Popliteal Artery Entrapment

6

Antonino Cavallaro

Popliteal artery entrapment is recognized as a rare but not exceptional causative factor of aneurysm. Gaylis [1] found that 4 out 53 (7.5%) popliteal aneurysms were due to entrapment; in the series of popliteal aneurysms reported by Batt et al. [2], the incidence was 2.6% (3/116); when the aneurysm was bilateral, the incidence of entrapment cases was, respectively, 8.3% (1/12) and 2.3% (1/43).

Stuart [3] is quoted as the first to describe muscular compression on the popliteal artery (Fig. 6.1): during the dissection of a limb amputated for gangrene consequent to thrombosis of a popliteal aneurysm, he observed the currently universally known anomaly in the course of the popliteal artery, looping around the inner margin of the medial gastrocnemius, and as well a variant in the site of proximal insertion of that muscle.

An anomaly similar to that described by Stuart had been reported in 1875 by Gruber [5] (Fig. 6.2): in his case, the popliteal artery presented a segmental chronic thrombosis supplied by a marked enlargement of the arteries of gastrocnemius.

Variants in the interrelationships between the neurovascular bundle, and, namely, the popliteal artery, and the neighboring musculotendinous

NOTE ON A VARIATION IN THE COURSE OF THE POPLITEAL ARTERY.—By T. P. Anderson Stuart, *Student of Medicine, University of Edinburgh.*

In May of last year, I was requested by Professor Spence to make for him a preparation of the popliteal space of the limb of a man aged 64, who had had to submit to amputation on account of gangrene of the foot, resulting from a very large popliteal aneurism. As the dissection proceeded a most striking abnormality in the course of the artery came to light, and, so far as I have been able to ascertain, it is now put on record for the first time.

The popliteal artery, after passing through the opening in the adductor magnus, instead of, as it usually does, coursing downwards and outwards towards the middle of the popliteal space, so as to lie between the two heads of the gastrocnemius muscle, passes almost vertically downwards internally to the inner head of the gastrocnemius. It reaches the bottom of the space by turning round the inner border of that head, and then passes downwards and outwards beneath it—between it and the lower end of the shaft of the femur. The inner head of the gastrocnemius arises much higher up than usual, namely, from the inner division of the linea aspera about an inch and a half above the condyle, thus leaving a considerable space between it and the condyle, over which space the artery passes. The other structures are normal. The preparation is now in the possession of Mr Spence.

Fig. 6.1 The short note published in 1879 by T. P. Anderson Stuart [3] (1856–1920) when he was a medical student in Edinburgh. He ended his career as the first professor and later the chairman of the board of the Medical School in Sidney [4]

structures were the object of several observations and reports by anatomists. Labatt [6] described in 1837 a double-headed medial gastrocnemius: vessels and nerve coursed, evidently compressed, between the two heads. In 1844, Quain [7] reported three observations about an unusual band of muscular fibers in close connection of the artery in the lower part of the popliteal space coursing down from a line above the femoral condyles to the gastrocnemius muscle: it

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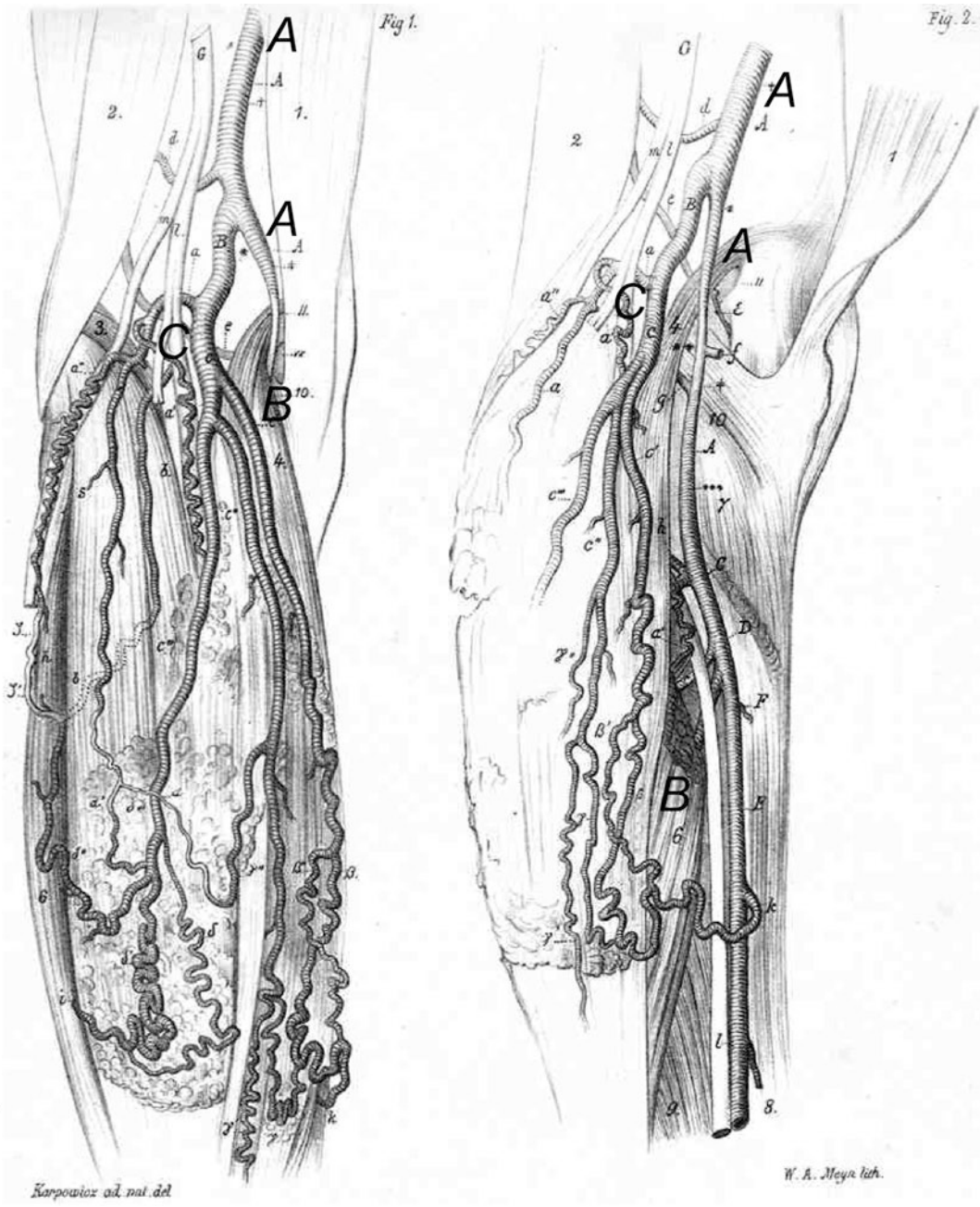

