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Neuropathic Pain

FACT SHEET

Identifying Neuropathic Pain in the Clinic: A Guide for Clinicians

- **Andreas C. Themistocleous, MBBCh, PhD,** Nuffield Department of Clinical Neuroscience, University of Oxford, Oxford, UK.
- **Annina Schmid, PhD,** MManipTher, MMACP, Nuffield Department of Clinical Neuroscience, University of Oxford, Oxford, UK.
- **Eva Sierra-Silvestre, PhD,** Nuffield Department of Clinical Neuroscience, University of Oxford, Oxford, UK.
- **Xavier Moisset, MD, PhD,** Neuro-DoI, Inserm/Université Clermont Auvergne, Clermont-Ferrand, France.
- **Sandra Sif Gylfadottir, MD, PhD,** Danish Pain Research Center, Department of Clinical Medicine, Aarhus University and Department of Neurology, Aarhus University Hospital, Aarhus, Denmark.
- **Joel Fundaun, DPT, DPhil,** Department of Anesthesiology, Perioperative, and Pain Medicine, Stanford University School of Medicine, Stanford, CA, USA.
- **Daniella M Menichella, MD, PhD,** Feinberg School of Medicine, Northwestern University, Chicago, USA.

Importance of diagnosing neuropathic pain

The International Association for the Study of Pain (IASP) defines neuropathic pain as pain caused by a lesion or disease of the somatosensory nervous system[7]. This definition distinguishes neuropathic pain from nociceptive pain arising from tissue injury or inflammation. Accurate identification of neuropathic pain in clinical practice is essential because it directly influences diagnosis, treatment selection, prognosis, and patient education. Neuropathic pain remains under-recognized or misclassified, particularly when it coexists with nociceptive pain[3].

Approach to diagnosis

Identification of neuropathic pain relies on careful integration of the patient's history and physical examination, supported by diagnostic investigations when appropriate[3]. The IASP Neuropathic Pain Special Interest Group (NeuPSIG) has proposed a grading system to improve diagnostic clarity and consistency[4]. The grading system applies when the history suggests pain arising from neurological pathology rather than non-neural tissue damage. It integrates symptom characteristics, neuroanatomical plausibility, examination findings, and confirmatory tests. As with most clinical guidelines, this framework has certain limitations, but it offers a helpful approach to increase the certainty of identifying or excluding neuropathic pain. Guidelines are designed to inform clinical practice rather than replace it, and their recommendations should be interpreted alongside clinical judgment and the individual patient context.

To reach the first level of diagnostic certainty for ***possible neuropathic pain***, two criteria must be fulfilled. First, there must be a history suggestive of a relevant lesion or disease affecting the somatosensory nervous system. This may include clearly identifiable events such as herpes zoster, traumatic nerve injury, or spinal cord injury, where pain develops immediately or within weeks. In contrast, conditions such as diabetic polyneuropathy may produce pain only after prolonged latency. While the interval between lesion onset and pain varies, a close temporal relationship strengthens diagnostic likelihood. In some disorders, pain or sensory symptoms represent the sole manifestation, as seen in trigeminal neuralgia.

In addition to temporal development, symptom characteristics should be considered. Characteristic symptoms include burning, shooting, stabbing, or electric shock–like pain, often accompanied by pins and needles. Both spontaneous pain and evoked phenomena occur. Positive sensory symptoms include paresthesia/dysesthesia (nonpainful/painful altered sensation such as tingling), and allodynia (pain evoked by normally nonpainful stimuli); negative symptoms include numbness or sensory loss. The combination of multiple characteristic features increases diagnostic probability. Screening tools such as DN4[2], painDETECT[6], or LANSS[1] may assist identification but should complement, not replace, clinical assessment.

Second, the distribution of pain should be neuroanatomically plausible (acknowledging variation in dermatomes and extraterritorial spread) and consistent with the suspected disease site within the

somatosensory nervous system. Neuropathic pain broadly conforms to recognized distributions. Examples include radiating pain that approximately follows dermatomal distributions in painful radiculopathy, peripheral nerve territories in focal neuropathies, a length-dependent "glove-and-stocking" distribution in polyneuropathy, or central patterns following stroke or spinal cord injury[5]. Pain outside recognizable neuroanatomical boundaries reduces diagnostic likelihood for neuropathic pain, although atypical presentations occur.

The next level of certainty, ***probable neuropathic pain***, requires a focused neurological examination demonstrating sensory abnormalities within a plausible distribution. Comparison of the painful area with a non-painful reference site may reveal sensory loss to light touch, pinprick, vibration, or temperature. Sensory gain phenomena, including allodynia or hyperalgesia, may also occur. Motor or autonomic signs may accompany sensory findings depending on lesion location.

Interpretation requires clinical context. Some neuropathic pain conditions are episodic, and sensory abnormalities may not be detectable at assessment. Central sensitization may also extend hyperalgesia or allodynia beyond the lesion site, reducing anatomical specificity. While positive sensory signs such as hyperalgesia are less predictive for neuropathic pain, they may still represent the predominant sensory features in certain neuropathic conditions. By contrast, sensory findings in non-neuropathic conditions, whether positive or negative, generally lack consistent neuroanatomical distribution and reproducibility.

The sensory examination has limitations. Clinicians cannot reliably assess deep somatic or visceral sensory function; therefore, certainty beyond "possible" neuropathic pain is rarely achievable in such conditions. Nevertheless, reproducible sensory abnormalities within a plausible neuroanatomical distribution provide strong support for probable neuropathic pain.

The final level of certainty, ***definite neuropathic pain***, requires confirmatory diagnostic evidence. Examples include: nerve conduction tests that can demonstrate a focal slowing in carpal tunnel syndrome; a skin biopsy showing reduced intraepidermal nerve fiber density in small fiber neuropathy; or magnetic resonance imaging showing demyelinating lesions in multiple sclerosis. Extensive testing is not necessary in all cases. Instead, investigation should follow clinical reasoning and consider whether findings are likely to influence management. In resource-limited settings, clinicians may not have access to diagnostic tests, and a diagnosis of probable neuropathic pain is usually sufficient to initiate treatment.

Different causes of chronic pain can coexist

Neuropathic pain frequently occurs alongside nociceptive pain, resulting in mixed pain presentations. Common examples include low back pain with neuropathic leg pain in painful radiculopathy and cancer-related pain, also causing neuropathic pain due to nerve compression or infiltration. In these situations, multiple pain mechanisms may contribute to the overall symptom profile, and a multidisciplinary approach is needed for optimal management.

Conclusion

Identification of neuropathic pain in clinical practice requires careful evaluation of the pain history, sensory symptoms, findings from neurological examination, and evidence of pathology affecting the somatosensory nervous system. Applying established diagnostic principles and grading criteria supports differentiation between neuropathic and non-neuropathic pain, recognition of mixed pain presentations, and selection of appropriate treatment strategies. Neuropathic pain should not be viewed as a binary diagnosis but rather as a spectrum of diagnostic certainty, reflecting the inherent complexity and uncertainty of clinical pain assessment. Improved identification of neuropathic pain ultimately contributes to better patient outcomes and more efficient use of healthcare resources.

Complementary Resource

A companion video resource is available to reinforce the key principles of neuropathic pain identification and to illustrate the NeuPSIG grading approach in clinical practice: https://www.youtube.com/watch?v=mVxA_8U4ekE.

Disclosures

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