

## A rare case of an acute soleus arcade syndrome complicated by a ganglion cyst: diagnosis by dynamic ultrasound

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### Abstract

The soleus arcade syndrome is a rare compression neuropathy of the tibial nerve that often remains undiagnosed due to low clinical awareness and difficult diagnosis. We present the case of a female patient admitted with acute worsening of a pre-existing sensory tibial neuropathy and acute tibial nerve palsy after knee joint injection. After a knee magnetic resonance imaging remained non-diagnostic, dynamic ultrasonography was performed. Constriction by the soleus arcade and proximal swelling of the tibial nerve could be demonstrated during plantarflexion of the ankle by means of a dynamic examination in the standing patient. The patient underwent surgery and recovered fully. This proposed diagnostic approach can be used to identify soleus arcade syndrome by ultrasound.

**Keywords:** soleus arcade syndrome; tibial nerve; ganglion cyst; dynamic ultrasonography

### Introduction

The soleus arcade syndrome (SAS) is a functional symptomatic compression of the tibial nerve (TN) at its entry under the soleus arcade (SA) – a tendinous arch forming a part of the origin of soleus muscle (fig 1a) [1,2] and an important differential diagnosis in patients with suspected TN neuropathy [1,3]. Comparable to other peripheral compression neuropathies such as the posterior interosseous nerve syndrome [4], the tibial nerve is thought to face repeated or sustained compression leading to oedema, perfusion imbalances and demyelination with axonal damage in late stages [5]. In general, the surgical treatment of focal compressive neuropathy of the TN is straightforward; the diagnosis itself can be complex due to low clinical awareness and a challenging workup with

often only subtle morphological changes and a primarily functional impairment [2,6].

Routinely, after a working diagnosis of SAS is established clinically, electrodiagnostic testing (EDx) is performed [1,3]: While EDx may demonstrate reduced sensory nerve conduction velocity, probably due to Schwann cell degeneration, findings can be unspecific due to the intermittent character of the strangulation mechanisms. The exact localization of nerve constriction is difficult due to the deep TN course. Yet, prior publications have focused on clinically and EDx-based diagnostic attempts in patients with clinical suspicion of SAS, confirmed by surgical exploration [1,3,6]. The inherent surgical risks (nerve injury or hematoma formation) without strong diagnostic findings are obvious. Imaging may further help to identify cases benefitting from surgical release.

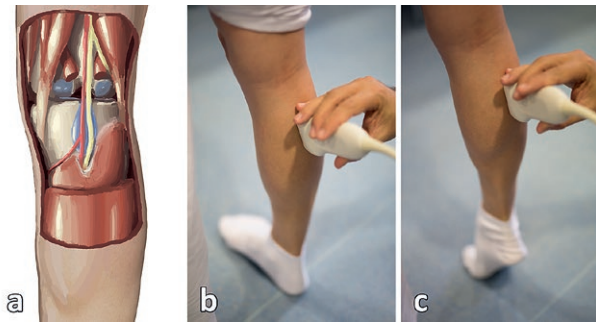
Imaging methods so far have not seen successful routine use in the diagnosis of SAS as mainly a functional pathophysiological process. Some authors have suggested the use of magnetic resonance imaging (MRI) [6–8], which can visualize focal nerve-impairment by neural swelling and muscular denervation oedema. Its rather low resolution, predefined examination region and geometry,

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**Fig 1.** Illustration of the course of the tibial nerve along the popliteal artery to the soleus arcade, where compression of the nerve occurred – complicated by a ganglionic cyst (blue colour) extending along the neurovascular bundle (a). Demonstration of the ultrasound probe positioning to examine the tibial nerve at its entry under the SA during rest (b) and during flexion of the calf muscles (c).

and impracticality of dynamic examinations [6] limit its diagnostic use. High-resolution ultrasound (HRUS) on the other hand can be tailored to the functional peculiarities in patients with TN neuropathies: compression and decompression of the TN as well as subsequent structural changes can be examined dynamically [9].

We hereby present the first case of a HRUS-based dynamic diagnosis of SAS in a 53-year old patient and propose a dynamic HRUS examination technique to corroborate the diagnosis of SAS.

### Case report

A 53-year old female patient with history of chronic diffuse pain of the left sole of her foot, which had not responded to various conventional analgetics as well as gabapentin, presented with acute worsening of symptoms and increasingly weakened plantarflexion of the toes after receiving an intraarticular corticosteroid-injection for degenerative knee pain two days prior. Clinical examination revealed the inability to flex and spread the toes; the patient reported hypaesthesias and painful dysaesthesias in the distal sensory area of the tibial nerve as well as a positive Tinel sign upon compression of the distal poplitea with aggravation of the symptoms.

