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Spinal Stenosis with Sacral Osseous Deformity Mimicking Chronic Inflammatory Demyelinating Polyneuropathy

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Abstract

BACKGROUND: Differential diagnoses of neurosurgical spinal disorders and polyneuropathies have been recognized to cause clinical perplexity, occasionally misdiagnosing chronic inflammatory demyelinating polyneuropathy (CIDP). When nerve conduction studies and cerebrospinal fluid (CSF) analyses reinforce a certain clinical presentation, the importance of imaging studies, conservative treatment response, and interdisciplinary clinical approach should be highly emphasized.

CASE PRESENTATION: We report a 51-year-old patient who presented with a 16-week history of neurogenic claudication and right-sided lower extremity monoparesis, with low back pain syndrome dating from 10 years ago. He was initially evaluated by a neurologist under the suspicion of CIDP, supported by nerve conduction studies and CSF analyses, without any subjective or objective improvements after systemic corticosteroid therapy. After performing magnetic resonance imaging (MRI) of the lumbosacral spine, he was referred to a neurosurgeon. Neurological examination revealed features of lower motor neuron lesion, consistent with the MRI findings of L4-L5 and L5-S1 stenosis with right-sided S1 vertebra osseous deformity, without any radiographic evidence of CIDP. The patient underwent surgery and improvements were noted early in the post-operative recovery phase and continuously throughout the regular monthly follow-ups, without any clinical features of CIDP. Histopathology results confirm sacral osseous deformity. No evidence of CIDP, osseous deformity residue, or recurrence was evident on the post-operative MRI control performed 11-month post-surgery.

CONCLUSIONS: Degenerative spinal stenosis compromising spinal canal dimensions can mimic CIDP due to sharing multiple clinical similarities. That scenario is especially highlighted when age-related spinal degenerative disease is unexpected and seldom aggravated by spinal osseous lesions. Avoiding misdiagnosis and providing adequate treatment can pose a serious challenge for neurosurgeons and neurologists, demonstrating the importance of an interdisciplinary approach toward diverse spinal disorders.

Introduction

Spinal pathology is a field of never-ending disease varieties, often filled with confusing and inextricably intertwined differential diagnoses, proving that an interdisciplinary approach is key to successful diagnosis and treatment. Spine and neurological disorders such as spinal stenosis syndromes and peripheral polyneuropathies have often inflicted multiple differential diagnosis dilemmas. Frequently, they can lead to misdiagnoses, especially regarding chronic inflammatory demyelinating polyneuropathy (CIDP) [1]. Lumbar spinal stenosis syndrome is a worldwide spread fairly frequent spine pathology. It is most common at L4-L5 and least at the L5-S1 level, first recognized as a distinct clinical entity in the 1950s and 60s [2], [3], [4], with well-established diagnostic methods and treatment modalities throughout the years, with decompressive spinal surgery remaining the gold standard for treatment of such cases. Kalichman *et al*. emphasized that there was no statistically significant association between genders and lumbar spinal stenosis, the mean age was 52.6 ± 10.8, and the prevalence of acquired lumbar spinal stenosis increased with age [5]. CIDP was first mentioned in 1975 [6], with the first case being described in 1899 [7]. CIDP is described as an immune-mediated disease of the peripheral nerves that causes sensory and motor impairment. Although the initial suspicions for CIDP are mainly clinical, the diagnosis is supported by nerve conduction studies, imaging findings, cerebrospinal fluid (CSF) analyses, or rarely, nerve biopsy, and thorough reviewing with the exclusion of other disorders that may cause or mimic CIDP. According to Michaelides *et al*., 62% of CIDP-diagnosed patients were males with mean age of 49.6 years [8]. Approximately 80% of patients respond well to corticosteroids [9], intravenous immunoglobulin [10], or plasma exchange [11]. Regarding these pathological entities, the available literature presents

multiple articles which describe CIDP mimicking a lumbar spinal stenosis syndrome, with the first dating from 1995 [12], but none vice versa. We describe a unique case of lumbosacral spinal stenosis with sacral osseous deformity mimicking CIDP.

