INTRANEURAL GANGLION CYST OF THE COMMON PERONEAL NERVE PRESENTING WITH FOOT DROP: A RARE CASE REPORT AND REVIEW OF THE LITERATURE

Jaimin Modh, Arvind Verma, Renish Padshala,Nazar Imam

Department of Neurosurgery, Smt. NHL Municipal Medical College, Ahmedabad, Gujarat, India.

ABSTRACT

Background: Intraneural ganglion cysts are rare benign mucinous lesions that arise within the epineurium of peripheral nerves. They represent an uncommon but surgically treatable cause of common peroneal neuropathy. Because their clinical presentation may mimic lumbar radiculopathy, entrapment neuropathy, or peripheral nerve sheath tumour, significant diagnostic delay is frequent.

Case Presentation: We report a 35-year-old man who presented with progressive foot drop and gait impairment. Magnetic resonance imaging (MRI) demonstrated a cystic lesion along the common peroneal nerve at the fibular neck, producing focal neural compression. Microsurgical exploration through a lateral approach was performed. The common peroneal nerve was identified, decompressed, and preserved. Intraoperative neurophysiological monitoring—including motor evoked potentials (MEPs) and direct nerve stimulation—guided safe fascicular dissection. A translucent cystic lesion containing gelatinous material was evacuated and excised. Histopathological examination confirmed a fibrous cyst wall with mucinous contents and no epithelial lining or malignant features, consistent with a ganglion cyst. The postoperative course was uneventful; foot drop resolved completely, and the patient regained normal independent walking.

CONCLUSION

This case highlights the importance of considering intraneural ganglion cyst in young adults with non-traumatic foot drop. Early MRI diagnosis, meticulous nerve-preserving microsurgery, and neurophysiological monitoring may permit excellent functional recovery. The articular/synovial theory of pathogenesis underscores the need to identify and address the joint connection and articular branch to reduce recurrence.

Keywords: Evoked Potentials, Motor; Foot Drop; Ganglion Cysts; Microsurgery; Monitoring, Intraoperative; Peroneal Nerve; Treatment Outcome

INTRODUCTION

Foot drop is a clinically important neurological deficit characterised by weakness of ankle and toe dorsiflexion, producing steppage gait, impaired foot clearance during the swing phase, an increased risk of falls, and major functional limitation. The responsible lesion may arise at multiple levels of the motor pathway, from the cerebral cortex, spinal cord, and lumbosacral nerve roots to the sciatic nerve, common peroneal nerve (CPN), deep peroneal nerve, neuromuscular junction, or anterior compartment musculature. In clinical neurosurgical practice, the most relevant peripheral diagnoses include L4–L5 radiculopathy, sciatic neuropathy, CPN entrapment at the fibular neck, traumatic nerve injury, compressive neuropathy from prolonged positioning, periarticular mass lesions around the knee, and iatrogenic injury after orthopaedic procedures [1].

The CPN is uniquely vulnerable at the fibular neck. It courses superficially around the lateral knee, becomes tethered near the fibular head, and bifurcates into the superficial and deep peroneal branches within a fibro-osseous tunnel near the peroneus longus origin. Even modest extrinsic compression at this site can produce weakness of ankle dorsiflexion, toe extension, and eversion, with sensory disturbance over the anterolateral leg and dorsum of the foot. Structural lesions must be actively considered when foot drop is progressive, atraumatic, associated with a palpable swelling, or imaging-positive at the fibular neck.

Intraneural ganglion cysts are rare benign lesions that develop within the epineurium and are filled with mucinous or gelatinous material. They are now understood to be joint-related rather than true neoplasms. The CPN is the most frequently affected peripheral nerve. In the largest systematic review, Desy et al. analysed 417 articles encompassing 645 patients and identified increasing recognition of articular connections and recurrence mechanisms over time [2]. A subsequent imaging analysis by Lenartowicz et al. catalogued 941 reported cases, confirming the CPN as the predominant site [3].

