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Letter to the Editor

No Nerve Biopsy in Cases of Unexplained Neuropathy if AL Amyloidosis has been Detected in Muscle

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Letter to the Editor

We read with interest the article by Driscoli *et al*. about a 48-year-old woman with a history of localized AL amyloidosis of the eye muscles that was treated with radiation therapy, steroids, and mycophenolate mofetil without positive effect. She subsequently developed sensory disturbances and muscle weakness [1]. The biopsy of the sural nerve was negative, but a second nerve biopsy of the ulnar nerve documented again AL amyloid [1]. It was concluded that a positive biopsy of the ulnar nerve allows for targeted treatment of neural amyloidosis [1]. The study is noteworthy, but some points should be discussed.

The first point is that it is not clear why a mixed motor and sensory nerve was selected for a second nerve biopsy. Since the patient had predominantly sensory deficits, a biopsy of a sensory nerve or branch would probably have been sufficient to detect amyloid neuropathy. A biopsy of a motor nerve carries the risk of permanent muscle weakness for the patient. The results of the rectal mucosa biopsy are also not mentioned.

The second point is that we disagree with the view that the documentation of AL amyloid in the nerve enabled targeted and specific treatment. The treatment is no different if AL amyloid is found in another tissue. What specific treatment did the index patient receive after AL amyloid was found in the ulnar nerve? To what extent did the results of the second nerve biopsy change the treatment?

The third point is that the NCS results are missing, except for those of the sural nerve. In order to assess whether the index patients actually suffered from axonal sensory-motor neuropathy, which is most common in AL amyloidosis [2], it is crucial to know the NCS results.

The fourth point is that the patient had developed bilateral ptosis, but it was not reported whether this was due to myopathy, myasthenia gravis, involvement of the autonomic fibers, or involvement of the oculomotor branch that innervates the levator palpebrae. Since the patient also had a history of amyloidosis of the eye muscles, it cannot be ruled out that the levator palpebrae was also affected by muscular amyloidosis.

The fifth point is that it is unclear why a nerve biopsy was performed at all. The index patient was diagnosed with localized amyloidosis of the eye muscles. The detection of amyloid in a tissue can also explain neuropathy if all other causes of neuropathy have been thoroughly ruled out.

The final point is that the authors appear to confuse electromyography (EMG) with nerve conduction studies (NCS). Sensory or motor neuropathies are usually documented by NCS, which detect either an axonal or a demyelinating lesion with or without conduction blocks. EMG usually distinguishes between a myogenic and a neurogenic lesion and can therefore only indirectly indicate neuropathy.

In summary, biopsy of a motor nerve is not necessary in patients with sensorimotor axonal neuropathy and a history of AL amyloidosis, especially if all other causes of neuropathy have been ruled out.

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