CLINICAL CASE

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ALCOHOL-RELATED TOXIC-METABOLIC AXONAL POLYNEUROPATHY MIMICKING AMYOTROPHIC LATERAL SCLEROSIS: A CASE REPORT

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Objective. To demonstrate the clinical and electrophysiological features that help distinguish amyotrophic lateral sclerosis (ALS) from alcohol-related toxic-metabolic axonal polyneuropathy in a patient presenting with fasciculations, progressive weakness, and gait impairment. **Material and methods.** The patient's medical history, neurological examination, spinal magnetic resonance imaging (MRI), laboratory investigations, and electroneuromyography (ENMG) findings were reviewed. The diagnostic course was analyzed with particular attention to degenerative spinal disease, radiculopathy, peripheral polyneuropathy, and motor neuron disease. Written informed consent was obtained for anonymous publication of the clinical information. **Results.** A male patient born in 1977 presented with progressive low back pain radiating to the lower limbs, weakness, gait difficulty, and fasciculations. Degenerative spinal disease was initially considered the principal explanation, and decompressive microdiscectomy was performed, but neurological symptoms persisted. Fasciculations and denervation findings subsequently raised suspicion of ALS, and ALS-directed treatment was initiated. Reassessment showed distal sensory abnormalities, reduced tendon reflexes with lower-limb hyporeflexia and areflexia, absence of pyramidal signs, and ENMG evidence of sensorimotor axonal polyneuropathy with a radiculopathic component. Spinal MRI demonstrated multilevel degenerative changes without spinal cord signal abnormality or myelopathy. Serological tests for HIV, hepatitis B surface antigen, hepatitis C antibodies, and syphilis were negative; marked hyperglycemia and major inflammatory changes were not identified. In the context of long-term alcohol use, the overall findings favored alcohol-related toxic-metabolic axonal polyneuropathy rather than isolated motor neuron disease. **Conclusion.** Fasciculations and denervation should not be interpreted in isolation as evidence of ALS. Careful integration of sensory findings, reflex changes, ENMG, neuroimaging, laboratory data, and exposure history is essential for identifying potentially treatable ALS mimics and avoiding diagnostic error.

Keywords: fasciculations, amyotrophic lateral sclerosis, alcohol-related polyneuropathy, axonal polyneuropathy, radiculopathy, diagnostic error

Introduction. Fasciculations are spontaneous, involuntary contractions of muscle fibers and are regarded during neurological examination as one of the signs of lower motor neuron involvement [1]. They are classically associated with amyotrophic lateral sclerosis (ALS), a severe neurodegenerative disorder characterized by progressive degeneration of upper and lower motor neurons [2–4].

However, fasciculations are neither specific nor pathognomonic for ALS and may occur in peripheral nervous system disorders, radiculopathies, metabolic abnormalities, and toxic conditions [1, 3, 5, 6]. Peripheral neuropathies may also occur as neurological manifestations of systemic diseases, including rheumatic disorders [7]. Therefore, interpreting fasciculations exclusively in the context of ALS may lead to an incorrect diagnostic pathway. The clinical overlap between ALS and various peripheral or spinal disorders may contribute to initial misdiagnosis and diagnostic delay [8].

Alcohol-related polyneuropathy is a common toxic-metabolic neuropathy that typically presents as a sensorimotor polyneuropathy characterized predominantly by axonal degeneration [5, 6]. Long-term alcohol use may damage the peripheral nervous system through direct neurotoxic effects as well as through nutritional disturbance and vitamin deficiency [5]. In some patients, weakness, reduced reflexes, muscle atrophy, pain, and fasciculations may create clinical overlap with ALS [3, 5].

Accordingly, diagnostic decisions in patients presenting with fasciculations should not be based on a single clinical sign. Medical history, neurological examination, MRI findings, and

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ENMG data should be interpreted together [3, 6, 9]. **The objective** of this case report was to illustrate the clinical and electrophysiological features relevant to the differential diagnosis between ALS and toxic-metabolic axonal polyneuropathy in a patient presenting with fasciculations, progressive weakness, and gait impairment.

Case presentation. A male patient born in 1977 presented with low back pain radiating to the lower extremities, progressive weakness, difficulty walking, and muscle fasciculations that had progressed over several months. A history of long-term alcohol use was documented. At the initial stage, the clinical presentation was attributed primarily to degenerative disc disease of the spine and radicular pain syndrome.

