

Anatomical Popliteal Artery Entrapment Syndrome Caused by an Aberrant Plantaris Muscle

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Purpose: We report on cases of anatomical popliteal artery entrapment syndrome (PAES) caused by an aberrant plantaris muscle and highlight the involvement of this muscle in PAES.

Materials and Methods: Seven symptomatic PAES legs in six patients treated at The Division of Vascular Surgery, Asan Medical Center, Seoul, Korea, between 1995 and 2011 were included in this study. We retrospectively analyzed patient records, magnetic resonance imaging (MRI) and/or computed tomography (CT) scans of the knee joint, Doppler pressure studies, CT angiographies, and conventional femoral arteriographies.

Results: Five males and one female patient with a median age of 32 (18-53) years old were enrolled in the study. All patients complained of intermittent claudication of the affected leg. All aberrant plantaris muscles were higher and more medially located than normal plantaris muscles, causing occlusion of the popliteal artery upon forced plantar flexion of the ankle. For arterial lesions, five occlusions of the popliteal artery and two patent popliteal arteries with positive provocation were noted. As for treatment, myotomy of the aberrant plantaris muscle was done for two non-occlusive PAES legs. For occlusive PAES legs, one thrombectomy, one saphenous vein graft interposition of the popliteal artery followed by myotomy, and two below-knee femoro-popliteal bypasses were performed. The median follow-up period was 88 (7-148) months.

Conclusion: An aberrant plantaris muscle can cause anatomical PAES. Classification or diagnosis of PAES should be based on axial studies using CT scans or MRI using various reconstruction methods. Treatment, including myotomy of the plantaris muscle, should be individualized.

Key Words: Popliteal artery entrapment syndrome, Plantaris muscle

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INTRODUCTION

Traditionally, anatomical popliteal artery entrapment syndrome (PAES) was explained and classified according

to the relationship between the medial head of the gastrocnemius muscle and the popliteal artery. With magnetic resonance imaging (MRI) and computed tomography (CT) scans of the knee joint, a more detailed anatomy of the

joint is now available, which highlights the mechanisms of popliteal artery entrapment [1-6]. This has allowed for anomalies such as those affecting the lateral head of the gastrocnemius muscle to be identified as causes of PAES [3,4].

Recently, we experienced a case of treatment failure in a patient who received a thrombectomy and autologous saphenous vein patch closure via posterior approach for an occluded popliteal artery; recurrent thrombosis occurred during the follow-up period and an aberrant plantaris muscle was observed in the CT scan.

Upon re-analyzing anatomical PAES cases not related to anomalies in the medial head of the gastrocnemius, we came across more cases of PAES related to an aberrant plantaris muscle, which we report in this study.

MATERIALS AND METHODS

We retrospectively analyzed 35 PAES legs of 23 patients treated at The Division of Vascular Surgery, Asan Medical Center, Seoul, Korea, between 1995 and 2011. By Rich's et al. [7] classification, type II anomaly, type III anomaly and aberrant plantaris muscle were noted in 51.4% (18/35), 20% (7/35) and 28.6% (10/35), while no cases of type I or type IV anomaly were found in our series. Among these symptomatic and asymptomatic PAES legs, 6 patients (7 legs) affected by symptomatic PAES caused by an aberrant plantaris muscle were included in this study. MRI and/or CT scans of the knee joint were used to diagnose PAES and evaluate musculotendinous anomalies around the popliteal artery. Doppler pressure studies (Doppler ankle-brachial index), CT angiographies, or conventional transfemoral

arteriographies of the lower extremity were used to evaluate the arterial lesions. Plantar flexion of the ankle was produced during the Doppler pressure study, the duplex scan, and the conventional angiographic study to induce



Fig. 1. Three patterns of arterial occlusion. We classified (A) arterial occlusion as segmental occlusion, (B) segmental occlusion with distal extension (diffuse), and (C) patent popliteal artery but tibial artery occlusion by distal embolization.

