Part VIII

Popliteal aneurysm and popliteal vascular entrapment

Critical Ischemia of the Lower Limb Associated with Popliteal Artery Aneurysms

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Thrombosis and distal embolization are the mechanisms by which popliteal artery aneurysms may lead to ischemia of the lower limbs. Thrombosis usually proceeds slowly and is anticipated by multiple embolic events that reduce the distal run off, thereby increasing resistance to outflow within the aneurysmal sac and finally precipitating occlusion. The incidence of distal embolization is estimated of about 25% of the patients bearing popliteal aneurysms (1-3). This figure, however, probably underestimates its real incidence, as this latter varies with the care paid to the interpretation of angiograms.

In a few patients the slowly occurring process of thrombosis allows a collateral circulation to develop, thus avoiding the clinical picture of acute ischemia of the limb. It should be remembered that aneurysms of the popliteal artery are often associated, up to 40% of the cases, with aneurysms in other arterial sites, including the infrarenal aorta, which should be looked for and subsequently treated (4,5).

The great majority of popliteal aneurysms is non specific in its aetiology, as they are improperly called atherosclerotic aneurysms. Septic, dysplasic, and false aneurysms are rare: the present chapter will deal exclusively with critical ischemia associated with non specific popliteal aneurysms.

The typical patient with critical limb ischemia due to a complicated popliteal aneurysm is a man in the seventh-eight decade of life, with sometimes a history of leg swelling and local pain, and an incidence of risk factors, which is superposable to that of atherosclerotic arterial diseases at other sites. At physical examination ipsilateral popliteal pulse is obviously absent, whereas the contralateral may be aneurysmal, the leg is ischemic, with rest pain, often with swelling and cyanosis, and/or distal trophic lesions. Motory and sensory loss is absent.

Although critical ischemia is a severe condition, it still leaves enough time for a thorough study of the patient, in order to plan optimal treatment. Intravenous heparin should be started immediately, in order to achieve a PTT twice the normal values, and a complete arteriogram of the abdominal aorta and the lower limbs should be obtained, with non longer than a couple of days delay. Although Duplex Ultrasound alone already enables correct diagnosis to be made, arteriography is the key in planning treatment. Nevertheless, Ultrasound remains a useful diagnostic tool, as it can reveal a concomitant thrombosis of the popliteal vein, to be kept in mind, for the risk of possible pulmonary emboli, due to mobilization during lytic therapy or surgical manipulation.

When a patient with popliteal aneurysm presents with critical ischemia, corresponding to grades II and III, categories 4 and 5 (6,7), angiographic aspects may be different.

A narrow-lumen popliteal artery with one outflow vessel to the leg may be present, with a scarce or absent outflow below the ankle. A thrombosed above/below knee popliteal artery with late opacification of the peroneal or a tibial artery may also be the case. Thrombosis of the popliteal artery, with absence of leg outflow vessels and more or less developped collaterals may be evident. This latter situation is more frequently associated with acute, sensory-motor ischemia, than with critical ischemia. When angiography demonstrates a tiny popliteal artery, with an outflow vessel, whose origin is abnormal, kinked or stenosed, by-pass grafting to this vessel should be performed immediately below its origin, as in this case the popliteal artery aneurysm involves the origin of the anterior tibial artery and tibio-peroneal trunk. This condition occurs in nearly 40% of the popliteal aneurysms (8). In this case, dissection and excessive manipulation beginning from the distal aneurysmal popliteal artery down to the outflow vessel beside being unuseful may expose to further dislodgement of fresh thrombus from the artery to distality, and from an eventually partially thrombosed vein to the lungs. When the aneurysm is thrombosed and no distal run off can be visualized on arteriogram, intraarterial catheter directed thrombolysis is the best alternative to blind surgical exploration. The rationale for intraarterial preoperative thrombolysis is restoration of limb perfusion and arterial run off, in order to identify the best recipient axis for bypass grafting. As patients in critical ischemia do not require immediate revascularization, but can withstand an additional period of ischemia, they are particularly fit for a staged treatment, namely preoperative thrombolysis/surgical revascularization (9). In a recent series, of 13 patients presenting with acute thrombosis of a popliteal aneurysm, thrombolysis was successful in restoring outflow and directing appropriate grafting in 77% of the patients (9).