Clinical course and previous intervention. At different stages, lumbar MRI demonstrated multilevel degenerative changes, disc protrusions, and discogenic-radicular changes at the L4–L5 and L5–S1 levels. The patient underwent decompressive neurosurgical treatment with microdiscectomy; however, the neurological symptoms did not completely resolve. Because weakness, tendon-reflex abnormalities, and fasciculations persisted, ALS was subsequently suspected.

Neurological examination. The patient was alert and oriented to time and place, with no clinically apparent cognitive impairment. Cranial nerve examination revealed no clinically significant abnormality. Motor examination showed peripheral-type weakness and muscle hypotonia, more pronounced in the distal lower extremities. Tendon reflexes were reduced in the upper extremities and more markedly reduced or absent in the lower extremities. Sensory examination disclosed distal polyneuritic-type abnormalities. Fasciculations were observed in the muscles, particularly in the back and proximal parts of the extremities. Pyramidal signs and pathological reflexes were absent. Although no major abnormality was identified on coordination testing, gait was unstable and limited by weakness.

Neuroimaging. MRI of the cervical, thoracic, and lumbosacral spine demonstrated multilevel degenerative changes, disc protrusions, foraminal narrowing at individual levels, and radicular changes. No pathological spinal cord signal abnormality, signs of myelopathy, or mass lesion were identified. The degenerative abnormalities could partly account for the pain syndrome and a local radicular component, but they did not fully explain the combination of distal weakness, sensory impairment, and reduced reflexes.

Electrophysiological assessment. ENMG demonstrated axonal-type peripheral nerve involvement, features of sensorimotor axonal polyneuropathy, and a radiculopathic component. Denervation and chronic neurogenic changes were present in some muscles; however, their distribution and electrophysiological characteristics were not considered specific for an isolated motor neuron disorder. When interpreted in the clinical context, the electrophysiological findings did not provide sufficient evidence of primary and progressive anterior horn motor neuron degeneration.

Laboratory assessment. Serological tests for human immunodeficiency virus, hepatitis B surface antigen, hepatitis C antibodies, and syphilis were negative. No marked hyperglycemia was identified, and the complete blood count showed no major inflammatory changes. These findings reduced the likelihood of selected infectious and metabolic causes of polyneuropathy.

Diagnostic course. The diagnostic process developed in four stages. First, degenerative spinal disease was regarded as the principal cause of the neurological symptoms, and conservative treatment, including physiotherapy, was provided; the clinical response was insufficient, and diffuse peripheral nerve involvement was not initially recognized. Second, decompressive neurosurgical intervention was performed for discogenic-radicular changes, but weakness, reflex abnormalities, and gait difficulty persisted. Third, fasciculations and denervation findings led to suspicion of ALS, and ALS-directed specific treatment was initiated before the diagnostic criteria had been fully substantiated; the fasciculations and denervation findings were interpreted largely outside the broader clinical context. Fourth, repeat clinical, anamnestic, and electrophysiological assessment demonstrated the importance of long-term alcohol exposure, sensory abnormalities, and ENMG evidence of sensorimotor axonal polyneuropathy. Alcohol-related toxic-metabolic axonal polyneuropathy with a radiculopathic component therefore became the leading diagnostic interpretation.

Discussion. Although fasciculations are a characteristic feature of ALS, they are not unique to this disorder [1–3]. Peripheral neuropathies, radiculopathies, and toxic or metabolic disorders should be considered in the differential diagnosis of conditions presenting with fasciculations [1, 5, 6]. Their diagnostic significance should therefore be assessed together with other clinical and electrophysiological findings.

Several features in the present case argued against ALS as the primary explanation. Sensory impairment, the distal and peripheral pattern of involvement, reduced lower-limb reflexes, absence of pyramidal signs, and ENMG evidence of sensorimotor axonal polyneuropathy were not typical of ALS [3, 6]. The presence of denervation alone is insufficient to establish ALS because similar changes may occur in radiculopathy and axonal neuropathy [3, 6].

Degenerative abnormalities identified on spinal MRI do not necessarily explain the entire neurological syndrome. In a case reported by Finsterer and Aliyev, an L5/S1 disc prolapse was initially considered the cause of lower-limb weakness, whereas subsequent evaluation identified a variant of chronic inflammatory demyelinating polyneuropathy with axonal and demyelinating changes [9]. Similarly, in the present patient, discogenic-radicular abnormalities could partly explain pain and local root symptoms but did not account for the complete neurological picture. The absence of spinal cord signal abnormalities and myelopathic features also reduced the likelihood of a central spinal cord process. MRI findings should therefore not be interpreted separately from clinical and electrophysiological information [3, 9].

