



Drop Foot Secondary to a Peroneal Intranural Ganglion Connected to the Superior Tibiofibular Joint: A Case Report

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Abstract

Peroneal neuropathy is the most frequent mononeuropathy of the lower extremity. Intranural ganglion cysts (INGCs) are among rare causes of peroneal nerve palsy. According to the articular (synovial) theory, the articular branch plays the key role in the pathogenesis. Patients present with pain around the fibular head and neck, motor weakness resulting in foot drop and paresthesia in the anterolateral calf and foot. Ultrasonography (US) and MRI are both useful in the diagnosis, but MRI is the best imaging modality in the demonstration of the articular connection and the relation of the cyst with adjacent structures, even without special neurography sequences. We present a 32-year-old male patient referred to our neurosurgery clinic with suspicion of lumbar radiculopathy. He presented with right foot drop which began 3 weeks prior. On examination, there was 90% loss in the ankle dorsiflexion and finger extension. Ankle eversion was also weakened. There was no low back or posterolateral thigh pain to suggest L5 radiculopathy and sciatic neuropathy. Following negative lumbar spine MRI, peripheral neuropathy was concerned. Electrodiagnostic evaluations findings were consistent with acute/subacute common peroneal nerve (CPN) axonal neuropathy. Subsequent MRI of knee showed a homogeneous, thin-walled tubular cystic lesion, extending along the course of the CPN and its articular branch. Full recovery of the neuropathy was achieved with early diagnosis and decompression via microsurgical epineurotomy. The diagnosis of INGC was confirmed by histopathologic examination. INGCs, although rare, should also be considered in the differential diagnosis of peripheral mononeuropathies.

Keywords

- ▶ foot drop
- ▶ intraneural ganglion cyst
- ▶ peroneal neuropathy

Introduction

Peroneal neuropathy is the most frequent mononeuropathy encountered in the lower extremity and is the third most common focal neuropathy after median and ulnar neuropathies.¹ It is most often caused by direct traumatic injury or

entrapment of peroneal nerve, especially at the level of fibular head and neck, where the nerve is quite superficial. Ganglion cysts, which are also referred simply as ganglia, are among the rare causes of peroneal nerve palsy. They are benign non-neoplastic cystic lesions containing mucinous fluid within a thin connective tissue capsule. They are thought to be

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first described by Hippocrates as knots of tissue containing mucoïd flesh. Although they are indistinguishable from synovial cysts on imaging, they are differentiated with the absence of synovial lining histologically. Ganglia can occur in various locations in the body, but they are most common in the hand or wrist (70–80%), more specifically at the dorsum of the wrist.² They are named according to their associated structures. If a ganglion cyst occurs within the epineurium of a peripheral nerve, it is called intraneural ganglion cyst (INGC). Various etiologies are suggested for their formation, but the articular (synovial) etiology proposed by Spinner et al, which emphasizes the key role of the articular branch, is the leading theory.³ According to this theory, a capsular defect in the adjacent joint allows synovial fluid to enter the epineurial sheath of the articular branch, which extends proximally by dissecting the epineurium along the path of least resistance. They stated that the injury of the joint is the main initiating factor in the development of these ganglion cysts.^{4,5} Patients present with signs and symptoms of peripheral neuropathy due to displacement and compression of the nerve fascicles. In advanced cases, denervation and atrophy in the muscles innervated by the affected nerves can occur. Positive Tinel's sign at the area of the ganglion cyst is commonly seen. Electrophysiological studies help to determine the level and the type of the injury. The diagnosis is based on MRI, which allows detection of the intraneural ganglia and its differentiation from other intra or juxtaneural cystic lesions. Once the diagnosis of an INGCs are made, early surgical decompression is standard treatment, because, when performed early, signs and symptoms are usually reversible. We report a case of a 32-year-old male patient with a 3-week history of foot drop caused by an intraneural ganglion of common peroneal nerve (CPN). Full recovery of the neuropathy was achieved with early diagnosis and immediate decompression of the nerve.

