

Pretrigeminal Neuralgia

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Over the years, this condition has been termed *dolor faciei Fothergillii* (after his 1776 description; there were earlier descriptions including Locke's in 1677), *trisma dolorificans*, epileptiform neuralgia, trifacial neuralgia, facial chorea, *tic convulsif*, and *tic douloureux*. In 1949, Symonds described a rare antecedent.

CLINICAL HISTORY

An 82-year-old woman presented with a 10-day history of facial pain. She described a severe sharp pain such as a flash of lightning lasting seconds over the right forehead or cheek with multiple episodes per day triggered by touching with her finger or a pillow, washing her face, or less often with moving her forehead or frowning. She recalled a similar pain in the same distribution lasting about 2 days about 3 years ago with no symptoms again until recently.

Two and one-half months previously, she developed an "uncomfortable" pain, such as a toothache with an intensity of 5/10 above the right upper teeth around the cheek which had been present most of the time. The pain did not interfere with sleep. There were no triggers. She was found to have a partially necrotic right upper canine and underwent treatment by an endodontist including root canal without any change

in the pain. Neurological examination was normal. A MRI of the brain including thin sequences, though the brainstem with and without contrast, were normal.

The patient was placed on controlled release carbamazepine 200 mg every 12 hours. Within 24 hours, all of the pain including the constant pain and the paroxysmal pain was completely gone and she could touch her face without triggering any pain.

Question.—Was the constant pain related to the trigeminal neuralgia?

EXPERT OPINION

The International Headache Society (IHS) and the International Association for the Study of Pain (IASP) classify trigeminal neuralgia (TN) as a neuropathic pain with distinct diagnostic criteria. Typical attacks of TN consist of brief paroxysms of excruciating pain limited to some part of the distribution of the trigeminal nerve. Stimulating an area of skin or mucous membrane will often trigger an attack.¹ The etiology is described as being associated with compressive force by a blood vessel on the trigeminal nerve intracranially. This may result in the paroxysmal pains triggered by non-noxious stimulus. Nearly all patients diagnosed with TN will receive drug therapy as their initial treatment. In the early 1960s, Blom found carbamazepine to be effective for most of his TN patients.² It remains a common medication, against which other drugs, effective in TN are compared. Surgical advances make this disorder curable in a high percentage.

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An atypical initial manifestation that, in some patients, precedes the classic presentation of TN, was introduced by Symonds. He described a continuous, aching pain in the upper or lower jaw, that later developed into classic paroxysmal pain. Mitchell termed this pain as “pretrigeminal neuralgia” (PTN). Descriptions of PTN have included pain that is mild to moderate in intensity, dull, aching, burning, throbbing, soreness of gums, and toothache.³ When localized to a tooth, PTN must be differentiated from pain of dental origin. In the absence of obvious clinical signs and of dental pathosis, irreversible dental procedures must be deferred and the pain history carefully re-examined.

Examination intraorally to define dental pathology requires inspection for structural change, probing for carious inclusions, percussion for periodontal or periapical pathology, and assessment of response to cold or hot stimulus. If there are abnormalities noted, radiographs or further assessment may be needed. If no clear pathology is noted, a default to nonodontogenic cause should be the standard.

The diagnosis of PTN or TN is sometimes hindered by the atypical symptoms and is not made until paroxysmal pain develops. However, an unexplained history of dull aching or burning pain that is recurrent and/or provokable by non-noxious stimulus warrants further investigation.³ The recognition of the dull, continuous, aching pain in the upper or lower jaw as PTN, and the understanding that it may precede the characteristic paroxysmal attacks of typical TN, expedites successful treatment. Appropriate medications may relieve the pain and unnecessary invasive and irreversible dental procedures may be avoided.¹

Though the clinical signs of PTN lack resemblance to those of TN, they reflect the same pathophysiologic condition.⁴ A response to membrane-stabilizing medications such as carbamazepine or other agents (listed in Table) is seen in both TN and PTN.

