

# Functional popliteal artery entrapment syndrome (FPAES) treatment with incobotulinumtoxinA: three successful clinical cases

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## Introduction

- Functional popliteal artery entrapment syndrome (FPAES) is an uncommon cause of lower-limb claudication, most commonly affecting young athletes.
- Untreated, FPAES may lead to popliteal artery (PA) damage, embolization and limb ischemia.
- The most frequent causes of FPAES include muscular hypertrophy and muscle anatomical traps of the popliteal fossa (PF).
- Possible treatments include surgery (i.e. dissection, bridging) and botulinum toxin type A (BoNT-A) injections under guidance.
- Literature is very limited regarding the use of incobotulinumtoxinA (INCO; Xeomin®) for the treatment of FPAES.

## Objective

- To describe the efficacy and safety of INCO for the treatment of FPAES using 3 clinical cases that presented in our clinical practice.

## Conclusions

- Following optimisation of treatment with INCO, all 3 patients resumed physical activities.
- No safety issues were reported by any of the patients.
- These 3 cases suggest that INCO can be an effective and safe treatment option for FPAES.

## Clinical cases

Table. Summary of clinical cases

	Medical history	INCO treatments	Clinical outcomes
<b>CASE 1</b> 56 y/o male	Had typical symptoms for the last 2 yrs. Conventional angiogram showed bilateral occlusion of the PA, at the PF level, during active plantar flexion from extrinsic compression by proximal calf muscles (Figure a). CT scan showed main contribution from the gastrocnemius medial (GM) on each side.	<ol style="list-style-type: none"> <li>The patient was injected for the first time with 50 U of INCO (2:1 dilution, one site in both GM), using CT guidance.</li> <li>The second treatment was performed 6 mos. later using ultrasound (US) guidance (Figure b), 50 U was injected in both GM and gastrocnemius lateral (GL), for a total dose of 200 U.</li> <li>A third treatment was performed 6 mos. later, using US guidance, 50 U in both GM and GL, and 50 U in left PM, for a total dose of 250 U.</li> </ol>	<ol style="list-style-type: none"> <li>No improvement was observed at 3 mo. follow-up.</li> <li>Patient noticed significant improvement of the symptoms in right leg, but not in left leg. MRI was performed 2 mos. later and showed bilateral focal atrophy at the injection sites (Figure c), without arterial occlusion, but hypertrophy of the left popliteal muscle (PM). Symptoms resumed after 3 mos.</li> <li>The patient saw complete remission of his symptoms after 3 wks., without recurrence at 48 mos. follow-up.</li> </ol>
<b>CASE 2</b> 47 y/o male	Bilateral progressive symptoms for many yrs., disabled when running, climbing stairs, or walking fast. US showed bilateral occlusion of the PA during active plantar flexion, at the PF level.	<ol style="list-style-type: none"> <li>The patient was first injected using US guidance, with 50 U INCO in both GM and GL, for a total dose of 200 U.</li> <li>A second series of injections was repeated 12 mos. later using the same protocol.</li> </ol>	<ol style="list-style-type: none"> <li>The symptoms completely disappeared after 3 wks., and the patient has been able to run two half-marathons in subsequent mos. MRI was performed 2 mos. after the injection and showed bilateral focal atrophy at the injection sites, without arterial occlusion. Symptoms recurred 9 mos. after the first treatment.</li> <li>The patient showed the same significant improvement and was still asymptomatic at 9 mos. follow-up.</li> </ol>
<b>CASE 3</b> 40 y/o male	Unilateral, typical symptoms in left leg for the last 2 yrs. MRI-angiogram first showed unilateral occlusion of left PA during active plantar flexion, at the PF level. US showed the same findings but the artery seemed mainly occluded by GM.	<ol style="list-style-type: none"> <li>75 U of INCO was injected in GM and 25 U in GL.</li> <li>Patient was then injected 5 mos. later with 75 U in left GM and 25 U in left PM.</li> </ol>	<ol style="list-style-type: none"> <li>The patient saw minimal improvement of his condition at 3 mos. follow-up. MRI was performed 4 mos. after the first treatment and showed no muscle atrophy but no artery occlusion during forced plantar flexion. A second evaluation was done 1 mo. later using US guidance and confirmed extrinsic arterial occlusion during plantar flexion, with contribution mainly from GM and PM.</li> <li>Symptoms disappeared 3 wks. later, without recurrence at 6 wks. follow-up.</li> </ol>

Figure. a) Angiogram showing bilateral occlusion of the PA. b) INCO injection using US guidance. c) Bilateral focal atrophy at the injection sites (Top: before; Bottom: 2 mos. post injection)