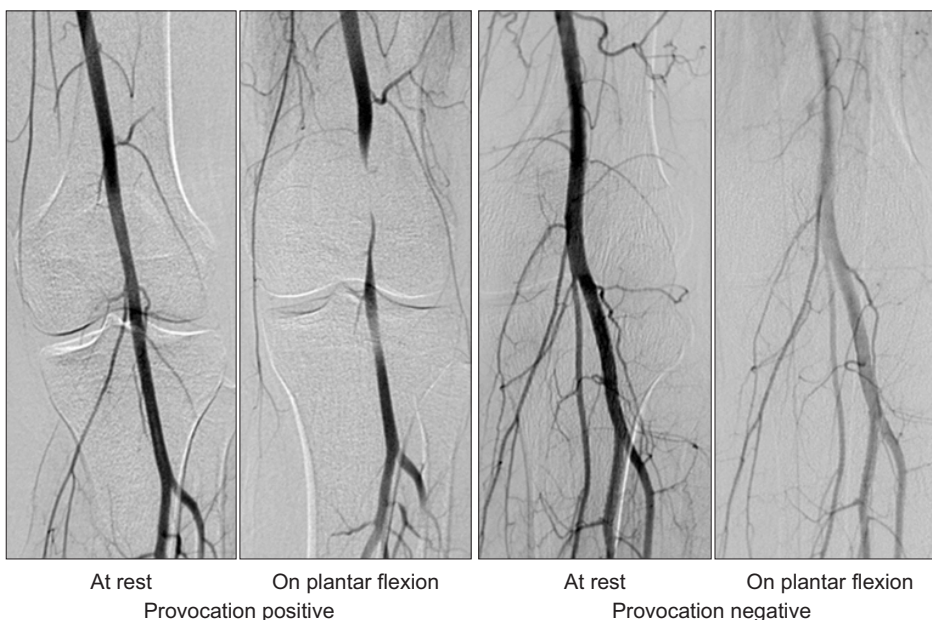


Fig. 2. Provocation study for a patent popliteal artery during transfemoral arteriography. When the popliteal artery is patent, we take an arteriogram with application of plantar flexion of the foot routinely and classified the artery as provocation positive or negative.

symptoms or occlusion of the popliteal artery in cases where no arterial occlusion was found upon examination, despite suspicions of PAES.

Patient records, MRI and/or CT scans of the knee joint, Doppler pressure studies, and CT or conventional transfemoral arteriographies of the lower extremity were completely reviewed. We classified gastrocnemius muscle anomalies according to a study by Rich et al. [7]. Arterial pathology was classified as segmental occlusion, segmental occlusion with distal extension (diffuse), and patent popliteal artery but tibial artery occlusion by distal embolization for occlusive lesions (Fig. 1). For non-occlusive lesions, the arterial pathology was classified as provocation positive or negative (Fig. 2). Follow-up was done on all patients in an outpatient department and/or by telephone.

RESULTS

1) Patient characteristics (Table 1)

Five males and one female patient with a median age of 32 (18-53) years old were included in the study. All patients were physically active and all complained of intermittent claudication of the affected leg. Symptomatic bilaterality was noted in one patient. Symptoms appeared insidiously and repeatedly, but all patients visited the hospital after the symptoms worsened with a median duration of 6 (1-36) months. There were no demonstrable causes for these symptoms except in the case of the female patient who had been taking steroids for 6 months to treat systemic lupus erythematosus (SLE). In this case, we believe hypercoagulability, which was confirmed by positive antiphospholipid antibody, may have been the cause of the popliteal artery occlusion. Upon physical examination of both legs in one of the patients, we observed the typical disappearance of palpable tibial pulses during forced plantar flexion of the foot. For the other patients, the popliteal arteries were already occluded so the tibial pulses were not palpable. All patients were active smokers, except one. There were no other atherosclerotic risk factors to report.