The prevailing pathogenic framework is the articular (synovial) theory: synovial fluid escapes from a neighbouring joint through a capsular defect, enters an articular branch of the nerve, and propagates proximally within the epineurium of the parent nerve. For CPN lesions, the source is typically the proximal (superior) tibiofibular joint [4,5]. This model has direct surgical implications—simple cyst decompression without addressing the articular branch and joint connection is associated with higher recurrence rates.

MRI is central to diagnosis and operative planning. Characteristic features include a T1-hypointense, T2-hyperintense cystic lesion following the nerve, sometimes exhibiting the tail sign, transverse limb sign, or signet-ring sign [3,6]. High-resolution ultrasonography may complement MRI, particularly for dynamic assessment and postoperative surveillance [7]. Despite these advances, diagnostic confusion persists because the lesion may be mistaken for schwannoma, neurofibroma, extraneural ganglion, Baker cyst, or lumbar radiculopathy.

The purpose of this article is to report a rare case of CPN intraneural ganglion cyst presenting as foot drop in a young adult male, successfully managed by nerve-preserving microsurgical excision under intraoperative neurophysiological monitoring, and to review the literature with emphasis on pathophysiology, diagnostic pitfalls, imaging signs, surgical

strategy, recurrence prevention, and expected neurological recovery.

Case Description

A 35-year-old male presented with a progressive history of foot drop. He reported increasing difficulty clearing his foot during the swing phase of gait, resulting in a characteristic steppage pattern and significant functional limitation of independent ambulation. There was no history of major trauma, penetrating injury, recent orthopaedic intervention, or known systemic neurological disease. The symptom onset was insidious, and the deficit had worsened over the months preceding presentation.

On clinical examination, the patient demonstrated foot drop consistent with CPN dysfunction. Ankle dorsiflexion and toe extension were weak; plantar flexion was preserved. Gait assessment confirmed impaired foot clearance during swing. Sensory examination revealed diminished sensation over the anterolateral leg and dorsum of the foot in the distribution of the superficial peroneal nerve. Preoperative muscle strength grading (Medical Research Council scale) and formal electrodiagnostic data should be inserted from the clinical record prior to journal submission.

MRI of the proximal leg demonstrated a well-defined, T1-hypointense and T2-hyperintense cystic lesion along the anatomical course of the CPN at the fibular neck—the region where the nerve winds around the fibular head and enters the peroneal tunnel (Figure 2). The lesion compressed and distorted the nerve, with no evidence of solid components or internal enhancement to suggest a nerve sheath tumour. Its cystic morphology, anatomical location, and relationship to the CPN supported the primary diagnosis of intraneural ganglion cyst; a cystic peripheral nerve sheath tumour remained a secondary differential pending histopathological confirmation.

Given the progressive motor deficit, gait impairment, and imaging evidence of focal nerve compression, surgery was recommended. The operative goals were decompression of the CPN, evacuation and excision of the cystic lesion, preservation of functional fascicles, prevention of iatrogenic nerve injury, and restoration of neural function.

The patient was positioned to expose the lateral proximal leg and fibular neck. A lateral skin incision was planned over the fibular head, aligned with the course of the CPN. Dissection proceeded in layers; subcutaneous tissues were divided with care to avoid traction on superficial neural structures. The operative field was maintained with self-retaining retractors.

Under magnification, the CPN was identified proximally in undisturbed tissue and traced distally toward the fibular neck. Vessel loops were placed for gentle mobilisation and protection. A cystic rather than solid lesion was encountered, intimately related to the nerve; the nerve appeared expanded and distorted by a translucent component containing gelatinous material, consistent with an intraneural ganglion cyst (Figure 3, Figure 4).

Continuous intraoperative neurophysiological monitoring was employed throughout. MEPs and direct electrical stimulation were used to identify functional fascicles and to distinguish viable neural tissue from the non-functional cyst wall. This was critical given the intimate relationship between the lesion and the nerve, where inadvertent fascicular injury could have produced permanent dorsiflexion weakness.

