Origin of torus palatinus and torus mandibularis: basis for clinical interpretation

Alberto **CONSOLARO**[1]
Renata Bianco **CONSOLARO**[2]

Abstract: *Torus palatinus and torus mandibularis are developmental anomalies of shape that become late manifest during growth and maturation of the jaws. Family history and the constant presence of tori with age and among edentulous patients attach a genetic origin to them and hinder their clinical interpretation as an adaptive response to occlusal overload, grinding and other external factors: tori are not a form of hyperplasia or adaptive hypertrophy. They are bone protuberances without a fibrous capsule, which differentiates them from osteomata and frees them from a neoplastic nature, albeit benign, especially because they do not grow continuously and uncontrollably in one's organism. The size of tori stabilizes by the end of maxillary growth, at around the age of 22 to 24 years. They are composed of normal bone, from a functional and structural perspective, and might be used as autograft harvesting site or osseointegrated implant placement site within clinically acceptable conditions.* Keywords: *Tori. Torus palatinus. Torus mandibularis. Anomalies.*

[1] Full professor, Universidade de São Paulo (USP), School of Dentistry, Bauru, São Paulo, Brazil. Professor of the postgraduate program, Universidade de São Paulo (USP), School of Dentistry, Ribeirão Preto, São Paulo, Brazil.
[2] Professor of Histology and Pathology, Faculdades Adamantinenses Integradas (FAI), Adamantina , São Paulo, Brazil.

How to cite this article: Consolaro A, Consolaro RB. Origin of torus palatinus and torus mandibularis: basis for clinical interpretation. Dental Press Implantol. 2015 Apr-Jun;9(2):39-54.
DOI: http://dx.doi.org/10.14436/2237-650X.9.2.039-054.oar

Submitted: May 13, 2015 - **Revised and accepted:** May 24, 2015

Contact address: Alberto Consolaro
E-mail: consolaro@uol.com.br

» The authors report no commercial, proprietary or financial interest in the products or companies described in this article.

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

THE TERM "TORUS" IS OF LATIN ORIGIN

The word "torus" (plural: tori) is of Latin origin. In Latin, "torus" means a rounded smooth volume enlargement, protuberance-like or knot-shaped. It is an anatomical trait used to identify structures resembling a cushion or a rope knot.[19,20]

For instance, the following anatomical structures: oral torus, better known as linea alba or occlusion line on the buccal mucosa; supraorbital torus, on the skull; torus tubarius, on the sidewall of the nasopharynx; torus metacarpalis, on the metacarpus; torus occipitalis; torus uretericus and torus uterinus. In Architecture, the term "torus" is defined as convex molding usually located at the base of a column.[19,20]

In maxillary bone, tori are bone protuberances found in two very specific regions: the first is the midline on the hard palate, or torus palatinus; whereas the second one is on the buccal mandibular surface, typically near mandibular canines and premolars, or torus mandibularis.

THE TERMS "EXOSTOSIS" AND "ENOSTOSIS"

Bone growth projecting from a bone surface might be identified as exostoses and occur in all types of bone. From the Greek, "exostosis" is composed of: ex (external) + osteon (bone) + osis (condition, state or process). Exostoses comprise anomalies, neoformation of reaction and neoplasm. In other words, the term is not related to the biological process it derives from.

Bone growth inside the bone might be identified by the terms enostoses or enosteoses.[4]

From the Greek, "enostosis" is composed of: en (internal) + osteon (bone) + osis (condition, state or process). The term "enostosis" might be used to identify, in a more precise manner, bone growth that occurs:

1. In surfaces faced towards natural body cavities, including the skull; for instance, maxillary sinus cavity;[17] or
2. Inside the bone, within cortical limits.[20]

Exostoses and enostoses might be a manifestation of several bone processes and vary from normal to aggressive diseases. The terms "exostoses" and "enostoses" refer to bone lesions compared to oral mucosal and skin lesions, such as vesicles, blisters, plaques, macule, among others. Torus palatinus and torus mandibularis are classic examples of exostoses (Figs 1, 2 and 3).

For instance, an osteoma might grow inside the bones or projecting towards the inside of a natural body cavity, such as the maxillary sinus, and be a clinical manifestation of enostosis. However, an osteoma might also grow projecting from the bone surface and be a manifestation of exostosis.

THE TERMS "FAMILIAL TENDENCY", "RACE FACTOR" AND "EPIGENETIC FACTORS"

The human skeleton is composed of 206 bones. At birth, it is composed of more bones than at adulthood, since some bones disappear or fuse together, thereby losing their anatomical identity.

Human anatomy varies in shape and volume of organs, including bones. Such variations might be inherited, but parents

