PALATAL AND MANDIBULAR TORI: DIAGNOSIS, CLINICAL SIGNIFICANCE AND CONCEPTUAL BASIS

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ABSTRACT

Palatal and mandibular tori are developmental disorders classified as morphological abnormalities of late palatal and mandibular growth and maturation. Familial cases and persistence in older and edentulous individuals suggest a genetic origin, which is just beginning to be unraveled. The interpretation of tori as an adaptive response to occlusal overload, bruxism and other external factors seems inadequate, because tori are not adaptive hyperplasia or hypertrophies. Tori are bony protuberances without a fibrous capsule, which distinguishes them from osteomas and indicates that they do not have any type of malignant or benign neoplastic nature, which is primarily confirmed by the fact that their growth is not continuous or uncontrolled. Torus size stabilizes at the end of maxillary growth, around the age of 22 to 24 years. Tori are masses of normal bone in terms of function and structure, and may be used as a harvest site for autogenous bone grafts to be used in other sites, or as the placement site for osseointegrated implants, if these procedures are clinically convenient. They may be removed if they interfere with any dental treatment.

KEYWORDS

Torus. Palatal torus. Mandibular torus. Anomalies.

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The frequency of palatal and mandibular tori in our population and all population groups, particularly in some ethnic groups, raises the interest of professionals and patients in understanding their clinical, as well as anthropological, significance.

In a previous study[1] about this topic, some clinical cases were described to illustrate some of the characteristics of palatal and mandibular tori especially for implantologists. This study brings new information and describes some cases that offer dentists in all specialties data necessary to explain the meaning of tori to all patients and, at the same time, to help them decide on the best management for each clinical case.

"TORUS" IN LATIN AND IN ENGLISH

In English, the origin of the word torus is the Latin term "torus". The plural form in English, as well as in Latin, is "tori".

This term, used to name a rounded and smooth increase in volume, a protuberance or knot, is often used in anatomy to name structures that are similar to a cushion, or couch.[2,3] Some examples of anatomic structures that match this description are:

- Oral tori, more often described as a linea alba or occlusal line on the buccal mucosa.
- Supraorbital torus and occipital torus of the skull.
- Torus levatorius on the lateral wall of the nasopharynx.
- Metacarpal torus of the hand, and
- Torus tubarius, torus uretericus and torus uterinus.

In architecture, torus refers to a convex semi-circular molding, often found in the base of columns.[2,3]

The jaws may have two very specific bone protuberances, in two distinctive regions, that have been lately classified as independent clinical entities:

- the first, in the hard palate midline, called palatal torus (Fig. 1); and
- the other, on the lingual aspect of the mandible, in the region of mandibular canines and premolars, called mandibular torus (Fig. 2).

In anatomy, the word torus is used to identify any increase in bone volume that is rounded and smooth, any protuberance in the form of a knot. Therefore, it is not wrong to assign the term torus to other bone protuberances in different areas (Fig. 3), which is also the case with other fundamental lesions, such as vesicles, nodules, blisters and ulcers. Such use, however, would not necessarily indicate a reference to the two classical clinical entities called palatal torus and mandibular torus.

MEANING OF EXOSTOSIS AND ENOSTOSIS

Superficial bone outgrowths, which may also be identified as exostosis, are found in all bones. The term exostosis, of a Greek origin, is made up of:

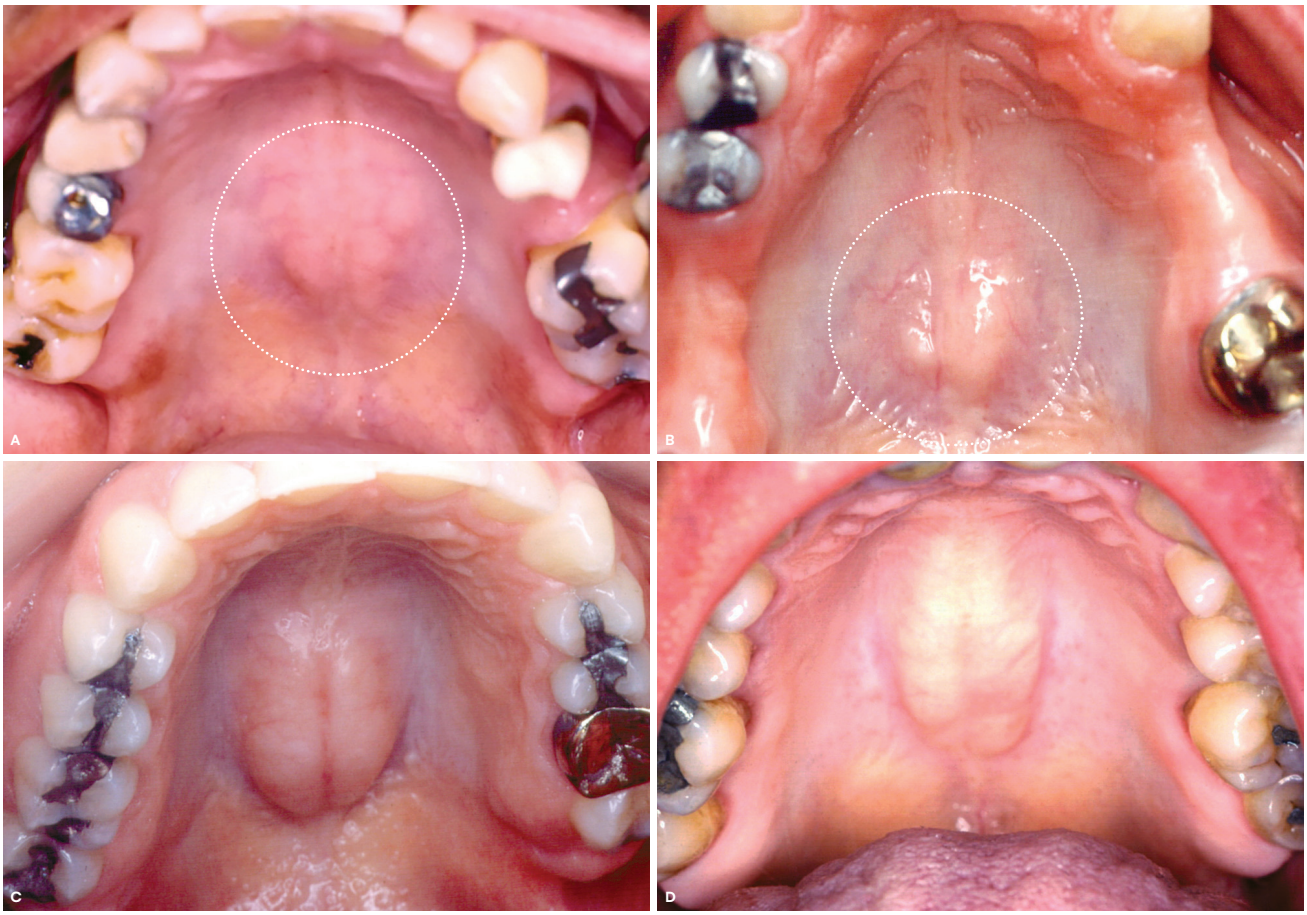

Figure 1

Flat palatal tori (**A**), nodular (**B**) and lobular (**C** and **D**). The most yellowish or whitish color of the mucosa that covers them, due to its thin thickness, stands out.

