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Letter to the Editor

The presence of sensory neuropathy should call into question the diagnosis of ALS

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Letter to the Editor

We were interested to read the article by Collins *et al*. about a 63-year-old man who had been diagnosed with slowly progressive proximal muscle weakness and a slight increase in creatine kinase (CK) for a year, which was initially attributed to long-term atorvastatin use (statin myopathy) [1]. As the patient also developed signs of upper motor neuron disease as the disease progressed, the diagnosis was changed to amyotrophic lateral sclerosis (ALS) [1]. The study is noteworthy, but several points should be discussed.

The first point is that we do not agree with the diagnosis of ALS [1]. Although the patient shows progressive involvement of upper and lower motor neurons, there are several arguments against the diagnosis of ALS. First, the patient had a sensory neuropathy [1]. The involvement of sensory fibers in ALS is unusual unless the patient has a second pathology. Second, the patient had "marked proximal muscle weakness" [1]. Limb onset ALS usually begins with weakness and muscle wasting in the distal muscles and progresses from distal to proximal as the disease progresses. Thirdly, the patient does not meet the revised Gold Coast criteria for the diagnosis of ALS, which are the current criteria for the diagnosis of ALS [2]. In addition to the progressive affection of the upper and lower motor neurons, these criteria require that all differential diagnoses that could explain the clinical picture must be thoroughly ruled out, which was not the case in the index patient [1]. Differential diagnoses that must be excluded are mitochondrial myopathy, beta-oxidation defects and other metabolic myopathies/neuropathies, compressive myelopathy, syrinngomyelia, chronic inflammatory demyelinating polyneuropathy, Guillain-Barre syndrome, multifocal motor neuropathy, inclusion body myositis, polymyositis, brainstem gliomas, Lamber-Eaton syndrome, Lyme disease, multiple sclerosis, neurosarcoidosis, primary lateral sclerosis, spinal muscular atrophy and hexokinase deficiency [3].

Secondly, we disagree that ALS is a subtype of a myopathy, as claimed in the discussion [1]. ALS is a motor neuron disease with upper and lower motor neuron involvement, with either distal, unilateral limb onset or bulbar onset, sparing sensory and autonomic fibers.

The third point is that no cerebral and spinal cord imaging findings have been reported [1]. There is also no report of MR tractography. Was there any evidence of atrophy of the corticospinal tract on routine imaging or tractography? Atrophy of the pyramidal tract is a common feature on imaging or tractography in ALS patients [4].

The fourth point is that no family history was reported. Since myopathy and neuropathy are often inherited, it is important to know whether or not one of the first-degree relatives also has a neuromuscular disease.

The fifth point is that no long-term follow-up data were reported. Since ALS is usually a rapidly progressive disease, it can be expected that the clinical symptoms and signs in the index patient also deteriorated rapidly. In particular, we should know whether the patient developed bulbar and respiratory muscle involvement leading to dysarthria, dysphagia and respiratory insufficiency. Did the patient last need a nocturnal oxygen supply or a CPAP mask? Was tongue atrophy present? Since certain familial forms of ALS can be complicated by cognitive impairment, it would be interesting to know whether the patient developed signs of dementia over time.

The sixth point relates to the discrepancy between the diagnosis of sensorimotor neuropathy and the absence of any sensory symptoms. Did the patient have sensory deficits on clinical neurologic examination or quantitative sensory testing? Did the patient complain of paresthesias, dysesthesias or allodynia? Did somato-sensory evoked potentials show prolonged cortical responses?

The seventh point is that no explanation was given for the swelling of the left foot [1]. Was this due to heart failure, renal insufficiency, venous thrombosis, hypostasis or lymphoedema?

To summarize, the diagnosis of ALS can only be made if the currently valid criteria are met. The diagnosis of ALS is particularly questionable in patients with sensory neuropathy.

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