2) Diagnosis of the aberrant plantaris muscle

MRIs of the knee joint (Fig. 3) were performed in four patients who showed typical symptoms of PAES. CT scans of the knee joint were obtained for two of these four patients during CT arteriography of the lower extremity (Fig. 4). No differences were observed between the CT scans and MRIs of the knee joint in these two patients (Fig. 5). For the remaining two patients, who complained of acute leg pain during CT arteriography on the lower extremity, we only

Table 1. Patient characteristics

Patient	Sex	Age (y)	Occupation	Affected leg	Symptom	Symptom duration	Risk factor	Preoperative ABL	Axial scan	Arteriography	Artery status	Provocation(+)	Operative finding of the popliteal artery	Treatment	Results	F-U (mo)
1	Male	20	Student	Right	Claudication	12	Smoking	0.95	MRI	Transfemoral	Patent	Provocation(+)	Normal	Myotomy	Nothing happened	141
2	Male	36	Farmer	Left	Claudication	12	Smoking	0.95	MRI	Transfemoral	Patent	Provocation(+)	Normal	Myotomy	Nothing happened	141
3	Male	53	Farmer	Right	Claudication	3	Smoking	0.63	MRI	Transfemoral	Occlusion	Segmental	Not explored	FEM-POP bypass, BK, RSV graft	Graft occlusion at 38 months postoperatively	151
4	Male	28	Office worker	Right	Claudication	1	Smoking	0.56	CT	CTA	Occlusion	Segmental	Aneurysm/acute thrombosis	Myotomy/interposition, RSV graft	Patent graft	103
5	Male	38	Office worker	Left	Claudication	6	Smoking	0.24	MRI, CT	Transfemoral, CTA	Occlusion	Segmental	Not explored	FEM-POP bypass, BK, RSV graft	Graft occlusion at 3 months postoperatively	84
6	Female	18	Student	Right	Claudication	36	Smoking	0.48	MRI, CT	Transfemoral, CTA	Occlusion	Diffuse	Not explored	Conservative	No more change	66
				Left	Claudication	1	SLE with steroids	0.52	CT	CTA	Occlusion	Segmental	Acute thrombosis	Thrombectomy	Reocclusion at 3 months postoperatively	29

MRI, magnetic resonance imaging; FEM-POP, femoro-popliteal; BK, below knee; RSV, reversed saphenous vein; CT, computed tomography; CTA, CT arteriography; SLE, systemic lupus erythematosus.

obtained a CT scan of the knee joint. All aberrant plantaris muscles were higher and more medially located than the normal plantaris muscle, and they ran along or across the popliteal artery in the upper portion of the popliteal fossa. This can cause occlusion of the popliteal artery during forced plantar flexion of the ankle. These findings were confirmed during surgery (Fig. 6).

3) Arteriography and arterial pathology

Transfemoral arteriography of the lower extremity was



Fig. 3. Magnetic resonance imaging of the knee joint. An aberrant plantaris muscle with its course is shown in left leg (circle) while there is no aberrant plantaris muscle in right leg.



Fig. 4. Computed tomography scan and arteriogram of the knee joint. An aberrant plantaris muscle (circle) and occlusion of the popliteal artery is shown in right leg while there is no arterial occlusion or aberrant plantaris muscle in left leg.

