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Adductor canal compression syndrome A forgotten disease



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Paolo Sapienza*, Elvira Tartaglia*, Luigi Venturini*, Piero Gallo**, Luca di Marzo*

*Department of Surgery "Pietro Valdoni", "Sapienza" University of Rome, Italy **Department of Radiological Sciences, Oncology and Pathological Anatomy, "Sapienza" University of Rome, Italy

Adductor canal compression syndrome. A forgotten disease

Superficial femoral vessels entrapped at the level of the adductor canal are rarely described. We first report the case of a young patient who practices professional soccer, affected with superficial femoral vein and artery occlusion due to a hypertrophied vastus medialis and adductor magnus at the Hunter's canal outlet. A careful literature search through MedLine was performed to elucidate the fascinating aspect of this occurrence.

KEY WORDS: Adductor canal syndrome, Entrapment

Introduction

Superficial femoral vessels entrapped at the level of the adductor canal are rarely described¹⁻⁴. A case of a patient affected with artery and vein outlet syndrome at the level of the abductor canal is firstly reported. A complete literature search of the International literature through MedLine (Pubmed. gov, U.S. National Library of Medicine, National Institute of Health) was also performed to elucidate the fascinating aspect of this syndrome.

Case report

A 30-year old Caucasian man was admitted at our Institution for severe calf claudication, hypoesthesia, numbness and mild edema of the left lower limb after moderate physical training. Symptoms arose 8 months earlier and the patient complained calf edema after intensive physical training partially improved after 3 months. The patient was a professional soccer player. Risk factors and associated diseases for atherosclerosis consisted of previous smoking habit (10 cigarettes per day for the past 2 years). No family history of early atherosclerosis was reported. No history of cannabis or other drugs abuse was recorded. On physical examination femoral pulses were normal bilaterally. There were non-palpable pulses below the groin on the left side, but normal distal pulses on the right side. The ankle-brachial index (ABI) was 0.54 in the left leg and normal on the contralateral leg. An ecocolorDoppler showed the occlusion of the popliteal vein from the knee articular rim up to the adductor canal. An echogenic thrombus was present into an incompressible popliteal vein. A rich collateral circulation well compensated vein occlusion. A CT-scan confirmed the occlusion of the superficial femoral artery

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Correspondence to: Paolo Sapienza, MD, PhD Department of Surgery "Pietro Valdoni" "Sapienza" University of Rome, Policlinico Umberto I°, Viale del Policlinico 151, 00161 Rome, Italy (e-mail: paolo.sapienza@uniroma1.it

(Fig. 1, Panel A) extended to the proximal portion of the popliteal artery due to the compression of hypertrophied vastus medialis and adductor magnus muscles (Figure 1, Panel B). Infrapopliteal vessels were all patent. At surgical exploration the vascular bundle was chronically impinged by a hypertrophied vastus medialis and adductor magnus at the Hunter's canal outlet (Fig. 2). No anomalous insertion of the muscles of the adductor canal or hypertrophied tendinous band at the Hunter's canal outlet was recorded. The patient underwent to the



Fig. 1: CT angiography demonstrates the occlusion of the left superficial femoral artery at the level of the adductor canal (Panel A) due to the compression of a hypertrophied vastus medialis and adductor magnus muscles (White arrows) (Panel B).



Fig. 2: The intraoperative picture shows that the vascular bundle (White arrows) is impinged by a hypertrophied vastus medialis and adductor magnus (Yellow arrows) at the Hunter's canal outlet.



