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Our experiences with popliteal artery entrapment syndrome

Popliteal arter tuzak sendromu deneyimlerimiz

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ABSTRACT

Background: In this study, we aimed to present our experiences on patients undergoing surgery for popliteal artery entrapment syndrome.

Methods: Between March 2007 and June 2010, 12 patients (9 males, 3 females; mean age 23.4±6.6 years; range 16 to 40 years) who underwent surgery for popliteal artery entrapment syndrome in our clinic were retrospectively analyzed. Eleven patients had unilateral and one patient had bilateral popliteal artery entrapment syndrome. Of all patients, eight had type 2 disease, while the remaining patients had type 3 disease. Physical examination revealed non-palpable pulses. The Ankle-Brachial indices of the affected extremity varied between 0.30 to 0.46. The diagnosis was based on magnetic resonance angiography or computed tomography angiography.

Results: The flow of distal popliteal artery was evaluated by the manual Doppler ultrasonography and pulsatile flow was detected. Postoperative ankle-brachial indices of patients were measured between 0.90 to 0.97 values in all patients. The patients were scheduled for the outpatient clinic at one and three months after discharge for magnetic resonance imaging or computed tomography angiography examinations. The results were normal, which indicated that femoro-popliteal artery bypass grafts were patent and popliteal artery lumens at the thrombectomy sites were open. Ischemic ulcers were completely healed. There was no in-hospital mortality.

Conclusion: Our study results suggest that popliteal artery entrapment syndrome should be kept in mind in young patients with complaint of claudication and a detailed vascular examination is of utmost importance for the differential diagnosis and treatment plan in this patient population.

Keywords: Claudicatio intermittens; popliteal artery entrapment syndrome; young adult.

ÖZ

Amaç: Bu çalışmada, popliteal arter tuzak sendromu nedeniyle ameliyat edilen hastalara ilişkin deneyimlerimiz sunuldu.

Çalışma planı: Mart 2007 - Haziran 2010 tarihleri arasında kliniğimizde popliteal arter tuzak sendromu nedeniyle ameliyat edilen 12 hasta (9 erkek, 3 kadın; ort. yaş 23.4±6.6 yıl; dağılım 16-40 yıl) retrospektif olarak incelendi. On bir hastada tek taraflı ve bir hastada iki taraflı popliteal arter tuzak sendromu vardı. Hastaların sekizinde tip 2 hastalık var iken, geri kalan hastalarda tip 3 hastalık vardı. Fizik muayenede nabızları alınamıyordu. Hastalıktan etkilenen ekstremitede ölçülen ayak bileği brakiyel indeksi 0.30 ile 0.46 arasında değişiyordu. Tanı, manyetik rezonans anjiyografi veya bilgisayarlı tomografi anjiyografi ile kondu.

Bulgular: Manuel Doppler ultrasonografi ile popliteal arterin distalindeki akım değerlendirildi ve pulsatil akım tespit edildi. Tüm hastalarda ameliyat sonrası ayak bileği-kol indeksi 0.90 ile 0.97 arasında ölçüldü. Hastalar taburcu sonrasında birinci ve üçüncü ayda manyetik rezonans görüntüleme veya bilgisayarlı tomografi anjiyografi muayeneleri için polikliniğe çağrıldı. Sonuçlar normaldi; femoro-popliteal arter baypas greftleri açıktı ve popliteal arterin trombektomi yapılan lümenleri açıktı. İskemik ülser tamamen iyileşti. Hastane mortalitesi gözlenmedi.

Sonuç: Çalışma bulgularımız, kladikasyon yakınması olan genç hastalarda popliteal arter tuzak sendromunun göz önünde bulundurulması gerektiğini ve bu hasta nüfusunda ayrıntılı vasküler muayenenin ayırıcı tanı ve tedavi planlaması açısından çok önemli olduğunu göstermektedir.

Anahtar sözcükler: Kladikasyon sürekliliği; popliteal arter tuzak sendromu; genç erişkin.

Popliteal artery entrapment syndrome (PAES) is a relatively rare, but potentially limb-threatening vascular disease due to anomalous anatomical relationships between the popliteal artery and the surrounding musculotendinous structures. Also, this compression of popliteal artery is mainly caused by the medial head of gastrocnemius muscle.[1] In general, patients with PAES are young adults and they often present

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with claudication during walking in short distances. Stuart first described the presence of this anomaly in an amputated leg in 1879.[2] The prevalence of this syndrome was reported to be 0.15% by Gumustas et al.[3] however, PAES is an uncommon entity of which its actual prevalence is unknown. Postmortem studies have found the prevalence to be 3.4%.[4] There are various types of PAES in the literature. On the other hand, PAES can be divided into categories according to its etiology. In congenital form or anatomical form, compression of the popliteal artery is associated with the disorders of embryogenesis, leading either directly to the popliteal artery anomalies or alterations of the adjacent structures. In functional form, on the other hand, compression of the popliteal artery is due to hypertrophy of the gastrocnemius muscle following exercise.[5] Treatment of PAES is usually surgical.[6]

In this study, we aimed to present our experiences on patients undergoing surgery for the treatment of PAES.

