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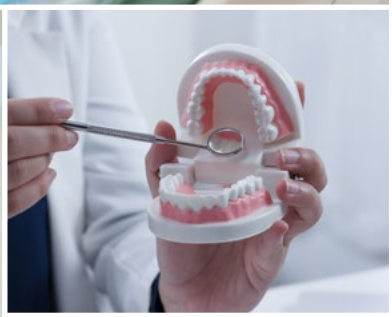

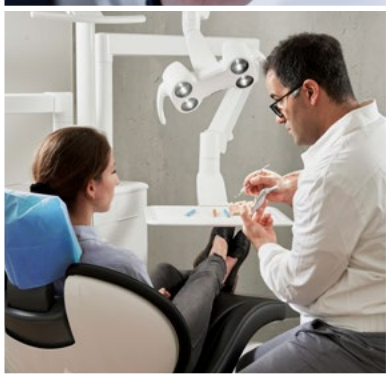

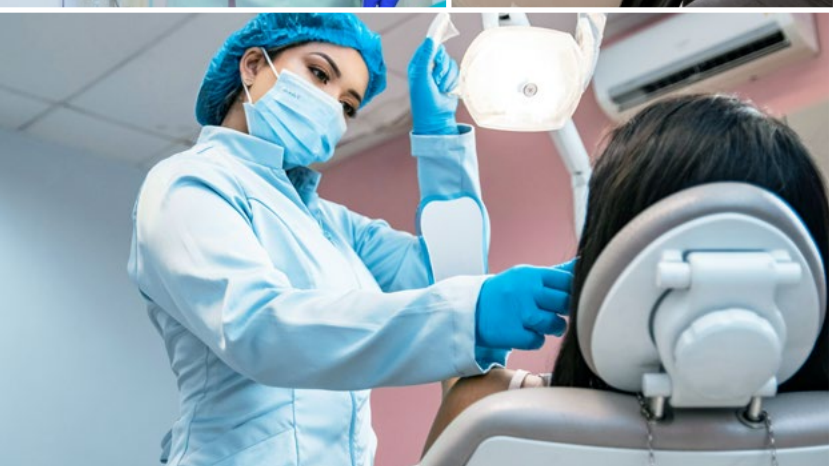

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Ilustre Colegio Oficial de Odontólogos y Estomatólogos de la Iª Región

SPECIAL SUPPLEMENT 2023
volume 20

contents

editorial

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Científica Dental

Dear colleagues and readers of the journal *Científica Dental* (Dental Science in English),

This special English-language supplement of *Científica Dental* features the best papers published in 2023 in the categories of best scientific article, best clinical case and best first-time author publication. A total of six papers are presented, which are the finalists of the previous categories.

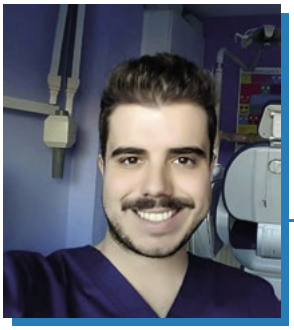
The subject matter of the papers is, as we always try to do at *Científica Dental*, current, varied and of interest to dental professionals. Our readers can access this issue free of charge at www.cientificadental.es.

The first prize in the category of original article goes to the study carried out by *González Fernández-Tresguerres, et al.,* which shows a broad and detailed analysis of the management of antiplatelet agents in oral surgery. In this same category, *Anitua* is the second prize-winner, with a study on the use of extra-short implants in crestal elevation with autologous bone and PRGF-Endoret in residual bone heights of 2-3mm.

As best clinical case, *Garrido Martínez, et al.* are the first winners with an excellent clinical case on the maxillary reconstruction of a patient with subperiosteal implants. *Anitua* repeats the award with a clinical case where they successfully show the explantation of a poorly positioned implant in the aesthetic sector and its subsequent regeneration.

In the best first author publication category, the work of *Galán Valero, et al.,* presents a clinical case on the surgical and orthodontic management of an autotransplantation of a maxillary impacted canine, and *Escobar Bores, et al.* analyses the influence of unilateral non-alternating chewing on maxillofacial development and early treatment in their review of the literature.

We would like to thank, as always, our authors for the high quality of the papers they present and for their trust in us to publish them in *Científica Dental*, as well as the editors and reviewers, whose work is essential for the production of each issue of this journal. We also thank our readers, of course, to whom this issue, which includes the most relevant papers published during the year 2023, is especially addressed.



Update

Antiplatelet therapy and oral surgery: to discontinue or not, that is the question

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SUMMARY

Cardiovascular disease is one of the most prevalent systemic pathologies worldwide; those patients usually have had an acute coronary event which is treated with antiplatelet therapy. These drugs represent a challenge for the dentist, who must face a major dilemma: either maintain the drug, with the consequent bleeding risk, or withdraw it, with the possibility of thromboembolic complications, entailing a risk to the patient's life. Therefore, dentists should know how to manage patients treated with these drugs when performing a surgical procedure or even a simple tooth extraction.

The objectives of this narrative review are, firstly, to recall platelet physiology and the mechanisms of platelet thrombus formation; secondly, to go more deeply into the mechanisms of action of the different antiplatelet drugs; and thirdly, since there are no clinical guidelines on this topic, to critically review the existing guidelines related to the dental management, in order to prevent the appearance of possible complications, not only local, but more importantly, systemic complications. In these cases, before interrupting antiplatelet therapy, the risk of bleeding should be evaluated against the risk of generating a new thromboembolic episode, such as stent thrombosis or recurrence of the acute coronary accident, events that could put the patient's life at risk.

KEY WORDS

Platelet antiaggregant; Platelets; Platelet thrombus; Acetylsalicylic acid; Clopidogrel.

INTRODUCTION

Cardiovascular disease is the systemic disorder that generates greater morbidity and mortality in the western world. In Spain, according to data from the National Institute of Statistics, in 2020, cardiovascular disease was the leading cause of death among the general population, above tumours and COVID-19. In this sense, ischemic heart disease is the leading cause of death in men and stroke in women.

Among cardiovascular diseases, atherosclerotic pathology is the main cause of morbimortality in our country, including acute coronary syndromes, cerebrovascular diseases and peripheral arterial disease[1]. The etiopathogenic mechanism underlying all these pathologies is the rupture of the atheroma plaque, which triggers the platelet aggregation which is the cause of the acute thrombosis process. For this reason, in recent years, new antiplatelet drugs have been developed, which constitute the cornerstone of the recurrence prevention of acute ischemic episodes, both short-term and long-term. These drugs have to be known by the odontologist, especially when performing a surgical procedure, to know what should be the correct dental management to avoid potential complications, not only from the point of view of local bleeding, but also, most importantly, to avoid systemic complications (such as stent thrombosis or the appearance of a new thromboembolic event), which could put the patient's life at risk[2].

The purpose of this narrative review is, firstly, to recall the platelet physiology and the platelet thrombus formation mechanisms; secondly, to delve into the mechanisms of action of the different platelet antiaggregants; and, thirdly, since there are no clinical guidelines in this regard, make a critical approach to the existing guidelines for their correct dental management, in order to perform a surgical procedure in the oral cavity or even a simple tooth extraction, with sufficient guarantees of success.

Physiology of the platelet

The platelet is one of the blood forming elements, along with red and white blood cells. Normally, there are between 150,000 and 400,000 platelets per mcL in the blood, and the average platelet volume is usually 7-9 cubic micrometers. Platelets come from the hematopoietic stem cells of the bone marrow, specifically from the myeloid lineage and have an average life of 7 to 10 days. Inside they have alpha granules and dense granules, where molecules of special relevance accumulate in platelet physiology[3].

Platelets play a primary role in haemostasis, since they initiate the repair of vascular lesions, forming the platelet plug, and also promote blood clotting, through the activation of thrombin released from the platelets themselves and the calcium released from the dense granules which are necessary for the formation of fibrin[3].

Platelet physiology involves several enzymes, such as cyclooxygenase (COX), which transforms arachidonic acid (AA) from membrane phospholipids, in prostaglandins (PG), which are the thromboxane A2 (TXA2), vasoconstrictor and platelet proaggregant and prostacyclin (PGI2), which is a vasodilator and antiaggregant and originates in the vascular endothelium[3]. Other platelet enzymes include: phospholipase A2, which releases AA from membrane phospholipids and the phosphodiesterase, which hydrolyzes the cAMP.

The platelets have receptors in their membrane which are glycoproteins (GP) and are inactive under normal conditions. The most relevant are GP Ia and GP VI that bind to collagen, GPIb that binds to the von Willebrand factor (VWF) and GP IIb/IIIa, which binds to several proteins, but the most important is fibrinogen. These receptors are involved in platelet adhesion phenomena (platelet binding to the injured vessel), activation (change of platelet morphology causing the secretion of granules) and aggregation (binding between several platelets)[3].

Platelet thrombus formation mechanism

Platelets circulate in the bloodstream inactively. But when a vessel injury occurs, subendothelial collagen is exposed, which is the stimulus to recruit the platelets that will form the platelet plug. Remember that platelets do not adhere to the intact endothelium, but they can

adhere to a foreign body inside the bloodstream (such as a coronary stent or a prosthetic heart valve)[3].

Adhesion: When a vessel is injured, the circulating platelets slow down their speed over the damaged area, against the blood flow that pushes them, thanks to the platelet GPIb binding to the von Willebrand factor (VWF) of the matrix under the endothelium. Then, the subendothelial collagen establishes a more stable binding by binding to platelets[3] GPIb and GPVI.

Activation: After adhesion, platelet activation occurs, appearing on the exterior of the receptors that were inactive. These activate intracellular molecules, which cause a change in platelet morphology, with the emission of pseudopods and release of certain substances that promote platelet aggregation, perpetuating the process. These molecules, known as platelet agonists are: TXA_2, ADP and thrombin. Of all of them, ADP is the most potent for recruiting platelets and spreading arterial thrombus, for which it is considered a platelet activation amplifier. Platelets have on their surface three receptors for ADP: $P2Y_1$, $P2Y_{12}$ and P2X. Each induces different platelet signalling pathways, but $P2Y_{12}$ is the most important since it favours the release of the content of the granules, the increase of intracellular calcium, the generation of TXA_2 and the activation of the GPIIb-IIIa receptor, which is key in platelet aggregation. Consequently, the platelet $P2Y_{12}$ receptor blockage is crucial to inhibit platelet activation and aggregation and thus prevent platelet thrombus formation. Therefore, in recent years new drugs have been developed capable of blocking this receptor[1].

Release: After activation, the molecules stored in the granules of the platelets are released. Activated platelets can release up to 300 different proteins. From alpha granules, proteins homologous to plasma (fibrinogen, fibronectin, factor XIII, VWF) and platelet-specific proteins (platelet factor 4-FP4), thromboglobulin, P-selectin, PDGF (platelet-derived growth factor) and thrombospondin) are released. From the dense granules ADP, ATP, calcium and serotonin (5-hydroxytryptamine or 5-HT) is released[3].

Aggregation: Once the platelets are trapped in the damaged area, new platelets are recruited from the bloodstream, known as platelet aggregation. Activation of the GPIIb/IIIa receptor is the final pathway leading to platelet aggregation. Once activated, it binds to its ligands, which have the sequence of RGD amino acids (Arg-Gly-Asp or arginine-glycine-aspartic), such as fibrinogen, but also VWF, fibronectin and vitronectin. This receptor is specific to platelets and binds in a bivalent manner to fibrinogen, forming binding bridges between two platelets[3].

Regarding the mechanisms that regulate platelet aggregation, there are the contribution and inhibition factors.

Thus, they favour the platelet aggregation the ADP, the thrombin, the collagen, adrenaline and TXA_2. While cAMP, cGMP and PGI_2 inhibit it, as well as nitric oxide

(NO), which in addition to being a platelet antiaggregant, is considered the most potent vasodilator of the organism[3].

Platelet antiaggregants drugs (APAs)

Antiplatelet agents (APAs) are drugs that inhibit platelet aggregation, acting as antithrombotics. They usually act irreversibly and their function cannot be monitored, but it is usually considered that their effect lasts as long as the average life of the platelet, that is, between 7 and 10 days[1]. The main indications of these drugs include acute coronary syndromes (ACS) (which include acute myocardial infarction-AMI and unstable angina), stable coronary artery disease, cerebrovascular disease, and peripheral artery disease, but they are also used after surgical treatments that are performed after the appearance of these conditions, such as percutaneous coronary intervention (PCI) or revascularization surgery, and in the prevention of recurrence of the same, that is, in the secondary prophylaxis of atherosclerotic disease[1].

AAPs are drugs whose action mechanism is based on inhibiting platelet enzymes, such as COX (acetylsalicylic acid) or phosphodiesterase (dipyridamole), on the platelet $P2Y_{12}$ receptor (such as thienopyridines) or GPIIb/IIIa receptor (such as tirofiban) or in acting as analogues of molecules that inhibit platelet aggregation (such as iloprost) (Table 1).

Table 1. Characteristics of platelet antiaggregants.

Type	Drug	Commercial name	Diana	Indications	Effect on platelets	Management
Antienzimatic	ASA	Adiro® Tromalyt®	COX	2nd SCA prevention	Pequeño	Oral
	Dipyridamole	Persantin®	Phosphodiesterase	2nd SCA prevention	Moderado	Oral
Anti-receptors	Clopidogrel	Plavix® Iscover®	ADP $P2Y^{12}$ receptor	2nd SCA prevention	Moderado	Oral
	Prasugrel	Efient®	ADP $P2Y^{12}$ receptor	2nd SCA prevention	Grande	Oral
	Ticagrelor	Brilique®	ADP $P2Y^{12}$ receptor	2nd SCA prevention	Grande	Oral
	Tirofiban	Agrastat®	GPIIb/IIIa receptor	Thrombolytic in AMI	Grande	IV
PGI_2 analogues	Iloprost	Ilomedin®	PGI_2	Raynaud phenom.	Grande	IV

Acetylsalicylic acid (ASA); Adenosine diphosphate (ADP); Cyclooxygenase (COX); Acute myocardial infarction (AMI); Prostacyclin (PGI2); Acute coronary syndrome (ACS).

APAs can be classified according to their action mechanism in[4-7]:

1. Antienzimatic
   - 1.1. Cyclooxygenase (COX) inhibitors
     - 1.1.1. Acetylsalicylic acid (ASA) (Aspirin®, Adiro®, Tromalyt®)
     - 1.1.2. Triflusal (Disgren®)
   - 1.2. Phosphodiesterase inhibitors
     - 1.2.1. Dipyridamole (Persantin®)
     - 1.2.2. Cilostazol
     - 1.2.3. Pentoxifylline
2. Receptor inhibitors
   - 2.1. Of ADP ($P2Y_{12}$): Ticlopidine, Clopidogrel, Prasugrel, Ticagrelor
   - 2.2. From GPIIb/IIIa: Abciximab, Tirofiban, Eptifibatide
   - 2.3. Of thrombin (PAR1): Vorapaxar
3. Prostacyclin analogues
   - 3.1. Iloprost (Ilomedin®)

1. Antienzimatic

1.1 COX inhibitors

1.1.1 Acetylsalicylic acid (Adiro® and Tromalyt®)

Acetylsalicylic acid (ASA) is the antiplatelet par excellence. It is an irreversible inhibitor of the cyclooxygenase COX-1 and COX-2 of the platelet and, therefore, inhibits the synthesis of TXA_2 platelet and of the PGI_2 of the vascular endothelium, but especially of the first.

In addition, small doses appear to affect only TXA_2. Also, since platelets do not have a nucleus, they do not have the ability to resynthesize COX, unlike endothelial cells, for which the TXA_2 inhibition lasts for the average platelet life, that is, between 7 and 10 days.

Inhibition of TXA_2 only suppresses one of the aggregation mechanisms, but does not affect the aggregation induced by ADP.

However, another effect of ASA in the platelets is that it decreases the secretion of dense granules, that is, decreases the release of proaggregant substances during platelet activation. This would explain why its effects

on platelets are more than one would expect from simple platelet inhibition dependent on a relatively weak agonist such as TXA_2[1].

ASA is the basic antithrombotic therapy, used as an antiplatelet treatment only in the secondary prevention of atherosclerotic disease[8].

1.1.2. Triflusal (Disgren®)

Triflusal is an ASA analogue, which selectively inhibits the platelet COX, but does not affect endothelial cells. Triflusal has fewer side effects than ASA, so it is indicated in patients with ASA resistance and in geriatric patients.

1.2. Phosphodiesterase inhibitors

1.2.1. Dipyridamole (Persantin®)

Dipyridamole is a phosphodiesterase inhibitor, which increases intracellular cAMP levels, inhibiting aggregation; it is also a vasodilator. It does not have advantages over ASA, but it can be associated with anticoagulant drugs and given to patients with cardiac valve prostheses with ASA intolerance.

1.2.2. Cilostazol

It increases intracellular cAMP levels and is a vasodilator.

1.2.3. Pentoxifylline

Pentoxifylline is a vasodilator phosphodiesterase inhibitor, currently used in the prevention of jaw osteonecrosis.

2. <u>Platelet receptor inhibitors</u>

2.1. Inhibitors of ADP $P2Y_{12}$

2.1.1. Irreversible inhibitors: Thienopyridines

2.1.1.1. 1st Generation: Ticlopidine (Tiklid®)

Ticlopidine is a thienopyridine derivative, which behaves like a prodrug, that is, it is metabolised in the liver resulting in an active metabolite, which antagonizes ADP induced aggregation. It was the first inhibitor of the $P2Y_{12}$ receptor, but the relative frequency of adverse reactions, such as diarrhoea and, above all, neutropenia (in 0.8% of cases), has made its use increasingly low.

2.1.1.2. Of 2nd generation: Clopidogrel (Plavix®, Iscover®)

It is a prodrug, which requires two oxidation reactions in the liver to transform into the active metabolite, which inhibits the $P2Y_{12}$ receptor. However, a large individual variability has been described in the induced antiaggregation response by clopidogrel. It is usually used at a dose of 75 mg a day, being more powerful than 100 mg of ASA.

A coronary stent may be administered in conjunction with the ASA for the treatment of ACS after placing or after percutaneous revascularization surgery, which constitutes the so-called “dual antiplatelet therapy” or “dual platelet antiaggregation” (DPA).

2.1.1.3. Of 3rd generation: Prasugrel (Efient®)

Prasugrel is another prodrug that inhibits the $P2Y_{12}$ receptor, and is more potent, faster and has less variability in the antiplatelet response than clopidogrel. It is the only one that has benefits in diabetics.

2.1.2. Reversible inhibitors

2.1.2.1. Ticagrelor (Brilique®)

Ticagrelor is an antagonist of the $P2Y_{12}$ receptor with a reversible effect. It is faster and more powerful than clopidogrel. In addition, it has extraplatelet effects that are beneficial from a cardiovascular point of view.

2.1.2.2. Cangrelor

Recently, new antagonists even more potent antagonists like Cangrelor and Elinogrel have been designed. These new antiaggregants achieve greater antithrombotic efficacy, but also involve an increased risk of bleeding.

2.2. GPIIb/IIIa receptor inhibitor

2.2.1. Abciximab, Tirofiban, Eptifibatide

They are antiaggregants for hospital use administered by IV, blocking the binding of fibrinogen and VWF to the glycoproteins of the platelet surface (mediated by the GPIIb/IIIa receptor).

They are used as emergency thrombolytic drugs in the treatment of AMI. The sooner therapy with GPIIb/IIIa inhibitors is provided, the most favourable the prognosis for AMI.