Fig. 3: Histology of the femoral artery showed an asymmetrical arrangement of thrombus, intimal and medial hyperplasia, focal calcification, cholesterol deposition, and chronic inflammatory cell infiltration (right side) typical of "vascular entrapment" and on the left side of the artery intimal thickening is negligible (Elastic trichrome, x50).

resection of the occluded arterial segment. The arterial continuity was re-established with a vein interposition graft from the mid-thigh superficial femoral artery to supragenicular popliteal artery. The hypertrophied vastus medialis and adductor magnus muscles were partially resected to allow the anatomical placement of the reconstruction. The vein was harvested from the contralateral limb at the level of the saphenous-femoral junction for a length of 6 cm. Pathologic examination of the excised right femoral artery segment showed an asymmetrical arrangement of thrombus, intimal and medial hyperplasia, focal calcification, cholesterol deposition, and chronic inflammatory cell infiltration thus representing the typical histologic pattern of arterial entrapment (Fig. 3). Hematochemical tests excluded Bechet's syndrome, immunological disorders (ANA, anti-ds DNA, LAC, aCI IgG, aCI IgM, anti-B2 GPI) and coagulative disorders (Factor V G1691A- Leiden, Factor II, Factor V H1299R mutations, plasma level of omocisteine and cisteine, Protein C and Protein S). Post-operative course was uneventful. The patient quitted professional soccer activity. At 50-month follow-up the patient is alive and the reconstruction is patent.

Discussion

A comprehensive computerized literature search from 1950 to 2011, through Medline using the following keywords: [adductor canal] and [femoral artery and vein entrapment] was performed. It was also supplemented by a thorough manual search using bibliographies of pertinent or not pertinent articles. The papers where the

Reference	N° pts.	Sex	Age	Race	Sport Activity	Side	Associated diseases	Symptoms	Location of occlusion structure	Compressing	Treatment	Follow-up (months)
Palma [1952]	1	М	41	Cau	N/A	Left	_	CC	SFA	HT	TEA, LS	N/A
	2	М	34	N/A	N/A	Right	_	CC	SFA	ΗT	TEA, LS	48
	2					Left	_	CC	SFA	ΗT	TEA, LS	48
	3	М	46	N/A	N/A	Right	_	CC, cramping	SFA	ΗT	TEA	N/A
	3					Left	_	CC, cramping	SFA	ΗT	TEA	N/A
	4	М	55	N/A	N/A	Right	_	CC	SFA	ΗT	TEA	24
	4					Left	_	CC	SFA	ΗT	N/A	24
	5	М	61	N/A	N/A	Left	Syphilides	CC, RP	SFA	HT	TEA, LS	N/A
	6	М	53	N/A	N/A	Right		CC	SFA	HT	Arterial liberation, LS	24
	6					Left		CC	SFA	HT	Arterial liberation, LS	24
Lee [1972]	1	М	59	Cau	N/A	Right	_	CC	SFA	HT	TEA	N/A
Balaji [1981]	1	М	26	Cau	Yes	Right	_	CC, H, NU	SFA	HT	Vein interposition	N/A
	2	М	51	N/A	Yes	Left	MI	CC, NU	SFA	HT	Vein interposition	N/A
	3	М	45	N/A	Yes	Left	_	CC, NU	SFA	HT	TEA, patch	N/A
Verta [1984]	1	М	36	N/A	N/A	Right	_	CC	SFA	HT	Vein interposition	6
di Marzo [2011]	1	М	30	Cau	Yes	Right	Smoking	CC, E	SFA	MH	Vein interposition	25

TABLE I - Adductor Canal Compression Syndrome

Legend:

pts=patients; M=male; N/A=not available; Cau=caucasian; RP=rest pain; N=necrosis; CC=calf claudication; NU=numbness; E=edema; H=hypoesthesia; MI=Myocardial Infarction; SFA= Superficial femoral artery; HT=Hypertrophied tendon; TEA=Thromboendarterectomy; LS=Lumbar Sympathectomy; MH=Muscle Hypertrophy.