Fig. 6.2 The anomalous course of the popliteal artery found by Gruber [5]. (A) Popliteal artery. (B) Medial gastrocnemius muscle. (C) Arteria gastrocnemialis communis

separated the nerve from the vessels or the artery from the vein and was defined as a supplementary or third head of the gastrocnemius. After more than a century, Iwai et al. [8] stressed the role of the third head of gastrocnemius in the mechanism of entrapment of the popliteal vessels and described the "gastrocnemius tertius" as a muscle arising from the posterior and inferior surface of the femur and joining the gastrocnemius, more frequently the medial head; in

Japanese, the frequency of gastrocnemius tertius would be higher than generally reported [9] (5.5% vs about 3%). The different types of gastrocnemius tertius had been thoroughly described in 1884 by Testut [10], who put into evidence that this unusual muscle could arise also from flexor muscles (semimembranosus or biceps femoris) or by the greater abductor. This was confirmed by Le Double [11], and, later, Frey [12] registered the variants (at least 12) of gastrocnemius tertius. In the last decades of the nineteenth century, several observations of an accessory, lateral, head of the medial gastrocnemius were reported [13–15]: the artery coursed between the principal and the accessory head [16] (Fig. 6.3), sometimes traversing the muscular substance.

Parona [17], in 1903, reported the anomalous cranial insertion of the medial gastrocnemius, more lateral than usual, onto the metaphysis: the muscle separated the artery from the vein. In 1938, Suzuki [18] described the compression of the popliteal artery by the tendon of the semimembranosus muscle.

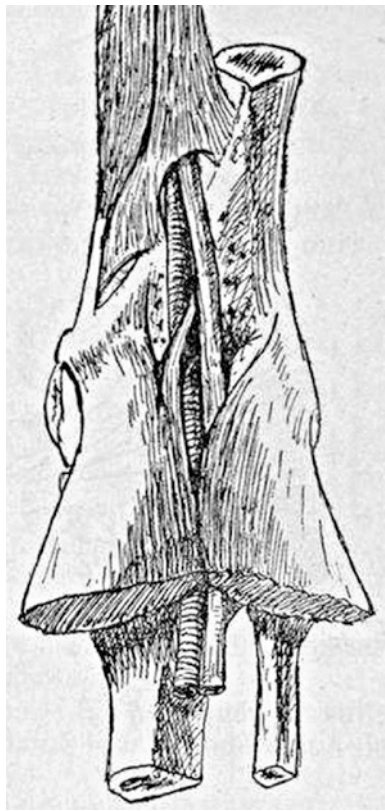

Fig. 6.3 Accessory head of the medial gastrocnemius as illustrated by Bouglé [16]

Popliteal artery entrapment entered the clinical stage in Leyden, with Hamming [19], who, in 1959, reported the treatment of a 12-year-old boy complaining of claudication. The young age of the patient and the peculiarity of the anatomical situation found at operation excited interest and attention, and by 1964 four additional cases had been reported [20–23]: of these, two were bilateral [22, 23], and a post-stenotic aneurysm was observed in two limbs [21, 22]. In 1965, the expression "popliteal artery entrapment syndrome" was proposed by Love and Whelan [24], being rapidly and almost universally accepted, even if French authors [25] continued to use preferentially the expression "syndrome de l'artère poplitée piégée." Literature reviews registered eight cases in 1970 [26] and 14 cases in 1971 [27]: the latter resulted in complication by aneurysm (one case) and post-stenotic dilation (four cases). From 1959 through 1975, clinical cases amounted to 65 [28]. In 1979, Rich et al. [29] pointed to the increasing interest of this probably underestimated pathology, textually asserting "specific consideration for the diagnosis of popliteal vascular entrapment should be given to those patients in the younger athletic male population in whom intermittent claudication with exercise develops, to all younger male and female patients in whom thrombosis of the popliteal artery develops without trauma, and to all middle-aged patients in whom popliteal aneurysm develops." The clinical field in which high suspicion for popliteal entrapment is demanded would be enlarged in the following years, including military recruits and an increasing number of female patients, and in 1999 Levien and Veller [30] again suggested that popliteal entrapment had been probably underestimated up to then.