Case Presentation

A 51-year-old male patient presented to the neurology department, with a 16-week history of intense low back pain (symptoms of intermittent low back pain syndrome date back 10 years ago), radiating toward both legs, especially affecting his right leg and the gluteal regions bilaterally. Furthermore, he was unable to run or walk on his toes with his right leg, unable to stand upright for more than 30 s, with a maximal walking distance of 10 m and lower body strength reduction with noted "thinning" of his right calf. The patient is an active member of a tactical unit that requires regular strenuous physical activity but due to the abovementioned complaints, his professional performance is limited. The patient underwent a course of physical therapy without any subjective or objective improvements, after which he was evaluated and treated under the suspicion of CIDP. Lower extremity (LE) electromyoneurography (EMNG) findings (Table 1) revealed prolonged distal motor latency (DML), decreased compound muscle action potential (CMAP), and conduction velocity (CV) for the right peroneal nerve and decreased CV for the left peroneal nerve in the fibular region, with both nerves showing signs of temporal dispersion, with tibial nerves bilaterally showing prolonged DML and F-wave latencies, with normal CMAP and CV.

Analyses of CSF revealed an elevated protein count of 0.782 g/L (normal reference values 0.15–0.45 g/L), with the rest of the routinely examined components in their normal reference ranges. The patient was prepared for conservative medical treatment under the suspicion of CIDP. After finishing a systemic corticosteroid trial treatment with no objective clinical improvements, the patient underwent a lumbosacral spine magnetic resonance imaging (MRI) and he was referred to a neurosurgeon for further evaluation while postponing any further neurological treatment. The neurological examination we performed revealed an antalgic posture and gait, right-sided positive straight leg raise (SLR) test at 70°, right-sided absence of the ankle jerk reflex, right-sided plantar flexion weakness (Oxford Muscle Strength Grading Scale 4-/5) with an inability to stand or walk on toes with the right LE, with neurogenic claudication and right-sided LE monoparesis, right-sided hypesthesias and bilateral paresthesias along L5 and S1 dermatomes, right-sided calf hypotrophy, and no other neurological deficits. The lumbosacral spine MRI revealed L4-L5 and L5-S1 spinal stenosis, L5-S1 intervertebral disc degeneration, and a right-sided spinal osseous deformity of the S1 vertebra, eminently compressing the right-sided S1 nerve root and cauda equina, without any nerve element thickening noted (Figure 1).

Based on all the above mentioned evaluations and examinations, after adequate pre-operative preparation, the patient underwent a bilateral L4-L5 laminotomy (interhemilaminectomy) with foraminotomy and right-sided S1 hemilaminectomy with ipsilateral L5-S1 foraminotomy. Maximal reduction of the sacral osseous deformity was achieved and bioptic material was sent for histopathological evaluation (Figure 2).

Furthermore, meticulous microdissection, decompression, and proper mobilization of the L5 nerve roots bilaterally, as well as the cauda equina, right-sided S1 nerve root, and thecal sac were performed (Figure 2). Furthermore, our intraoperative findings did not reveal any enlargement of the bilateral L5 nerve roots and right-sided S1 nerve root, as well as cauda equina (Figure 2). There were no new deficits postoperatively and the patient was fully mobilized on the 1st post-operative day. In the early post-operative recovery phase, the patient reported improved standing in the upright position as opposed to his pre-operative state and he reported significant improvements regarding his maximal walking distance as he became able to walk pain-free for more than 100 m, without a need to rest. The patient was discharged from the hospital in stable and improved condition, with recommendations for post-operative physical therapy and rehabilitation course, and appropriate lifestyle modifications for proper post-operative recovery. During regular monthly follow-ups, the patient reported further improvements in maximal walking distance (more than 3 km without a need to rest) and when standing upright, with reduced but still present bilateral LE paresthesias, with complete absence of the pre-operative painful low back and LE sensations, and no other significant changes.

Table 1: Motor and sensory nerve conduction studies

I. Motor nerve conduction study					
Side R/L (right/left)	Nerve	DML (ms)	M-wave amplitude (mV)	Motor conduction velocity	F-wave latency (ms)
R	n. peroneus profundus	11.6	1.0	38.1	
L	n. peroneus profundus	5.4	1.5	43.0 /// 34.5	
R	n. tibialis	13.3	2.6	37.2	71.9
L	n. tibialis	8.7	4.1	37.9	65.4
II. Sensory nerve conduction study					
Side R/L (right/left)	Nerve	Neurography - latency (ms)	Neurography - amplitude (μs)	Sensory conduction velocity	
R	n. suralis	3.5	6.1	32.1	
L	n. suralis	2.1	7.9	41.7	