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Several large series are available on the overall clinical features and surgical treatment of popliteal aneurysms (1,2,4,10-17). On the other hand, series dealing with management of thrombosed popliteal aneurysms and the ensuing ischemia are limited to a relatively small number of patients (9,18). Overall, graft patency and limb salvage rate are respectively of 50-70%, and over 90% at 5 years in the largest series (1,2,4, 10-20). In the acute or critical setting results are less encouraging, with an amputation rate reaching 30% (5).

Personal experience is limited to 4 cases, all males, of a mean age of 74 years. All presented with critical lower limb ischemia as defined above (6,7). At arteriography, a thrombosis of the popliteal artery at the knee joint was present in two patients. Both of them underwent thrombolysis which failed to restore a distal run off in one patient, who subsequently required an above-knee amputation. In the other patient thrombolysis restored a narrowed popliteal artery ending on a good quality peroneal artery. He underwent a successful superficial femoral to peroneal artery bypass graft with a reversed, orthoanatomic saphenous vein, which remains patent at 23 months (Figure 1).

The remaining two patients presented with a partially thrombosed popliteal artery continuing respectively with a peroneal and posterior tibial artery in one case and the anterior tibial artery in the other. In the first one a reversed saphenous vein was grafted from the above knee to the below knee popliteal artery.

In the second one aneurysm was excluded and a reversed saphenous vein was grafted from the superficial femoral to the anterior tibial artery htrough the interosseous membrane. Both grafts are patent, respectively at 44 and 37 months.

In conclusion, cririal ischemia associated with aneurysms of the popliteal artery is a challengig sondition for treatment directed to limb salvage. Arteriography is mandatory for planning appropriate treatment. Whenever the aneurysm is trhombosed and distal run off is not clearly opacified, intraarterial thrombolysis is the preliminary treatment of choice, in order to identify a possible, good quality receiving axis for bypass grafting. Whenever the distal popliteal artery appears unsuitable for grafting, revascularization should be directed without hesitating to the best patent distal axis.

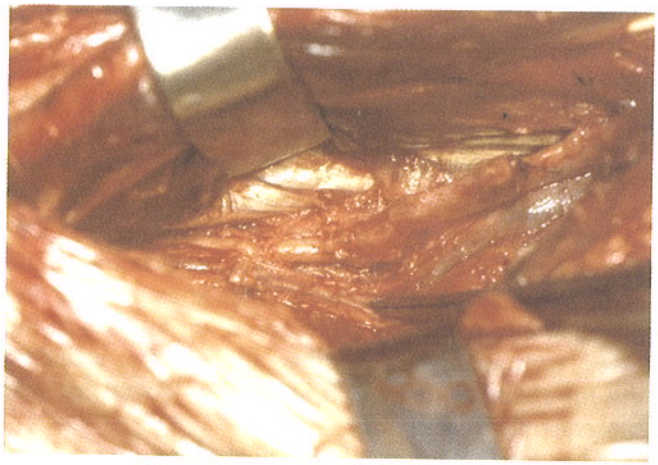

FIGURE 1
Intraoperative view: detail of distal anastomosis in a superficial femoral to peroneal artery saphenous by-pass for thrombosed popliteal aneurysm.

REFERENCES

(1) ANTON GE, HERTZER NR, BEVEN EG, O'HARA PJ, KRAJEWSKI LP. *Surgical management of popliteal aneurysms: trends in presentation, treatment, and results from 1952 – 1984.* J Vasc Surg 1986; 3: 125-34.

(2) VERMILION DB, KIMMINS SA, PACE WG, EVANS WE. *A review of one hundred forty-seven popliteal aneurysms with lomg term follow-up.* Surgery 1981; 90: 1009-14.