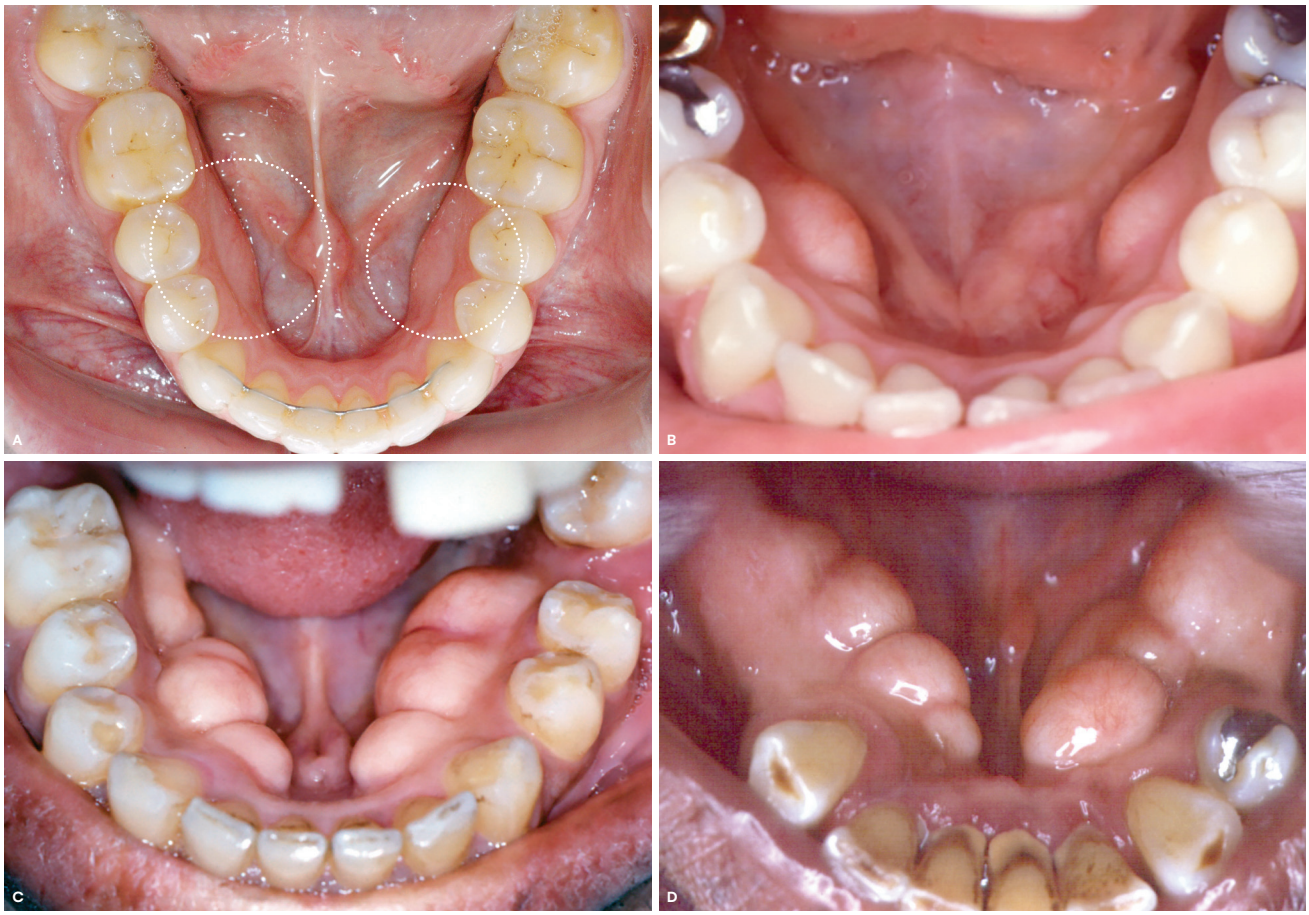

Figure 2

Flat bilateral mandibular tori (**A**), nodular (**B**) and lobular (**C** and **D**).

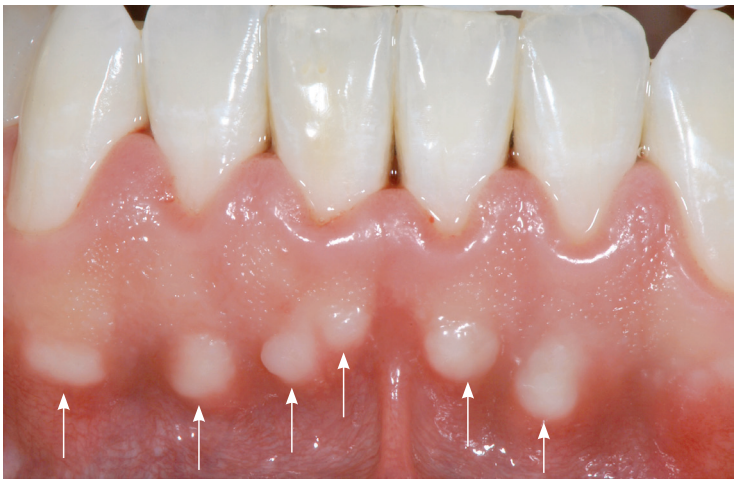

Figure 3
Bone exostosis (arrows) in the gingiva and bucco-alveolar mucosa of a young patient, unrelated to any other disease. When multiple exostosis appear, an association with Gardner's syndrome must be sought with precision.

ex = external + osteon = bone + osis = condition, action or process.

In the group of exostoses, there are anomalies, reactive bone formation and neoplasias. That means that the term is not associated with the nature of the biological process that produces it.

An outgrowth that occurs within bone may be described as an enostosis[4]. The term enostosis, also of a Greek origin, is made up of: en = internal + osteon = bone + osis = condition, action or process.

The term enostosis may be used for the accurate description of bone outgrowths that develop:

1. On surfaces facing natural cavities of the body, including the skull and, as an example, the cavity of the maxillary sinus;[5] or
2. Within the bone, in the space limited by the cortical plates.[3]

Exostoses and enostoses may be signs of several events that involve bones, ranging from normal changes to aggressive diseases. The terms exostosis and enostosis refer to bone lesions that compare with the fundamental lesions of the oral mucosa and skin, such as vesicles, blisters, plaques and macules. Palatal and mandibular tori are classical examples of exostoses (Fig 1 and 2)

An osteoma, for example, may grow only within bone, or, alternatively, into a natural cavity, such as the maxillary sinus. In this case, it is an enostosis clinically. However, osteomas may also grow out of the bone and have their base on the bone surface In this case, they are exostoses clinically. Osteomas are benign neoplastic lesions, whereas palatal and mandibular tori are the result of developmental disorders.

FAMILIAL TRENDS, RACIAL FACTORS AND EPIGENETIC FACTORS

A human skeleton has 206 bones. Newborns have more bones than adults, as some disappear or are incorporated by others and lose their anatomic identity.

Familial tendency — Human anatomy may have variations of form and volume of organs, which includes bones. These variations may be inherited, but the carriers of the genes responsible for these disorders are not necessarily the parents, as other family members may have them. In some conditions, when the genetic and hereditary details of transmission patterns have not yet been studied, etiopathogenesis is often referred to as a change in familial tendency.