2.3. PAR1 antagonist (protease 1 activating receptor)

2.3.1. Vorapaxar (Zontivity®)

It inhibits thrombin-mediated aggregation, since it is an antagonist of the thrombin PAR1 receptor. Accepted by the FDA, but not by the EMA.

3. Prostacyclin analogues

3.1. Iloprost (Ilomedin®)

Iloprost is an analogue of prostacyclin, which increases intraplatelet cAMP and is also a vasodilator. It is used in peripheral artery disease, thromboangiitis obliterans and Raynaud's disease.

CLINICAL CONSIDERATIONS

The Working Group on Cardiovascular Thrombosis of the Spanish Society of Cardiology[8], coordinated by the Spanish Society of Cardiology (SEC) and made up of representatives of the majority of Spanish medical societies, considers that dental surgical procedures are of low haemorrhagic risk, since haemostasis can be achieved properly, a possible haemorrhage does not pose a vital risk to the patient or compromise the outcome of surgery and usually do not require transfusion. While some maxillofacial surgery procedures may be medium or high risk; medium risk would be when the haemorrhage may result in a transfusion or reoperation (such as removal of tumours, radical resection of the maxilla or jaw, or reduction of complicated bone fractures); and the high risk would exist in those surgeries in which the perioperative haemorrhage can be life-threatening for the patient or the outcome of surgery, such as in LeFort I, II or III surgery.

However, the authors of this study consider that the haemorrhagic risk of dental procedures should be stratified in another way. Therefore, we have classified the risk of bleeding following González et al[9] in: low haemorrhagic risk, if a simple exodontia or transmucosal implant is to be done; medium risk, if less than 3 simple exodontia, less than 3 implants, second phases or scaling and root planning (SRP) are to be performed; and high risk if regenerative procedures are to be performed, soft and hard tissue grafts, sinus elevations, impacted teeth extractions, more than 3 implants or more than 3 extractions (Table 2).

Table 2. Surgical procedures in oral surgery according to bleeding risk.

Bleeding risk	Procedure
Low	A simple exodontia A transmucosal implant
Medium	2-3 simple exodontia 2-3 implants SRP Second phases
High	More than 3 exodontia More than 3 implants Sinus lifts Bone regeneration techniques Soft tissue grafts Bone grafts

Modified by González et al. (2016)[8].

Table 3. Stratification of thrombotic risk.

HIGH	SCA	<3 months of medical treatment with AAP after ACS <6 months after PCI + metal stent or DES <12 months after PCI + metal stent or DES + risk factors <12 months after PCI + 1st DES generation
	Stable coronary artery disease	<3 months after PCI + metal stent or DES <6 months after PCI + metal stent or DES + risk factors <12 months after PCI + 1st DES generation
MODERATE	SCA	3-6 months of medical treatment with AAP after ACS 6-12 months after PCI + metal stent or DES > 12 months after PCI + metal stent or DES + risk factors > 12 months after PCI + 1st DES generation
	Stable coronary artery disease	3-6 months after PCI + metal stent or DES 6- 12 months after PCI + metal stent or DES + risk factors > 12 months after PCI + 1st DES generation
LOW	SCA	> 6 months of medical treatment with AAP after ACS > 12 months after PCI+ metal stent or DES
	Stable coronary artery disease	>6 months after PCI + metal stent or DES >12 months after PCI + metal stent or DES + risk factors

AAP: platelet antiaggregants PCI: Percutaneous coronary intervention *ACS: acute coronary syndrome DES: pharmacoactive stent* *Modified from Vivas et al., 2018[8].*

The incidence of post-extraction bleeding rate in patients treated with AAP varies in literature from 0 to 17.4%[10-13]. However, it is necessary to take into account that in these patients the thrombotic risk is more important than the bleeding[7] risk. Discontinuation of antiplatelet therapy may trigger thromboembolic events with serious consequences[7]. Stent thrombosis is a rare but potentially catastrophic event, leading to ACS or even death in 25-45% of the cases[14], especially in the first 6 years of its placement[8].

To stratify the thrombotic risk, the most important thing is the time elapsed since the transient ischemic attack, but the type of implanted stent must also be considered and how the ischaemic episode occurs. The lack of clinical trials should be noted[8].

Patients with a stent are at higher risk of thromboembolic complications than those with a stable coronary artery disease[7,14], especially when a drug-eluting stent or pharmacoactive stent (DES) has been inserted, especially the first generation, which are associated with a higher thrombosis possibility than the second generation, which have improved their safety profile[8].

According to Vivas et al.[8], (2018), a high thrombotic risk has been reported when less than 3 months of ACS have passed and is only under medical treatment (especially during the first month) or less than 6 months from the implantation of a second generation DES or less than 12 months from the first generation DES. The thrombotic risk will be moderate when 3 to 6 months have passed after the ACS under medical treatment, between 6 and 12 months after placement of second-generation ACS or more than one year of first-generation ACS. Thrombotic risk is considered low if more than 6 months of ACS under medical treatment or more than one year since the placement of second-generation ACS[8] (Table 3) have passed.

In general, without having to consult the cardiologist for the type of stent, the thrombotic risk can be summarized as: high if less than 6 months have passed since the implantation of any type of stent; medium risk, if 6 to 12 months have passed since the implantation of any type of stent; and low risk, if more than 12 months have passed since the implantation of any stent or if it does not have a stent.

In general, the carriers of one or more stents undergo double antiplatelet therapy (DAPT) during the year following the implantation surgery[8], for which during this period their thrombotic risk is considered moderate to high and also their bleeding risk.

The removal of ASA produces a rebound effect on the platelet physiology, in such a way that it decreases fibrinolysis and increases TXA_2[6] production. Each day that the ASA or clopidogrel is suspended, a platelet regeneration of 15-20%[3] occurs, that is, after stopping the antiplatelet therapy, platelet aggregation returns to baseline level after 5 days[6].

REVIEW OF THE MOST USED DRUGS LITERATURE

ASA

Haemorrhagic risk

Omar et al.[15], in 2015, observed that taking ASA did not increase the risk of bleeding in patients who underwent the extraction of all the teeth (full-mouth extraction), recommending to continue taking ASA and use local haemostatic measures.

In the study of Lu et al.[16] (2015), the incidence of haemorrhage in the group with ASA was 1.2% vs 0.7% in the control group, so they do not advise stopping the drug.

Eapen et al.[17], in 2017, designed a prospective study with 80 patients receiving a low ASA dose treatment for tooth extractions. In one group the ASA was stopped and the other continued the treatment. In no case was there prolonged postoperative bleeding and only one case with ASA local haemostatic measures had to be performed. This group recommends not to interrupt ASA before tooth extraction.

In the prospective study by Gupta et al.[18], 2018, they concluded that stopping ASA prior to tooth extractions is not necessary, as local haemorrhage can be solved with local haemostatic measures.

Thrombotic risk and ASA interruption

In the meta-analysis by Burger et al.[19], 2.3% to 6.1% of acute cardiovascular events were observed when ASA was stopped before surgery.

In the meta-analysis by Biondi-Zoccai et al.[20], with more than 50,000 patients it was found that the interruption of ASA produced adverse cardiovascular effects, so they advised not to stop ASA therapy.

According to Mahmood et al.[7], ASA should not be stopped, especially if it is indicated in secondary prevention of ACS, stroke or after revascularization surgery[7].

For patients with simple antiaggregation (SAA), it is recommended to continue with ASA, since it has been shown to reduce the ischemic risk without significantly increasing the risk of bleeding, according to the Working Group on Cardiovascular Thrombosis of the Spanish Society of Cardiology[8].

Clopidogrel

Haemorrhagic risk

Omar et al.[15] observed that Clopidogrel administration did not increase the risk of bleeding in patients who had all their teeth extracted (full-mouth extraction), recommending to maintain Clopidogrel and use local haemostatic measures.

Thrombotic risk and Clopidogrel interruption

Stent carriers have a higher risk of thrombotic complications, especially with drug coatings. There are examples in the literature where it is reported that after stopping Clopidogrel, a stent thrombosis occurs[14,21,22].

Clopidogrel discontinuation is a risk factor for stent thrombosis. When the patient is in AAP treatment only with Clopidogrel it is advised not to stop it[7], When the thrombotic risk is high or moderate, Clopidogrel should not be stopped before dentoalveolar surgery[7]. It is preferable to postpone surgery until the thrombotic risk is low[8]. When more than 12 months have passed since stent insertion and the thrombotic risk is low, if

the dental procedure involves a low haemorrhagic risk, such as a simple exodontia, clopidogrel should not be stopped. But if the surgical procedure is regenerative surgery with a high bleeding risk, the cardiologist should be consulted to discontinue Clopidogrel 5 days before surgery and take it again after 24-48 hours.

DAP

Haemorrhagic risk

Lillis et al.[23] compared bleeding after tooth extractions in patients with DAP vs SAA and observed increased bleeding in DAP. However, all cases were successfully handled with local measures.

In the study by Lu et al.[16], the incidence of bleeding in the DAP group was 4.4% vs 0.7% in the control group, however, they do not advise discontinuing this treatment before tooth extractions.

Napeñas et al.[24], found no significant differences in intraoperative bleeding between the DAP and SAA groups, although they observed greater bleeding in the immediate postoperative period in the DAP group. His opinion is that it is not necessary to stop dual antiplatelet therapy before the dental surgical procedure.

Olmos-Carrasco in 2018[10] found 8.3% of haemorrhagic complications in the first 30 minutes after tooth extraction in patients with DAP, which were resolved with local haemostatic measures.

Nathwani and Martin in 2016[25] conducted a literature review and in the consulted articles all cases of bleeding under DAP were resolved with local measures, so they do not advise to interrupt the antiaggregant.

In the Ockerman systematic review of 2019[26], they found greater postoperative bleeding with DAP than with SAA, but all cases were solved with local measures and the authors do not recommend stopping any DPA before exodontia.

Sánchez-Palomino et al.[12] consider that an impregnated gauze in tranexamic acid (Amchafibrin®) for 30 minutes is advisable to avoid postoperative bleeding in patients with SAA and DAP.

Mahmood et al. in 2020[7] commented that there is no article of uncontrolled bleeding after dental surgery in patients under DAP and conclude that there is no indication to stop DAP prior to a surgical procedure of the oral cavity.

Thrombotic risk and DAP discontinuance

In the article jointly published by the American Dental Association, the American Heart Association, the American College of Cardiology, the Society for Cardiovascular Angiography & Interventions and the American College of Surgeons[27] highlighted the importance to continue with DAP in patients with coronary stents. Discontinuation of dual treatment with aspirin and clopidogrel in patients with stents is associated with a 5 to 10 higher risk of myocardial infarction and even mortality. This risk is inversely proportional to the time elapsed since stent insertion. The thrombosis risk is greater than the risk of bleeding, so stopping ASA or clopidogrel should be avoided[27].

In patients under DAP, dental surgeries should be postponed until they stop this therapy. They can only undergo surgery if their life depends on the same and without interrupting the DAP7. In the systematic review by Childers et al. 28, they reaffirm the need to conduct a risk-benefit assessment before performing dental surgery with these patients.

According to the Working Group on Cardiovascular Thrombosis of the Spanish Society of Cardiology[8], which seeks a consensus to homogenize protocols, the first consideration with patients under PAD is to assess the need for elective intervention while the thrombotic risk is moderate to high; if the intervention can be delayed, it is best to postpone it until the thrombotic risk of the patient is considered low.

When the thrombotic risk is low, that is, more than 12 months have passed after the implantation of any stent, but the cardiologist has preferred to maintain the DAP beyond the first year, and they will undergo minor dental surgical procedures, with low bleeding risk, as a simple extraction, ASA and clopidogrel

should be maintained. If the thrombotic risk is low and the bleeding risk is moderate to high, for example, in a regenerative surgery procedure, the possible discontinuance of clopidogrel 5 days before surgery will have to be discussed with the cardiologist.

DENTAL MANAGEMENT OF THE PATIENT UNDER ANTIAGGREGANTS

For many years, dentists have overestimated the risk of local bleeding when performing exodontia or implants in patients who are taking antiaggregants and, on the other hand, we have underestimated the risk of thrombosis, promoting the discontinuance of the drug between 5 and 7 days before the surgical procedure. However, the current recommendations go in the opposite direction, since it has been seen that when antiaggregants are stopped, new cardiovascular events can occur[2,6,7,27].

Regarding thrombotic risk, surgical procedures may be performed only when it is low, that is, when more than one year has passed after the stent placement[8]. if not more than one year has passed, it is preferable to postpone dental surgery[6,8].

It should be borne in mind that, at present, the single antiaggregant medication should not be stopped before performing an exodontia or a surgical procedure in the oral cavity and, if modified, it should be as little as possible and after consultation with the cardiologist[17,18,23].

When a patient is being treated with ASA at low doses (100-300 mg/ day), it is not stopped[6,7,19].

When a patient is exclusively taking clopidogrel (75 mg) and dental extractions or surgical procedure are required, it is not stopped. Only if the thrombotic risk is low and regenerative surgery procedures are required can the drug be satopped for 5 days after consultation with the cardiologist[8,15].

If the patient is receiving dual antiplatelet therapy with ASA plus clopidogrel, it is because they have had one or more stents inserted in the last year, therefore, it will have a moderate to high thrombotic risk and it is not advisable to discontinue any of the drugs prior to an exodontia or surgical procedure of any kind[26-30]. It is preferable to postpone the surgical procedure[6,8].

If the patient has a low thrombotic risk and a low bleeding risk is expected, SAA should be continued with both ASA and clopidogrel. If the patient is still under DAP after more than one year of the stent insertion, the cardiologist who knows the hemodynamic state and the type of stent the patient has, should be consulted to be able to discontinue exclusively the clopidogrel 5 days before the dental surgical procedure. The ASA will always be continued.

Each case should be individualized, but in general[6-13,15-20,23-31] (Table 4):

1. When the patient is treated with ASA 100- 300 mg it is always continued.
2. When the patient is taking clopidogrel at low doses (75 mg/ day), it is not stopped.
3. If the patient is being treated with another AAP more potent than ASA or clopidogrel (ticagrelor or prasugrel), the cardiologist should be consulted.
4. When the thrombotic risk is high (for example, a patient with a stent that has been inserted for less than 6 months) and the patient is undergoing DAP, none of the AAPs can be stopped. It is preferable to postpone the procedure[8].
5. If the thrombotic risk is moderate (for example, a patient who has a stent that has been inserted between 6 and 12 months), according to the Spanish Society of Cardiology, the ideal is to wait for the thrombotic risk to be low[8].
6. If the thrombotic risk is low and the bleeding risk is expected to be low, such as exodontia, antiaggregants are maintained.
7. If the thrombotic risk is low, but the haemorrhagic risk is expected to be moderate to high, as in

Table 4. Surgical management protocol of the patient under treatment with AAP.

		HAEMORRHAGIC RISK			
		DRUG	**HIGH**	**MEDIUM**	**LOW**
THROMBOTIC RISK	**High (<6 months)**	ASA 100	Postpone surgery	Postpone surgery	Postpone surgery
		Clopidogrel 75	Postpone surgery	Postpone surgery	Postpone surgery
		Dual AAP	Postpone surgery	Postpone surgery	Postpone surgery
	Medium (6-12 months)	ASA 100	Postpone surgery	Postpone surgery	Postpone surgery
		Clopidogrel 75	Postpone surgery	Postpone surgery	Postpone surgery
		Dual AAP	Postpone surgery	Postpone surgery	Postpone surgery
	Low (>12 months)	ASA 100	Continue	Continue	Continue
		Clopidogrel 75	Consult if stopping 5 days and continue after 48hr	Consult if stopping 5 days and continue after 24hr	Continue
		Dual AAP	Continue ASA Consult Clopidogrel	Continue ASA Consult Clopidogrel	Continue ASA Consult Clopidogrel

regenerative procedures, the cardiologist should be consulted to stop clopidogrel 5 days before the procedure and resume it after 48 hours, if the bleeding risk is high after 24 hours, or if the risk is moderate[8].

8. When the patient is undergoing a triple antiaggregant therapy, the cardiologist should be consulted.
9. In all cases it will be necessary to carry out local haemostatic procedures that, according to the consulted authors, are effective to prevent local haemorrhagic complications[6-19,24-31].

Local tranexamic acid is an effective option to reduce the bleeding risk in patients who are being treated with platelet antiaggregants[12].

CONCLUSIONS

- ASA is the basic antithrombotic therapy used as a secondary prevention of atherothrombotic events.
- ASA causes the least bleeding. A dose of 100 mg of ASA is always maintained.
- Although it is necessary to individualize each case, if the thrombotic risk is high or moderate, antiaggregants cannot be stopped, therefore it is better to wait for the thrombotic risk to be low, postponing dental surgery.
- If the antiplatelet therapy is with a single drug (SAA), either ASA 100 mg or clopidogrel 75 mg, it is always maintained and only local haemostatic procedures will be used. Only if the thrombotic risk is low and regenerative surgery procedures are required, clopidogrel can be stopped for 5 days, after consulting the cardiologist.
- If the patient is being treated with another more powerful AAP (ticagrelor, prasugrel), the cardiologist should be consulted.
- If the thrombotic risk is low, because more than 12 months have passed after the stent insertion, but the patient is still on dual antiplatelet therapy, the ASA is not stopped. In this case, if in addition the bleeding risk is moderate to high, the cardiologist can be consulted to stop clopidogrel 5 days before surgery and resumed after 48 hours if the risk is high or at 24 if the risk is moderate.
- It is time to stop interrupting antiplatelet therapy before performing a tooth extraction.

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Original article

Implants of 4.5 and 5.5 mm length inserted directly in crestal elevation with autologous bone attached to PRFG-Endoret in residual bone heights of 2-3 mm: retrospective study

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SUMMARY

Introduction. The approach to the atrophic maxilla in height is a challenge for the dentist, and new techniques that allow us to place implants with less invasiveness for the patient are becoming more and more common. The incorporation of the transcrestal approach and the reduction in implant length have been key to treating more patients with a smaller number of surgical interventions. In this study we present a series of cases with extreme bone atrophy in height rehabilitated using transcrestal elevation and 4.5 and 5.5 mm long implants.

Methods. A retrospective study was carried out in patients in whom extra-short implants (4.5 and 5.5 mm in length) were inserted directly by transcrestal elevation with residual ridge heights between 2 and 3 mm. The implant was the unit of analysis for descriptive statistics in terms of location, implant dimensions, and radiographic measurements. The patient was the unit of measurement for the analysis of age, sex and medical history. The primary variable was the gain in height above the implant apex 6 months after surgery and one year after loading, comparing the two measurements. Biological complications and implant failure were recorded as secondary variables.

Results. Ten patients who met the inclusion criteria were recruited and 20 implants were inserted. The mean residual bone volume height was 3.1 mm (+/- 0.3 mm with a range of 3-4 mm). In all cases a transcrestal sinus lift performed, with autologous particulate bone obtained from the drilling of the neoalveolus generation zone for implant insertion, the mean of this elevation above the implant apex in millimetres being 2.8 mm (+/- 0.99 range 1.9 -5 mm). In the control TC at one year of loading of the implants studied, the bone gain achieved was maintained, with no decrease in the volume gained being observed; only three cases showed a decrease of between 0.4 and 0.5 mm of the initial volume at the end. No implants failed during the follow-up period and no biological complications were noted during surgery.

