

Steering Peripheral Neuropathy Workup

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KEYWORDS

• Peripheral neuropathy • Polyneuropathy • Etiologic workup • Pattern recognition

KEY POINTS

- Having diagnosed peripheral polyneuropathy, the astute neuromuscular physician proceeds with an informed and targeted etiologic investigation, which is paramount to guiding available treatment, rather than resigning immediately to palliative care strategies.
- Pattern recognition is a useful tool for investigating the underlying etiology of peripheral neuropathy, using Barohn and Amato's 3-6-10 approach to categorize into 10 distinct phenotypic patterns that inform a more targeted etiologic workup.
- Cryptogenic idiopathic neuropathy accounts for 25% of patients with peripheral neuropathy, yet after additional workup, a proportion can be attributed to hereditary forms, immunologic causes (acquired demyelinating polyneuropathies, eg, chronic inflammatory demyelination polyradiculoneuropathy and variants), paraproteinemia, or undiagnosed medical conditions.
- Diagnostic workup should be guided by history, examination, electrodiagnostic findings, laboratory testing, and additional assessment tools. A balanced view of cost, risk, and benefit enables clinicians to capitalize on the diagnostic clues to achieve not just a diagnosis, but clinical value.

INTRODUCTION

Physicians commonly encounter peripheral neuropathy regardless of specialty or subspecialty, owing to its prevalence in the general population of 1% to 3%, increasing to 7% in those older than 50.¹ Unfortunately, many of the known etiologies of polyneuropathy cause irreversible peripheral nerve pathology. Lured by the perception that little can be offered, time-pressured clinicians may fail to see value in the workup of peripheral neuropathy. Additionally, peripheral neuropathy may be erroneously diagnosed based solely on reported symptoms in the face of known risk factors and assumptions about causation. Among those referred to specialists, the etiology for 25% of patients with peripheral neuropathy remains elusive despite a careful search,

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aply ascribing the term “cryptogenic” to idiopathic neuropathy.² After additional workup, however, a proportion can be attributed to hereditary forms, immunologic causes (acquired demyelinating, eg, chronic inflammatory demyelination polyradiculoneuropathy [CIDP] and variants), paraproteinemia, or undiagnosed medical conditions.²⁻⁴

Learning to recognize the various clinical and electrodiagnostic patterns of peripheral neuropathy enables a targeted approach to etiologic investigation, and subsequently guides patient discussions of self-management, disease course, and prognosis. Moreover, as advancements in neuropathology and pharmacotherapy inform the many etiologies of polyneuropathy, it is imperative for clinicians to identify the underlying etiology to appropriately guide treatment options and prevent complications.

PATHOPHYSIOLOGY OF PERIPHERAL NEUROPATHY

Polyneuropathy refers to pathology affecting multiple peripheral nerves and involves the cell body, axon, myelin sheath, or a combination thereof. Regardless of the etiology of peripheral neuropathy, pathophysiology devolves into predictable patterns of response to injury: demyelination, axonal degeneration, or a combination of both.

Demyelination can have a focal segmental distribution, such as neurapraxia, when compression is insufficient to damage axons, yet leads to injury involving a focal segment of myelin, resulting in action potential failure (conduction block) or conduction velocity slowing. Typical examples include acute compression and chronic entrapment. Generalized demyelination results in conduction block and/or temporal dispersion in multiple nerves often asymmetrically and in anatomic locations outside common areas of compression/entrapment.

Axonal (Wallerian) degeneration, represented by axonotmesis and neurotmesis, results from prolonged, focal crush injury or transection resulting in disintegration and removal of axon and myelin distal to injury followed by an alteration of neural properties proximal to injury (so-called “dying back”), and ultimately, cell body death. Axonopathy also can be the result of toxic degeneration or a generalized insult to the peripheral nervous system.

Many conditions affect both peripheral nerve myelin and axon, but ultimately, Wallerian axonal degeneration represents the final common pathway of neuronal injury. With significant axonal loss, secondary demyelination occurs, and in cases of severe demyelination, axonal injury also occurs. Furthermore, acute conduction block, axonal loss of varying durations, and chronic muscle atrophy can all manifest as amplitude loss on motor conduction studies. In chronic polyneuropathy, this often obscures the initial distinguishing pathophysiologic features that might steer toward a specific underlying etiology, contributing to the diagnostic quandary.

ASSESSMENT OF PERIPHERAL NEUROPATHY

History

Because the diagnosis of peripheral neuropathy relies heavily on pattern recognition, a thorough history will provide initial clues⁵:

- Symptoms: onset, timing, character, severity, location/distribution and symmetry, course, exacerbating and relieving factors. Nocturnal foot and leg pain is a common complaint, yet not well understood. Many patients describe hypersensitive dysesthesias on contact with footwear or bed sheets.

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