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Bruxism: two case reports

Bruxismo: descrizione di due casi

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Key words

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Parole chiave

Bruxism • Ipertrofia dei masseteri • Diagnosi • Trattamento

Summary

The present report refers to two patients presenting with grinding of the teeth (bruxism). A brief review is made of the literature concerning the aetiology of the disease, the clinical manifestations and diagnosis, as well as the therapeutic approach.

Riassunto

Vengono descritti due casi di digrigno dentale (bruxismo) e viene effettuata una revisione critica della letteratura relativa all'eziologia, alle manifestazioni cliniche ed alla diagnosi di tale problema odontoiatrico, e si espongono le diverse modalità terapeutiche.

Introduction

Bruxism, a disease generally considered a parafunctional buccal habit, usually manifests whilst sleeping, and is characterised by a non-functional convergence and grinding of the teeth [1 2], resulting in odontoiatric problems and disorders, not only of the temporomandibular articulation, but also of the muscles used in chewing.

The ENT specialist, even if not usually directly involved in the diagnosis and laboratory investigations related to the disease, is often called upon in the management of the clinical manifestations of this condition.

Two patients presenting typical characteristics and treated in the ENT Clinic of the "Tzanio" Hospital, Piraeus, Greece, have been studied and the most important characteristic data concerning pathogenetics, diagnostic and clinical aspects of the disease, as well as management, are discussed.

Case reports

Case n. 1

Female, 28 years old, a foreign language teacher of English origin, came for attention to our ENT clinic, complaining of a "swelling" in the left temporal region, which she had first noticed six months previously whilst combing her hair. The mass was asymptomatic, however, from the case history, it was revealed that her family had observed that, whilst asleep, the patient ground her teeth.

With further questioning, it was found that the patient had difficulty in opening her mouth in the morning, and, during chewing of cold foods, teeth showed sensitivity. The patient who had a history of frequent bouts of allergic rhinitis, was of anxious disposition, and presented marked neurovegetative disorders.

Upon clinical examination, a widespread painless mass was detected, in the left temporal region, which was immobile and appeared to be in contact with the temporal muscle, on that same side (Fig. 1). During palpation of the muscles, in that area, both the origin of the left temporal muscle, in the coronoid process, and the origin of the left masseter, in the mandible, gave rise to slight pain, upon exerting pressure.

The remaining ENT examination was negative to the tests, and also the odontoiatric examination was within physiological limits; as far as concerns the presence of disorders in dental occlusion, wear was detected in the occlusional surfaces of the molars.

Computed axial tomography (CAT) revealed a disproportion of the lateral soft tissues with a protuberance of the left temporal muscle compared to the right (Fig. 2). The patient was sent for surgery, not only for diagnostic purposes, but also for aesthetic rehabilitation.

The left temporal muscle was found to be hypertrophic, and a specimen was removed. Biopsy examination revealed hypertrophy of the skeletal muscle. No other pathological tests were performed. The post-operative period was uneventful. Temporal hy-

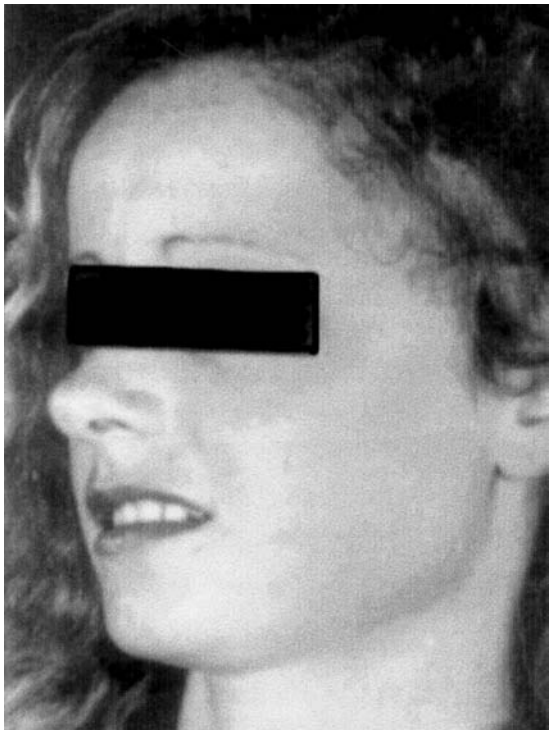

Fig. 1. Case 1. Swelling of left temporal muscle.