Due to the recent knee intervention, a MRI (1.5 Tesla Magnetom Symphony TIM; Siemens, Erlangen, Germany) of the region was performed. Here, a large ganglion cyst was described to dorsally displace the neurovascular bundle in the popliteal fossa alongside some fluid ventral to the medial gastrocnemius head, leading to the diagnosis of a ruptured ganglionic cyst (fig 2d,e).

Due to these unsatisfactory results, HRUS was performed (Philips Epiq 7, 12-3 MHz linear-array broadband transducer; Philips, Mortsels, Belgium): substantial

swelling prior to the entry under the SA was demonstrated for the TN (fig 2a). By dynamic examination (fig 1b,c) severe constriction of the TN by the SA was seen during ankle plantarflexion with the patient standing on her tip-toes (fig 2c) with a prompt release in a neutral foot-position (fig 2b). Furthermore, ventral displacement of the neurovascular bundle was seen due to a ganglionic cyst. Thus, the diagnosis of a motion-dependent compressive TN neuropathy was established and an acute surgical intervention was performed, confirming the above findings intraoperatively. After surgical preparation and resection of the ganglion, the neurovascular bundle was found to contain a markedly swollen TN segment proximal to its entry under the SA.

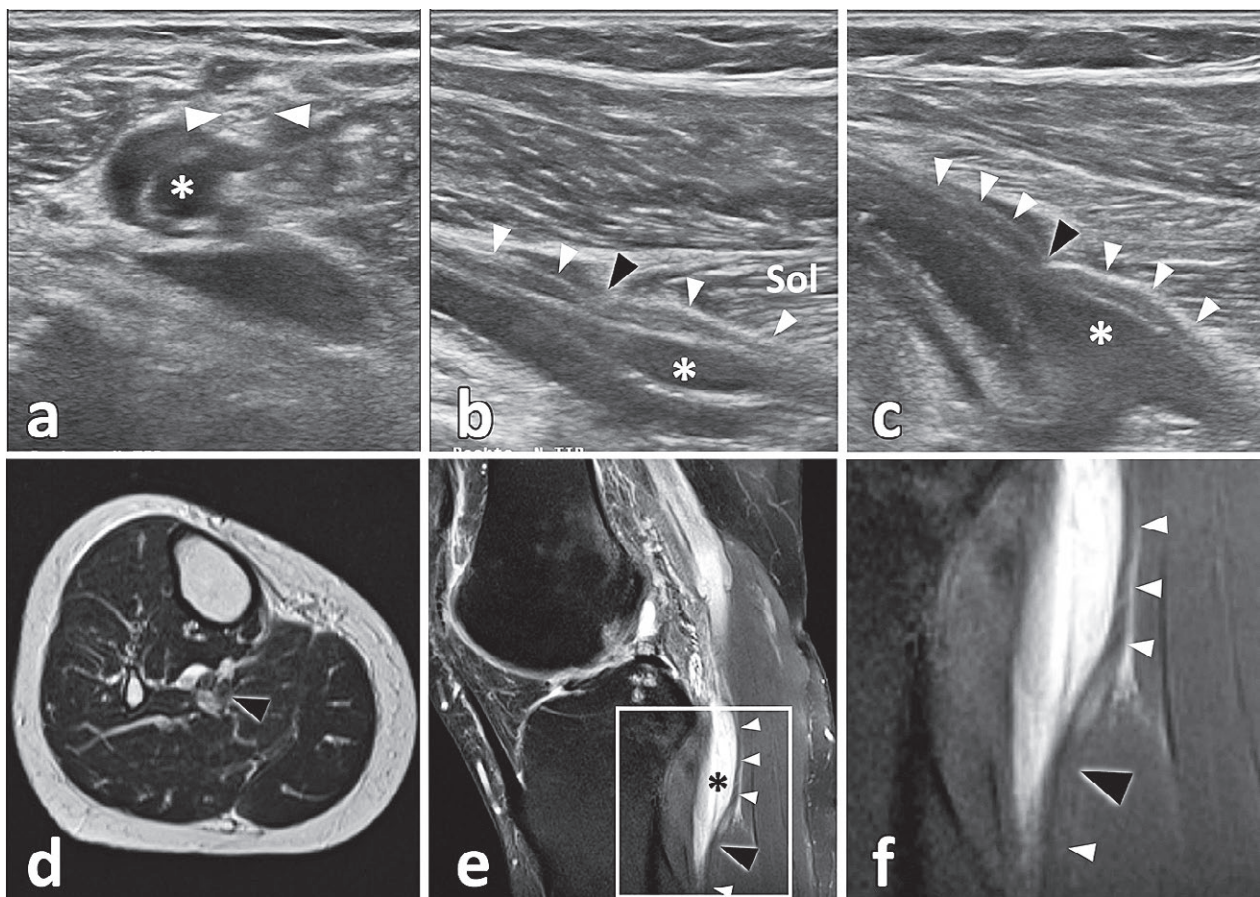
The patient reported immediate relief of specific symptoms after surgery with complete restitution of strength and reduction in pain intensity.