(3) WHITEHOUSE WM, JR, WAKEFIELD TW, GRAHAM LM, ET AL. *Limb-threatening potential of arteriosclerotic popliteal artery aneurysms.* Surgery 1983; 93: 694-9.

(4) DAWSON I, VAN BOCKEL JH, BRAND R, TERPSTRA JL. *Popliteal artery aneurysms: long-term follow-up of aneurysmal disease and results of surgical treatment.* J Vasc Surg 1991; 13: 398-407.

(5) GRAHAM LM. *Femoral and popliteal aneurysms.* In: Rutherford RB (Ed). "Vascular Surgery" 5th edition, WB Saunders, Philadelphia, 2000; 1345-560.

(6) DORMANDY J. *European working group on critical chronic ischemia. Second european consensus document on chronic critical leg ischemia.* Circulation 1991; 84: 1-26.

(7) RUTHERFORD RB, BAKER JD, ERNST C, ET AL. *Recommended standards for reports dealing with lower extremity ischemia: revised version.* J Vasc Surg 1997; 26: 517-38.

(8) BUDA JA, WEBER CJ, MCALLISTER FF, VOORHEES AB. *The results of treatment of popliteal artery aneurysms: a follow-up study of 86 aneurysms.* J Cardiovasc Surg 1974; 15: 615-9.

(9) MARTY B, WICKY S, RIS HB, ET AL. *Success of thrombolysis as predictor of outcome in acute thrombosis of popliteal aneurysms.* J Vasc Surg 2002; 35: 487-93.

(10) FARINA C, CAVALLARO A, SCHULTZ RD, FELDHAUS RJ, DI MARZO L. *Popliteal aneurysms.* Surg Gynecol Obstet 1989; 169: 7-13.

(11) WYCHULIS AR, SPITTELL JA, WALLACE RB. *Popliteal aneurysms.* Surgery 1970; 68: 942-52.

(12) BOUHOUTSOS J, MARTIN P. *Popliteal aneurysms: a review of 116 cases.* BR J Surg 1974; 61: 469-75.

(13) REILLY MK, ABBOTT WM, DARLING RC. *Aggressive surgical management of of popliteal artery aneurysms.* AM J Surg 1983; 145: 498-502.

(14) ROGGO A, BRUNNER U, OTTINGER LW, LARGIADER F. *The continuing challenge of aneurysms of the popliteal artery.* Surg Gynecol Obstet 1993; 177: 565-72.

(15) VARGA ZA, LOCKE-EDMUNDS JC, BAIRD RN. *A multicenter study of popliteal aneurysms. Joint vascular research group.* J VASC SURG 1994; 20: 171-7.

(16) LOWELL RC, GLOVICZKI P, HALLETT JW, JR, ET AL. *Popliteal artery aneurysms: the risk of nonoperative management.* Ann Vasc Surg 1994; 8: 14-23.

(17) DAVIDOVIC LB, LOTINA SI, KOSTIC DM, ET AL. *Popliteal artery aneurysms.* World J Surg 1998; 22: 812-7.

(18) STEINMETZ E, BOUCHOT O, FAROY F, ET AL. *Preoperative intraarterial thrombolysis before surgical revascularization for popliteal artery aneurysm with acute ischemia.* Ann Vasc Surg 2000; 14: 360-4.

(19) INAHARA T., TOLEDO AC. *Complications and treatment of popliteal aneurysms.* Surgery 1978; 84: 775-83.

(20) SZILAGYI DE, SCHWARTZ RL, REDDY DJ. *Popliteal arterial aneurysms. Their natural history and management.* Arch Surg 1981; 116: 724-8.

Popliteal Vascular Entrapment And Critical Limb Ischemia

Luca di Marzo, Paolo Sapienza, Antonino Cavallaro

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 Surgical Treatment of PAE was reported in 1959, in a 12- year-old boy complaining claudication after walking 300 meters. At surgical exploration, Dr. Hamming, at the 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 thromboendoarterectomy (2).

