Case Report

Thalidomide-induced sensory axonal polyneuropathy: A case report

Abstract

Introduction: Several chemotherapeutic agents are currently available for the management of various malignancies; however, many are associated with adverse effects. A case of thalidomide- induced polyneuropathy is described below.

Case Description: A 65year old man, with a history of secondary myelofibrosis in management with thalidomide, consulted for 5months of neuropathic pain and paresthesia in both hands. On physical examination, he presented hypoesthesia and hyperalgesia in both hands. In the electrodiagnostic studies (EDX) a sensory polyneuropathy of axonal type was concluded.

Discussion: Chemotherapy-induced peripheral neuropathy (CIPN) is a side effect that occurs in up to 70% of patients. Symptomatology includes motor and sensitive deficit. EDX studies confirm diagnoses and characteristics. For the particular case of thalidomide involvement is mainly axonal. There is no standard treatment for CIPN, but several drugs can improve symptoms.

Conclusions: CIPN is a frequent complication in patients managed with chemotherapy. The physiatrist is the ideal professional to provide a comprehensive approach to this entity, in order to improve the patient's functionality.

Keywords: polyneuropathy, thalidomide, electrodiagnosis, drug-related side effects and adverse reactions, hematologic neoplasms

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Abbreviations: CIPN, chemotherapy-induced peripheral neuropathy; EDX, electrodiagnostic study; SNAPs, sensory nerve action potentials

Introduction

The increase in life expectancy leads to an increase in chronic diseases, including cancer. Currently, there are several chemotherapeutic agents available for the management of different neoplasms; however, many are associated with adverse effects, such as neuropathies. The diagnosis of chemotherapy-induced peripheral neuropathy (CIPN) represents a clinical challenge, due to the multiple coexisting or previous conditions that predispose to peripheral nerve injury. Currently, there is no standard treatment, however many experimental agents show promise.[1]

Clinical case

Male in the sixth decade of life, right-handed, with a history of essential thrombocythemia diagnosed 10 years ago, reclassified 5 years ago as secondary myelofibrosis. Under management with ruxolitinib, thalidomide and hydroxyurea. He consulted for a 5-month clinical picture, consisting of pain with neuropathic characteristics and paresthesias in both hands, predominantly on the left. Occasionally, objects have fallen from his hands and he has had difficulty performing manual activities that require a fine pincer grasp. Due to the above, peripheral neuropathy was suspected.

On physical examination, he presented hypoesthesia and hyperalgesia both on the back and on the palm of both hands. There was no evidence of atrophy in the intrinsic muscles of the hand, nor alteration of muscle strength. The coin rotation test was abnormal, obtaining a time of 23 seconds on the right side and 25 on the left.

In the electrodiagnostic study (EDX), no sensory response from the left ulnar nerve was found and the amplitude of sensory nerve action potentials (SNAPs) of the median nerves and the right ulnar nerve was reduced (Figure 1). Motor nerve conduction of the median and ulnar nerves, (Figure 2) as well as conventional needle electromyography, were normal. It was concluded that the findings were compatible with a sensory polyneuropathy of the axonal type. It was recommended to complement the examination with the evaluation of the lower limbs to improve the characterization.

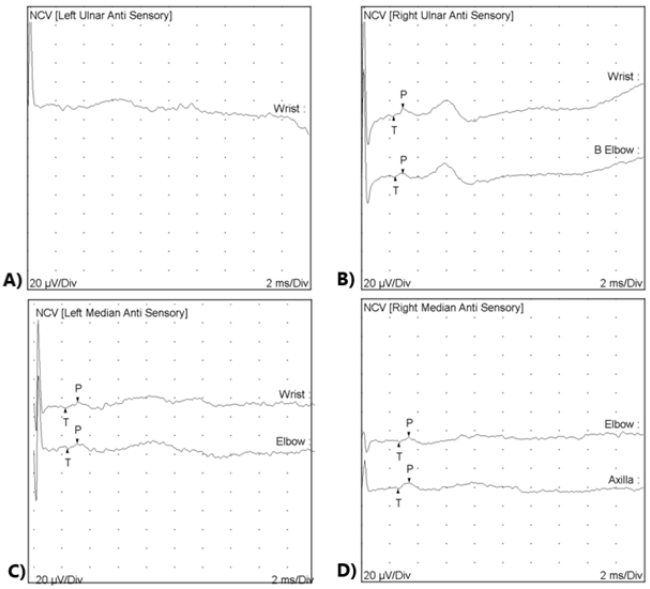

Figure 1 Sensitive nerve conduction. A. Absence of response of the left ulnar nerve. B,C,D. Amplitude disminution of the right and median nerves.

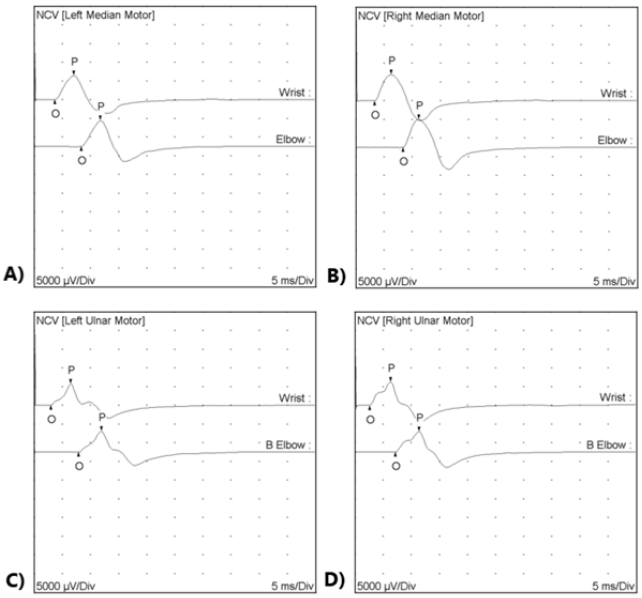

Figure 2 Motor nerve conduction. A and B Ulnar nerves. C and D Median nerves.

Discussion

Thalidomide is an immunomodulator currently used for the treatment of hematologic neoplasms.[2] Its mechanism of action is uncertain, but it is believed to be associated with the suppression of tumor necrosis factor alpha. CIPN is a side effect that occurs in up to 70% of patients. This effect is due to axonal degeneration, without demyelination, and can be severe and permanent. The symptoms generally start with pain, hypoesthesia, and paresthesia in hands and feet, but in severe cases hyporeflexia, weakness, and ataxia occur.[3]

Although thalidomide CIPN is known to be dose-dependent, the cause of neuropathy is still poorly understood. Theories exist that involve amyloid deposits, the complement cascade, and cytokine-mediated injury.[4]

EDX studies confirm CIPN, identifying the nerve structures involved, the presence of axonal degeneration, and its severity. In the particular case of thalidomide, axonal damage compromises the amplitude of SNAPs, evidencing a decrease of 50% or more.[5] Quantitative sensory tests have been introduced in recent years to assess small fibers; however, they are not widely available and require the collaboration of the patient.[5]

There is no standard treatment for CIPN. Tricyclic antidepressants, selective serotonin and norepinephrine reuptake inhibitors may be recommended in first-line management.[6]

Conclusion

CIPN is a frequent complication in patients managed with chemotherapeutics. The physiatrist, having knowledge in oncological rehabilitation, electrodiagnosis and pain management, is the ideal professional to give a comprehensive approach to this entity, in order to improve the functionality of the patient and, as far as possible, reintegrate him to his normal activities.

Acknowledgments

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Conflicts of interest

Authors declare no conflict of interest exists.

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