### Discussions

Several case reports have been published [1,10] on syndromes caused by compression of the TN by the SA [3,7]. Williams et al published a retrospective report on the surgical release of the proximal TN in 49 cases, 39 of whom with an atraumatic history including ten with proven neuropathy [11]. Most authors report on how the diagnosis was made clinically and electrophysiologically and only confirmed during surgery, while to our knowledge an *a priori* radiological definition has not been reported. Even though MRI is recommended in some publications [2] to detect Baker cysts, nerve sheath tumours or synovial cysts [2], only a few publications with a prospective focus on the diagnosis are available. Ladak et al published a retrospective report on MRI findings in nine patients with proximal TN decompression, with the causative mechanisms ranging from compression by the SA to ganglia or intraneural cysts [6].

Within this case report a special focus was put on the diagnosis of SAS by means of a focused and dynamic HRUS examination. While the patient had been suffering from longstanding chronic pain in the areas associated with the TN, the symptoms became specific only after an intraarticular injection for degenerative knee arthritis.

The diagnostic algorithm is easy to follow by any (musculoskeletal) radiologist with experience in HRUS and similar to the plethysmographic algorithm presented by Turnipseed et al for patients with atypical lower leg claudication [12]: first, the entry of the TN under the SA is located by following the TN distally. The SA is usually found around 9 cm distally to the medial tibia plateau [13], where cross-section areas and regional blurring/loss of fascicular texture of the TN can be evaluated. The TN



**Fig 2.** Axial HRUS imaging (a) of the tibial nerve demonstrates a part of the ganglionic cyst (\*) in close relation to the tibial nerve (white arrowheads). By a dynamic sagittal examination, the constriction of the nerve could be demonstrated: a minor indentation was visible in the neutral standing position (b), while the nerve showed a marked constriction by the soleus arcade (Sol) as the patient was standing in ankle plantarflexion (c). Even though a large ganglionic cyst (\*) showed broad contact with the TN, the nerve's swelling was localized just proximal to the soleus arcade, where the nerve also appeared constricted (black arrowhead). Corresponding axial 1.5 Tesla T2w TSE (d) and sagittal fat-saturated reconstructed PD TSE maximum-intensity projection MRI studies (e) of the calf at the entry of the tibial nerve (white arrowheads) under the soleus arcade (for magnification [white box] see also (f)). While the MRI findings retrospectively confirmed the diagnosis, the nerve compression was not appreciated in the initial workup.

is then longitudinally visualized using an oblique sagittal imaging plane with the patient standing in neutral position and during ankle plantarflexion. Here, compression not discernible in a relaxed position can potentially be demonstrated. Furthermore, this algorithm allows to quickly assess the popliteal fossa for other causes mimicking SAS [14]. The corresponding surgical findings were in our case in line with older reports on intraoperative findings [1].

Nonetheless, the diagnosis of SAS still requires thorough history taking and clinical examination. However, patients may present with atypical or fluctuating symptoms and may not always remember a precipitating event [1,3,10] such as trauma or prolonged physical activity involving holding positions or training [3]. Weakness may or may not be present and is not a reliable indicator

of SAS; reduced flexion and abduction of the toes was described in five out of nine patients in a case series by Mastaglia [3].

Even though the use of EDx is recommended, inconclusive EDx may be misleading as unstable nerve compression is rarely represented by means of EDx. As shown by Mastaglia [3], out of 9 cases with surgically confirmed SAS, none showed motor latency abnormalities; signal amplitudes were lowered only in 2 out of 9. Of the 4 cases that had undergone sensory conduction studies, tibial sensory action potential at the ankle was absent in only one case. Denervation findings were present in 2 out of 9 cases. Even inching of the TN – usually employed in superficial nerves of the upper extremity [15,16] – may be futile due to the TN's deep course. Furthermore, as EDx employs an exclusory approach the



EDx workup of proximal tibial neuropathy requires extensive measurements [2].

