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Review Article

# Risk in Patients with Orthodontic Treatment

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## Abstract

Orthodontic treatments are long, progressive and have a relatively slow clinical evolution. Most consultations are scheduled, with predictable clinical results and there are usually few emergencies. Therefore, a bibliographic review was carried out with the objective of showing the main risks and complications of orthodontic treatments.

**Keywords:** Risk; Orthodontics; Preventive Orthodontics; Removable

## Introduction

The comprehensive assessment of the patient and their stomatognathic system, together with mastery of the technique used and biomechanics, guarantee that unforeseen unwanted effects are rare. However, orthodontic treatment is not without risks. Avoiding them depends largely on a correct diagnosis and careful planning. 2. Risk and risk factors. The health – disease process is based on a balance between the general resistance of people to preserve physical and mental well-being in opposition to the action of aggressive agents that promote its destruction. These agents, or risk factors, predispose to biological damage and threaten the correct outcome of the treatment. Risk factors increase the possibility of certain health damage occurring. This probability of damage is called risk. Therefore, there is a difference between the terms "risk factor" and "risk." The first refers to any characteristic or circumstance present in an individual or group of people that makes them especially susceptible to suffering from a morbid process; the second refers to the probability that said process will occur.

## Objective

Describe the main risks and complications of orthodontic treatments.

### Reference search methods

Scientific information was collected through a search using the following descriptors in English: The Medical Subject Headings (MeSH): "risk, orthodontics, preventive orthodontics,

### Analysis strategy

The search was based solely on risks and complications of orthodontic treatments.

### Developing.

### Risk factors in orthodontics.

Identifying the risk factors in a patient before starting orthodontics allows the clinician to work to eliminate them, mitigate them or, ultimately, carry out the treatment under the influence of it, taking additional measures that reduce the probability of causing damage. Risk factors may or may not be modifiable depending on the existing possibilities of influencing them. They may be associated with the patient or the treatment. Taking into account the above, it is possible to classify them as follows:

### Patient's own risk factors.

### Modifiable

Its existence is related to individual aspects of the patient; Its frequency and severity will vary considerably from one individual to another. It can be thought that habits are modifiable risk factors par excellence. However, changing lifestyles is usually extremely difficult if there is not the patient's strong will and full awareness of the need for change to successfully complete the treatment. In the vast majority of cases it is preferable to carry out habit control before installing any type of device, unless said device is aimed precisely at eradicating the habit [1,2].

## Poor tooth brushing

Taking into account that orthodontic movements are based on the tooth's supporting apparatus, any habit that threatens periodontal health will negatively affect orthodontic treatment. Tooth brushing is a beneficial habit when done efficiently. Carrying it out with the necessary frequency and thoroughness prevents the proliferation of dental plaque, the main etiological agent of periodontal disease.

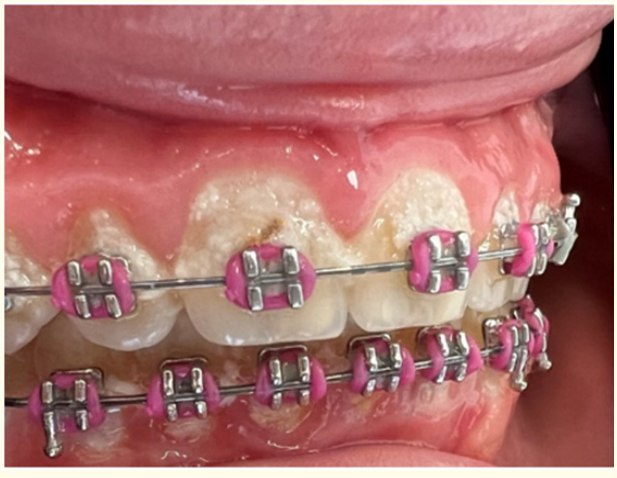

**Figure 1:** Presence of bacterial plaque in a patient with fixed appliances.

White spot lesions (WML) are one of the main adverse effects of orthodontic treatment with fixed appliances, as a consequence of insufficient brushing. Its presence shows an imbalance in the process between demineralization and remineralization of the enamel surface. These lesions can be defined as a porosity in the subsurface of the enamel due to demineralization by organic acids.

Patients undergoing orthodontic treatment may present a high risk of developing BML, with risk factors for its formation such as poor compliance with treatment, poor oral hygiene, prolonged treatment time and the age of the patients. These characteristics mean that LMB have a high incidence in these patients, reaching prevalences of up to 84% according to various authors.

The way in which spot lesions are evaluated can affect these values, with different clinical strategies, the use of photographs or devices and techniques that allow the presence of BML to be identified in early stages. Various proposals have been described for the management of BML, such as the topical application of fluoride, amorphous calcium phosphate, application of sealants, and incorporation of antibacterial agents into orthodontic cements with the intention of avoiding or preventing its formation [3,4].

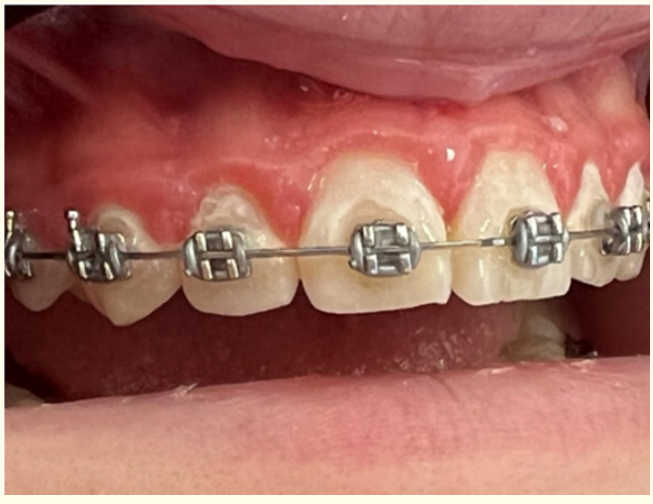

**Figure 2:** White spot lesions in a patient with fixed appliances.

Smoking. Not only dental plaque irritates periodontal tissues. A risk factor that has the ability to act locally on the periodontium, but also systemically, is smoking. This habit should not be downplayed; it can act on the stomatognathic system in very early stages of life. Maternal smoking is an important etiological factor in the genesis of cleft lip and palate. The hypoxia that it produces on the tissues interferes with the movements and fusion of the nasal and palatine processes of the embryo.

Smokers can be classified as light, when they consume less than 5 cigarettes per day on average, moderate, between 6 and 15 cigarettes per day, and severe, when they smoke more than 16 cigarettes per day. It is the main cause of oral cancer and among the harmful oral effects we can highlight the increase in the progression of periodontal disease. The average number of smokers wearing orthodontic appliances is around 15%, an insignificant figure. Different studies have been published on the molecular and cellular effects of nicotine, which have proposed that it may affect bone resorption and apposition [3-5].

The mechanism that exposes this speaks of an increase in the expression of cyclooxygenase 2 (cox 2) and prostaglandin E2. Cox 2 is the main enzyme that converts arachidonic acid to prostaglandins, which are important factors in bone remodeling and orthodontic tooth movement. Nicotine can increase orthodontic tooth movement, at the expense of increasing prostaglandins. The study carried out by Henemyre and collaborators concludes that nicotine leads to an increase in the number and function of osteoclasts, and this effect is what can help accelerate tooth movement. It is stated

that the increase in the dose of nicotine also leads to the greatest increase in tooth movement, being, therefore, a dose-dependent effect.

Authors such as Hapidin., *et al*. recommend that severe smoking patients first start treatment to stop smoking or reduce nicotine doses before starting orthodontic treatment, due to the high risk of bone loss that can occur throughout active treatment. In light or moderate users, the use of light forces and spaced appointments is recommended, in addition to strict periodontal control (Figure 3).

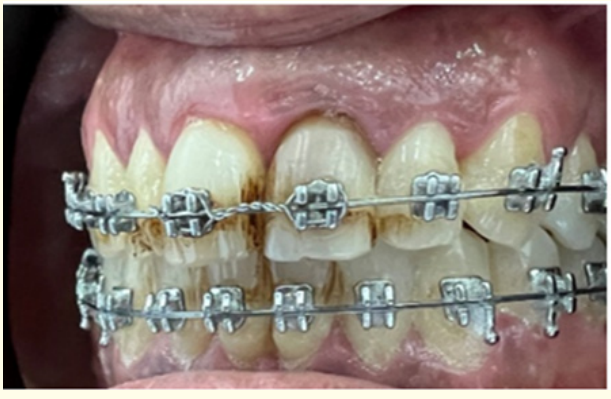

**Figure 3:** Smoking patient with fixed appliances.

Note the stains on the enamel produced by nicotine.

