



Delayed Arterial Spasm after Low Energy Trauma: A Case Report

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Abstract

Case: We report a unique case of a 30-year-old patient with Kawasaki Disease who presented with concern for an ischemic distal lower extremity after suffering a low energy twisting mechanism to his ipsilateral knee ten days prior. Angiogram demonstrated popliteal artery spasm and magnetic resonance imaging demonstrated no internal derangement of the knee. The patient regained full function and resolution of symptoms after administration of intraoperative nitroglycerine. The patient was discharged in stable condition with vasodilatory medication and did not have any recurrent complications.

Conclusion: We advocate for providers to be aware of this rare scenario of an ischemic presenting limb after a low energy trauma. Evaluation and quick diagnosis of arterial spasm and limb ischemia is paramount to help avoid long-term consequences.

Keywords: Trauma; Arterial Spasm; Vasospasm; Ischemia; Kawasaki disease

Introduction

Acute knee injuries are common and result in more than two million emergency department visits each year [1,2]. Initial management of knee injuries often includes evaluation for knee effusion, ligamentous injury, as well as plain films to rule out any potential fracture or ligamentous avulsion [3]. Additionally, due to their complex anatomic relations, evaluation and documentation of neurovascular status to the extremity is paramount [4].

The popliteal artery is tethered to the popliteal hiatus proximally and to the soleus arch distally [5]. Due to these structural relations, Popliteal Artery Injuries (PAIs) are frequently seen with high energy mechanisms including distal femur fractures, tibial plateau fractures, knee dislocations, or penetrating injuries [6]. PAIs can occur in the form of arterial transection, intimal injury, occlusion, or spasm, with the former being the most common [7,8]. These injuries often occur during the same time as the initial trauma due to the aforementioned tethering of the artery. Furthermore, these injuries typically present with rapid changes in clinical status and lower extremity perfusion.

Timeliness in recognition and diagnosis of PAI and maintenance of limb perfusion is critical for avoidance of limb ischemia as amputation rates are reported as high as 30% to 60% [9,10]. Initial management of patients with concern for PAI should begin with a basic physical exam with a focus on distal extremity pulses and digital capillary refill. Doppler ultrasonography may be utilized if there is any concern for lack of palpable pulses [11]. Ankle-Brachial-Indices (ABIs), or Arterial Pressure Indices (APIs), are an additional clinical tool used to assess for PAIs which can indicate possible arterial injury [12]. Should the aforementioned results raise concern, generally a computerized tomography angiography of the affected extremity may be obtained before considering possible surgical intervention.

In contrast to high energy mechanisms, low energy knee injuries rarely present with popliteal artery injury [13]. Additionally, incidences of arterial injury become exceedingly rare with blunt trauma knee injuries without radiographic evidence of fracture, ligamentous avulsion, or dislocation and are limited to case reports [14]. One notable exception to this is the classic “ultra-low energy” injuries seen among obese patients that can result in traumatic damage and arterial injury to an

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extremity [15].

To our knowledge there is no literature describing delayed popliteal artery spasm after a low energy knee injury. The purpose of this study is to report a unique presentation of delayed subacute limb ischemia in a young patient with Kawasaki Disease secondary to popliteal artery spasm who sustained an otherwise relatively atraumatic knee injury.

Case Presentation

A 30-year-old male with a past medical history significant for hypothyroidism and Kawasaki disease presented to our tertiary care facility with concern for acute limb ischemia of his left lower leg. Ten days prior the patient sustained a low energy twisting injury to his knee when the left knee became caught in a rafter while at work. There was minimal pain associated with the initial injury; however, it became increasingly painful 4 days after the injury. The patient presented to his primary care provider 8 days after the injury due to increased pain and difficulty ambulating. At that time, the patient was neurovascularly intact without skin changes and had palpable pulses. Radiographs taken at the time did not show any acute osseous abnormalities of the left knee (Figure 1).

The patient then presented to an outside facility two days later with a cool left lower extremity and non-palpable pulses. The patient had minimal leg pain at that time but reported some moderate left knee pain. On examination, the patient had minimal tenderness to palpation with a delayed capillary refill in the toes. Knee ligamentous exam was stable without any knee effusion present. Paresthesia's were present in the left lower extremity. Poikilothermia and a pale extremity were present. The Dorsalis Pedis (DP) pulse was neither palpable nor Dopplerable. Computerized Tomography Angiogram (CTA) was performed and demonstrated occlusion of the left anterior tibial artery with patent peroneal and Posterior Tibial (PT) arteries (Figure 2); however, the popliteal artery appeared to be shrinking in size (Figure 3). As there was concern for ischemia due to the clinical image of lack of perfusion, the patient was started on unfractionated heparin and transferred to our tertiary care center.