Many case reports have been published in the following years but the majority of the series comprised a small number of patients (3,4) and collecting series were uncompleted for a lack of details and patient follow-up (5). In 1979 we treated our first case of arterial entrapment in a 15-year old girl. The popliteal artery was compressed by the soleus muscle, causing intermittent claudication. Surgical resection of the soleus muscle relieved symptoms. The patient returned to a normal life, married and had a child after 7 years. She is still in good condition and does not complain symptoms related to her syndrome. 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. Great effort was addressed to collect different series with comparable criteria. The criteria established by the Society for Vascular Surgery were reviewed and accepted with enthusiasm and minor changes. Common opinion was to consider both the arterial and venous entrapment as a common disease defined vascular entrapment. A large discussion was made on the functional form of entrapment. This was first described by Rignault et al. (6) in 1985 and describes cases in which the anatomy of the popliteal fossa is normal. Symptoms are usually caused by hypertrophy of the muscles determining a compartment syndrome.

Functional entrapment was included in the classification as type F (Table I).

Embryology and anatomy

The arterial system of the leg develops from two embryonic arteries, both of which take 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 give 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 leg, 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 is divided in 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 obliterates. The adult popliteal artery is therefore the product

TABLE I
Classification of compressing structures causing PAE

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 and medial head of gastrocnemius.
Type V:	Primary venous.
Type VI:	Variants.
Type F:	Functional.

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of fusion of several embryonic elements. Both heads of the gastrocnemius muscle arise from the femoral epiphysis. As the infant develops, the origins of this muscle migrate cranially across the epiphysial plate on to the femoral metaphysis. As a results 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 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 error 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 sleeps 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 can not explain the final situation: often in fact the abnormalities are complex thus not permitting a complete embryologic explanation (7). The popliteal vein is involved more rarely because its embryologic development happens later as compared to the artery. The recently proposed modification of the classification tried to cover the most frequent embryologic anomalies and to classify the most rare 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 out of 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 formerly 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. The latter differs from the data of the literature in which up-to-now a ratio of 10:1 has been reported. This is probably due to the fact that we are a referring center for PAE and we see and operated on for 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 having a bilateral PAE. Two limbs were left untreated because they were asymptomatic and were followed-up. One patient was surgically explored but vascular reconstruction was felt at risk of early thrombosis because of the poor run-off status. The patient is still claudicant 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: 1-early and 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 meters) 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 the absence of them 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 between intermittent claudication to rest pain and necrosis. Because of the richness of the collateral circulation at 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 supra-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 aneurismal 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 and aneurysm complication. It is well known that complication occurs in 18% to 31% of all patients with popliteal aneurysm who did not undergo operation and that the incidence of limb loss increases dramatically once complications of the aneurysm have occurred, even with operative intervention (14). Overall in our experience we observed 26 (45.6%) limbs having symptoms of class 1, 20 (35.1%)