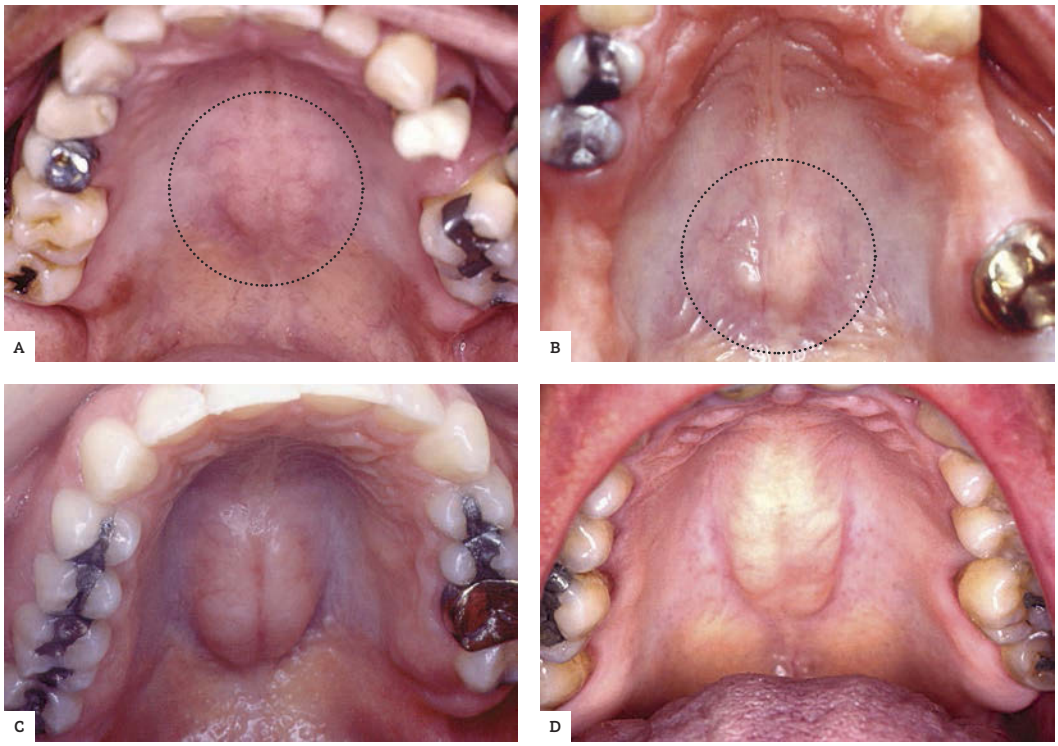

Figure 1. Types of torus palatinus: **A**) flat; **B**) nodular and **C**, **D**) lobulated.

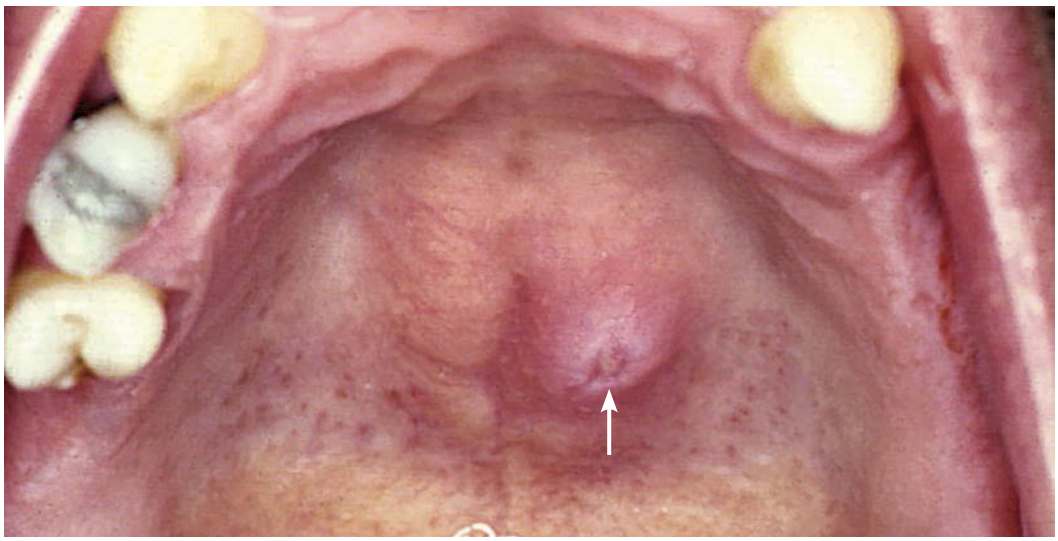

Figure 2. Torus palatinus ulcered by removable denture with metal bar directly placed over extremely thin mucosa. Within a few hours, the patient presented signs of ulcer over the torus.

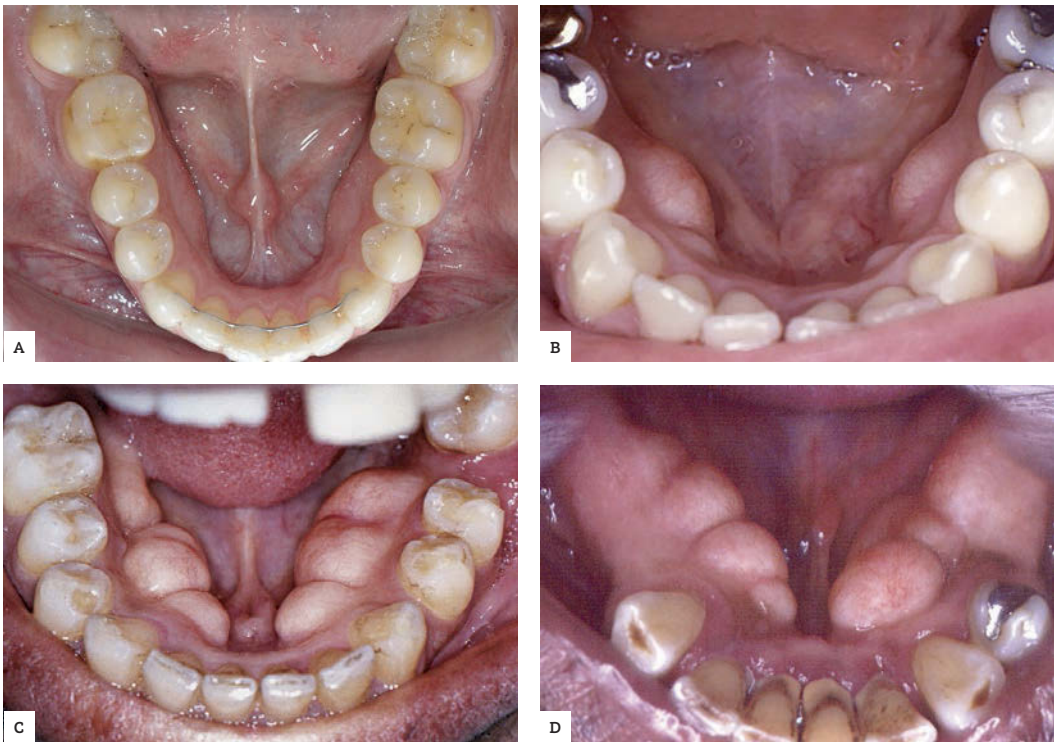

Figure 3. Tori mandibularis on both sides of the jaws: **A**) flat; **B**) nodular and **C**, **D**) lobulated.