### Deleterious habits. a) Altered respiratory pattern

An altered breathing pattern can lead to serious problems in facial and body development. When breathing habitually through the mouth, pathological changes occur in the posture of the jaws, tongue and head. Mouth breathing leads to imbalances in the forces that act on the facial skeleton and dental arches. Mouth breathing requires practically constant inocclusion of the arches when the jaw rotates clockwise and the tongue is in a low position.

The pressure exerted by the cheeks on the teeth, without the antagonistic action of the tongue, will favor the development of narrow arches with an ogival shape and protrusion of the incisors. Furthermore, poor occlusal contact favors egression of the posterior teeth, which predisposes to an open bite, unless there is compensatory vertical growth of the mandibular ramus. Whatever the case, the vertical dimension of the face will be increased. Poor nasal airflow does not allow the air column to serve as an adequate functional matrix in the development of the maxillary basal bone. The resulting skeletal hypoplasia is accompanied by poor development of the nasal cavity, paranasal sinuses, and a deep palatal vault, easily identifiable during physical examination [4-6].

The postural changes of the head with respect to the trunk are the result of the unconscious adjustment made by the patient to reduce air resistance during breathing. All these characteristics are part of the so-called adenoid facies. It is necessary for the orthodontist to know how to early identify the presence of an altered respiratory pattern and the cause that causes it. At rest there is more resistance to the passage of air when breathing through the nose than through the mouth.

This leads to a greater inspiratory effort, which is totally physiological. However, when air resistance through the nasal passage is abnormally high, the inspiratory effort is insufficient and the patient unconsciously switches to mouth breathing. Narrowing of the nasal cavity and nasopharynx increases resistance and can sometimes cause a total obstruction of the airway, making it impossible to carry out the function correctly. In these cases, mouth breathing is functional since it is essential for the patient to maintain life.

Patients with functional mouth breathing can continue to breathe through the mouth even when the narrowings and/or obstructions have been eliminated. In these cases, mouth breathing can be considered a habit [3,4,7].

It is necessary to establish a correct treatment plan to determine what type of mouth breathing the patient has. If this is aimed at eliminating the habit when in reality it is a functional problem, it will inevitably lead to failure. It will also be accompanied by a feeling of frustration and guilt on the part of the patient who, despite having cooperated with the treatment, will feel unable to achieve the proposed objectives. It is not necessary to refer to an otorhinolaryngology specialist to establish a differential diagnosis. Multiple mechanisms have been described that can be carried out in the consultation to achieve this.

One of them, which is really simple, is to ask the patient to hold a tongue depressor with their lips for at least one minute. If the patient manages to keep it in position, it can be ensured that the nasal airway is patent. However, we must be attentive to the appearance of symptoms that may reveal pathological narrowing, such as the appearance of dyspnea, increased heart rate, or simply the patient removing the lip depressor. In these cases, the patient must be referred. It should also be taken into account that the absence of these symptoms does not rule out the existence of problems, since air requirements are lower at rest than during any type of exercise, as basic as speech [7,8].

That is why it is recommended that the patient also be asked, while holding the depressor with his lips, to perform some type of light exercise such as squats or jumping. If, despite this, he is able to hold the depressor, you can be completely sure that the nasal airway is patent and that we are dealing with a mouth breathing habit. In these cases, referral is not necessary and treatment is provided by the orthodontist. Narrowings and obstructions can be temporary, such as those observed in colds and allergies, resulting from inflammation of the nasal mucosa, or permanent due to hypertrophies of the turbinates, adenoids, and pharyngeal tonsils.

This must also be taken into account, because in allergic patients, the high frequency of temporary obstructive events may be equivalent to chronic obstruction. In these cases the patient should also be referred to an allergist. No orthodontic treatment should be started without oral breathing control because the risk of failure is high. For this reason, its treatment must be among the first therapeutic objectives. It can be established as a consensus that, from a physiological point of view, nasal breathing is the most appropriate for the correct development of the stomatognathic system. However, in certain situations, we all need to breathe through the mouth without this constituting a functional problem [3,4].

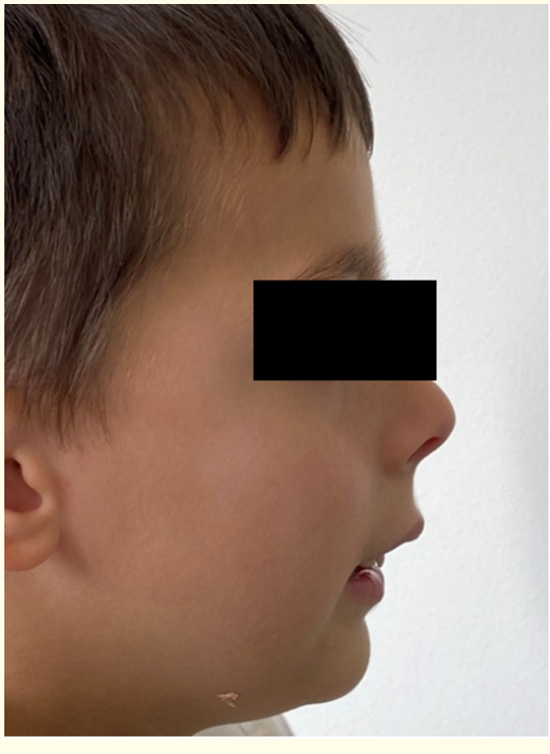

**Figure 4:** 5-year-old boy with oral respirator.

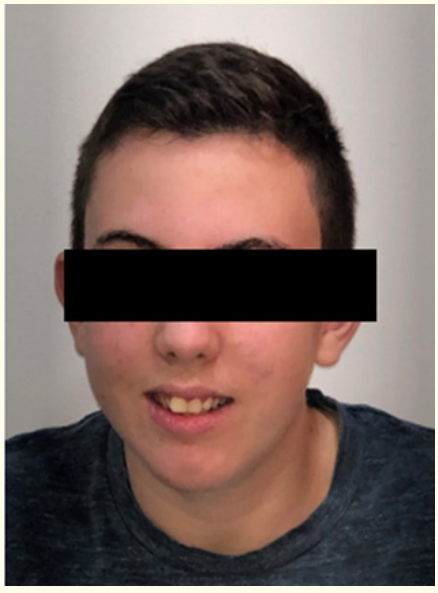

**Figure 5:** Adolescent at rest with oral breathing.

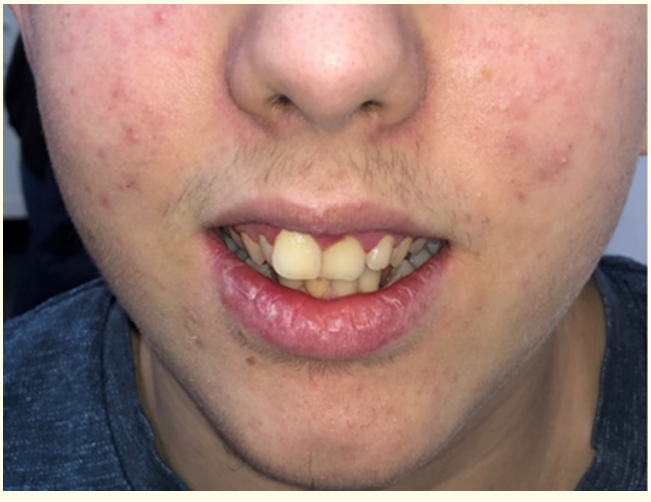

**Figure 6:** Adolescent in the previous image, oral respirator at rest.

## Digital suction

The characteristic malocclusion resulting from sucking is due to a combination of direct pressure on the teeth and an alteration in the pressure pattern of the cheeks and lips at rest. As in all abnormal habits, the repercussions that thumb sucking has on dentofacial structures will depend on the chronology, intensity, duration and frequency of the habit. In general, thumb or pacifier sucking during the first 18 months of life does not present risks for teething. Between 18 months and 3 years, the damage is limited to the

anterior sector (or to one of the sides in less frequent forms of suction). But, if these habits persist once the permanent teeth have erupted, it can cause malocclusion, consisting of open and spaced upper incisors, lingually deviated lower incisors, anterior open bite and narrow upper jaw. If the patient has a normal growth pattern and other factors do not have an impact, such as oral breathing due to chronic obstruction of the upper airways, it is very likely that by stopping the habit early, the occlusion will normalize [7-9].

