain or hypothermic [10,11]. Lundin, et. al. demonstrated an underdiagnosis rate of 30 % among vascular surgeons when an ankle:brachial index (ABI) of 0.76 was used for the cutoff of whether or not a pulse would be palpated [10]. In light of the poor accuracy when using CRT and/or distal pulse examination, some objective means of testing the circulation must be encouraged if there is any suspicion of a vascular injury, pain or neurological changes outside of the operative field or suspected ischemia. An ABI is no harder to obtain than simply taking a blood pressure. In patients who do not have normal pulses or any history of peripheral vascular disease we recommend that an ABI be obtained both pre-operatively either in the surgeon's office or pre-operative holding area and post-operatively in either the OR or PACU to diagnose any potential arterial injury in a timelier fashion. If a Jones dressing is utilized it should leave part of the foot uncovered and/or plethysmography can be used. Simply releasing the dressings when there is an absent or questionable pulse and monitoring the patient's pulse if there is any question of ischemia is highly unreliable and prolongs the ischemia time and the potential for permanent injury.

Post-operative pain that is not at the operative site or that is more severe than expected requires an objective assessment of the arterial circulation since pain is usually the first sign of ischemia. Similarly, any post-operative TKA patient who develops sensory or motor changes in the distal limb must be fully and accurately assessed both neurologically and from a vascular standpoint. Simply attributing these findings to a tight dressing, regional block or neuropraxia can delay the diagnosis if any of these symptoms are secondary to ischemia. Pulse examinations are unreliable in patients in pain who may be hypothermic, especially when performed by a busy surgeon [10]. This leads to delays the diagnosis and increases the risk of harm. "Maintaining a high index of suspicion" is essential [3]. Any suspicion of ischemia should lead to obtaining objective arterial testing and/or a vascular Surgery consult rapidly.

If compartment syndrome is suspected an ischemic etiology must be considered. It must be remembered that "normal" pressures in the compartments after TKA are not known [3]. Also, if the popliteal artery has been disrupted, the perfusion pressure to the compartments will be much lower than normal. This will make it possible for a compartment syndrome to occur at a much lower compartment pressure. An ankle pressure must be obtained and used when calculating the  $\Delta P$ .

This will markedly improve the likelihood of accurately diagnosing a compartment syndrome, if one exists, and leading to the correct diagnosis of ischemia.

## Conclusion

Arterial injuries that occur during a TKA may not be totally preventable even in the best of hands. As this study clearly demonstrates the effects of ischemia may slowly evolve. Eliminating the devastating consequences of a delayed diagnosis of ischemia is possible. Simply performing an ABI or digital plethysmography both pre-operatively and post-operatively in a patient with diminished pulses or a history of PVD or post-operatively in a patient with any suspicion of ischemia or unexplained pain or neurological change can accurately diagnose the change in perfusion pressure and facilitate a timely repair before permanent neurological damage or limb loss occurs. These tests are easy to perform and can be accurately done by an appropriately trained nurse. There is no contraindication to obtaining either an ABI or digital plethysmography in a post-operative TKA patient. Surgeons must be familiar with the distribution that a nerve block affects and the length of time that the nerve block can reasonably be expected to last. Dressings should be modified so that the foot and ankle are not covered with bulky dressings that make accurate assessment of the circulation difficult. It is essential that Vascular Surgeons educate their orthopedic colleagues about the risks of arterial injury during TKA.

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