

Minimally Invasive Management of Cystic Adventitial Disease in the Popliteal Artery: A Case Report and Review

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SOUHRN

Kontext: Cystická degenerace adventicie (cystic adventitial disease, CAD) je vzácnou příčinou stenózy tepen, které postihuje převážně popliteální tepnu.

Kazuistika: K lékaři se dostavil 46letý muž s náhlou bolestí v pravém lýtku. Fyzikální vyšetření a CT angiografie odhalily významnou stenózu pravé popliteální tepny. Protože se nepodařilo iniciální trombektomií odstranit trombotický materiál, bylo rozhodnuto provést hybridní výkon zahrnující PTA a implantaci stentu. Vyšetření MR potvrdilo CAD a při kontrole po 24 měsících nebyly u pacienta zjištěny žádné příznaky.

Diskuse: Etiologie CAD je stále předmětem diskuse, přičemž byly předloženy teorie o místním poranění a synoviální/ganglionové cystě. Tradiční léčba byla chirurgická, nicméně tato kazuistika prokazuje účinnost minimálně invazivního přístupu.

Závěr: Minimálně invazivní endovaskulární léčba může být v případě CAD účinná; po výkonu je pro zajištění dlouhodobé průchodnosti tepny nezbytné pacienta pečlivě sledovat.

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ABSTRACT

Background: Cystic adventitial disease (CAD) is a rare cause of arterial stenosis, primarily affecting the popliteal artery.

Case presentation: A 46-year-old male presented with sudden right calf pain. Physical examination and CT angiography revealed significant stenosis of the right popliteal artery. Initial thrombectomy failed to remove thrombotic material, prompting a hybrid approach with PTA and stent implantation. MRI confirmed CAD, and the patient remained asymptomatic at 24-month follow-up.

Discussion: The etiology of CAD is debated, with theories including local trauma and synovial/ganglion origin. Traditional management has been surgical, but this case demonstrates the efficacy of a minimally invasive approach.

Conclusion: Minimally invasive endovascular treatment may be effective for CAD, with close monitoring needed to ensure long-term patency.

Keywords:

Cystic adventitial disease

Endovascular treatment

Hybrid surgery

Introduction

Cystic adventitial disease (CAD) is a rare but well-documented form of nonatherosclerotic arterial stenosis. While it predominantly affects the popliteal artery (PA), it can also involve other arteries or veins.¹ The incidence of CAD is estimated to be 1 in 1,200 cases of claudication and 1 in 1,000 femoral angiograms, with a notable male predominance of 15 : 1. Approximately 85% of CAD cases are localized in the popliteal region.² Patients typically present as young, otherwise healthy individuals with no signs of multilevel atherosclerosis. Due to the rarity of the condi-

on and the limited literature, primarily consisting of case reports, the management of CAD remains individualized and patient-specific.

Case presentation

A 46-year-old male presented with a sudden onset of pain in the right calf following prolonged squatting while gardening. The symptoms began seven days prior to admission. Unlike classic claudication, he was able to walk approximately one kilometer without pain, but experienced pain onset when ascending stairs. The patient had no

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Fig. 1 – Sagittal view of a subtotal occlusion of the right popliteal artery.

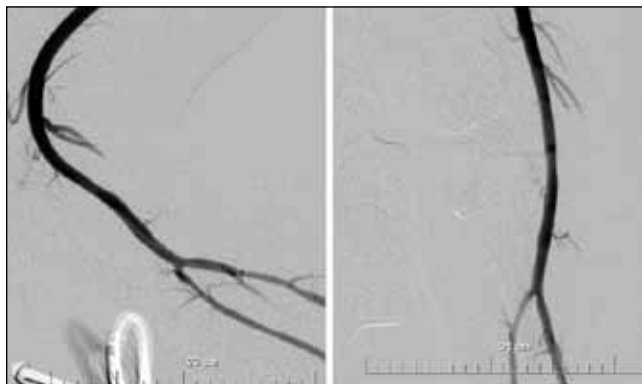


Fig. 2 – Post-thrombectomy angiography with flexed knee on the left and extended knee on the right. The angio suprisingly shows no residual stenosis.