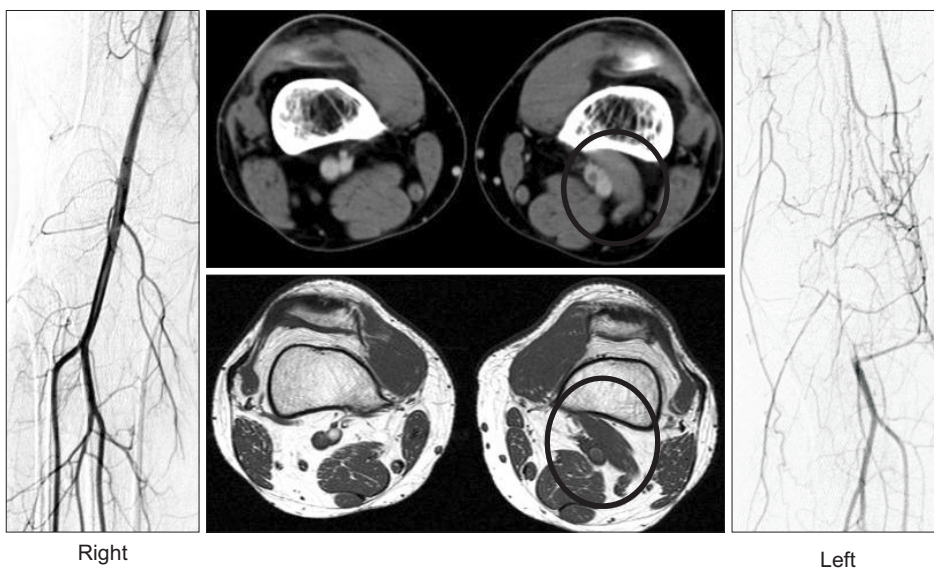


Fig. 5. Magnetic resonance imaging (MRI) and computed tomography (CT) scan of the knee joint. There is no discrepancy in findings between CT scan (upper box) and MRI (lower box). An aberrant plantaris muscle with arterial occlusion is shown in left leg (circle) while there is no aberrant plantaris muscle or arterial occlusion in right leg.

performed in four patients who showed typical symptoms of PAES. For one patient with bilateral PAES, we performed the provocation study during transfemoral arteriography of the lower extremity, which showed a patent popliteal artery at rest but occlusion of the popliteal artery during plantar flexion of the foot (Fig. 2A). This finding was confirmed by passive plantar flexion of the foot during the operation. One of the objectives in performing the arteriography was to evaluate the asymptomatic contralateral leg upon provocation. For patients with unilateral PAES, we observed occlusion of the popliteal artery in the affected leg, but the provocation study was negative on asymptomatic contralateral legs (Fig. 2B). CT arteriography of the lower extremity was obtained for two out of these four patients. We observed no differences in the findings between the CT and transfemoral arteriographies of the lower extremity in these two patients. The remaining two patients, who complained of acute leg pain, only received a CT arteriography of the lower extremity. Arteriography findings for all four patients showed segmental occlusion of the popliteal artery (Fig. 1A) as the most common pattern.

4) Treatment

Surgery was performed on six PAES legs in five patients

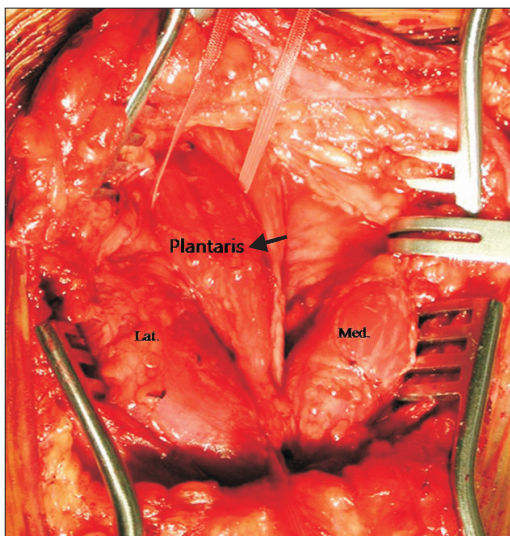


Fig. 6. Operative finding of the aberrant plantaris muscle (black arrow) in left leg. This muscle is almost vestigial in man but aberrant plantaris muscle is higher and more medially located than the normal plantaris muscle, and runs along or across the popliteal artery in the upper portion of the popliteal fossa. This can cause occlusion of the popliteal artery during forced plantar flexion of the ankle. Lat.: lateral head of the gastrocnemius muscle, Med.: medial head of the gastrocnemius muscle.

and conservative treatment was recommended for one patient. The popliteal artery was explored posteriorly in four legs, including two patent and two arteries segmentally occluded by acute thrombosis. In field, by repeating dorsi-flexion and plantar-flexion of the ankle passively, we could easily find the muscle compressing the artery. Myotomy of the aberrant plantaris muscle at the origin was performed in two non-occlusive PAES legs (in one patient). Thrombectomy and saphenous vein graft interposition of the popliteal artery followed by myotomy of the aberrant plantaris muscle were performed, respectively, in the other two patients. Two below-knee femoro-popliteal bypasses using a reversed saphenous vein graft via a medial approach were carried out on two segmentally occluded popliteal arteries. In these two cases, we did not explore the plantaris or medial head of the gastrocnemius muscle at the knee, but placed the graft in the subcutaneous layer. Conservative treatment was conducted in one patient with a diffusely occluded popliteal artery.