Conclusions. Achieving success of implants placed in areas of extreme horizontal atrophy by transcrestal sinus approach with extra-short implants is possible provided that correct stabilization of the implant is achieved in the initial phase (primary stability) and a careful protocol of drilling, implant insertion, type of bone graft (100% bone and autologous plasma processing) and subsequent loading of the implant (progressive loading) is used.

KEY WORDS

Transcrestal elevation; Extra short implant; Primary stability.

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INTRODUCTION

In implantology, as in other areas of medical medicine, surgical techniques undergo an evolution, usually to adapt to new scientific advances to new materials and to achieve techniques that solve the same problems in less time, more predictability and with lower morbidity for the patient[1-3]. The advent of shorter implants such as short, extra-short and ultra-short has made possible the rehabilitation of large height atrophies with fewer surgeries, avoiding, in many cases, accessory bone regeneration techniques[4-9]. In the posterior areas of the maxilla, the most used technique to produce a gain in hight when bone has been lost vertically has long been sinus elevation by lateral approach[10]. Subsequently, a variant of this technique was developed, generating access through the alveolar crest, from the neo alveolus formed for the insertion of the future implant. This technique initially described by Summers[11], which received his name, used osteotomes and hammer to raise the lower cortical of the sinus floor once the approach was made through the crest. As a result of achieving a constant improvement of the crestal approach surgery, different access systems (ultrasound and milling cutters mainly)[12,13] and alternatives designed to detach the Schneider membrane once exposed (controlled pressure instruments, pneumatic balloons, spatulas with different shapes...)[14,15], as well as variations regarding the type of material used as a graft, even performed out without filling material[18- 20].

The crestal lifting technique is currently among the most used to treat the height deficit of the posterior maxilla, when there is a bone remnant of at least 5 mm[21-23] in height, although currently there are also research studies that indicate that this technique can be used even if the bone volume is less than this height[20-25].

The use of a careful milling technique adapted to the receiving orifice, increasing the primary stability with the diameter of the implant and the anchorage in the vestibular cortex, palatal, mesial and distal (instead of looking for apical anchorage), they are the success keys of these works where extra-short implants have been inserted at residual heights of less than 5 mm even when the residual density was low[20-25]. In addition, in the follow-up of these implants, no lower success rate or complications related to low residual height or migrations of implants to the maxillary sinus have been found[20-25].

In this type of approach to the sinus, an important point is the stability of the grafted bone, located above the apex of the implant and with a bone tissue little vascularized (as usually occurs in these large atrophies with low density), so assessing what happens with the bone volume gained by this long-term procedure is also a key fact[26,27]. The mineralization of the bone graft and its maintenance once the implant loading is performed can make the difference in the success of the technique, especially in increasingly extreme cases. Therefore, the material used as a graft and the surface of the implant are two factors to take into account when performing this type of procedure[28-31]. Hydrophilic and osteoconductive surfaces in implants are of vital importance in these complex cases, as well as the filling materials that stimulate the formation of new bone[28-31]. The implants with UnicCa® (Biotechnology Institute, Vitoria, Spain) surface, have a superhydrophilic surface. It is a very rough surface (Optima®), with a calcium ions layer. This implies that the contact of blood and plasma with all points of the surface increases to the maximum the active surface for regeneration, by being completely coated with fluids due to its high capillarity. In the following series of clinical cases, patients treated by transcrestal sinus lift, with extra-short and ultra-short implants (4.5 and 5.5 mm), BTI (Biotechnology institute), of internal connection and universal plus platform in residual bone heights below 3 mm, studying the behaviour of implants regarding their survival as well as the maintenance of the bone volume achieved in the crestal elevation.

MATERIAL AND METHOD

Patients were retrospectively selected in which extra-short implants were inserted using the crestal lift technique using frontal drills, according to the techni-

que used and described by our study group[13], which surgery was carried out before 2015 to have a load follow-up period of at least 5 years, and in which the residual bone height was less than 3 mm.

In all cases, a diagnosis was made based on study models, intraoral examination of the patient and performing a Cone-beam analysed using the specific BTI-Scan 3 software (Biotechnology Institute, Vitoria, Alava, Spain).

Prior to the implant insertion, an antibiotic premedication consisting of amoxicillin 2 g orally one hour before the procedure and paracetamol 1 g orally (as analgesic) was used. Patients continued with an amoxicillin treatment 500- 750 mg orally every 8 hours (depending on weight) for 5 days. As a filling material in all cases, using the autologous bone obtained from the milling (from the same transcrestal elevation area where the implants were inserted simultaneously) embedded in PRGF-Endoret fraction 2. The milling was carried out at low revolutions (biologic milling)[32], the membrane is accessible by crestal perforation, it is detached, the graft is placed and then the implant is inserted with the surgical motor fixed at 25 Ncm and 25 rpm, finishing the implant insertion with the torque wrench (Figure 1).

Patients attend subsequent check-ups performing a control Cone-Beam after 5 months (before loading the implant) and after one year of the load, performing a new measurement in these images to analyse the bone gain and the maintenance of the same. In these check-ups, data are collected on prosthetic complications or crestal bone loss in these patients, as well as possible failures.

The implant was the analysis unit for the descriptive statistics regarding location, implant dimensions, and radiographic measurements. The patient was the measurement unit for the analysis of age, sex and medical history. The main variable was the gain in height over the apex of the implant after 6 months of surgery and one year after the load comparing the two measurements and the biological complications and implant

Figure 1. The step-by-step technique: a) is marked with the starter drill keeping 0.5-1 mm of margin to prevent drilling the Schneider membrane. b) Continue widening maintaining 0.5 mm of safety margin. c) With the frontal drill the sinus membrane is reached. d) With the bone recovered from the milling agglutinated with the plasma and with a PRGF clot, the membrane is lifted. e) Lifting is finished with the insertion of the implant.

failure were recorded as secondary variables. The Shapiro-Wilk test was performed on the data obtained to verify the normal distribution of the sample.

The qualitative variables were described using a frequency analysis. Quantitative variables were described by average and standard deviation. Implant survival was calculated using the Kaplan-Meier method. The data were analysed with SPSS v15.0 for Windows (SPSS Inc., Chicago, IL, USA).

RESULTS

Ten patients who met the inclusion criteria were recruited, in which 20 implants were inserted. Three of them were women with an average age of 72 (+/- 6 years). None of the patients were smokers at the time of surgery and did not have any active periodontal disease. The majority of the implants diameter included in the study was 5 mm (60%), followed by 4.5, 5.5 and 4.75 mm (13.33% each). The predominant length was 5.5 mm (86.6% of the cases), with 13.4% of the remaining implants with a length of 4.5 mm. The most common positions were for the second molars (molars 27 and 17) representing 55% of the cases.

The mean height of the residual bone volume was 3.1 mm (+/- 0.3 mm with a range of 3-4 mm). In all cases, transcrestal sinus elevation was performed, with particulate autologous bone obtained from milling the neo alveolus generation zone for implant insertion, being the average of this elevation above the apex of the implant of 2.8 mm (+/- 0.99 range 1.9 -5 mm). In the CT control scan after one year of inserting the studied implants, the bone gain achieved was maintained, no decrease in the volume gained was observed, only three cases showed a decrease of between 0.4 and 0.5 mm of the initial volume at the end (Table).

All implants were rehabilitated in two phases and all of them were ferulized to other implants in the rehabilitation. In all cases, screwed prostheses of more than one implant with intermediate elements (transepithelial) were performed, ferulizing to other implants with a length of 4.5 or 5.5 mm. In all situations, a progressive prosthesis load was carried out 6 months after insertion of the implant consisting of a provisional structure finished in resin to move to a definitive metal-ceramic prosthesis. In all cases, the initial transepithelial are preserved to maintain the hermeticity achieved in the first phase of prosthesis manufacture.

No implants failed in the follow-up period and no biological complications were found in the surgery. The postoperative condition of all patients was excellent with minimal discomfort in the surgery area and with minimal postoperative inflammation.

Figures 2-19 show one of the cases included in the study.

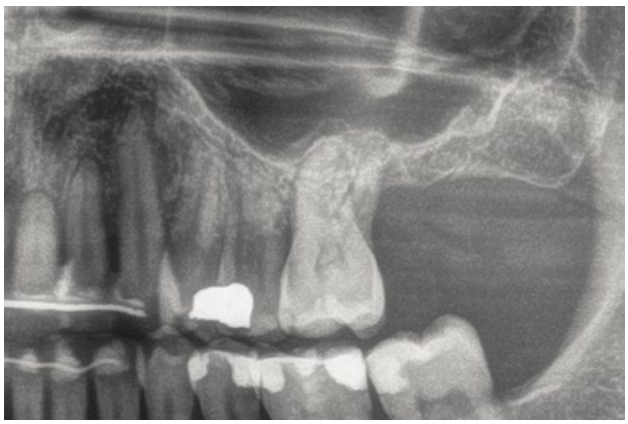


Figure 2. Radiological image of molar 26. The bone loss can be seen, in addition to a mobility grade III.

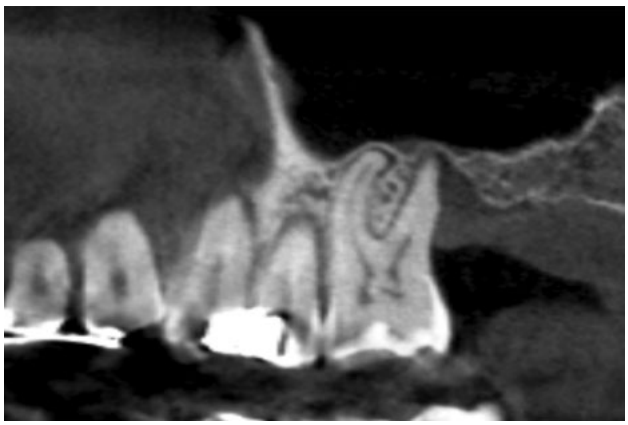
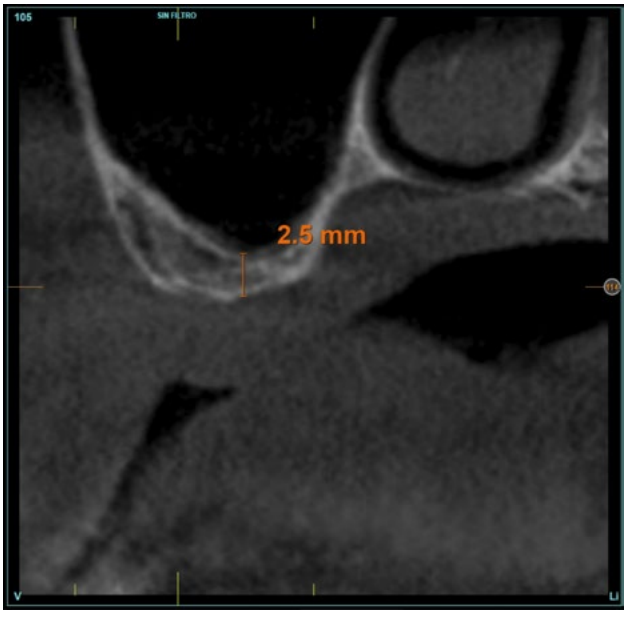
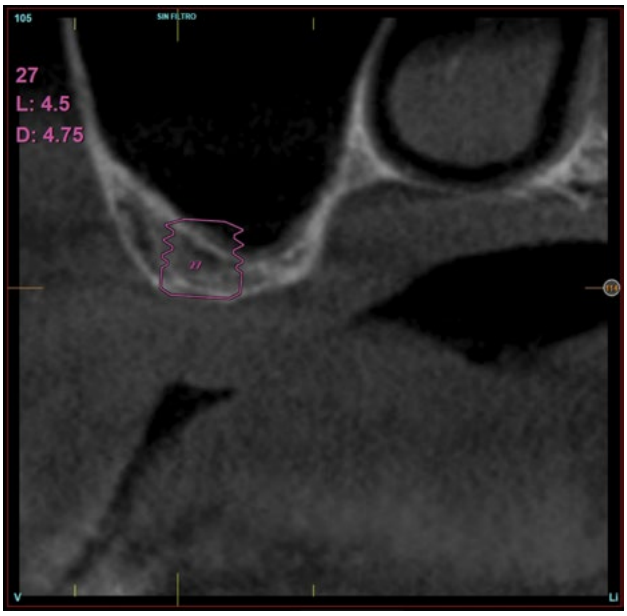


Figure 3. Panoramic cut of the CBCT where an apical image is observed in the distal root. It was decided to proceed with the extraction and regeneration with PRGF–Endoret in addition to insertion of an implant in the area corresponding to molar 27.

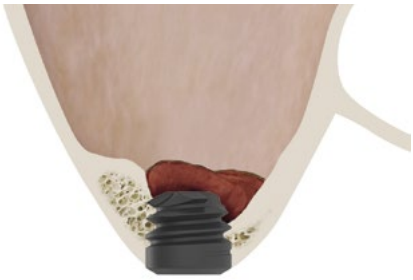
Table. Characteristics of the implants included in the study and initial and final bone gain

Implant	Position	Age	Sex	BONE GAIN OVER THE INITIAL APEX (mm)	BONE GAIN OVER THE APEX AFTER A YEAR (mm)
1	27	76	M	1.9	1.9
2	17	71	M	1.9	1.9
3	17	72	M	5.00	4.6
4	27	69	M	2.10	2.10
5	16	82	W	2.80	2.80
6	15	68	M	2.30	2.30
7	26	69	M	2.87	2.87
8	15	63	M	2.98	2.98
9	17	74	W	2.79	2.79
10	17	76	W	2.56	2.56
11	27	76	M	2.80	2.80
12	16	71	M	3.10	3.10
13	17	72	M	2.27	1.87
14	26	69	M	2.99	2.99
15	27	82	M	2.89	2.89
16	25	68	M	2.88	2.88
17	27	69	M	2.09	2.09
18	26	63	M	3.00	3.00
19	17	74	M	3.88	3.88
20	26	76	M	3.90	3.4

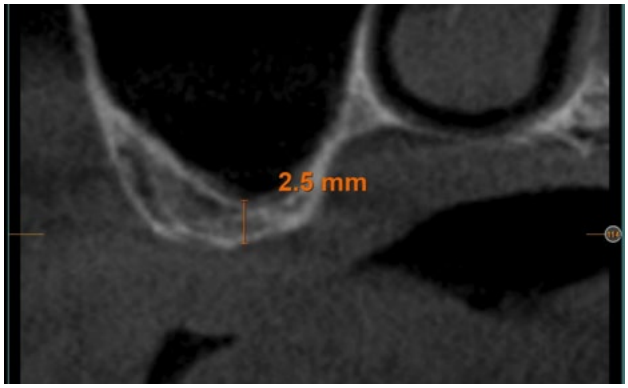


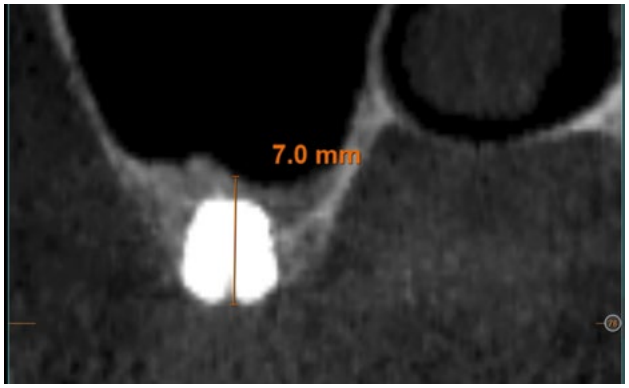


Figures 4 and 5. CBCT cuts corresponding to molar zone 27. A bone height of less than 2.5 mm is seen and the planned implant is 4.5 mm long by 4.75 mm diameter that will be anchored in the remaining cortical in the vestibular area.



Figures 6 and 7. Schematic of the planning of bone graft placement using a transcrestal lift, as previously described with the anchoring of the implant in the vestibular portion of the bone crest.



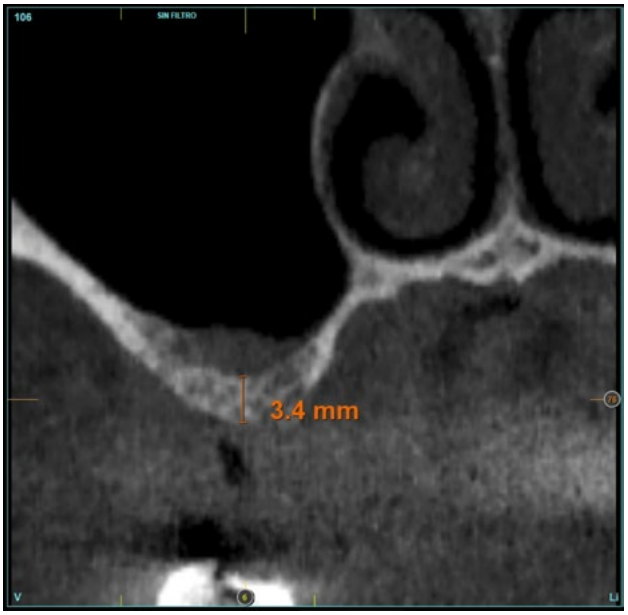


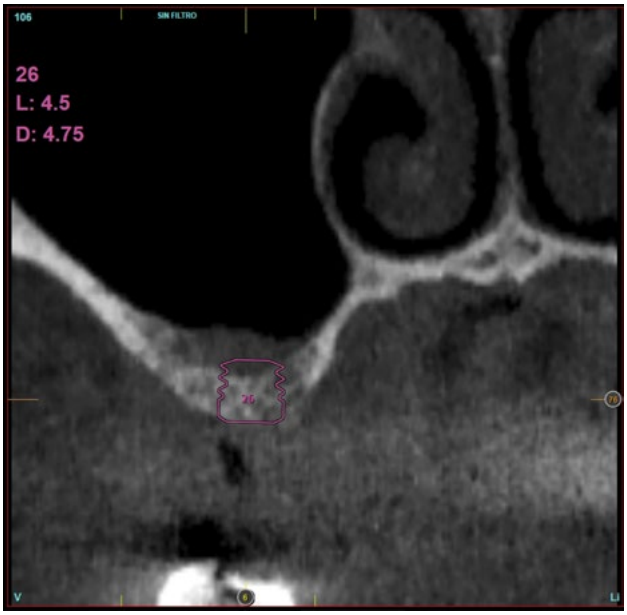
Figures 8 and 9. CBCT cuts prior to implant insertion and the transcrestal lift, and after graft healing and implant osseointegration 6 months after surgery. A bone gain of 4 mm is seen.

DISCUSSION

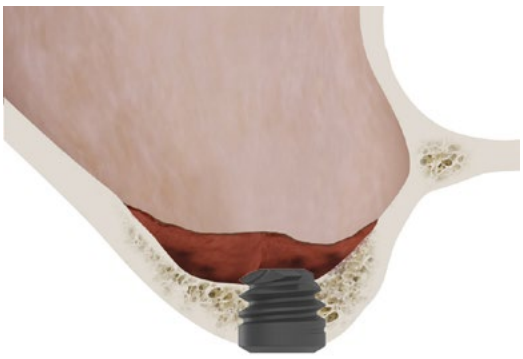
Transcrestal sinus lift with short and extra-short implants is a highly predictable technique with high success rates for elevated implant, finding slight differences in residual bone volume. The survival of short and extra-short implants inserted by a transcrestal procedure when residual bone crest is 5 mm high or more is found in 94.9%, compared to 92.7% reported for cases where these implants are inserted in areas with severe vertical resorption (less than 5 mm of residual bone height)[21,22]. This decrease in predictability may be due to the milling sequence, stabilization of the implant and the surface of the implant, since they are elements that play in favour of achieving a primary stability in areas with low density and this can make the difference in limit cases[13,24,27,30-33]. In implant insertion surgery, we can modify all the parameters except bone density to play in our favour and achieve greater primary stability in cases where the bone does not offer a correct stability by itself. It is possible to vary the implant to use, its diameter and in many cases its length (extra-short implants 4.5-5-5 mm), as well as adapt the milling sequence to achieve compression.