carriers of those genes do not necessarily present the manifestations of variation. Other relatives, however, might as well present them. In some cases, when the transmission of some alterations has not been studied in unprecedented detail from a genetic and hereditary perspective, its pathophysiology is commonly referred to as familial tendency.

Likewise, whenever an anatomical variation or lesion is most commonly shared by a specific ethnic group, its pathophysiology is commonly referred to as a "race factor".

Epigenetic factors might exert strong influence over the conditions and genetically determined morphology of variations, since they modulate or constitute the micro and macro environment where genes are activated and the other parts of cells perform the activities transmitted by the genes. It is worth noting that epigenetic factors do not promote structural changes or genetic mutations, although they influence how they function.

It is possible to assert that the final outcomes of a given body function or structure result from genetically transmitted

information as well as from the action of factors external to the genes. For this reason, they are termed "epigenetic". This might occur, for instance, with the morphology of the jaws: some people from the same family might differ due to the different epigenetic factors each family member is exposed to, including twins.

This might explain why several members of the same family have torus palatinus and/or torus mandibularis of different morphology and sizes. The same applies to tooth and lingual features of cleft lip and palate at the palatal rugae. The same epigenetic factors, equal in manner and intensity, will hardly be shared by two genetically identical people.

CORRECT TERMS FOR DISORDERS ACCORDING TO THE PERIOD OF DEVELOPMENT AFFECTED: APPLIED CONCEPTS

Developmental disorders are occasionally termed dysgenesis or malformation. They are grouped into anomalies, dysplasias and deformities.

Anomalies are alterations of several aspects of an organ or tissue — including number, shape and position. In general, the function of the affected structure is hindered, as the cause of a given anomaly is established at the early stages of development (or organogenesis). Two examples: cleft lip and partial anodontia.

Dysplasias affect the function of a given organ or tissue because they affect the latest stages of development, or histogenesis, especially differentiation and maturation. The following are two classic examples of dysplasia: Hereditary Ectodermal Dysplasia and Cervical Enamel Projection or Invagination.

Deformities are characterized by physical causes, such as lack of space or movement during fetal development, which impairs the ultimate formation of an organ or one of its anatomical parts. Two examples: micrognathia due to lack of upward movement of the fetus' head, as it occurs in Pierre Robin sequence, and root dilaceration.

Torus palatinus and torus mandibularis are classified as late developmental anomalies established at the final stages of formation of the jaws. They are considered anomalies of shape, not a dysplasia, because bone tissue of which they are composed of is completely normal from a structural and functional perspective.

THE TIME OF OCCURRENCE AND DIAGNOSIS: TERMS USED

Developmental disorders might occur and be diagnosed before birth, especially if the diagnostic technology currently available is taken into account, regardless of pathophysiology. Thus, it is possible to assert that developmental disorders are prenatal alterations.

Regardless of its pathophysiology, should the disorder be present at the time of birth, it is classified as and termed "congenital" which means "born with", etymologically speaking.

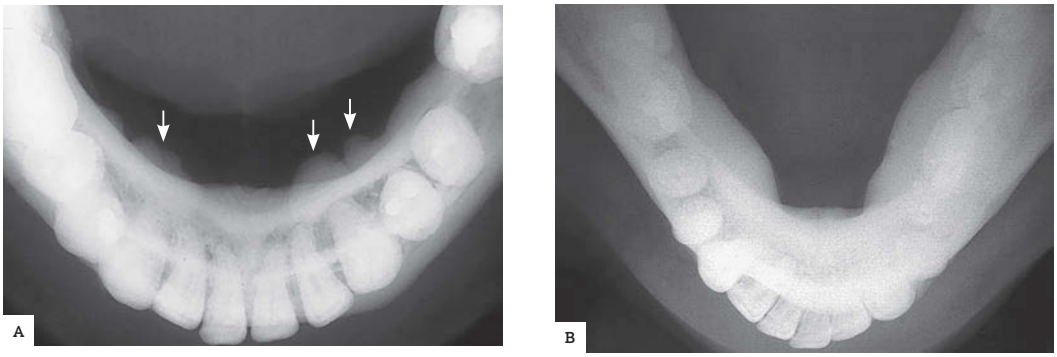

Figure 4. Radiographic images of nodular tori mandibularis on both sides of the jaws: **A**) multiple protuberances; **B**) wider basal bone in the mesiodistal plane.

Developmental disorders might occur and/or become manifest after birth, during childhood, adolescence or adulthood. Should that be the case, the developmental disorder is postnatal.

Torus palatinus and torus mandibularis become manifest after adolescence and have growth ceased by the end of maxillary and mandibular growth, between 22 and 24 years old. Despite associate familial tendency and ethnic factors, tori are postnatal developmental disorders. They are not congenital because tori are absent in one's jaws at birth. Obviously, they are not prenatal.

THE NATURE OF ITS CAUSES: TERMS USED

Based on their causes, developmental disorders are grouped into two basic types: hereditary and acquired (also, multi factorial inheritance).

