Popliteal Artery Occlusion in a Young Baseball Athlete

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Popliteal artery occlusion is a rare vascular complication in athletes and has not been previously documented in baseball players. A 21-year-old male baseball player presented with a 10-month history of progressive claudication because of repeated trauma-induced popliteal artery occlusion from frequently practicing stealing bases by sliding down onto his right leg. He was found to have a transient deficiency in both protein C and protein S. The patient underwent percutaneous transluminal recanalization angioplasty followed by anticoagulation therapy, with good results. This case illustrates the importance of awareness of this potential complication in baseball athletes, work-up for a hypercoagulable state and the feasibility of angioplasty therapy in the management of ischemic limbs after trauma. (Circ J 2007; 71: 283–285)

Key Words: Baseball; Popliteal artery occlusion; Protein C; Protein S; Sport injury

Popliteal artery injury has been reported for various athletic activities, such as rugby, football, kayaking, weight lifting and wind surfing, but has been rarely associated with baseball.2 However, symptoms of arterial injuries are easily mistaken for neuromusculoskeletal problems, particularly in young athletes, and the diagnosis may be missed or delayed. We describe a young male baseball player who had occlusion of the right popliteal artery resulting from repeated trauma to the right popliteal area because of frequently practicing stealing bases by sliding down onto the right leg.

Case Report

A 21-year-old male presented with a 10-month history of progressive claudication in his right leg. He had been healthy and had no history of trauma except for frequently practicing stealing bases by sliding down onto his right leg while playing baseball. Over this period of time, he continued to play baseball as long as he could tolerate the pain, but the severity had been worsening to the point where he had to stop playing, as he had trouble walking for 100–200 m without several rests. He sought medical attention at another hospital and based on a Doppler ultrasound study was diagnosed as having an occlusion of the right popliteal artery without other visualized vascular abnormalities. The patient had no family history of peripheral arterial or venous thromboses.

On physical examination, the femoral artery pulse was normal on both sides. However, the pulse of the right popliteal artery was impalpable. The pulses of the right dorsalis pedis and posterior tibialis were diminished when compared with those of the left leg. The right calf muscle was found to be noticeably weak. The right mid-calf circumference was measured at 38 cm, which was 1.5 cm less than the left.

Most laboratory values were unremarkable, including complete blood count, prothrombin time and activated partial prothromboplastin time, liver enzymes, creatinine, and antinuclear antibody. However, both protein C (43%; normal range 70–130%) and protein S (35%; normal range 75–107%) levels were lower than normal. Chest X-ray was normal. Abdomen-to-knee computed tomographic angiography (CTA) was essentially normal, except for a segmental occlusion of the normally coursed right popliteal artery (Fig 1). An arterial angiogram confirmed a long occlusion (∼8 cm) of the right popliteal artery with patent collateral circulation to the calf (Fig 2).

The patient underwent percutaneous transluminal angioplasty to the occluded artery. The lesion was firm and barely crossed with a Terumo 0.035 guidewire (Terumo Corporation, Japan) following predilatation to the very proximal segment of the lesion with a Wanda 5.5×40 mm balloon (Boston Scientific, Galway, Ireland). The Wanda balloon was then replaced with a 6.0×40 mm balloon for inflation to open up the lesion with a residual stenosis of 30% and a satisfactory distal flow (Fig 2). After the procedure, he received warfarin for 3 months with the internationally normalized ratio adjusted to between 2 and 3.

The patient has had no further claudication. At 1-year follow-up, a Duplex/Doppler-ultrasound study of the right popliteal artery was normal. His antithrombin III (111%), protein S (111%) and protein C levels (81%) were normal. Magnetic resonance angiography (MRA) revealed patent right popliteal vessels (Fig 1).

Discussion

We believe this is the first reported case of lower-limb ischemia caused by repeated trauma-induced popliteal arterial occlusion in a baseball player.

There are various possible causative mechanisms for popliteal artery occlusion. Knee dislocation and fracture, tethering at the abductor hiatus and the soleus arch, epiphyseal separations of the proximal tibia and distal femur...
frequently result in popliteal artery injury and occlusion. However, the present patient did not have these problems. Popliteal arterial entrapment syndrome is a rare cause of occlusion most commonly found in athletes with hypertrophied calf muscles because of the abnormal positioning of the popliteal artery in relation to the medial head of the gastrocnemius muscle. However, compression can also occur to a normally coursed artery by anomalous musculotendinous formations lying between the 2 gastrocnemius heads, such as the aberrant head of the gastrocnemius, excessively hypertrophied soleus, plantaris, or popliteal muscle. The present patient did not have calf muscle hypertrophy by physical examination and the cross-sectional studies of CTA or MRA also showed no evidence of the surrounding muscles in the popliteal fossa compressing the normally coursed artery. Furthermore, the artery has remained patent after angioplasty alone, a good outcome that would not be expected in patients with popliteal entrapment syndrome if they did not undergo surgical release of compression or revascularization. Deficiency in both protein C and protein S has a much higher prevalence than deficiency of antithrombin III in Chinese patients. The patient had abnormally low values of both proteins at presentation but the values of each were normal at 1-year follow-up, suggesting a transient consumption of the 2 proteins, such as an ongoing thrombotic event, but we did not observe obvious thrombus in the popliteal artery. However, the trauma was intermittent and spanned a period of 10 months and residual microthrombus might escape detection. Similar transient low values of protein C and protein S have been reported in patients with previous lower limb revascularization who present with either recent occlusion (<6 months) or old occlusion (>6 months). Acquired deficiency in protein C and protein S can occur in other situations such as infection, severe liver disease, disseminate intravascular coagulation etc. but the present patient was healthy and well nourished. Thus, we postulate that the repetitive and intermittent vessel trauma and an associated hypercoagulable state because of low levels of protein C and protein S led to complete occlusion of the popliteal artery. Other coagulation markers that were not measured, such as D-dimer, fibrin degradation product, antiphospholipid, or abnormal platelets, may have given a comprehensive picture of the patient’s coagulation state.

Hypercoagulability caused by a deficiency of both protein C and protein S usually results in venous thromboembolic events such as deep vein thrombosis and pulmonary embolism and more rarely causes arterial thrombosis. In a recent study by Cho et al, 11 of 133 patients with cystic adventitial disease should be excised and a bypass performed to yield a good long-term result; which was not the case in this patient.

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(aged between 38 and 72 years) with peripheral arterial insufficiency were found to have levels of protein C and protein S that were less than 60% of normal. All those patients had occlusions in the lower extremities, and 2 with no history of trauma developed popliteal artery occlusion that was described as having "characteristic angiographic findings" of long segmental thrombotic occlusion without clear evidence of atherosclerosis in the main arterial trees. Of note, the arterial angiogram of the present patient with transient low levels of protein C and protein S fits this description.

Popliteal artery occlusion is treated with bypass surgery or thrombolytic therapy. We did not consider thrombolytic therapy useful in this case because the lesion was a long, firm, and chronic total occlusion even though the low values of protein C and protein S at presentation suggested an ongoing thrombotic event. Percutaneous transluminal angioplasty is reported to be the preferred treatment for traumatic popliteal artery occlusion as long as the extent of the disease allowed. The patient underwent successful percutaneous transluminal revascularization angioplasty followed by anticoagulation therapy for 3 months with good results.

This case suggests that although rare, doctors should be aware of the potential complication of trauma-induced popliteal arterial occlusion in baseball athletes. If popliteal artery occlusion occurs, the possibility of a hypercoagulable state should be investigated and treated accordingly. Percutaneous transluminal angioplasty appears to be a feasible treatment modality for revascularization.

References