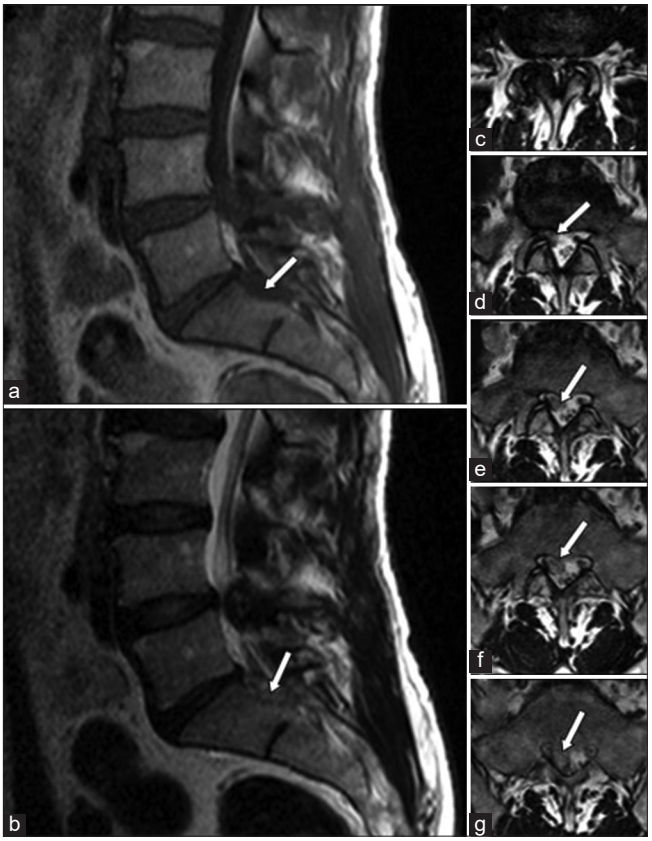

Figure 1: Pre-operative lumbosacral MRI sequences. (a) Sagittal T1-weighted image (WI) presents the osseous deformity with a homogeneous hypointense signal on T1WI (white arrow); (b) Sagittal T2WI shows L5-S1 disc degeneration and posterior S1 osseous deformity, without perilesional edema or signs of inflammation (white arrow), and no nerve element thickening noted, with normal nerve element signal intensity; (c) Axial T2WI at L4-L5 presents spinal stenosis due to facet joint and lig. flavum hypertrophy; (d-g) [descending order – craniocaudal direction] – Axial T2WI at L5-S1 level shows an osseous deformity (white arrows) originating from the posterior part of the inferior right half of the S1 vertebral body lower margin, expressing significant compression on the cauda equina and the right-sided S1 nerve root. The deformity is clearly separated from the posterior part of the S1 body, ascending superiorly, eventually following a trend of narrowing and ending at the level of the lower margin of L5-S1 disc space. The deformity shows low signal areas centrally and intermediate to moderately higher signal parts surrounding it

The histopathological analysis of the bioptic material obtained from the abovementioned osseous lesion revealed cancellous bone with regular bony trabeculae and moderately cellular bone marrow (Figure 3), confirming our initially suspected diagnosis of osseous S1 deformity.

A postoperative MRI control was performed after 11 months, showing no residual or recurrent osseous deformity present, with adequately decompressed thecal sac and cauda equina, bilateral L5 nerve roots, and right-sided S1 nerve root, without any nerve element thickening, noted in Figure 4 and Figure 5. No further neurology consultations were indicated or performed. Further follow-ups are adequately scheduled.

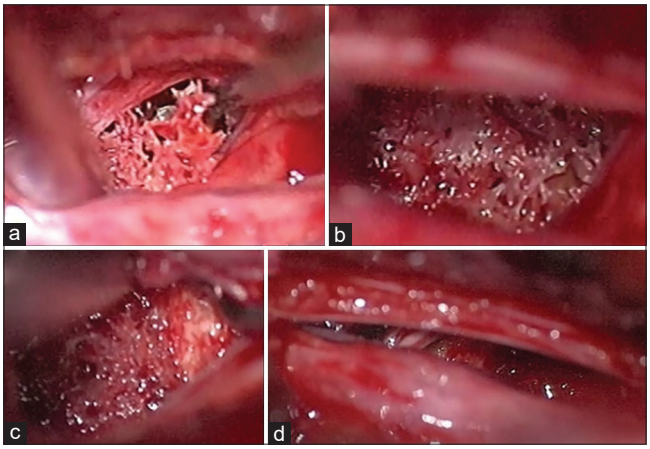

Figure 2: Intraoperative findings (obtained through surgical microscope). (a-c) osseous deformity fragments with a firm to bony consistency, built of trabeculae adjacent to irregular cavities that contain red bone marrow, sent for histopathological analysis; (d) decompressed cauda equina after osseous deformity reduction and widened spinal canal at L5-S1 level