In addition, the use of bioactive implant surfaces, which accelerate bone formation in contact with the graft substrate can lead to substantial improvement of the results obtained when increasing the height of bone volume above the apex as well as the posterior maintenance. The load received by the implant is also key in maintaining the increased bone. A poor design of the prosthesis can result in the best surgeries not being successful. Therefore, the work of prostheses on transepithelial instead of directly to implant, the maintenance of implant-prosthesis hermeticity and the distancing of the critical attachment area of the implant with the prosthesis (with the height of the transepithelial next to or slightly supra-gingival areas) it can make the success achieved in the first phase of the treatment to be retained after loading[24-25].

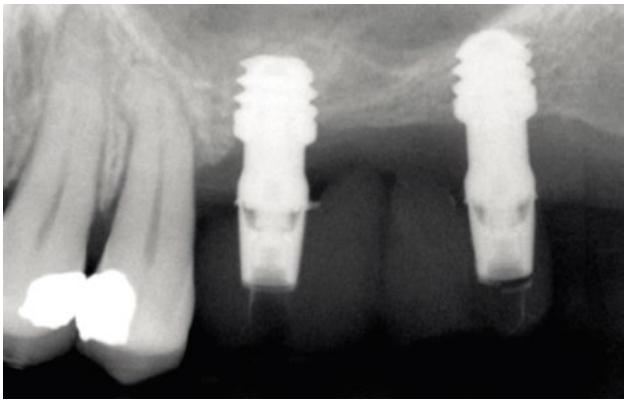


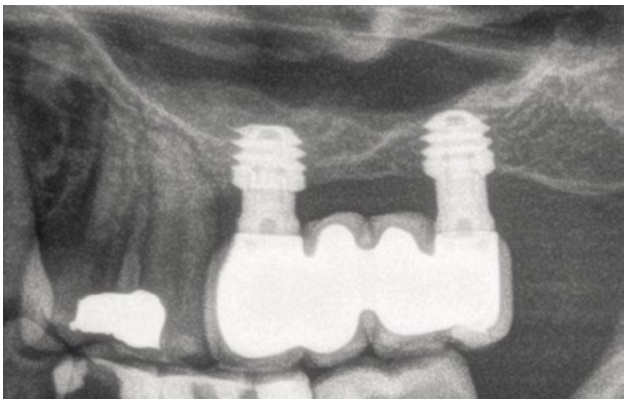


Figures 10 and 11. CBCT planning cuts after regeneration of the alveolus of zone 26. A residual bone volume is seen with a height of 3.4 mm, being more uniform on this occasion throughout the crest area. An implant of 4.5 mm in length and 4.75 mm in diameter is planned.

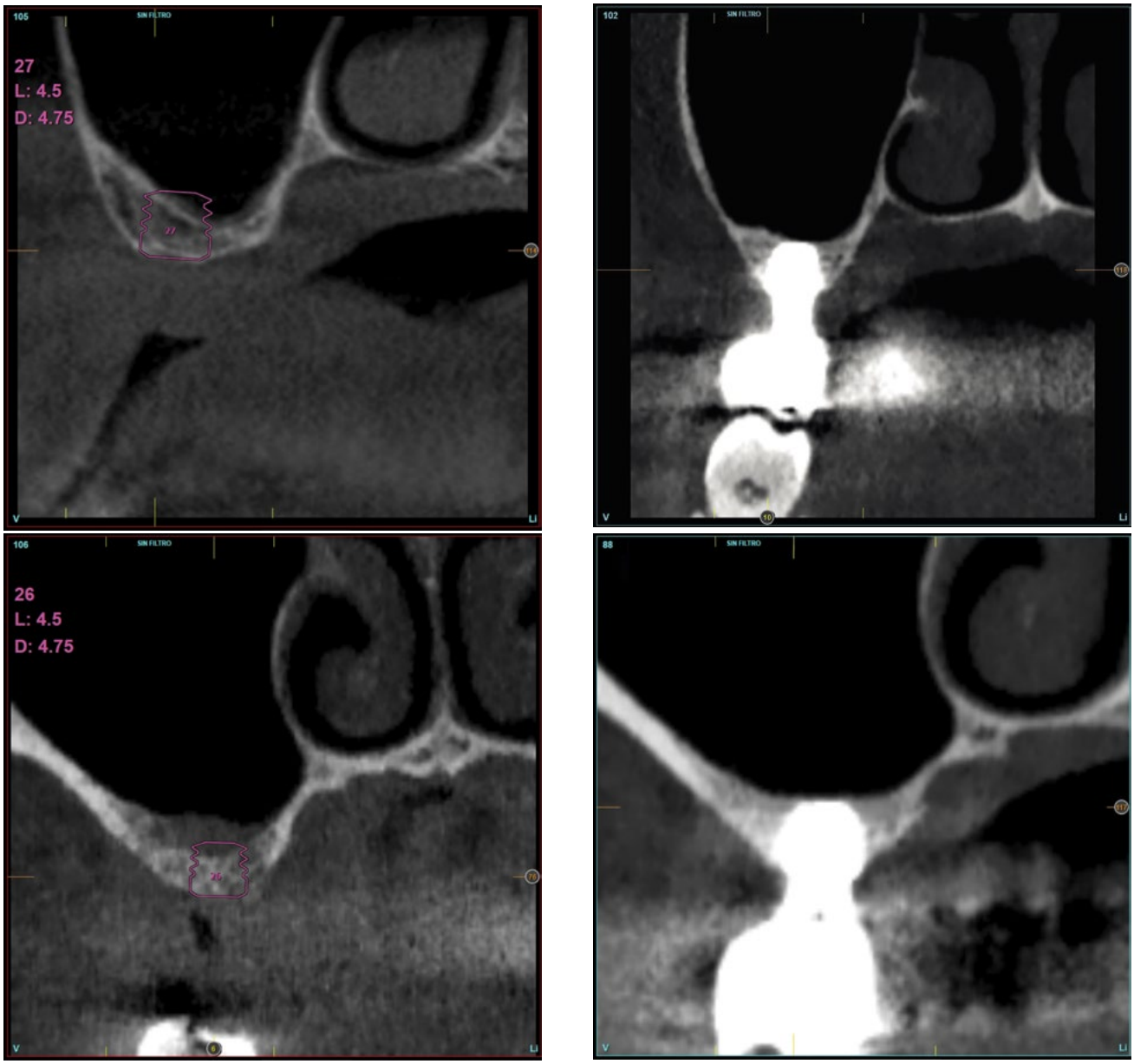


Figures 12 and 13. Schematic of a transcrestal lift for the insertion of the implant in the area corresponding to the 26.





Figures 14 and 15. X-ray of the provisional prosthesis of a progressive load (performed in PMMA by CAD-CAM) and final prosthesis performed on MULTI-IM transepithelial, screwed.



Figures 16-19. Planning and final cuts after two years of loading. It is observed in both areas (26 and 27) before the implant insertion and the gained and preserved bone volume after the function of the implants.

CONCLUSIONS

To achieve the success of the implants placed in areas of extreme horizontal atrophy by a transcrestal sinus approach with extra-short implants is possible, provided that a correct stabilization of the implant is achieved in the initial phase (primary stability) and a careful milling protocol is used, insertion of the implant, management of the graft, 100% processing of bone and autologous plasma and subsequent loading of the same (progressive loading).

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CLINICAL CASE

Maxillary reconstruction with a subperiosteal implant in a case of severe atrophy. From planning to rehabilitation following a fully digital protocol. About a clinical case and bibliographic review

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SUMMARY

Introduction: Customized titanium subperiosteal implants, designed from a planning and manufacturing software, are an alternative in the rehabilitation of severe maxilla atrophies, avoiding more complex bone and soft tissue reconstructive surgeries and reducing healing times. The aim of this work is to present the rehabilitation of a clinical case with severe atrophy in the upper maxilla, using a structure with subperiosteal implants, through a digital protocol.

Description of the case: A subperiosteal structure of sintered itanium was designed with six transepithelial connections that were rehabilitated with an immediate implant-supported fixed prosthesis manufactured in PMMA. Two months later, a sintered chromium-cobalt structure was made with mechanized bases covered with acrylic resin teeth as final restoration. In the one year follow-up, the case remains stable.

Conclusions: Nowadays, rehabilitation with subperiosteal implants are an alternative tool in cases of complex surgeries with large atrophies with the possibility, in addition, of performing an immediate load.

KEYWORDS

Subperiostal implants; Oral rehabilitation; Digital planning; Cad/Cam.

INTRODUCTION

The use of endoosseous dental implants to replace the absence of teeth has shown a great predictability over the years, being today one of the main techniques for dental rehabilitation[1]. However, bone in quantity and quality is necessary for its placement. In cases of severe bone resorption, more advanced surgeries are needed for bone regeneration, in which there may be more complications, morbidity and longer treatment time[2].

Subperiosteal implants (SI) were developed in Sweden in the early 1940s. The SI consisted of a custom-made implant, inserted under the periosteum and fixed with screws and the mucous tissue that covered it[3,4]. They were manufactured in chromium-cobalt or titanium alloys and were rehabilitated by transmucous pillars that emerged in the oral cavity[5]. Although they were used for years in cases of maxilla atrophies, they were replaced by endoosseous implants designed by Branemark[6]. This was due to its complex manufacturing. It was necessary to take an impression of the residual bone ridge, which was sent to the laboratory for the structure design, with the consequent imbalances since they were not very stable models. In this way, its placement in the patient was very difficult, and several complications could appear[7,8]. However, advances in the planning and manufacturing field with various materials have allowed these structures to be made digitally with an excellent predictability and fit, thus avoiding more complex surgeries[9].

The objective of this work is to present implant-supported rehabilitation in a clinical case with severe atrophy in the upper maxilla, using a structure with subperiosteal implants, through a digital protocol, and the evolution one year after its placement.

CLINICAL CASE

We present the clinical case of a 65-year-old patient with an implant-supported dentoalveolar rehabilitation on implants in the upper arch, placed in 2010, on which he referred pain, mobility and suppuration. After an orthopantomography, we observed a severe generalised perimplantitis in the upper arch, affecting all implants (Figure 1). It was explained to the patient that it was not possible to perform a bone regeneration of the lost tissues.

Under local anaesthesia, we disassembled the dentoalveolar hybrid prosthesis. It was only fixed by two implants that showed lower mobility. Periodontal curettage was performed in all areas adjacent to the lost implants and preprosthetic surgery to favour the closure of tissues with nonresorbable suture. A provisional complete prosthesis was placed during

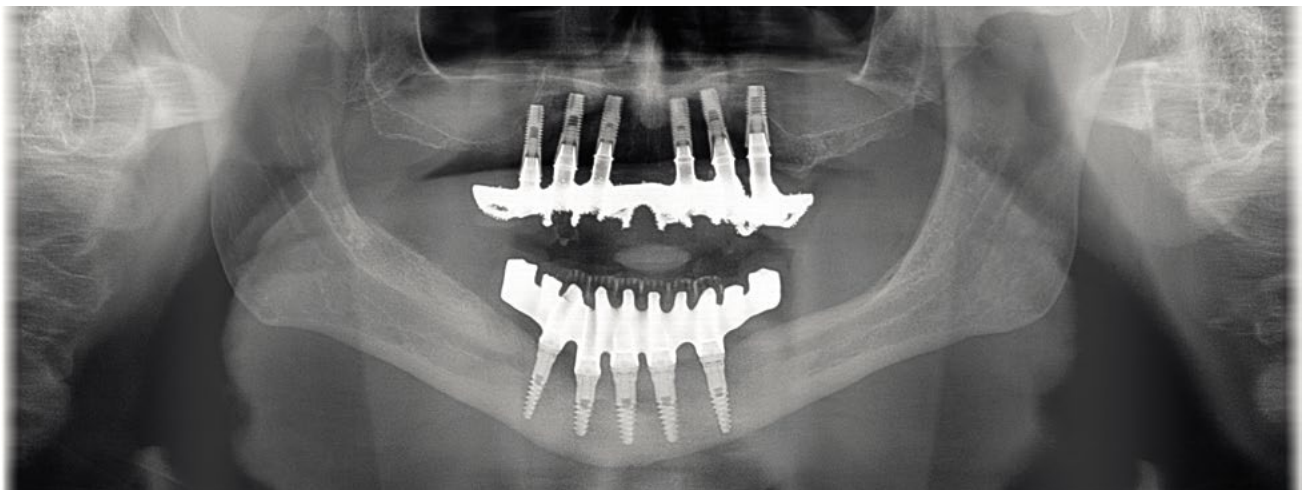

Figure 1. Initial orthopantomography.

tissue healing time. Two months later, tissue healing was complete and no clinical signs of inflammation or pain were observed (Figure 2).

Given the degree of severe bone resorption after perimplantitis, the manufacture of an SI was planned as an alternative to the reconstruction of the upper maxilla with bone grafts, bilateral sinus lifts and subsequent placement of endosseal implants or surgery with zygomatic implants. With this type of reconstructions, it is also possible to perform an immediate load on the structure at the same time of the surgery.

For this purpose, intraoral photographs were made and the complete prosthesis of the patient was used for the different planning tests. First, the double scanning technique was used, adding different radiopaque markers with gutta percha in the prosthesis[10] (Figure 3). Then, the scanning was obtained with a conical beam computerized tomography (CBCT), (Planmeca ProMax 3D, Helsinki, Finland), both the prosthesis and the patient with his prosthesis stabilized with a silicone bite registration (Figure 4). In addition, an intraoral scan of the patient's prosthesis was performed. From this, a personalized structure was designed Subperiosteal sintered in Titanium (Ti-6-4) (Custom 3D®) with 6 Multi-Unit® type connections (Branemark, Nobel Biocare) and fixed with osteosynthesis

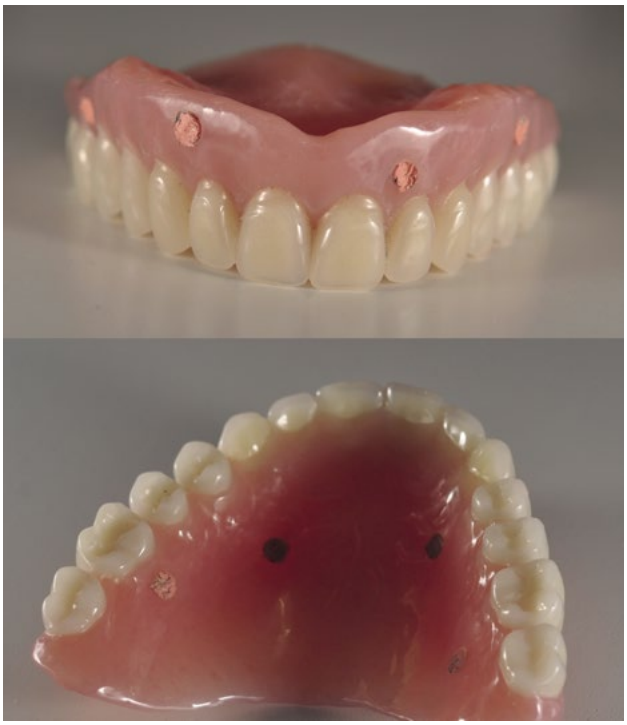

Figure 3. Immediate complete prosthesis with gutta percha markers.

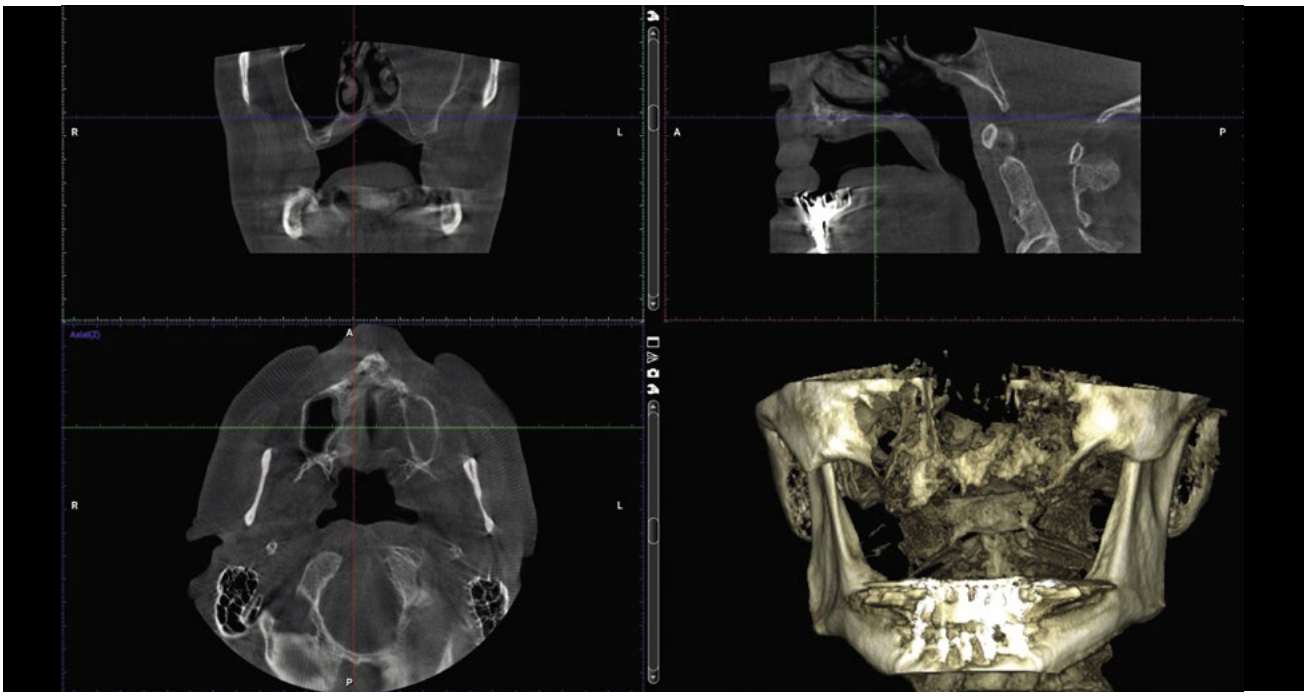

Figure 2. CBCT of the patient two months after extraction.

screws in the higher density and volume areas of the malar bone and upper jaw (Figure 5 and 6). At the same time, with the STL digital test of the planning of the mesh, and with the scanning of our complete prosthesis, the laboratory technician made an implantosupported rehabilitation milled in polymethylmethacrylate (PMMA) with Multi-Unit® type titanium interfaces for immediate loading (figures 7 and 8).