Microsurgical decompression was performed along a nerve-preserving dissection plane. The cystic component was carefully opened; gelatinous material was evacuated and the cyst excised as completely as safely achievable without sacrifice of functioning fascicles. Following decompression, the nerve appeared adequately released with no residual focal compression. Haemostasis was achieved, and the wound was closed in anatomical layers.

Histopathological examination of the excised specimen demonstrated a fibrous cyst wall with mucinous material, no epithelial lining, and no malignant features, confirming the diagnosis of ganglion cyst (Figure 5). The postoperative course was uneventful. No new neurological deficit was observed. Ankle dorsiflexion and gait improved progressively; foot drop resolved clinically, and the patient regained normal independent walking. No wound complication, infection, or early postoperative deterioration was documented. The patient was advised to continue neurological follow-up to monitor recovery and detect any radiological recurrence.

DISCUSSION

Rarity and Clinical Significance

Intraneural ganglion cyst of the CPN is a rare but surgically reversible cause of foot drop that deserves early recognition. Unlike foot drop due to degenerative lumbar pathology or traumatic peroneal nerve injury—which may produce irreversible axonotmesis or neurotmesis—intraneural ganglion cysts typically cause pressure-mediated neuropathy without complete axonal disruption. Timely surgical decompression before irreversible axonal loss is therefore associated with excellent neurological recovery.

Diagnostic delay is common because the entity is unfamiliar to many clinicians. Patients with foot drop are frequently referred for lumbar spine evaluation first, particularly when incidental degenerative disc disease is present on imaging. Young et al. demonstrated that peroneal intraneural ganglia can present with pain, fluctuating weakness, and sensory symptoms localising to the CPN—features that, if recognised, should prompt peripheral nerve-directed imaging rather than further spinal workup [8]. The present case reinforces this principle: a young man with focal pathology at the fibular neck recovered fully after microsurgical decompression that would not have been offered if the deficit had been attributed reflexively to lumbar disease.

Pathophysiology: The Articular (Synovial) Theory

The modern understanding of intraneural ganglion cysts rests on the articular theory. Earlier hypotheses—de novo mucinous degeneration within the nerve, traumatic origin, or embryological rests—failed to explain the consistent anatomical relationship between intraneural cysts and adjacent synovial joints or the recurrence patterns seen after isolated cyst evacuation.

The articular theory proposes that synovial fluid escapes from a joint via a capsular defect and enters an articular branch of the peripheral nerve. Hydrostatic pressure then drives the fluid proximally within the epineurium of the parent nerve. For CPN lesions, the relevant source is usually the superior

tibiofibular joint; the articular branch arising from the CPN or deep peroneal nerve provides the anatomical conduit. Progressive fluid accumulation expands the epineurium, displaces and compresses fascicles, and produces the clinical deficit.

Spinner et al. established the primacy of the articular branch through clinical series and a unifying theoretical analysis [4,5], shifting management from simple cyst evacuation toward interruption of the joint–nerve fluid pathway. Desy et al. confirmed in 645 patients that recurrence correlates with percutaneous aspiration and failure to disconnect the articular branch or treat the joint [2]. Lenartowicz et al. further demonstrated that contemporary MRI identifies joint connections more reliably than ultrasonography and that their detection is increasing with improved imaging protocols [3].

Practically, this model means that the lesion is not a neoplasm, that decompression alone may not be curative, and that high-resolution preoperative imaging should be scrutinised specifically for the joint connection—not merely the dominant cyst mass.

Anatomical Vulnerability of the Common Peroneal Nerve

The CPN is predisposed to symptomatic compression at the fibular neck for anatomical reasons: it is superficial, tethered proximally and distally, and traverses a fibro-osseous tunnel near the peroneus longus origin. Even modest cyst expansion may therefore produce significant neural compression. Deep peroneal fascicles, which subserve ankle and toe dorsiflexion, are often disproportionately affected, explaining presentations dominated by foot drop; involvement of superficial peroneal fascicles adds eversion weakness and sensory symptoms over the dorsum of the foot.