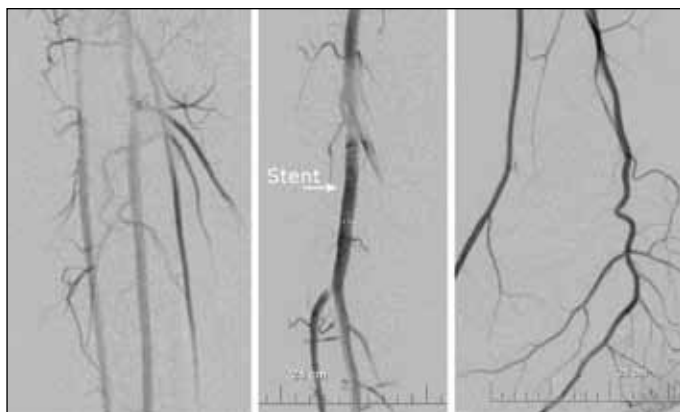


Fig. 3 – Post operative angiographies showing a patent popliteal stent in the middle, and three below the knee arteries run-off.

comorbidities and had undergone arthroscopic cruciate ligament repair of the right knee five years earlier due to trauma.

Physical examination revealed the absence of pulsations in the anterior and posterior tibial arteries of the right leg, while the popliteal artery pulse remained palpable. An ankle-brachial index (ABI) was measured, showing significant discrepancies between the two legs, with an ABI of 0.5 on the right compared to 1.0 on the left.

The patient underwent a computed tomography angiography (CT-Angio), which revealed a significant 90% isolated stenosis of the right popliteal artery without an apparent underlying cause (Fig. 1).

Taking in consideration the sudden onset of symptoms and the high probability of fresh thrombus formation, we initially opted for an open thrombectomy of the popliteal artery via a femoral incision using a 4Fr Fogarty catheter. No thrombotic material was evacuated, prompting us to proceed with a hybrid approach. Our angiography post thrombectomy surprisingly showed no residual stenosis and excellent distal run-off (Fig. 2). Our hypothesis is that the outward force exerted by balloon of the Fogarty catheter itself was enough to push back the cystic formation.

Those findings prompted us to use IVUS which showed homogenous stenosis on the back wall of the PA. This involved percutaneous transluminal angioplasty (PTA) of the popliteal artery using a drug-eluting balloon. Follow-up angiography revealed no residual stenosis but a slight hint of a dissection flap leading to the decision to implant a short self-expandable stent. The final angiography showed excellent results – three patents below the knee arteries run-off, with palpable pulses restored in the anterior and posterior tibial arteries (Fig. 3)

Post-operative magnetic resonance imaging (MRI) revealed a cystic formation on the posterior wall of the popliteal artery in the previously stenosed area, confirming the pathogenesis of the lesion. The stent remained widely patent with no signs of residual stenosis (Fig. 4).

The patient has started on clopidogrel 300 mg loading dose immediately post procedure and 75 mg once daily. Following the intervention, the patient experienced

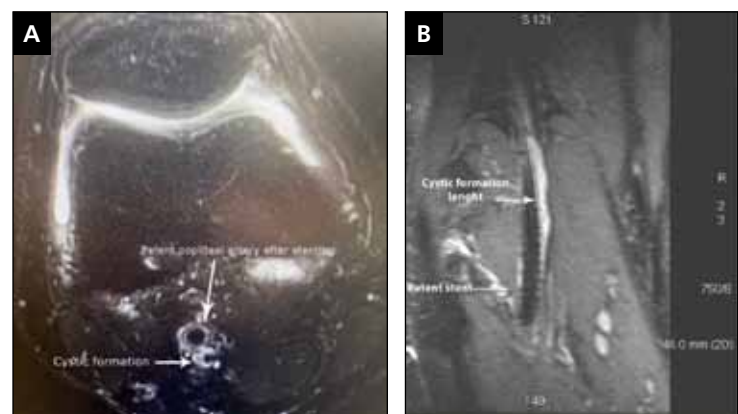


Fig. 4 – MRI of the right popliteal artery showing a cystic formation on the back artery wall with patent stent and no sign of compression. (A) Transversal view. (B) Sagittal view.