The wide spectrum of anatomical variants found to be responsible for popliteal artery flow impairment gave origin to several attempts to classification [31–35], but none looked entirely satisfactory, due to the continuing reports on new types of compression and the relevant number of

variants (some of which really rare or unique) sometimes variously interlacing reciprocally. The concomitant involvement of the popliteal vein was observed since the early reports [36–38], and occasionally a "pure" venous entrapment was observed [39]. On a whole, vein entrapment is present in about 30% of the cases [8, 40]. In a comprehensive review [28] of 172 patients (210 limbs) treated surgically, 17 anatomical variants were identified: the medial gastrocnemius, alone or in combination with other structures, was involved in over 80% of the cases, including Stuart's original variant (21% of all cases) and lateral attachment onto the femoral metaphysis (31% of all cases). In 1998, in Rome, the **Popliteal Vascular Entrapment Forum** was established [41], headed by Norman M. Rich, and, accepting with minor changes what stated by the Society for Vascular Surgery, a classification was proposed (Fig. 6.4), based on the following:

- Popliteal arterial and venous entrapment represent a common disease defined as popliteal vascular entrapment.
- Anomalies are complex, and in several instances a single embryologic variant cannot fully explain the situation.

Seven main types of popliteal vascular entrapment were identified:

- Type I—the anatomical position of the medial head of gastrocnemius is normal or almost normal (insertion onto the superior and posterior surface of the medial condyle); **the popliteal artery loops around the inner margin of the medial gastrocnemius and courses beneath the muscle to reach its normal place in the lower part of the popliteal space.**
- Type II—**the medial head of the gastrocnemius is laterally attached**, onto the femoral metaphysis.
- Type III—**accessory slip of the medial gastrocnemius**. This accessory bundle may be tendinous, muscular, or both. If the muscular component is particularly developed, the artery looks as passing within the muscle [34, 36, 42–45].

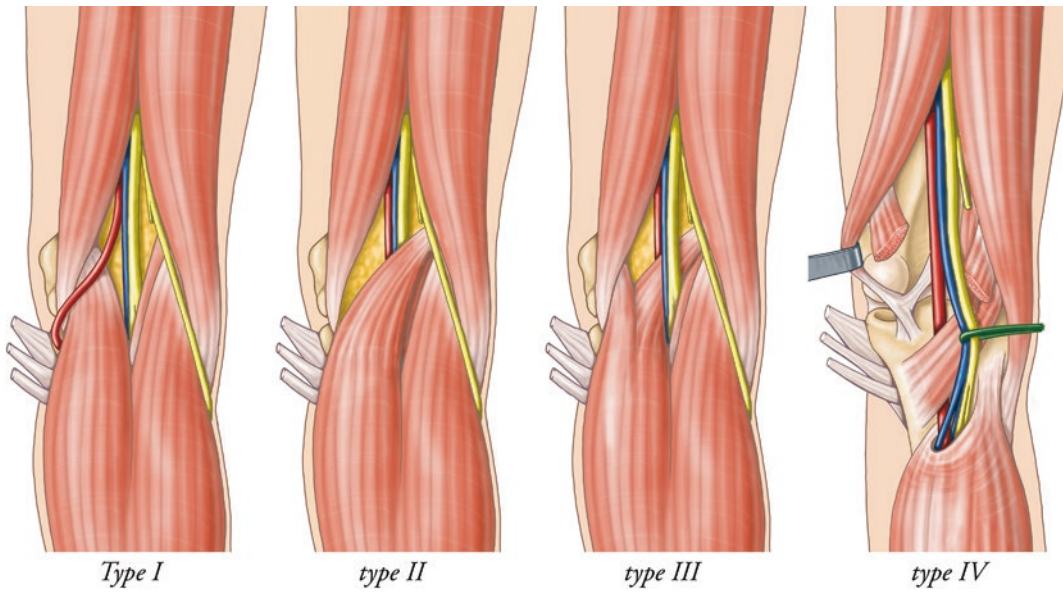

Fig. 6.4 The four more frequent types of entrapment (see text). In type II, an example is given of the concomitant compression over the artery, the vein, and the nerve

- Type IV—**the popliteal artery passes ventrally to the popliteus muscle**. Entrapment from fibrous band has been sometimes assimilated to type IV [46–48]. Type IV is considered the only variant in which the nerve too may not be involved [49].
- Type V**—vein entrapment, primary or associated with artery entrapment**. The rarity of vein entrapment may be due to the fact that deep limb veins, which are accompanying veins to the arteries, are the last to develop, being unavailable for entrapment in the embryological stage in which it may occur [50].
- Type VI—**variants**. It includes complex anomalies and rare anatomical derangements. Ezaki et al. [51] found compression by an aberrant tendinous band of the greater adductor muscle. This had been already recognized by Maistre [52] as a frequent and important component of popliteal entrapment. Compression by the plantaris muscle was identified occasionally [53] but with impressive frequency by Bouhoutsos and Daskalakis [35]. Other anatomical variants responsible for compression upon the popliteal vessels are accessory tendon of the semitendinosus muscle [33, 54], fused head of gastrocnemius [55], and medial accessory tendon of the lateral gastrocnemius [56]. Entrapment from branches of the tibial nerve has been described [29]. Reviewing the operative reports, one is brought to think that a certain overlapping of plantaris muscle, gastrocnemius tertius, and accessory bundle of medial gastrocnemius is possible in defining the anatomy and embryology of the compressing structure.
- Type VII or F—**functional** entrapment.

Levien and Veller [30] synthesized the embryological causes of the more frequent types. Persistence of the primitive popliteal artery explains type IV. Under normal circumstances, the cranial migration of the medial gastrocnemius occurs before the development of the definitive popliteal artery. Delay in the migration of medial gastrocnemius or early formation of the latter may give origin to type I (the muscle catches the artery which is swept medially) or to type II (the artery partially arrests the final migration of the muscle) or to type III (the artery develops within the migrating muscle). Therefore types I–III would represent the result of a unique embryologic derangement, which may variously produce either the aberrant path of the popliteal vessels [57] or an abnormal attachment of medial gastrocnemius [31] over the normally positioned vessels.