Racial factors — When a certain anatomic variation or lesion is more frequent in an ethnic group, the existence of a "racial factor" is usually mentioned as part of its etiopathogenesis, although, in terms of accuracy, this term is very general and undetermined.

Epigenetic factors — Epigenetic factors may have an important effect on genetically determined conditions and morphology, as they modulate and constitute the micro- and macro-environment where genes are activated and where the other parts of the cell operate on the information transmitted by these factors. Epigenetic factors do not promote structural changes or gene mutations, but they affect their functions substantially.

Bodily functions and structures result from genetically transmitted information and the action of external factors on genes, and are, therefore, called epigenetic. An example of this is the morphology of the jaws: some people in the same family may be different because of the different epigenetic factors to which each person is submitted, even in twins. This may explain why palatal or mandibular tori found in several members of the same family may have different forms and sizes. The same is true of cases of cleft lip and palate, palatine rugae and tooth and tongue characteristics. Two genetically identical individuals will rarely have the same epigenetic factors acting in the same way and intensity.

DEVELOPMENTAL DISORDERS ARE NAMED ACCORDING TO THEIR TIME OF DEVELOPMENT: APPLIED CONCEPTS

Developmental disorders, in general, are sometimes called dysgenesis or malformations. They may be nosologically distinguished into anomalies, dysplasia and deformities.

Anomalies — Changes in several aspects of the affected organ or tissue, such as in number, form and position; they usually affect the function of the structure significantly, because their cause is pres-

ent at organogenesis, a very early phase of development. Two examples are cleft lip and partial anodontia.

Dysplasias — They compromise the function of an organ or tissue by affecting its later development, or histogenesis, particularly its differentiation and maturation phases. Two classical examples are hereditary ectodermal dysplasia and projection or invagination of cervical enamel.

Deformities — Their causes are physical, such as lack of space or movement during fetal development, and affect the final morphology of the organ or of one of its anatomic parts. Two examples are micrognathia, resulting from poor upward movement of the fetal head, such as found in the Pierre-Robin sequence, and root dilaceration.

Palatal and mandibular tori may be classified as late developmental anomalies, as they occur in the final phase of maxillary formation. They are anomalies that affect the morphology of teeth, and not a dysplasia, as the structure and function of the bone tissue that forms them is normal.

TIME OF OCCURRENCE, DETECTION OF DEVELOPMENTAL CHANGES AND THE NAMES THEY RECEIVE

Prenatal changes — These cases are classified as prenatal. Developmental disorders may occur and be detected before birth regardless of their etiopathogeny, particularly because of diagnostic technologies currently available.

Congenital changes — Changes are classified and called congenital when the disorder, regardless of type of etiopathogeny, is present at birth. Congenital means "born with" etymologically.

Postnatal changes — Developmental disorders may occur or appear after birth, during childhood, in adolescence, or even in adulthood. When this is the case, these developmental disorders are called postnatal.

Palatal and mandibular tori appear in adolescence or later, and their growth ends when maxillary and mandibular growth is complete, at the age of 22 to 24. Despite any familial tendency and associated ethnic factors, tori are disorders of postnatal development. They are not congenital, as the jaws do not have them at birth, and they are also prenatal.

NATURE OF THE CAUSES AND NAMES ASSIGNED

Developmental disorders may be classified into two basic types according to their causes: hereditary and acquired.

Hereditary — These disorders are inherited due to genetic changes transmitted by parents, and some examples are albinism and hereditary amelogenesis imperfecta. In adult life and during development, human beings may undergo genetic changes in their cells, which will not be transmitted to their children because they do not affect germ cells Enamel hypoplasia induced by infections and trauma are examples of this condition.

Hereditary disorders may be genetic, when changes affect one or a few genes in structurally normal chromosomes, which have about one thousand genes each. However, some developmental disorders are the result of chromosomal changes and affect a large number of genes. In these cases, several organs, tissues and functions are compromised, which characterizes a syndrome.

Hereditary developmental disorders may be classified as: genetic, when they result from changes in a single or a few genes; and chromosomal, when they affect part of a chromosome or all of one chromosome, or when there is an increase in the number of chromosomes.

Acquired — these developmental disorders result from external factors that promote changes in the formation of organs or tissues without first having affected the germ cells of carriers. An example is the anterior open bite due to finger sucking for a long time: maxillary development is affected, but this is not later transmitted to children, as it does not affect genes.

Multifactorial inheritance — The changes are transmitted by parents and will only manifest themselves if the modified genes interact with environmental factors. The child receives the altered gene, but the change will only manifest itself if it interacts with acquired factors. Classical examples of multifactorial inheritance are cleft lip and palate and diabetes mellitus.

Palatal and mandibular tori, according to cases reported and reviews of the literature, are greatly suggestive of a hereditary nature, but there are still no accurate methodological data to assert that nature. They are still classified as a morphological anomaly of the jaws with a familial tendency. However, some genetic changes, which may participate in the etiopathogeny of tori, have already been detected.[6]

THE NUMBER OF DISORDERS: DEFINITIONS

Developmental disorders may be simple or multiple. When multiple, they may be divided into three groups:

Syndromes — Disorders are defined as syndromes when their cause is known and clearly determined, and when the changes are seen in numerous patients, characterizing a typical condition, such as Down syndrome, also called trisomy 21.

Associations or syntropy — These are disorders of an unknown origin, in which the changes are found in numerous patients, which characterizes the same typical condition, although the cause remains undetermined.

Sequences — Multiple events that occur in a sequence, in a ripple effect. Only the first has a cause that induces it, and all the others result one from the other, as in the Pierre-Robin sequence.

TORI ARE NOT BENIGN NEOPLASIAS AND DO NOT BECOME MALIGNANT

Benign or malignant neoplasias are cell proliferation disorders that occur after the loss of control over this important mechanism. Neoplasias, or new tissue formations, are cells and tissues that proliferate continuously, at a more or less rapid pace, but not under the control of the organism to which they belong. Their growth is autonomous and unlimited.

Osteomas, for example, proliferate continuously and do not stabilize along time. Therefore, they should be treated as soon as diagnosed. Even when benign, neoplasias grow, and thus gradually compromise structures and occupy spaces, affecting functions and aesthetics. Benign neoplasias require treatment: they do not stop growing! Only a small number of neoplasias, about 10%, usually malignant, are classified as hereditary. All others have causes that are defined differently.

Palatal and mandibular tori are not neoplasias. They begin to grow and receive a diagnosis during puberty, reaching their maximum size at the age of 22 to 24, the time when cranial and mandibular growth is complete in girls and boys. After that age, their size stabilizes and remains stable until old age. Therefore, their development in the jaws has no neoplastic nature. Tori do not have a fibrous capsule.

Exostoses and enostoses may be signs of several events that involve bones, ranging from normal changes to aggressive diseases. An osteoma, regardless of where it is found in the skeleton, may grow within the bone only, or into a natural cavity, such as the maxillary sinus. In this case, it is clinically classified as an enostosis. However, osteomas may also grow out of the bone, and the bone surface serves as their base. In this case, their clinical diagnosis is exostosis.

