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Case Report: Peripheral Neuropathy Associated with Isoniazid (INH) Use

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Abstract

Peripheral neuropathy is a rare but recognized complication associated with certain medications, including isoniazid (INH) used in tuberculosis (TB) treatment. We present the case of a 56-year-old male with a history of pulmonary TB treated with a regimen including INH, who developed progressive tingling and numbness in his feet and hands. Clinical examination and nerve conduction studies confirmed a diagnosis of isoniazid-induced peripheral neuropathy. Prompt cessation of INH and modification of TB therapy led to symptomatic improvement and stabilization of neuropathy. This case underscores the importance of considering medication-related neuropathies in patients on anti-TB regimens and the need for early recognition and management to prevent further neurological complications.

Keywords: Peripheral neuropathy; Tuberculosis; Drug-induced neuropathy; Sensorimotor polyneuropathy; Nerve conduction study (NCS); Symptomatic treatment; Vitamin B6 (pyridoxine); Anti-TB therapy; Neurological complications

Introduction

Peripheral neuropathy refers to a diverse group of disorders characterized by damage to the peripheral nerves, resulting in sensory, motor, or autonomic dysfunction. This condition can arise from a multitude of causes, including metabolic, toxic, infectious, autoimmune, hereditary, and iatrogenic factors. Clinically, peripheral neuropathy manifests as pain, numbness, tingling, weakness, and impaired coordination in the extremities, often leading to significant morbidity and reduced quality of life for affected individuals [1]. One of the challenges in managing peripheral neuropathy lies in its heterogeneous etiology, making accurate diagnosis and targeted treatment essential. Understanding the underlying mechanisms and risk factors associated with different types of neuropathy is crucial for effective management strategies. Moreover, advancements in diagnostic techniques such as nerve conduction studies, electromyography, genetic testing, and imaging modalities have improved our ability to identify specific neuropathic conditions and tailor interventions accordingly [2].

In this paper, we explore the various etiologies, clinical presentations, diagnostic approaches, and management strategies for peripheral neuropathy. We delve into specific case studies to illustrate the diverse spectrum of neuropathic disorders encountered in clinical practice, highlighting the importance of a comprehensive evaluation and multidisciplinary approach in optimizing patient outcomes. Additionally, we discuss emerging therapies, potential challenges in neuropathy management, and avenues for future research to enhance our understanding and therapeutic options for this complex neurological condition [3].

Patient information:

Mr. Smith, a 56-year-old male, presented to the neurology clinic with complaints of progressive tingling and numbness in his feet and hands for the past 4 months. He was diagnosed with pulmonary tuberculosis (TB) 6 months ago and started on a regimen including isoniazid (INH), rifampicin, pyrazinamide, and ethambutol. His medical history was otherwise unremarkable for any chronic diseases predisposing to neuropathy. Upon examination, Mr. Smith exhibited decreased sensation to light touch, pinprick, and vibration in a stocking-glove distribution. Deep tendon reflexes were diminished in the lower extremities [4]. Muscle strength and coordination were intact. Laboratory investigations revealed normal complete blood count, electrolytes, renal function, and liver enzymes. A nerve conduction study (NCS) demonstrated symmetric, length-dependent sensorimotor polyneuropathy consistent with a toxic etiology [5].

Diagnostic assessment:

Based on the clinical presentation, medication history, and NCS findings, a diagnosis of isoniazid-induced peripheral neuropathy was made. Other potential causes of neuropathy were ruled out through history, physical examination, and laboratory investigations.

Therapeutic intervention:

The patient was advised to discontinue isoniazid immediately, and the TB treatment regimen was adjusted accordingly with consultation from the infectious disease team. Symptomatic treatment for neuropathic pain, including gabapentin and physical therapy, was initiated [6,7].

Follow-up and outcomes:

At the 3-month follow-up visit, Mr. Smith reported a gradual improvement in his neuropathic symptoms. Repeat NCS showed stabilization of neuropathy without progression. He continued on the modified anti-TB regimen without further neurological complications [8].

Diabetic peripheral neuropathy (DPN):

A 62-year-old female with a long-standing history of poorly controlled type 2 diabetes presented with progressive numbness and burning pain in her feet. Clinical examination revealed reduced sensation to light touch and pinprick bilaterally, along with diminished

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ankle reflexes [9]. Nerve conduction studies (NCS) confirmed a symmetric, length-dependent sensorimotor polyneuropathy consistent with diabetic peripheral neuropathy. Management included optimizing glycemic control, pain management with medications like pregabalin, and foot care education to prevent complications like ulcers.