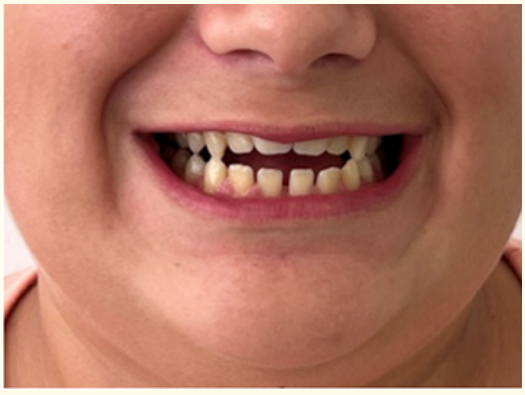

**Figure 7:** 15-year-old adolescent with digital sucking habit and permanent dentition.

Other habits There are habits with the capacity to generate malocclusions if they are maintained for a sufficient duration and intensity. Among them are onychophagia, postural habits, especially when sleeping, bruxism, interposition of the lower lip or the use of musical instruments. Bruxism requires a multidisciplinary approach by the pediatrician, dentist and psychologist, since complex problems lie behind it and its occlusal, aesthetic, and temporomandibular joint risks cannot be neglected.

On the other hand, the interposition of the lower lip sometimes leads to protrusion of the upper incisors with retrusion of the lower incisors. When this habit is severe, it is usually accompanied by dermatological lesions on the lower lip. The use of musical instruments can also lead to malocclusion. Previously, it was suspected that wind instruments could modify the position of the anterior teeth. An example is the clarinet, which could increase the overjet due to the way the tongue is held between the incisors, therefore, the clarinet could be considered to represent a potential class II malocclusion factor [9,10].

For some stringed instruments such as the viola and violin, the head and jaw must be positioned in a certain position that contributes to the pressure of the tongue and lips/cheeks, and which could lead to asymmetries in the shape of the arches. These types of tooth displacement have been observed in professional musicians, but even so they are not very striking and hardly any effects are seen in the majority of infants. It could be concluded that labial and lingual pressures during the use of instruments are too ephemeral and do not cause any damage, except for musicians with longer duration.

## Mobile devices

Mobile devices are relatively new, versatile, accessible and very attractive technological instruments, especially for young people, whose use carries a risk of abuse and addictive behavior. There is a wide variety of devices with mobile functions such as cameras, phones, computers, tablets, GPS devices, music players and video game consoles, among others. Abuse or addiction are not the only harmful effects, other risks have been investigated such as mobile harassment, a greater risk of work and traffic accidents, related to sensory interference during employment. It also presents neurological and neuropsychological risks due to loss of sleep, greater interpersonal anxiety and a lower level of self-esteem. Craniofacial posture is also highly influenced by prolonged use of these devices.

## Phrenic adhesions

The frenulum is a connective tissue that is wrapped in a membrane that runs from the alveolar cord to the lip, cheek or tongue. Its function is to ensure complete movement of the tongue. But, incorrect development of this can generate different complications, not only for oral health but also for orthodontic treatments. In the oral cavity there are three types of frenulum, the upper and lower labial and the lingual.

As for the upper labial frenulum, it is a fibrous fold of mucosa that can have various shapes and sizes. Normally, it begins on the inner surface of the upper lip, inserting into the gum and is present in both children and adults. The lower labial frenulum is located in the lower midline of the mouth and unites the lip with the inserted gum under normal conditions. And the lingual frenulum is a fibrous cord that extends from the base of the tongue to the floor of the mouth, where it is inserted. It is formed during the gestation process and can develop into all possible sizes and thicknesses. Phrenic adhesions present risks in different aspects such as orthodontic treatment, occlusion and speech, but everything will depend on each case. The dentist must carry out a thorough analysis of the braces before applying any type of orthodontic treatment, since the alterations that are usually found in this area harm the teeth in their position and size over time. If the upper labial frenulum is too

low, not only will the mobility of the upper lip be compromised, but the upper teeth will end up separated from each other [11,12].

The lower frenulum does not usually cause so many inconveniences in orthodontics, but a pathological insertion can cause gingival recessions. If it is the lingual frenulum, due to ankyloglossia, this condition is capable of influencing and harming the patient's speech, causing diction problems and affecting the position and size of the jaws.

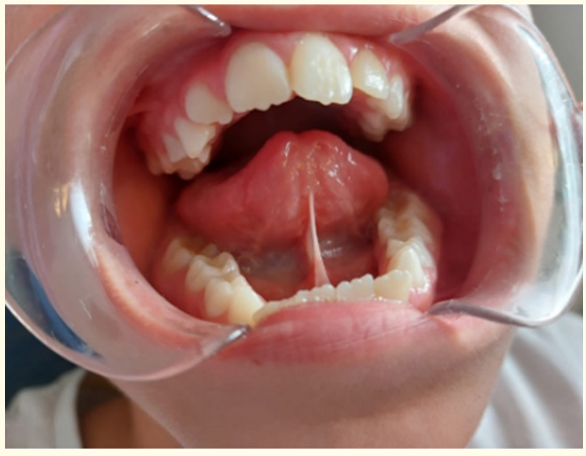

**Figure 8:** Pathological lingual frenulum.

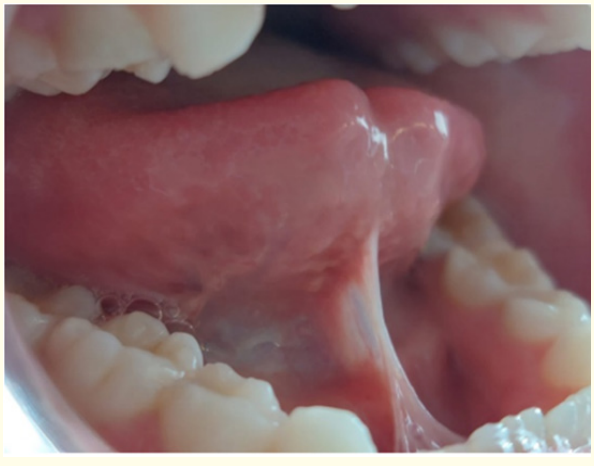

**Figure 9:** Pathological lingual frenulum.

## Not modifiable

In most cases, the origin of a malocclusion is due to a distortion of normal development. It is very difficult to find the specific origin or risk factor in a concrete way and it is most often the result of the synergy of different factors. Among the non-modifiable risk factors the following can be described:

## General health and medication

The general health of the patient is an important point when carrying out orthodontic treatment, because many pathologies refer to occlusal alterations that can represent a significant challenge for the professional when it comes to improving the patient's occlusion or improving their conditions in the as the pathology allows, since they can generate limitations in the orthodontic objectives of the treatment. The most common situations with which orthodontic treatment and the general health situation of a patient are related are genetic deformities. Among the most common we can find: cleft lip or palate, cerebral palsy, torticollis, cleidocranial dysostosis, congenital syphilis and examtematous fever. Cleft Lip and Palatine: Represents a deformity of congenital origin resulting from the lack of coalescence of the segments that form the lips and palate. When cleft palates occur, allowing communication between the mouth and the nasal passages, the functional disorders are more serious and the prognosis is more dismal. Suction becomes impossible and swallowing is difficult, with fluid reflux through the nose being very common. The nasal passages, having direct communication with the mouth, are frequently inflamed, causing chronic coryzas [10-12].

This malformation consequently generates skeletal, functional and psychological changes, with a complete imbalance of the dental arch, difficulty in chewing, gingivitis, periodontitis, early loss of permanent teeth, among others. Cerebral palsy: It consists of a lack of muscle coordination attributed to an intracranial injury. It causes disturbances in muscle function when chewing, swallowing, breathing and speaking. In addition to this, these patients usually eat a soft diet due to the lack of chewing, which generates hypotinicity of the perioral muscles, lack of eruption of the temporary and permanent teeth according to the age of the patient, generating a pseudo ankylosis. Malocclusions in children with cerebral palsy involve dento-skeletal and joint problems such as

- Dental class II with increased overjet and overbite, relationship with muscle hypotonia and oral breathing.
- Open bite with protrusion of incisors due to lingual interposition.
- Forward projection of the head.
- Alterations of the temporomandibular joint: limitation of movement, crepitation, condylar dislocation, pain.

