[Orthopaedic Surgery]

Death After Closed Adolescent Knee Injury and Popliteal Artery Occlusion: A Case Report and Clinical Review

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A healthy adolescent male soccer player sustained a radiograph-negative, effusion-negative physeal injury of the proximal tibia from a ground-level fall with traumatic occlusion of the popliteal artery. Orthopaedic evaluation and arteriography were delayed for 72 hours after the injury. He arrived at a tertiary referral center in multisystem organ failure secondary to lower extremity ischemic necrosis, septic pulmonary thromboembolism, and systemic shock. Emergent medical evaluation, a high index of suspicion, and a careful neurovascular examination are imperative after every closed knee injury in the young athlete.

Keywords: pediatric knee; injury; dislocation; proximal tibia fracture; popliteal artery; occlusion

Popliteal artery injury is an infrequent but devastating diagnosis after trauma to the adolescent knee during contact sports. Delay in presentation and restoration of blood supply are common[8,11,24,32] and increase the likelihood of subsequent permanent disability or amputation.[4,5,25] Even low- or moderate-energy trauma may result in popliteal artery injuries[4,22] that threaten the limb; however, more serious consequences are possible.

CASE REPORT

A healthy 13-year-old male athlete (body mass index, 21.8 kg/m^2) fell at full speed after contact with an opposing soccer player. He struck the ground with his right proximal tibia with the right knee hyperflexed as the foot plantar-flexed beneath his buttock. He was slow to rise and limped off the field with pain and difficulty when bearing weight on the extremity. The following day, he extended his knee and felt a sudden crack, continuing to favor his left leg. He vomited and had decreased appetite but did not seek medical care. On postinjury day 2, his right lower extremity was swollen with increasing pain, and he developed shortness of breath. On postinjury day 3, he experienced increased dyspnea, heavy palpitations, and slurred speech. His lower trunk showed mottled skin and purple striae. At this point, the patient was evaluated at an urgent care center and promptly referred to a regional emergency department (ED).

Upon arrival in the ED, he was afebrile but tachycardic and in mild respiratory distress. Right knee range of motion was diminished without knee effusion; there was significant calf tenderness and pedal edema. Radiographs of the knee and leg did not reveal fracture or dislocation (Figure 1). Ankle-brachial indices were not obtained. An ultrasound examination of the right lower extremity demonstrated deep venous thrombosis (DVT) of the popliteal vein extending cephalad to the common femoral vein. A computed tomography (CT) angiogram of the chest and abdomen demonstrated multiple lower lobe cavitary lesions, along with a 30% to 40% right pneumothorax (Figure 2), consistent with pulmonary emboli (PE)[20,36] and an infectious process. A chest tube was placed. Abdominal CT demonstrated mucosal edema in the esophagus, stomach, and small bowel. The white blood cell count was 1.0 × 10^3/μL (3.28-9.29 nL), platelets 45 × 10^3/μL (143-398 nL), lactate 29 mg/dL (6-20 nL), total bilirubin 1.7 mg/dL (0.2-1.1 nL), and D-dimer was >10,000 ng/mL FEU (<500 nL), consistent with sepsis and disseminated intravascular coagulopathy. Clinically, he was confused, combative, vomiting, and in persistent respiratory distress. He was transfused with packed red blood cells and fresh frozen plasma, sedated, intubated, and subsequently transferred to a tertiary care center.

In the pediatric intensive care unit, the patient's temperature was 39.7°C. He was tachycardic and hypotensive with delayed capillary refill throughout all extremities. Both lower extremities were edematous with absent pulses. Creatine kinase was 7787 IU/L (31-335 nL). Blood cultures grew methicillin-sensitive *S. aureus*. Antibiotics, fluids, packed red blood cells, fresh frozen plasma, inotropic medications, and intravenous steroids

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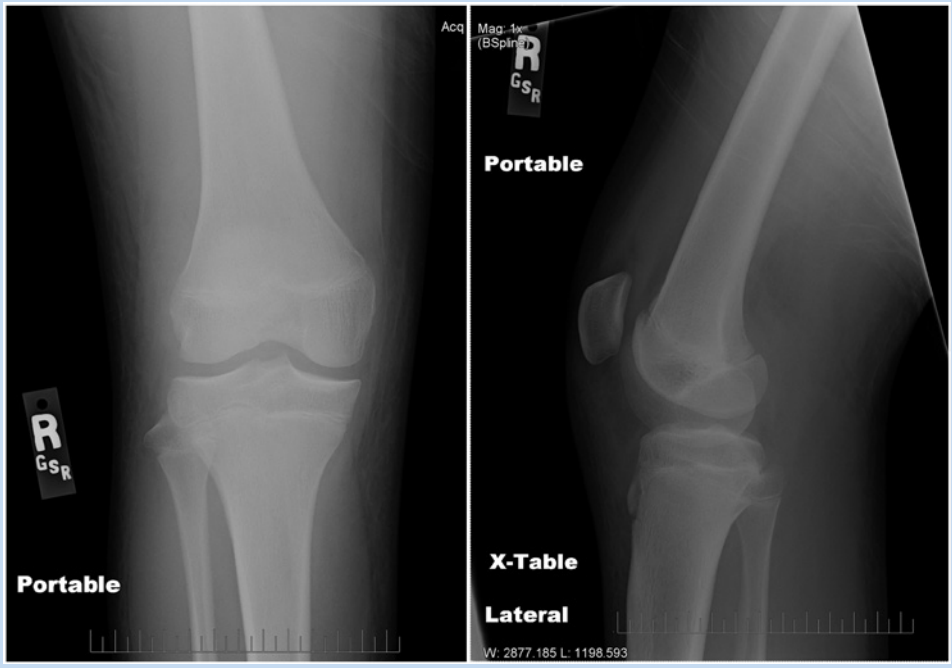

