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Popliteal Vascular Entrapment

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Popliteal Arterial Entrapment (PAE) was first reported in 1879 by Stuart, a medical student at the University of Edinburgh. Mr. Stuart observed, during the dissection of an amputated leg of a 64-year-old man, a popliteal artery coursing around the medial head of the gastrocnemius muscle and aneurysmal changes in the popliteal artery distal to the point of external muscular compression [1].

The first case of PAE was surgically treated in 1959, in a 12-year-old boy complaining of claudication after walking 300 meters. At surgical exploration, Hamming, at Leyden University in The Netherlands, found an occluded artery with an anomalous course medial to the medial gastrocnemius muscle. He transected the muscle and performed a successful popliteal artery thromboendarterectomy [2]. In 1979 Rich et al. published the first series of patients treated for PAE [3]. Many case reports were published in the ensuing years, as well as other small series [4]. Efforts to revise collective series were incomplet for lack of details and patient follow-up [5]. The functional form of entrapment was first described by Rignault et al. in 1985 in a report describes cases in which the anatomy of the popliteal fossa is normal [6].

Popliteal Vascular Entrapment Forum

In 1998, in Rome, the Popliteal Vascular Entrapment Forum was founded. Surgeons with the largest experience on this field all over the world were invited as founding members of the Forum. Significant efforts were made to collect different series with comparable criteria. The criteria established by the Society for Vascular Surgery were reviewed and accepted with minor changes (Table 1). Consensus was reached to consider both arterial and venous entrapment as a common disease defined *vascular entrapment* Venous entrapment was included in the classification as type V, being defined as primarily venous. A great dial of discussion was devoted to the functional form of entrapment. Symptoms are usually caused by hypertrophy of the muscles. *Functional entrapment* was included in the classification as type F.

Several other meetings followed, and the membership expanded to include representatives from the five continents of the world. A decision was made to schedule an annual muting, and a great effort was made to establish a common database cases from different countries. This was judged to be the only way to increase the amount of data to be analyzed to bring greater consistancy to reports published series.

Embryology and Anatomy

The arterial system of the leg develops from two embryonic arteries, both of which take their origin from the umbilical artery, a branch of the dorsal aorta. The axial artery is first identifiable in the 5–6 mm embryo (30 days intrauterine life). The external iliac artery appears in the 8 mm embryo (32 days intrauterine life) and subsequently gives rise to the femoral artery when the embryo reaches, the 11–12 mm stage (38 days intrauterine life). The axial artery runs longitudinally within the posterior compartment of the lower limb, while the femoral artery elongates in the anterior compartment. In its course behind the knee, the axial artery lies deep to the developing popliteus muscle (14 mm embryo, 42 days intrauterine life).

At this stage the axial artery may be divided into three segments: 1-proximal to (termed *arteria ischiadica*), 2-deep to (termed *arteria poplitea profunda*), and 3-distal to (termed *arteria interossea*) the popliteus muscle. At this stage a bridging vessel, the ramus communicans superius, enters the popliteal fossa through the adductor hiatus, acting as a conduit between the femoral artery and the arteria ischiadica. At the 18-mm stage (48 days intrauterine life), the arteria ischiadica gives rise to a branch at a point just proximal to the upper border of the popliteus muscle. This vessel runs superficial to the popliteus muscle (termed *arteria poplitea superficialis*) and distally joins the arteria interossea. The arteria poplitea profunda disappears. The adult popliteal artery is therefore the product of fusion of several embryonic elements. Both heads of the gastrocnemius muscle arise form the proximal tibial. As the infant develops, the origins of this muscle migrate cranially across the epiphysial plate onto the femoral metaphysis. As a result of differential movement of the two heads of gastrocnemius, the medial head eventually comes to lie in a more proximal position on the femoral shaft than

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Table 1. Classification of compressing structures causing politeal entrapment.

Type I: Popliteal artery running medial to the medial head of gastrocnemius
Type II: Medial head of gastrocnemius laterally attached
Type III: Accessory slip of gastrocnemius
Type IV: Popliteal artery passing below popliteal muscle
Type V: Primary venous entrapment
Type VI: Variants
Type F: Functional entrapment

the lateral head. The origin of the medial head of gastrocnemius in the adult lies directly caudal to the adductor hiatus, with the popliteal artery lying immediately lateral to it.