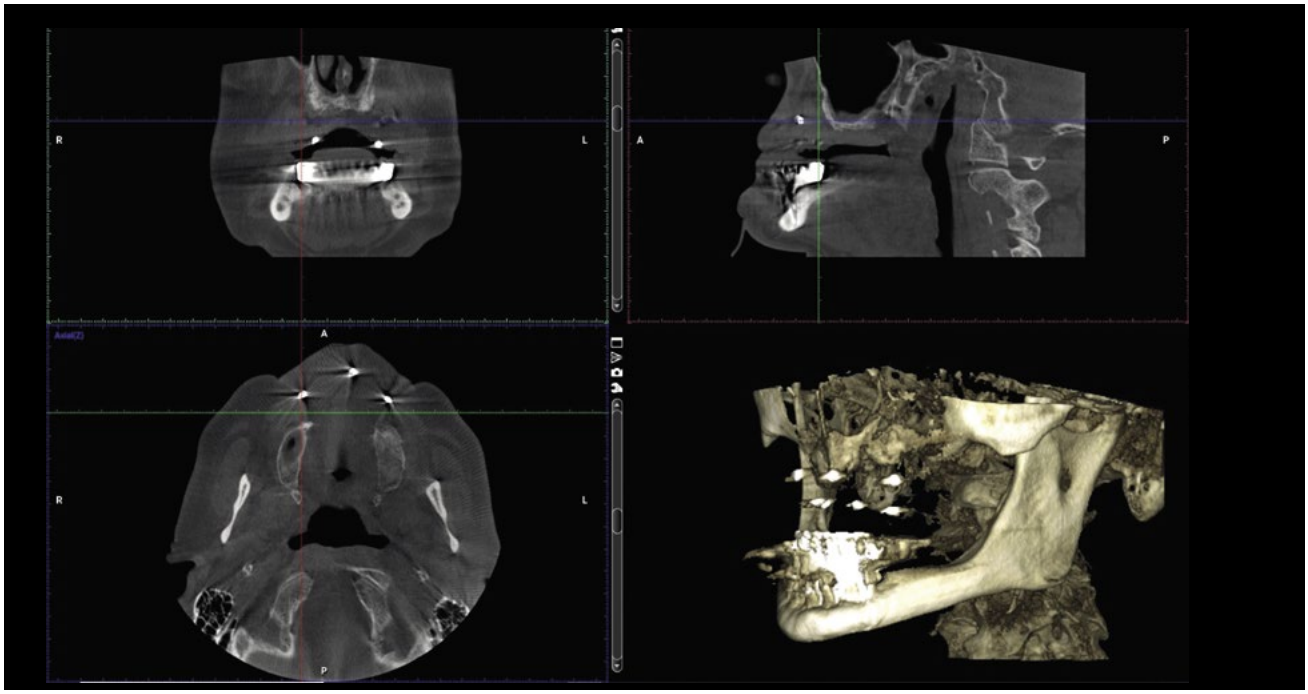

Figure 4. The patient CBCT with complete prosthesis and gutta-percha markers.

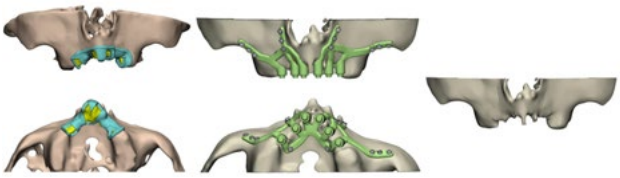

Figure 5. Digital planning of the SI design.

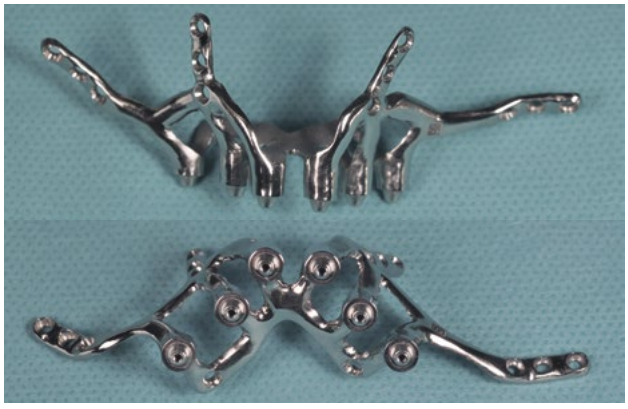

Figure 6. Subperiosteal implant.

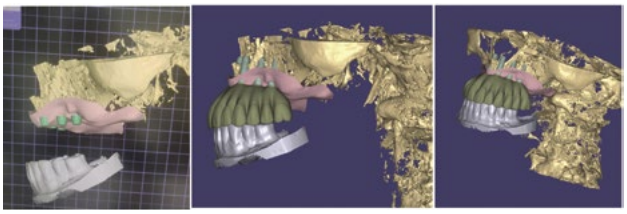

Figure 7. STL of the implant-supported immediate provisional prosthesis design.

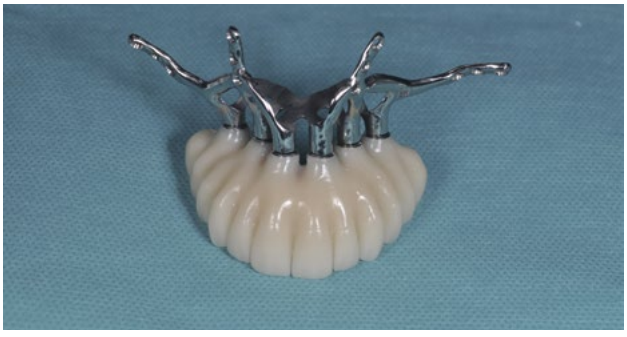

Figure 8. Immediate implant-supported provisional prosthesis manufactured in PMMA together with the SI.

The surgical procedure was performed under general anaesthesia and nasotracheal intubation. A supracrestal incision and detachment of a maxillary flap of total thickness was made (Figures 9 and 10). The boundaries of the dissection were both infraorbital ridges, both laterally malar bodies and the anterior half of the hard palate caudally. In addition, a customized cutting guide was used so that the SI was completely in direct contact with the bone (Figure 11) and the mesh was fixed with the different 1.5 mm osteosynthesis screws in the nasal and zygomatic buttresses (KLS Martin, Freiburg, Germany) (Figure 12). The closure was performed with nonresorbable suture. Finally, the PMMA provisional rehabilitation was screwed for the immediate load, with a torque of 20 N on the implants (Figures 13-15).

Two months later, with the soft tissues healed around the connections (Figure 16), a new intraoral scanning was made of the implants and the provisional prosthesis for future restoration. A FRI type passivity test (rigid impression splint) was manufactured with an aluminium structure to assess the correct fit on the implants (Figure 17). For implant-supported rehabilitation, a sintered structure was made in chrome-cobalt with machined bases covered with acrylic resin teeth from Bredent® (Figures 18-21).

The patient has been checked every six months this year, performing X-rays and cleaning the structure, without finding any prosthetic or periodontal complications.

DISCUSSION

Implantological rehabilitation in patients with severe maxillary atrophies has always been a challenge for the surgeon[11]. The progress in diagnosis and planning, the improvement in regeneration techniques and the design of materials, represent an improvement in the resolution of these complex cases. However, complications may arise in these surgeries, increasing the morbidity, time and cost of the treatment for the patient[12].

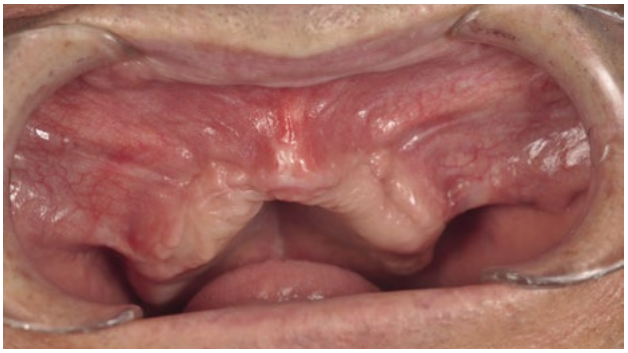

Figure 9. Upper maxilla photograph.

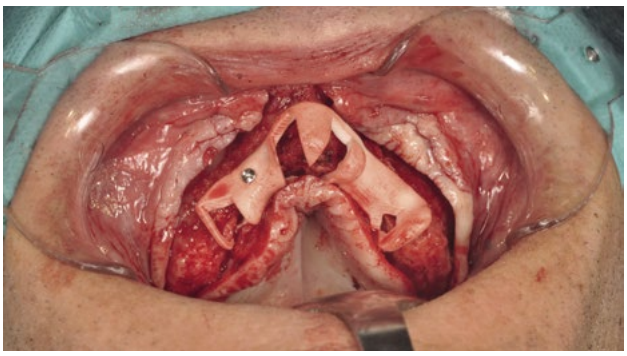

Figure 11. Placement of the surgical guide.

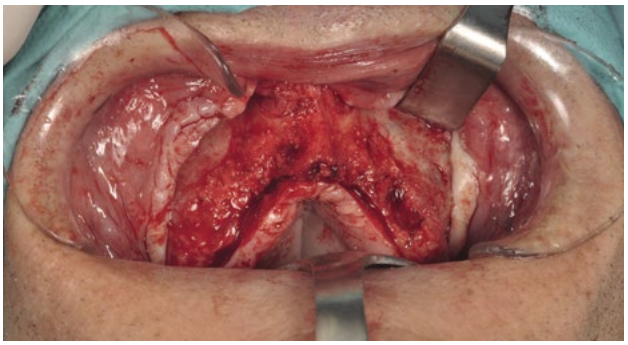

Figure 10. Incision and detachment of the upper flap.

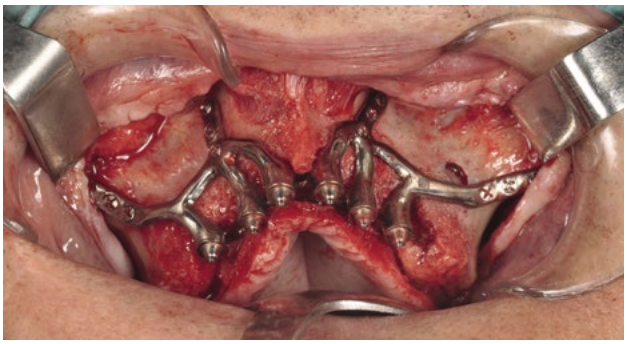

Figure 12. SI fixed with osteosynthesis screws.

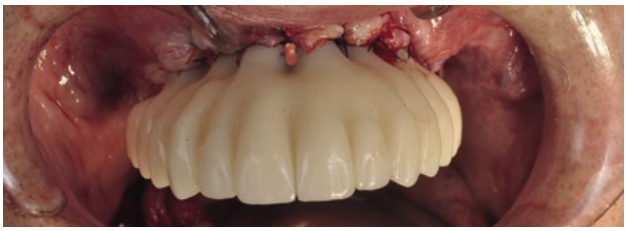

Figure 13. Flap suture and immediate upper load.

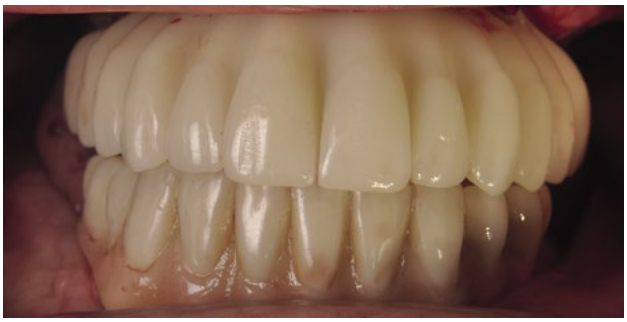

Figure 14. Intraoral photograph maximum intercuspidation with the provisional prosthesis.

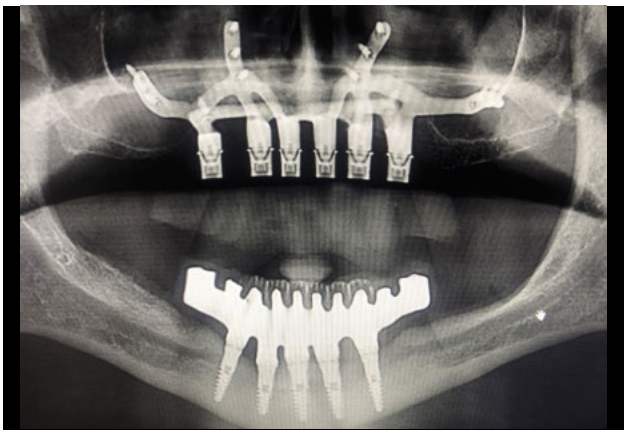

Figure 15. Orthopantomography with SI and provisional prosthesis.

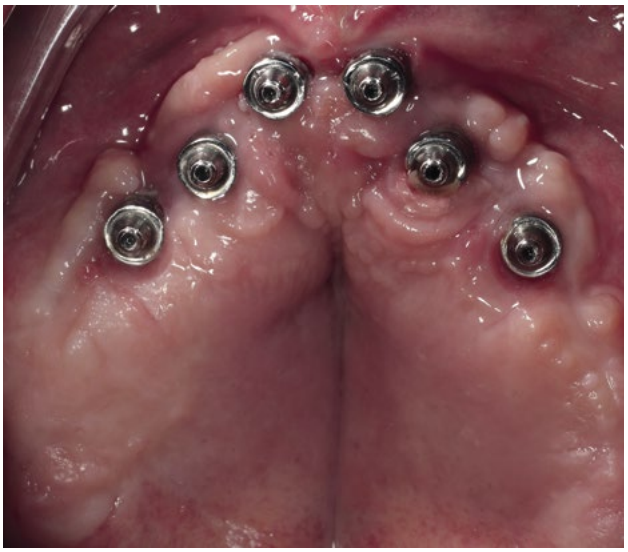

Figure 16. Upper arch with SI two months after surgery.

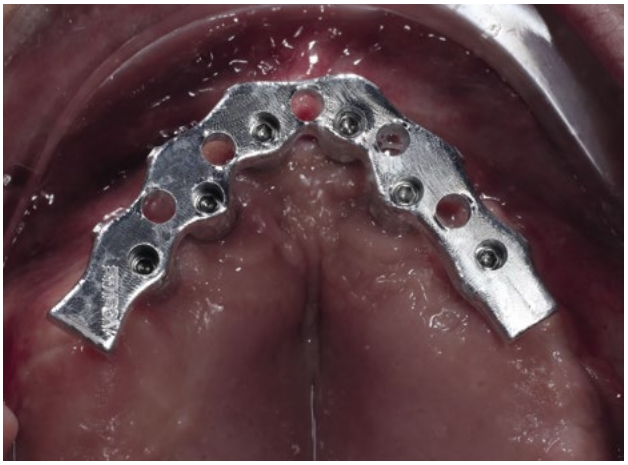

Figure 17. Rigid splint impression (RSI) manufactured in aluminium.

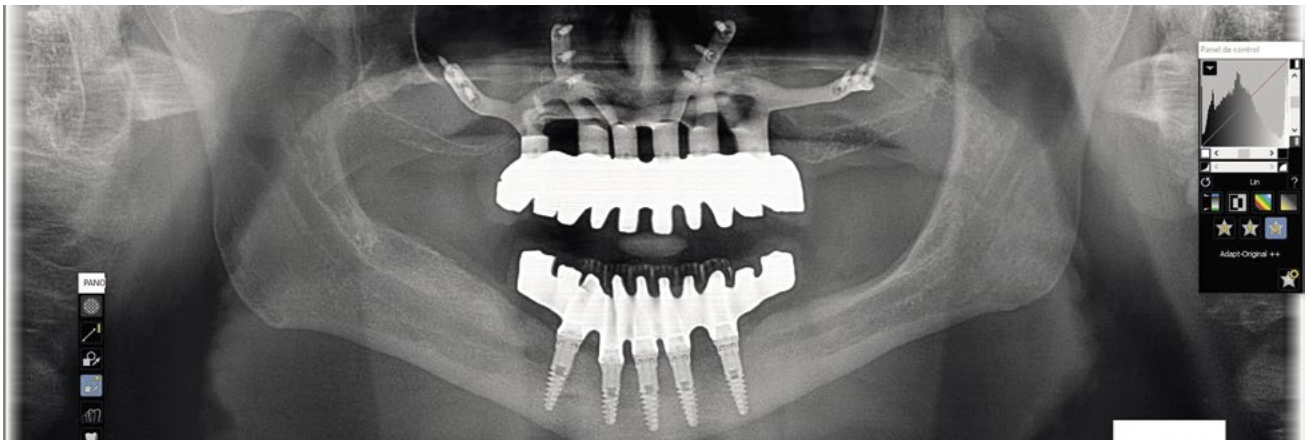

Figure 18. Orthopantomography with implantosupported final rehabilitation.

The SI were widely used in the mid-50s, until the appearance of endoosseous implants, due to the ease of placement and rehabilitation[13]. However, the new technologies in titanium additive printing and 3D planning return to SI its role as a viable therapeutic alternative in severe maxillomandibular atrophies. The use of high-quality imaging tests and 3D planning programs allow to represent a patient's residual bone volume with submillimetre accuracy. These data allow us to generate a virtual bone model, with which to adapt accurately the SI to the pre-existing anatomy[14].

In 2009, Kusek published a clinical case of a rehabilitation on SI using CAD/CAM technology for the manufacture of a sterolythographic model in epoxy resin that was subsequently sent to the laboratory for the casting of structures[15]. In recent years, the additive technique of laser sintering for the manufacture of various structures in titanium and chromium-cobalt, is the one being used to process these subperiosteal implants.

In 2016, Cohen et al. published an in vitro study on the biological behaviour of SI structures produced in Ti6Al4V, using laser sintering and post-machining on different surfaces (SL)[16]. It demonstrated a high bone-implant contact, with a vertical growth histologically and histomorphometrically demonstrated.

In 2017, Mommaerts exposes a new design for the SI, using osteosynthesis screws fixed in the maxilla and malar zones, and various transmucosal connections are housed in the structure to screw the temporary or permanent prosthesis, using a digital protocol for this[17].

The Cerea et al.[18] retrospective clinical study includes the largest number of rehabilitated patients with this technique. It was performed on 70 patients with a two year follow-up. The maxilla and jaw of these patients were partially or totally rehabilitated with subperiosteal implants manufactured on laser sintered structures and a subsequent electropolishing process, making the surfaces completely smooth. The survival rate of the implants was 95.8% and the main postsurgical complications were pain, discomfort and swelling. There was an 8.9% rate of prosthetic complications.

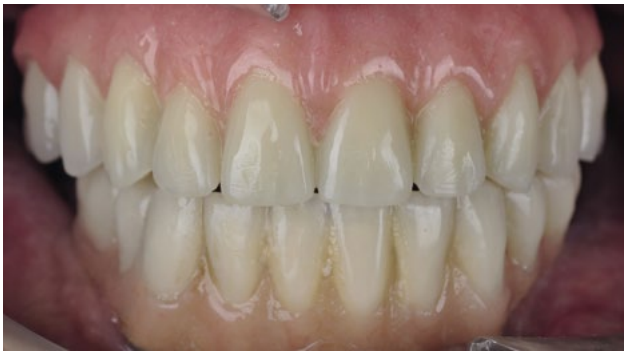

Figure 19. Intraoral photograph of the patient with the completed rehabilitation.

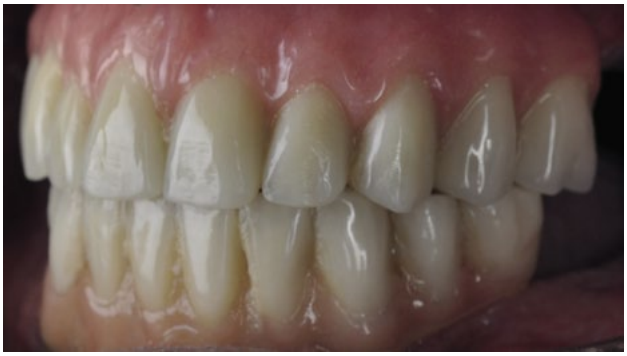

Figure 20. Lateral intraoral photography of the patient with the completed rehabilitation.