Figure 1. Antero-posterior (AP) and lateral radiographs of the affected right knee 3 days after closed injury. The AP view shows a congruent joint line, no interruptions of the subchondral or metaphyseal bone, regular physeal morphology, and normal tibiofemoral alignment. The lateral projection is slightly caudad without disruption of the tibial apophysis, the physeal line, or the posterior cortex through the physis. No significant effusion was present.

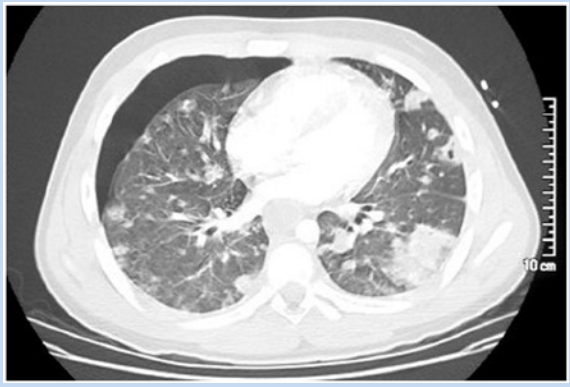

Figure 2. Representative axial cut from a computed tomography angiogram of the chest 3 days after knee injury demonstrating the right pneumothorax with scattered cavitary lesions, filling defects, and focal hemorrhages.

were administered. A follow-up CT angiogram confirmed left upper lobe PE, bilateral pulmonary hemorrhages, and multiple cavitary lesions. Extracorporeal membrane oxygenation was initiated. A right lower extremity arteriogram was consistent with spasm of the profunda femoris, the superficial femoral, and the common femoral arteries. The popliteal artery below the knee was not visualized, and there was no reconstitution of anterior tibial, posterior tibial, or peroneal arterial flow, even on delayed imaging (Figure 3). Following the arteriogram, a right leg fasciotomy was performed, but no viable muscle was found. Through-knee amputation was deferred because of persistent hemodynamic instability, and the ischemic, infected limb segment was wrapped in dry ice. The patient was monitored on full cardiopulmonary support and intravenous antibiotics over the next 5 days. On postinjury day 8, the family withdrew supportive care, and the patient expired from multisystem organ failure.

DISCUSSION

Significant pediatric knee injuries are relatively rare, with an incidence an order of magnitude less than those in the adult population.[17,27] From a series of 23,842 accidents sustained by children and adolescents, only 0.5% involved the knee.[17] Soft tissue injuries, fractures, and dislocations can present with or without findings on plain radiography. Several mechanisms are possible, including overt knee dislocations[29] and proximal tibial physeal injuries.[6]

Despite negative radiographs, the most likely mechanism of popliteal artery injury is transient proximal tibial physeal disruption.[14] This mechanism is supported by a history of loading a hyperflexed knee, posteriorly displacing the tibia at the epiphysis, and an audible reduction with extension.