## Torticollis

Consists of atrophy of the sternocleidomastoid muscle, as consequences of which facial asymmetries occur as a result of mandibular development. There is also a marked deviation of the dental

midline and the treatment must be carried out as early as possible, to prevent the malocclusion from being corrected. Cleidocranial dysostosis: it is a congenital and hereditary defect, which triggers maxillary retrusion and protrusion of the mandible with delayed eruption of permanent teeth and with a high frequency of presence of supernumerary teeth. Congenital syphilis: Systemic syphilis infection can cause Hutchinson's teeth, which are screwdriver-shaped anterior teeth or mulberry-shaped molars. The spirochetes of syphilis penetrate the blood vessels of the areas where there is odontogenesis, interfering with the levels of differentiation and maturation, thus generating malformations [3,13,14].

### Exanthematous fever

Rubella, chickenpox, measles and scarlet fever can also affect the development of teeth. The high fever that accompanies these infections alters the tissues of ectodermal origin, resulting in grooves in the enamel. In children with congenital rubella, the most common dental anomalies are partial or total hypoplasia of the enamel of primary teeth, as well as delayed eruption. Down syndrome: it consists of a congenital disorder that is characterized by having a replica of the paired chromosome 21, it is also known as trisomy 21. It is the most frequent congenital anomaly, with the highest prevalence and that affects all races equally, encompassing mental and behavioral changes as well as physical and oral malformations.

As these patients present alterations in the oral area, chewing and phonation function may sometimes be compromised, with harmful consequences for their nutritional status and social incorporation. Patients with DS present specific sorofacial characteristics such as: brachycephaly, persistent metopic suture, absent sphenoid sinuses, hypoplastic maxillary sinuses, maxillary, mandibular and skull base hypoplasia. Likewise, some of these anomalies influence the most frequent presentation: class III malocclusion, posterior crossbite, anterior open bite and oral breathing, which are increased and made chronic by hyperplasia of the tongue, which also adopts a low position in the mouth. oral cavity stimulating mandibular prognathism and stimulating the development of class III [3,4].

The use of drugs or polymedicated patients during treatment does not usually represent a problem, however, it is necessary to know the type of medication and the risks they may represent for the positive evolution of orthodontic treatment, but above all the impact that not following it may have. some guidelines according to the pathology that the patient presents. The patient undergoing orthodontic treatment may be consuming a wide variety of medications, but those that can reach the mechanically stressed bone tissue, through blood circulation, interacting with local "target" cells, are the ones that can mainly affect the treatment. orthodontic. The combination of the mechanical effect of forces and some of these agents can result in inhibition or stimulation of tooth movement. Within this group are nonsteroidal anti-inflammatory drugs (NSAIDs) and bisphosphonates that inhibit MDO and corticosteroids that stimulate it [14,15].

The group of drugs most frequently used during orthodontic treatment are NSAIDs, used to control pain after the application of forces to the teeth. The anti-inflammatory effect of this group of drugs is the result of blocking the synthesis of prostaglandins from arachidonic acid, through the inhibition of the cyclooxygenase enzyme. Clinical studies have identified the role of prostaglandins in the bone resorption process, determining that they have a direct action in increasing the number and size of osteoclasts and in stimulating their resorptive activity. This is why the use of NSAIDs for pain control in orthodontics has been questioned and studied in recent years, because not only would they have a positive effect on pain management after orthodontic activations, but they could also affect the sequence of movement. tooth, by inhibiting, or at least decreasing the relationship between inflammation and the bone resorption process.

Most studies demonstrate the effectiveness of NSAIDs in pain control, but they also establish that in some way they can affect the effectiveness of tooth movement, which is why today the lines of research try to determine which would be the anti-inflammatory with the least impact. in bone remodeling, and the latest publications point to paracetamol as a good therapeutic alternative. Paracetamol is one of the most used drugs for the safe and effective control of pain during treatment. It acts by decreasing the products of cyclooxygenase, preferably in the central nervous system, without significantly altering the peripheral secretion of PGs, thus experimental studies in animals they have concluded that the use of paracetamol would not alter bone remodeling, that is, it would have no effect on the range of tooth movement [15,16].

The continuous increase in corticosteroids for the treatment of medical conditions, such as arthritis, allergies, kidney disorders, collagen disorders and others, has made both clinicians and researchers interested in understanding the mechanisms by which these medications could alter bone metabolism. Corticosteroids are related to the control of carbohydrates, fat and protein metabolism, but they also have anti-inflammatory properties. They also play a role in bone physiology, although their role has not been

clearly defined. The long-term side effects of these therapies lie in alterations in hard tissue mineralization and healing, in addition to alterations in chondrogenesis and osteogenesis, bone loss and osteoporosis. In acute administrations remodeling appears to be slower. Clinically, the results suggest that patients can be treated orthodontically with corticosteroid therapies, but certain considerations must be taken. In cases of patients undergoing corticosteroid therapy for a short time, it is advisable to postpone orthodontic mechanics until they stop receiving the drug. In cases of chronic corticosteroid therapies, as bone formation is limited, it is recommended to perform orthodontic mechanics with reduced forces and monitor the patient more frequently. A common drug associated with orthodontic patients and with an impact on oral health is bisphosphonates [15-17].

In patients who have a low risk, low intake of the drug or short administration time, tooth movements tend to be quite predictable, unlike patients with high risk. Therefore, altered bone metabolism and the greater extent of side effects are factors that must be taken into account in treatment planning, especially in cases of dental extractions or high-risk patients. Regardless of a longer duration of treatment, tooth movements must be slowed down and more side effects must be taken into account, such as incomplete closure of spaces as well as poor root parallelism, especially in cases of dental extractions and closures. of spaces.

## Risks in embryonic development

There are teratogenic risks such as chemical agents or substances that can pose risks to embryonic development during pregnancy. These patients do not exceed 1% of children who require orthodontic treatment.

## Risks in skeletal growth

Intrauterine molding. The development of the maxilla and/or mandible can be altered if the prenatal pressure that the head suffers during growth suffers distortions in the areas of rapid growth. The risks of this growth not being compensated for after childbirth are greater when the original problem disappears after birth. If the facial pressure that caused the developmental problem is eliminated after birth, the risk of pathological growth is very low and a complete recovery can be expected. On the other hand, uterine fibroids cause asymmetry of the skull and face. - Mandibular trauma during childbirth. Using forceps during childbirth can damage one or both temporomandibular joints. Currently, it is known that the condylar cartilage is not essential for the adequate growth of the mandible, therefore, it is not easy to relate the lack of mandibular development with injuries produced at birth, with congenital origin being the most probable cause of condylar cartilage. mandibular development.

## Childhood mandibular fractures

Falls and blows during childhood can fracture the jaw, with the condylar neck being the most at risk, but regeneration is quite good afterwards. The prognosis is more favorable the earlier the fracture, possibly due to the potential for greater growth early in life. The vast majority of fractures of the condylar process go unnoticed, and the best treatment is conservative.

## Muscle dysfunctions

Facial muscles can influence jaw development in two ways. One way is the formation of bone in the muscle insertions that depends on their activity, and secondly, the growth of the muscles directs the jaw forward and down. Muscles can be lost due to injury to the motor nerve, leading to inadequate development of that part of the face. Excessive muscle contraction can cause facial asymmetry as a result of growth restriction on the affected side. The reduction in tonic muscle function seen in muscle paralysis or weakness allows excessive mandibular displacement, triggering greater vertical growth and greater eruption of the posterior sectors, with the risk of presenting an anterior open bite. - Acromegaly, mandibular hypertrophy and hemimandibular elongation. The presence of an adenohypophyseal tumor in Acromegaly increases the secretion of growth hormone, generating growth of the jaw and development of a class III skeletal malocclusion. Hemimandibular hypertrophy and hemimandibular elongation sometimes appear in adolescents and/or adults in whom unilateral excessive growth of the jaw of unknown origin occurs. This growth can stop spontaneously, but if it does not, a resection of the affected condyle may be necessary [17,18].

## Risks in dental development

The main congenital defects can be accompanied by risks in dental development. Among the most significant are: - Congetic absence of teeth. It may be a total absence (anodontia), a large number of teeth (oligodontia) or a few (hypodontia). Cases of anodontia are very rare and are related to syndromes such as anhidrotic ectodermal dysplasia. Hypodontia is more common, with the most affected teeth being the distal ones of each group (lateral incisor, second premolar, third molar).