Fromm and Graff-Radford described the progression from PTN to TN as progressive in weeks to years. The age distribution ranged from 22 to 81 years with a mean age of 56.2 years. There was no difference related to gender, and the pain occurred equally on the left and right sides. Two groups of patients were described, one with PTN who responded to carbamazepine and an-

Table.—Medical Therapies Used in Trigeminal Neuralgia

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| I. Antiepileptics/ Membrane-Stabilizing Agents |
| 1. Carbamazepine* |
| 2. Oxcarbazepine |
| 3. Gabapentin |
| 4. Topiramate |
| 5. Zonisamide |
| 6. Levetiracetam |
| 7. Tiagabine |
| 8. Valproate Sodium |
| 9. Phenytoin |
| 10. Clonazepam |
| 11. Lamotrigine |
| II. Antidepressants |
| 11. Nortriptyline |
| 12. Desipramine |
| 13. Venlafaxine |
| 14. Amitriptyline |
| 15. Doxepin |
| 16. Protriptyline |
| 17. Imipramine |
| III. Muscle Relaxants |
| 18. Baclofen |
| 19. Tizanidine |

*FDA approved for the treatment of trigeminal neuralgia.

other with a history of PTN who had transformed and responded to carbamazepine.

The present case exemplifies the progression of PTN to TN. The patient described severe sharp pain “like a flash of lightning” lasting seconds fitting the accepted criteria for TN. Additionally, she had an uncomfortable toothache-like pain that preceded the paroxysmal pain by 2.5 months. This prodromal pain matches the proposed criteria for PTN. The normal MRI suggests that neither pain was due to a demyelinating process or neoplasm in the cerebellopontine angle. We are now able with higher-resolution imaging and focus on the posterior cranial fossa, able to identify the presence of a blood vessel in contact with the trigeminal nerve. The presence of this vessel is not read as abnormal or looked for by most neuro-radiologists unless specifically requested. Identifying the offending blood vessel prior to surgical treatment may improve predicted outcome. Microvascular decompression (MVD) has been reported to have an initial complete pain relief rate between 87% and 98%. The success rate falls to 75% to 80% after 2 years and under 65% after 10 years.⁵ The predicted complete

response of the pain to MVD is lower when there is a constant pain component (<60%) in addition to the paroxysmal pain. However, with or without a persistent pain, the cure rate is higher with evidence of greater compression of the trigeminal nerve by a blood vessel.⁶

The continued pain after the endodontic treatment indicates that the toothache was not due to dental pathology. Merrill and Graff-Radford reported that a very high percentage of patients (65%) with TN receive unnecessary dental therapies.⁴ The positive and complete response of the pain to carbamazepine provides further evidence that the constant pain was indeed related to the TN and typifies PTN.

It is imperative that the clinician seeing trigeminal pain be aware that this rare but treatable condition exists. This will prevent the unnecessary pain and suffering and minimize the need for invasive dental therapies.

REFERENCES

1. Fromm GH, Graff-Radford SB, Terrence CF, Sweet WH. Pre-trigeminal neuralgia. *Neurology*. 1990;40:1493-1495.
2. Fromm GH, Sessle BJ. Introduction and historical review. In: Fromm GH, Sessle BJ, eds. *Trigeminal Neuralgia: Current Concepts Regarding Pathogenesis and Treatment*. Boston: Butterworth-Heinemann; 1991:1-26.
3. Mitchell PG. Pre-trigeminal neuralgia. *Br Dent J*. 1980;149:167-170.
4. Merrill RL, Graff-Radford SB. Trigeminal neuralgia: how to rule out the wrong treatment. *JADA*. 1992;123:63-68.
5. Nurmikko TJ, Eldridge PR. Trigeminal neuralgia—pathophysiology, diagnosis and current treatment. *Br J Anaesth*. 2001;87:117-132.
6. Szapiro J, Sindou M. Prognostic factors in microvascular decompression for trigeminal neuralgia. *Neurosurgery*. 1985;17:920-929.