relief of claudication symptoms and remained asymptomatic at the 12-month follow-up. A noninvasive Doppler ultrasound examination confirmed patency of the stent. The ankle-brachial index at the 12-month follow-up was normal at 1.0.

At the 24-month interview, the patient reported remaining asymptomatic and continued to tolerate exercise well.

Discussion

The etiology of cystic adventitial disease (CAD) remains controversial, with several potential theories proposed, including: 1) repetitive local trauma, 2) systemic disease, 3) synovial/ganglion origin, and 4) embryological development.^{3,4}

The repetitive local trauma theory postulates that vessels located near joints are subject to repetitive microtrauma, leading to disruption of the adventitia from the media. This disruption can result in intramural bleeding and subsequent cyst development due to enzymatic activity within the vessel wall.² There have been reports of trauma induced cysts; however, this theory is controversial as CAD can occur in children and is rare in athletes.⁴

Linquette et al. proposed the systemic disease theory, suggesting that CAD is related to a generalized disorder of myxomatous degeneration. However, this theory has failed to gain adequate support, as patients did not show systemic lesions on follow-up.² The synovial/ganglion theory and the embryological theory have recently gained more favor in the etiology of CAD. The synovial/ganglion theory proposes that adventitial cysts develop due to migration of synovial ganglions from the adjacent joint capsule or tendons, along vascular branches to the adventitia.⁵ The synovial/ganglion theory seems to fit best in our case keeping in mind the fact that he had previous surgical repair in the knee joint. The embryological theory hypothesizes that during development, mesenchymal cells are incorporated into the adventitia from adjacent joint tissue. These cells then secrete mucin over a number of years giving rise to adventitial cysts which have the potential to encroach on the arterial lumen.^{2,4} Because there is no single unifying theory able to account for the pathogenesis of all clinical cases, CAD may be better explained by a combination of multiple theories.

Management of CAD has predominantly been surgical^{6,7} involving either cyst drainage to allow full arterial lumen expansion or arterial reconstruction via bypass. Results generally favor arterial reconstruction, although evidence has been primarily derived from case reports and occasional small series. A minimally invasive approach using percutaneous cyst drainage⁸ has been described but has shown mixed results. Endovascular techniques have been infrequently utilized, typically involving balloon angioplasty or focusing on secondary interventions.^{9–11}

Due to the location of the cyst beneath the adventitia, balloon angioplasty is likely to be ineffective. Unless there is disruption of the intima, media, or adventitia allowing drainage of the cyst contents either into the artery or towards the popliteal space, the cystic lesion is

expected to remain unchanged or potentially refill with fluid over time.

At the time of the procedure, there was suspicion of CAD in this particular patient. However, we were surprised to observe a near-excellent result following Fogarty thrombectomy. This outcome is puzzling because significant force would theoretically be required to maintain artery patency if there were indeed compressions in this segment.

This observation led us to consider that stent placement alone might suffice, offering the potential for long-term patency with minimal surgical trauma, as it was the case ultimately.