PATIENTS AND METHODS

Between March 2007 and June 2010, 12 patients (9 males, 3 females; mean age 23.4±6.6; range 16 to 40 years) who underwent surgery for PAES in our clinic were enrolled in this retrospective study. A written informed consent was obtained from each patient. Eleven patients had unilateral PAES. Four of them were on the right side and seven of them were on the left side. One patient had bilateral PAES. Eight of 12 patients had type 2 disease, while the remaining patients had type 3 PAES. Main complaints included leg pain during exercise, claudicatio intermittens (limited walking distance of approximately 100 meters) and coldness on their toes. One of our patients was a 16-year-old girl and suffered from ischemic ulcers on her first and second toes. Physical examination revealed nonpalpable pulses in all patients. Complaints and findings on admission are shown in Table 1. Preoperative popliteal artery flow was evaluated using manual Doppler ultrasound, which showed low flow signals associated with deteriorated blood flow on the affected legs. The Ankle-Brachial indices (ABI) of these patients varied between 0.30 and 0.46 on the affected side. The diagnosis was based on magnetic resonance angiography (MRA) or computed tomography angiography (CTA). Computed tomography image of the right-sided PAES in a 28-year-old male patient is shown in Figure 1.

Table 1. Complaints and findings of patients

Complaints and findings	n	%
Leg pain during exercise	12	100
Claudicatio intermittens	12	100
Coldness of toes	12	100
Ischemic ulcers	1	8.3
Nonpalpable pulses	12	100

RESULTS

The patients applied to the outpatient clinic and hospitalized when diagnosed. One patient suffered from diabetes mellitus (DM), two from hypertension (HT), and one from dyslipidemia. Four patients were smokers. Medical history of a patient with bilateral PAES revealed that she was previously misdiagnosed with Raynaud syndrome and Buerger's disease, and she was, therefore, mistreated. Under general anesthesia, a curvilinear incision was made over the posterior aspect back of the knee. Skin and subcutaneous tissues were passed. We explored the structures of the popliteal fossa and found the compression due to the medial head or accessory muscle bundle (abnormal) of the gastrocnemius muscle on the popliteal artery. We turned the popliteal artery and vein and hanged them with silastic loops. We excised the abnormal head or accessory muscle bundle of the gastrocnemius muscle, which was compressing on the popliteal artery. After heparinization, transverse arteriotomy was performed on the popliteal artery. We opened the popliteal artery and realized that artery lumen was occluded by a blood clot (thrombus) in nine legs, whereas seven patients had left-sided PAES and one patient had bilateral.

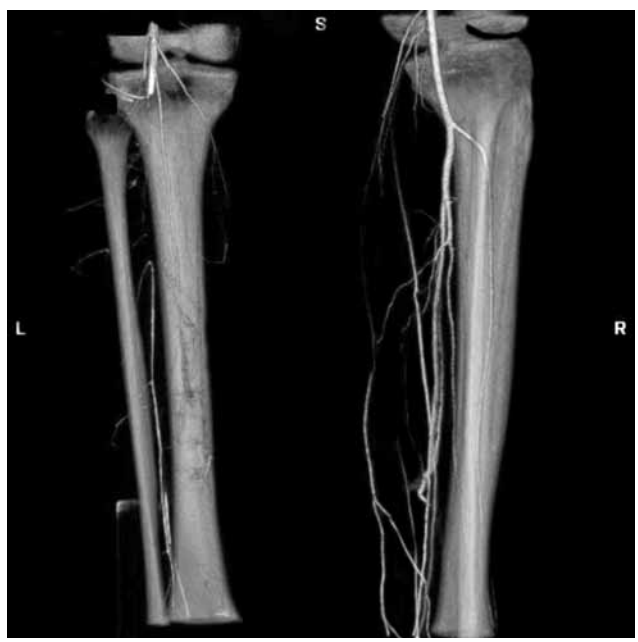

Figure 1. Computed tomography image of right-sided popliteal artery entrapment syndrome in a 28-year-old male patient.