Discussion and Conclusion

The process of reaching the correct diagnosis and implementing adequate treatment in spinal pathology can frequently be one followed by many challenges and obstacles in various aspects. The case we present is a unique finding of L5-S1 spinal stenosis with right-sided S1 vertebra compressive osseous deformity and L4-L5 spinal stenosis, mimicking CIDP. This case differs in comparison to the findings of multiple authors that described CIDP mimicking spinal stenosis [12], [13], [14], [15], [16], [17], [18], once again proving the utmost importance of an interdisciplinary approach toward treating patients presenting with spinal pathology. Given the fact that suspicion for CIDP is initially clinical, the clinical presentation and neurological examination findings tend to differ among the described cases.

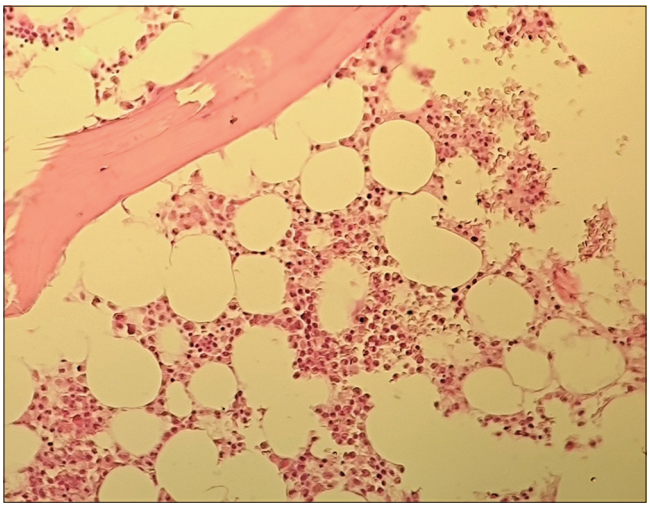

Figure 3: Histopathologic findings of the sacral (S1 vertebra) osseous deformity. The deformity consists of cancellous bone with regular bony trabeculae and moderately cellular bone marrow.

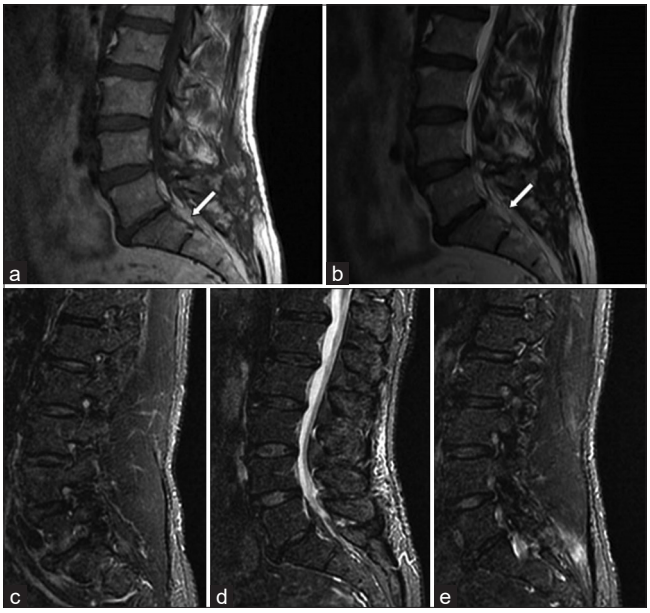

Figure 4: Post-operative lumbosacral MRI sagittal sequences (after 11 months). (a) T1WI; (b) T2WI; (c) [left of midline], (d) [midline], (e) [right of midline] – short tau inversion recovery (STIR) sequences. The MRI sequences showed no residual or recurrent osseous deformity present (white arrows), with normal signal intensity in T1WI, T2WI, and STIR sequences of the nerve elements, without any nerve element thickening noted