Palatal and mandibular tori are classical examples of exostosis. Another important difference between tori and osteomas is the fibrous capsule. In an osteoma, there will always be condensation of the fibrous connective tissue circumscribing and isolating the neoplasia from neighboring tissues, including bones. In imaging studies, neoplastic proliferations of bone are outlined by a radiolucent halo.

Tori should not be classified as benign neoplasias or a type of osteoma, which is a continuously growing benign neoplasia that is clearly outlined by a fibrous capsule. Tori are developmental disorders that have different features, which characterize them as separate clinical entities. Tori do not have a fibrous capsule and do not generate a radiolucent peripheral halo on imaging studies (Fig 4).

TORI ARE NOT HYPERPLASIA OR THE RESULT OF HYPERTROPHIC GROWTH!

Normal cells control proliferation to respond to functional and adaptive demands of the organism according to their metabolic needs or their need to adapt to the environment.

Hyperplasia — When a part of the body is under greater functional or adaptive demands, the organism induces proliferation to increase the number of cells at that site until the demand has been successfully met. This type of adaptive response of the organism is called hyperplasia: an increase in the number of cells and tissue of a certain area as an adaptive response.

Microscopically, palatal and mandibular tori do not appear as hyperplasia, as the organization and distribution of bone, as well as the number of cells, is normal. In adaptive hyperplastic responses, the sites have a greater number of cells, such as in periostitis ossificans.

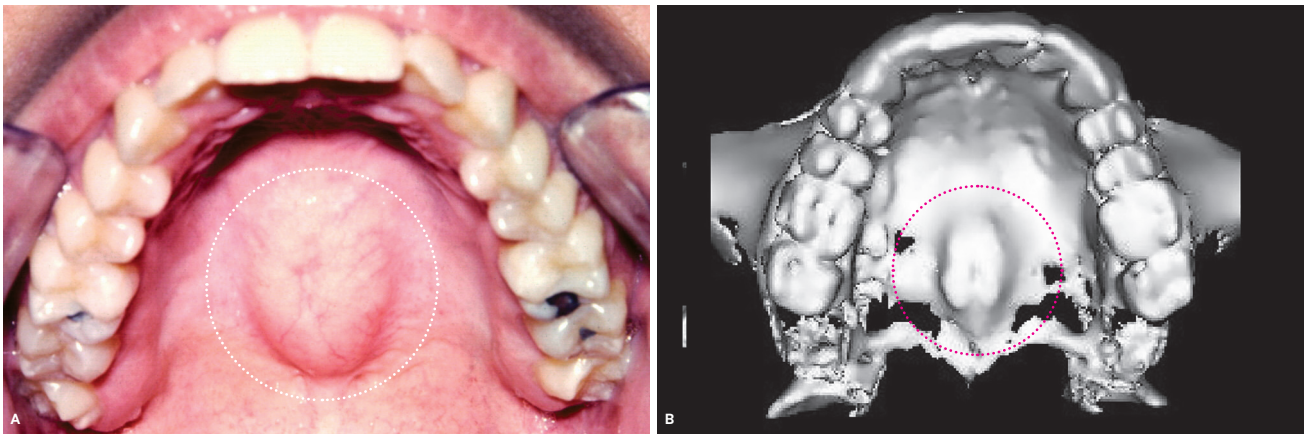

Figure 4
Palatal torus and 3D tomographic image revealing the bony volumetric increase (circle) in the hard palate midline without a hypodense halo separating from jaw bone. The palatal torus represents a volumetric increase in the usual morphology of the hard palate, without representing a benign neoplasm.

Hypertrophy – The term hypertrophy describes a form of functional adaptation of tissues that cannot proliferate anymore, such as skeletal muscle cells. In these cases, the functional demand is met by an increase in cell volume, which becomes larger but cannot proliferate. The increased size of the cells is usually seen under microscopy in tissue analyses. Microscopic sections of tori do not show this increased size because the proliferative capacity of osteoblasts is preserved, if necessary.

The terms hyperplasia and hypertrophy are used only to identify phenomena of a cellular nature that refer to the adaptation of cells to increased functional demands. Palatal and mandibular tori do not have signs of hyperplasia or hypertrophy under microscopy (Fig 5).

PALATAL TORI: ASPECTS OF THEIR CLINICAL AND IMAGING DIAGNOSIS

As its name implies, a torus is a bone protuberance or exostosis with a flat base and very slow growth found in the midline of the hard palate (Fig 1 and 4). Its shape differs from patient to patient, and tori are morphologically classified as flat, spindle-shaped, nodular or lobular.[7]

The oral mucosa covering the palatal torus is normal, but very thin, and the whitish color of the bone immediately below may be seen, which gives the torus a whitish or yellowish appearance (Fig 1 and 5). This delicate oral mucosa may easily ulcerate when prosthetic devices or any type of appliance is placed in the mouth, be it metal archwires, jackscrews or an acrylic plate[8]. If any of these is placed in the mouth, there will be ulceration and pain in the region of the palatal torus (Fig 6). A palatal torus alone is asymptomatic.

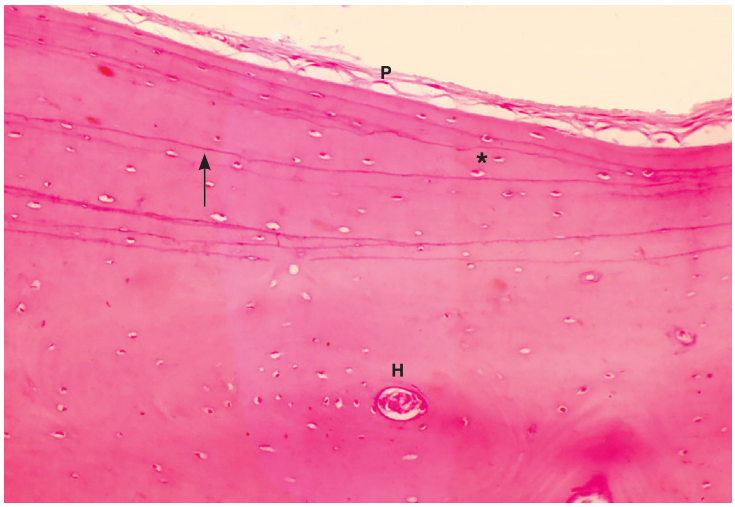

Figure 5
Microscopic aspects most common in the palatal tori represented by normal bone with its apposition layers (arrows), Haversian canal (**H**), osteocyties (*) and periosteum (**P**) (**HE, 25X**).

PROBABLE CAUSES OF PALATAL TORI

Palatal tori are developmental disorders classified as anomalies of late maxillary growth and maturation. Some familial cases have been described, and an autosomal pattern has been determined.[6,9]

There is no methodological evidence to justify assigning a neoplastic or hyperplastic nature to palatal tori, although they may, sometimes, be found under these classifications in the literature, in a few studies.

Their hereditary nature may be supported by studies that found a greater prevalence in some ethnic groups, such as Eskimos and native peoples. Their frequency ranges from 20% to 25%,[10,11,12] and they are more common in women, at a 2:1 ratio. Diagnoses are usually made in the third decade of life.