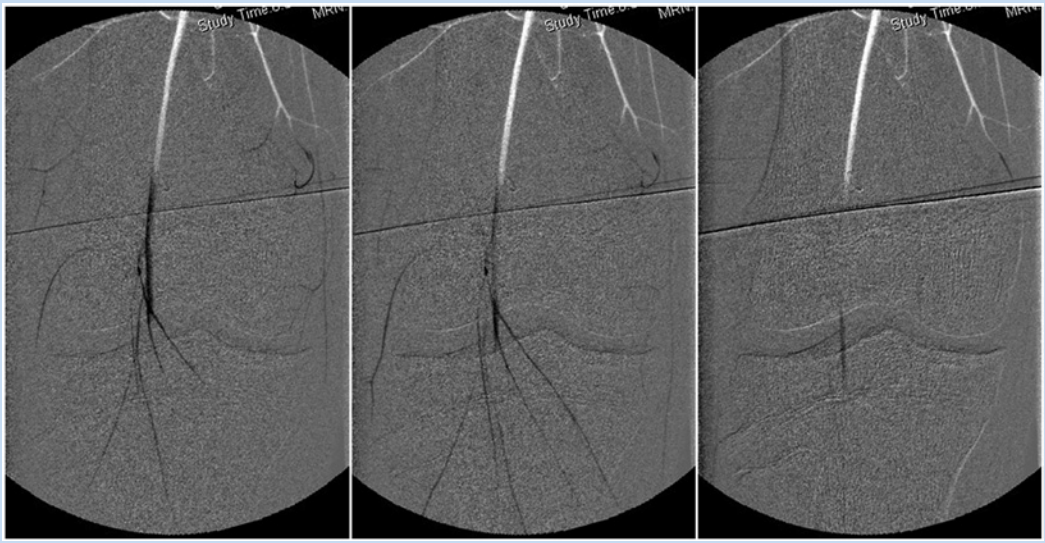

Figure 3. Sequential antero-posterior fluoroscopic images of the right lower extremity at the level of the knee joint 4 days after injury. There is an acute cutoff of the popliteal artery at the level of the knee joint with small faint and slow-filling collaterals. There is no reconstitution of the popliteal or any of the named tibial vessels.

Because the proximal tibial physis is extra-articular, there was no palpable knee effusion or hemarthrosis.

Proximal tibia physeal fractures are relatively rare.[15,30,37] From a single institution series of 2,650 long bone fractures, there were 16 (0.6%) proximal tibial physeal injuries, primarily in male adolescents.[19] The low incidence of this injury is related to the distal insertion of the collateral and capsular ligaments, shielding epiphyseal bone from shear and translational forces.[1] Additional protection is afforded by an extended metaphyseal insertion of the semimembranosus and a curvilinear physeal plane.[10] Disruption is most likely to occur during sporting activities,[34] classically by a hyperextension mechanism. However, similar to the present case, Hartley and Ricketts reported a football injury from a posterior force to the tibia where the knee locked in flexion and then autoreduced with forced extension.[14]

Following physeal disruption, the distal tibial fragment can translate posterolaterally to compress the popliteal artery.[1] The popliteal artery is tethered at several levels: the Hunter canal, the collaterals and branching geniculate arteries adherent to the posterior capsule, and the distal "trifurcation," where the anterior tibial artery dives through the interosseous membrane and bifurcates into multiple compartments.[15] In either hyperextension or hyperflexion, physeal displacement places tension and shear over this arterial segment, which can result in intimal tears or more severe vessel damage.[2]

Two small series over the past 50 years have documented vascular trauma after proximal tibial physeal fracture at 7.1% to 7.7%,[2,34] although it may be much higher.[1] The diagnosis of any popliteal artery injury is often delayed; presentation after low-energy events may be benign.[4,22] Delayed vascular injuries after long bone fractures include false aneurysm, thrombosis, and arteriovenous fistula.[31,35] Two recent reports describe posttraumatic popliteal arteriovenous fistula formation more than 10 weeks after closed treatment of simple proximal tibia fracture.[7,39] Although often coincident and highly morbid, venous injury and DVT occur in association with arterial occlusion.[9] Once arterial injury occurs, subsequent arterial rupture and thromboembolic events threaten the limb.[22] According to data from a 2002 to 2006 multicenter United States trauma registry, isolated blunt trauma to the popliteal artery in adult and pediatric patients still carries a 10% amputation rate.[16] Surprisingly, blunt trauma to the knee may have a 10-fold greater amputation risk than penetrating trauma,[8] perhaps due to a less dramatic presentation and resultant lower index of suspicion.

In children, there may be further misleading cues. Arterial injury and occlusion may not eliminate a dorsalis pedis pulse.[12] Even with successful closed reduction of a proximal tibial physeal fracture, vascular occlusion may persist.[2] When symptoms of ischemia are present, considerable debate exists as to the utility of early arteriography versus intervention without arteriography.[11,23,25,38] In lieu of a formal angiogram, duplex ultrasonography has comparable sensitivity and specificity for extremity trauma.[3] Noninvasive ankle-brachial indices may also be considered.[13,33]

Following a diagnosis of popliteal artery thrombosis, early revascularization is recommended due to generally insufficient collateral flow and elevated risk to the limb.[24] Excellent success rates have been reported in children after early vascular intervention.[32] There is an isolated report of successful thrombectomy 3 days after proximal tibial disruption and

popliteal thrombosis in a 13-year-old male patient.[21] However, in general, there is a precipitous decline in lower extremity limb preservation when treatment occurs more than 24 hours after vascular occlusion.[8,16,18,24,26,28,32]

CONCLUSION

This case illustrates the mortal risks of amputation and systemic inflammatory illness after popliteal artery trauma in the pediatric population. Emergent medical evaluation, a high index of suspicion, and a careful vascular examination are imperative after every closed knee injury in the young athlete.

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