ENMG played a central role in redirecting the diagnostic process. The findings indicated axonal peripheral nerve injury, sensorimotor polyneuropathy, and a radiculopathic component. In combination with fasciculations, such abnormalities may be misinterpreted as ALS; however, involvement of sensory fibers and a polyneuropathic pattern are not characteristic of ALS [3, 6]. ENMG should therefore be interpreted together with the medical history, neurological examination, and neuroimaging findings [3, 6].

Laboratory evaluation is also important in the etiological differential diagnosis of polyneuropathy. In distal symmetric polyneuropathy, blood glucose testing, measurement of vitamin B12 and its metabolites, and serum protein immunofixation electrophoresis are among the investigations with relatively high diagnostic yield [10]. In the present case, the investigations that were available reduced the probability of selected infectious and metabolic causes, although the documented laboratory work-up was not comprehensive.

Long-term alcohol use may cause toxic-metabolic injury to the peripheral nervous system [5]. Axonal degeneration, pain, sensory impairment, reduced reflexes, distal weakness, and muscle atrophy may occur in this setting [5]. Partial overlap of these manifestations with the clinical features of ALS may allow alcohol-related polyneuropathy to act as an ALS mimic [3, 5]. In this patient, long-term alcohol exposure, sensory abnormalities, and ENMG evidence of sensorimotor axonal polyneuropathy formed the principal basis for the final diagnostic interpretation.

Limitations. This report describes a single patient and therefore cannot establish the frequency with which alcohol-related polyneuropathy mimics ALS or determine the diagnostic performance of individual clinical or electrophysiological features. The available documentation did not include detailed numerical ENMG parameters, a comprehensive report of all laboratory investigations recommended for distal symmetric polyneuropathy, or long-term follow-up after the revised diagnostic assessment. These limitations should be considered when interpreting the case.

Conclusion. This case illustrates that fasciculations are not pathognomonic for ALS and should not be used in isolation to establish the diagnosis. Sensory abnormalities, peripheral-type reflex reduction, sensorimotor axonal polyneuropathy, and radiculopathic findings should prompt targeted evaluation for ALS mimics. ENMG findings should be interpreted together with the clinical examination, exposure history, and neuroimaging. Degenerative abnormalities on spinal MRI do not by themselves prove that they are the principal cause of neurological symptoms. Alcohol-related toxic-metabolic axonal polyneuropathy may clinically resemble ALS, and potentially treatable peripheral nervous system disorders should be considered before a diagnosis of ALS is finalized.

Patient consent. Written informed consent was obtained from the patient for anonymous publication of the clinical information.

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XÜLASƏ

ALKOQOL MƏNŞƏLİ TOKSİK-METABOLİK AKSONAL POLİNEYROPATİYANIN YAN AMİOTROFİK SKLEROZU İMİTASİYA ETMƏSİ: KLİNİK HAL

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Məqsəd. Fassikulyasiya, proqressiv zəiflik və yeriş pozuntusu ilə müraciət edən pasiyentdə yan amiotrofik skleroz (YAS) ilə alkoqol mənşəli toksik-metabolik aksonal polineyropatiyanın fərqləndirilməsinə kömək edən klinik və elektrofizioloji xüsusiyyətləri göstərmək. **Material və metodlar.** Pasiyentin anamnezi, nevroloji müayinəsi, onurğa sütununun maqnit-rezonans tomoqrafiyası (MRT), laborator müayinələri və elektroneyromioqrafiya (ENMQ) nəticələri təhlil edilmişdir. Diaqnostik gediş degenerativ onurğa xəstəliyi, radikulopatiya, periferik polineyropatiya və motor neyron xəstəliyi baxımından qiymətləndirilmişdir. Klinik məlumatların anonim dərcinə dair pasiyentdən yazılı məlumatlandırılmış razılıq alınmışdır. **Nəticələr.** 1977-ci il təvəllüdlü kişi pasiyent aşağı ətraflara yayılan proqressiv bel ağrısı, zəiflik, yeriş çətinliyi və fassikulyasiyalarla müraciət etmişdir. İlkin olaraq degenerativ onurğa patologiyası əsas səbəb hesab edilmiş və dekompressiv mikrodiskektomiya aparılmış, lakin nevroloji simptomlar davam etmişdir. Fassikulyasiyalar və denervasiya əlamətləri sonradan YAS şübhəsi yaratmış və xəstəliyə yönəlik spesifik müalicəyə başlanmışdır. Təkrar qiymətləndirmədə distal hissiyyat pozuntuları, vətər reflekslərinin azalması, aşağı ətraflarda hiporefleksiya və arefleksiya, piramid əlamətlərin olmaması, ENMQ-də isə radikulopatik komponentlə birlikdə sensor-motor aksonal polineyropatiya aşkar edilmişdir. Onurğa MRT-sində çoxsəviyyəli degenerativ dəyişikliklər olsa da, onurğa beynində patoloji siqnal dəyişikliyi və mielopatiya müəyyən edilməmişdir. İİV, HBsAg, anti-HCV və sifilis üzrə seroloji testlər neqativ olmuş, aşkar hiperglikemiya və əhəmiyyətli iltihabi dəyişikliklər qeyd edilməmişdir. Uzunmüddətli alkoqol istifadəsi fonunda ümumi klinik və elektrofizioloji mənzərə izolə olunmuş