Upon arrival at our facility, the patient reported worsening paresthesia's and the left lower extremity felt "asleep" below the knee. Clinically, the patient had no palpable popliteal, DP or PT arterial pulses on examination. Motor function appeared to be grossly intact. On the basis of initial mechanism of injury, there was concern for a torsion type injury causing popliteal artery dissection and subsequent thrombotic occlusion by the vascular surgery team. After careful discussion, the patient elected to undergo angiography and



Figure 1: Anteroposterior and lateral radiographs of the left knee demonstrating no acute osseous abnormalities.



Figure 2: Coronal computerized tomography angiogram imaging demonstrating occlusion of the anterior tibial artery.



Figure 3: Coronal computerized tomography angiogram imaging demonstrating diminished size of the popliteal artery.

thrombolytic therapy.

The patient was taken to the endovascular suite where angiography demonstrated an infrapopliteal artery spasm. Intra-arterial nitroglycerin was utilized, and obvious correction of the spasm was seen. There did not appear to be any intimal injury to the artery. No plaque was observed, and 0% occlusion of the popliteal and AT arteries were noted (Figure 4). The patient's popliteal, DP and PT pulses were palpable after intervention.

Postoperatively, the patient was placed on amlodipine. Orthopedics was consulted to evaluate for ongoing knee pain. On examination the patient had an atraumatic appearing knee without any appreciable effusion, significant tenderness, or ligamentous laxity. Magnetic Resonance Imaging (MRI) did not show any abnormalities to the left knee (Figure 5).

Patient subsequently followed up one week after and was found to have a normal ankle brachial index of 1.1, which was equal to the contralateral side. The left knee pain was much improved and there did not appear to be any subsequent instances of the cool foot. At final follow up, the patient was back to work at his previous level of activity without any subsequent occurrences.

Discussion

This report presents unique circumstances of a young patient who suffered a low energy knee trauma and presented with a delayed

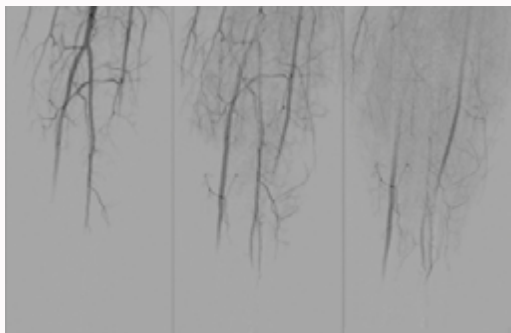


Figure 4: Intraoperative angiography demonstrating patent popliteal, anterior tibial, posterior tibial and peroneal arteries.

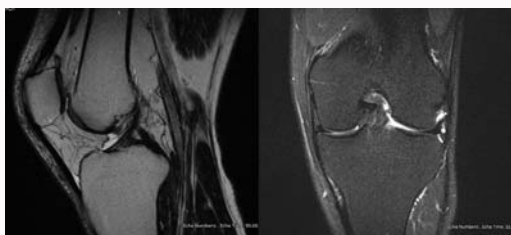


Figure 5: Sagittal T2 and coronal short tau inversion recovery MRI sequences demonstrating and intact knee without any acute abnormalities.

popliteal artery spasm. This case poses several unique circumstances regarding mechanism of injury, timing of symptom onset, and intraoperative findings. Despite a substantial delay from initial injury to eventual presentation, there were clinical concerns for delayed acute limb ischemia.

Prompt intervention is critical with any concern of PAI after blunt trauma as studies have demonstrated that delay in treatment can lead to long term functional deficits [16,17]. Multiple studies have evaluated that an ABI (or API), are one of the most effective and least invasive measures to evaluate for lower extremity arterial injury [4]. An ABI with a normal value (>0.9) is highly specific for ruling out a vascular injury [4,18]. Mechanistically, vascular injury associated with blunt trauma to the lower extremity is due to intimal disruption with subsequent thrombi and vascular occlusion. Additionally, PAIs associated with blunt trauma and arterial insufficiency can manifest over time which adds to diagnostic difficulty [19].