The embryologic studies of the popliteal artery entrapment syndrome pointed to a developmental variant in the stage of embryonic life between 14 and 22 mm, consisting of an abnormal migration of the medial gastrocnemius muscle, which does not complete its medially and cranially directed course or it catches the popliteal artery, which sweeps along medially with it. Developmental anomalies of the popliteal artery, with persistence of its ventral component, could account for the rare instances of entrapment caused by the popliteus muscle. However, a single anomaly cannot explain the final situation; often, in fact, the abnormalities are complex, not permitting a complete embryologic explanation [7]. The popliteal vein is involved more rarely because its embryologic development occurs later than that of the artery. The recently proposed modification of the classification tried to cover the most frequent embryologic anomalies and to classify the rarest of them as variants.

Incidence and Demographics

Since its first mention in 1879 [1], PAE has been described with an increasing frequency in the world literature. Gibson et al. [8] observed an incidence of 3.8% in a series of 86 postmortem limb examinations. Bouhoutsos and Daskalakis [9] treated 33 patients screened from a series of approximately 20,000 young vascular patients; the incidence was 0.165%. In a previous study [10] at our institution, we detected a similar incidence (2 cases out of 1212 patients studied during a period of 21 months). The real incidence of PAE is still difficult to calculate precisely because the diagnosis may be difficult at an early stage of the disease and some cases may be dismissed. However, all data from the literature indicate that the syndrome is more prevalent than has heretofore been appreciated [11].

In our series, comprising a total of 35 patients, PAE usually affected young males (age range 30–40 years) with a male/female ratio of 3.2:1. This sex ratio differs from the data of the literature in which a ratio of 10:1 has been reported. This is probably because we are a referring center for PAE and we see and operate on a great number of patients. Bilateral PAE has been described during the years more and more often. In our experience we found 20 (57.1%) patients with bilateral PAE. Two limbs were left untreated because they were asymptomatic and the patients were followed-up. One patient underwent surgical exploration, but vascular reconstruction was not carried out because of the risk of early thrombosis as a result of poor run-off status. The patient is still experiencing claudication with a walking distance of 300 meters. The popliteal vein was concomitantly involved in 9 (15.8%) limbs of our series.

Signs and Symptoms

Symptoms and signs in patients affected with PAE may be extremely variable. We categorized [12] two stages of this disease: stage 1, early and stage 2, advanced. A grading scale of symptoms was also proposed. According to the following classification, symptoms should be categorized: class 0, asymptomatic; class 1, pain, paresthesia, and cold feet after physical training (jogging and heavy work) in nonprofessional athletes; class 2, claudication (>100 m) while walking; class 3, claudication (<100 meters) while walking; class 4, rest pain; class 5, necrosis. In addition, all tests proposed by the Society for Vascular Surgery/North American Chapter should be adopted to better categorize the disease [13]. At an early stage, symptoms may range from none to pain, paresthesia, and cold feet after physical training (jogging or heavy work) in nonprofessional athletes.

In the advanced stages, the popliteal artery may undergo the typical changes seen in the advanced atherosclerotic process with the occurrence of post-stenotic aneurysmal dilatation or thrombosis. In such cases the usual presenting symptoms range from intermittent claudication to rest pain and necrosis. Because of the richness of the collateral circulation of the knee level through the superior, middle, and inferior genicular arteries when the popliteal artery is occluded, complaints may be limited to mild claudication while walking. Prolonged disease with a super-imposed atherosclerotic process may progress to a stable degeneration of the distal branches (tibioperoneal trunk and anterior tibial) with a deterioration of the run-off.

The occurrence of post-stenotic aneurysmal dilatation is rare (in our series 8 limbs out of 57). In such cases the popliteal aneurysm may be a source of embolization with the consequent risk, if left untreated, of limb loss or aneurysm complications. It is well known that complications occur in 18% to 31% of all patients with popliteal aneurysm who do not undergo operation, and that the incidence of limb loss increases dramatically once such complications have occurred, even with operative intervention [14]. Overall in our experience we observed 26 (45.6%) limbs with symptoms of class 1; 20 (35.1%) of class 2; 8 (14.1%) of class 3; and 1 (1.7%) of class 5. Two (3.5%) limbs were asymptomatic, and none of the patients were professional athletes. Seven (20%) patients participated in sports such as soccer, volleyball, and horseback riding, however. The occurrence of moderate to severe symptoms accounts for less than 16% of all patients affected with PAE, and these data support the finding that the progression of the atherosclerosis in PAE is a rare phenomenon.