Hereditary: developmental disorders inherited by genetic alterations passed on by one's parents; for instance, albinism and hereditary amelogenesis imperfecta. During adulthood, and also during the stages of development, humans might undergo genetic alterations that might not be passed on to the next generation, provided that they have not affected germinative cells which remain unaltered. Enamel hypoplasia induced by infection and trauma is a good example.

Hereditary disorders might be of genetic origin whenever one or a few genes, located inside a chromosome with normal

structure, are altered. On average, each chromosome has one thousand genes. Nevertheless, some developmental disorders are caused by chromosome alterations, which affects a large number of genes. Whenever this happens, several organs and tissues are damaged and have normal functioning hindered, which leads to syndromic cases.

In short, hereditary developmental disorders are classified into: **genetic,** whenever they result from single or multiple genetic alterations; and **chromosomal**, when they partially or completely affect a single chromosome or increase the number of chromosomes.

Acquired: developmental disorders resulting from external factors that affect the formation of organs or tissues without affecting germinative cells. A good example is anterior open bite caused by long-term thumb sucking: maxillary development is affected, but not passed on to the next generation.

Multi factorial inheritance: alterations passed on to the next generation, which only become manifest if modified genes interact with environmental factors. Someone might receive an altered gene, but it will only become manifest upon interaction with acquired factors. A classic example of multi factorial inheritance is cleft lip and palate and diabetes mellitus.

Case reports and literature reviews strongly suggest that torus palatinus and torus mandibularis are of hereditary origin; however, more precise methodological data are warranted to support such statement. In this case, it remains as an anomaly of shape of the jaws with familial tendency.

NUMBER OF DISORDERS PRESENT: CONCEPTS

Developmental disorders might be classified as single or multiple. Multiple developmental disorders are divided into three different groups:

Syndromes: of well-determined and well-known cause, with alterations recurring in a high number of patients and characterizing a typical condition, such as Down Syndrome or Trisomy 21.

Associations or syntropies: of unknown cause, with alterations recurring in a high number of patients and characterizing a typical condition, despite their undetermined cause.

Sequences: whenever multiple manifestations occur as a consequence of another, in ripple effect. Only the first manifestation had a cause and induced all the others that, in turn, derive from one another. For instance, the Pierre Robin sequence.

TORI ARE NOT NEOPLASMS!

Benign or malignant neoplasms represent a cell proliferation disorder, with loss of control over this important mechanism. Neoplasms — or tissue neoformation — represent cells and tissues growth that results from uncontrolled continuous rapid proliferation. Neoplasm growth is autonomous and unlimited.

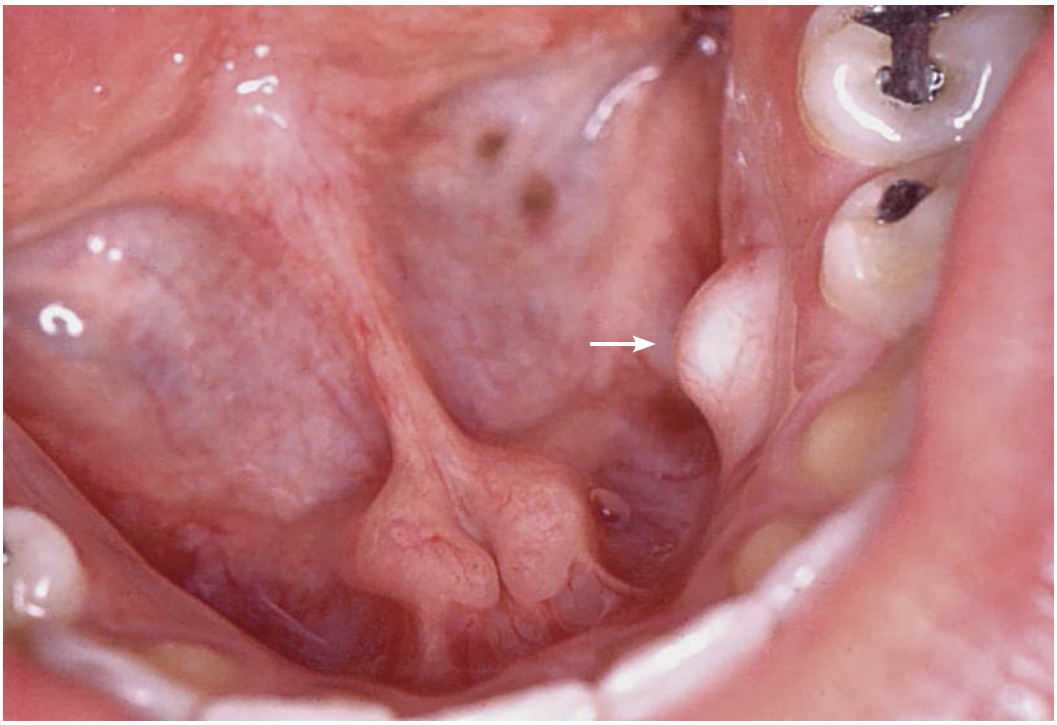

Figure 5. Torus mandibularis on one side, as it occurs in 20% of cases. Note its nodular shape and whitish color.

Osteoma, for instance, proliferates continuously and does not stabilize with time. For this reason, it requires treatment soon after diagnosis. Despite being benign, neoplasms expand, gradually promote structural damage and occupy spaces, thereby affecting function and esthetics. Benign neoplasm requires treatment: it does not stop growing! Only a small number of neoplasms, around 10%, usually malignant ones, are hereditary. All the others are of well-known cause.

