Multiple Sclerosis Presenting to the Dental Practitioner: A Report of Two Cases

SIMON HUTCHINSON AND STUART CLARK

Abstract: **In this paper, two patients are described who presented with trigeminal neuralgia which turned out to be an early symptom of multiple sclerosis. General dental practitioners need to be aware of the possible causes for atypical facial pain, especially in patients under 40 years.**

***Dent Update* 2001; 28: 516–517**

Clinical Relevance: **The dental practitioner should be aware of the differential diagnoses of atypical facial pain.**

Facial pain can be one of the prodromal symptoms of multiple sclerosis. The two patients described in this paper presented to their general dental practitioners with facial pains suggestive of trigeminal neuralgia. They were subsequently diagnosed with multiple sclerosis and its rapidly progressive form acute disseminating encephalomyelitis. Practitioners should be aware of these differential diagnoses in patients presenting with atypical facial pain, especially patients under 40. If there is any doubt in the clinician's mind, he or she should refer the patient to his/her general medical practitioner for a full neurological assessment.

CASE 1

A 42-year-old Caucasian woman was referred by her GDP to the Oral and Maxillofacial Surgery Department complaining of an intermittent shooting pain on the left side of her face centred over her temporomandibular joint. This pain had been present for three months and occurred approximately twice per week, lasting for about two hours. The patient was otherwise medically fit and well.

***Simon Hutchinson**, BDS, MFDS RCPS (Glasg.), Senior House Officer, and **Stuart Clark**, FDS RCS (Edin.), FRCS (Edin.), Specialist Registrar, Oral and Maxillofacial Surgery, Sunderland Royal Hospital.*

Clinical Findings

On examination, the patient had no muscle tenderness and good mandibular movement. A click was evident in her left temporomandibular joint towards the end of opening. There was no evidence of intra- or extra-oral pathology.

An initial diagnosis of myofacial pain was made and conservative treatment was commenced.

After two months, the pain was worsening and electric shock-like, and a trial course of carbamazepine 100 mg twice daily was instigated. However, soon her symptoms worsened to include lethargy and unsteadiness of gait. Initially, the lethargy was thought to be a side-effect of the carbamazepine, although a neurological assessment was requested.

Neurological Findings

Neurological examination showed that the patient had normal visual acuity and pupillary reflexes but optic atrophy in her right eye. Her extraocular movements were abnormal with nystagmus in all directions of gaze. Her peripheral nervous system showed normal power but pathologically brisk reflexes. She had vibration impairment up to her knee bilaterally and mild limb ataxia with severe ataxia of gait. Progressive brainstem deterioration was diagnosed.

Other Findings

Routine haematological and haematinic investigations were normal; however, a lumbar puncture showed increased levels of IgG in the cerebrospinal fluid, and oligoclonal bands were present. A magnetic resonance imaging scan revealed marked florid changes of demyelination with multiple punctate high signal intensity lesions in the pons and brainstem (Figure 1).

Diagnosis and Treatment

The findings were diagnostic of multiple sclerosis. The patient was started on steroids and sodium valproate which had limited effect. Her facial pain has improved and she continues to be under oral and neurological review.

CASE 2

A 23-year-old Caucasian woman was referred to the Oral and Maxillofacial Surgery department by her GDP with numbness to the left side of her face,