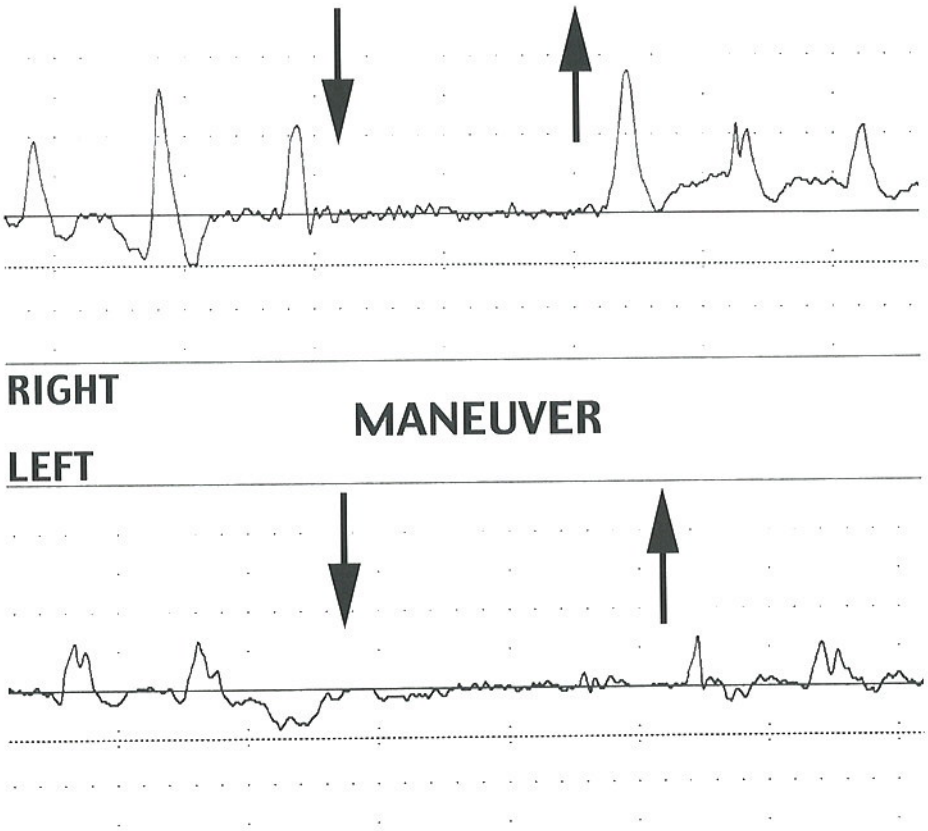

FIGURE 1
Doppler CW during maneuvers.

of class 2, 8 (14.1%) of class 3 and 1 (1.7%) of class 5. Two (3.5%) limbs were asymptomatic and none were professional athletes, but 7 (20%) patients participated in sports such as soccer, volleyball, and horseback riding. The occurrence of moderate to severe symptoms accounts for less than 16% of all patients affected with PAE, 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 presenting with signs and symptoms of pain, paresthesia, cold feet and calf claudication after intensive physical training to avoid more dramatic clinical presenting symptoms such as aneurysmal changes and arterial atherosclerotic changes. An other important aspect we should stress to better understand PAE, are the differences between the anatomic popliteal vascular entrapment and the functional form. Several Authors describe the two forms of entrapment as the same entities. 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 musculotendineous structures. Others (15,16) described a compartment syndrome resembling a popliteal entrapment in well-conditioned athletes. Athletes can, in fact, complain of 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 a 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 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 (Figure 1). 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 (Figure 2). 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, there may be false-positive results.

The noninvasive techniques have at times been found to lack specificity. To minimize their false-positive rate, computed tomography and magnetic resonance imaging have been used to confirm PAE, simultaneously

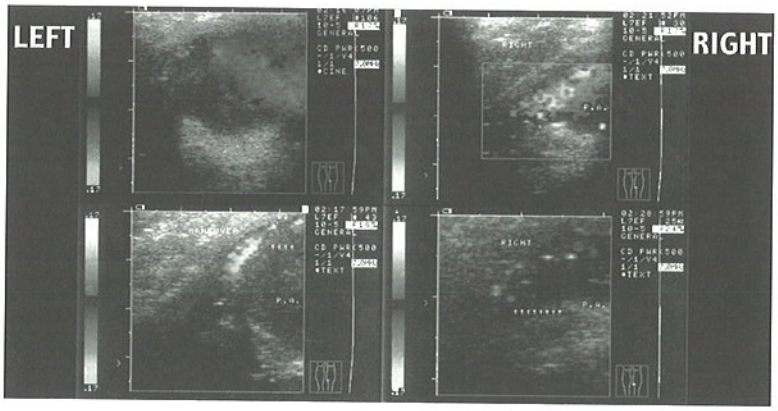

FIGURE 2

Color Doppler at rest (above) and during maneuvers (below). Patient with bilateral popliteal aneurysm caused by PAE (same patient).

revealing detailed anatomic information of the popliteal fossa. The increasing specificity and sensibility of computed tomography and magnetic resonance in studying the popliteal fossa could represent a valid alternative to the more invasive digital angiography. At present we still continue to study the PAE with angiography during maneuvers but we are performing at the same time 3D computed tomography and magnetic resonance angiography. However, at present a clear and precise preoperative characterization of the compressing structure is not always possible and the visualization of the distal run-off in the cases of advanced disease is underestimated. Therefore, we still recommend digital angiography with maneuvers in all cases in whom the occurrence of atherosclerotic changes at the popliteal artery and its distal branches are present.