Torus palatinus and torus mandibularis are not neoplasms because they start to growth and become noticed at puberty, reaching their maximum size between the age of 22 and 24 years old. It is during this stage of development that craniomandibular growth ceases in boys and girls, respectively. From this age on, tori stabilize in size and remain stable until old age, without denoting any neoplastic features when established in one's jaws.

Exostoses and enostoses might be a manifestation of several bone processes that vary from normal to aggressive diseases. An osteoma, regardless of its location in

one's skeleton, might grow inside the bones or projecting towards the inside of a natural body cavity, such as the maxillary sinus, and be a clinical manifestation of enostosis. However, an osteoma might also grow projecting from the bone surface and be a manifestation of exostosis. Torus palatinus and torus mandibularis are classic examples of exostoses.

Torus and osteoma also differ in relation to the fibrous capsule. An osteoma always has a capsular condensation of fibrous connective tissue circumscribing it and isolating the neoplastic process of neighboring tissues. In imaging examinations, this is revealed as a radiolucent halo that delineates the benign neoplastic proliferation of bone tissue. Tori should not be compared to or considered as benign neoplasms or some type of osteoma which is a benign neoplasm of continuous growth clearly circumscribed by a fibrous capsule. Tori are developmental disorders with peculiar features that characterize them as well-determined clinical conditions.

TORI ARE NOT HYPERPLASIAS NOR HYPERTROPHIES!

Normal cells control cell proliferation in order to satisfy one's organism functional and adaptive demands, according to its metabolic and/or adaptive environmental needs.

Whenever functional and/or adaptive demands are placed on a given body part, the organism induces the mechanism of proliferation in order to increase the amount of cells until such demands are satisfied. This type of adaptive response given by one's organism is known as hyperplasia: an increase in the number of cells within a given area and tissue, as an adaptive response.

Microscopically, torus palatinus and torus mandibularis do not present as hyperplasia because the bone they are made of is normal in terms of cell organization, distribution and number (Fig 8). The sites of hyperplasia, as an adaptive response, present with a higher number of cells, as it occurs in cases of ossifying periostitis.

On the other hand, hypertrophy represents a functional adaptation of tissues unable to proliferate; for instance, skeletal muscle cells. In these cases, in order to satisfy functional demands, the cells increase in volume, i.e., become bigger, but are unable to proliferate. In general, these larger cells can be viewed microscopically in tissue analyses. Microscopic sections of tori do not reveal larger cells, since, should it be necessary, osteoblasts have their proliferative ability preserved.

It is worth noting the terms "hyperplasia" and "hypertrophy" are used to identify phenomena of cellular nature relative to cell adaptation to increased functional demands.

TORUS PALATINUS: CLINICAL ASPECTS OF DIAGNOSIS

As indicated by the term itself, torus represents a protuberance or bone exostosis, with a flat base and extremely slow growth,

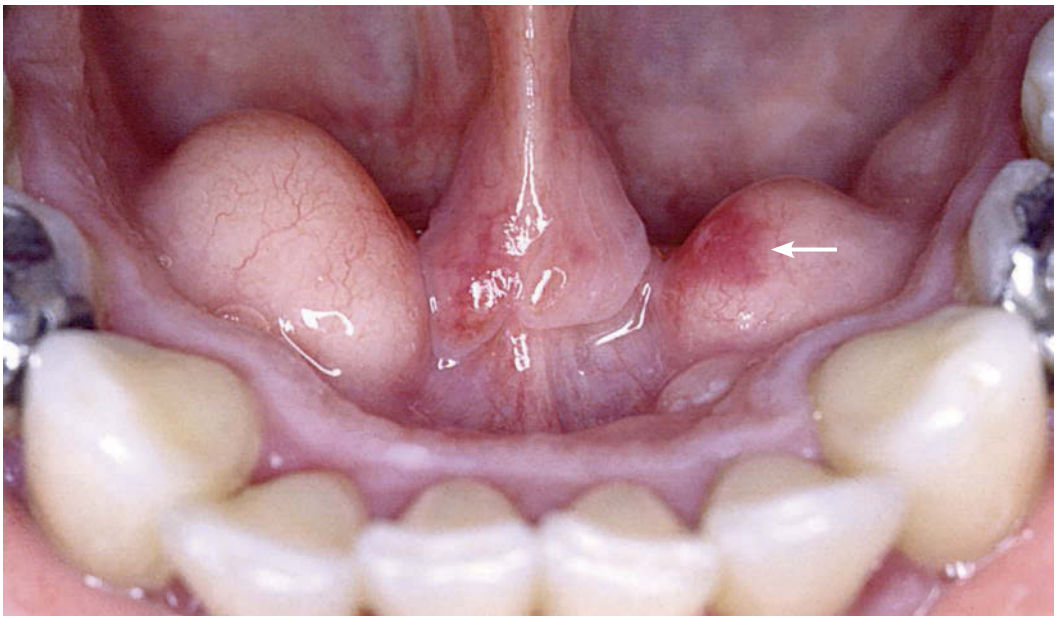

Figure 6. Asymmetrical tori mandibularis on both sides. One of them was ulcered (arrow) by solid rough food touching the thin oral mucosa.

located at the midline of the hard palate (Fig 1). It varies significantly in shape from one patient to another, as it can be flat, fusiform, nodular or lobulated.[16]

The oral mucosa covering the torus palatinus is normal; however, it is usually extremely thin and reveals the whitish color of the bone underneath, which renders the torus a protuberance of whitish or yellowish color (Fig 1). Due to being delicate, the oral mucosa becomes ulcerous with ease whenever prostheses or appliances of any kind (metal bars, expanding screws or acrylic plates) are placed (Fig 2). Should that be the case, within a few hours, ulcers and pain will appear near the torus palatinus. Torus palatinus per se is asymptomatic.