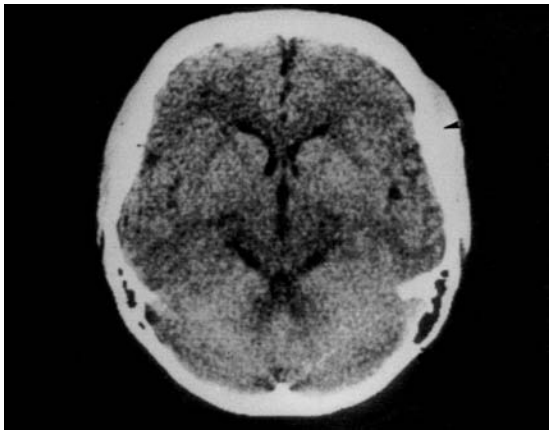

Fig. 2. Case 1. Horizontal CT section showing swelling of left temporal muscle.

pertrophy was considered to be due to bruxism, in this patient, also since the patient did not present impaired dental occlusion or other objective findings. The patient, however, presented an anxious disposition with neurovegetative disorders. Psychiatric therapy was, therefore, suggested and the patient accepted.

At one-year follow-up, after surgery, the patient no longer presented bruxism and superficial masseter muscles no longer presented sensitivity, hypertrophy or other pathological findings.

Case n. 2

Male, 37 years old. The patient presented with severe pain in both temporo-mandibular articulations, hypertrophy of both masseters and toothache.

These symptoms had been present for several years and the patient himself was not aware of having an intense form of bruxism. He had, in the past, been examined by an orthodontic specialist who had prescribed the use of a soft maxillar tutor, which he wore when sleeping, without, however, obtaining useful results. In general, the patient's physical status was very good, and he was found to be a psychologically well-balanced individual.

The main problem, in this patient, was the presence of cranio-facial pain and headache localised in the temporal region, particularly upon awakening in the morning.

From a clinical viewpoint, besides hypertrophy and sensitivity during palpation of the chewing muscles (Fig. 3), pain was felt upon applying pressure to both temporo-mandibular articulations.

Enlargement of the masseters increased further in size when the patient clenched his teeth.

Odontoiatric examination showed wear upon the molars, on both sides, and also attrition of the free borders of the incisors. The attrition was so intense, that, in several areas, the dentine was visible.

Furthermore, abnormal closure of the mouth, type II (overbite), due to the small size of the mandible, was found, resulting in a difference of 7 mm.

Since the patient was particularly distressed by this problem, and since she did not present psychological

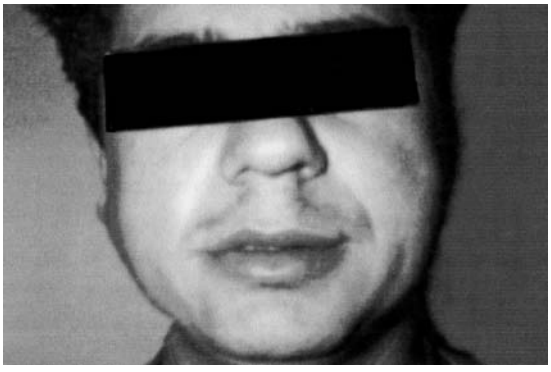

Fig. 3. Case 2. Hypertrophy of both masseters.

disorders or other personality problems, it was decided to collect biopsies of the muscle and salivary gland, which resulted negative to the pathological examination.
The presence of bruxism and hypertrophy of both masseters were considered to be due to the dental occlusion disorder and it was, therefore, held necessary to correct this abnormality.
Since this was a case of severe malocclusion which had previously been unsuccessfully submitted to treatment by an orthodontist, the patient was sent to a specialist in maxillo-facial surgery, for further appraisal.
Surgical treatment was advised. The patient accepted and the outcome was successful, even if the patient has not yet been seen at long-term follow-up.