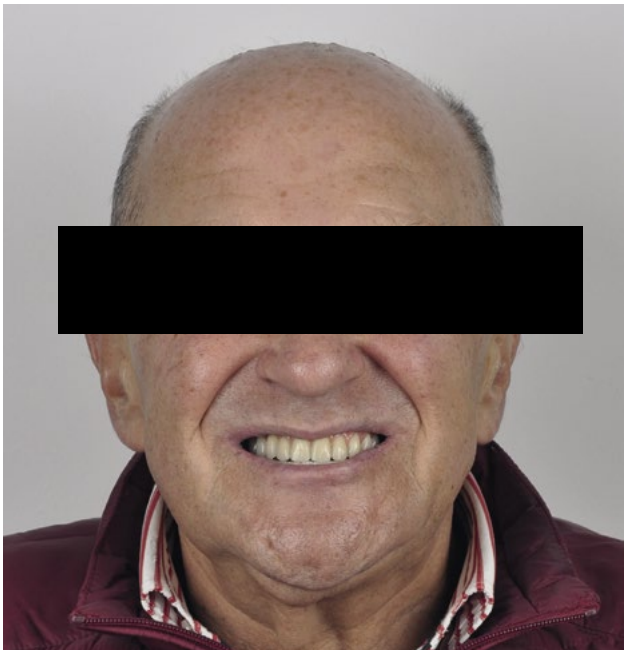

Figure 21. Extraoral photography of the patient with the completed rehabilitation.

In 2020, Mangano et al.[19] published a study of 10 patients with mandibular atrophic posterior sectors. These were rehabilitated with SI manufactured on laser sintered structures and subsequent decontamination and sterilization with organic acids. Within a year, no implants had been lost and all complications were minor.

CONCLUSIONS

Today, SI rehabilitation has improved significantly due to the great advances in digital planning and CAD/CAM. Although more studies are needed, it is an alternative tool in cases of complex surgeries with large atrophies with the possibility, in addition, to perform an immediate load.

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CLINICAL CASE

Implant explantation poorly positioned in an aesthetic sector and subsequent regeneration with block grafting. Clinical case

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SUMMARY

Performing an explantation in the aesthetic sector produces a bone defect that often leaves an area for rehabilitation with implants that must be regenerated with different procedures. Block grafting can be an alternative in cases where simultaneous vertical and horizontal bone regeneration is required. In the present clinical case, the explantation of a malpositioned implant in the aesthetic sector is shown, with an impossible rehabilitation that must be removed, subsequently regenerating the defect to be able to position a new implant, this time in a situation that allows a predictable and aesthetically satisfactory rehabilitation.

KEYWORDS

Aesthetic sector; Bone regeneration; Explantation.

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INTRODUCTION

Replacement with an immediate post-extraction implant is a very common technique for teeth located on the aesthetic front, especially the maxilla. In many cases, in order to stabilise these implants, the aim is to increase their length (apical anchorage) and the morphology or the previous defect left by the alveolus sometimes forces us to an excessive over-angulation of the implant or a too vestibularised placement, with the subsequent difficulty of an adequate prosthetic rehabilitation[1,2]. Therefore, to perform a correct planning of each of the cases in this area it should be carried out leaving for a later phase (either completely postponed after healing or during early bone healing) the placement of the implant, allowing us to correct some of these errors[1-3].

When a patient presents an implant located in the aesthetic sector with infectious problems or incorrect placement, which prevents its correct rehabilitation, the treatment plan is complicated, since the case must be started again, but in both hard and soft tissue with worse conditions that at the beginning of the treatment[4]. Therefore, having a technique that allows removing the implant with the least possible bone loss in the area is ideal, since this ensures a better starting situation for the new implant regeneration and rehabilitation[6]. The used atraumatic explantation kit (KEXIM- Biotechnology Institute®)[5-8] ensures the removal of the implant without damaging the bone tissue in which it is located, in a quick and simple way, being able to subsequently insert a new implant in the same area and surgical procedure in those cases where it is indicated.

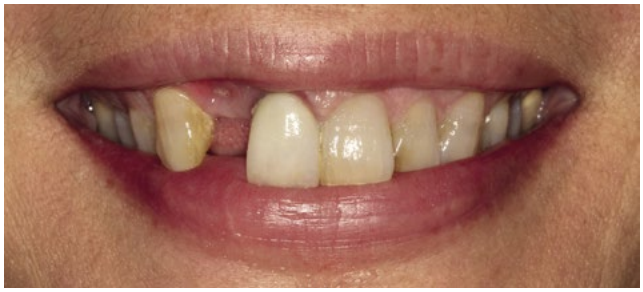
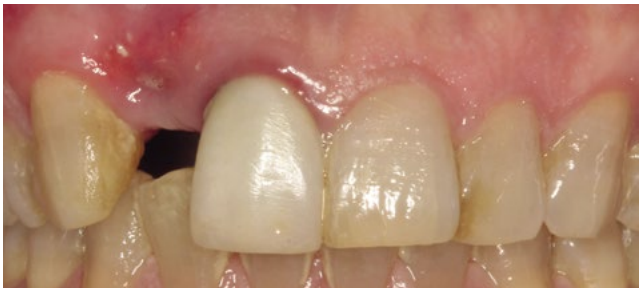
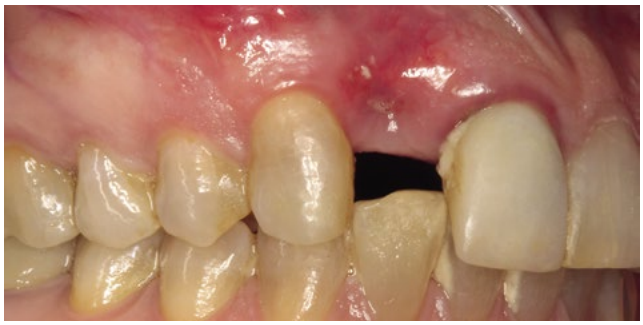
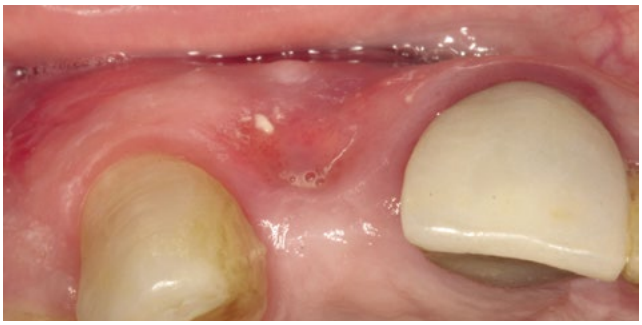
In this clinical case, there is an implant placed in an erroneous position in the anterior sector, in an area where the rehabilitation space is seriously compromised (lateral incisor zone), and where one must be as conservative as possible in the explantation to have a better chance. In the development of the case, the planning performed for its explantation, regeneration and subsequent insertion of a new implant is shown, along with its prosthetic rehabilitation and the follow-up of the case over time to verify that the stability of the treatment performed is maintained.

CLINICAL CASE

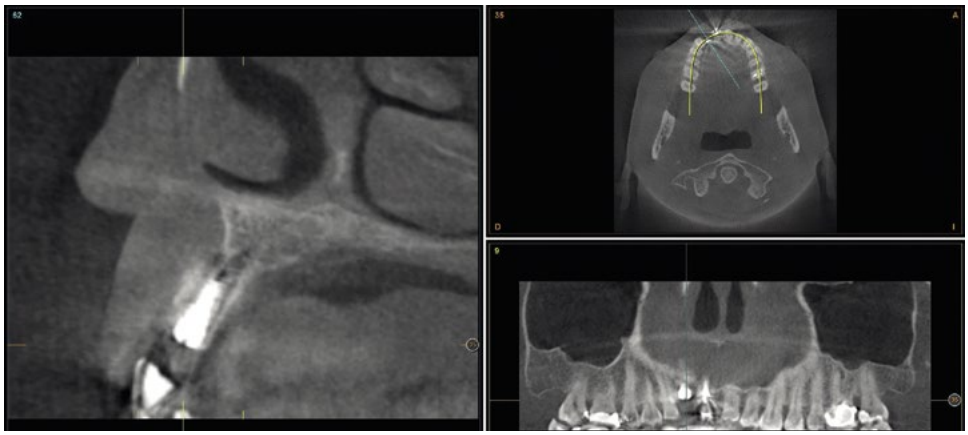
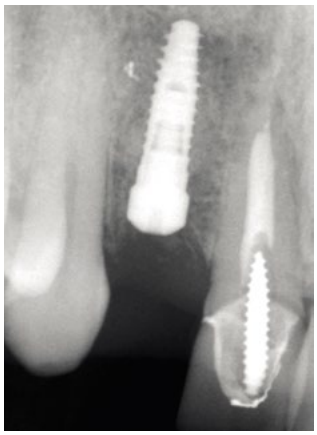
We present the case of a 36-year-old woman who comes to the clinic demanding solution for an implant located in position 1.2. This implant has carried a rehabilitation with a crown for a short period of time in which an important mucositis and loss of the thickness of the gingival tissue occurred, for which the crown was removed. The patient has a provisional removable crown and the soft tissue surrounding the area of the initial emergency of the implant-supported prosthesis is in bad condition, with a significant loss of thickness and the remains of a soft tissue fistula (Figures 1-4).

To continue with the study of the case, a Cone Beam is performed where we can observe three-dimensionally the position of the implant located in position 1.2. In the sectional cuts it is visualized completely positioned toward vestibular with an almost complete resorption of the cortical bone of this area, which explains the underlying soft tissue problems. The periapical radiography shows the position of the implant with respect to adjacent teeth (Figures 5 and 6). With this image we proceed to create a flap and the explantation of the implant. The crown located in tooth 1.1 is also removed to be able to make another crown that will serve to support the provisional extension for zone 1.2, while the first procedure heals. In this first approach a block grafting obtained from the mandibular ramus is also placed which is fixed with a microscrew in the area to be regenerated where the vestibular cortical has been lost. Once fixed and positioned, it is filled around the block grafting with particulate bone obtained with a bone-scraper of the same donor zone embedded in PRGF-Endoret for better fixation and cellular viability (Figures 7-9).

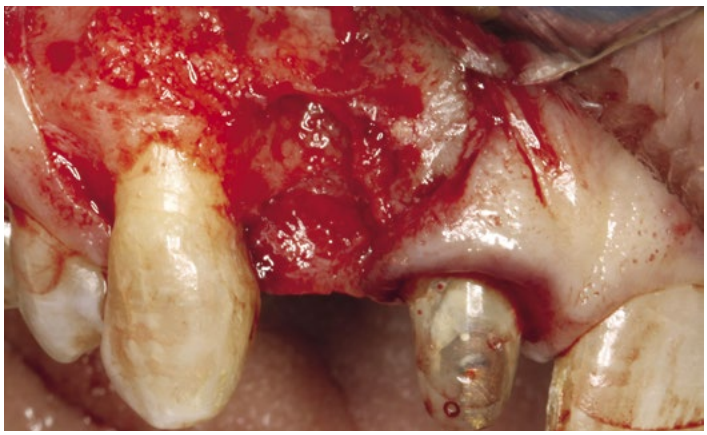
Four months later, a new dental cone-beam is performed to quantify the gain obtained in width with the regenerative procedure. It is observed in the sectional cut that a crest width of more than 7 mm and a complete regeneration of the alveolar ridge has been achieved, allowing the insertion of a new dental implant in the adequate position (Figure 10). In the surgical reentry, the information of the Cone-Beam is verified with a total integration

Figures 1-4. Appearance of the soft tissue of the implant area generating the problem, with loss of gingival thickness and an evident fistula.

Figures 5 and 6. Diagnostic image of the implant located in position 1.2




Figures 7 and 8. Explantation of the implant where the defect left by the loss of the vestibular cortical is observed.

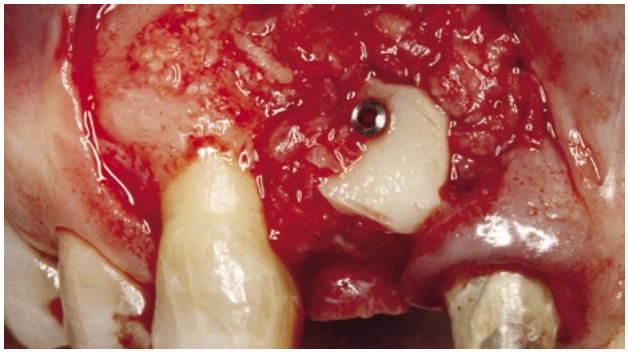


Figure 9. Image of the placement of the block and particulate graft to achieve filling the defect and obtain the 4 mm vertical growth needed for the new implant.

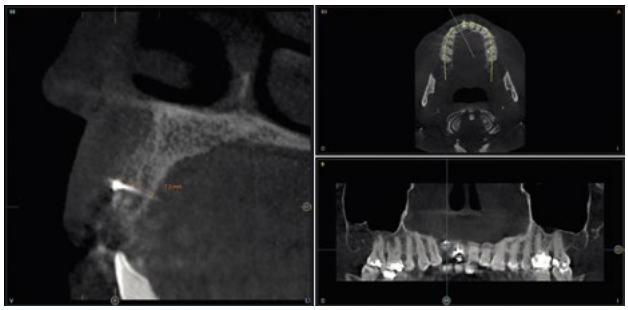
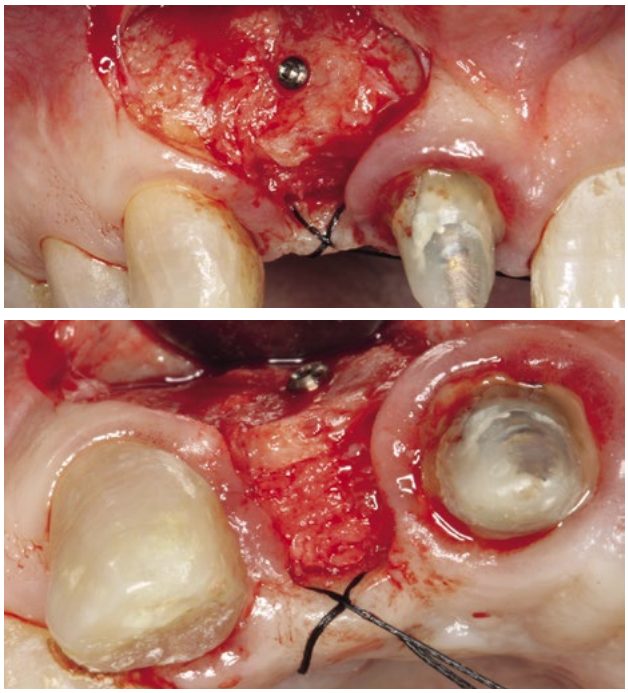


Figure 10. Cone Beam planning where a complete regeneration of the defect is observed.



Figures 11 and 12. Clinical images of the defect regeneration. It is observed how a complete integration of the block grafting has been achieved.

of the graft material, as shown in the clinical images at the time of lifting the flap for the insertion of the implant (Figures 11 and 12). The microscrew is removed and the implant is placed, which is performed by vestibular compression of the graft to gain in this manner even more contour in this area (Figure 13). The implant is left in a surgical phase, with a low healing abutment that allows a subsequent location of the same without an aggressive soft tissue surgery when the construction of the prosthesis is initiated. The provisional crown remains as an extension from the tooth 1.1.

Three months later, the prosthetic phase begins. The state of the gingival tissue is correct, although at vestibular level we would like to achieve a decrease of the zenith of the future tooth, so a connective tissue graft is planned that allows this more appropriate emergence profile conformation. The case ends with E-max crowns at the level of 1.2 and 1.1, achieving a harmonious smile and fully integrated in the rest of the smile, regarding colour, emergence and disposition of the gingival margins (Figures 14-15). The patient continues a follow up for years, maintaining the stability of the performed rehabilitation (Figure 16).

DISCUSSION

Advanced perimplantitis explantation or removal of implants that are incorrectly positioned and which prosthetic rehabilitation is not possible, complying with functional or aesthetic criteria, is a problem that we have to face in our consultations and for which tools are needed to facilitate the approach. Within the techniques for dental implants removal, there are different procedures, being those based on counter-torque that have proved to be more simple, reliable and conservative with the bone tissue, key to later rehabilitate the area with new dental implants[5-10].

The concept of being able to perform an osseodisintegration by counter-torque was introduced by different authors in the 90s, performing experiments in which different implant surfaces were tested and their abili-

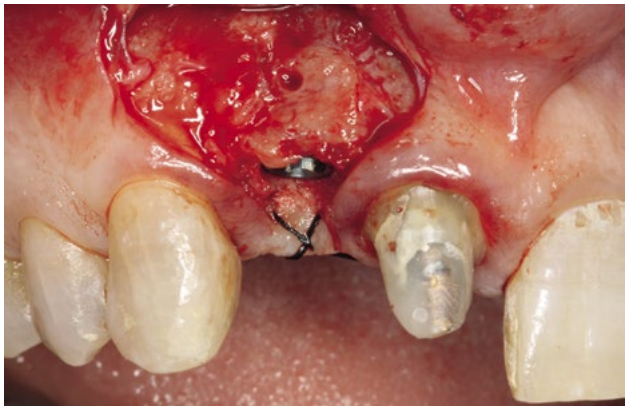
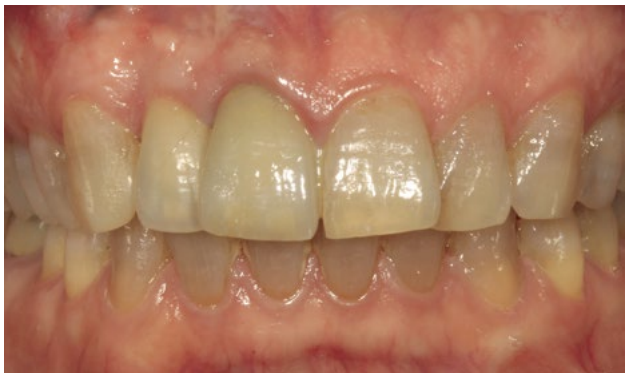
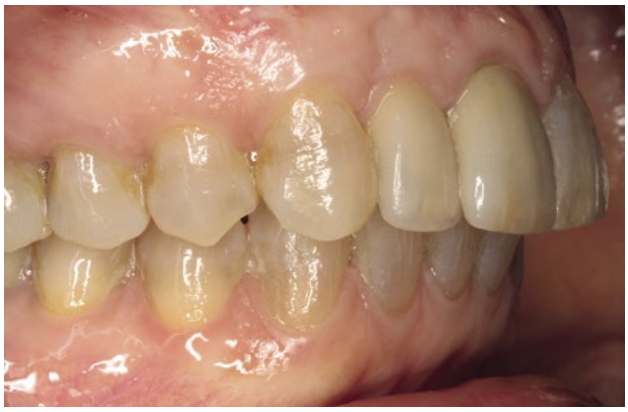


Figure 13. Juxtacrestal placement of the implant, which is used in turn as an expander moving the graft toward the vestibular area to gain a greater bone contour at this level.