Causes associated with torus palatinus

Torus palatinus is a developmental anomaly of shape with late manifestation during growth and maturation of the jaws. There have been some family case reports, and an autosomal dominant pattern has been granted to it.[21]

There is lack of adequate methodological evidence that allows torus palatinus to be reported as being of neoplastic or hyperplastic nature, although it might occasionally be classified as so in a few studies in the literature.

The hereditary nature of the disorder is reinforced by research that reveals its high prevalence within certain ethnic groups, as the Eskimo and indigenous people. Its frequency varies from 20 to 25%,[6,14,18] with the disorder being more commonly found among women in a 2:1 ratio. It is usually diagnosed at the third decade of life.

Although the majority of studies classify torus palatinus as a variation of normality and consider the genetic-hereditary factor of the disorder as its major etiological factor,[21,22] a few studies occasionally highlight that environmental factors might exert some influence on its development.[10,12,13]

Prognosis and protocols for torus palatinus

Microscopic examination reveals that torus palatinus has normal bone structure, without any morphological sign of neoplasm or hyperplasia. In general, it is composed of thick cortical bone tissue and, deeper inside, a small area of trabecular bone.[5]

Stable growth at around the age of 22-24 years and the predominantly small size of torus palatinus prevent many patients from acknowledging its presence in the hard palate. On occasion, they are bigger, and the patient might have some concerns about his health and/or carcinophobia. For this reason, it is paramount to give reliable and through explanation about torus palatinus in order to calm the patient down.

From a biological standpoint, there is no need to surgically remove a torus palatinus. The former is only performed in cases requiring:

1. A metal or acrylic piece of appliance or prosthesis to go near the torus palatinus. In these cases, an ulcer will form within a few hours, causing the patient to feel a lot of pain and discomfort.
2. Speech, masticatory and hygiene function or even patient's comfort to be reestablished; always taking the size and shape of lesion into account.
3. A source of autogenous bone, based on the need determined by previous oral restorative treatment planning. Bone tissue harvested from torus palatinus is normal in function and structure (Fig 8).

TORUS MANDIBULARIS: CLINICAL ASPECTS OF DIAGNOSIS

Similarly to torus palatinus, torus mandibularis is a protuberance or exostosis mostly (80%) found on both sides of the jaw (Figs 3, 4, 5). It has a flat base, presents with slow growth and is located on the lingual surface of mandibular cortical bone, near canines and premolars, always above the mylohyoid line.[2,10]

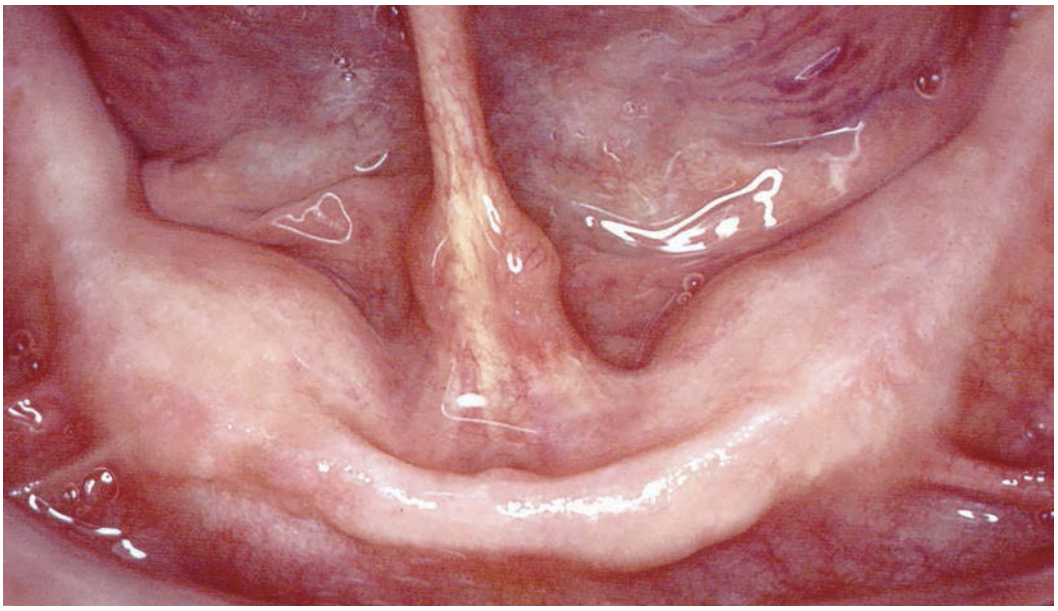

Figure 7. Tori mandibularis on both sides in an edentulous patient without alteration in shape and volume over the years.

Its clinical manifestation varies considerably, from a discreet protuberance to fusiform, nodular or lobulated large exostoses occupying almost the entire floor of the mouth (Figs 3, 4 and 5). It becomes clinically manifest as a single or multiple lobule.[2]

The oral mucosa covering the torus palatinus is extremely thin and reveals the whitish color of the bone underneath, which renders the torus a protuberance of whitish, pinkish or yellowish color (Figs 3, 5, 6) The oral mucosa becomes ulcerous and painful within a few hours whenever prostheses or appliances of any kind (metal bars, expanding screws or acrylic plates) are placed. This contrasts with its asymptomatic characteristic.

Causes associated with torus mandibularis

Family history reinforces torus mandibularis disontogenic origin, which is

associated with developmental disorders, as an anomaly with late manifestation during growth and maturation of the jaws. Research on the prevalence of torus mandibularis reveals it is present in 6 to 24% of the overall population. Additionally, it becomes manifest or are identified by patients until their third decade of life. Many patients are unaware of having this morphological alteration of the jaw, especially when torus mandibularis is small-sized.