## Malformed and supernumerary teeth

Alterations in the size and shape of teeth are the result of anomalies produced during the morphological differentiation phase of

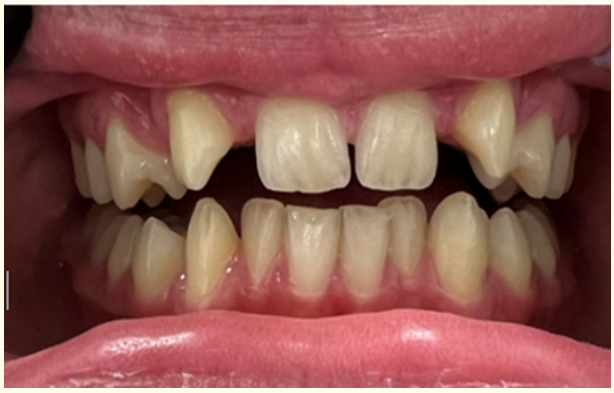

**Figure 10:** Patient with agenesis of upper lateral incisors.

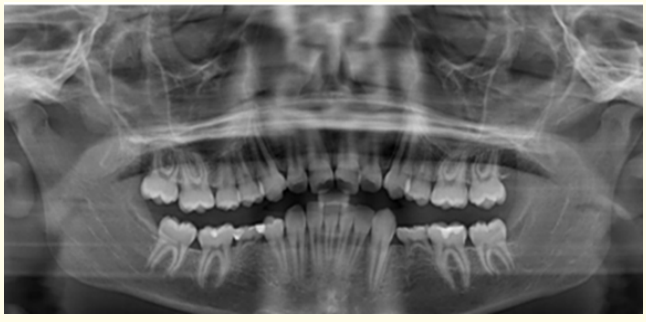

**Figure 11:** Orthopantomography showing the agenesis of teeth 4.5 and 3.5.

development. The most common alteration is the variation in size, specifically of the upper lateral incisors and the upper second premolars, which entails a risk in normal occlusion.

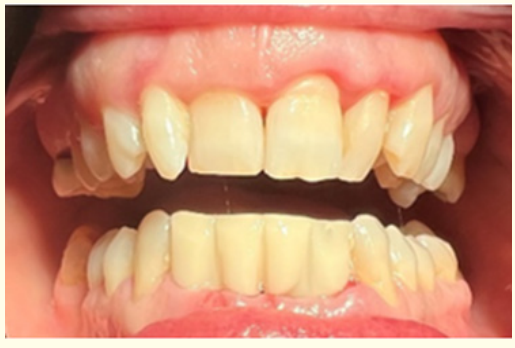

**Figure 12:** Conoid teeth in position 1.2 and 2.1.

5% of the population has a significant size discrepancy between the upper and lower teeth. Tooth germs can also fuse or germinate during their development. It is almost impossible to achieve a normal occlusion when there is the presence of fused or germinated teeth, or with any other malformation. Supernumerary teeth are also due to alterations during the initiation and proliferation phases of dental development. The most common area is the midline of the upper jaw, called mesiodens.

The presence of supernumeraries presents risks for normal occlusal development and their early detection is essential to avoid incorrect occlusal relationships. It is worth highlighting the congenital syndrome of cleidocranial dysplasia, which is characterized by the absence of clavicles and lack of eruption of permanent teeth due to the presence of extra teeth. - Interferences in the eruption. Tooth eruption may be affected by supernumerary teeth, sclerotic bone, and intense gingival fibrosis.

These interferences can be observed in cleidocranial dysplasia. It is indicated to clear the eruption path of the permanent teeth so that they can erupt. Therefore, it will be necessary to extract the supernumerary teeth, osteotomy of the bone that covers the permanent teeth and retract the fibrous gum so that they can erupt.

### Ectopic eruption

This is the malposition of a tooth germ, with the risk of causing it to erupt in the wrong area. In the case of a more mesial eruption of the upper first molars, the distal root of the temporalis will be resorbed and the arch length will be reduced. The teeth that encounter the most difficulty erupting are the upper canines that remain retained or erupt buccally and the lower second premolars that remain impacted or erupt lingually [18-20].

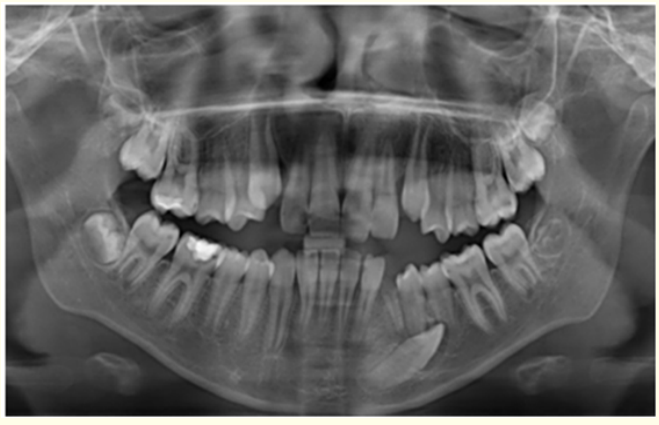

**Figure 13:** Ectopic position of 3.3.

The upper central incisors are also affected with some frequency.

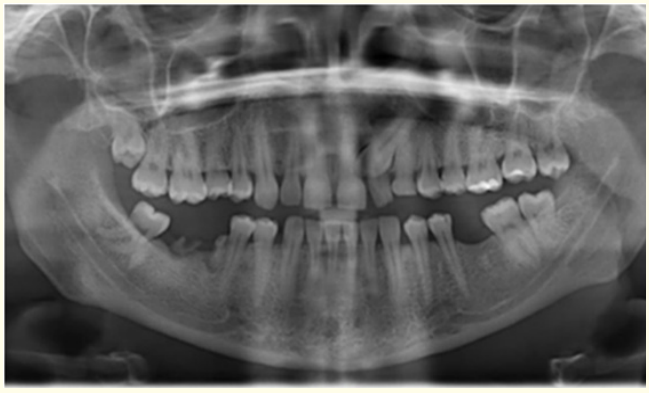

**Figure 14:** Ectopic position of 2.3 that has not been able to erupt in a 35-year-old patient.

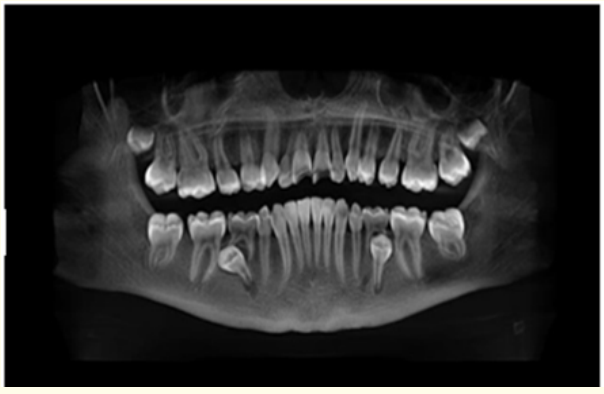

**Figure 15:** Ectopic position of 4.5.

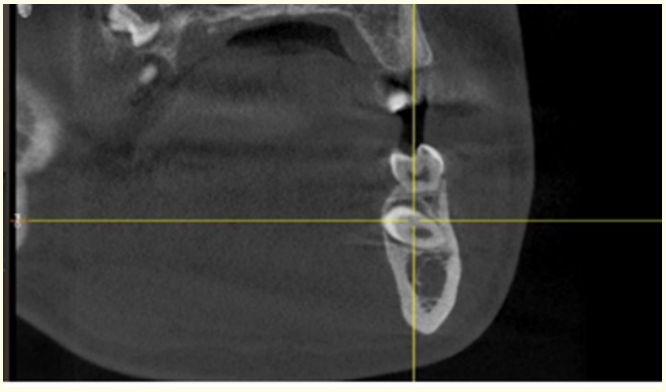

**Figure 16:** CBCT section of the previous image showing the lingual eruption of 4.5.

Early loss of temporary teeth. If a primary canine or first deciduous molar is lost, the movement that frequently occurs is distal drift of the incisors. If the loss is unilateral, an asymmetric movement will occur, with risks of causing crowding and occlusion problems.

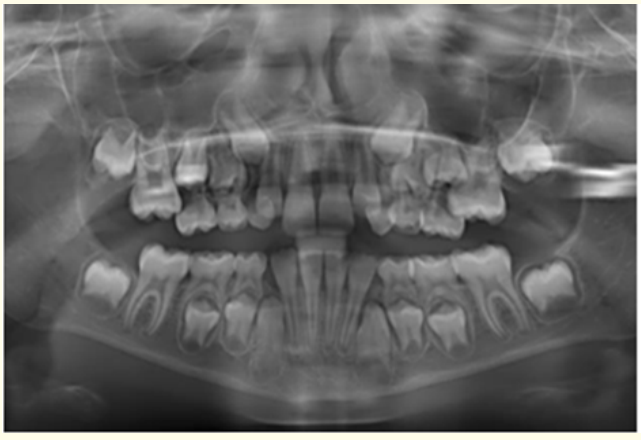

**Figure 17:** Orthopantomography showing the mesial eruption of the upper first molars that have caused the early loss of the upper second temporary molars.