The characteristic MRI signs reflect this anatomy. The "tail sign" suggests continuity with the joint source; the "transverse limb sign" represents cystic material within the articular branch as it crosses the anterior fibular neck; and the "signet-ring sign" reflects eccentric fascicular displacement within the epineurium [6]. Recognition of these signs can reliably distinguish intraneural ganglion cyst from extraneural ganglion and from peripheral nerve sheath tumour, both of which require different operative strategies.

Diagnostic Challenges and Differential Diagnosis

Because foot drop is a sign rather than a diagnosis, a structured clinico-anatomical approach is required before attributing it to any single cause. The distribution of motor weakness, pattern of sensory loss, reflex changes, and presence or absence of upper motor neuron signs help localise the responsible lesion. L5 radiculopathy may closely mimic CPN neuropathy because both impair ankle dorsiflexion; however, radiculopathy typically also affects hip abduction and foot inversion, may be accompanied by back pain, and produces paraspinal denervation on electromyography. CPN neuropathy selectively impairs dorsiflexion and eversion while sparing inversion (tibialis posterior is tibial-nerve innervated). Sciatic neuropathy additionally involves hamstrings and tibial-nerve muscles, and central causes produce upper motor neuron signs.

At the fibular neck, the structural differential diagnosis encompasses entrapment neuropathy, traumatic neuroma, extraneural ganglion cyst, intraneural ganglion cyst, schwannoma, neurofibroma, malignant peripheral nerve sheath tumour, lipoma, vascular malformation, synovial cyst, Baker cyst extension, and soft tissue sarcoma. Cystic peripheral nerve sheath tumours may exhibit heterogeneous enhancement or solid components; ganglion cysts typically display uniform fluid signal. Panwar et al. emphasised that intraneural ganglion cysts are frequently under-recognised and stressed that identifying the joint connection is central to correct diagnosis [9]. In the present case, the lesion was initially considered a peripheral nerve tumour; the operative approach—and therefore the outcome—would have differed substantially if this misclassification had not been corrected by careful MRI review.

Role of Electrodiagnosis and Neuroimaging

Electrodiagnostic testing localises the lesion, quantifies severity, and distinguishes demyelinating conduction block from axonal loss—information directly relevant to prognosis and operative urgency. Nerve conduction studies in CPN intraneural ganglion cyst may show reduced compound muscle action potential amplitude, focal slowing, or conduction block at the fibular head. Needle electromyography typically demonstrates denervation in tibialis anterior, extensor hallucis longus, extensor digitorum brevis, and peroneal muscles, with normal findings in tibialis posterior and paraspinal muscles—a pattern that effectively excludes both L5 radiculopathy and sciatic neuropathy [8].

MRI remains the most informative preoperative modality, defining cyst extent, nerve displacement, the presence of muscle denervation changes, and the possible joint connection. Among 941 cases reviewed by Lenartowicz et al., MRI was more likely than ultrasonography to detect joint connections, a finding critical to planning recurrence-prevention surgery [3]. High-resolution ultrasound usefully complements MRI through dynamic evaluation, easy comparison with the contralateral side, and convenient postoperative follow-up [7], though it is operator-dependent and less sensitive for subtle joint connections.

Surgical Management Principles

The surgical objectives are nerve decompression, cyst evacuation and excision, fascicular preservation, management of the articular branch when identified, and reduction of recurrence risk. A safe strategy begins with exposure of the CPN proximal to the lesion in undistorted tissue, followed by distal tracing toward the fibular neck and peroneal tunnel. External neurolysis releases compressive fascial structures. The cystic region is inspected under magnification; dissection avoids blind coagulation, aggressive traction, and circumferential nerve stripping. Direct stimulation identifies functioning fascicles and guides the choice of a safe entry zone for epineurial opening if required.