Figures 14 and 15. Rehabilitation completed with the integration of pieces 1.1 and 1.2 in the rest of the aesthetic front.

ty to achieve a better osseointegration with the counter-torque removal of the implants, based on the fact that a better surface area would give higher removal values (implant removal would be more expensive)[11-16]. Later, this idea is taken up in different tests, among them, those developed by our study group to design

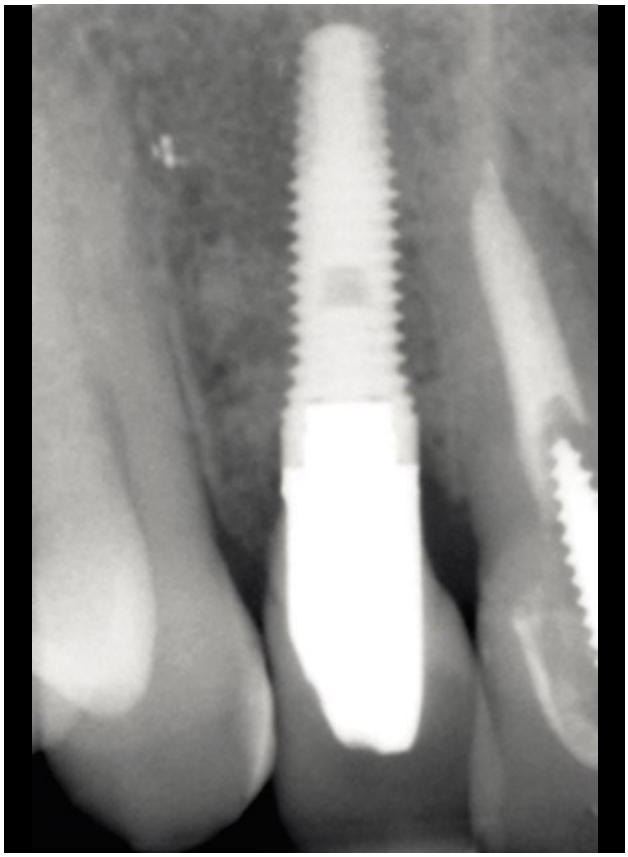


Figure 16. Follow-up radiography after 8 years, where the obtained result is maintained.

in an animal model an explantation kit based on counter-torque that is effective to remove different types of implants with different morphologies and surfaces, in the least traumatic possible manner[7].

In the case of incorrect positioned implants, sometimes this defect is due to an initial bone deficit that has caused the position of the implant to be conditioned by the residual bone volume. On other occasions, a greater defect has been generated in the bone than originally existed when the implant technique was performed incorrectly, resulting in a resorption of the alveolar bone[17,18], this resorption frequently affects the vestibular cortical, as in the present case. On numerous occasions it is necessary to rebuild the alveolar ridge in order to generate sufficient volume that allows the insertion of the implant safely and with a suitable position for its rehabilitation. When there is a total or partial loss of the vestibular cortical bone one of the most

used techniques for regeneration of the area is usually a block graft[19]. With this procedure we can regenerate the width and height of the defect creating a new absent cortical, thus completely restoring the lost bone architecture.

In cases like the one shown, of a single implant, an additional expansion can also be generated with the implant itself to correct the present defect. The insertion of the implant with expansion gives us an extra correction that makes it gain even more volume at this level. This technique of vestibular expansion of the graft once integrated, through the preparation of the alveolus in a way that generates compression, has been described by our study group with good stable and lasting results over time[20]. In this case, this procedure has also been used restoring the contour in such an aesthetically important area as the lateral incisor.

CONCLUSIONS

Implants that are incorrectly positioned in the aesthetic sector can be extracted in an atraumatic way and reshape the bone tissue that allows the insertion of a new implant in the correct position from the point of view of the posterior prosthetic rehabilitation. For this, atraumatic extractors and different regeneration techniques can be used depending on the bone defect to be restored.

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CLINICAL CASE

Integral surgical and orthodontic treatment of an autotransplanted maxillary canine: case report

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SUMMARY

Introduction: The treatment of canines included in the maxilla, is mainly based on orthodontic traction. When this fails, the extraction of the canine and the subsequent placement of a dental implant is usually considered as an alternative. Autotransplantation is proposed as a treatment option provided that the complete extraction of the canine is viable since it provides many advantages such as the capacity of being mobilized with orthodontics.

Clinical case: We describe a clinical case in which an autotransplant of an included canine was performed after traction failure in a young patient. After performing regenerative surgery, she underwent orthodontic and restorative treatment. After a follow-up of 20 months, the patient is asymptomatic, without mobility or resorption signs and with a stable periodontal status of the tooth.

Conclusion: Despite the limitations, it can be concluded that, whenever feasible, autotransplantation can be a valid alternative when orthodontic traction does not work, mainly in young patients in whom implants are not indicated.

KEYWORDS

Included canine; Dental autotransplant; Orthodontics.

INTRODUCTION

Included canines are considered those that, at their normal eruption age (13.9 years in women and 14.6 years in men), remain retained in the jaw surrounded by the pericoronary sac and with their bone tissue intact[1]. The prevalence of impacted teeth varies between 5.6 and 18.8%, being the maxillary canines up to 2% of the total impacted teeth[2].

The treatment of choice against impacted canines is orthodontic traction to guide them to their ideal position, which is not always viable due to factors such as the location of the canine, age or the refusal of the patient to undergo orthodontic treatment. Faced with this impossibility of conservative traction treatment, two treatment philosophies are presented: the expectant attitude with follow-up periods, leaving the impacted canine while it does not cause any pathology, or the extraction of the same[2].

Exodontia is usually associated with the insertion of a dental implant and its rehabilitation with an implant-supported prosthesis. But an alternative to inserting alloplastic grafts is autotransplantation, an ideal replacement of the tooth in the dental arch[2,3]. Autotransplantation is defined as the process of moving an impacted or erupted tooth from one site to another in the same individual, either to a natural alveolus or surgically created, usually with implant drills[3,4]. This technique is considered an alternative with an adequate success rate to rehabilitate function and aesthetics, especially in young patients[5].

The aim of this article is to describe a clinical case in which an autotransplantation of an impacted canine after failure of the orthodontic traction was performed in a young patient who was subsequently subjected to orthodontic and restorative treatment.

CASE PRESENTATION

Description of the case

A 13-year-old female patient, with no previous medical history, attends the Master of Dentofacial Orthopaedics at the Universidad Rey Juan Carlos (URJC) where she is diagnosed with an impacted 2.3. For this, she is sent to the Master of Oral Surgery and Implantology of the same University. A bone window is made through which the crown of the canine is exposed, to finally adhere an orthodontic button and perform traction.

After 17 months since said surgery, it is observed that the canine has not moved and it is decided to try the autotransplantation of the same.

Diagnostics

A conical beam computed tomography (CBCT) scan is performed to evaluate the exact position of the impacted canine which is located in mixed position, with the root completely formed in Nolla[6] stage 10 and Moorrees stage 6[7], in contact with the cortical of the sinus and the crown with the cusp breaking the vestibular cortical. Around the crown a radiolucency is observed that corresponds to the pericoronary sac.

Regarding the adjacent teeth, no resorption of the roots was observed in the radiological tests and the clinical examination showed positive vitality and no discomfort on percussion.

Planning

Measurements were made with the CBCT to determine the exact dimensions of the canine and the receiving area to ensure that the space was correct and was not necessary to modify it with orthodontics. In addition, anatomical relationships with adjacent structures were evaluated to avoid possible complications during surgery (Figure 1).

Surgical treatment

Local supraperiosteal anaesthesia (Articaina 4%, 1:100000 IU) was administered at the bottom of the vestibule from the lateral incisor of the first quadrant (1.2) to the first molar of the second quadrant (2.6). Both the anterior palatine nerve and the nasopalatine nerve were anaesthetized.

Intrasulcular incisions and a crestal incision were made in the edentulous space of the left upper canine (2.3). A

vestibular flap was lifted at total thickness from the first left upper molar (2.6) to the contralateral lateral incisor (Figure 2). Using a round osteotomy drill number 8 from a hand piece, a vestibular bone window was made until the whole canine crown was exposed (Figure 2). The tooth was then loosened using two elevators, without exceeding the amelocementary limit to avoid damaging the periodontal ligament fibres (Figure 2).

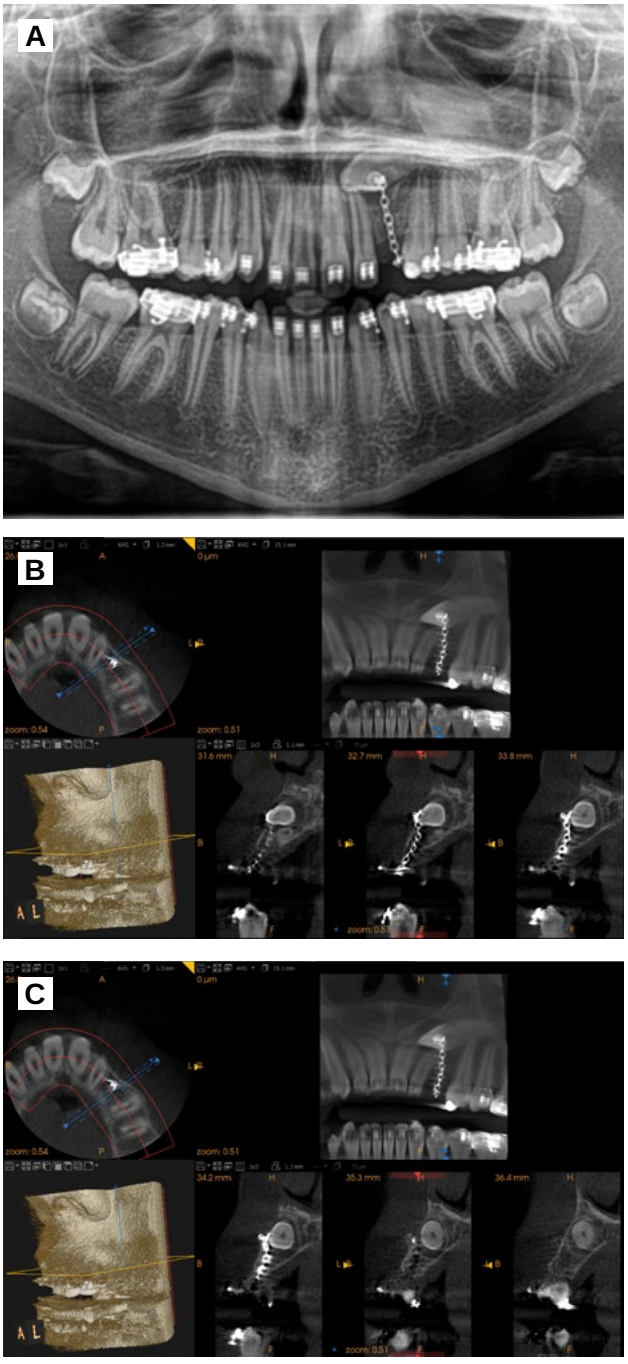

Figure 1. A. Panoramic radiography where it is observed that an orthodontic button was placed to try to pull the canine, without success. B, C. CBCT cuts prior to surgery.

The osteotomy was performed in the crestal region in order to make a surgical alveolus (Figure 2) with an anatomy similar to the future canine autotransplantation using surgical implant drills from the same business (Biomet 3i, Barcelona, Spain). A tooth exodontia was performed (Figure 2) and moved to the surgical alveolus (Figure 2). Guided bone regeneration was performed in the vestibular and palatal region of the same, both to surround the tooth in a favourable bone frame and to regenerate the vestibular defect that remained after the extraction of the canine.

For palatal regeneration, local supraperiosteal infiltrative anaesthesia was placed in the bottom of the lower molars of the 4th quadrant and an intrasulcular incision was made in the first and second right lower molar (4.6 and 4.7) that was continued with a linear incision through the mandibular ramus. The total thickness of the vestibular flap was detached and autologous particulate bone of the mandibular ramus was obtained with a bone scraper (Figure 3). Once enough autologous bone was obtained, the retromolar gap was stitched with 5/0 monofilament suture with simple stitches.

In the palatine a titanium mesh was placed fixed with two osteosynthesis screws (Stryker, Michigan, USA).

The created space was filled with particulate autologous bone mesh in order to regenerate the area of the tooth that was outside the bone (Figure 3) and the vestibular was regenerated with biomaterial (Apatos, Osteógenos, Madrid, Spain), since it was a self-contained cavity (Figure 3).

Finally, the flap was sutured with simple stitches repositioning the papillae, with a monofilament suture 5/0. The occlusion was reduced until it did not make contact with any other tooth and the canine was ferulized with the orthodontic arch itself (Figures 3 and 3) for 2 weeks.

Postoperative antibiotic (amoxicillin + clavulanic acid 875/125), anti-inflammatory (Dexketoprofen 25mg) and analgesics (paracetamol 1g) were prescribed for 7 days.

Endodontic treatment

After 2 weeks of the surgery, periapical X-rays were taken and a root canal of the canine was performed under absolute isolation (Figure 3).

The ferulization was removed and it was verified that the tooth had no mobility or percussion sensitivity.

Orthodontic treatment

After two weeks of the root canal, orthodontic movements started in order to place the canine in a correct position in the arch. For this, fixed brackets and arches were used, first of 0.16 nickel titanium (NiTi) and later of 16x4 NiTi x16.

Completion of the case and aesthetic treatment

After a follow-up of 12 months, the brackets were removed. The patient was asymptomatic and the canine did not feel mobility or any pathology signs to clinical and radiographic examination (Figures 4, 4, 4).

Finally a crown elongation was performed to level the gingival margin to the contralateral canine. A guided surgery splint was planned, based on a previous digital waxing, which marked the position of both the new gingival margin and the bone level (Figures 4, 4, 4).

Once the gum is stabilized, the aesthetic result will be improved with a composite veneer.

DISCUSSION

The prevalence of inclusion of the upper canines varies according to the literature. Zufia et al.[2] describe a 2% of the general population. Most canine inclusions occur in the maxilla, with a prevalence of 1 - 3% compared to 0.07 - 3% in the jaw[8].

The aetiology of inclusion is considered multifactorial, two thirds of the upper canines retention take place in the palate, of which, 85% have space to erupt but are impacted due to a very complex eruption trajectory or genetic factors. The remaining third are retained in the vestibular bone, in most cases due to lack of space due to maxillary compression[1-9].

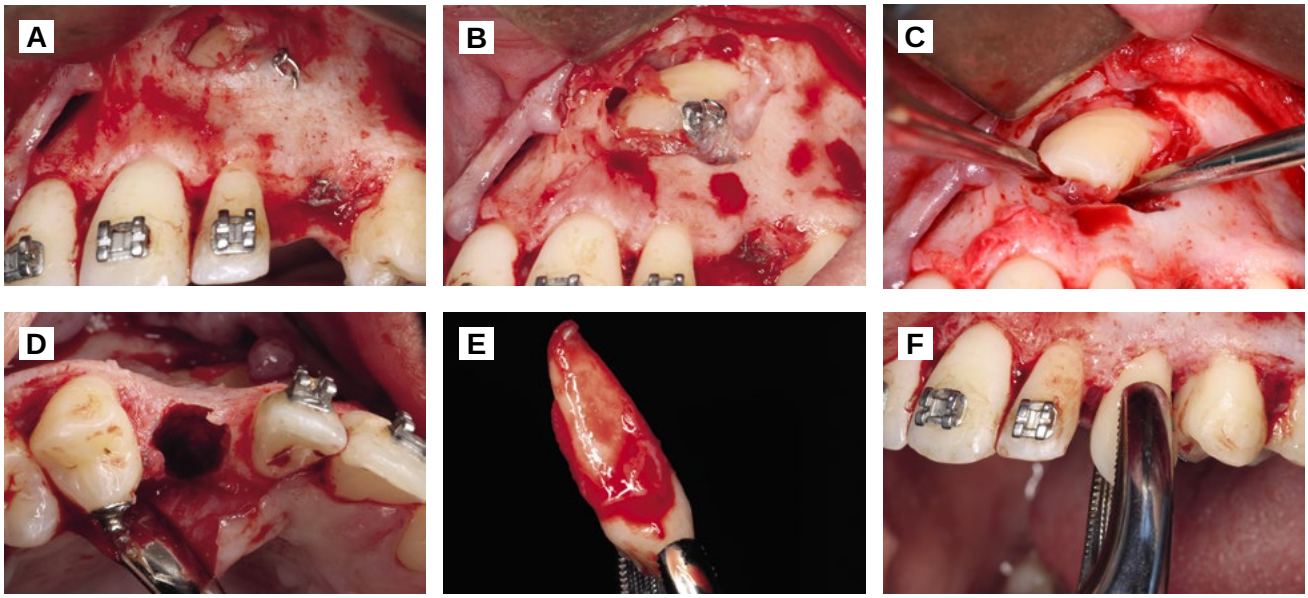

Figure 2. A. Full thickness detachment. B. Osteotomy of the clinical crown of the canine. C. Luxation of the canine. D. Creation of a new alveolus with implant drills. E. Canine extraction. F. Placement of the canine in the new alveolus.

The first treatment option in any scenario will always be orthodontic traction, either with open or closed window. It is a treatment that offers good results but is not always viable, either because of a high position of the canine or simply because of the refusal of the patient to undergo a long and expensive orthodontic treatment[2]. Davarpanah et al. mention success rates of 100% in patients up to 20 years of age, while in adults (between 20 and 47 years of age) success rates of 69.5% of the described cases[8].

When traction is not feasible, different treatment options are described, including autotransplantation of the included canine, extraction and placement of an implant[3], or even some articles break with the principles of osseointegration and describe the placement of an implant through the included canine without performing the extraction[10].

To plan a case of these characteristics, it must be taken into account that autotransplantation, unlike implants, adapts to the eruption, it can be moved with orthodontics, stimulates bone regeneration, maintains proprioception and preserves the gingival architecture of the ligament[3-11]. In addition, it delays the placement of implants, constituting an alternative if the autotransplantation does not work[12].