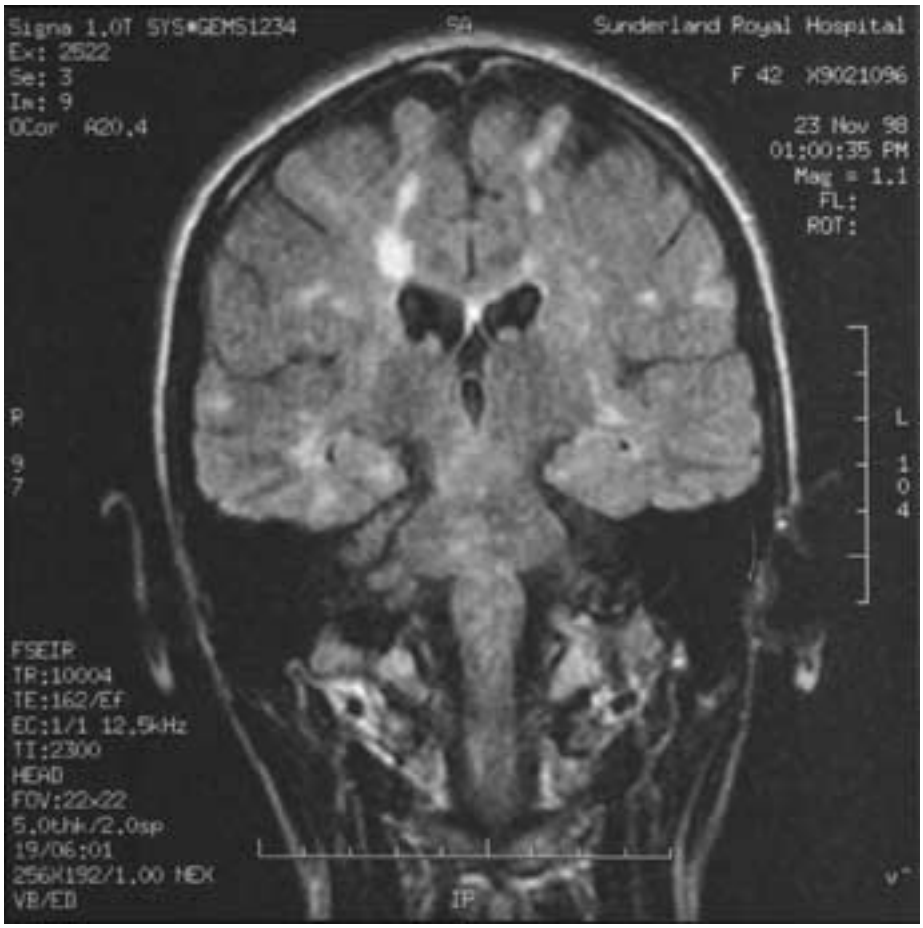

Figure 1. *Case 1: florid changes of demyelination are apparent, with multiple punctate high signal intensity lesions in the pons and medulla.*

roof of mouth and tongue which had commenced four months before: she awoke one morning with this altered sensation, which felt like 'an injection from a dentist'. This sensation was worse in cold weather, sometimes affecting her forehead but never crossing the midline. The patient was being treated with propranolol for hypertension but was otherwise fit and well.

Neurological examination revealed reduced sensation of the maxillary and mandibular branches of the left trigeminal nerve although her cranial nerves were normal. It was felt that this neuropathy should be investigated further.

An MRI scan revealed a focus 1 cm in diameter within the white matter of the right posterior parietal lobe and with a 2 mm hyperintense focus within the left pons towards the midbrain related to the fifth nerve nucleus. The appearance of the scan was consistent with acute disseminated encephalomyelitis. The patient continues to be reviewed by the neurological team.

DISCUSSION

Trigeminal neuralgia is a debilitating disorder that typically occurs in middle to old age, with a slight female predominance.

Multiple sclerosis is a disease characterized by progressive demyelination of nervous tissue, usually presenting in women between 20 and 40 years of age.[1] Between 2% and 7% of patients with trigeminal neuralgia also have multiple sclerosis.[2,3] Where multiple sclerosis is associated with trigeminal neuralgia, there appears to be an earlier age of onset, the symptoms are commonly bilateral and the condition is frequently refractory to carbamazepine,[4] although carbamazepine remains the drug of first choice in its treatment.

Disseminating encephalomyelitis is a descriptive term applied to rapidly progressive cases of multiple sclerosis. The onset is acute, sometimes with fever, leukocytosis, and a raised CSF cell count. The disease usually follows a rapidly fatal course and death can occur within a few weeks.

There is no specific diagnostic test for multiple sclerosis, although MRI scans are the most useful in demonstrating areas of demyelination in the brain. The drug of choice in the treatment of trigeminal neuralgia, whether idiopathic or associated with multiple sclerosis, is carbamazepine. However, as in the first case, this drug may have misleading neurological side-effects which can mask the underlying condition.

Trigeminal neuralgia can also be misdiagnosed for the more common temporomandibular joint dysfunction.

These cases clearly indicate the advisability of a neurological consultation in patients of atypical facial pain.[4,5] Practitioners should refer patients presenting with neurological symptoms for specialist diagnosis and management.[1,6] This is particularly pertinent in patients under 40 years of age or who present with signs and symptoms suggestive of a generalized neurological disorder.

ACKNOWLEDGEMENTS

The authors thank Mr J.M. Ryan, Consultant Oral and Maxillofacial Surgeon, Sunderland Royal Hospital.

REFERENCES

1. Neilson K, Field EA. Trigeminal neuralgia: a cautionary tale. *Br Dent J* 1994; **176:** 68.
2. Friedman CE. Trigeminal neuralgia in a patient with multiple sclerosis. *J Endodont* 1989; **15:** 379–380.
3. Brisman R. Trigeminal neuralgia and multiple sclerosis. *Arch Neurol* 1987; **44:** 379–381.
4. Zakrzewska T. Surgical management of trigeminal neuralgia. *Br Dent J* 1991; **170:** 61–62.
5. Darlow LA, Brooks ML, Quinn PD. Magnetic resonance imaging in the diagnosis of trigeminal neuralgia. *J Oral Maxillofac Surg* 1992; **50:** 621–626.
6. Gale D, Prime S, Campbell MJ. Trigeminal neuralgia and multiple sclerosis. *Oral Surg Oral Med Oral Pathol* 1995; **79:** 338–401.