Surgical Options for Complex Craniofacial Pain

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KEYWORDS

• Craniofacial pain • Neuropathic pain • Surgical treatment • Peripheral field/nerve stimulation
• Spinal cord stimulation • Ganglion neuromodulation • Motor cortex stimulation
• Deep brain stimulation

KEY POINTS

- Surgical treatment of complex craniofacial pain syndromes has been shown to achieve significant pain relief (>50%) in medically refractory pain syndromes.
- Appropriate patient selection, risks, and benefits associated with each of these therapies, presence of comorbidities, sensory deficits accompanying the area of pain, and insight into the programming parameters are the key points that need to be considered before selecting these surgical options for complex craniofacial pain syndromes.
- Given the reversibility and minimal invasiveness of peripheral nerve/field stimulation, this modality is being explored at a rapid pace and is preferred by both patients and surgeons.
- Motor cortex stimulation and deep brain stimulation can be considered for patients who fail other less invasive neurostimulation therapy for complex craniofacial pain syndromes.
- There is paucity of reliable data in the literature on the efficacy of these therapies for complex craniofacial pain syndromes and prospective randomized controlled studies are warranted to establish their therapeutic value.
- Peripheral nerve/field stimulation therapy, ganglion stimulation, motor cortex stimulation, and deep brain stimulation therapy are still investigational and off-label therapies for complex craniofacial syndromes.

INTRODUCTION

Craniofacial pain is a common condition that affects approximately 10% to 25% of the adult population worldwide with a significant impact on their quality of life.[1–3] The International Headache Society (2004) classified craniofacial pain into 14 different categories, which provides a useful template in establishing a uniform clinical diagnosis.[4] Women are more frequently affected with craniofacial pain, in the ratio of 2:1.[1] The common causes of face pain include trigeminal neuralgia (tic douloureux or Fothergill disease), trigeminal neuropathic pain, and persistent idiopathic facial pain (PIFP, or atypical face pain). A population-based study reported the lifetime prevalence of trigeminal neuralgia and PIFP to be 0.3% and 0.03% respectively.[5] Of these, trigeminal neuropathic pain and PIFP are complex pain syndromes that are often difficult to manage with medications alone.

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According to the International Association for the study of pain, neuropathic pain is defined as "pain initiated or caused by a primary lesion or dysfunction in the nervous system."[6,7] Neuropathic pain can be further classified as peripheral or central (depending on the site of the disorder) and acute or chronic (lasting >3 months).[6,7] Trigeminal neuropathic facial pain (TNP) is defined as a constant burning, cramping, pricking, deep aching, or electric shock–like facial pain along the distribution of trigeminal nerve branches.[6,8] TNP is often associated with sensory dysfunctions such as paresthesia or dysesthesia, which can manifest as cold, pricking, tingling, or itching sensations along the distribution of pain.[6] The most severe form of facial pain, with complete numbness in the distribution of pain, is referred to as anesthesia dolorosa. TNP can result from surgery, traumatic injury, and herpetic infection (shingles) of the areas innervated by the branches of trigeminal nerve, including the sinuses, teeth, face, or skull.[9,10] TNP develops in a delayed fashion typically many days to months following the initial insult.[11] In addition, iatrogenic injury to the trigeminal nerve by nerve ablation, rhizotomy, or ganglion ablation to treat trigeminal neuralgia can initiate trigeminal deafferentation or neuropathic pain.

PIFP (atypical face pain) is defined as "Persistent facial pain that does not have the characteristics of the cranial neuralgias and cannot be attributed to other disorders" (International Headache society, 2004).[4] This entity was first described by neurosurgeons Frazier and Russell, in 1924.[12] This condition is often described as severe persistent unilateral facial pain that is deep or poorly localized and usually burning or crushing in nature. Furthermore, there is a normal work-up without an associated sensory loss or other neurologic deficits.[2,4] There may be a history of surgical or traumatic injury to the face, teeth, or gums before the onset; however, there is no demonstrable local cause that can endorse the persistence of this facial pain. PIFP is usually not confined within the anatomic distribution of the branches of trigeminal nerve and is often a diagnosis of exclusion.[2,13] This condition affects approximately 1 in 100,000 adults, with no clear gender predilection, although the clinical presentation is more common in women.[2]

PATHOPHYSIOLOGY OF COMPLEX FACE PAIN

Multiple mechanisms have been postulated in the pathophysiology of neuropathic face pain, which accounts for a wide variety of clinical presentations in patients with similar diseases.[6] Therefore, patients with different pain generators and clinical presentations differ in their responses to treatment and overall outcome. As such, patients with posttraumatic TNP may have a different mechanism of pain onset compared with those with a spontaneous origin of TNP.[11,14] Surgical or traumatic injury to the trigeminal nerve results in impaired functioning of both small unmyelinated and large myelinated nerve fibers with subsequent demyelination of the trigeminal nerve.[2,14] The phenomenon of abnormal temporal summation of pain signals and reduced temperature thresholds with hot/cold hyperalgesias in patients with TNP can be attributed to hyperexcitability of central neurons and hypersensitization of peripheral C fibers/free nerve endings respectively.[2,11,14] This process initiates as a result of interaction between chemicals (histamine, substance P, calcitonin gene–related peptide, glutamate, prostaglandins, bradykinins) released following tissue injury and peripheral nociceptors/free nerve endings, which subsequently can result in alteration in central pain pathways (central hypersensitization) over a period of time. Gender differences have been implicated in the interaction of chemical mediators with peripheral nociceptors and peripheral pain pathways, which may account for the higher incidence of chronic pain conditions in women.[15] The pathophysiology of postherpetic trigeminal neuropathic pain (PHN) involves dysfunction of both peripheral and central pain pathways in varying proportions in different patients.[16,17] This phenomenon can be attributed to the differences in clinical findings between patients with facial and truncal PHN and those with acute and chronic PHN.[17]

The pathophysiology of PIFP is also not completely understood. PIFP was initially thought to be a component of a somatoform disorder because most patients had associated psychiatric disorders.[2,9] However, various neuropathic mechanisms have recently been implicated in the pathogenesis of this disorder.[2] The specific pathophysiologic mechanisms underlying the onset of neuropathic pain have not yet been elucidated, but with ongoing research it might be possible to identify precise mechanisms and thus target therapy accordingly.

PAIN PATHWAYS AND NODES OF INTERVENTION

The pathways related to pain transmission are complex and include both sensory and affective components. The somatic sensations from the skin of the face, forehead, scalp up to the vertex, and mucous membranes of the nasal cavity and paranasal sinuses are carried by branches of the