### Dental trauma. Dental trauma can lead to malocclusion problems

Trauma to a temporary tooth can displace the germ of a permanent tooth. The enamel may also be affected if the permanent crown is forming. Distortion of the angle between the crown and the root once the crown has already formed. A direct injury to the permanent tooth can also occur, causing damage to the alveolar process [20,21].

In situations in which an intrusion or reimplantation of an avulsed tooth has occurred, there is a greater risk that the tooth will subsequently present with ankylosis, making orthodontic treatment more complex.

### Ankylosis

Caused by some type of injury that, due to the rupture of the periodontal membrane, determines the formation of a bone bridge, joining the cement to the alveolar dura lamina, delaying or preventing the tooth from erupting. The primary tooth does not erupt and sinks into bone tissue that continues to grow. It carries the risk of adjacent permanent teeth acquiring incorrect positions in the arch, with the opposing tooth being extruded and the subsequent tooth failing to erupt or erupting incorrectly.

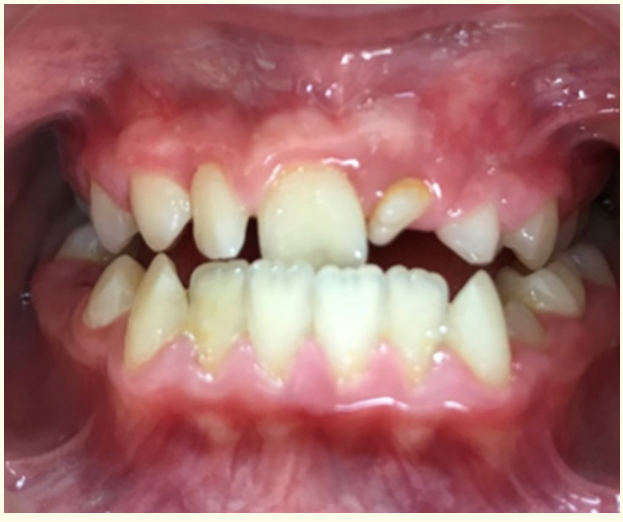

**Figure 18:** Malocclusion problems at the anterior level due to loss of tooth 2.1 due to trauma and eruption of 2.2 in position 2.1 poorly positioned.

## Genetics

Currently, it is considered that in most cases malocclusions are the result of a bone-dental discrepancy and a disharmony in the development of the maxillary bone bases. Even so, it is a mistake to determine that there is a single origin of the malocclusive problem, since it is a multifactorial etiopathogenesis [21-23].

The prominent factors involved in the etiopathogenesis, for example, of Class III malocclusion are genetic, congenital and acquired factors, which act at three levels, general, proximal and local. Currently, there are advances in genetic research that are allowing the analysis of complex aspects in human biology: growth, development and response to external stimuli, such as orthodontic treatment. According to various reviews, genetics is one of the main factors that directly affects both the shape and arrangement of the teeth.

Various lines of genetic research are being developed in the field of orthodontics in order to deepen the knowledge of different pathologies and be able to establish a clinical application through preventive or therapeutic measures. Malocclusion is a manifestation of the interaction of genetic as well as environmental factors during the development of the orofacial region. In the orthodontic field, genetics is of great interest to understand why a patient presents a specific occlusion and the risk it represents during orthodontic treatment.

Consideration of a patient's genetic factors is an essential element for diagnosis, which theoretically underlies all dentofacial anomalies [3,4,24,25].

## Risk factors inherent to orthodontic treatment

As orthodontics acts practically on the entire stomatognathic system, each of its components presents, to a greater or lesser extent, a risk of suffering damage. Depending on the structures that may be negatively affected, the risks can be classified as follows:

Periodontal risks:

- **Inflammatory:** Gingivitis induced by bacterial plaque
- **Allergy:** - Nickel - Acrylic
- **Dystrophic:** - Periodontal recession - Alveolar dehiscence - Fenestration - Loss of bone height - Thinning of the attached gum - Open gingival embrasures
- Proliferative - Plaque-induced gingival hyperplasia
- Periodontal pain

## Dental risks

- Orthodontic induced root resorption (OIRR)
- Cavities, enamel decalcification
- Tooth devitalization
- Damage to the enamel when removing the fixed technique
- Pulp pain
- Pain when removing the fixed technique Joint risks:
- Joint pain
- Dysfunction Risks inherent to the appliance:
- Loss of anchorage
- Unwanted or unexpected movements Skeletal risks:
- On palatal expansions

The isolated description of the risks associated with orthodontic treatment and its predisposing factors is a complex task due to the close link that exists between them and the need to carry out a holistic analysis of each patient. Furthermore, a factor may be associated with one or several risks. In turn, several factors can affect the same risk.

## Periodontal risks

All chronic periodontal disease is a progressive, generally painless process of which the patient is often unaware. Its main etiological agent is dental plaque. The elements that contribute to increasing retention should be considered risk factors that should not be undervalued. Cemented elements such as brackets, bands, ligatures

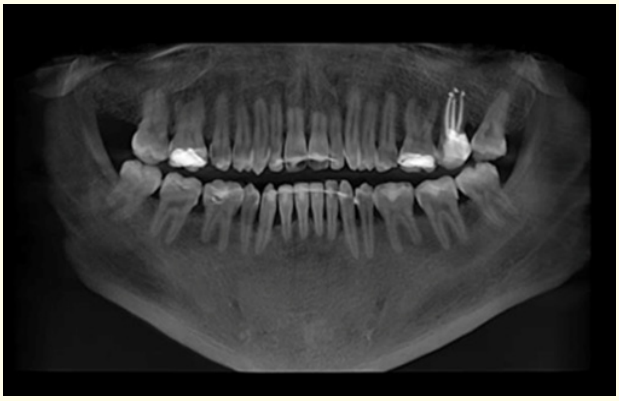

**Figure 19:** RRIIO after orthodontic treatment.

and arches have the potential to make tooth brushing difficult and reduce the physiological self-cleaning mechanism exerted by the tongue, lips and cheeks on the tooth surface. Regarding mouth breathing, it is necessary to determine its origin [24-26].

When there are pathological obstructions of the airways, plaque retention during the use of a fixed orthodontic appliance is an important etiological factor in the development of demineralization and chronic hyperplastic gingivitis. Bogren., *et al*. suggested that the accumulation of microbial plaque on teeth is a direct cause of gingivitis and periodontitis. The correction of malocclusions is beneficial for periodontal health. Orthodontic treatment is associated with a lower prevalence of periodontitis in the future. Adult patients who were treated by orthodontics show a lower prevalence of periodontitis compared to those patients who did not receive it [3,4,26].

There is an association between putative periodontal pathogens of subgingival dentobacterial plaque and early onset periodontitis (PIT). It has been found that the presence and microbial levels of Porphyromonas gingivalis (PG), Prevotella intermedia (PI), Fusobacterium nucleatum (FN), Campylobacter rectus (CR) and Treponema denticola (TD) are significantly higher in sick patients compared to healthy ones. PG, TD, and PI species are most associated with generalized and/or rapidly progressive PIT. Generally, orthodontic treatment does not increase the risk of high levels of periodontal pathogens. Although after 6 months of treatment with fixed appliances, the counts of Tannerella forsythia (TF), Eikenella corrodens (EC), PI, FN, TD and CR increase significantly, they decrease to the pre-treatment level after 12 months of therapy [3,4,27].

There is a relationship between malocclusion and the presence of periodontal disease. Dental plaque causes periodontal disease and its removal is more difficult when teeth are poorly aligned. The presence of gingivitis is usually greater in patients with malocclusion, which reinforces the link between dental crowding and plaque accumulation. In most malocclusions there are different degrees of gingival recession with mild to moderate periodontal pockets. However, the association between the severity of malocclusion and the presence of periodontal disease is contradictory. While some studies affirm this approach, others find no relationship. Additional studies should be performed to achieve a consolidated consensus on the association between malocclusion and periodontal disease. The severity of the poor positioning of the teeth can influence the intensity of periodontal disease, occlusal trauma as a consequence of the poor position of the teeth has deleterious effects on the supporting periodontium [28].