This diagnostic tool permits a better planning of the reconstructive operation (Figure 3).

Treatment

Management of the PAE syndrome is surgical, an exception is made in cases diagnosed very late with an occluded popliteal artery without aneurismal changes in which collateral circulation is considered satisfactory for the patient. 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 a progressive degeneration of the popliteal artery. However, time can not be considered the only factor causing stable popliteal degeneration. We treated in fact 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 can not 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.

Reconstructions of the popliteal artery should be limited in length to the impaired arterial segment, avoiding anastomoses to the tibial vessels whenever it is possible. The presence of aneurysmal changes does not modify the surgical tactic. We always approach the popliteal artery through a Z-shaped posterior incision.

This is useful when more musculotendinous structures are involved. The medial incision is more anatomic but does not allow complete exposure of the popliteal fossa and the identification of the compressing structures may be difficult. Autogenous veins should be preferred to prosthetic materials. In our experience we never dealt with the typical complications of critical limb ischemia caused by embolizing

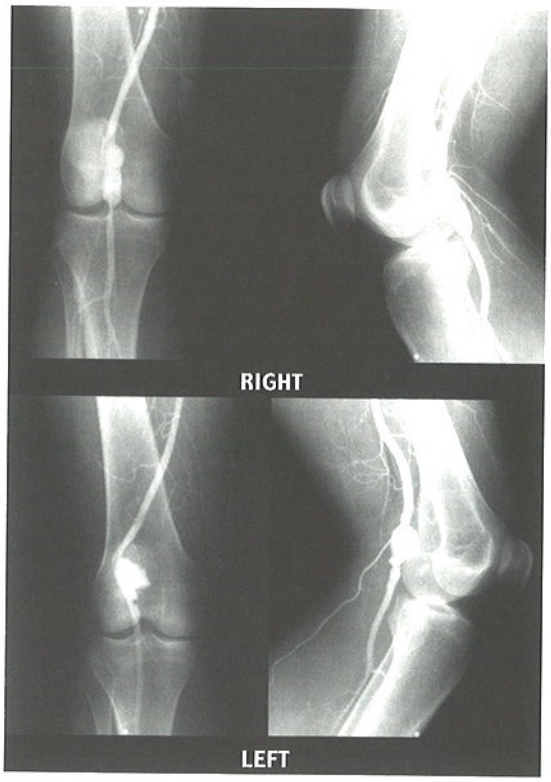

FIGURE 3

Arteriography demonstrating a bilateral popliteal aneurysm caused by PAE (same patient)

aneurysms in patients affected with PAE but in 2 (3.5%) limbs we observed a well compensated peripheral circulation after popliteal aneurysm embolization. In the presence of critical limb ischemia caused by distal embolization we recommend the use of thrombolytic agents (streptokinase, urokinase or tissue plasminogen activator) if the episode lasts less than 24 hours before the attempt at surgical resection of the aneurysm. However, this therapy could create in a small percentage of the cases serious complications. Reported rates of stroke and major hemorrhage during thrombolytic therapy are 1 and 5%, respectively (18) and acute deterioration of limb during intrarterial thrombolysis, possibly secondary to thrombus destabilization, may also occur. In the other case, an emergent operation to improve the run-off should be performed. Experiences (19) with thrombosed popliteal aneurysms and poor vessels run-off status have clearly demonstrated in these cases a significantly higher risk of limb amputation. The best results in popliteal artery management are in fact achieved by elective repair of patent aneurysms with good run-off vessels.