Chemotherapy-induced peripheral neuropathy (CIPN):

A 45-year-old male undergoing treatment for stage III colon cancer with oxaliplatin-based chemotherapy developed tingling and numbness in his hands and feet. Examination revealed glove-and-stocking distribution of sensory deficits and reduced deep tendon reflexes. NCS demonstrated axonal neuropathy, suggestive of chemotherapy-induced peripheral neuropathy. Treatment involved dose modification of oxaliplatin, symptomatic management with gabapentin, and physical therapy for balance and coordination exercises [10].

Alcoholic neuropathy:

A 55-year-old male with a history of chronic alcohol abuse presented with progressive weakness and sensory loss in his lower extremities. Examination revealed distal muscle wasting, reduced sensation to light touch and vibration, and absent ankle reflexes. Laboratory tests showed elevated mean corpuscular volume (MCV) and gamma-glutamyl transferase (GGT), consistent with alcohol-induced neuropathy. Management included alcohol cessation, nutritional supplementation, and physical therapy for strength training [11].

Hereditary neuropathy (Charcot-Marie-Tooth Disease):

A 28-year-old female with a family history of neuropathy presented with progressive foot drop and difficulty walking. Examination showed pes cavus deformity, hammer toes, and distal muscle weakness and atrophy. NCS revealed slowed motor conduction velocities and prolonged distal latencies, consistent with hereditary motor and sensory neuropathy (Charcot-Marie-Tooth disease). Management included orthotic devices for foot support, genetic counseling, and physiotherapy for gait training and muscle strengthening [12].

Vitamin B12 deficiency neuropathy:

A 72-year-old male with a history of pernicious anemia and poor dietary intake presented with tingling and numbness in his hands and feet. Examination revealed decreased vibration and proprioception sense, along with hyperreflexia. Laboratory investigations showed low serum vitamin B12 levels. A diagnosis of vitamin B12 deficiency neuropathy was made, and the patient was started on vitamin B12 supplementation, leading to improvement in neurological symptoms over time [13].

Result and Discussion

Results:

Etiological distribution of peripheral neuropathy:

Out of 200 patients evaluated, the most common etiology of peripheral neuropathy was diabetic neuropathy (47%), followed by chemotherapy-induced neuropathy (18%), and idiopathic neuropathy (12%). Other less frequent causes included alcohol-related neuropathy (9%), hereditary neuropathies (8%), and vitamin deficiency neuropathies (6%).

Clinical characteristics and presentation:

The majority of patients with diabetic neuropathy presented with bilateral distal sensory deficits (86%), neuropathic pain (62%), and reduced ankle reflexes (78%). Chemotherapy-induced neuropathy predominantly manifested as sensory symptoms (paresthesia, numbness) in a glove-and-stocking distribution (72%) and motor weakness in the distal extremities (40%).

Diagnostic findings:

Nerve conduction studies (NCS) revealed evidence of axonal neuropathy in 65% of diabetic neuropathy cases and demyelinating features in 30% of chemotherapy-induced neuropathy cases. Electromyography (EMG) findings showed denervation patterns in distal muscles in 82% of alcoholic neuropathy patients [14].

Treatment outcomes:

Patients with diabetic neuropathy who underwent intensive glycemic control and received neuropathic pain medications showed a 30% improvement in neuropathic symptoms after 6 months of treatment. Those with chemotherapy-induced neuropathy who underwent dose modification or cessation of neurotoxic agents experienced partial resolution of symptoms in 50% of cases. Alcoholic neuropathy patients who achieved sobriety and received nutritional supplementation demonstrated stabilization of neuropathy progression and modest improvement in motor function.

Complications and prognosis:

Patients with severe diabetic neuropathy had an increased risk of foot ulcers (22%) and lower extremity amputations (5%) over a 2-year follow-up period. Chemotherapy-induced neuropathy patients who developed motor deficits had a longer recovery time and poorer functional outcomes compared to those with sensory symptoms alone. Hereditary neuropathy cases showed variable progression rates, with some patients experiencing significant disability while others remained relatively asymptomatic.

Discussion:

Isoniazid is a well-known cause of peripheral neuropathy, typically presenting as a symmetrical sensorimotor polyneuropathy affecting the distal extremities. The mechanism of INH-induced neuropathy is thought to involve interference with pyridoxine (vitamin B6) metabolism, leading to neuropathic symptoms. Early recognition, discontinuation of the offending agent, and supportive care are crucial in managing drug-induced neuropathies.

Conclusion

This case highlights the importance of considering medication-related causes, such as isoniazid, in patients presenting with peripheral neuropathy, especially in the context of anti-TB therapy. Timely intervention and appropriate adjustments to treatment can lead to favorable outcomes and prevent further neurological complications.

Acknowledgment

None

Conflict of Interest

None

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