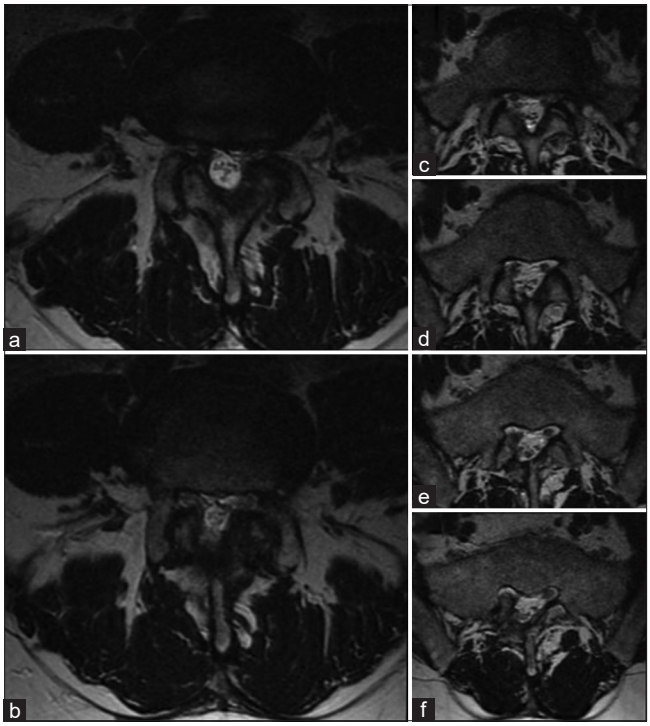

Figure 5: Post-operative lumbosacral MRI T2WI axial sequences (after 11 months). (a), (b) [descending order – craniocaudal direction] – L4-L5 level presenting adequate decompression of the thecal sac and the bilateral L5 nerve roots; (c-f) [descending order – craniocaudal direction] – L5-S1 level showing adequately decompressed thecal sac and cauda equina, as well as right-sided S1 nerve root, without any nerve element thickening noted, with no residual or recurrent osseous deformity present

A 2019 systematic review and meta-analysis defines the classic presentation of CIDP as manifesting sensory and motor impairment in the distal and proximal segments of all limbs evolving over more than 8 weeks, with neuropathic pain being a common symptom among these patients [8]. The disease course was classified as relapsing-remitting in 43% of patients and chronic-progressive in 57% [8]. Different patients have manifested diverse clinical complaints and varying degrees of worsened neurological status, corresponding to some of the findings of our presented patient. However, the majority of his neurological findings were asymmetrical, eventually pointing to asymmetrical sensory-motor CIDP [19]. In addition, his age and gender further justified the initial investigations and treatment under the suspicion of CIDP [8]. Furthermore, as Kalichman *et al*. emphasized, there are no statistically significant gender differences regarding lumbar spinal stenosis, and its prevalence increased with age, especially in 60+ years old patients [5], additionally justifying the CIDP suspicion, evaluation, and treatment. Despite all this, none of his findings were consistent with a specific pattern other than motor and sensory impairments in the affected LE, with diverse findings throughout all the cases in the available literature regarding deep tendon reflexes, SLR test, peroneal palsy, Romberg test, muscle atrophy, pathological reflexes, paraparesis, and bowel and bladder dysfunction [12], [13], [14], [15], [17]. Regarding EMNG characteristics, the findings, in this case, were presented as suggestive of CIDP, according to the currently accepted electrodiagnostic criteria [20], initially affecting the neurologist's suspicion and further treatment course. On a side note, Bostelmann *et al*. [21] described the possibility of spinal canal stenosis-induced polyneuropathy (unspecified type), affecting electrodiagnostic values of LE nerves, contrary to the findings of Jang and Lee [22], which specified that the severity of compression of cauda equina caused by spinal stenosis did not significantly affect the electrodiagnostic values of LE nerves. Turning our attention toward CSF analyses, this case brings a new degree of clinical confusion with the abovementioned elevated protein count in our patient and normal leukocyte count, further supporting the systemic corticosteroid trial treatment that the neurology doctors applied but without any evident objective clinical improvements [20]. As emphasized by London and Nowacek [23], in the absence of supportive clinical and electrodiagnostic data, the specificity of elevated CSF protein is low, and the primary value of CSF analysis is to rule out alternative diagnoses. In addition, CSF analyses have been described as overutilized in the routine evaluation of CIDP and it has been suggested that they may contribute more to the misdiagnosis than the correct diagnosis of CIDP [23]. Furthermore, according to Allen [1], CSF protein values may be influenced by degenerative spinal stenosis, presenting one of the potential pitfalls to CIDP misdiagnosis. Regarding MRI findings, CIDP reveals diffuse thickening of the cauda equina, sometimes resembling an intradural tumor, with low signal intensity in T1 & T2WI and abnormal post-contrast enhancement