Discussion

The term "bruxism" comes from "bruxomania" which first appeared in the literature in an article by Marie and Pietkiewcz cited by Ramfjord [3] in 1907, but was first used by Frohman, in 1931[4], to define the problem of a dental nature, resulting from non-physiological movements of the mandible, stressing, in particular, the psychological element of the disease.
The frequency of bruxism according to various Authors ranges between 5% and 96%, depending upon the diagnostic criteria used and the synthesis of the study population under examination [5].
Albeit, according to more recent data, bruxism, whilst sleeping, affects only 5-8% of the adult population, whereas it is more frequent in children, reaching 14-20% in those < 11 years of age [2].
Clinical manifestations are usually moderate, and the condition is thus often diagnosed by chance, during odontoiatric examination of the patient, and manifests clinically with typical wear of the teeth [6].
Causes of grinding are shown in Table I.
Usually, the aetiology is multifactorial and is often difficult to explain. In the past, it was generally held that bruxism was primarily due to anatomo-morphological factors, as in dental occlusion disorders and in abnormalities of the bone structures of the cranio-facial region.
In a well-known study published, in 1961 [3], by Ramfjord, who was the first to systematically study this disease, it was reported that approximately 93% of the patients with problems related to bruxism were cured following rehabilitation of the disorders, due to incomplete closure of the mouth. Case no. 2, described here, would appear to belong to this category. Indeed, this patient underwent rehabilitation for correct maxillary occlusion in order to treat an intense and long-term bruxism.
However, the latest data would appear to suggest that these factors are of secondary importance and involve only a small number of patients [7].
Psychological factors, and, in particular, emotional and psychosocial stress, anxiety, and personality disorders have, long since, been held to play a role in the aetiopathogenesis of the disease both as the principal and auxiliary cause [8 9].
Case n. 1, described here, presented psychological problems, namely nervous anxiety and emotional disorders, treatment of which was attempted primarily with consultations and behaviour strategies and, later, consultation with a specialist in psychiatry, resulting in a stable improvement.
Most of the studies, today, however, tend to agree that pathophysiological disorders of the central type play a major role in the pathogenesis of the condition [9 10].
These are considered to be sleep disorders, and, in particular, the reaction is characterised as "microarousal" (small or brief awakening) and precedes, in most cases, the episodes of bruxism which occur during night-time sleep [11].
This term is used to define the sudden change in deep sleep, which becomes lighter, and is associated with electroencephalographic and electromyographic changes, changes in cardiac and respiratory rhythm, as well as disorders of the autonomic nervous system.
Another important anomaly, frequently found, is impairment of the neurotransmitter system of the central nervous system (CNS), and, in particular, of the dopaminergic system of the basal ganglia [10].
This would explain the slight repressive action of levodopa in bruxism (on the contrary, its prolonged use results in deterioration), but also explains why the use of drugs with an immediate action on the dopaminergic system may induce bruxism, for example, the selective inhibitors of the reuptake of the serotonine (selective serotonine reuptake inhibitors, S.S.R.I.) [12].
Furthermore, nicotine stimulates central dopaminergic action, which may explain why some studies have reported that smokers present bruxism twice as often as non-smokers, and that episodes of bruxism are 5-fold that of non-smokers during night-time sleep [13].
Thus, various other factors, such as genetic predisposition, allergy, alcohol abuse, use of drugs, encephalic damage of an organic nature and mental retardation, are often considered responsible for the onset of bruxism [2].
Over the last few years, Lavigne and Montplaisir [14], formulated a theory of the model inducing bruxism, according to which, the causes of bruxism derive from the rhythm existing between sleep and the activity of the masseter muscles, as found in almost 60% of normal subjects.

Table I. Causes of bruxism.

I. Anatomo-morphological factors
– Dental occlusion anomalies (malocclusion)
• Various morphological types of malocclusion
• Functional malocclusion
– Anomalies of the oro-facial region
• Condyle height asymmetry
• Larger cranial and bizygomatic widths
• Rectangular form of dental arch of maxilla
• Rectangular morphology of face
II. Psychophysiological factors
– Stress (emotional, physical, psychosocial)
– Anxiety (states of anxiety, psychosocial)
– Emotional disorders
– Psychosomatic disorders
– Personality disorders (hyperactivity, rage, aggressiveness, perfectionist tendency)
III. Pathophysiological factors
– Sleep disorders
• Poor quality of sleep
• Microarousal episodes (short awakening)
• Frequent movements of body
• Behaviour disorders during REM sleep
• Periodic movements of feet
• Agitated sleep syndrome
• Sleep apnoea syndrome
• Sleep epilepsy
• Sleepiness during the day
– Rhythmic muscular activity of masseter muscles
– Sensitivity disorders of central dopaminergic neurotransmission
IV. Other causes
– Genetic predisposition
– Allergy (allergic rhinitis, bronchial asthma, swallowing of allergenic foods)
– Hemifacial spasm
– Various syndromes (Gilles de la Tourette S., Rett S., Shy-Drager S.)
– Whipple disease
– Neurological disease (brain haemorrhage, coma, Huntington disease, Parkinson disease, olivopontocerebellar atrophy)
– Oromandibular dystony
– Drug intake
• Antidepressants (SSRI) (Fluoxetine, sestraline, paroxetine)
• Chronic use of neuroleptic and levodopa
• Amphetamine and analogous drugs (OTC)
– Smoke and alcohol abuse