Case Presentation

In April 2018, a 32-year-old male patient who presented with right foot drop, which began 3 weeks prior, was referred

to our neurosurgery outpatient clinic by his general practitioner with suspicion of lumbar radiculopathy. In addition to foot drop, there was pain in the right knee and right lateral calf. In addition, paresthesia was present in the anterolateral part of the right lower leg and foot. He denied any recent history of trauma. On physical examination, there was obvious motor weakness in the right foot, with 90% loss in the ankle dorsiflexion and finger extension. Ankle eversion was also weakened. He denied any low back or posterolateral thigh pain to suggest L5 radiculopathy and sciatic neuropathy. Following negative lumbar spine MRI, which showed only diffuse mild bulging and annular tear at L5-S1 intervertebral disc (►Fig. 1), peripheral neuropathy was concerned. At second look, tenderness and positive Tinel's sign at the level of fibular head and neck was revealed. There were no signs of atrophy in the leg muscles. Plantar flexion and ankle inversion were normal. Patellar and Achilles reflexes were normoactive and the Babinski sign was negative (normal flexor plantar response). There was hypoesthesia in the peroneal sensory area. Electrodiagnostic evaluations were performed and showed decreased nerve conduction velocity, motor amplitude and denervation potentials in the right extensor hallucis longus, anterior tibial and peroneus longus muscles. Overall findings were consistent with acute/subacute focal neuropathy of CPN associated with partial axonal loss. Subsequent MRI of the knee showed a homogeneous, thin-walled multilocular tubular cystic lesion, extending along the course of the CPN and its articular branch. Its length was approximately 9 cm and thickness was 6.3 mm. It was hypointense on T1-weighted images and hyperintense on T2-weighted images, having a few septations (►Fig. 2). The articular extension from the superior tibiofibular (STF) joint was demonstrated (►Fig. 3). Following administration of IV gadolinium, thin peripheral enhancement was seen (►Fig. 4). In addition, diffuse hyperintensity associated with acute denervation was present in the anterior tibial and extensor digitorum muscles located at the anterior compartment of the lower leg (►Fig. 5). Findings were considered as compatible with INGC of CPN.

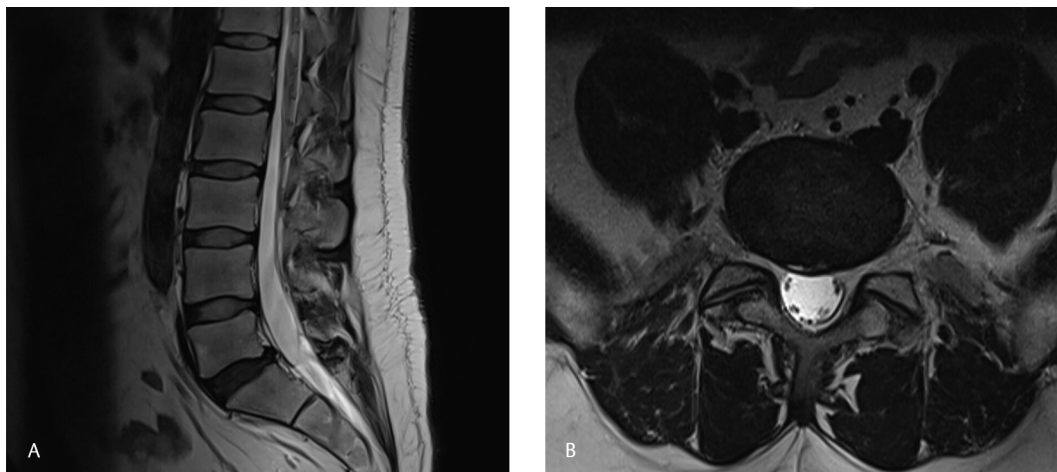


Fig. 1 Lumbar spine MRI (A) sagittal T2-weighted and (B) axial T2-weighted images showing diffuse mild bulging and annular tear at L5-S1 intervertebral disc.