The type and severity of the malocclusion with which the patient comes to the consultation is one of the most important risk factors to take into account when deciding on any type of orthodontic treatment. Its complexity can determine the presence of periodontal disease, dental and joint disorders. From an etiological point of view, periodontal problems will be associated with the accumulation of dental plaque, occlusal trauma and the need to perform orthodontic movements. Bacteria are the main cause of inflammatory periodontal disease. The health of periodontal tissues depends largely on the balance between bacterial action and the response established by the host. The bacterial action will be determined by the nature of the oral microbiota, its virulence and whether it is predominantly protective or pathogenic. The bacterial metabolism of plaque produces irritating agents that act on tissues directly or indirectly by stimulating immune and inflammatory reactions. The first line of defense of the host against the oral microbiota is the barrier effect of the periodontal tissues. In the gingival sulcus, the junctional epithelium has a relative permeability that allows bidirectional movement of various substances. Gingival fluid, immune system cells, immunoglobulins and complement are poured from the chorion into the sulcus. With inflammation, its permeability increases, so the movement of fluid and cells is increased.

In a perfect state of health, microorganisms cannot pass through it, but it is possible for foreign materials to penetrate from the sulcus to the chorion and subsequently reach the bloodstream. A large number of high molecular weight substances have the ability to pass through their intercellular spaces. The genesis of gingival inflammation may be due to the diffusion of bacterial metabolites and enzymes from the sulcus to the chorion through the junctional epithelium [29,30].

Something similar happens with the cervicular epithelium. Being healthy it is impermeable to bacteria; However, it is not immune to the action of bacterial antigens, metabolites and enzymes that have the potential to weaken it and achieve its destruction. Both the junctional and cervical epithelium react to bacterial aggression by releasing signaling molecules that trigger an early host inflammatory response with vascular changes and corresponding leukocyte activation. Bacterial periodontal damage is produced by a progressive and simultaneous effect of events. Firstly, bacteria manage to colonize the gingival sulcus by evading the host's defense. They then damage the cervical epithelial barrier and produce harmful substances that damage the tissue directly or indirectly: Proteolytic enzymes: They degrade tissues and proteins such as collagen and proteoglycans, which are the main structural proteins of the gingival connective tissue and the periodontal ligament. They affect the basement membrane and extracellular matrix proteins. In this way, the periodontal connective tissue is destroyed, which facilitates bacterial invasion [31,32].

They also interfere with repair by inhibiting clot formation or lysing the fibrin matrix in periodontal lesions. They have the potential to activate host tissue collagenases, which increases tissue destruction and inactivates proteins with an important function in defense. Hydrolytic enzymes: They degrade non-protein elements of the periodontal connective tissue, which contributes to increasing the permeability of the epithelium and causing its ulceration. Bacterial metabolites and toxic factors: Ammonia, toxic amines, indole, organic acids, hydrogen sulfide, methyl mercaptan, and dimethyl disulfide can damage tissues or stimulate inflammation. Polysaccharides, lipoteichoic acid and peptidoglycans are found in the cell walls of gram-negative bacteria, which are released upon death and stimulate bone resorption. Lipopolysaccharides of gram-negative bacteria: They are found as part of their cell wall and are released when they die or lyse. They are key inflammatory mediators that stimulate bone resorption and are potent antigens that can alert the host to the existence of a bacterial infection. Bacterial antigens: They stimulate the immune system and cause different immune and hypersensitivity reactions that can contribute to the protection of the host, but also to damage to its tissues. The main periodontal damage is caused by the action of these substances on the tissues and immune system of the host. In most cases, there is no direct bacterial invasion, however, soft tissue infection is usually rare due to the ability of the inflammatory and immune systems to destroy bacteria in the tissues [32,33].

## Dental risks

Root resorption in the primary dentition is a physiological process that derives from the forces generated by the eruption of permanent teeth. On the other hand, root resorption in permanent teeth is never physiological. There are two types of root resorption depending on their location: Internal (RRI) and external (RRE). IRR is due to a chronic inflammatory process in the pulp tissue whose origin is related to cavities, trauma, tooth whitening procedures, root canals or reimplanted teeth. RRE is related to acute or chronic mechanical trauma, with dental impactions, frequently due to eruptive anomalies or inflammatory processes of pulpal and periodontal origin, but also in most cases they are of idiopathic origin. Among the RRE, it is worth highlighting those that originate during orthodontic treatment. Orthodontics may be the only dental specialty that is based on the inflammatory process as a vehicle to solve both aesthetic and functional problems.

The RRE that accompanies orthodontic treatment is anatomopathologically different from those that occur idiopathically, which is why they are called orthodontic-induced inflammatory root resorption (ORIOR). RRIIO is an adverse clinical phenomenon that can clinically debut with excessive mobility and/or pain on percussion. Its definitive diagnosis is made with periapical and/or panoramic radiographs. The extent and severity of the root condition can condition the future function and stability of the tooth, making its prevention very difficult due to its multifactorial origin and, in many cases, its idiopathic etiology. For years, RRIIO has been related to the use of fixed and removable appliances during orthodontic treatment. The determining factors of IRRIO are the impulse and the critical periodontal barrier as primary determinants and bone density as a secondary determinant. In an incipient stage of movement, resorption depends fundamentally on an external factor, strength, but as the treatment is prolonged it will depend on intrinsic factors and time. The critical barrier is the narrowing of the periodontal space that transforms the osteoclastic action into osteo-odontoclastic action [30-33].

IRRIO comes from the damage suffered by the periodontal ligament due to the compression that is inflicted on its structure while there is tooth movement, which determines the impairment of capillary circulation. Vascular injury gives rise to a phenomenon of aseptic coagulative necrosis better known as hyalinization. There is a certain individual tendency, even of a family nature, to develop RRIIO. Of all the factors mentioned, the one that can best be controlled currently to prevent root resorption is the biomechanical

one and as J.A. says. Canut "prudently carried out treatment, with light forces and three-dimensional control of the root, very rarely causes medium-sized root resorptions. Prolonged treatments with continuous forces and the application of intense forces or extensive torque and intrusion movements can lead to severe root injuries. RRIIO is a type of superficial resorption or transient inflammatory resorption. Reconstructive resorption exceptionally appears with orthodontic therapy. The considerations to which special attention must be paid to try to reduce the appearance of IRRIO and its risks, both clinical and legal before, during and after treatment are the following.

## Before treatment

All patients must be informed before orthodontic treatment of the risk of suffering IRRIO as a consequence of it, and sign the informed consent. - Take a good medical and dental history. It is known that IRRIO is more frequently associated with various pathologies such as asthma, diabetes, hypothyroidism or allergies. The consumption of corticosteroids is also related to the appearance of IRRIO.

- If the patient has different general or local risk factors for IRRIO, a treatment plan should be designed using light forces, reducing duration and limiting the objective.
- Patients who present onychophagia or thumb sucking over 7 years of age, parafunctions or bad habits such as tongue or lip interposition will be considered high-risk patients.
- It is advisable to carry out the treatment as soon as possible, because teeth in formation are more resistant to suffering from IRRIO [30-33].

Adults have less capacity to adapt to occlusal modifications than children or adolescents. During treatment

- Try to use superelastic and low-friction materials.
- Space out activations.
- Pay attention to intrusive movements, with or without radiculolingual torsion.

Large displacements for the correction of severe interincisal overjets in cases that require extraction or not.

- The anterior vertical elastics for closing the bite, as well as the class II ones. - Carry out radiographic controls 6 months after the start of treatment in order to check if morphological changes have begun in the apices. To reduce the risk of progression, it is recommended to stop treatment for 2 or 3 months and perform quarterly radiographic checks [30-33].

## After treatment

The most frequent evolution after the removal of the appliances is that the lesions begin a process of recurrence. On the contrary, if there has been extensive root resorption with an unfavorable crown-radicular proportion and with the affected teeth subject to atypical and pathological movements during chewing, the resorption process may continue. Pathological mobility occurs especially in the anterior teeth and in these cases it is recommended to stabilize the teeth with a disocclusion splint. If the resorption process is not stopped, it is indicated to perform root canal treatment on the affected teeth [30-33].

If RRIIO is observed in the final radiographs, the patient should always be informed.