Each of the above-mentioned causes may lead to an increase in frequency, duration and intensity of muscle activity, even exceeding physiological levels and resulting in the onset of bruxism.

This model offers a satisfactory explanation regarding the multifactorial aetiology of bruxism.

The clinical manifestations of the disease are shown in Table II.

Table II. Clinical manifestations of bruxism.

- Dental wear (wear of dental elements), particularly in eccentric sites (bruxofacet), incompatible with a history of physiological wear, special diet, odontoiatric treatment, adaptation of teeth or professional wear of teeth
- Rigidity or fatigue of mandibular muscles every morning or also during awakening at night
- Sensitivity of temporomandibular articulation
- Sensitivity and hypertrophy of masseter muscles
- Cranio-facial pain syndrome, with chronic headache, in particular of temporal region
- Hypersensitivity of teeth to cold air or liquids
- Feeling of weight in teeth
- Frequent movements of mandible, for no reason
- Ulceration of oral mucosa, located behind molar teeth, or border of tongue

The main manifestations characterising the severe forms of bruxism, which usually allow the diagnosis to be made, are: recent history of grinding of the teeth (at least 3-5 nights a week, for at least six months), wear of the teeth, early morning pain, tired feeling in the region of the masseter muscles with evidence of hypertrophy [15].

Hypertrophy of the temporal muscle is very rare, particularly unilateral hypertrophy which was observed in our first patient and which is, to our knowledge, only the third case to have been described in the international literature.

In the other two cases [16 17], the aetiology was not well defined, whilst in the present patient, there was a very definite relationship with bruxism, emerging not only from the case history and clinical pattern but also from the outcome of treatment. Diagnosis is often difficult, since grinding of the teeth usually occurs during sleep, and, therefore, the patient is not fully aware of the problem. Albeit, grinding of the teeth during sleep, reported by the patient's partner, may be helpful in this respect.

Clinical and laboratory investigations, in these patients, should include collection of a detailed history of the disease, referring both to medical and odontoiatric disorders, orofacial and odontoiatric examinations, X-ray control of the jaws and temporo-mandibular articulation, CT of the facial skull as well as kinesiologic study of the oro-mandibular system [2 6 15].

Final diagnosis is possible in the sleep laboratory, with polysomnographic recordings which include electroencephalographic examinations (EEC), electromyographic (EMG), electrocardiographic (ECG) and thermosensitive signals with simultaneous audio-video recording [18].

Unfortunately, high costs represent one of the great disadvantages of this laboratory investigation. It is advisable to collect a biopsy of muscle tissue, when diagnostic problems are encountered, in order to proceed with differential diagnosis due to inflammatory muscular enlargement, benign or malignant tumours, or the parotid-masseter syndrome.

A muscle biopsy was collected from both our patients. Results were negative as far as concerns tumour or other disorders.

No specific and generally accepted treatment exists, as yet, for this condition; medical treatment needs to be "tailored" since the aetiology of this disease is multifactorial.

The main treatments will include: an odontoiatric and orthodontic approach, together with psychological and pharmacological treatment [6].

Dental malocclusion disorders are corrected by means of a rigid acrylic jaw tutor, particularly at night-time [19]. Indeed, soft jaw tutors are not suitable for long-term use.

Psychological support, communication strategies, biofeedback methods and sleep therapy have been shown to be particularly useful in many patients [15 20 21].

Pharmacological treatment comprising the use of benzodiazepine, central muscle relaxants, tricyclic antidepressants, and, ocasionally, L-dopa, propranolol or type A botulin toxin, has not been shown to be effective [14].

In conclusion, we wish to stress that bruxism is rarely encountered by the ENT specialist, who may be called upon to treat the patient on account of clinical manifestations. Albeit, it is important to be aware of the latest developments, in this field, not only as far as concerns pathogenesis and diagnosis, but also the therapeutic approach.

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