saphenous nerve was entrapped at the level of the adductor canal were excluded. Four papers1-4 dealing with 11 patients affected with superficial femoral artery occlusion due to adductor canal compression syndrome, which the Authors added 1 case of their own, were identified. A total of 16 limbs were affected, no data were available to evaluate limb dominance. Table I describes demographics, risk factors and associated diseases of the series. Mean age was 45 ± 11 (min. 26 - max. 61). After the analysis of the literature review, we firstly noted that all the cases were published prior 1984 and secondly that Palma¹ reported 6 patients with a bilateral involvement in 4. These aspects are very intriguing and we like to debate these observations. The syndrome might be misdiagnosed in some cases, or the interest to investigate on its occurrence is decreased. Furthermore, since the age at presentation of patients reported by Palma¹ and histologic feature depicting case n° 2, the younger of the series, we speculated that most of these patients might be affected with a usual localization of the atherosclerotic process at the level of the Hunter canal.

The pathogenetic mechanism is an extrinsic compression of the neurovascular bundle inside the adductor canal. Two different clinical presentations have been described for the adductor canal outlet syndrome; 1-compression of the saphenous nerve at the hiatus, causing pain on the medial aspect of the knee without arterial or vein involvement; 2-arterial compression, usually presenting as moderate to severe claudication during vigorous exercise^{5,6}. Two different pathophysiological mechanisms might be hypothesized. The first is a sort of fibrous band or structure compressing the contents of the canal, usually constituted by a tendinous loop of the adductor magnus, which is the main cause of arterial occlusion. The second is a "scissor-like compression mechanism" during the exercise over the femoral artery at the adductor hiatus, caused by a hypertrophied vastus medialis and adductor magnus, in absence of anomalous insertion of the muscles of the adductor canal. We first described a case of hypertrophied muscle compressing the superficial femoral artery at the level of the Hunter canal.

Recently, ultrastructural studies on the normal aspects and relations of the perivascular fibrous tissue of the adductor canal in asymptomatic adult individuals, exhibited a sort of adherence to the vessels and adjacent muscles of the adductor hiatus rather than functioning normally as a 'buffer' or 'cushion' during motion7. The lost of the physiologic scissors-like sliding mechanism, due to connective tissue modification normal in the aging process, affect the normal vessel-canal wall inter-action⁸. We hypothesized that there is a direct correlation between the loss of the physiologic scissors-like sliding mechanism and the vigorous training schedules of endurance athletes. An intensive sporting activity could led to a modification of perivascular fibrous tissue of the adductor canal, with consequent loss of the sliding mechanism during motion, and the development of compressive syndrome and vascular complications. Although in the pre-

sent review it is not possible to reach a definitive conclusion, a direct correlation between the arterial segment involved and the dominant limb might explain the monolaterality of the disease. We propose this simple algorithm to help clinicians to distinguish this condition from early atherosclerosis. Patients younger that 35 years old, highly trained athletes, competing at a professional level (such as skiers, soccer or rugby players, runners etc.), without a family history or risk factors for atherosclerosis should be suspected to have an adductor canal compression syndrome in the presence of calf claudication after physical training. Therefore, a colorDoppler followed by a CT-scan should be performed to confirm the diagnosis. These exams should also exclude the presence of a popliteal artery entrapment syndrome^{9,10}. It is also of interest that this syndrome resembles external iliac endofibrosis¹¹ in high-performance cyclists a relatively well known distinct pathologic entity.

We believe that surgery is the treatment of choice and consists of resection of the occluded arterial segment and vein interposition graft.

In conclusion, adductor canal compression syndrome is rare and it has been not reported for a long time, thus, probably, indicating a loss of interest on this occurrence.

Riassunto

La compressione dei vasi femorali a livello del canale degli adduttori è raramente descritta in letteratura.

Nell'articolo è presentato il caso clinico di un giovane paziente, calciatore professionista, affetto da occlusione dell'arteria e vena femorale superficiale a livello del canale di Hunter causata dall'ipertrofia dei muscoli vasto mediale e adduttore magno.

Nell'articolo è presente inoltre un'attenta revisione della letteratura effettuata su MedLine per delucidare gli affascinanti aspetti di questa patologia.

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