Contrary to acute presentations, arterial injuries following lower extremity injury rarely presents in a delayed fashion. The distinctive aspect of this case was the substantial delay from initial presentation to eventual clinical manifestation of progressive limb ischemia. There have been few reports in the literature of similar instances. In contrast to our relatively atraumatic mechanism of injury, a study by Wynes et al. discussed a similar instance of subacute lower extremity vascular ischemia weeks after definitive fixation of pilon-type ankle fracture dislocation that resulted in lower extremity amputation [16]. They further highlighted the necessity of identification and intervention for associated vascular injuries [16].

Post-traumatic arterial spasm of the upper extremity is a more frequently encountered phenomenon, especially in the setting of a dysvascular fracture or Pulseless Perfused Hand (PPH). While expectant management is thought to be an acceptable strategy due to the assumption of vasospasm, one systemic review by White et al.

found that only to 8% of the post-reduction cases were secondary to arterial spasm [20]. The PPH does differ from our case in that it is not a true ischemic insult, it has similar implications for the mechanism of vascular response to trauma. Delayed arterial occlusion after upper extremity fracture has been previously documented, however these occlusions have been secondary to intimal dissection or thrombosis as opposed to arterial spasm [21,22].

The patient in the current study presented with a low energy twisting injury after an injury at work. Retrospectively, there was question as to whether the patient sustained a spontaneous knee dislocation or other ligamentous injury during his initial injury. Data indicate that knee dislocations with spontaneous relocation occur up to 50% of the time [23]. Physical examination of the knee should be directed toward evaluation of any ligamentous insufficiency as well as monitoring of neurovascular status. Kim et al. report a similar presentation of delayed PAI due to blunt trauma without evidence of fracture or dislocation [15]. In their case, the patient presented one week after initial injury with MRI evidence of bicruciate ligamentous injury and CTA evidence of popliteal artery occlusion. Intraoperatively, emergent surgical intervention revealed evidence of thrombotic occlusion of the popliteal artery with collateral flow from the medial genicular artery. Despite similar timing and presentation of acute limb ischemia, the patient in the current study did not reveal any concern for ligamentous injury. Plain films did not reveal any fracture or persistent subluxation or dislocation of the knee joint. Further, MRI results did not demonstrate any ligamentous or soft tissue injury that would otherwise support this theory.

Another important aspect of this case were the CT and intraoperative angiographic findings. In contrast with vasospasm of small to medium sized vessels which is well documented in the literature, popliteal artery spasm as a cause of lower extremity ischemia is rare and uniquely appears most commonly after traumatic events such as Gunshot Wounds (GSW) [18,19]. A case report by Peach et al. discusses a case of angiographically confirmed tibial artery spasm acutely after a GSW to the lower extremity with spontaneous resolution [19]. They hypothesized that these traumatic spasms are due to external mechanical stimulation or kinetic energy dispersion can result in a “concertina-like” appearance on angiography [19]. Further, they recommended that in the absence of overt arterial wall injury, diagnostic modalities such as digital subtraction angiography and CTA should be utilized to better ascertain the etiology of the present ischemia. In our case, the initial CTA demonstrated a patent popliteal artery, peroneal artery, and posterior tibial artery with evidence of occlusion of only the anterior tibial artery. Intraoperative angiography demonstrated an infrapopliteal arterial spasm without evidence of thrombus in any of the aforementioned arteries which resolved with intraarterial nitroglycerine leading to restoration of arterial flow and distal capillary perfusion.

Finally, the patient has had a longstanding diagnosis of Kawasaki disease. Arterial inflammation is a known factor in the pathogenesis of Kawasaki disease [20]. Classically, this pathology manifests mainly within small to medium sized arteries, particularly in the coronary arteries. One such limitation of this case report is the inability to draw conclusion between association and causation regarding the patient’s prior medical diagnosis and relatively atraumatic injury. There are case reports of arterial spasm and thrombi in young adolescents without evidence of traumatic events [21]. However, this presentation remains relatively undocumented and unheard of in adults.

Conclusion

Popliteal artery injuries often occur after high energy trauma and resulting fractures, knee dislocations, or ligamentous injuries. This article presents a rare case of a delayed popliteal artery spasm after a low energy trauma in a patient with Kawasaki Disease. This case highlights the importance of continued evaluation and a clinical suspicion for this rare presentation. Providers should be aware of this rare presentation and not exclude it as a potential diagnosis.

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