5) Treatment results and follow-up

Myotomy in PAES legs showed excellent results. The symptoms disappeared after the operation and no symptoms were noted within a 141 month follow-up period. As for direct repair of the occluded popliteal artery, graft interposition followed by myotomy showed excellent results with a patent graft over a 103 month follow-up period. On the other hand, thrombectomy with a saphenous vein patch angioplasty showed re-occlusion of the popliteal artery within 3 months of the operation in spite of anticoagulation therapy (Fig. 7). In this case, the patient's blood tested positive for lupus antigen and SLE, so we believe that hypercoagulability was the cause of occlusion of the popliteal artery. Regarding bypass surgery, the results were not promising. Both grafts became occluded within 3 months and 38 months of the operation, with a median follow-up period of 88 (7-148) months.

DISCUSSION

PAES has been classified into various types according to the abnormal relationship between the medial head of the gastrocnemius muscle and the popliteal artery in the popliteal fossa. A deeper understanding of developmental embryology and the mechanisms of how to impinge the popliteal artery have allowed the classification to be modified [7-9]. The most widely accepted classification was made by Whelan and modified by Rich, and divides PAES into six types [7]. However, MRI or CT scans of knee joints have provided a more precise anatomy of the knee and

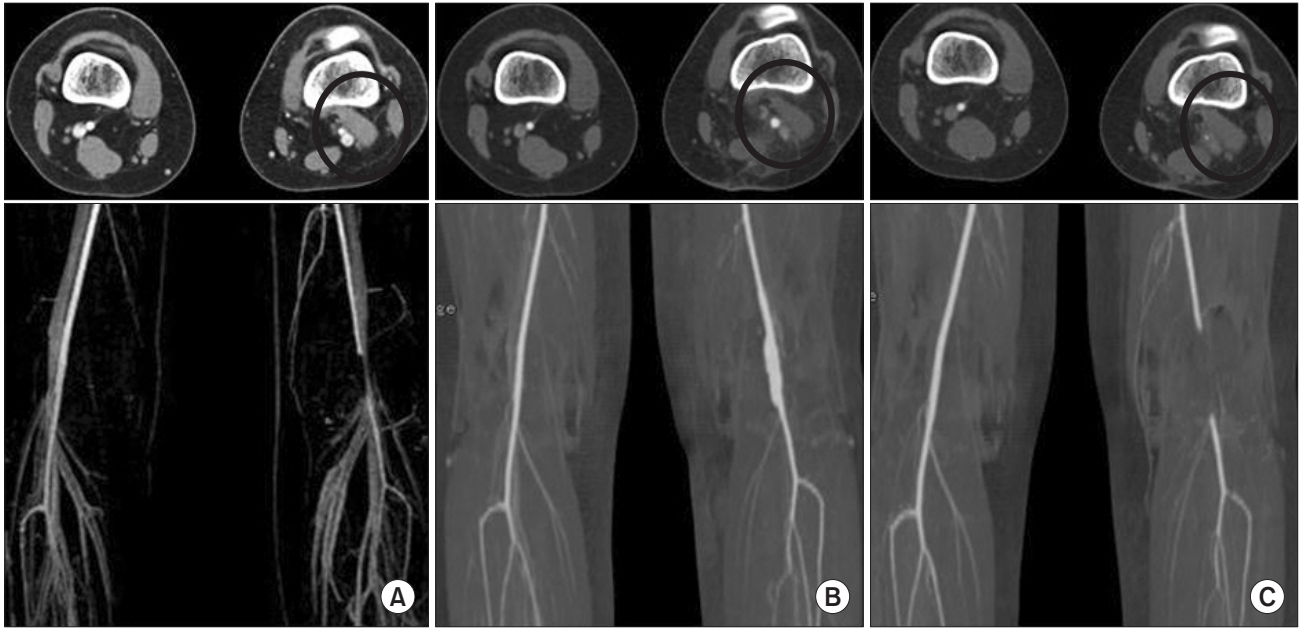


Fig. 7. Re-occlusion of the popliteal artery in a patient without myotomy of the aberrant plantaris muscle. This result suggested that myotomy of the aberrant plantaris muscle is a key procedure to maintain arterial patency. Computed tomography scan and arteriogram: Initial (A), following thrombectomy with autologous saphenous vein patch closure without myotomy of the plantaris muscle via posterior approach (B), re-occlusion of the popliteal artery following the same pattern as the original lesion 3 months postoperatively (C). An aberrant plantaris muscle is shown in axial scans (black circle).

explained the mechanisms of popliteal artery entrapment, and so new types of PAES, such as anomalies of the lateral head of the gastrocnemius muscle, have also been introduced as a cause of PAES [3,4].

The plantaris muscle belongs to a superficial group of posterior crural muscles and plays a role in plantar flexion of the ankle. However, this muscle is almost vestigial and presumed to act in combination with the gastrocnemius in humans, while it is well developed in many mammals. It has been reported to be aberrant in approximately 10% of cases [5,10,11]. When the plantaris muscle develops abnormally, it can entrap the popliteal artery during contraction, which occurs during plantar flexion of the ankle [12-15]. In our study, all aberrant plantaris muscles were higher and more medially located than the normal plantaris muscle, and ran along or across the popliteal artery in the upper portion of the popliteal fossa. Here, they caused occlusion of the popliteal artery during forced plantar flexion of the ankle. It was proven in one of our series (Fig. 6).