MRI examinations may in principle be used for the identification of SAS, yet two key factors reduce the viability of such a diagnostic strategy: first, MRI offers a relatively low resolution depending on the sequence and time invested in acquiring it, impeding the appreciation of neural changes such as oedema or swelling. Secondly, while dynamic musculoskeletal MRI examinations are basically feasible [17], they are rarely used in routine examinations due to planning requirements, time and patient compliance. In our patient, neural compression at the SA was not appreciated in the initial knee MRI workup and the most likely cause for the patient's symptoms was considered to be a ruptured ganglionic cyst. Furthermore, other differential diagnoses difficult to assess by MRI such as venous claudication [14] can be identified by HRUS.

Thus, HRUS appears to be useful in the workup of suspected SAS as it allows the dynamic high-resolution assessment of the TN, pinpointing the exact localisation of neural damage and identify other causative mechanisms, potentially increasing diagnostic reliability and avoiding unnecessary surgical interventions.

## References

1. Mastaglia FL, Venerys J, Stokes BA, Vaughan R. Compression of the tibial nerve by the tendinous arch of origin of the soleus muscle. *Clin Exp Neurol* 1981;18:81-85.
2. Oh SJ, Meyer RD. Entrapment neuropathies of the tibial (posterior tibial) nerve. *Neurol Clin* 1999;17:593-615.
3. Mastaglia FL. Tibial nerve entrapment in the popliteal fossa. *Muscle Nerve* 2000;23:1883-1886.
4. Loizides A, Baur EM, Plaikner M, Gruber H. Triple hour-glass-like fascicular constriction of the posterior interosseous nerve: a rare cause of PIN syndrome. *Arch Orthop Trauma Surg* 2015;135:635-637.
5. Rempel DM, Diao E. Entrapment neuropathies: pathophysiology and pathogenesis. *J Electromyogr Kinesiol* 2004;14:71-75.
6. Ladak A, Spinner RJ, Amrami KK, Howe BM. MRI findings in patients with tibial nerve compression near the knee. *Skeletal Radiol* 2013;42:553-559.
7. Chhabra A, Williams EH, Subhawong TK, et al. MR neurography findings of soleal sling entrapment. *AJR Am J Roentgenol* 2011;196:290-297.
8. Dong Q, Jacobson J, Jamadar DA, et al. Entrapment neuropathies in the upper and lower limbs: anatomy and MRI features. *Radiol Res Pract* 2012;2012:230679.
9. Sikdar S, Wei Q, Cortes N. Dynamic ultrasound imaging applications to quantify musculoskeletal function. *Exerc Sport Sci Rev* 2014;42:126-135.
10. Iida T, Kobayashi M. Tibial nerve entrapment at the tendinous arch of the soleus: a case report. *Clin Orthop Relat Res* 1997;334:265-269.
11. Williams EH, Rosson GD, Hagan RR, Hashemi SS, Dellon AL. Soleal sling syndrome (proximal tibial nerve compression): results of surgical decompression. *Plast Reconstr Surg* 2012;129:454-462.
12. Turnipseed WD, Pozniak M. Popliteal entrapment as a result of neurovascular compression by the soleus and plantaris muscles. *J Vasc Surg* 1992;15:285-294.
13. Williams EH, Williams CG, Rosson GD, Dellon LA. Anatomic site for proximal tibial nerve compression: a cadaver study. *Ann Plast Surg* 2009;62:322-325.
14. Franco A, Lachenal G, Morzol B, et al. The soleus syndrome. Apropos of 3 cases of intermittent claudication of venous origin. *Phlebologie* 1982;35:695-702.
15. Azrieli Y, Weimer L, Lovelace R, Gooch C. The utility of segmental nerve conduction studies in ulnar mononeuropathy at the elbow. *Muscle Nerve* 2003;27:46-50.
16. Campbell WW. The value of inching techniques in the diagnosis of focal nerve lesions. *Muscle Nerve* 1998;21:1554-1556.
17. Shapiro L, Harish M, Hargreaves B, Staroswiecki E, Gold G. Advances in musculoskeletal MRI: technical considerations. *J Magn Reson Imaging* 2012;36:775-787.