Intraoperative neurophysiological monitoring was employed throughout the present case. MEPs and direct stimulation proved particularly valuable for identifying functional fascicles within the distorted intraneural anatomy, allowing decompression to proceed without permanent dorsiflexion loss. Although the literature on routine neurophysiological monitoring specifically for peroneal intraneural ganglion cysts is limited, the rationale is compelling in cases with preoperative motor deficit and intraneural pathology.

The articular branch must be specifically sought. When identified, most authorities recommend ligation or transection

to interrupt the synovial fluid conduit [4,5,10,11]. For first presentations without significant joint degeneration, decompression combined with articular branch disconnection is usually sufficient. For recurrent or large cysts with prominent proximal tibiofibular joint pathology, more targeted joint-directed treatment—such as synovial resection or joint curettage—may be necessary. The literature consistently warns that cyst evacuation alone, without addressing the articular connection, predisposes to recurrence [2–5,10,11].

Outcomes and Recurrence in the Literature

Functional recovery after microsurgery is generally favourable when the deficit is treated before prolonged axonal degeneration. Wilson et al. reported outcomes in 65 surgically treated patients; median dorsiflexion improved from 2/5 preoperatively to 5/5 at last follow-up, and radiological recurrence occurred in only 6 patients, all extraneural [12]. König et al. similarly demonstrated significant motor recovery and used postoperative nerve ultrasonography to correlate morphological changes with clinical outcomes [13]. Kankam et al. concluded from a case series and systematic review that surgical excision combined with articular branch neurectomy was associated with improved motor and sensory function [15].

The present case aligns with these favourable outcomes. Complete resolution of foot drop and restoration of independent walking after microsurgical decompression suggest that the preoperative deficit was predominantly compressive and reversible. Despite this excellent result, long-term recurrence surveillance remains important, particularly if the status of the articular branch and proximal tibiofibular joint connection was not definitively documented or treated intraoperatively.

Joint-directed procedures carry additional considerations, as Wilson et al. demonstrated when evaluating joint stability after treatment of the superior tibiofibular joint source [14]. Balancing recurrence prevention with preservation of joint function is important; more aggressive joint management is generally reserved for recurrent lesions or cases with prominent joint pathology.

Comparison with Similar Reported Cases

Several recent reports illustrate the clinical spectrum. Broekx et al. described a 12-year-old child with CPN intraneural ganglion cyst causing foot drop, emphasising the importance of articular branch ligation and transection in minimising recurrence [16]. Pemmari et al. reported a large intraneural cyst in the peroneal nerve and highlighted persisting under-recognition of this entity in cases of unexplained peroneal palsy [17]. Della Vecchia et al. described acute non-traumatic foot drop due to peroneal intraneural ganglion cyst and reviewed the diagnostic-therapeutic pathway [18]. Adil et al. reported an intraneural synovial cyst with four-year follow-up, confirming that substantial motor disability can respond well to surgery [19]. Smith et al. demonstrated that high-resolution MRI can delineate the knee joint origin of a peroneal intraneural ganglion cyst, reinforcing the value of detailed preoperative imaging for operative planning [20].

The present case contributes to this literature in several respects. First, the patient was a young adult male—an age group in which lumbar disc disease, trauma, or entrapment neuropathy are more commonly suspected, potentially delaying recognition of a rare cystic intraneural lesion. Second, the use of intraoperative MEPs and direct stimulation enabled safe dissection and nerve preservation. Third, the postoperative outcome was excellent. Fourth, the case illustrates that cystic lesions at the fibular neck should not be reflexively categorised as solid nerve sheath tumours without careful MRI evaluation.

Limitations of the Present Report

This case report has several limitations. Formal preoperative MRC grading, detailed sensory mapping, nerve conduction study values, and long-term follow-up data are not included in the available record and should be added from the clinical notes before journal submission. The intraoperative status of the articular branch and proximal tibiofibular joint connection should be confirmed from the operative note, as this information is important for assessing recurrence risk. The exact wording of the histopathology report must be inserted; if pathology reveals schwannoma, neurofibroma, or another peripheral nerve sheath tumour, the title, abstract, discussion, and references require revision accordingly.

