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Pain management of painful legs

Schmerztherapie bei schmerzenden Beinen

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ABSTRACT

Background Leg pain is a common problem in daily phlebological practice.
Methods The manuscripts shows an actual overview of principles in the management of pain.

ZUSAMMENFASSUNG

Hintergrund Beinschmerzen sind ein häufiges Symptom in der phlebologischen Praxis.
Methode in der vorliegenden Arbeit wird ein strategisch sinnvolles Management von Schmerzen/Beinschmerzen dargestellt.

Introduction

Patients with painful legs present a broad differential diagnostic field with respect to determining the cause and its appropriate treatment. But, on the other hand, the question arises as to how the symptom itself can best be managed.

EXAMPLES OF THE DIFFERENTIAL DIAGNOSIS OF PAINFUL LEGS, MODIFIED FROM [1, 2]

- Vascular causes:
  - venous: acute venous occlusion, superficial and deep vein thrombosis, chronic venous insufficiency, varicose veins
  - arterial: arterial occlusion, arteriosclerosis
- Lymphatic causes: inflammation of the lymphatics, lymphatic stasis, lymphoedema
- Skin diseases
- Orthopaedic causes: osteoarthritis, pelvic tilt, aseptic bone necrosis, osteoporosis, spinal diseases, herniated discs, sciatica, leg deformities
- Rheumatic causes: rheumatoid arthritis, collagen diseases
- Inflammation: bones, tendons, joints
- Muscular causes: myogelosis, myositis, muscle diseases, muscle stiffness, calf cramps
- Traumatic causes: muscle tears, cartilage injuries, fractures, sprains, dislocations, ligament/tendon injuries, complex regional pain syndrome (CRPS), compartment syndrome
- Neurological causes: multiple sclerosis, polyneuropathy, nerve compression syndrome
- Restless legs syndrome: uncontrollable urge to move the legs, especially at night
- Psychogenic causes: depression
- Metabolic causes: gout
- Tumours: sarcoma

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The pathophysiology of leg pain is multifaceted and the subsequent pharmacotherapeutic and non-drug management of pain is just as complex.

According to the International Association for the Study of Pain (IASP) the definition of pain that has been valid since 1994 is "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" [3].

This description indicates the ever-present subjective and emotional character of pain as an unpleasant experience. Even though there is an association with actual or described tissue lesions, the action potentials that run through the nociceptive system from the impulse-triggering source to awareness are subject to a large number of individual modulations. However, it cannot be explained by the still widely held model of a bell pull between the painful trigger and perception of the pain, which was first formulated by Descartes in 1632 [4]. It was not until three hundred years later that Melzack and Wall in 1965 proposed the image of a regulatory "gatekeeper" in the posterior horn of the spinal cord: here, inhibitory mechanisms modulate ascending nociceptive transmission in the central nervous system (CNS). This is known as the gate control theory [5].

Since then research has brought further insight into the complex processes in the recognition of harmful events, the processing and integration of the triggered signals, and the development of awareness of this information – but we are still far from presenting a comprehensive model [6].

Classification of leg pain

The symptoms should first of all be classified according to the time dimension:

- Acute pain (present for less than six months)
- Chronic pain (for six months or more)

Pain can also be categorised by the pathophysiological processes concerned:

- Nociceptive pain
- Neuropathic pain
- Mixed pain – a combination of the two

Methodical approach to the diagnostic investigation and treatment of pain

General conditions: The therapeutic approach – take patients with pain seriously

Even though the experience of pain usually arises from a functional or structural tissue lesion, it is also significantly impacted by the patient's subjective determinants. In addition, this impact becomes more noticeable the longer the pain persists. In order to obtain a better understanding, therapists ask questions such as: has the patient already experienced a similar event? How did they cope with it? What was the result? What situations cause the pain? Do they have useful resources available? Is there a good social network? What do they associate with the pain? Are they well informed? Do they trust the therapist and their own strategies for combatting the pain?

The more similar patients are to their therapists in dealing with the pain, the greater the chances of successful pain management. The starting point – namely how well the therapist understands the patient's behaviour towards the pain and considers it plausible – determines how seriously the patient concerned is taken in his or her suffering.

The results of more sophisticated diagnostic investigations play a subordinate role. The diagnosis of pain is made mainly on verbal and non-verbal information, as well as the clinical examination of the patient, aided by the evaluation of questionnaires.

Acute or chronic pain?

To establish the multidimensionality of pain, we first have to distinguish whether the pain is acute or has already become chronic. While acute pain can usually be well controlled by pharmacological measures, considerable individually established modulatory changes at the biological, psychological and social level have to be reckoned with when the pain has persisted for three to six months. These aspects have to be addressed accordingly. The chances of success with analgesic therapy alone dwindle as the pain becomes progressively chronic. The patient instead needs a coordinated combination of counselling with corrective and motivational treatment from doctors, psychotherapists, physiotherapists and other professional groups.