There is lack of adequate methodological evidence that allows torus mandibularis to be reported as being of neoplastic or hyperplastic nature, although it might occasionally be classified as so in a few studies in the literature. Studies have been conclusive for some families: some cases are inherited with complete or incomplete penetrance.[1,7,8]

Its hereditary nature is reinforced by studies revealing a higher probability (from 40 to 60%) of having torus mandibularis whenever one or both parents have either torus palatinus or torus mandibularis.[21] Should neither one of parents have torus, the probability is reduced to 6-8% of the overall population.

Prognosis and clinical protocols for torus mandibularis

Microscopic analysis reveals bone tissue of torus mandibularis is normal, without morphological signs of neoplasm or hyperplasia. Microscopic sections of torus mandibularis reveal they are composed of thick cortical bone (Fig 8). On rare occasions, it presents with a small area of trabecular bone deeper inside.

Stable growth at around the age of 22-24 years and the predominantly small size of torus mandibularis prevent many patients from acknowledging its presence in the mandible. On occasion, they are bigger, and the patient might have some concerns about his health and/or carcinophobia. For this reason, it is paramount to give reliable and through explanation about the lesion in order to calm the patient down.

From a biological standpoint, there is no need to surgically remove a torus mandibularis. The former is only performed in cases requiring:

1. A metal or acrylic piece of appliance or prosthesis to go near the torus mandibularis. In these cases, an ulcer will form within a few hours, causing the patient to feel a lot of pain and discomfort.
2. Speech, masticatory and hygiene function or even patient's comfort to be reestablished; always taking the size and shape of lesion into account.
3. A source of autogenous bone, based on the need determined by previous oral restorative treatment planning. Bone tissue harvested from torus mandibularis is normal in function and structure.

From a therapeutic and prognostic perspective, tori do not become malignant

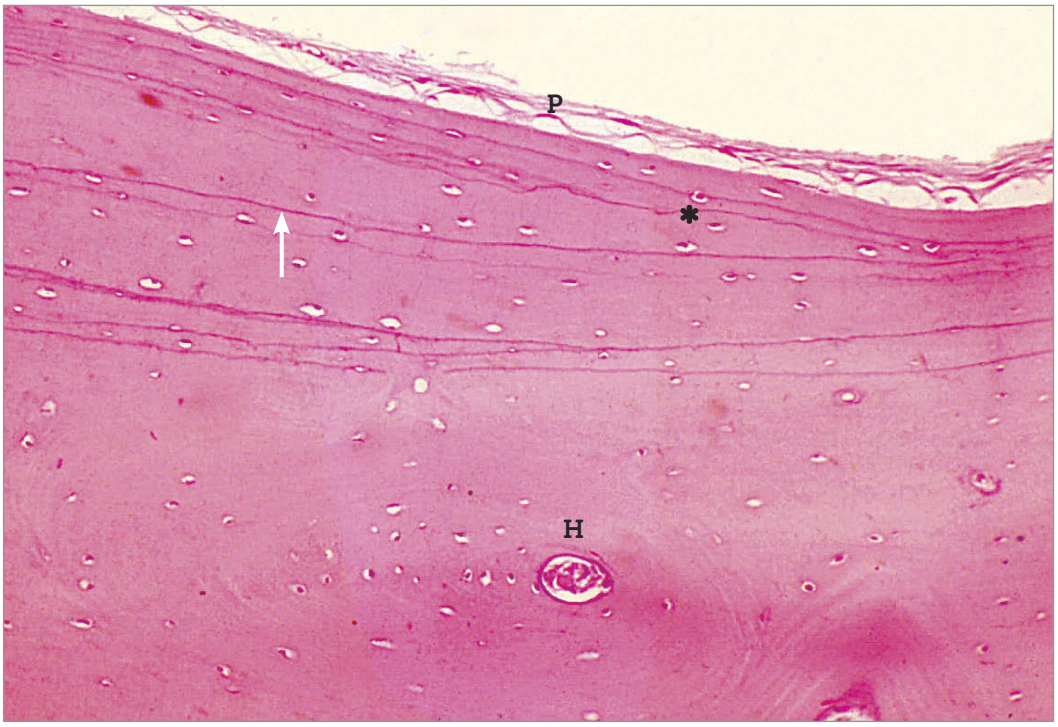

Figure 8. Most common microscopic aspect of torus palatinus and torus mandibularis represented by normal bone with apposition of layers (arrow), Haversian canal (H), osteocytes (*) and periosteum (P) (HE, 25X).

lesions. In restorative oral treatment planning, should it be necessary to place implants near tori (Fig 7), they are biologically considered normal bones in terms of function and structure.

VULNERABLE POINTS "OCCLUSAL HYPOTHESIS" TO EXPLAIN TORI

There is a hypothesis used to explain the origin of tori, especially those found in the mandible, which is not exclusively genetic. In this hypothesis, environmental and functional factors also act during adolescence,[3,9,11] contributing to tori growth; even though such factors decrease during adulthood and remain stable at an old age. Based on the prevalence and age group of patients, as well as on the shape of their mandible, there is some correlation between the development of tori and the use of drugs, attrition and temporomandibular

disorders.[15] These variables represent environmental factors that somehow interfere in the development of tori.

Many patients, with or without tori, present with occlusal overload, grinding, attrition and clenching that begin during adolescence and persist throughout life. Should there be a correlation between occlusal stress, grinding and temporomandibular disorders, why would tori growth not vary with age and increase after 22 and 24 years old when the growth of the jaws ceases?