Popliteal artery entrapment is diagnosed predominantly in young adults, being 47% of patients aged 21–40 and 28% aged 20 or less [58]; extremes are at 7 years [59] and 65 years [60]. Males outnumber females 9:1 according to Andre et al. [61]; in a review of 375 cases reported from 1959 through 1990 [62], a 7.5:1 ratio was found. The number of female patients is steadily increasing (in our personal experience[1] the M/F ratio is 3.5:1): currently, popliteal artery entrapment should be suspected in any young patient, regardless of sex, with symptoms of pain, paresthesia, cold foot after intensive physical activity, and as well if complaining of claudication mainly when walking upgrade or ascending stairs.

Bilateral involvement was already reported in the early clinical experiences [22, 23, 53] and accounts for about 38% of all cases. Occurrence of the syndrome in consanguineous has been repeatedly observed [63–66] and once in monozygotic twins [67].

True incidence of popliteal entrapment is still obscure. Gibson et al. [57] found three cases in a series of 86 dissections (3.8%); the incidence was of 3.3% in the postmortem study published by Paulo [68]. O'Donnell [69] registered a similar incidence (4.3%) in 92 cadaver studies. Going to the clinical setting, Bouhoutsos and Daskalakis [35] treated 33 patients in a population of about 20,000 young subjects (0.16%) purposedly screened; di Marzo et al. [70] observed the same percentage in a smaller series (2/1212; 0.16%).

[1]Personal experience has been gained at the Dept. of Surgery "P. Valdoni," University "La Sapienza," Rome, and consists of 40 patients (62 limbs) treated from 1979 through 2009.

It looks convenient to consider two stages of popliteal artery entrapment [71]: early and advanced. This relies on the pathology of the involved artery but mainly corresponds with the type of treatment required. In the early stage, the artery does not present any stable alteration, at least macroscopically; cure requires simple freeing from the compressing structure. In the advanced stages, significant stenosing lesions or thrombosis or dilation/aneurysm is present, and an associated reconstructive surgery is required, if possible; moreover, distal branches may be seriously damaged by acute or chronic embolism arising from the popliteal artery lesion.

Lesions of the arterial wall have been studied in several specimens [72–75] and derive from the chronically repeated compression by a musculotendinous structure against an osseous plane; on the muscular side, they are *stretch* lesions, consisting of the appearance of a longitudinal smooth muscle bundle as observed in the bronchial artery as a consequence of hypertension [76]; on the osseous side they are *crush* lesions, with atrophy of the smooth muscle cells and of the internal elastic membrane, thickening of the intima, and eventually organized thrombus. Naylor et al. [77], studying five specimens of occluded and resected artery, were able to define three stages of histopathologic lesions, starting with neovascularization of the adventitia and proceeding with fibrosis of the media and final fragmentation of the internal elastic lamina, fibrointimal proliferation, and superimposed thrombosis. It has been suggested [29, 47] that type IV entrapment (from popliteal muscle or fibrous transverse band) may be the most aggressive for artery disease; this could be true, as bilateral aneurysm was observed in a very young patient [46]; moreover Levien and Veller [30] reported that severe ischemia affected more than 60% of type IV cases (5/8), while its incidence was less than 30% in the whole series of 58 cases of entrapment and that the eight aneurysms observed were in type I and type IV; however anatomic lesions of the popliteal artery have been found in many musculotendinous variants.

Midpopliteal artery thrombosis affected 3 out 12 patients reported by Rich et al. [29]; emphasis on its significance and the required type of surgical approach was made by several authors [78, 79]. Post-stenotic popliteal aneurysm, first observed by Servello [21] in Padua, has been repeatedly reported [22, 25, 35, 44, 45, 48, 80], sometimes bilaterally [28, 46]. In our experience, aneurysm was present in 17% of patients (13% of limbs); in different series or reviews, the incidence of aneurysm ranged from 7 to 21% [27, 29, 81], while a simple post-stenotic dilation was observed in about 30% of the cases [27, 74]. Occasionally, the aneurysm was the first sign of entrapment [82]. As a consequence of the popliteal artery lesion, distal flow may be impaired by emboli; these may obviously arise from an aneurysm [29, 35, 83, 84] but also from mural thrombus of normal size artery [57, 85, 86]. It is a matter of fact that, especially in older patients, involvement of the tibial vessels may be observed in absence of signs of atherosclerosis: this can endanger or preclude the required reconstructive procedure.

From the clinical point of view, early stage is diagnosed in younger patients with mild symptoms after intensive physical training or claudication >100 m, being rare heavy claudication (<100 m) or chronic ischemia; in the advanced stage, symptoms are frequently invalidating (Table 6.1). This grossly corresponds with

Table 6.1 Popliteal artery entrapment: distribution of age, symptoms, and angiographic findings according to the stage of the disease

	Early stage	Advanced stage	Fisher's exact test
Age <25 years	86/129 (68%)	87/216 (40%)	$p < 0.001$
Mild symptoms	30/73 (41%)	25/193 (13%)	$p < 0.001$
Claudic. >100 m	37/73 (51%)	87/193 (45%)	N.S.
Invalidating symptoms	6/73 (8%)	81/193 (42%)	$p < 0.001$
A = normal at rest	63/105 (60%)	7/180 (4%)	$p < 0,001$
A = stenosis at rest	42/105 (40%)	45/180 (25%)	N.S.
A = occlusion, aneurysm	0/105	128/180 (71%)	$p < 0,001$

From di Marzo et al. [62], modified

angiographic findings; however, symptoms may depend also on the effectiveness of the knee collateral network and on the eventual impairment of the distal arterial tree caused by embolism or, in older patients, by concomitant and independent chronic arterial disease. Acute ischemia has been scantily reported, affecting also the youngest patient in the world literature [59]. Popliteal artery thrombosis may be responsible for sudden onset of symptoms, but generally an acceptable compensation occurs avoiding a really limb-threatening condition.