motor neyron xəstəliyindən daha çox alkoqol mənşəli toksik-metabolik aksonal polineyropatiyanı dəstəkləmişdir. **Yekun.** Fassikulyasiya və denervasiya YAS-ın sübutu kimi klinik kontekstdən ayrılıqda şərh edilməməlidir. Hissiyyat dəyişikliklərinin, refleks pozuntularının, ENMQ, neyrovizualizasiya, laborator məlumatlar və təsir amilləri anamnezinin kompleks qiymətləndirilməsi müalicə oluna bilən YAS imitatorlarının vaxtında tanınması və diaqnostik səhvlərin qarşısının alınması üçün vacibdir.
Açar sözlər: fassikulyasiya, yan amiotrofik skleroz, alkoqol polineyropatiyası, aksonal polineyropatiya, radikulopatiya, diaqnostik səhv

РЕЗЮМЕ

ТОКСИКО-МЕТАБОЛИЧЕСКАЯ АКСОНАЛЬНАЯ ПОЛИНЕЙРОПАТИЯ АЛКОГОЛЬНОГО ГЕНЕЗА, ИМИТИРУЮЩАЯ БОКОВОЙ АМИОТРОФИЧЕСКИЙ СКЛЕРОЗ: КЛИНИЧЕСКИЙ СЛУЧАЙ

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Цель. Показать клинические и электрофизиологические признаки, позволяющие дифференцировать боковой амиотрофический склероз (БАС) и токсико-метаболическую аксональную полинейропатию алкогольного генеза у пациента с фасцикуляциями, прогрессирующей слабостью и нарушением ходьбы. **Материал и методы.** Проанализированы анамнез, данные неврологического осмотра, магнитно-резонансной томографии (МРТ) позвоночника, лабораторных исследований и электронейромиографии (ЭНМГ). Диагностический процесс оценивали с учетом дегенеративного заболевания позвоночника, радикулопатии, периферической полинейропатии и болезни мотонейрона. Получено письменное информированное согласие пациента на анонимную публикацию клинических данных. **Результаты.** Пациент мужского пола, 1977 года рождения, обратился с прогрессирующей болью в пояснице с иррадиацией в нижние конечности, слабостью, затруднением ходьбы и фасцикуляциями. Первоначально основным объяснением считалась дегенеративная патология позвоночника; была выполнена декомпрессивная микродискэктомия, однако неврологическая симптоматика сохранялась. В дальнейшем фасцикуляции и признаки денервации вызвали подозрение на БАС, и была начата специфическая терапия. При повторной оценке выявлены дистальные чувствительные нарушения, снижение сухожильных рефлексов с гипорефлексией и арефлексией в нижних конечностях, отсутствие пирамидных знаков и ЭНМГ-признаки сенсомоторной аксональной полинейропатии с радикулопатическим компонентом. МРТ позвоночника показала многоуровневые дегенеративные изменения без патологического сигнала в спинном мозге и признаков миелопатии. Серологические тесты на ВИЧ, HBsAg, anti-HCV и сифилис были отрицательными; выраженная гипергликемия и значимые воспалительные изменения не выявлены. С учетом длительного употребления алкоголя совокупность данных больше соответствовала токсико-метаболической аксональной полинейропатии алкогольного генеза, чем изолированной болезни мотонейрона. **Заключение.** Фасцикуляции и денервационные изменения не следует интерпретировать изолированно как доказательство БАС. Комплексная оценка чувствительных нарушений, рефлекторных изменений, ЭНМГ, нейровизуализации, лабораторных данных и анамнеза воздействующих факторов необходима для своевременного выявления потенциально лечимых состояний, имитирующих БАС, и предупреждения диагностических ошибок.
Ключевые слова: фасцикуляции, боковой амиотрофический склероз, алкогольная полинейропатия, аксональная полинейропатия, радикулопатия, диагностическая ошибка

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