In order to include occlusal overload, grinding and other conditions in the set of epigenetic factors behind the development of tori, there should be stronger and more direct methodologically determined evidence.

Nevertheless, some aspects of this hypothesis, which explains that occlusal overload, grinding and other environmental factors would influence the development of tori, cannot coherently explain the following:

1. Should tori be an adaptive response to mechanical stress resulting from grinding, clenching or occlusal overload, tissues would present with hyperplasia or hypertrophy. However, microscopic examination does not reveal such condition (Fig 8).
2. If bone remodeling is continuous in order to promote ongoing functional adaptation of dynamic bone restructuration, why do tori not disappear in edentulous or elderly patients (Fig 7), given that they would no longer have any function, as it occurs with the alveolar process upon tooth loss?

Tori do not disappear with a reduction in occlusal overload, with age or with tooth loss because they are most likely to represent part of the shape of a maxilla and mandible genetically determined, with variation in anatomical structures, as evinced by systematic studies conducted with families and certain ethnic groups.

FINAL CONSIDERATIONS

Current knowledge about torus palatinus and torus mandibularis reveals that they are part of the development of the jaws, within a given population and fitting a genetic pattern, as an anatomical variation of normality.

On occasion, tori are larger and might prevent patients from performing some functions, including hygiene, in addition to hindering appliance and prosthesis placement. For this reason, tori are classified as developmental anomalies of shape. There is not enough strong methodological evidence capable of attributing the origin and cause of tori to mechanical factors, such as occlusal overload, grinding, temporomandibular disorders, systemic conditions or drugs.

Tori are composed of normal bone in terms of function and morphology, without microscopic signs of hyperplasia, hypertrophy or neoplasm. This bone might as well be as autograft harvesting site or osseointegrated implant placement site within clinically acceptable conditions.

REFERENCES

1. Barbujani G, Rolo M, Barrai I, Pinto-Cisternas J. Torus palatinus: a segregation analysis. Hum Hered. 1986;36(5):317-25.
2. Choi Y, Park H, Lee JS, Park JC, Kim CS, Choi SH, et al. Prevalence and anatomic topography of mandibular tori: computed tomographic analysis. J Oral Maxillofac Surg. 2012 Jun;70(6):1286-91.
3. Cortes ARG, Jin Z, Morrison MD, Arita ES, Song J, Tamimi F. Mandibular tori are associated with mechanical stress and mandibular shape. J Oral Maxillofac Surg. 2014;72(11):2115-25.
4. Costeira O. Termos e expressões da prática médica: elementos greco-latinos de uso médico. Rio de Janeiro: Ed. Farmoquímica; 2001.
5. García-García AS1, Martínez-González JM, Gómez-Font R, Soto-Rivadeneira A, Oviedo-Roldán L. Current status of the torus palatinus and torus mandibularis. Med Oral Patol Oral Cir Bucal. 2010 Mar 1;15(2):e353-60.
6. Gorsky M1, Raviv M, Kfir E, Moskona D. Prevalence of torus palatinus in a population of young and adult Israelis. Arch Oral Biol. 1996 Jun;41(6):623-5.
7. Gorsky M, Bukai A, Shohat M. Genetic influence on the prevalence of torus palatinus. Am J Med Genet. 1998 Jan 13;75(2):138-40.
8. Gould AW. An investigation of the inheritance of torus palatinus and torus mandibularis. J Dent Res. 1964 Mar-Apr;43:159-67.
9. Haugen LK. Palatine and mandibular tori: a morphologic study in the current Norwegian population. Acta Odontol Scand. 1992 Apr;50(2):65-77.
10. Ihunwo AO, Phukubye P. The frequency and anatomical features of torus mandibularis in a Black South African population. Homo. 2006;57(4):253-62.
11. Jainkittivong A, Langlais RP. Buccal and palatal exostoses: Prevalence and concurrence with tori. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2000 Jul;90(1):48-53.
12. King DR, Moore GE. An analysis of torus palatinus in a transatlantic study. J Oral Med. 1976 Apr-Jun;31(2):44-6.
13. King DR, Moore GE. The prevalence of torus palatinus. J Oral Med. 1971 Dec;26(3):113-5.
14. Kolas S, Halperin V, Jefferis K, Huddleston S, Robinson HBG. The occurrence of torus palatinus and torus mandibularis in 2,478 dental patients. Oral Surg Oral Med Oral Pathol. 1953;6(9):1134-41.
15. Morrison MD, Tamimi F. Oral tori are associated with local mechanical and systemic factors: a case-control study. J Oral Maxillofac Surg. 2013 Jan;71(1):14-22.
16. Nogueira AS, Gonçales ES, Santos PS, Damante JH, Alencar PN, Sampaio FA, Garcia AS. Clinical, tomographic aspects and relevance of torus palatinus: case report of two sisters. Surg Radiol Anat. 2013 Nov;35(9):867-71.
17. Salvat. Dicccionario médico. Barcelona: Salvat; 1972, pg. 162.
18. Sathya K, Kanneppady SK, Arishiya T. Prevalence and clinical characteristics of oral tori among outpatients in Northern Malaysia. J Oral Biol Craniofac Res. 2012 Jan-Apr;2(1):15-9.
19. Skinner HA. The origin of medical terms. 2.ed. Baltimore: Williams & Wilkins, 1961.
20. Stedman TL. Dicionário médico. 23. ed. Rio de Janeiro: Guanabara Koogan; 1979. pg. 1415.
21. Suzuki M, Sakai T. A familial study of torus palatinus and torus mandibularis. Am J Phys Anthropol. 1960;18(4):263-72.
22. Woo JK. Torus palatinus. Am J Phys Anthropol. 1950 Mar;8(1):81-111.