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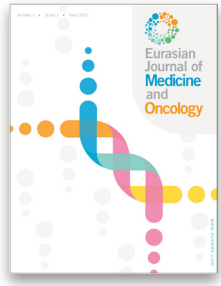

Case Report

Effect of Alpha Lipoic Acid in the Treatment of Multiple Sclerosis-Induced Neuropathic Pain: A Case Report

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Abstract

Multiple sclerosis (MS) is a chronic demyelinating disease of the central nervous system (CNS) resulting in motor, sensory, and cognitive impairment. MS symptoms greatly affect the quality of life of patients with one of the most common symptom being pain. Neuropathic pain (NP) that develops secondary to demyelination, neuro-inflammation, and axonal damage in the CNS is the most distressing and difficult type of pain to treat in patients with MS. A patient with MS presented to our department with dysesthetic extremity pain that was characterized by burning and tingling predominantly in both feet and was worse at night. She was evaluated and diagnosed with MS-induced NP and treated with alpha lipoic acid (ALA) because of unresponsiveness and intolerance to amitriptyline, pregabalin, and gabapentin treatments. In this report, we present and discuss the ALA treatment in a patient with MS-induced NP.
Keywords: Alpha lipoic acid, multiple sclerosis, neuropathic pain

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Multiple sclerosis (MS) is a chronic demyelinating disease of the central nervous system (CNS) resulting in motor, sensory, and cognitive impairment.[1] MS greatly affects the quality of life (QOL) of patients and one of the most common complication is neuropathic pain (NP). Alpha lipoic acid (ALA) is a potent antioxidant, which has been extensively evaluated in prospective, placebo-controlled studies in patients with diabetic neuropathy. Only one study suggested that oral ALA is a promising therapy for MS.[2] We aimed to see whether ALA would be an effective treatment in a patient with MS-induced NP, who could not tolerate classical NP medications.

Case Report

A 49-year-old woman with MS presented to our hospital with complaints of shooting and burning pain, tingling, and numbness in both her feet. She was diagnosed with MS 8 years previously. She had no other systemic diseases. She was treated with pulse steroid in the acute phase but she did not receive any medication in the remission phase. She was diagnosed with NP at the same time and was treated with 10 mg amitriptyline for 4 years and 25 mg for the next 3 years because of an increase in her symptom severity. Although amitriptyline dose was increased as the drug tolerability of the patient was good, her symptoms did not abate and her QOL was affected. According to the patient, she could not wear socks or footwear for the last 3 years because of shooting and burning pain in her feet. Last year, she consulted the neurology department of a hospital, and she was prescribed 75 mg pregabalin twice a day in addition to amitriptyline; the pregabalin dose was increased to 150 mg twice a day after a week. However, the patient experienced side effects of pregabalin, such as balance disorder and fall, dizziness, and somnolence. After

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this drug intolerance, she consulted with the same department and pregabalin treatment was discontinued; instead, gabapentin treatment was started. However, similar side effects occurred and the treatment had to be discontinued. The patient then consulted our hospital with these disturbing and life-limiting symptoms, and after the evaluations, she was diagnosed again with MS-induced NP. We also considered restless leg syndrome in the differential diagnosis, but the patient had no complaints of typical "irresistible urge to move the limbs." The initial Lanss pain scale score was 14, and the physical and mental scores of Short form 36 were 28.9 and 28.1, respectively. The patient did not have diabetes mellitus, but we decided to initiate treatment with ALA 600 mg once a day because of the patient's good tolerability to ALA, unresponsiveness to amitriptyline, and intolerance to pregabalin and gabapentin treatments. In addition, the amitriptyline dose was decreased to 10 mg once a day. Although there were no side effects due to ALA, the symptoms persisted with the same intensity in the first and second week. However, the patient said that the symptoms started to decrease by the third week after which they continued to decrease gradually each week. After 2 months, the patient said "I could wear socks after 3 years." After 3 months, the symptoms decreased by nearly 60% as the patient's expression and the Lanss pain scale score decreased to 6, and the physical and mental scores of SF-36 increased to 41.3 and 51.7, respectively, after the treatment. The patient is still currently on ALA treatment and she has not yet experienced side effects till now.

Discussion

MS is a chronic demyelinating disease of the CNS and greatly affects the QOL of patients, and one of the most common complications is NP. Evidence-based recommendations for the pharmacological management of NP promulgated by the NP Special Interest Group of the International Association for the Study of Pain include the treatment of MS-induced NP.[3] The recommended first-line treatment includes tricyclic antidepressants (such as nortriptyline and amitriptyline), selective noradrenaline reuptake inhibitors (such as duloxetine and venlafaxine), and voltage-gated calcium channel a2-d subunit ligands (such as gabapentin and pregabalin). Strong opioid analgesics (such as morphine, oxycodone, methadone, and fentanyl) and tramadol (alone or in combination with a first-line drug) are generally regarded as second-line treatments.[3, 4] Third-line drugs that may be used in second-line treatment under some circumstances include other antiepileptic drugs (such as carbamazepine, lamotrigine, oxcarbazepine, topiramate, and valproic acid), mexiletine (orally active lignocaine analog), N-methyl-D-aspartate receptor antagonists (such as ketamine and memantine), and topical capsaicin.[3, 4] Although ALA was not recommended as a treatment for MS-induced NP, we decided to treat the patient with ALA because of the side effects and ineffectiveness of the recommended first-line treatment. ALA is a potent antioxidant, which has been extensively evaluated in prospective, placebo-controlled studies in patients with diabetic neuropathy. Some studies have shown that the symptoms and deficits of diabetic polyneuropathy significantly decreased after ALA treatment.[5, 6] However, few studies have shown limited benefit in symptom scores with some improvement in nerve electrophysiology.[7] There are no randomized controlled studies of ALA treatment in patients with MS-induced NP. One study suggested that oral ALA represented a promising therapy for MS because ALA was capable of decreasing the levels of two immunologic markers of MS activity, i.e., serum matrix metalloproteinase-9 and soluble intercellular adhesion molecule-1, which was indirectly associated with T cell migration into the CNS.[2] Based on these cellular mechanisms and side effects of antiepileptic drugs, we considered using ALA to treat the symptoms of MS-induced NP for this patient as ALA treatment had very few side effects and patients with diabetic neuropathy showed good responses. After the treatment, the patient's response to ALA was very good as mentioned above, and we considered using ALA alone or in combination with the recommended first-line drugs in our clinic to treat MS-induced NP in some selected cases. We believe that ALA should also be considered as a choice in the treatment of MS-induced NP; however, further studies are needed to evaluate the efficacy of ALA in MS-induced NP.

Disclosures

Informed consent: Written informed consent was obtained from the patient for the publication of the case report.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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