As for arterial pathology, the pathogenesis has been explained as a repeated compression of the popliteal artery leading to mechanical damage of the vessel wall, which may present as normal, occlusion, aneurysm, thrombosis, and distal embolization [16-19]. We could not determine how the arterial pathology developed, although thrombotic

occlusion was believed to be the cause based on our operative findings, but we could classify the arterial lesion as segmental occlusion, segmental occlusion with distal extension, and distal embolization without popliteal artery occlusion for occlusive lesions, and provocation positive or negative for patent arteries. In our study, occlusion of the popliteal artery (5/7) was more common than a patent popliteal artery, and in five cases of arterial occlusion, segmental occlusion of the popliteal artery was the most common pattern.

As for the surgical approach, we routinely used a posterior approach for myotomy as recommended in several studies [16,18,19]. It was useful to identify muscle anomalies and abnormal relationships between the popliteal artery and the gastrocnemius or any other muscles including the plantaris muscle.

Regarding occlusive arterial lesions, we performed surgery to treat segmental occlusion of the popliteal artery, while conducting conservative treatment in the case of diffuse arterial occlusion. Bypass surgery using a reversed saphenous vein graft has been reported to yield good results with long-term patency [1,8,16,17,19]. However, in our case, the results of bypass surgery were not as promising as we expected. Bypass might have shown good results if the arterial occlusion was not caused

by atherosclerosis, but the reason for this poor result is uncertain. Both grafts used for the femoro-popliteal bypass became occluded soon after surgery, while interposition of the popliteal artery followed by myotomy of the plantaris muscle via a posterior approach was still patent. When direct repair of the popliteal artery is performed via a posterior approach, myotomy of the anatomically deranged muscle should follow. In the case of the patient who underwent a thrombectomy with autologous saphenous vein patch closure without myotomy of the plantaris muscle, as we didn't have a concept of aberrant plantaris muscle at the time, we observed re-occlusion of the popliteal artery following the same pattern as the original lesion within 3 months of the operation, even though

adequate anticoagulation therapy with warfarin had been administered. Additional myotomy wasn't performed as the patient refused a second operation.