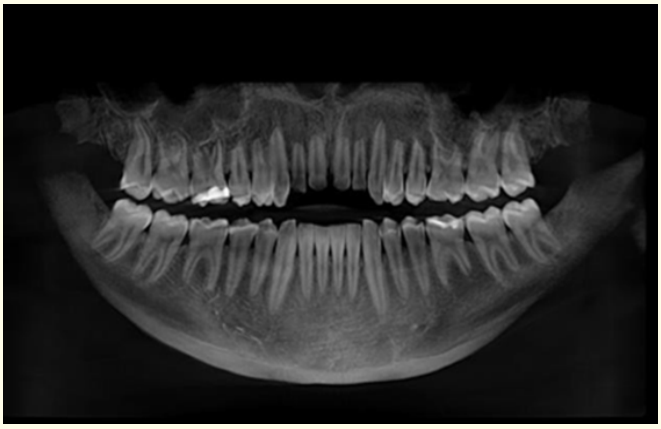

**Figure 20:** Internal and external resorption of the tooth 1.6.

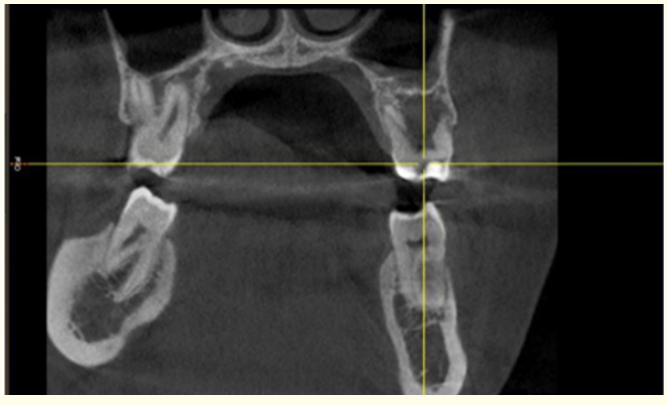

**Figure 21:** CBCT section of tooth 1.6 in the previous image.

## Joint risks

The existence of a functional or structural temporomandibular disorder in a patient with malocclusion does not contraindicate orthodontic treatment, regardless of the patient's age. However,

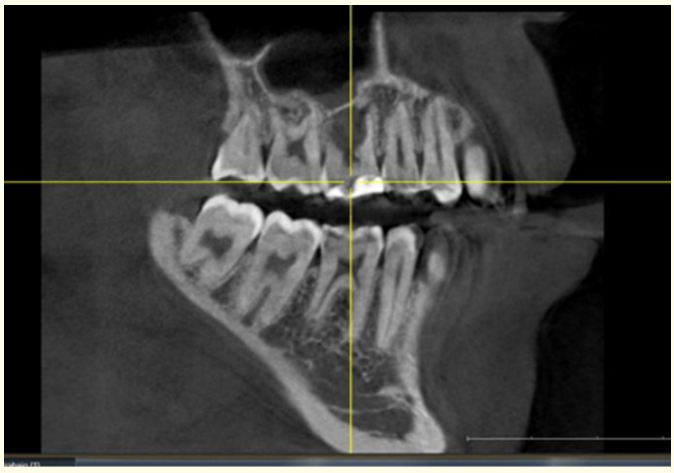

**Figure 22:** CBCT section of tooth 1.6 in the previous image.

the comprehensive treatment of these patients may include the correction of the malocclusion with the aim of achieving greater orthopedic joint stability, eliminating possible factors that perpetuate the disorder. These treatments must be multidisciplinary between the TMJ specialist and the orthodontist.

The orthodontist must keep in mind that muscular alterations and the condyle-disc complex are frequently accompanied by alterations in the position and function of the jaw, so it is contraindicated to begin orthodontic treatment if the patient presents pain. It is worth highlighting in this chapter idiopathic condylar resorption (ICR), which is also known as condylar atrophy, idiopathic condylosis, progressive condylar resorption or "cheerleader" syndrome. A series of local and systemic factors and diseases have been described that can increase the risk of causing resorption of the condyle of the mandible. Local factors include IUGR, osteoarthritis, reactive arthritis, avascular necrosis, infection, and trauma. Systemic autoimmune and connective tissue diseases that favor condylar resorption include: rheumatoid arthritis, psoriatic arthritis, scleroderma, systemic lupus erythematosus, Sjögren's syndrome, ankylosing spondylitis, among others. RCI can cause occlusal and skeletal instability, dentofacial deformities, temporomandibular joint dysfunction, and pain. It has also been linked to orthodontic treatment and orthognathic surgery [30-33].

The people at greatest risk of suffering from IUGR are women, between 10 and 40 years old, with predominance in the pubertal growth phase, high angles of the occlusal plane and mandibular plane and with a skeletal and occlusal class II relationship with or without open bite. This pathology is also associated with the consumption of oral contraceptives. The diagnosis is based on the patient's symptoms and complementary imaging tests. Patients may present with progressive worsening of their occlusion and aesthetics with or without TMJ symptoms and associated pain. In bilateral cases they usually debut with a posterior displacement of the mandible and the development of an anterior open bite is observed. And in unilateral cases, the mandibular dental midline and chin are displaced towards the affected side with class II occlusion, crossbite and presence of posterior occlusal prematurities on that side. It should be noted that TMJ symptoms may be present, although they are often very mild or non-existent. Clicks and/or crepitations are absent because hyperplastic synovial tissues increase joint space [30-33].

Skeletal risks. There are also risks or adverse effects of orthopedic therapy such as maxillary expansion using any of the Hass, Hyrax or McNamara type expanders. Regarding the unwanted effects on skeletal patterns, the literature indicates that palatal expansions can produce a downward displacement of the maxilla and an extrusion of the supporting teeth, causing a downward and backward mandibular rotation. This rotation in turn produces cephalometric changes, such as increases in the inclination of the mandibular plane, in lower and anterior facial height, in facial convexity and also causes an anterior open bite in the growing patient. In addition to this, the overcorrection of two to three millimeters of the perimeter of the maxillary arch causes occlusal interferences, occluding palatal cusps of the upper teeth with the buccal cusps of the lower teeth contributing to an increase in the vertical dimension. This is why some orthodontists have advised against using maxillary expansion in patients with vertical growth patterns and convex facial profiles to avoid greater severity of malocclusion. It should also be noted that during orthopedic treatments using maxillary expanders, patients are unable to maintain proper hygiene and effective control of bacterial plaque, which can generate moderate to severe gingival inflammation. In the study carried out by Rosa., *et al*., with Hass-type maxillary expanders, 50% of the patients developed a transient bacteremia after removal of the device that persisted until the fifth day. The bacteria corresponded to Gram + bacteria where Streptococcos Oralis was isolated [32,33].

At the same time, in the study by Guerel., *et al*., various blood samples were taken before and after removal of the circuit breaker, and bacterial growth was observed in 32% of the samples, and Streptococci Viridans could be isolated in 16% of them. There are few bacterial species that are capable of generating distant infections once they enter the bloodstream, one of these is S. Viridans,

which is capable of causing bacterial endocarditis. Therefore, in patients with moderate to severe risk of developing bacterial endocarditis, antibiotic prophylaxis should be considered, in addition to complementing the use of oral antiseptic rinses such as chlorhexidine at the time prior to removal of the expander to reduce the risk of developing it. In addition to the changes that occur in the maxillary suture, some studies have presented a direct association between maxillary expansion and mandibular changes, such as increased mandibular width and rotation. Due to this mandibular movement, it is suggested that the condyles present a spatial change. Many investigations have studied the movement of the condyles during maxillary expansion. This effect has not been specified if it represents a positive or negative effect. A hypothesis about the relationship between maxillary expansion and condylar movement determines that it generates more tipping and dental dilation than skeletal expansion, therefore causing the mandible to rotate and have premature posterior contact. Thus the mandible is positioned more posteriorly and can cause extra pressure from the condyle towards the glenoid fossa [30-33].

Another associated risk is accidental swallowing of the instruments used for activation, which may be just a wire or a key made up of a handle plus the wire. When this occurs, the instrument can pass through the gastrointestinal tract without incident or complications until it is eliminated naturally; or also cause serious complications, such as: obstruction of the airways or gastrointestinal tract, organ perforation, internal bleeding, sepsis or even death. The indication is that the situation must be monitored, if the patient does not report symptoms or suffers from some type of intestinal disease, monitoring is recommended and the removal of the object will be eliminated without difficulties. On the other hand, if the object has an active tip and measures more than 5cm, the risk of damaging a structure increases and must be controlled by x-rays until the removal of the artifact is observed. If the object does not move normally in the intestine, it is recommended to remove it surgically [30-33].

## Conclusion

Orthodontics is a specialty in which different dentomaxillofacial anomalies are treated, where each treatment can present multiple risks and complications that will affect the quality of life of the patients and their expectations would be affected.

## Acknowledgements

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