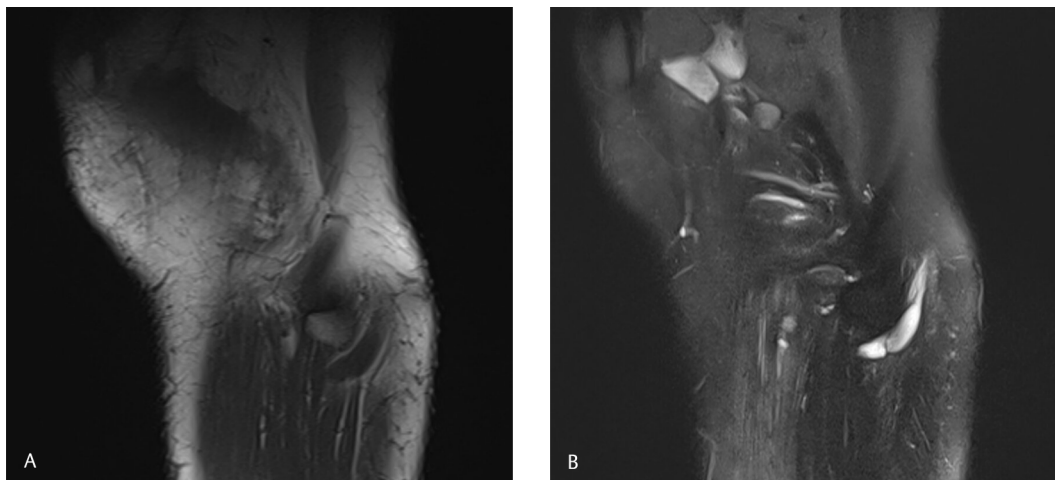


Fig. 2 (A) Sagittal T1-weighted MRI and (B) sagittal T2-weighted MRI of the right knee at the level of the fibular head and neck. The cystic tubular lesion within the common peroneal nerve (CPN), which is hypointense on the T1-weighted image and hyperintense on the T2-weighted image, with a few septations.

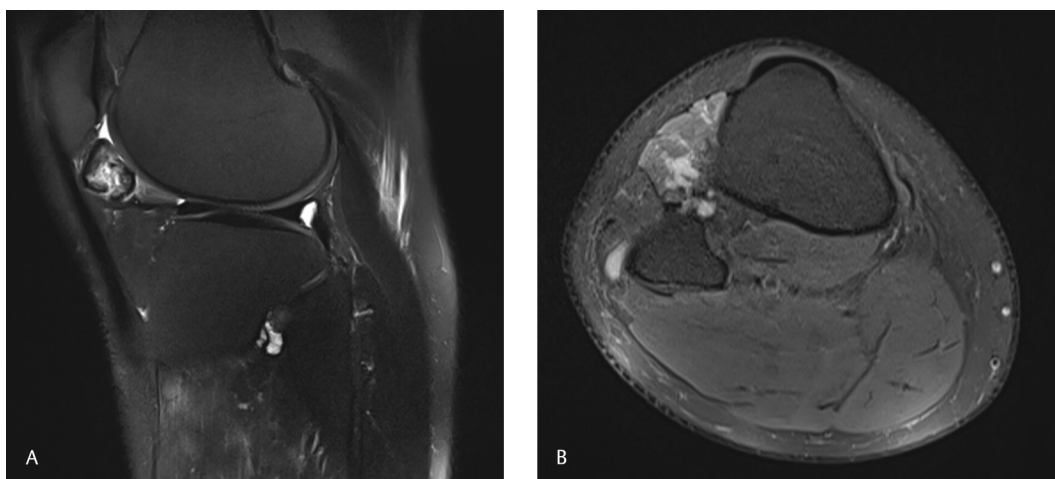


Fig. 3 The articular extension of the cystic lesion from the superior tibiofibular (STF) joint on (A) the sagittal T2-weighted image and on (B) the axial proton density (PD) fat-saturated image.

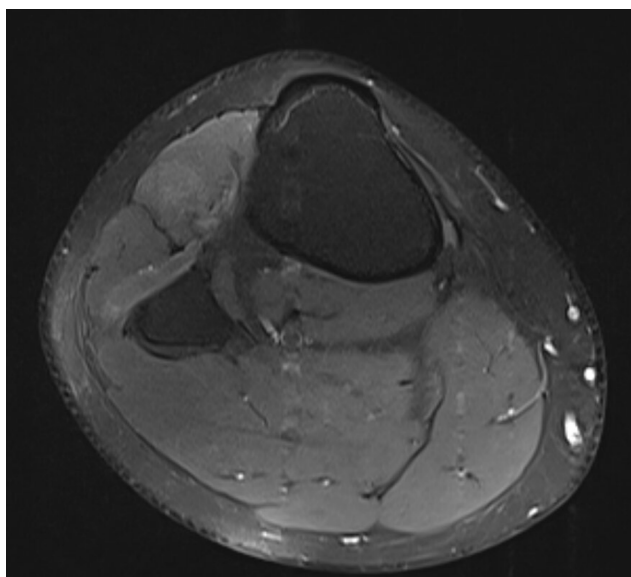


Fig. 4 Axial T1-weighted fat-saturated image following administration of IV gadolinium showing thin peripheral brightness of the cystic lesion.

