NEUROPATHIC PAIN

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Introduction

Neuropathic pain (NP) is a major disability in common neurological diseases, such as neuropathy, myelopathy, multiple sclerosis, or stroke (Table 1). Pain is complex sensation, strongly modulated by cognitive influences, and understading nocioceptive function and dysfunction is a hard task not only for „general" neurologists but also for all pain specialists[1-2]. The distiction between nocioceptive and neuropathic pain mechanisms is often a difficult clinical exercise. The underlying pathophysiological mechanism that result in chronic pain can only be inferred, and this inference is based primarily on verbal description supported by examination and investigations[2]. NP is a neurological disorder with a high prevalence, thus it is essential that neurologists get involved in its diagnosis and manamgement[1], and of course that not exlude other specialists for this „job", including phamily practitioners.

Table 1. Some common causes of neuropathic pain

Periferal nerve lesion or dysfunction
Painful diabetic neuropathy
Post-herpetic neuralgia
Post-surgical pain (including post-mastectomy and phantom limb pain)
Complex regional pain syndrome
Trigeminal neuralgia
Chemotherapy-induced neuropathy
Neuropathy secondary to tumour infiltration
Central nerve lesion or dysfunction
Central post-stroke pain
Multiple sclerosis pain
Spinal cord injury pain

Epidemiology

In 1991 Browsher[3] suggested that the prevalence of neuropathic pain in the Unided Kingdom (UK) general population was probably about 1% , and in USA similar estimate was in 1997[1]. A recent UK study[1] found that the population prevalence of chronic pain of predominantly neuropathic origin was 8.2% in adults, and it was concluded that neuropathic pain in the community seems to be more common than previously estimated. It assumed that prevalence of NP can be expected to increase in the future as the population ages.

Diagnosing neuropathic pain in clinical practice

Neuropathic pain can be difficult to identify becuse it is subjective, evidence of neuropathy does not always imply neuropathic pain, and multiple pathological mechanisms ae variously expressed, some of which overlap with nocioceptive pain[2].

The examination of a pain patient aims at clarifying underlying disease and understanding whether the pain is nocioceptive, neuropathic, psychogenic, or a combination of such. In case of neuropathic pain, abnormal sensory finding should be neuroanatomically logical, compatible with a definite lesion site, and location, quality and intensity of pain should be assessed. It means that neurological examination in suspected neuropathic pain should include quantification and maping of motor, sensory and autonomic phenomena to identify all signs of neurological dysfunction[1].

Although there are no validated studies on bedside examination, good clinical practice teaches that in pain patients a through neurological examination

is invaluable – the sensory testing being the most important part of it – and is prleiminary to any quantitative assessment[1].

Quantitative sensory testing (QST) may be defined as the analysis of perception in response to external stimuly of controlled intensity. So, QST measures detection thresholds (i.e. sensory responses) to thermal and electrical stimuli. The most sensitive of these is thermal threshold sesitivity (TTS) testing which involves the application of a thermode to the skin[2]. According to EFNS Panel on Neuropathic Pain[1], although QST is not conclusive to demonstrate neuropathic pain, it is helpful to quantify the effects of treatments on allodynia and hyperalgesia.

It is recommended to rate the intensity and the unpleasantness of pain separately[5]. The intensity of the different pain components that the patient may report (spontaneous ongoing pain - burning or deep pressuure pain, spontaneous paroxysmal pain, dysestheseae, paresthesiae) or the evoked pains (allodynia and hyperalgesia), as wll as pain worsening with movement, should be rated separately, but using the sam scale. If different pain components involve different territories, these can be documented on a template body map. The simlest scales are probably the best. Whereas verbal raing (VRS) scale is found to be easier by many patients, visual analog scale (VAS) is more apt to treatment trials because it permits parametric statistics. The Likert 0-10 numerical rating is good compromise[1].

Treatment of neuropathic pain

Although monotherapy is the ideal approach, rational polypharmacy is often pragmatically used. Several classes of drugs are moderately effective, but complete or near-complete relief is unlikely. Antidepressants and anticonvulsants are most commonly used. Opioid analgesics can provide some relief but are less effective than for nociceptive pain; adverse effects may prevent adequate analgesia. Topical drugs and a lidocaine-containing patch may be effective for peripheral syndromes. Sympathetic blockade is usually ineffective except for some patients with complex regional pain syndrome (Ad hoc Committee of the Crotian Society for Neurovascular Disorders and Croatian Medical Association)[6]. According to EFNS Panel Neuropathic Pain[7]. the main peripheral pain conditions respond similarly well to tryciclic antidepressants, gabapentin and pregabalin, but some conditions, such as HIV-associated polyneuropathy, are more refractory. There are only few studies on central pain, combination therapy, and head-to-head comparison. It is recommended for future trials to assess quality of life and pain symptoms or signs with standardized tools.
This presentation gives an overview of diagnosing and treatment of neuropathic pain syndromes.

References

1. CRUCCU G, ANAND P, ATTAL N, GARCIA-LARREA L, HAANPAA M, JORUM E, SERRA J, JENSEN TS. Assesment of neuropathic pain. In: Hughes R, Brainin M, Gilhus NE (eds). European Handbook of Neurological Management. Oxford: Blackwell Publishing, 2006: 109-122.
2. BENNETT MI. Diagnosing neuropathic pain in clinical practice. In: Bennett M (ed). Neuropathic pain. Oxford: Oxford University Press, 2006: 25-35.
3. BROWSHER D. Neurogenic pain syndromes and their management. Br Med Bull 1991; 47: 644-646.
4. TORRANCE N, SMITH BH, BENNETT MI, LEE AJ. The epidemiology of chronic pain of predominantnly neuropathic origin. Results from a general population survey. J Pain 2006; 7(4): 281-289.
5. SMITH WB, GRACELY RH, SAFER MA. The meaning of pain: cancer patient's rating and recall of pain intensity and affect. Pain 1998, 78: 123-129.
6. DEMARIN V, BASIĆ-KES V, ZAVOREO I, BOSNAR-PURETIĆ M, ROTIM K, LUPRET V, PERIĆ M, IVANEC Z, FUMIĆ L, LUSIĆ I, ALEKSIĆ-SHIHABIS A, KOVAC B, IVANKOVIĆ M, SKOBIĆ H, MASLOV B, BORNSTEIN N, NIEDERKORN K, RUNDEK T. Recommendations for neuropathic pain treatment. Acta Clin Croat 2008; 47 (3): 181-191.