Although most studies describe palatal tori as a normal variation and assign genetic-hereditary factors as their main origin,[9,13] some authors claim that environmental factors may affect their development.[14,15,16,17]

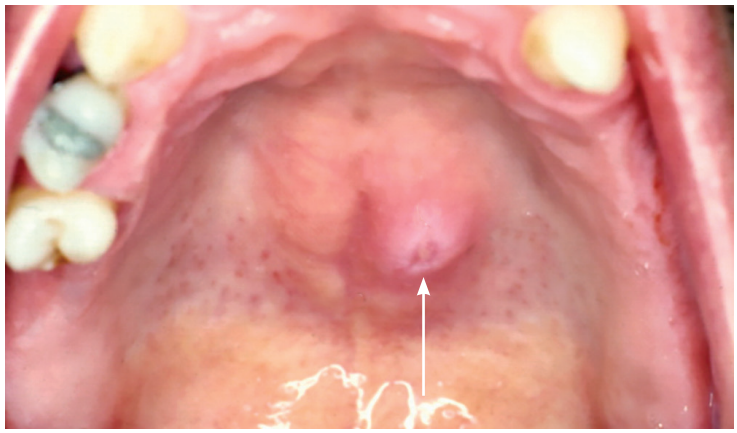

Figure 6
Palatal torus ulcerated by placing a removable prosthesis with a metal bar directly on the extremely thin mucosa. Within a dew hours, the patient presented with symptoms and an ulcerated lesion (arrow) overlapping the torus.

PROGNOSIS AND CLINICAL AND SURGICAL MANAGEMENT OF PALATAL TORI

Bone structure is normal in palatal tori examined under microscopy, and there are no morphological characteristics of neoplasia or hyperplasia in their tissues (Fig 6). In general, they are composed of dense cortical bone with a small area of trabecular bone at a deeper level.[18]

Their growth stops at around 22 to 24 years of age, and they are usually small, which explains why several patients do not detect their presence in their hard palate. When larger, patients may present with signs of excessive concern about their presence, or even cancerphobia, which requires a clear and detailed explanation about palatal tori to reassure them.

Biologically, there is no need to remove a palatal torus surgically, except when it is necessary to:

3. Place any metal or acrylic element of an appliance or prosthesis in the region of the palatal torus; in these cases, an ulcer will develop in a few hours, with great pain and discomfort for the patient.
4. Reestablish phonetic function or adequate mastication, sleep, hygiene or even comfort for the patient, because of the size and form of the lesion.
5. Use it as a source of autogenous bone, when therapeutically necessary according to previous planning of oral restorative treatments. The bone obtained from a palatal torus is structurally and functionally normal (Fig 5).

MANDIBULAR TORI: ASPECTS OF ITS CLINICAL AND IMAGING DIAGNOSIS

Mandibular tori, as palatal tori, are usually bilateral (80%) protuberances or exostoses (Fig 2, 7, 8, 9, 10). Their base is flat, their growth is slow, and they are found in the lingual aspect of the mandibular cortical bone, in the region of canines and premolars, always above the mylohyoid ridge.[14,19] Their clinical presentation varies greatly, from a discrete protuberance, to large spindle-shaped, nodular or lobular exostoses that take up all the floor of the mouth (Fig 2, 7, 8, 9 and 10). Clinically, they have one or more lobules.[19] Because the oral mucosa covering the palatal torus is very thin, the whitish color of the bone immediately below may be seen, which gives the torus a whitish pink or yellowish appearance (Fig 2, 8 and 11). Tori are naturally asymptomatic, but a prosthesis or appliance component of any type, such as a metal arch, palatal expander or acrylic plate, placed on the mucosa of the mandibular torus will cause ulceration and pain in the region in a few hours (Fig 12).

PROBABLE CAUSES OF MANDIBULAR TORI

Familial cases confirm the dysontogenetic nature of tori: associated with developmental disorders, they are described as an anomaly that occurs during the late phase of growth and maxillary maturation. Studies about their prevalence revealed that mandibular tori are found in 6% to 24% of the general population, and appear or are perceived by patients up to the third decade of life.[20] Several patients are unaware of this morphological change in their mandible, especially when the torus is small.

Assigning a neoplastic or hyperplastic nature to palatal tori lacks any adequate methodological evidence, although they may, sometimes, be found under these classifications in the literature, in a few studies. Studies have been conclusive in some families: some cases are inherited, with complete or incomplete penetrance.[21,22,23]

Their possible hereditary nature has been confirmed in studies that revealed a greater likelihood, of about 40% to 60%, of mandibular tori when one or both parents have one of the types of tori. When neither parent has tori, this percentage goes down to 6% to 8% in the general population. In a study with monozygotic twins, 93.6% of the 81 pairs examined had mandibular tori, whereas for dizygotic twins, this percentage was 79.4%[24]. Genetic changes associated with tori were reported by Dou et al.[6] in 2017.

PROGNOSIS AND CLINICAL AND SURGICAL MANAGEMENT OF MANDIBULAR TORI

Microscopic analyses revealed that mandibular tori are normal bone and have no morphological signs of neoplasias or hyperplasia[25]. Microscopy sections of mandibular tori reveal that they are composed of dense cortical bone; only a few times a small deeper area of trabecular bone is seen. Their growth stops at around 22 to 24 years of age, and they are usually small, which explains why several patients do not detect their presence in their mandible. When larger, patients may present with signs of excessive concern about their presence or even cancerphobia, which requires a clear and detailed explanation about mandibular tori to reassure them.

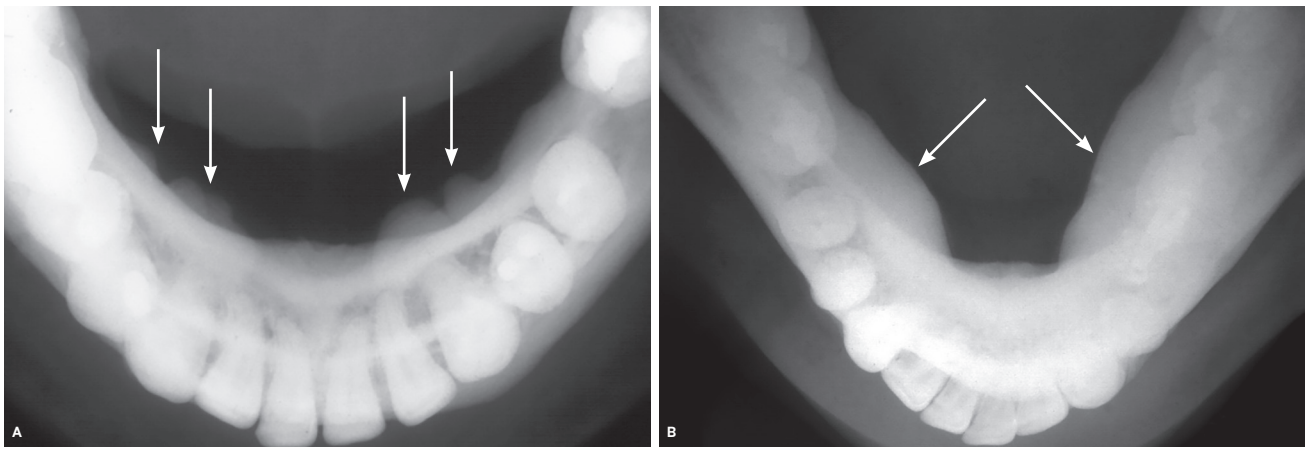

Figure 7
Radiographic images of bilateral Mandibular Tori in nodular form as multiple protuberances in **A** and with a broades and mre extensive bone base in the mesiodistal direction in **B**.