of the thickened nerve roots [12], [13], [15], [17], [24] with marked variability of nerve root enlargement seen with CIDP in the literature [25]. Contrary to the above mentioned most common MRI findings in CIDP cases, our patient's MRI did not manifest a radiographic feature of CIDP, but a classic MRI presentation of lumbosacral spinal stenosis Grade C [26] at L4-L5 level and Grade B [26] at the L5-S1 level, additionally aggravated by the abovementioned and histopathologically verified right-sided S1 osseous deformity. The MRI findings of our case did not suggest the S1 vertebra osseous lesion to be a malignant lesion according to its radiographic characteristics [27], initially suspecting sacral osseous deformity, but also considering hyperostosis, enostosis, osteoid osteoma, and osteoblastoma among other less probable diagnoses [27]. It is imperative that in adequate cases, differential diagnoses of spinal osseous benign and malignant lesions should be considered [27], thoroughly reviewed, and adequately excluded before considering further steps in treatment. Our intraoperative findings did not reveal any evident thickening of the abovementioned neural elements, with significant cauda equina and right-sided S1 nerve root compression elicited by the right-sided S1 osseous deformity with the existing stenosis at the L5-S1 level, and compression of the thecal sac and bilateral L5 nerve roots due to L4-L5 level spinal stenosis. The post-operative spine MRI performed after 11 months, showed no residual or recurrent S1 vertebra osseous deformity present, with adequately decompressed neural elements, as stated above. Analyzing the STIR sequence did not reveal any radiographic CIDP characteristics [28], although Oudeman *et al*. [29] demonstrated the limited value of this sequence when differentiating CIDP from normal healthy volunteers. Finally, the systemic corticosteroid trial therapy did not demonstrate any objective clinical improvements [20], but contrary to that, significant objective and subjective improvements after the surgical treatment were evident. The patient presented with continuous improvements in standing and walking abilities, history of pain, and sensory-motor parameters on the regular monthly follow-ups, without any evident pointers towards suspicion of CIDP (relapsing-remitting or chronic-progressive). The aforementioned improvements have additionally confirmed our diagnosis of spinal stenosis and eliminated the initial suspicion of CIDP. Regarding improvements in CIDP diagnostic accuracy, of particular importance are: (1) Heightened attention to "atypical" variants of CIDP; (2) astute clinical correlation when electrophysiologic findings show only amplitude-dependent slowing, are mild or moderate in diabetic patients, are restricted to compressible sites, do not satisfy demyelinating criteria, or are confined to the lower limbs; (3) cautious interpretation of CSF protein values between 0.45 and 0.6 g/L; and (4) adoption of objective metrics of "improvement" if "improvement after immunotherapy" is used to support the diagnosis [1]. Furthermore, it has been suggested that in pursuit of reducing misdiagnosis of CIDP, improvement of the utilization, and adherence to CIDP diagnostic guidelines [23], [30], with correct interpretation of electrodiagnostic data are imperative [23]. In this unique case, we theorize that the facet joint and lig. flavum hypertrophy at L4-L5 and L5-S1 levels, and the S1 osseous deformity, are the main and only causative factors of the patient's clinical presentation of spinal stenosis, decisively excluding CIDP. In addition, this rare case reveals the importance of anticipating multiple differential diagnoses while considering an interdisciplinary approach to solve complex medical dilemmas. The general conclusions from this case report point to the fact that specific disease characteristics, thorough neurological examination, and regular patient follow-ups as fundamental clinical approaches are just as important in defining the correct diagnosis and proper treatment, as well as proper utilization and interpretation of nerve conduction studies, CSF analyses, and imaging methods.

Declaration of patient consent

Declaration of consent has been obtained from the patient for publication of this case. The patient was informed that no personal details will be revealed in the publishing of this case.

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