Considering that popliteal vascular entrapment is a congenital lesion, it is not easily explainable why symptoms become evident in so different ages of life. Type of compression, muscular development, and lifestyle look as important factors. Certainly muscular activity and especially abrupt changes of it play a significant role: this explains the common finding of young people becoming symptomatic after beginning military service. On the other hand, one wanders how many bearers of the consequences of this embryologic variant remain permanently asymptomatic.

Diagnosis of popliteal artery entrapment was made during surgical exploration in the first two cases [19, 20]. In 1962, Servello [21] suspected, on the basis of arteriography, the anomalous position of the popliteal artery and observed an abrupt decrease in the amplitude of the oscillometric pulse wave during forced plantar flexion of the foot. Hamming and Vink [36] tried to standardize the maneuvers apt to procure reduction in pedal pulses in case of entrapment: forced plantar flexion (active contraction of gastrocnemius) and forced dorsal flexion (stretching of gastrocnemius). These maneuvers became routine components of the diagnostic iter when popliteal artery entrapment was suspected, from the digital appreciation of pulses to noninvasive and imaging examinations. Foot hyperextension (or plantar flexion) is an active maneuver, possibly against a stop represented by the examiner's hand or [87] a folded broadsheet wrapped under the plantar surface of the foot (the subject grasping firmly both ends of the looped sheet, thus providing counteractive resistance). Forced dorsal flexion is generally a passive maneuver. In any case, it is important that the knee is fully extended: this happens almost automatically with active maneuver but should be assured passively in passive maneuver. In this case, pressure exerted upon the knee of the supine subject should not be excessive to avoid false results in case of recurvated knee. The importance of full knee extension derives primarily by clinical observations: ability to run with semi-flexed knee but problems in walking [23], sleeping with the knee bent because straightening of the knee joint causes numbness and tingling of the foot [44], and difficulty in walking upgrade [88]. During the initial clinical examination, it is important to reproduce the type of muscular activity that, according to the patient, first evoked symptoms. It is not unusual, in case of popliteal thrombosis, to observe the sign of "hot knee" [89], but this is quite aspecific, indicating only the excellent development of knee collateral network.

CW Doppler evaluation of ankle systolic pressure [90] was initially considered highly reliable [91], and details for optimally performing the examination have been described [70], but with increasing experience, the number of false positive became impressive. Several observations [92, 93] demonstrated that forceful plantar flexion may compress and laterally displace the artery in up to 50% of normal individuals. In 1974, Darling et al. [83] tried to correlate noninvasive testing (pulse volume recorder) with angiographic findings in the diagnosis of popliteal artery entrapment, but their initial enthusiasm successively faded [94] because, while positivity was obtained in all entrapment cases, false-positive results were observed in about 40% of subjects without entrapment.

Duplex scan of the popliteal artery at rest and during maneuvers may be an important diagnosic tool [70, 95, 96]. Di Marzo et al. [70] focused attention on the fact that sample volume must be exactly in the artery after this has been forced deep in the popliteal fossa by the maneuver and that measurements must be repeated three to four times. However, Akkersdijk et al. [97] observed that duplex scan may demonstrate compression or occlusion of the popliteal artery during active

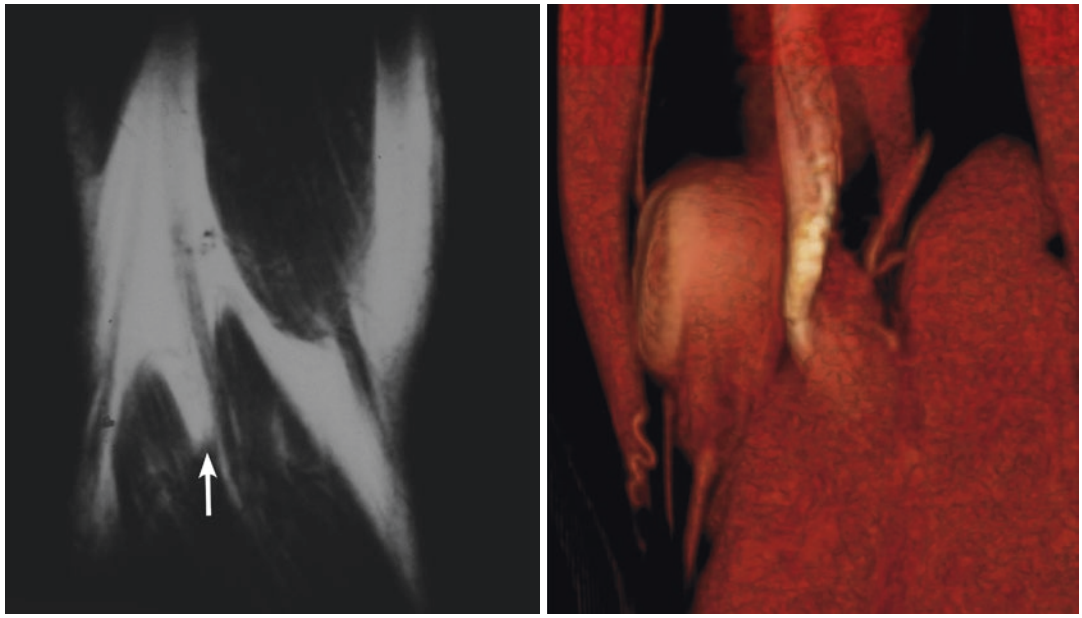

Fig. 6.5 Left: MRI (performed in 1987) demonstrating accessory head of medial gastrocnemius. Right: MR angiography showing the medial gastrocnemius laterally inserted and compressing the popliteal artery. (From di Marzo et al. [69] with permission)

plantar flexion in up to 70% of healthy highly trained subjects.