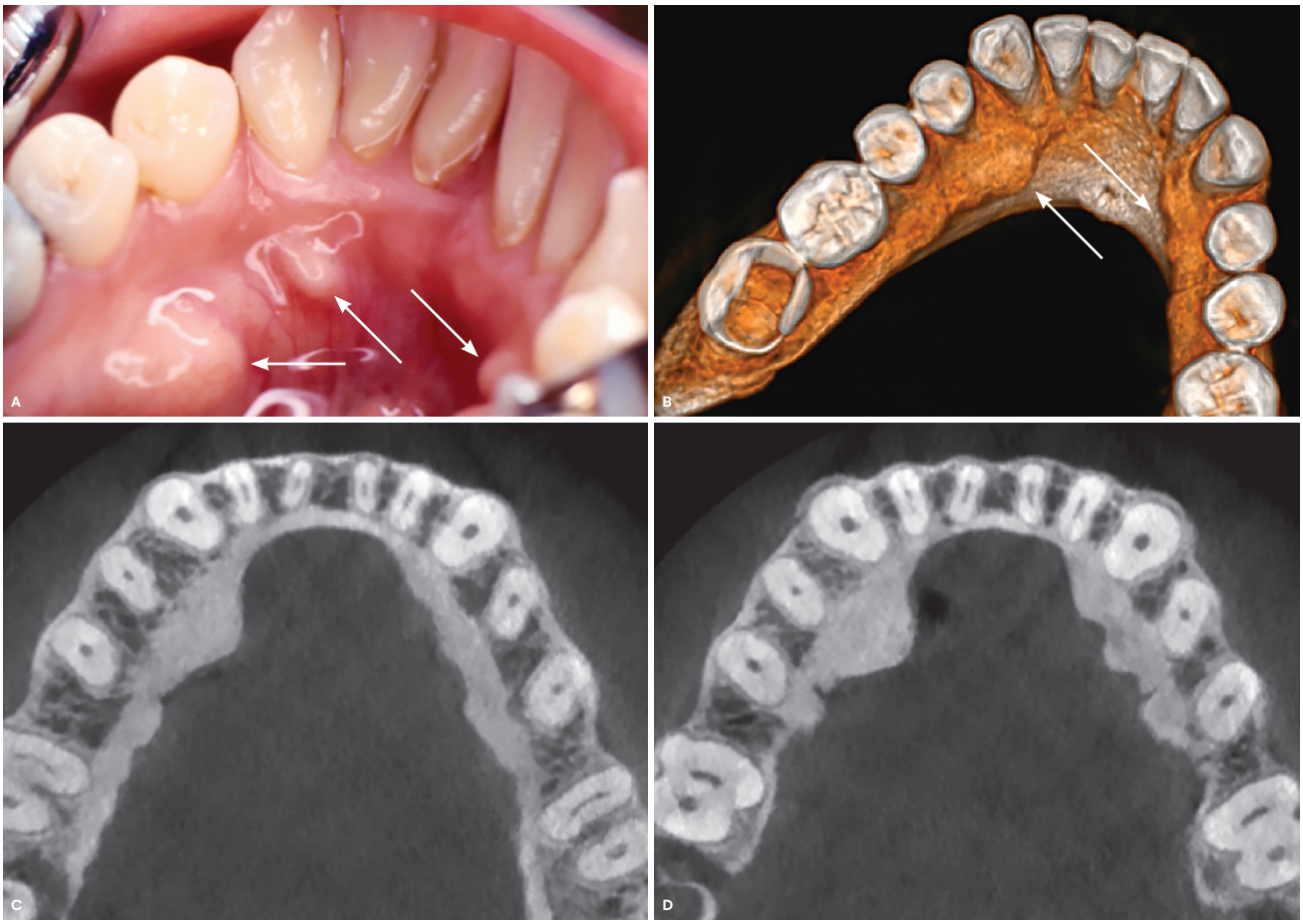

Figure 8

3D tomographic, clinical image and axial sections of bilateral Mandibular Tori in nodular form as multiple protuberances (arrows).