The rationale behind deploying a bare-metal, self-expanding stent is to utilize its radial force to induce remodeling of the arterial wall. This mechanism theoretically involves “pushing” the contents of the cyst out of the adventitia, potentially utilizing anatomical connections similar to those described with the knee joint. This process aims to collapse the cystic cavity and stimulate an inflammatory reaction that could lead to cyst sealing. However, proving this concept is challenging definitively.¹²

Intimal hyperplasia and restenosis are significant concerns with this procedure. Minimizing arterial injury by avoiding balloon angioplasty before or after stent deployment is crucial. Close patient follow-up is essential, with re-intervention if necessary. Given the impracticality of directly intervening on the cyst, a short bypass graft would be considered as the primary option.

Conclusion

Cystic adventitial disease of the arteries presents a challenging diagnostic and therapeutic dilemma, characterized by cystic formations within the adventitial layer, predominantly affecting the popliteal artery. Management has traditionally been surgical, with options ranging from cyst drainage to arterial reconstruction. Our case highlights the efficacy of a minimally invasive approach using bare-metal, self-expanding stents, which leverages their radial force to potentially remodel the arterial wall and address the underlying pathology without the need for extensive surgical trauma.

However, the management of CAD remains complex, with considerations including the risk of intimal hyperplasia and restenosis post-stent placement. Close monitoring and prompt reintervention are essential to ensure long-term patency and symptom relief. Further research is warranted to elucidate the optimal treatment strategies and long-term outcomes of endovascular interventions in CAD.

Conflict of interest

The authors declare that they have conflict of interest.

Funding

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Ethical statement

Our institution does not require an ethical approval for reporting individual cases or case series.

Informed consent

Verbal informed consent was obtained for anonymized patient information to be published in this article.

References

- Desy NM, Spinner RJ. The etiology and management of cystic adventitial disease. *J Vasc Surg* 2014 Jul;60:235–245.e11.
- Levien LJ, Benn CA. Adventitial cystic disease: A unifying hypothesis. *J Vasc Surg* 1998;28:193–205.
- Mathiasen RE, Luo L, Norton A, Vargo C. Cystic Adventitial Disease of the Popliteal Artery in an Adult Male Soccer Player: A Case Report. *Curr Sports Med Rep* 2018;17:287–289.
- Desy NM, Spinner RJ. The etiology and management of cystic adventitial disease. *J Vasc Surg* 2014;60:235.e11–245.e11.
- Crolla RM, Steyling JF, Hennipman A, et al. A case of cystic adventitial disease of the popliteal artery demonstrated by magnetic resonance imaging. *J Vasc Surg* 1993;18:1052–1055.
- Chervakov V, Valchev D, Kirova G, Daskalov A. Popliteal Adventitial Cystic Disease. Case Report. *Cor Vasa* 2022;64:560–563.
- Baxter AR, Garg K, Lamparello PJ, et al. Cystic adventitial disease of the popliteal artery: is there a consensus in management? *Vascular* 2011;19:163–166.
- Van Rutte PWJ, Rouwet EV, Belgers EHJ, et al. Treatment of Popliteal Artery Cystic Adventitial Disease, Primary Bypass Graft not Always First Choice: Two Case Reports and a Review of the Literature. *Eur J Vasc Endovasc Surg* 2011;42:347–354.
- Khoury M. Failed Angioplasty of a Popliteal Artery Stenosis Secondary to Cystic Adventitial Disease – A Case Report. *Vasc Endovascular Surg* 2004;38:277–280.
- Fox RL, Kahn M, Adler J, et al. Adventitial cystic disease of the popliteal artery: Failure of percutaneous transluminal angioplasty as a therapeutic modality. *J Vasc Surg* 1985;2:464–467.
- Maged IM, Kron IL, Hagspiel KD. Recurrent Cystic Adventitial Disease of the Popliteal Artery: Successful Treatment With Percutaneous Transluminal Angioplasty. *Vasc Endovasc Surg* 2009;43:399–402.
- Mertens R, Bergoeing M, Mariné L, et al. Endovascular Treatment of Cystic Adventitial Disease of the Popliteal Artery. *Ann Vasc Surg* 2013;27:1185.e1–1185.e3.