From the beginning, arteriography has been considered the chief diagnostic tool, showing the anomalous course of the popliteal artery ("deviation angulaire brutale" as defined by Maistre [52]) and its displacement and/or compression/occlusion during forced plantar flexion [98–100]. Stable segmental lesions of the popliteal artery (occlusion at rest, dilation, aneurysm) should suggest entrapment on the basis of clinical and noninvasive findings and lack of other signs of arterial disease. However, Rich et al. [29] warn about the fact that angiographic compression of the popliteal artery cannot be considered a really pathognomonic sign as there is no adequate control study to know how this might happen in normal anatomy. Jeffery et al. [101] reported two emblematic cases in which both Doppler fall in ankle pressure and occlusion at arteriography during active plantar flexion allowed to establish the diagnosis of entrapment: the first case was operated on, but anatomy was normal; the second one was simply followed up with improvement at 18 months. Arteriography continues however to be used [102].

Currently, imaging studies (Fig. 6.5) allow a detailed insight into the anatomy of the popliteal fossa. Both CT [103–106] and MR [107–110] can fully delineate muscle arrangement, and CT and MR angiography [46, 111–115] are highly reliable in defining vascular and muscular anatomy and the reciprocal interrelationships at rest and during stress maneuvers.

Popliteal artery disease caused by entrapment is progressive, evolving toward stenosis, thrombosis, and eventually aneurysm; heavy consequent complication may be the post-embolic damage of the distal arterial branches threatening limb function or survival. Acute limb ischemia is not frequent but not exceptional. Every effort should be made to achieve diagnosis at the early stage to allow timely surgical treatment, which assures excellent and long-lasting results. Early diagnosis depends on the self-attention of patients, who should ask for medical attention at the first symptoms, even if mild and not worrying (and this may happen in children and adolescents) and as well on the awareness of pediatricians, general physicians, and sports physicians about this peculiar disease. Generally they are knowledgeable about popliteal entrapment;

timely involvement of vascular surgeons became progressively more frequent after few years from the initial clinical experiences. In fact, of the 129 cases treated by simple musculotendinous section up to 1990 [62] (when the reported cases totaled 375), only 21 (17%) belong to the period from 1959 through 1980, and 108 (83%) were treated after 1980. However, a series consistent only on advanced cases have been reported also recently [116]. A *perfect* early diagnosis is that made in an asymptomatic limb, as it happens in patient with a symptomatic limb when the contralateral one is purposedly studied on account of the frequent bilaterality of entrapment.

It is a common statement that all cases of entrapment, once diagnosed, should be treated regardless of symptoms. At surgery, the compressing mechanism should be abolished, associating a reconstructive procedure if required. However, if thrombotic occlusion is well compensated, simple musculotendinous section (to avoid continuing mechanical stress upon the arterial wall) has been suggested [117]; as well, vascular reconstruction may look not feasible on account of extensive involvement of the distal arterial tree (as in one of our patients). There is general agreement on the utility of complete section of the compressing structure; complete section of the head of medial gastrocnemius (which is frequently the entrapping muscle) is well tolerated [30]. Controversies exist about the surgical approach, which largely depends on surgeon's experience and preference. Many surgeons (including ourselves) prefer the posterior approach in the prone patient through a lazy S-shaped incision as reported in the first case by Hamming [19]; others, the medial approach classically described for the extensile exposure of the popliteal artery by Gryska et al. [118]. The exposure gained through the posterior approach allows to identify any musculotendinous anomaly and fibrous bands and as well the adequate possibility of managing any segmental lesion of the popliteal artery [29]. Objections, besides the prone position of the patient, are the limited segment of saphenous vein that may be harvested if required and the difficulties in performing extended reconstructions involving the arterial tree proximally and/or distally to the popliteal artery. As for the first objection, some surgeons [116] harvest the vein preliminarily, with patient supine, and then turn the patient to the prone position; however, an adequate segment of vein, to perform an interposition graft or a short bypass, may be easily harvested through the posterior approach. The second objection represents undoubtedly an indication to medial approach. The accuracy of the currently available preoperative studies should allow a precise refinement about the choice of the best surgical approach. It seems reasonable to use the posterior approach when the arterial lesions, if any, are strictly limited to midpopliteal artery (Fig. 6.6) or, in any case, a convenient segment of proximal popliteal artery is disease-free and as well the distal popliteal (or the proximal tibioperoneal trunk) looks available for distal anastomosis. When a bypass is planned starting from superficial femoral or ending onto tibial branches, the medial approach is necessary.

The chief problem with medial approach is the limited control of all the structures of the popliteal fossa and the risk of missing anatomical anomalies: for the patient, this may be irrelevant in case of long bypasses [29], but might represent a risk of failure in case of reconstruction localized in the popliteal fossa, like endarterectomy or interposition graft [78] (Fig. 6.7). As a prudential measure, Kim et al. [116] fashion a subcutaneous route for bypass when musculotendinous section has not been performed.

Medial approach is generally preferred when dealing with acute limb ischemia [46], and in these cases preoperative thrombolytic treatment may be of great advantage [59, 119]. Local reconstruction of the popliteal artery relies mainly on endarterectomy + patch graft or vein interposition graft; the latter, when possible, should be preferred to femoropopliteal or femorotibial bypass, allowing a better long-term (80 months) patency, according to Kim et al. [116] (>80% vs 55%). Other types of reconstruction have been occasionally reported: resection and end-to-end anastomosis [26, 59] and grafting with autologous hypogastric artery [32].

