



ESRA Italian Chapter

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Peripheral neuropathic pain Differential diagnosis









Disclosures

Consultant for

- Abbott
- Boston Scientific

diagnosis nociceptiv heuropathic/ Differential



A Comprehensive Algorithm for Management of Neuropathic Pain

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Differential diagnosis of peripheral neuropathic pain Etiological



- Polyneuropathies: metabolic, autoimmune, hereditary, idiopathic
- Mononeuropathies: due to entrapment, post-traumatic, postsurgical, amputation or continuity neuroma, multiple
- Radicolopathies: due to compression (+/- inflammation), infective, metabolic

Etiological diagnosis is related to therapy?



Andrea M. Trescot *Editor*

Peripheral Nerve Entrapments

Clinical Diagnosis and Management





Differential diagnosis Pathogenetic mechanisms

- Since studies on the effectiveness of pharmacological therapies in neuropathic pain of various etiologies have demonstrated unsatisfactory responses, a therapeutic approach based on pathogenetic mechanisms has been proposed
- Forstenpointner et al propose to group patients in three main clusters based on sensory profiles (QST)
 - "Sensory loss": prevalence of negative signs to all stimulation modalities
 - "Thermal hyperalgesia": prevalence of positive signs (allodynia)
 - "Mechanical hyperalgesia": loss of thermal sensitivity (small fibers) and allo allodynia to mechanical stimuli

Bedside clinical evaluation

Bedside sensory examination.

Modality	Bedside assessment
Touch	Cotton bud or ball, painter's brush
Vibration	Tuning fork
	Pin, toothpick, cocktail stick
Cold	Cold metal, tube with cold water, cloth with surgical spirit, Lindblom roller ²¹
Warm	Warm metal, tube with warm water, Lindblom roller ²¹

Finnerup NB et al. Pain, 2016

From clusters to pathogenetic mechanisms

Sensory loss: loss of small and large diameter fibre function, paradoxical heat sensation to QST. Spontaneous pain can be due to ectopic action potentials generated in proximal sites of lesioned nociceptors: dorsal root ganglion or deafferented central neurons

Thermal hyperalgesia: function of small and large fibres relatively preserved in combination with thermal allodynia and minimal mechano-dynamic allodynia (DMA). Spontaneous hyperactivity of preserved nociceptors can be responsable for spontaneous pain and can induce second order neurons sensitization

Mechanical Hyperalgesia: predominant loss of function of small and medium diameter (thermal hypoesthesia) associated with pressure hyperalgesia, pinprick hyperalgesia, and severe DMA. Central sensitization is prevalent for mechanical stimuli. In this group, spontaneous pain is related to spontaneos activity in the nociceptive system, in the periphery or in the spinal cord

R. Baron et al. Pain, 2017; 58: 261–272

Sensory profiling and etiology



R. Baron et al. Pain, 2017; 58: 261–272

From physiology to pathophysiology



Laura Demartini. All'origine del dolore

Sites of possible functional alterations

Reduced transduction threshold For increased receptor expression (eg.: TRPV1) or modified functionality (sensitization) **Reduced coding** threshold due to altered ionic Ectopic action potential generation in channel expression the site of lesione, in the DRG or II Spontaneous pain or order neuron evoked by non-noxious

stimuli

Proceeding in differential diagnosis The stimulating factors





Mechanical stimuli in the peripheral territory, on the ectopic site or the ganglion

Non-painful mechanical stimuli, eg.: disk pressure on a lesioned root or ganglion (ectopic site) or Tinel on the median nerve

Inflammatory mediators and/or hypoxia make the ectopic site hypersensitive to stimuli. Body temperature can became an effective stimulus

For a targeted therapy

- Hypothesize the diagnosis of neuropathic pain based on the diagnostic criteria^{1,2}
- Identify the site of injury
- Identify the pathogenetic mechanisms and the site of altered functionality
- Identify the presence of stimulating factors or sensitization
- Choose therapies base on mechanisms and disite of action

- 1) Treede RD et al. Neurology 2008.
- 2) Finnerup Nd et al. Pain, 2016.

Diagnostic tools

- Identify the site of injury
- Identify the pathogenetic mechanisms and the site of altered functionality
- Identify the presence of stimulating factors or sensitization

Diagnostic/therapeutic injections with local anesthetic and steroid

Diagnostic instruments



RL-68 y M

Postherpetic neuralgia since 20 y in the territory T1-T3 on the left Continuous spontaneous pain (NRS 7) plus pain parxysms evokes by light touch (NRS 10)

At clinical evaluation, mechanical allodynia in the painful territoy, light cold allodynia in the same territory, cold anesthesia in the axillary region, warm anesthesia in the painful area

Cluster Mechanical Hyperalgesia with prevalen loss of small fibres function

RF stimulation on T1 evoked paresthesias in the forearm but not in the arm; on T2, at 1 V pain was evoked in painful territory without paresthesia demostrating a prevalent large fibres lesion; on T3, stimulartion at 0,4 V evoked paresthesias in the caudal part of painful area

PRF on DRG: a diagnostic instrument for ganglion functionality

Another hystory



PC-42 y F

At the age of 16, amputation at the middle third of left thigh for a sarcoma Postoperative period complicated by infection with pain in the lateral posterior aspect of the foot

In the following years episodes of severe pain in the same area with long periods without pain

Now she presents 3-4 episodes/year of severe pain lasting about 48 h interferin with every activity

Why every 4 months?

How to treat this patient?



