Painful post-Traumatic Trigeminal Neuropathy (PTTN)

Definition

Unilateral facial or oral pain following injury to the trigeminal nerve, with other symptoms and/or clinical signs of trigeminal nerve dysfunction. PTTN is characterized by continuous burning and/or shooting pain in an area that has a clear history of trauma associated with the onset of pain. Neurophysiological, psychophysical, and imaging modalities should support damage to the somatosensory system. Clinically, there are positive and/or negative neurological signs and symptoms, and these are hallmark of PTTN.

Epidemiology

Unclear what the exact prevalence in the population is. The incidence following injuries to the peripheral branches of the trigeminal nerve following implants, 3rd molar extractions, orthognathic surgery, mid-face fractures and root canal therapy is around 3-5%. Considering the wide prevalence of such injuries and procedures PTTN is suspected to be common. Although it may occur at any age typical age of onset is around 50 years, and patients are largely female [1, 2].

Pathophysiology

The pathophysiology of PTTN involves a cascade of events in nervous system function and form. Generally events are time dependent, progressing from the peripheral to the central nervous system. These events include alterations in functional, biochemical and physical characteristics of neurons and glia on a background of genetic sensitivity.

Clinical Features

Location, radiation: Unilateral pain in the area of injury, or at the distal dermatome of an injured nerve, reflecting the anatomy of the injury. Occasionally may spread to adjacent dermatomes but unless both sides have been injured seems to remain unilateral.

Character: Burning. May have shooting like qualities.
Severity: Moderate to severe, mean visual analog scale ratings 5-8 [3, 4].

Duration: Mostly pain is continuous and patients may report superimposed attacks of short-lasting pain.

Accompanying Features: Positive (e.g. dysaesthesia) and negative neurologic symptoms (e.g. numbness) [5] in the area of injury and the distal dermatome. Hyperalgesia and other sensory changes may also be found in extratrigeminal sites suggesting more extensive changes in central somatosensory processing. Thermal modalities are usually preserved [6]. Local redness and mild swelling may be seen. Often there is a complaint of swelling from the patient which may not be verifiable clinically.

Modifying factors: Stress may increase pain. Certain areas in or around injured area may act as a trigger and when stimulated induce an attack of shooting pain. There is no latency or associated refractory period.

Psychological Comorbidity: PTTN is associated with a substantial psychosocial burden. Patients with more severe pain demonstrate elevated levels of depression and pain catastrophizing, as well as substantially reduced quality of life (QoL) and coping efficacy levels. Pain intensity is a good predictor for QoL measures and emotional problems such as depression.

Prognosis: Poor. There is a limited response to available and recommended pharmacotherapeutic interventions[7]. Peripheral surgical interventions aimed at pain relief are generally contraindicated, however microsurgical nerve repair may improve sensation.

Investigations
Depending on the type of injury appropriate imaging studies are usually be needed to assess the location and extent of injury. Quantitative sensory testing should be performed. Neurophysiologic testing is recommended but not always readily available.

Management
Pharmacotherapy
The mainstays of pharmacologic treatment of PTTN remain the antiepileptic drugs (AEDs) and the tricyclic (TCAs) and newer antidepressants [8, 9]. In contrast to the traditional 50% pain reduction for clinical significance, research has shown that about a 30% reduction represents meaningful pain relief for NP patients. This is difficult to attain.

Based on current evidence an antidepressant possessing mixed serotonin/noradrenaline (e.g. amitriptyline and nortriptyline) or serotonin and noradrenaline reuptake inhibition (e.g. venlafaxine and duloxetine) is the usual starting therapy. The newer antidepressants drugs, such as duloxetine, are effective in and since they have fewer side effects than the TCAs, are an alternative.

If the patient's medical history precludes the use of an antidepressant, the anticonvulsants pregabalin and gabapentin are the recommended drugs, although they are generally inferior to the antidepressants. Failure of either of the above strategies is an indication to begin a trial of the alternate drug, if the medical status of the patient allows - i.e. move patients on antidepressants to the anticonvulsants and vice versa.

Failure of this second phase is an indication to try combined therapy, duloxetine or amitriptyline may be combined with one of the anticonvulsants such as gabapentin or pregabalin.

If the above strategy fails opioids and opioid combinations may be a viable alternative.

Surgical Treatment
The role of surgery in the management of non-painful neuropathies is well established and nerve repair may improve the level of sensation in injured patients [10]. Surgery is marginally more successful in inferior alveolar than in lingual nerve injuries [11]. Repair should be performed early, within one year of injury there are good rates of sensory recovery. The efficacy of surgery for painful trigeminal neuropathies is unclear. Further peripheral surgical procedures (exploration, further apicectomies) for PTTN may result in more pain. Unless there are specific indications we advise patients with painful traumatic neuropathies not to undergo further surgery.

Cognitive Behavioral Therapy
Neuropathic pain is associated with comorbid anxiety and depression. A meta-analysis did not show a significant effect of CBT on pain intensity and quality of life measures in neuropathic pain. Notwithstanding, psychotherapeutic support should be offered to distressed patients.

References

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