CONCLUSION

Common peroneal nerve intraneural ganglion cyst is a rare but surgically treatable cause of foot drop. It should be considered in any patient with non-traumatic dorsiflexion weakness and a cystic lesion near the fibular neck or proximal tibiofibular joint. MRI is essential for diagnosis, operative planning, and identification of the articular joint connection. Microsurgical decompression with fascicular preservation can result in excellent neurological recovery. Intraoperative neurophysiological monitoring is a valuable adjunct when the lesion is intimately related to functioning fascicles. To minimise recurrence, the articular branch and proximal tibiofibular joint connection should be specifically evaluated and addressed at surgery. Early diagnosis and anatomically informed nerve-preserving microsurgery are the keys to functional restoration.

Figure Legends



Figure 1. Preoperative clinical photographs demonstrating foot drop. The left panel shows the lateral profile of the affected limb with the foot held in a plantarflexed, adducted position and the toes pointing downward, confirming failure of active ankle dorsiflexion. The right panel shows the anterior view with both feet suspended; the affected foot hangs dependently, contrasting with the contralateral limb, and illustrates the asymmetric neurological deficit. These clinical signs are consistent with dysfunction of the common peroneal nerve at the fibular neck.



Figure 2. Preoperative magnetic resonance imaging of the proximal leg. (a) Axial proton-density or T1-weighted image demonstrating a well-defined, smoothly marginated cystic lesion at the level of the fibular neck, situated in the expected course of the common peroneal nerve. The lesion displaces and compresses adjacent neural structures. (b) Coronal T2-weighted or fat-saturated sequence showing the same lesion as markedly hyperintense with homogeneous fluid signal and no internal solid components or enhancement, consistent with an intraneural ganglion cyst arising in relation to the proximal tibiofibular joint.

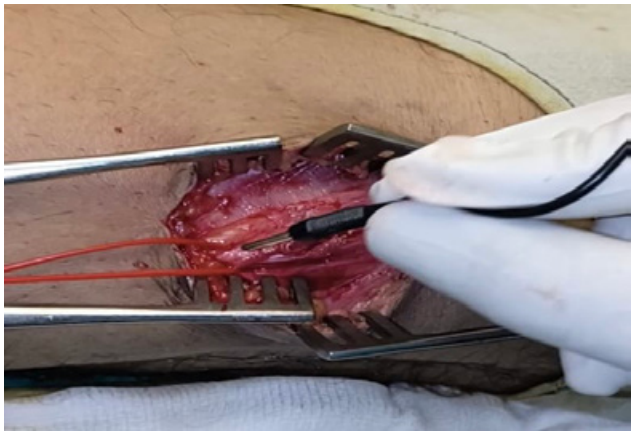


Figure 3. Intraoperative photograph demonstrating microsurgical exposure of the common peroneal nerve at the fibular neck through a lateral approach. The nerve is held with red vessel loops, and a direct electrical stimulation probe (black-tipped instrument) is applied by the surgeon to identify functional fascicles within the distorted intraneural anatomy. Self-retaining retractors maintain the operative field. Neurophysiological monitoring was used throughout the procedure to guide safe dissection and preserve dorsiflexion function.

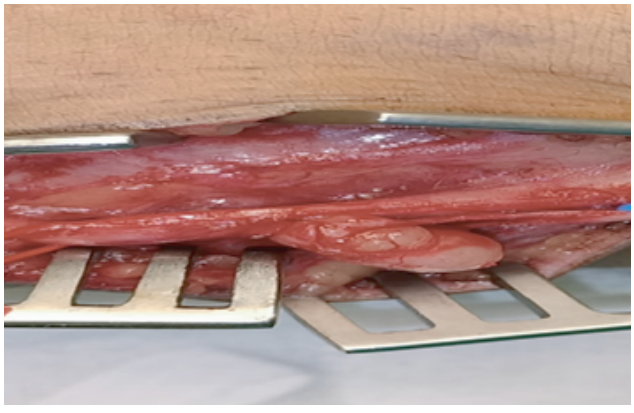


Figure 4. Intraoperative view showing the exposed cystic lesion in close anatomical relationship to the common peroneal nerve. A globular, glistening cystic mass is visible inferior to the main nerve trunk, which is secured with a red vessel loop proximally. A blue sucker is positioned laterally for retraction. The appearance—smooth surface, tense cystic consistency, intimate but distinct relationship to the parent nerve—is consistent with an intraneural ganglion cyst prior to decompression and excision.