The goal is often to guide patients back to self-determination of their lives (i. e. to overcome the passive expectation that an expert will take the pain away) and restore their sense of responsibility. In this respect, medication may help by lowering the inhibition threshold, while physiotherapists can assist in finding the appropriate physical activity to overcome the fear of movement.

The treatment of chronic pain is therefore time consuming and expensive. This makes prompt efficient treatment in the acute stage is all the more important, as well as prophylaxis to prevent the pain becoming chronic.

Nociceptive and/or neuropathic pain?

The choice of medication depends on whether the pain is generated and conducted by an intact nervous system and the extent to which normal action potentials are generated by undamaged nociceptors and transmitted along intact peripheral and central tracts, i. e. nociceptive pain. This is the most common type of pain, and often occurs in association with wounds or operations. There is tissue damage with inflammatory processes. Prostaglandins play an important role.

However, if the nervous system itself is pathologically altered by mechanical or metabolic injury (for example, by polyneuropathy or amputation), the symptoms are those of neuropathic pain. The patient's description of pain with a burning/shooting character points towards this type of pain.

Combinations of the two types are common (e. g. with ischaemic conditions).

While nociceptive pain responds well to non-opioid drugs (which are almost exclusively prostaglandin synthesis inhibitors) and opioids, the treatment of neuropathic pain is often unsatisfactory. Medication includes antidepressants, which improve the efficiency of the descending inhibition by inducing an increase in the serum levels of the neurotransmitters serotonin and noradrenaline, and

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anticonvulsants that, as sodium and potassium channel blockers, increase the stability of the resting potential of the transmitting nerve structures. Both these classes of drugs have to be introduced gradually, titrating the dose, and do not become effective until they have been taken daily for a few weeks. Rapid effective treatment of peaks of neuropathic pain is therefore not really possible. The patient has to show good compliance with treatment, since the possible side effects start as soon as the medicine is taken but the desired pain relief does not kick in for a couple of weeks.

ANTIDEPRESSANTS AND ANTICONVULSANTS IN NEUROPATHIC PAIN

Both classes of drug have to be introduced gradually. Side effects may be experienced right from the first dose but the analgesia does not take effect for 1–2 weeks. Diverse interactions and contraindications have to be taken into account. The patient must also be well informed (i. e. on the use of antidepressants for pain relief).

Antidepressants

Most of these drugs have a sedative effect so they should be given in the evening, e. g. start amitriptyline with 10–25 mg in the evening and increase every 3–4 days up to 75–100 mg. Caution in elderly people: anticholinergic side effects. Alternative: mirtazapine or duloxetine. Both these drugs have an anxiolytic effect and lighten the patient's mood, which positively impacts their ability to cope with the pain. Duloxetine (starting dose 30 mg) is therefore given in the morning because of its drive-enhancing affect and mirtazapine (e. g. 15 mg) is usually prescribed in the evening. Pure selective serotonin reuptake inhibitors (SSRIs) such as citalopram have no effect on neuropathic pain.

Anticonvulsants

Examples of drugs: carbamazepine at a dose from 300 mg/day to a maximum of 1200 mg/day (may induce enzymes in the liver) and gabapentin from 100 mg once daily to a maximum of 1200 mg three times daily, increasing the dose every 2–3 days.

Twice daily dosage: titrate the dose of pregabalin from 75 mg twice daily to a maximum of 300 mg twice daily. Pregabalin is also approved for anxiety states. All the products mentioned cause the patient to feel tired.

Non-opioids and opioids are usually not effective in neuropathic pain or only mildly effective.

Depending on the pathophysiology of the pain, a combination of pharmacological mechanisms of action is often required. While non-opioids such as ibuprofen, diclofenac, metamizole, and paracetamol reduce cyclo-oxygenase and hence decrease prostaglandin synthesis, opioids inhibit the pre- and post-synaptic conduction of action potentials. Antidepressants and anticonvulsants work on other principles, as previously mentioned.

All the mechanisms of action can be combined: in each case, the safety profile and risks have to be weighed up against the patient's comorbidities and checked for compatibility.

Conflict of interest

The author declare that they have no conflict of interest.

References

[1] https://www.medizin-kompakt.de/beinschmerz

[2] http://www.productivemedic.com/differential-diagnosis/limb-pain/

[3] Merskey H., Bogduk N. (Ed) Classification of Chronic Pain, Part III: Pain Terms, A Current List with Definitions and Notes on Usage. 2. ed, IASP Task Force on Taxonomy, IASP Press, Seattle, 1994: 209–214

[4] Descartes R. L'homme. Angot, Paris 1664, reprinted from "Oeuvres de Descartes", XI, Adam and Tannery, Paris, 1957: 119–209

[5] Melzack R, Wall PD. Pain mechanisms—a new theory. Science 1965; 150: 971

[6] Egloff N, Egle UT, von Kähnel R. Weder Descartes noch Freud? Aktuelle Schmerzmodelle in der Psychosomatik. Praxis. Hogrefe AG. 2008; 97: 549–557. Online 2012 https://doi.org/10.1024/1661-8157.97.10.549.

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