Experience with endovascular treatment of popliteal arterial entrapment is still limited. Bürger et al. [120] reported a 6-month positive result (duplex control) in a case of popliteal

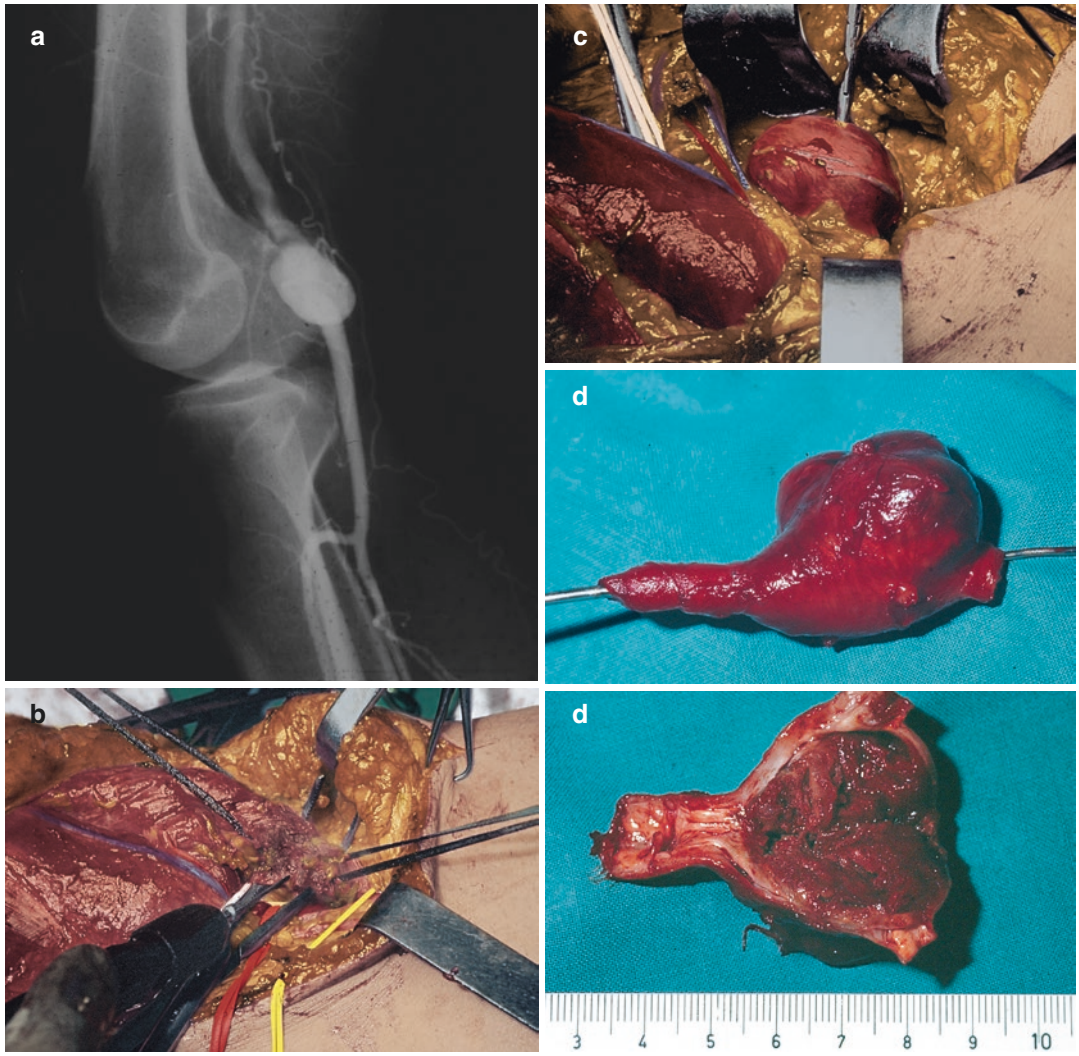

Fig. 6.6 Popliteal aneurysm due to entrapment. (**a**) Arteriography. (**b**) Preparation and section with cautery of medial gastrocnemius attached laterally. (**c**) The aneurysm (treated by resection and interposition vein grafting). (**d**) The operative specimen

thrombosis (possibly caused by an abnormal muscle bundle revealed by CT) treated by thrombolysis and subsequent stenting with a nitinol stent coated with PTFE. The general feeling is that endovascular stenting without removal of the compressing structure may not be effective [121, 122] as indicated by some reports on fracture and/or occlusion of the stent [69, 123]. As well, simple transluminal angioplasty resulted ineffective in a case of stenosis from entrapment erroneously attributed to atherosclerosis due to the advanced age of the patient [124]. Percutaneous balloon angioplasty may look attractive if followed by muscle correction, as it allows preservation of the natural popliteal artery [125], but aneurysm formation seems to be a complication in the short- and mid-term [126].

Functional popliteal entrapment was described by Rignault et al. [127] in 1985. The accepted definition of this still obscure and controversial pathologic condition is external compression of the popliteal artery without identification of any anatomical alteration. Turnipseed [128] defines functional entrapment as a form of overuse injury associated with hypertrophy of medial gastrocnemius, plantaris, and soleus muscles. Lane et al.