CONCLUSION

An aberrant plantaris muscle can cause PAES and it is more common than we thought. Therefore, classification or diagnosis of PAES should be based on image studies using CT scan or MRI with various reconstruction methods. We believe this to be a more practical approach than the previous classification based on embryology and operative findings. In addition, treatment should be individualized, including myotomy of the plantaris muscle.

REFERENCES

- 1) Turnipseed WD. Popliteal entrapment syndrome. *J Vasc Surg* 2002;35:910-915.
- 2) Chernoff DM, Walker AT, Khorasani R, Polak JF, Jolesz FA. Asymptomatic functional popliteal artery entrapment: demonstration at MR imaging. *Radiology* 1995;195:176-180.
- 3) Liu PT, Moyer AC, Huettl EA, Fowl RJ, Stone WM. Popliteal vascular entrapment syndrome caused by a rare anomalous slip of the lateral head of the gastrocnemius muscle. *Skeletal Radiol* 2005;34:359-363.
- 4) Kim HK, Shin MJ, Kim SM, Lee SH, Hong HJ. Popliteal artery entrapment syndrome: morphological classification utilizing MR imaging. *Skeletal Radiol* 2006;35:648-658.
- 5) Pillai J, Levien LJ, Haagensen M, Candy G, Cluver MD, Veller MG. Assessment of the medial head of the gastrocnemius muscle in functional compression of the popliteal artery. *J Vasc Surg* 2008;48:1189-1196.
- 6) Tanaka H, Higashi M, Fukumoto Y, Ogino H. Entrapment of the popliteal artery. *J Vasc Surg* 2010;52:479.
- 7) Rich NM, Collins GJ Jr, McDonald PT, Kozloff L, Clagett GP, Collins JT. Popliteal vascular entrapment. Its increasing interest. *Arch Surg* 1979;114:1377-1384.
- 8) Persky JM, Kempczinski RF, Fowl RJ. Entrapment of the popliteal artery. *Surg Gynecol Obstet* 1991;173:84-90.
- 9) Levien LJ, Veller MG. Popliteal artery entrapment syndrome: more common than previously recognized. *J Vasc Surg* 1999;30:587-598.
- 10) Vishy Mahadevan. Plantaris muscle in chapter 83 leg/muscles. In: Standring S, editor. *Gray's anatomy*. 40th ed. London: Churchill Livingstone; 2008. p. 1421.
- 11) Moore KL, Dalley AR. Clinically oriented anatomy. 6th ed. Philadelphia: Lippincott Williams and Wilkins; 2008. p. 652-660.
- 12) Erdoes LS, Devine JJ, Bernhard VM, Baker MR, Berman SS, Hunter GC. Popliteal vascular compression in a normal population. *J Vasc Surg* 1994;20:978-986.
- 13) Ferrero R, Barile C, Bretto P, Buzzacchino A, Ponzio F. Popliteal artery entrapment syndrome (report on seven cases). *J Cardiovasc Surg (Torino)* 1980;21:45-52.
- 14) Bouhoutsos J, Daskalakis E. Muscular abnormalities affecting the popliteal vessels. *Br J Surg* 1981;68:501-506.
- 15) Turnipseed WD, Pozniak M. Popliteal entrapment as a result of neurovascular compression by the soleus and plantaris muscles. *J Vasc Surg* 1992;15:285-293.
- 16) Murray A, Halliday M, Croft RJ. Popliteal artery entrapment syndrome. *Br J Surg* 1991;78:1414-1419.
- 17) di Marzo L, Cavallaro A, Sciacca V, Mingoli A, Tamburelli A. Surgical treatment of popliteal artery entrapment syndrome: a ten-year experience. *Eur J Vasc Surg* 1991;5:59-64.
- 18) Kwon TW, Kim SK. Popliteal artery entrapment syndrome-case report. *Kor J Vasc Surg* 1996;12:85-88.
- 19) Lambert AW, Wilkins DC. Popliteal artery entrapment syndrome. *Br J Surg* 1999;86:1365-1370.