We recommend the careful investigation of a PAE in all young patients, regardless the sex, who have signs and symptoms of pain, paresthesia, cold feet, and calf claudication after intensive physical training. Early diagnosis avoids more dramatic clinical presenting symptoms such as aneurysmal changes and arterial atherosclerotic changes. Another important aspect of PAE involves the differences between anatomic popliteal vascular entrapment and the functional form. Several authors have described the two forms as the same entity. Rignault et al. [6] first described a form of functional entrapment in an athletic subject who presented with hypertrophied gastrocnemius muscle without an anomalous relationship between the popliteal artery and its surrounding musculotendinous structures. Others [15, 16] have described a compartment syndrome resembling popliteal entrapment in well-conditioned athletes. Athletes can, in fact, experience deep calf muscle cramping, rapid limb fatigue, and occasional paresthesia

on the plantar surface of the foot. Akkerdijk et al. [17] showed that in healthy, highly trained subjects, popliteal artery compression or occlusion can be provoked by active plantar flexion of the foot (up to 72% of the arteries investigated).

Diagnosis

Signs and symptoms in a young patient should always raise the suspicion of possible PAE, and a careful examination followed by noninvasive evaluation should be performed in each case. The diagnostic evaluation of PAE is an important step in the treatment of this syndrome [10]. Doppler Continuous Wave (CW) with maneuvers that tighten the calf muscles, such as active plantar flexion against resistance, if correctly performed, can permit diagnosis, although false-positive results may occur. The pencil probe should be placed on the posterior tibial artery, avoiding sudden movements during calf muscle contraction, and the examination should be repeated at least three to four times.

Color Doppler represents a useful tool in diagnosing PAE and is best performed when the Doppler CW is positive. The popliteal artery should be scanned during calf muscle contraction. During maneuvers calf muscles push the artery deep into the popliteal fossa. The color Doppler should be carefully repeated at least three to four times after the artery displacement, placing the sample volume in the artery. In fact, if the sample volume is not correctly placed in the popliteal artery, be a false-positive that may result.

The noninvasive techniques have at times been found to lack specificity. To minimize their false-positive rate, computed tomography (CT) and magnetic resonance imaging (MRI) have been used to confirm PAE, simultaneously revealing detailed anatomic information about the popliteal fossa. The increasing specificity and sensitivity of CT and MRI in studying the popliteal fossa could represent a valid alternative to the more invasive digital angiography.

At present we continue to study the PAE with angiography during maneuvers, but at the same time we perform 3-D CT and MRI. However, a clear and precise preoperative characterization of the compressing structure is not always possible, and visualization of the distal run-off in cases of advanced disease is underestimated. We therefore still recommend digital angiography with maneuvers in all patients in whom the occurrence of atherosclerotic changes at the popliteal artery and its distal branches are present. This diagnostic tool permits better planning of the reconstructive operation.

Treatment

Management of the PAE syndrome is surgical, except in cases of very late diagnosis of a patient with an occluded popliteal artery without aneurysmal changes in which collateral circulation is considered satisfactory. Current data encourage the identification of patients with PAE at an early stage, when surgical treatment can be limited to musculotendinous section. In an our previous study [12] we clearly demonstrated that the only parameter influencing long-term outcome is age at presentation. This finding could be interpreted as signifying progressive degeneration of the popliteal artery. However, time cannot be considered the only factor causing stable popliteal degeneration. In fact, we have patients affected with bilateral PAE in whom arterial reconstruction was required in one limb and muscolotendinous section was the treatment of choice in the other. It is obvious that in these cases, time cannot be the only factor to explain the natural history of PAE.

We considered whether other factors, such as affected side, musculotendinous structures, or dominant limb, would explain our observations. However, no statistical differences were recorded. Reconstruction of the popliteal artery should be limited in length to the impaired arterial segment, avoiding anastomoses to the tibial vessels whenever possible. The presence of aneurysmal changes does not modify the surgical strategy. Autogenous veins should be used, rather than prosthetic materials. We recommend approaching the popliteal artery through a Z-shaped posterior incision. This is useful when more musculotendinous structures are involved. The medial incision, in fact, does not allow complete exposure of the popliteal fossa, and identification of the compressing structures may be difficult. However, this approach should be considered when extensive arterial impairment requires reconstruction with distal anastomosis to the tibial vessels.

In conclusion, we believe that attention should be placed on the diagnosis of PAE at an early stage in symptomatic patients, thus limiting surgical treatment to simple myotomy and avoiding vascular reconstruction. Myotomy offers the best results and eliminates the need for vascular reconstruction.

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