REFERENCES

(1) STUART PTA. *Note on a variation in the course of the popliteal artery.* J Anat Physiol 1879; 13: 162.

(2) HAMMING JJ. *Intermittent claudication at an early age due to anomalous course of the popliteal artery.* Angiology 1959; 10: 369-70.

(3) RICH NM, COLLINS GJ, MCDONALD PT, KOZLOFF L, CLAGETT PG, COLLINS JT. *Popliteal vascular entrapment. Its increasing interest.* Arch Surg 1979; 114: 1377-84.

(4) COLLINS PS, MCDONALD PT, LINC RC. *Popliteal artery entrapment an evolving syndrome.* J Vasc Surg 1989; 10: 484-90.

(5) DI MARZO L, CAVALLARO A, SCIACCA V, MINGOLI A, STIPA S. *Natural history of entrapment of the popliteal artery.* J Am Coll Surg 1994; 178: 553-6.

(6) RIGNAULT DP, PAILLER JL, LUNEL F. *The functional popliteal entrapment syndrome.* Int Angiol 1985; 4:341-3.

(7) CAVALLARO A, DI MARZO L, GALLO P, CISTERNINO S, MINGOLI A. *Popliteal artery entrapment. Analysis of the literature and report of personal experience.* Vasc Surg 1986; 20: 404-23.

(8) GIBSON MH, MILLS JG, JOHNSON GE, DOWNS AR. *Popliteal entrapment syndrome.* Ann Surg 1977; 185: 341-8.

(9) BOUHOUTSOS J, DASKALAKIS E. *Muscular abnormalities affecting the popliteal vessels.* Br J Surg 1981; 68: 501-6.

(10) DI MARZO L, CAVALLARO A, SCIACCA V, LEPIDI S, MARMORALE A, TAMBURELLI A, STIPA S. *Diagnosis of popliteal artery entrapment syndrome: the role of duplex scanning.* J Vasc Surg 1991; 13: 434-8.

(11) LEVIEN L, VELLER MG. *Popliteal artery entrapment syndrome: more common than previously recognized.* J Vasc Surg 1999; 30: 587-598

(12) DI MARZO L, CAVALLARO A, MINGOLI A, SAPIENZA P, TEDESCO M, STIPA S. *Popliteal artery entrapment syndrome: the role of early diagnosis and treatment.* Surgery 1997; 122: 26-31.

(13) RUTHERFORD RB, BAKER JD, ERNST C, JOHNSTON KW, PORTER JM, AHN S, JONES DN. *Recommended standards for reports dealing with lower extremity ischemia: revised version.* J Vasc Surg 1997; 26: 517-38.

(14) SHORTELL CK, DEWEESE JA, OURIEL K, GREEN RM. *Popliteal artery aneurysms: a 25-year surgical experience.* J Vasc Surg 1991; 14: 771-9.

(15) TURNIPSEED WD, POZNIAK M. *Popliteal entrapment as a result of neurovascular compression by the soleus and plantaris muscles.* J Vasc Surg 1992; 15: 285-94.

(16) ERDOES LS, DEVINE JJ, BERNHARD VM, BAKER MR, BERMAN SS, HUNTER GC. *Popliteal vascular compression in a normal population.* J Vasc Surg 1994; 20: 978-86.

(17) AKKERSDIJK WL, DE RUYTR JW, LAPHAM R, MALI W, EIKELBOOM BC. *Colour duplex ultrasonographic imaging and provocation of popliteal artery compression.* Eur J Vasc Endovasc Surg 1995; 10: 542-5.

(18) BERRIDGE DC, MAKIN GS, HOPKINSON BR. *Local low dose intrarterial thrombolytic therapy: the risk of stroke and major hemorrhage.* Br J Surg 1989; 76: 1230-3.

(19) CARPENTER JP, BARKER CF, ROBERTS B, BERKOWITZ HD, LUSK EJ, PERLOFF LJ. *Popliteal artery aneurysms: current management and outcome.* J Vasc Surg 1994; 19: 65-73.