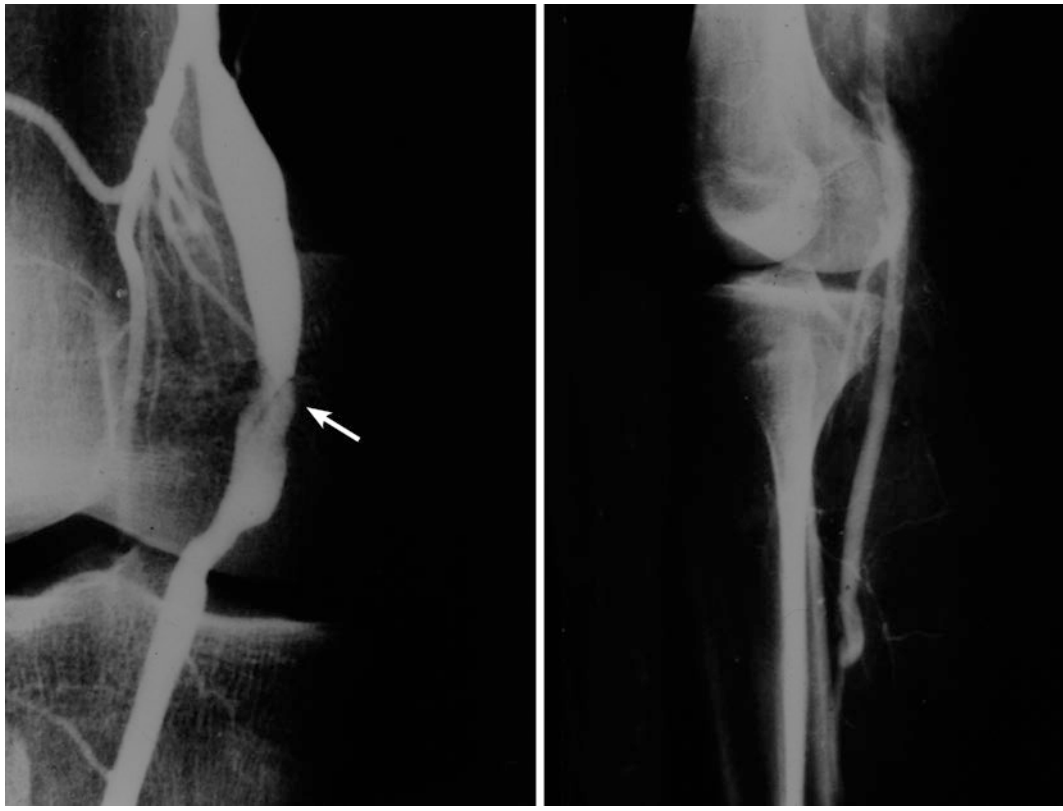

Fig. 6.7 Patient treated with interposition graft for popliteal artery occlusion. Diagnosis of entrapment was missed and consequently musculotendinous section was not performed. After a few months, reappearance of symptoms. Control arteriography (left) demonstrates compression upon the graft, which eventually occluded after few days. Owing to dense fibrous reaction around the artery and the graft, a tibial bypass was necessary (right)

[129] assert that the typical patient is a sports person with claudication but without a definitive anatomical anomaly, pointing to the fact that symptoms may be so invalidating as to produce significant lifestyle interference. The importance of gastrocnemius hypertrophy had been already put into evidence by Darling et al. [83]. In 1971, Evans and Bernhard [130] described a case of acute popliteal artery entrapment attributable to a relevant post-traumatic edema of gastrocnemius. The concept of space competition within the popliteal fossa between different structures anatomically normal but some of them modified in volume was in some way outlined.

In 1999, Levien and Veller [30] reported on 88 limbs (48 patients) affected with popliteal entrapment: this was defined as functional in 30, of which three presented severe ischemia; vein compression was never associated. The hypothesis was that mild anatomical variants, usually clinically silent, might become evident in the subject undergoing muscular hypertrophy [131]. This was supported by the observations [132, 133] made by MRI in healthy asymptomatic subjects without muscle hypertrophy: in those who underwent popliteal artery occlusion during active plantar flexion (called occluders) a more extensive midline position of the medial head of gastrocnemius was demonstrated. Why in a small number of patients severe ischemia develops is still unexplained: Levien [131] suggested the coexistence of unrecognized hypercoagulability, but this was excluded in the case reported by Kim et al. [116].

In 2009, Turnipseed [128] reported on a large series of athletic subjects complaining of symptoms evoking popliteal entrapment or chronic recurrent exertional compartment syndrome: out of a population of 854 patients, anatomical entrapment was diagnosed in 14 (1.6%) and

functional entrapment in 43 (5%). Impressive, through the years (the study started in 1987), was the increasing proportion of female patients due to the progressively larger number of young women playing sports requiring prolonged and strenuous muscular activity. In functional entrapment patients, gratifying results were obtained with a *release* technique performed through the medial approach and consisting of the detachment of gastrocnemius and soleus from the tibia, subtotal resection of the plantaris, and resection of the fibrous superior border of the soleus.

Similar satisfactory results were reported by Levien and Veller [30] in 23 limbs with the simple section, at the level of the tibial plateau, of the muscular portion of the hypertrophied medial gastrocnemius leaving only the tendon, and by Deshpande and Denton [134] with the section of medial gastrocnemius in subjects with failed fasciotomies.

It is evident that the reported experiences are not yet conclusive and that differential diagnosis between functional popliteal entrapment and chronic recurrent exertional compartment syndromes may be very challenging, also because, frequently, positive entrapment studies do not correlate with symptoms, suggesting that popliteal artery impingement in patients with atypical claudication does not necessarily constitute a pathologic condition [135].

Another problem with functional entrapment is the involvement of the vein (observed in about 10% of the cases by Lane et al. [129]). Popliteal vein obstruction during plantar flexion, on which Raju and Neglen [136] focused attention, was observed by Leon et al. [137] in subjects in the supine position and later by Lane et al. [138] when the knee is locked in the standing position and relaxed muscles may *crowd* in the popliteal space.

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