We cleaned the arterial lumen and irrigated with isotonic fluid in these patients. Thrombectomy was performed at the proximal and distal parts of the artery. Thrombectomy material was removed from the artery and the distal part of the artery was, then, flushed with diluted papaverine solution (50 mg papaverine in 50 mL of NaCL 0.9%). Closure of arteriotomy was performed. Meanwhile, complete occlusion and dense endothelialization of the luminal surface of the right popliteal artery as a result of trauma caused by compression at the vessel wall were detected in four patients after the transverse arteriotomy. The same-side saphenous vein was harvested and prepared as a graft. In these patients, the femoral artery was found at the adductor hiatus, in other words, Hunter's canal exit on the lower third of thigh and popliteal artery was found in the upper calf. Then, femoropopliteal artery bypass was performed using this graft and distal artery flow was maintained. In all patients, incisions were managed for bleeding and were completely closed. The flow of distal popliteal artery was evaluated by the manual Doppler ultrasonography. Pulsatile flow was detected. Postoperative ABI's of the patients were measured between 0.90 and 0.97 values. The patients received 175 IU/kg/day low-molecular-weight-heparin. Following coumadinization, low-molecular-weight-heparin treatment was discontinued.

During the follow-up postoperative vital signs of the patients were stable and the patients were discharged with anticoagulant treatment (warfarin) to attain an international normalized ratio (INR) of 2-2.5. At three months, warfarin was discontinued and the patients received antiaggregant therapy (100 mg/day N-acetyl salicylic acid).

The patients were called for the outpatient clinic after discharge at one and three months to perform MRA or CTA. The results were normal, which indicated that femoropopliteal artery bypass grafts were patent, and popliteal artery lumens at the thrombectomy sites were open. Ischemic ulcers were completely healed in all patients. There was no in-hospital mortality.

DISCUSSION

It is known that popliteal artery is a continuation of the femoral artery. It begins at the opening (hiatus magnus) in the adductor magnus muscle and runs downward and enter the popliteal fossa which is a diamond-shaped space posterior to the knee joint bordered by the biceps femoris tendon proxylaterally, the semimembranosus muscle proxymedially, and the medial and lateral heads of the gastrocnemius muscle distally.[7] The popliteal artery normally runs downward and ends at the lower border of popliteus muscle by dividing into anterior and posterior tibial arteries.[7] During this course, the popliteal artery passes between the medial and lateral heads of the gastrocnemius muscle. The popliteal artery can be entrapped by neighboring muscles and tendons due to variations in embryologic development of the muscles and arteries.

Popliteal artery entrapment syndrome usually affects males younger than 30 years of age with a male to female ratio of 15:1 (male predilection).[8] Although bilateral popliteal arterial occlusion secondary to PAES is rare, PAES can be seen bilaterally in 25% of cases.[8] On the other hand, there are only a few reports on patients with bilateral PAES in the literature. Yavas et al.[9] reported one bilateral PAES patient with type 2 in their study.

In addition, Ivaki et al.[10] reported that a rare case of bilateral PAES who presented with the same symptom 11 years after his first experience of PAES of his left leg. In consistent with the literature, the number of male patients was more than female ones and the mean age of our patients was 23.4±6.6 years in our study. Also, we detected bilateral involvement in one patient (8.3%) who had type 2 PAES. As a result, patients with unilateral PAES should be carefully monitored for PAES on the controlateral side.[10] Also, there are some studies which reported familial PAES,[11] although none of our patients had this pathology. Similarly, Murray et al.[12] reported that PAES is an uncommon cause of peripheral vascular disease in young and normal weighted individuals, presenting as progressive claudication or sudden limb ischaemia and it may also present later in life with insidious symptoms relating to popliteal thrombosis or aneurysm. Claudicatio intermittens, coldness of toes, leg pain during exercise, and ischemic ulcers were remarkable in our patients. However, none of our patients suffered from sudden limb ischemia.

Furthermore, PAES be classified into six types according to the relation of popliteal artery base with surrounding structures.[5] Collins et al.[13] reported that incidences of type 1, 2, 3, 4, and 5 PAES were as 5%, 32%, 26%, 37% and 0%, respectively. However, most of our patients had type 2 PAES.

The most common risk factors of peripheral arterial disases were DM, dyslipidemia, smoking, and HT in our study population (Table 2). The patients with DM had well-regulated glucose levels thanks to oral antidiabetic drugs. Drug therapy was started in patients with dyslipidemia and/or hypertension. Smoking an

Table 2. The most common peripheral arterial disease risk factors in patients with popliteal artery entrapment syndrome

Complaints and findings	n	%
Diabetes mellitus	1	8.3
Hypertension	2	16.6
Dyslipidemia	1	8.3
Smoking	4	33.2

average of one pack of cigarettes per day for two to five years was identified in our smoker patients. However, we did not found any findings relating to chronic obliterative peripheral arterial disease with MRA or CTA in any patients.