Surgery

Decompression via microsurgical epineurotomy was performed under spinal anesthesia in the supine position. After making a linear longitudinal incision, extending distally from the head of the right fibula, the skin and subcutaneous tissue and the fascia were crossed and the CPN was identified. It was expanded and edematous. The mucinous material was drained via an epineurotomy. With the resection of the sheath of the cyst, it was removed totally. To prevent recurrence, the articular branch of CPN was primarily ligated and resected. Following bleeding control, the layers were primarily sutured and the operation was terminated. No postoperative complication was developed. The diagnosis of INGC was confirmed by histopathologic examination, showing cystic formation within the neural tissue (► Fig. 6). The patient was discharged with recommendations and intensive physiotherapy program. Successful results were obtained with almost full recovery of the neuropathy, with clinical and EMG evidence 3 months after the operation. Follow-ups with telephone interviewing

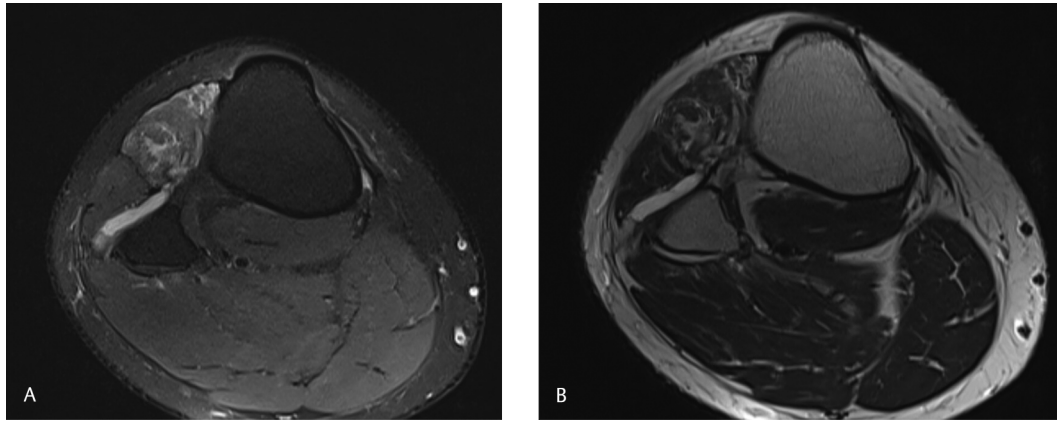


Fig. 5 Diffuse heterogenous hyperintensity due to acute denervation in the anterior tibial and extensor digitorum muscles on (A) the axial proton density (PD) fat-saturated image and on (B) the axial T2-weighted image.

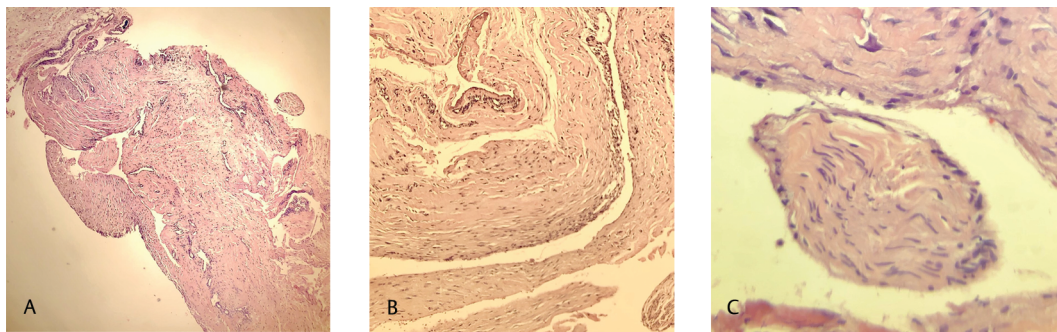


Fig. 6 Histological section shows (A) irregular cystic spaces within the neural tissue. (B) cystic formation within the neural tissue (c) an island of neural tissue within the cystic formation.

was continued at 6-month intervals over the course of 2 years, and any recurrence of the symptoms did not occur.