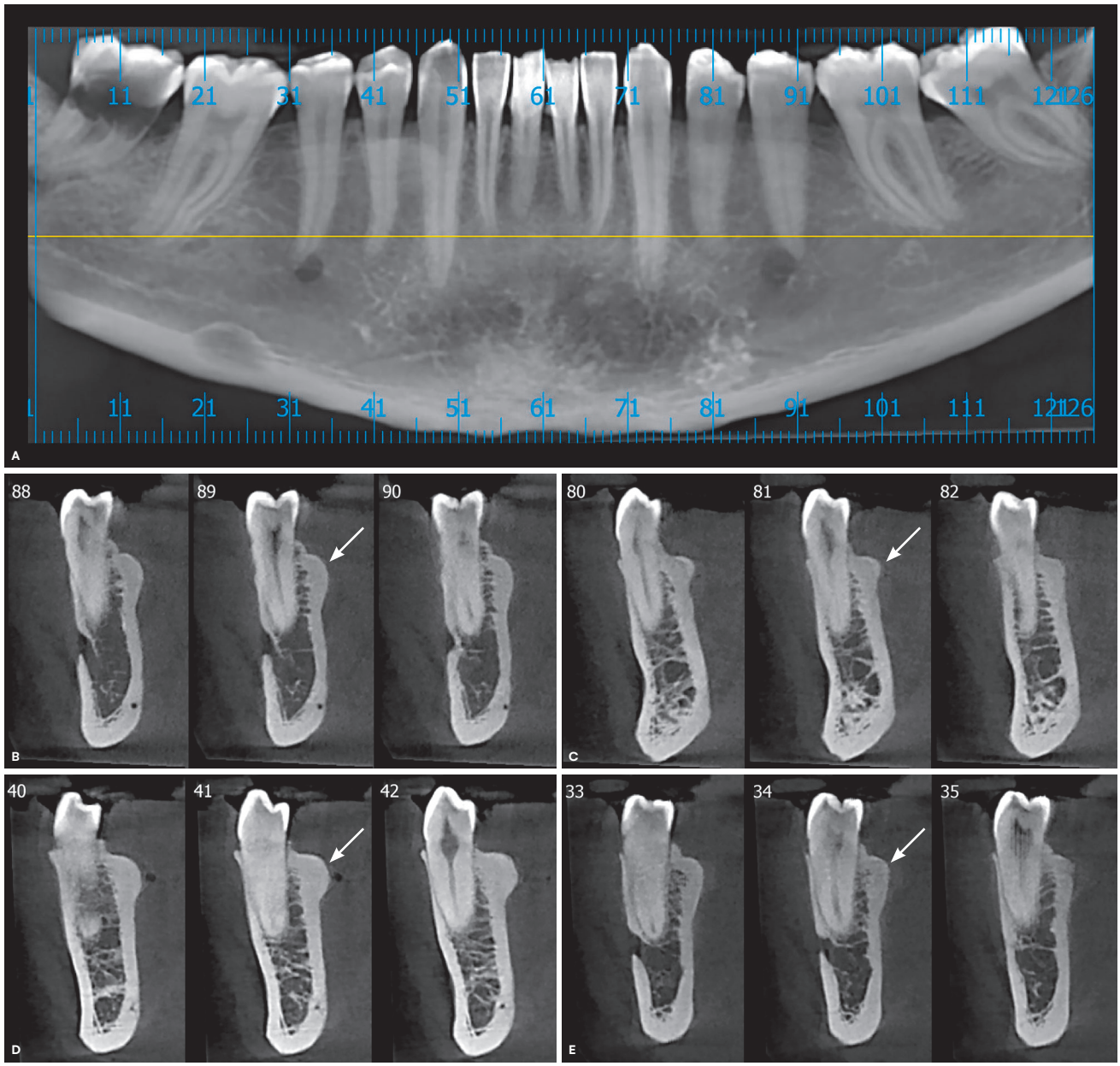

Figure 9

Tomographic images in bilateral Mandibular Tori sections in nodular form with focal thickening of the bone cortex and volumetric increase (arrows) in the region of the lower premolars. The thickening of dense bone continues with cortical bone without radiolucent halo or capsule.

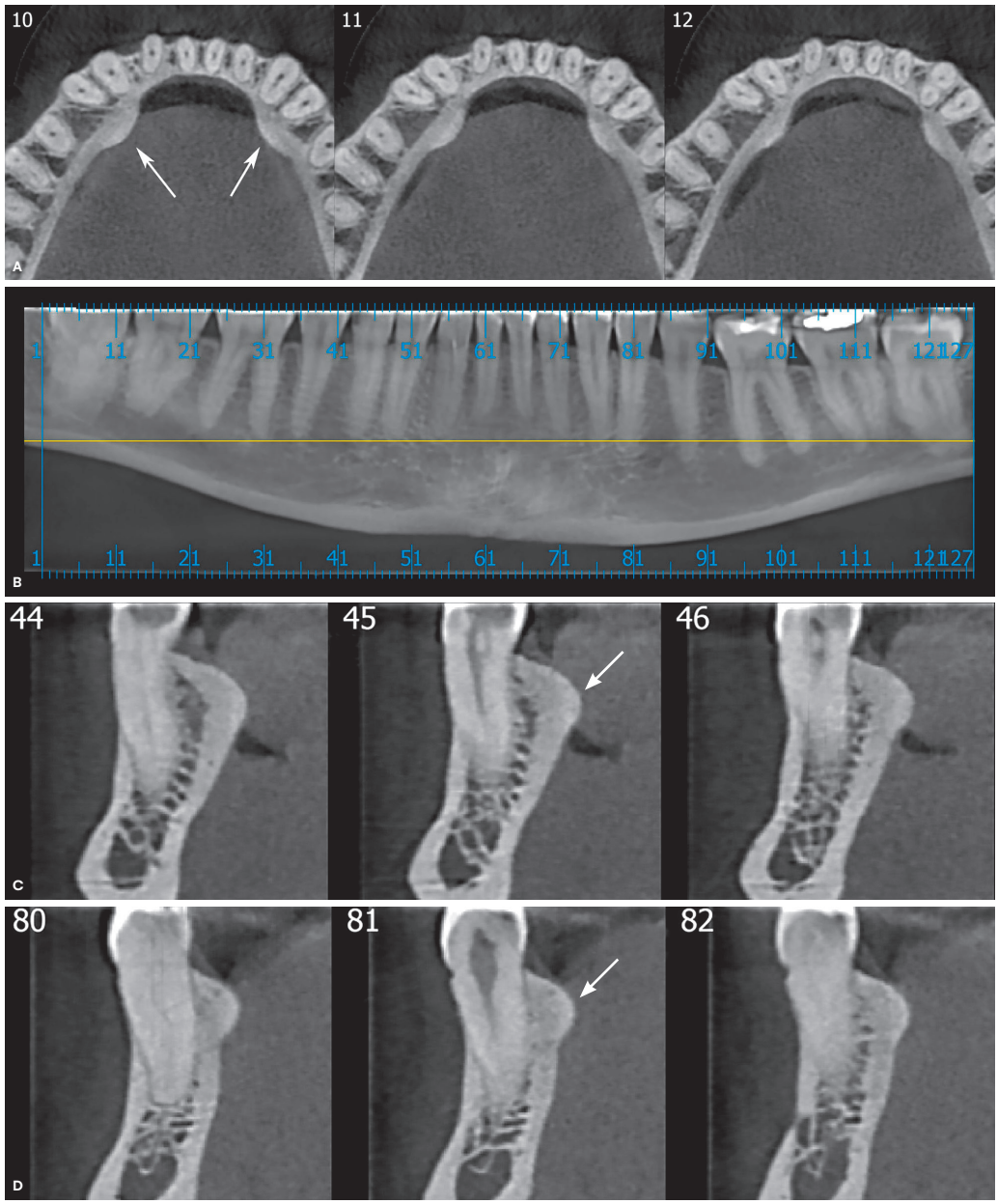

Figure 10

Axial sections of bilateral nodular Mandibular Tori with focal thickening of the bone cortex and volumetric increase (arrows) in the region of the lower premolars highlighted in the buccolingual section.

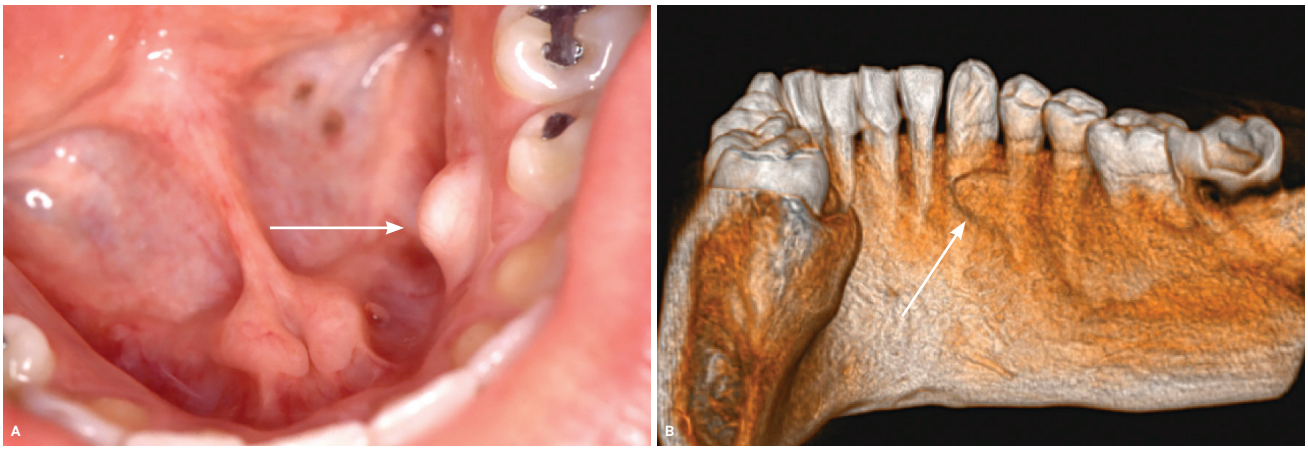

Figure 11
Unilateral Mandibular Torus that occurs in 20% of cases. Its nodular shape and white color stand out. In 3D reconstruction, the bony protuberance of the Mandibular Torus stands out (arrow).