Sinha et al.[14] reported that diagnosis and appropriate management of PAES were complicated by several factors: mainly its actual prevalence is unknown and the condition is rare. On the other hand, we believe that early diagnosis and accurate treatment can be established by using detailed vascular examination, medical history, and appropriate imaging studies.

Chronic compartment syndrome, medial tibial syndrome and PAES may cause claudication in young individuals.[15] Furthermore, the most significant clinical manifestation of PAES is limited walking distance in young patients who do not have any risk factors for atherosclerosis.[16] In a study, physical examination findings of the patients were found to be non-specific.[8] Also, Almeida et al.[5] reported that on physical examination, patients without thrombotic complications have normal pulses at rest. Otherwise, De Oliveira et al.[17] reported that a 20-year-old man with bilateral PAES was admitted to their medical service and was operated after the examination. The physical examination also produced normal findings, except the absence of right tibial pulses and decreased tibial pulses in his left leg in plantar flexion.

In another study, Tercan et al.[18] pointed out that the absence of foot pulses during passive dorsiflexion and active plantar flexion was characteristic. In addition, most patients, similar to our patient population, are diagnosed years after the initiation of symptoms and complications have already developed at the time of diagnosis. The main complaint of our patients was claudicatio intermittens with nonpalpable popliteal and distal arterial pulses. Extrinsic arterial compression of popliteal artery in PAES may cause chronic vascular micro-injuries and thrombus formation results in distal ischemia of the affected limb.[6,19] Early detection of PAES is important to prevent its progression.[10] However, diagnostic delay is common, as it usually occurs in young and athletic patients, in particular. The diagnosis and the graduation of both severity of dynamic circulatory insufficiency and arterial damage are usually made by CTA,[6] CT[20] or MR[20,21] and MRA.[21]

These radiological imaging studies can express the precise anatomical relations of muscle, bone, and the artery, as well as the degree of stenosis or occlusion of the popliteal artery.[19] Also, these imaging methods become more widespread in the diagnosis of popliteal artery disease.[3] In our study, we accurately diagnosed PAES after physical examination using MRA or CTA. The main goal of the treatment is surgical creation of normal anatomy within the popliteal fossa.[22] Also, Tercan et al.[18] suggested that PAES should be treated by surgery, regardless of the degree of symptoms.

Surgical treatment of PAES includes releasing of the vessel by extracting the muscle which causes entrapment and reconstruction of narrow lumen of the popliteal artery by endarterectomy or bypass grafting.[6] When PAES is detected early as the popliteal artery is intact, treatment of choice is to divide the abnormal muscle or fibrous tissue is required.[8] Furthermore, if the diagnosis is made after the popliteal artery is occluded or stenotic, the treatment of choice is vascular reconstruction by endarterectomy or bypass grafting in addition to the division of the anomalous musculotendinous structures.[8]

On the other hand, endovascular treatment such as percutaneous catheter directed with or without balloon angioplasty was another treatment modality. However, there were some studies suggesting that endovascular treatment was not effective without removing underlying reason of vessel entrapment, in which the risk of re-occlusion was high.[6,18] Also, Kwon[23] reported that endovascular stent application to the popliteal artery in a patient with PAES resulted in occlusion one year after the stent placement, and was treated with surgical bypass graft by reversed saphenous vein. Otherwise, treatment of the occlusion of popliteal artery in patients with PAES by angioplasty may be a feasible approach after exclusion of the factors which causes entrapment.[18]

In the present study, we performed surgical intervention, as they were ineligible for endovascular treatment modalities. During the operation, we detected compression of the popliteal artery in all patients. Furthermore, thrombus and complete occlusion were found in the popliteal artery in eight and four patients, respectively. We excised abnormal head or accessory muscle bundle of the gastrocnemius muscle in eight

patients and thrombectomy was performed in these patients; however, femoropopliteal artery bypass using the same side saphenous vein graft was performed in other patients. In the scheduled visits, femoropopliteal artery bypass grafts were patent and popliteal artery lumens at the thrombectomy sites were open.

However, our study has some limitations. First, this study was a retrospective study. Second, we had a small sample size and many patients with various types of PAES were excluded. However, we suggest that PAES may be missed during the diagnosis and, therefore, mistreatment may lead to undesired complications of PAES. Also, we believe that the correct diagnosis of the etiology of claudication is of utmost importance to avoid unnecessary medical, endovascular, or surgical treatments.

In conclusion, popliteal artery entrapment syndrome is a relatively rare, but potentially limb-threatening disease, which is predominantly seen in younger population. Popliteal artery entrapment syndrome should be kept in mind in young patients with a complaint of claudication and a detailed vascular examination is essential in the differential diagnosis and treatment plan.

Declaration of conflicting interests

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