*Written informed consent was obtained from the patients for publication of this case report.

Discussion

INGCs have been reported to affect many peripheral nerves around the synovial joints, but the most common location is the CPN and its branches.⁶ It is the lateral branch of the sciatic nerve, which courses from the posterolateral side of the knee and the fibular head to the anterolateral side of the lower leg. When exiting the fibular tunnel, it typically trifurcates in the deep and superficial peroneal nerves and a smaller articular branch. The deep peroneal nerve (DPN) provides motor innervation to the anterior compartment muscles, which are responsible for foot dorsiflexion and toe extension. The superficial peroneal nerve (SPN) supplies ankle evertors located in the lateral compartment. The smaller articular branch provides sensory information from the STF and serves as a conduit in the formation of the intraneural ganglia.³ The event begins with the degenerative or traumatic capsular defects of the STF joint, which allows transmission of synovial fluid into the articular branch with formation of a cyst inside the epineural sheath, having a tendency to extend proximally, following the path of the least resistance up to the CPN and sciatic bifurcation.⁷ From there, the tibial nerve can also be

potentially affected as a secondary descending pathway.^{8,9} In a retrospective analysis of MRIs of 245 patients presenting with different peripheral nerve palsies, 13 INGCs were established and seven of these were associated with CPN. An intra-articular connection was established in 12 out of these 13 lesions.¹⁰ In another systematic review of the literature, a joint connection in 27 of 79 case reports was retrospectively confirmed, which were previously unrecognized.¹¹ Patients present with pain around the fibular head and neck, and various degrees of motor weakness of the anterior and lateral compartment muscles associated with paresthesia in the anterolateral part of the calf and foot.^{11,12} Obvious motor weakness resulting in foot drop may occur like in our case. The onset of symptoms is usually gradual, but presentations with acutely developing foot drop has also been reported.¹³ Our patient presented with gradually worsening foot drop with 3 weeks of history. In the differential diagnosis, other pathological conditions, including L5 radiculopathy, sciatic neuropathy and, less frequently, lumbosacral plexopathy should be excluded. This differentiation is important because early surgical decompression is the main factor in achieving successful outcomes in cases of INGCs. On clinical examination, careful evaluation of lower extremity muscles and sensory areas is very helpful in the determination of the approximate localization of the pathology. Weakness in hip abduction suggests a pathology that is proximal to the sciatic nerve. In addition, since coexistent presence of impairment in

the plantar flexion and foot inversion shows involvement of the tibial nerve, the pathology is thought to be proximal to the sciatic nerve bifurcation. Moreover, because the only muscle innervated by the peroneal nerve above the knee is the biceps femoris muscle, coexistent weakness of this muscle also suggests a more proximal lesion.¹⁴ In our patient, there was no complaint of low back or posterolateral thigh pain to suggest L5 radiculopathy. Plantar flexion and ankle inversion were normal. Electrodiagnostic evaluation, including motor and sensory nerve conduction studies (NCSs) and electromyography (EMG) of the relevant muscles, is used to distinguish peroneal neuropathy from other disorders presenting with weakness of the ankle dorsiflexion. On needle EMG, at least two muscles innervated by the DPN: anterior tibial muscle, extensor hallucis longus, at least one muscle innervated by the SPN: peroneus longus, peroneus brevis muscles, the short head of the biceps femoris, and one muscle innervated by the tibial nerve: posterior tibial muscle should be evaluated. If there is abnormality in these muscles, the examination should be extended to exclude a more proximal pathology. In addition, these testings are also useful in planning the treatment by establishing the potential for recovery. However, there are some limitations in these testings. They are relatively invasive and operator-dependent. They do not provide a complete examination of a muscle, and the deeply located muscles are difficult to evaluate. In addition, denervation in the earliest phases is not revealed.¹⁵ In radiological imaging of INGCs, ultrasonography (US) and MRI are both useful in the diagnosis. On US, they appear as anechoic or hypoechoic well-margined cystic lesions with frequent septations as well as acoustic enhancement.^{16,17} MRI, even without special neurography sequences, is the best imaging modality in the demonstration of these lesions along the course of the nerve and the relation of the cyst with adjacent structures. It can reliably demonstrate the articular connection in many cases.¹⁸ Spinner et al demonstrated tail sign (connection of the cyst to the STF joint) as a MRI feature in the identification of the joint connection.¹⁹ In addition, denervation changes in the affected muscles are shown as secondary findings of the peripheral neuropathy. Therefore, it is suggested that knee MRI should be performed in all cases of non-traumatic peroneal nerve palsy.²⁰ Axial T1-weighted images are the best in evaluation of the CPN in routine MRI and 3 tesla MRI is much more useful in neural examinations.³ INGCs appear as elongated uni- or multilocular cystic lesions within the nerve. They are generally hypointense on T1-weighted images, although high-protein content or hemorrhage may result in increased signal intensity. On T2-weighted/proton density (PD) images, they are typically hyperintense. They often show no contrast enhancement, however some amount of peripheral brightness may be present, like in our case.²¹ On MRI, muscles innervated by the affected nerves should be evaluated for denervation changes. In acute stages, denervation edema is seen as hyperintense on short tau inversion recovery (STIR) and on fat-suppressed T2-weighted/PD images. In later stages, if nerve conduction has not been restored, atrophy occurs after approximately 7 days. It is still a reversible finding but indicates that these muscles are at