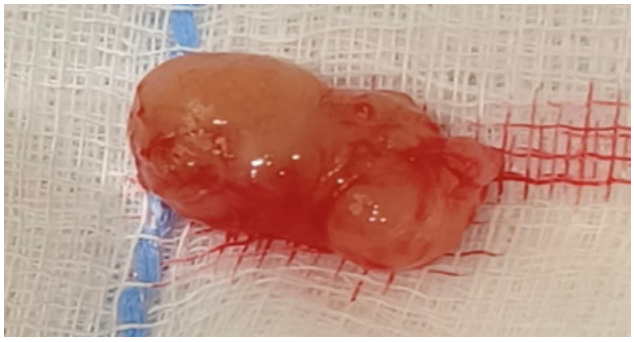


Figure 5. Gross specimen photograph of the excised cystic lesion placed on surgical gauze. The specimen is ovoid, measuring approximately 1.5 cm in greatest dimension, with a smooth, glistening, translucent surface and a bilobated configuration. Its amber-coloured, gelatinous gross appearance is consistent with the mucinous contents of a ganglion cyst. Histopathological examination confirmed a fibrous cyst wall with mucinous material, no epithelial lining, and no malignant features.

Declarations

Patient consent: Written informed consent for publication of clinical details, imaging, and intraoperative photographs was obtained from the patient prior to submission.

Conflict of interest: The authors declare no conflict of interest.

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Ethical approval: Institutional ethical approval was not required for this anonymised single-patient case report.

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MULTICRANIAL NEUROPATHY IN REACTIVATED VARICELLA ZOSTER VIRUS INFECTION—A CASE REPORT

Garima Shant[1], Deepak Jain [2], Manisha Dolia[3]

[1] Department of Medicine, PGIMS, Rohtak, India,
[2] Department of Medicine, PGIMS, Rohtak, India
[3] Department of Medicine, PGIMS, Rohtak, India,

ABSTRACT

Herpes zoster typically presents with dermatomal rash and radicular pain. Although rare, but it can cause various neurological complications such as meningitis, encephalitis, myelitis, cerebral angiitis, subacute amyotrophy and cranial nerve palsy. We report a case of a 63-year-old male who presented with otalgia, facial asymmetry, hearing loss, and subsequently developed dysphagia and hoarseness. Clinical examination revealed involvement of multiple cranial nerves (VII, VIII, IX, and X), consistent with polyneuritis cranialis. Notably, there were no characteristic vesicular eruptions. Cerebrospinal fluid analysis demonstrated lymphocytic pleocytosis with elevated protein, and polymerase chain reaction confirmed VZV DNA. A diagnosis of herpes zoster meningitis with cranial polyneuropathy was established. The patient was treated with intravenous acyclovir and corticosteroids, resulting in complete recovery of neurological deficits on follow-up. This case underscores that Varicella zoster virus can be an important and often underrecognized cause of polyneuritis cranialis, highlighting the need for high clinical suspicion to enable timely diagnosis and management and to prevent long-term sequelae

KEYWORDS

Varicella zoster virus; Polyneuritis cranialis; Herpes zoster meningitis

INTRODUCTION

Varicella Zoster Virus is a neurotropic virus which cause two distinct clinical syndromes: varicella and herpes zoster. Herpes zoster is caused by spontaneous reactivation of VZV which become latent in sensory ganglia, predominately in trigeminal and thoracic ganglion cells during primary infection with chickenpox. The waning of immunity with ageing, sys-