

## Case report

# Severe amphetamine-induced bruxism: treatment with botulinum toxin

See S-J, Tan E-K. Severe amphetamine-induced bruxism: treatment with botulinum toxin. Acta Neurol Scand 2003; 107: 161–163. © Blackwell Munksgaard 2003.

While chewing and grinding movements have been observed in amphetamine addicts, recognition and management of this problem have rarely been highlighted. Botulinum toxin (BTX) has previously been demonstrated to be effective for bruxism associated with movement disorders, such as cranial-cervical dystonia. However, there is little information on its use in tardive bruxism. Here we report an amphetamine addict who presented with medically intractable bruxism, and discuss its pathophysiology and successful treatment with BTX.

### S.-J. See, E.-K. Tan

Department of Neurology, Singapore General Hospital, Singapore

Key words: amphetamine; bruxism; botulinum toxin

Eng-King Tan, Parkinson's Disease and Movement Disorder Program, Department of Neurology, Singapore General Hospital, Outram Road, Singapore 169608, Singapore

Fax: 65 62 20 3321

e-mail: gnrtk@sgh.com.sg

Accepted for publication May 16, 2002

Bruxism refers to diurnal or nocturnal parafunctional activity including clenching, grinding, bracing and gnashing of the teeth (1). The prevalence of bruxism has been reported from 5 to 96% of adult populations (2–5), and about 15% in children (6). While most of the nocturnal bruxism seen in the general population is apparently 'idiopathic', bruxism has been reportedly caused by, or associated with, numerous conditions such as cranio-cervical dystonia (7), post-anoxic brain damage (8), coma (9), cerebellar damage (10), Huntington's disease (11), Rett's syndrome (12), Whipple's disease (13), mental retardation (14), and exposure to dopamine receptor-blocking medications (15) and selective serotonin re-uptake inhibitors (16–18).

Although chewing and grinding movements have been observed in amphetamine addicts (19), recognition and management of this problem have rarely been highlighted. Here we report an amphetamine addict who presented with medically intractable bruxism, and discuss its pathophysiology and successful treatment with botulinum toxin (BTX).

### Case report

Our patient, a 37-year-old man, first sought medical attention because of severe tooth wear

resulting from bruxism. His symptoms of loud grinding and tooth clenching started 10 months earlier and resulted in excessive tooth wear, frequent headaches and insomnia. The loud grinding sound also caused much distress to him and his family members. The bruxism persisted in sleep, although it was less intense than when the patient was awake. His bruxism was accompanied by involuntary orolingual movements which had appeared 8 months before the onset of bruxism. Neurological examination did not reveal any focal deficits. Functionally, the bruxism disturbed his swallowing and speech, resulting in significant social embarrassment and depression.

Several months before the onset of bruxism, our patient had inhaled and consumed various types of amphetamines, including 3,4-methylenedioxymethamphetamine or 'Ecstasy' on a daily basis. The consumption of amphetamines ended abruptly when he was incarcerated for the illegal abuse of drugs. Since the incarceration, he had not used any recreational drugs. He first experienced bruxism 1 month after the cessation of amphetamine use.

Medical treatment with drugs such as trihexyphenidyl and tetrabenazine had no effect on bruxism. The patient was subsequently treated with *Dysport*® (Speywood Biopharm Limited, UK). *Dysport*® was reconstituted with 2.5 ml of

sodium chloride injection BP (0.9%) to yield a solution containing 200 units per ml of *Dysport*<sup>®</sup> (One unit is defined as the median lethal intraperitoneal dose in mice). Using a 29G (0.33 mm × 12.7 mm) needle, 0.25 ml (50 units) of *Dysport*<sup>®</sup> was injected at two to three sites intramuscularly into each side of the masseter muscles. The temporalis muscles were not injected. Within a month, the patient reported significant improvement of his bruxism. The therapeutic effect lasted 3–4 months. His family members corroborated his report. There was no complaint of any significant side-effects such as jaw weakness and dysphagia.

### Discussion

Bruxism can lead to many dental and neuromuscular problems, such as tooth wear, periodontal disease, headaches and temporomandibular disorders (2). In 1965, Ashcroft et al. (19) drew attention to the observation that chewing and tooth grinding movements can be a useful clinical sign to recognize amphetamine addicts. However, the diagnosis and management of bruxism in these addicts have rarely been highlighted.

Our patient suffered severe tooth wear, frequent headaches, insomnia, depression, and his loud grinding was a constant source of distress to his family members. His grinding and headaches significantly improved following a course of BTX injections. The exact pathogenesis of bruxism is still not clear. Occlusal discrepancies resulting in reflex contraction of the jaw muscles is frequently (although unproven) thought to be a cause of bruxism (20, 21). Based on animal and human studies, dopaminergic system dysfunction may be implicated in bruxism. Drugs that alter dopaminergic stimulation can cause bruxism (22–25). Recently, investigators demonstrated that an increase in non-functional masticatory activity in rats can be induced by repeated stimulation of the dopaminergic system with apomorphine, a dopamine receptor agonist. There was a positive correlation of these masticatory movements and incisal attrition rate (26). It has been observed that bruxism occurs predominantly during transition from sleep to wakefulness at which time dopaminergic neurons exert their excitatory effects on cortical and limbic jaw motor areas (27). Disruption of the norepinephric (NE) system could also be involved. It has been shown that the masseteric reflex in anesthetized cats can be enhanced by NE and by the NE agonist, phenylephrine (28, 29). Some authors hypothesized that there may be a yet unidentified 'bruxism generator' in the brain which

controls the timing and pattern of bruxism. This generator is likely to be influenced by complex interactions of the motor, limbic and autonomic systems (30).

While some clinical studies drew attention to the potential usefulness of medications such as propranolol (31), bromocriptine (32) and levodopa (33) in alleviating sleep bruxism, more scientific stringent studies are still required. It is also not clear whether these drugs are effective for iatrogenic causes of bruxism, which are usually more severe and disabling. BTX has been demonstrated to be effective for bruxism associated with movement disorders such as cranial-cervical dystonia (7) and coma (8). To our knowledge, its use in drug-induced bruxism has rarely been highlighted. In a study of eight patients with diurnal bruxism secondary to chronic antidopaminergic drug exposure, it was found that the response to pharmacologic treatment was generally poor (15). Our case suggests that BTX is safe and effective for severe intractable amphetamine-induced bruxism. While jaw dislocation has been reported with the use of BTX in a patient with amyotrophic lateral sclerosis (34), serious side-effects are rare when it is administered in patients with bruxism (7, 35). Some authors reported that a single course of BTX can possibly lead to total resolution of bruxism (35). In addition to weakening the muscles of mastication, it has been postulated that BTX may inhibit periodontal mechanoreceptors, which may have a facilitatory effect on jaw closure motor neurons (35). Presently it is not clear whether injecting masseter muscles alone, or in combination with temporalis muscles, is more effective. Further controlled clinical trials are needed to address this issue in both nocturnal and diurnal bruxism.

In conclusion, we highlight the observation that severe bruxism can be a serious complication in amphetamine addicts, and this may be aggravated by neuroleptic exposure. BTX appears to be an effective treatment for severe amphetamine-induced bruxism.

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