continued risk of developing fatty replacement, which occurs in longer standing denervation. It is best visualized on axial nonfat suppressed T1-weighted images.¹⁸ This differentiation is very important, because fatty replaced fibers are usually inevitably lost, and they do not recover after surgical nerve repair.²² This classical anatomical location within the nerve and the characteristic articular communication with adjacent joints allow differentiation of INGCs from other intra or juxta-neural cystic lesions, such as a cystic schwannoma or extraneural ganglion cysts (ENGCS). Intraneural cystic necrosis may rarely occur in large and longstanding schwannomas and may be confused with INGCs showing peripheral enhancement.²¹ Extraneural ganglia, which are located adjacent to a peripheral nerve but outside the epineural sheath, should also be differentiated. When they are large enough, they may compress the nerve and cause similar clinical findings. INGCs usually present with more pronounced clinical and electromyographic findings because in an INGC, the perineurium restricts the expansion of the ganglia, which increases the pressure on the nerve fascicles, even when the cyst is small in size. On imaging, while INGCs form a tubular-shaped appearance along the course of the nerve, ENGCS are seen in ovoid or rounded form. In addition, Spinner et al demonstrated transverse limb sign (cyst within the articular branch of the CPN) and signet ring sign (cyst within the CPN) at the STF joint for differentiating between INGCs and ENGCS.¹⁹ The treatment of INGCs involves early surgical decompression of the nerve with excision of the ganglion cyst. Conservative treatment is not recommended, because functional recovery would not be seen due to the enlarging cyst. It has been reported that to have successful surgical outcomes, decompression should be performed within 4 months of the appearance of the foot drop.²³ In our patient, the diagnosis of the INGC was established on knee MRI at the day of presentation, 3 weeks after the appearance of foot drop, and surgical decompression was performed 2 days later. In the surgical decompression, the articular disconnection and the treatment of the articular pathology is important in prevention of intraneural cyst recurrence.^{3,11,24} In our patient, surgical decompression via complete excision of the cyst as well as disconnection of the articular extension was achieved. Sensory loss and motor functions were almost fully recovered 2 months after the operation. No recurrence was observed at 2 years postoperatively.

Conclusion

INGCs, although rare, should also be considered in the differential diagnosis of peripheral mononeuropathies. Their differentiation from other more common pathologies that may present with similar clinical findings is important, because early surgical decompression is the main factor in the recovery of the neuropathy. Careful physical examination and electrodiagnostic studies are helpful in directing the appropriate radiological imaging. MRI, even without special neurography sequences, is the best imaging modality in the demonstration of the articular connection and the relation of the cyst with adjacent structures. Therefore, knee MRI should be

performed in the evaluation of nontraumatic peroneal neuropathy, and articular connection should be demonstrated. To prevent intraneural cyst recurrence, articular disconnection and the treatment of the articular pathology are important in the surgical decompression of the nerve.

Ethical Approval

Ethics committee approval was not needed. Written informed consent was obtained from the patient.

Funding

None.

Conflict of Interest

None declared.

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