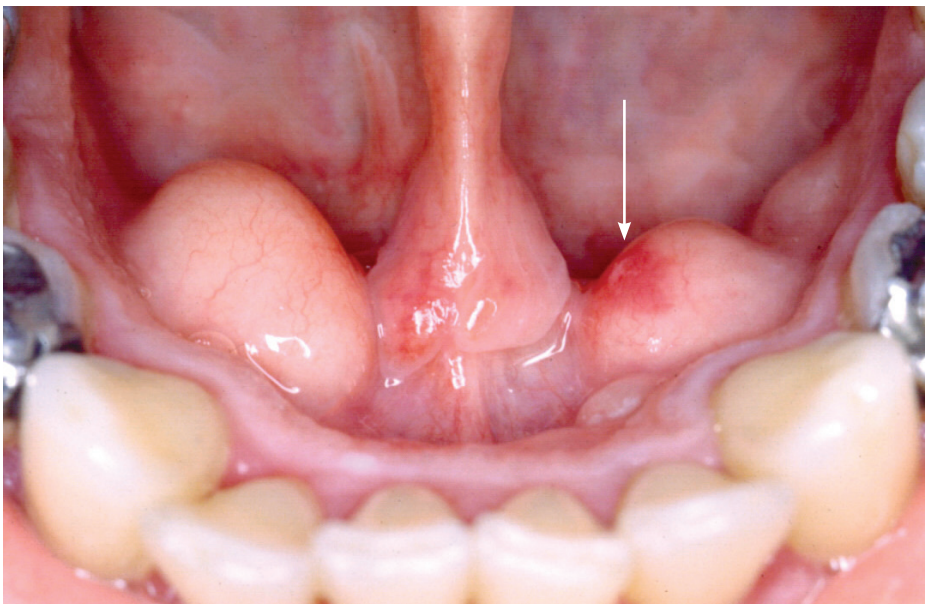

Figure 12
Ulceration due to the passage of solid and rough foods in the thin oral mucosa overlying one of the bilateral and asymmetric Mandibular Tori (arrow).

Biologically, there is no need to remove a mandibular torus surgically, except when it is necessary to:

1. Place any metal or acrylic component of an appliance or prosthesis in the region; in these cases, an ulcer will develop in a few hours, with great pain and discomfort for the patient;
2. Reestablish phonetic function or adequate mastication, sleep, hygiene or even comfort for the patient, because of the size and form of the lesion;
3. Use it as a source of autogenous bone, when therapeutically necessary according to previous planning of oral restorative treatments. The bone obtained from a mandibular torus is structurally and functionally normal.

Treatment and prognosis do not indicate that tori become malignant. Rehabilitation planning that requires the use of the region where a torus is found for implant placement (Fig 7) should take into consideration that a torus is normal bone functionally and structurally.

VULNERABLE POINTS OF THE OCCLUSAL HYPOTHESIS TO EXPLAIN TORI

There is a hypothesis to explain the appearance of tori, particular those in the mandible, in which their origin is not assigned exclusively to genetics. In this hypothesis, environmental and functional factors in adolescence[26,27,28] also play a role and contribute to their growth; in adulthood, however, the role of these factors is reduced, and tori remain stable until old age.

Some studies about prevalence and age of carriers, as well as mandible shape, have suggested that there might be a correlation between tori and drug use, attrition and temporomandibular joint dysfunction.[17,29] These variables may represent environmental factors that somehow affect the development of tori, but do not explain their appearance. Bertazzo-Silveira et al.[30], in 2017, conducted a systematic review and concluded that, based on available evidence, abnormal tooth wear may be associated with tori, particularly mandibular tori. Moreover, they found that there is not enough evidence to confirm the association between tori and other signs and symptoms of bruxism.

Several patients, with or without tori, present with occlusal overload, bruxism, attrition and tooth clenching in adolescence and for the rest of their lives. If tori were associated with occlusal stress, bruxism or temporomandibular joint dysfunctions, why would their growth not vary with age, increasing in size also after the age of 22 to 24, exactly when maxillary development stops?

Methodologically defined evidence will have to be more robust and direct if occlusal overload, bruxism and other conditions are to be included in the list of epigenetic factors of the development of tori.

This hypothesis, which suggests that occlusal overload, bruxism and other environmental factors affect the appearance of tori, is unable to explain some of its aspects accurately:

1. If tori were an adaptive response to the mechanical stress of bruxism, tooth clenching or occlusal overload, there should be hyperplasia or hypertrophy in the tissue examined, but this has not been confirmed microscopically (Fig. 8).
2. If bone remodeling is constant and provides a continuous functional adaptation to dynamic bone restructuring, should tori not disappear in edentulous and older patients (Fig. 13), as there would be no more function for them, in the same way as the alveolar process when teeth are lost?

Tori do not disappear because of the reduction of occlusal load with age or tooth loss. They are part of the maxillary and the mandible, and are genetic variations of these normal anatomic structures, as demonstrated by systematic observations of families and ethnic groups.

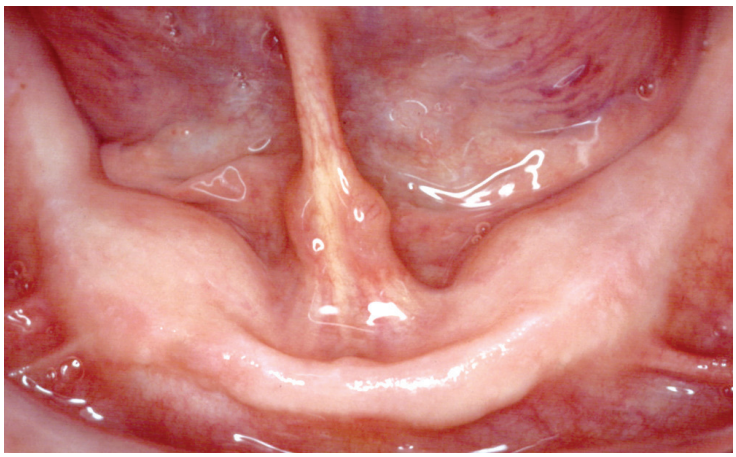

Figure 13
Bilateral Mandibular Tori in a toothless patient without any change in shape and volume over the years.

FINAL CONSIDERATIONS

Current knowledge about palatal and mandibular tori reveals that they are part of the development of the jaws in certain populations, according to a genetic pattern and as a variation of normal anatomy.

In some cases, they may be large and sometimes disturb some functions, make hygiene difficult and complicate the placement of appliances and prostheses. They are developmental disorders classified as form anomalies. There is not enough methodological evidence to assign their origin to mechanical factors, such as occlusal overload, bruxism, temporomandibular joint dysfunction, systemic conditions or drug use.

Tori are composed of bone that is functionally and morphologically normal, with no signs of hyperplasia, hypertrophy or neoplasias. Because of that, they may be used in procedures that require autogenous bone grafts, or may receive osseointegrated implants, if clinically convenient.

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» Patients displayed in this article previously approved the use of their facial and intraoral photographs.