Intractable pain from trigeminal neuralgia

Introduction

Trigeminal neuralgia is recognized as a cause of severe facial pain, although this usually responds to conventional strong analgesics and/or anticonvulsants. This article describes a patient literally 'petrified' by pain caused by primary trigeminal neuralgia.

Discussion

Trigeminal neuralgia was first described in 1773 among patients with excruciating bouts of unilateral facial pain (Fothergill, 1773). With an estimated incidence of only 4.5 cases per 100 000, other disease processes including migraine, dental caries and temporomandibular joint dysfunction are often mistakenly diagnosed.

The underlying cause of trigeminal neuralgia remains speculative. Evidence suggests vascular compression, predominantly from the superior cerebellar artery, contributes to focal demyelination at the trigeminal root (Prasad and Galetta, 2009). This results in ephaptic transmission (electrical cross-talk between axons) and ectopic generation of spontaneous impulses, which may arise from mechanical effects of the pulsating vessel, triggering neuropathic pain. Demyelinating disease or compression from a cerebellopontine tumour are possible secondary causes.

Opiate analgesia is generally insufficient and the anticonvulsant agents carbamazepine or oxcarbazepine are frequently used. Reports indicate that carbamazepine is effective in controlling 70–90% of cases (Shaikh et al, 2011; Wang and Bai, 2011). However, side effects, including blood dyscrasias, hepatic dysfunction and drug interactions, limit use. Alternatives include phenytoin and sodium valproate but evidence is lacking. Gabapentin and pregabalin are often also used, as is baclofen for spasm. Combining gabapentin with an anaesthetic block (weekly ropivacaine injections) into known facial trigger sites has been effective as a second-line therapy (Lemos et al, 2011).

Surgical intervention is reserved for cases with intractable pain despite maximal medical therapy. The Cochrane Collaboration has reviewed experimental and established techniques (Zakrzewska and Akram, 2011). In essence, there are interventions that aim to destroy (ablative methods) nerve fibres at one of three anatomical levels: the posterior fossa root entry zone, Gasser's ganglion (where the trigeminal nerve divides into its three branches), or peripherally at the trigger zone (*Table 1*), e.g. glycerol injection into Meckel's cavity or radiofrequency thermocoagulation. Advantages include moderate pain relief through minimally invasive day-case procedures. However, nerve damage inevitably results in variable sensory loss, a disturbing symptom itself.

Microvascular decompression is the only non-ablative intervention, where the culprit blood vessel is dissected free from the trigeminal nerve. This procedure requires an invasive post-auricular craniotomy and

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Table 1. Surgical interventions for trigeminal neuralgia

Source	Ablative	Non-ablative
Posterior fossa at root entry (central)	Partial sensory rhizotomy	Microvascular decompression
	Stereotactic radiosurgery	
At Gasser's ganglion (point of trifurcation)	Radiofrequency thermocoagulation	
	Glycerol rhizolysis (bathing nerve in glycerol)	
	Balloon compression	
	Stereotactic radiosurgery (gamma knife)	
Distal to ganglion at trigger zone (peripheral)	Cryotherapy	
	Neurectomy (cutting nerve)	
	Alcohol injection	
	Streptomycin injection	

Case Report

A 47-year-old man presented with extreme right-sided facial discomfort of a lancinating character. Any facial movement triggered pain. He was unable to eat, take oral fluids or talk and communicated using written notes and phone text messages.

Previously diagnosed with a right-sided cluster headaches, these transformed 12 months ago into typical maxillary division trigeminal neuralgia.

Initial management consisted of a multi-modal cocktail of analgesics (paracetamol, diclofenac, tramadol, pregabalin and carbamazepine), with morphine as required. After 7 days, the pain management team prescribed subcutaneous infusion of ketamine (50 mg/24 hours) with rectal diazepam to suppress spasms. Subsequently, the ketamine was increased to 100 mg/24 hours. On transfer to a neurosurgical unit, he underwent two injections of glycerol, a mild neurolytic agent, into Meckel's cave (a bony cavity within the skull containing the trigeminal ganglion). Repatriated on day 24, he was still unable to speak and was being fed via nasogastric tube. By day 27 he was able to speak without pain. He had now lost 12.7 kg in weight and had been bed-bound for almost a month. Clinical psychologists, dieticians, physiotherapists, occupational therapists and speech therapists became involved. He was discharged home 48 days after his admission.

Six weeks later he was re-admitted with a further exacerbation and intravenous ketamine was re-commenced. The patient now expressed suicidal ideology. Re-referred for surgical microvascular decompression, he underwent root section as no vascular loop was found at surgery. This produced almost complete resolution of the pain although he volunteers a 'phantom-limb type' appreciation of the start of an attack. He is currently left with deafness in the right ear and some right facial numbness.

complications include ipsilateral hearing loss and 0.3% mortality rate. Outcomes are encouraging, with 70% remaining pain free at 10 years (Zakrzewska et al, 2005; Zakrewska and McMillan, 2011).

Conclusions

Trigeminal neuralgia is a debilitating condition that may initially respond to a selection of analgesics and anticonvulsants, but in some cases will rapidly progress. This case highlights the plight of patients suffering such extreme discomfort and the management options available. **BJHM**

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LEARNING POINTS

- Trigeminal neuralgia producing intractable pain requiring a continuous intravenous infusion of anaesthetic agent (ketamine) is unusual.
- In this case a surgical section of the trigeminal nerve root was required to resolve symptoms.
- Extreme symptomatology and a protracted duration of incapacity can result in both medical and psychological issues.