One of the keys to the success of autotransplantation is a healthy periodontal state, therefore, extraction should be as atraumatic as possible. Extraoral tooth time is also a key factor. Ji – Hyun et al. performed a series of 19 cases where the extraoral time was between 3 and 16 minutes, with a success of 84% of cases; to reduce the risk of complications the tooth should not exceed 18 minutes out of mouth[13]. The included canines, being teeth that have never had occlusion, have a periodontal ligament that is poorer in fibres, which is why some authors propose the application of orthodontic forces prior to autotransplantation surgery[4]. Phutinart et al. observed changes in the periodontal ligament after one, two, three and four weeks of orthodontics and found that the ligament size reached its maximum level after applying forces for one month[14]. That is why, in included canines, it is advisable to try orthodontic traction prior to surgery to increase the fibres of the periodontal ligament, since it is demonstrated that an early application of orthodontic forces increases

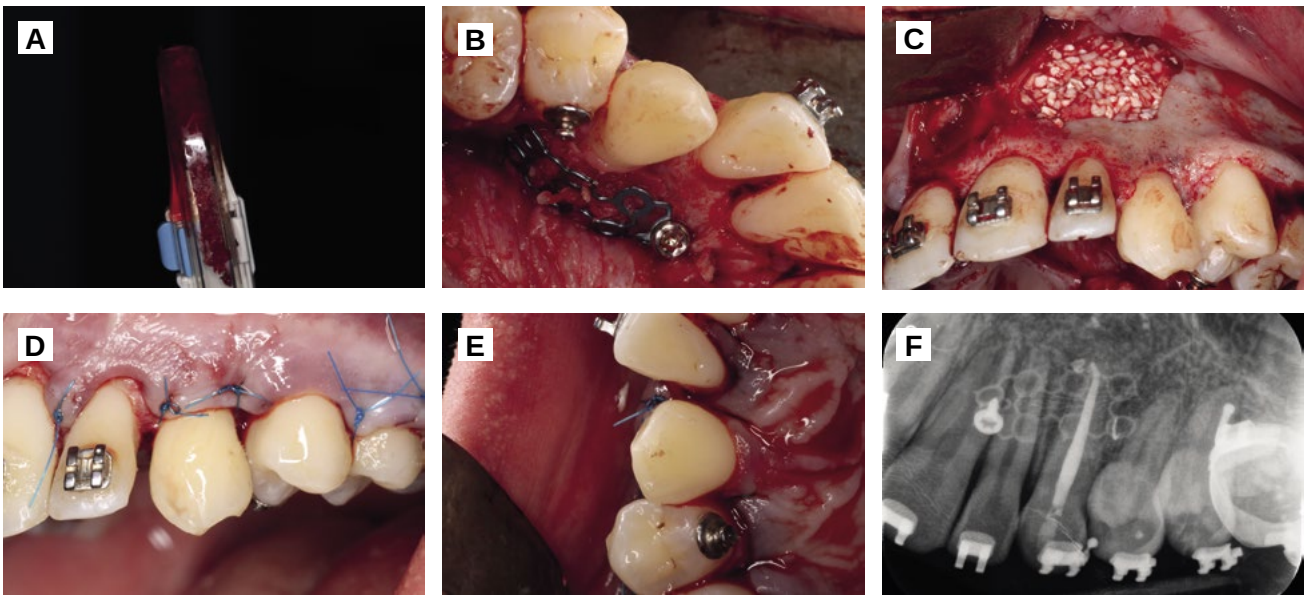

Figure 3. A. Autologous bone of the mandibular ramus. B. Placement of palatal mesh and filling of the defect with autologous bone. C. Vestibular gap filling with xenograft. D. Vestibular view of the sutured flap. E. Occlusal view. F. Post endodontic periapical radiography after 2 weeks of the surgery.

the autotransplantation success rate[15]. In addition, it can be favourable, when placing the canine in a better position to facilitate extraction and achieve occlusal space before autotransplantation[4].

Autotransplantation success rates are described at 98%[4], 90% for included canines[16] and 93% when they have been subsequently treated with orthodontics[15].

According to the studies consulted, orthodontic movements can begin to be performed between four and eight weeks after surgery, after the endodontic treatment has been completed and the ferulization removed[15]. Keep in mind that if ferulization exceeds six weeks, the ankylosis risk increases, then it will be much more difficult to move it with orthodontics[7]. Another possible complication is the resorption of the root, which occurs if during surgery the periodontal ligament of the tooth is damaged, since the formation of bone over dentin is stimulated. Resorption rates may increase if orthodontic forces are very large. Lacerda-Santos et al. indicate that resorption associated with orthodontic treatment ranges between 6 and 64%. Therefore, in autotransplanted teeth the forces applied must be minimal[17].

Autotransplants are also an option that allows combining different regenerative techniques. In the case described, it was decided to regenerate the palatal area with autologous scratched bone of the ramus since it is an area where a particulate is obtained with many morphogenetic proteins (BMPs), therefore, it increases the osteogenic capacity of the graft making it suitable to regenerate defects outside the bone[18].

The autotransplantation of included canines is, therefore, a technique that provides advantages such as greater proprioception, the possibility of moving them with orthodontics or immediacy in young patients where the placement of implants is not feasible. However, it is a sensitive technique that depends on the experience of the operator, the conservation of the periodontal ligament or the position of the canine among other factors. These disadvantages, coupled to the lack of scientific evidence from the articles in which the technique is described, make it difficult to evaluate the effectiveness of the same.

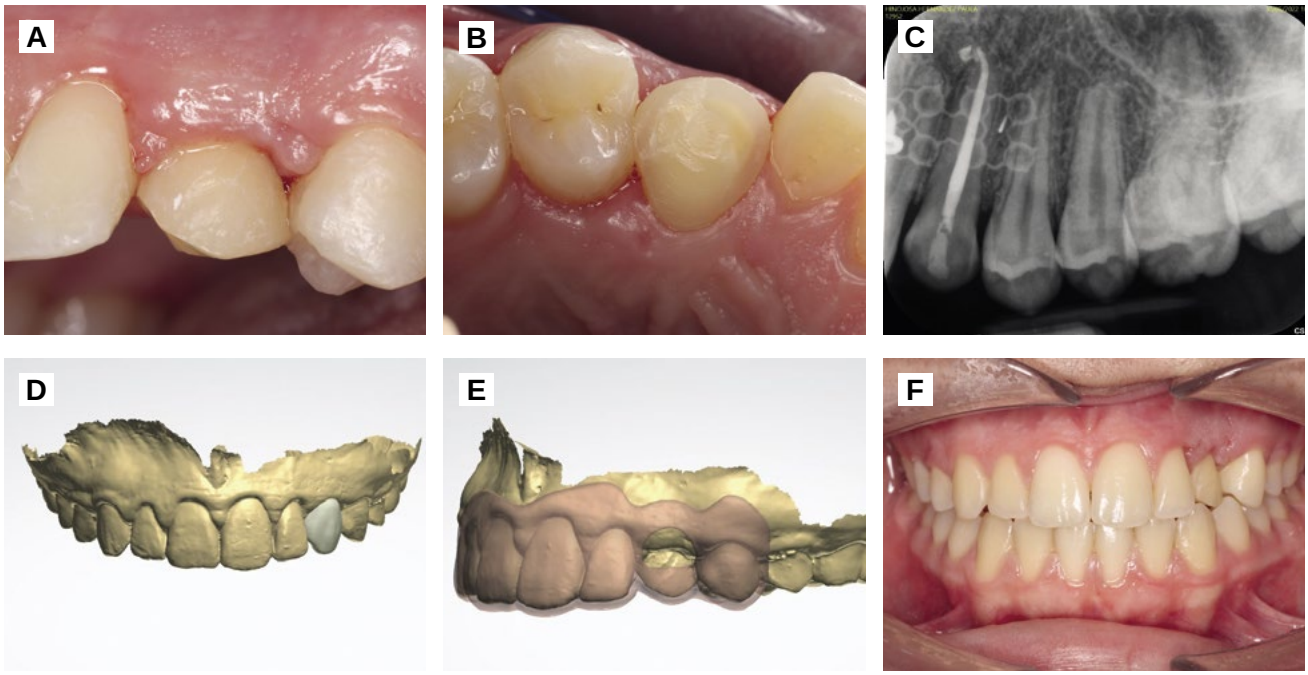

Figure 4. A. Vestibular view of the orthodontic removal day 1 year after surgery. B. Occlusal view. C Periapical radiography. D. Digital waxing. E. Digital preparation of guided splint for crown elongation. F. Post crown elongation.

CONCLUSION

Autotransplantation of included maxillary canines is an alternative to implant placement when orthodontic traction is not viable. It should be emphasized the importance of good planning, the possibility of the full extraction of the canine according to its relationship with adjacent anatomical structures and a good conservation of the periodontal ligament. It can also be combined with regeneration and orthodontic procedures whenever necessary.

More studies with more scientific evidence are needed to objectively evaluate the success rates of this technique.

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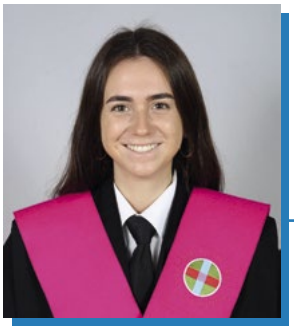

BIBLIOGRAPHIC REVIEW

Influence of non-alternating unilateral mastication in maxillofacial development and early treatment

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SUMMARY

Introduction: Non-alternating unilateral chewing is a harmful habit consisting of exclusively or predominantly chewing on one of the two sides of the dentition that, maintained over time during growth, causes an asymmetric development of the craniofacial structure affecting bone, muscle, joint and dental structures.

Objective: To describe how unilateral chewing influences maxillofacial growth and occlusion, as well as the early approach to avoid the consequences of this habit.

Material and method: A bibliographic search was made in the EBSCO-Dentistry Oral Science Source meta-search engine and in PubMed, selecting full-text articles in English and Spanish related to the subject, from which 45 were extracted to make this review.

Results: Non-alternating unilateral mastication generates the mandibular ramus of the non-masticatory side to undergo a greater growth because the condyle of this side only performs translational movements with an enormous growth stimulus. In addition, the greater load in the condyle of the chewing side generates anatomical changes, the neck being thickened and the head area increased. These changes at mandibular level produce unilateral posterior crossbite on the mastication side with a class II subdivision.

Conclusions: The hyperfunction of the mastication muscles and the vertical mandibular movement on the chewing side, as well as the eminently translational trajectory on the steady swinging side, generates asymmetric growth of the jaw and TMJ, deviation of chin and inferior dental midline to the working side, unilateral posterior crossbite and class II subdivision on the mastication side, among other alterations. Therapeutics in early stages consists of function rehabilitation, applying composite tracks on the cross side and maxilla expansion.

KEYWORDS

Unilateral mastication; mandibular asymmetry; unilateral crossbite; malocclusion.

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INTRODUCTION

The growth and development of the craniofacial structure is the result of the interaction between genetic and environmental factors where there is an increase in size, remodelling and displacement of structures1. They are morphogenic processes aimed at a state of functional and structural equilibrium between all the regional parts of the hard and soft tissue in growth and development. To achieve a physiological occlusion, the jaw will assume a greater growth in length than the maxilla[1,2]. The bones grow by the apposition of new bone tissue on one side of the cortical and resorption in the opposite area. This compound process is called drift and creates a direct growth movement of any given bone area.

The genetic and functional determinants of bone growth lie in the set of soft tissues (muscles, tongue, lips, cheeks, tonsils, adenoids...) that activate, deactivate, accelerate and delay the histogenic actions of osteogenic connective tissues[1]. During this growth, physiological habits (speech, normal swallowing and chewing) are stimuli for the growth of these structures. However, there are a number of harmful habits such as digital suction, onychophagia, oral breathing or lingual interposition that can interfere in maxilla and mandibular development and be part of the aetiology of malocclusions[3].

According to Proffit's theory of equilibrium, intense and intermittent functional forces are resisted by physiology, while light and continuous postural forces lead to adaptive mechanisms that produce biological remodelling[4]. These parafunctional oral habits of the stomatognathic system modify the position of the teeth and the relationship between them, as well as normal growth and function of the orofacial musculature, producing an imbalance between internal and external muscle forces[5,6]. Early diagnosis of abnormal habits is crucial for the prevention or early correction of malocclusions that may develop[6].

A basic postulate of functional cranial analysis is that the structure of the head and neck is organized operatively in terms of function: digestion, vision, olfactory sense, speech, etc.[7]. According to the Moss functional matrices hypothesis, each of the functions is performed by a functional cranial set. These components consist of two parts: a functional matrix that performs the function and a skeletal unit whose biomechanical role is to protect and/or support its specific functional matrix. Skeletal units are those formed by bone, cartilage or tendon tissues. Functional matrices include muscles, glands, nerves, vessels, fat, and teeth[8].

Unilateral chewing occurs when chewing is performed constantly or predominantly by one of the two sides of dentition. Both the jaw and the condyle modify their shape and size if the habit is maintained during development[9,10]. The objective of this bibliographical review is to elucidate the changes that can be produced by non-alternating unilateral mastication during development in the different structures of the craniofacial structure, as well as determine the causes for which this habit develops and describe the early approach during growth to prevent these alterations.

MATERIAL AND METHODS

To perform this bibliographical review, two searches were carried out. The first was carried out in the Discovery Service (EBSCO) metasearch engine, specifically in the section: Dentistry & Oral Science Source. The boolean operators "OR" and "AND" were used and the following search terms were introduced: "unilateral chewing", "mandibular asymmetry", "unilateral posterior crossbite", "treatment" and "causes". 175 articles were found. The search was then filtered with the following inclusion and exclusion criteria:

Inclusion criteria:

- Articles in Spanish or English.
- Full text.
- Academic publications.

Exclusions criteria:

- Articles in a language other than Spanish or English.

The result was reduced to 142 articles.

The second search was carried out in the PubMed search engine using the same boolean operators and the same terms as in the first search. A result of 144 articles was obtained. The search was then filtered using the same inclusion and exclusion criteria as above. The result was reduced to 97 articles. Given the low volume of publications, all articles were considered without publication date limitation.

Then, we proceeded to reading the titles in both information resources discarding those articles which were not in line with the topics to be addressed in this bibliographical review. Among the publications obtained from Discovery Service (EBSCO) and PubMed, 92 articles were selected. Of these 92 publications, 17 were repeated. This article selecting process relevant to this study is presented in the following flowchart (Figure 1):

We proceeded to read the 75 articles and we discarded 30 in which no outstanding information was found for the development of the work. 45 were used to make this bibliographical review. In addition, two reference books, three web pages publications and six articles with relevant information from other sources were included.

RESULTS

All growth changes in size, shape, spatial position and maintenance of all skeletal units are always secondary to the primary temporal changes in their specific functional matrices. Noticeable changes are perceived in the jaw when the mastication temporalis, masseter and internal pterygoid muscles increase their function and cross-sectional area[1,8].

When chewing is performed with predominantly vertical movements, as in the case of non-alternating unilateral mastication, the dominant muscles are the masseter, external pterygoid and anterior fascicle of the temporalis, generating a hinge movement on the masticatory side. The articular eminence of the temporal is accentuated because there is no condylar translation[11].

The neck and the mandibular condylar show traces of the reaction against major masticatory loads, especially of asymmetric type as in this type of chewing. To face these loads, the neck of the condyle thickens and the mandibular condylar area increases significantly[1]. The condyle on the chewing side performs only a rotational movement, so it lacks a growth response. In addition, it suffers an excessive burden resulting in

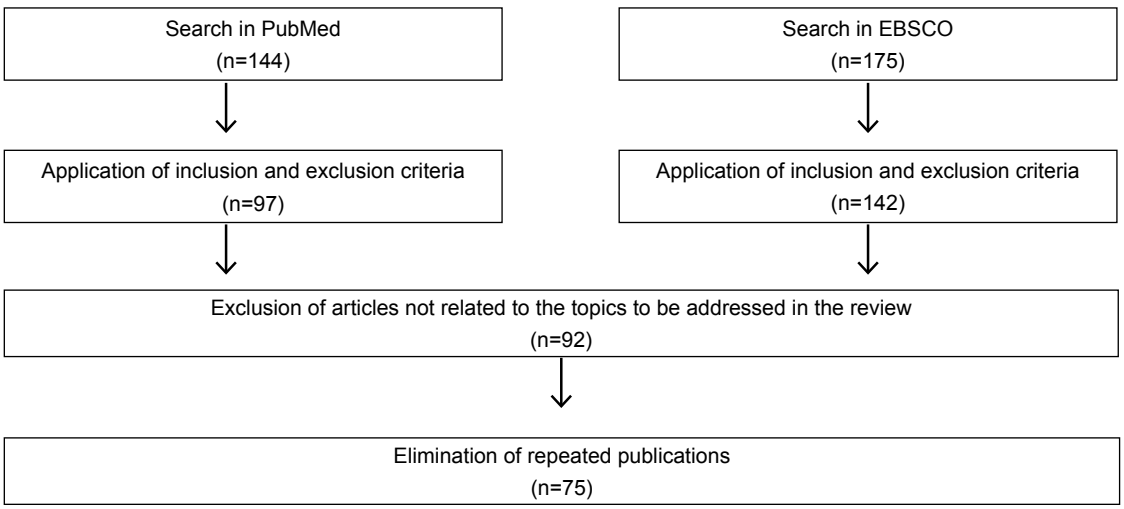

Figure 1. Flowchart representing the article selection process.

anatomical and structural changes in the TMJ. On the opposite side, the condyle performs only translational movement, causing a growth response with elongation of the neck and mandibular ramus and remodelling of the condyle-disc structure[2].

According to Planas' "Anteroposterior and Transverse Growth Law", the anteroposterior excitation of the TMJ of the swinging side produces as response the growth in length of the mandibular ramus of this side. However, on the working side the functional occlusal rub produces a mandibular thickening and expansion, thus producing a mandibular asymmetry[12].

To analyse this condition, a combined clinical analysis is necessary with front and side photographs, lateral and anteroposterior cephalogram, oblique mandibular X-rays at 45° and panoramic X-rays. The clinical analysis includes the electromyographic study, observing the location of the bolus in the oral cavity and the mandibular movement in the closing phase of the mastication[13,14]. In addition, when analysing the vertical dimension in these patients, it will be observed that, during the right to left lateral excursions, the vertical dimension is smaller on the chewing side. These measurements can be recorded in the front plane when performing the lateralities, the "Planas' functional masticatory angle"[11] being defined.

There are several types of foods that have been used as a test to determine the preferred chewing side in patients. In most studies chewing gum is used, but carrots and almonds have also been used. Other products such as silicone tablets can be used. The size, hardness and texture of the bolus influences the masticatory cycle and its muscle activity[15].

In addition, other evaluations can be performed such as video recording, kinesiography or electromyography[16].

Non-alternating unilateral mastication can occur for several reasons: mediation of the Central Nervous System and relationship with other laterality preferences such as being right or left-handed, peripheral factors such as avoiding one side because it produces pain or preference on one side for greater chewing efficiency. It may also be due to dental factors, size and type of ingested food, and the number and duration of chewing cycles until swallowing[17].

According to Larato, the teeth of the side that does not chew accumulate more dental calculus and plaque in their crowns and roots and as a consequence produce bone loss. This side presents a higher height of the canines, resulting in interference instead of acting as a canine guide. As a consequence, a worse occlusion adjustment occurs, making the patient chew with the easier half arch[18,19].

Patients with facial asymmetries not only present this deformity on the outer surface, but also in the internal structure as to the shape of the dental arch and the oral or lingual pressure, when comparing the two half arches. A deviation of the lower midline can be observed along with the chin toward the mastication side with a displacement of the lower interincisive point, being common the appearance of a crossbite. A class II subdivision occurs on the chewing side due to mandibular displacement toward that side[2,20].

As it is known, posterior crossbite is a transversal malocclusion in which the palatal cusps of one or several superior teeth do not occlude in the central fossa of their antagonists and it is the vestibular cusps that do so. It occurs on the side where mastication predominates, where the mandibular ramus is shorter[13,21]. On the crossbite side -the side used to chew- a hypertrophy of the masseter occurs due to the excessive use. These patients appear to have a dolichofacial pattern in half of their face on -the side without chewing-, while in the other half of the face they appear to have a brachyfacial pattern[22].

It has been suggested that an altered morphological relationship between the upper and lower arch is associated with differences in the condylar fossa relationship between the right and left side[23]. Any association between a temporomandibular disorder and a malocclusion, such as posterior crossbite, indicates the need for early orthodontic treatment to avoid future problems with joints and masticatory muscles[24].

It is very important to make a good anamnesis to know the patient's history, looking for possible factors that trigger and favour this type of habit[4]. Elimination of the habit and early treatment are necessary, since malocclusions with a skeletal component tend to worsen over time. The prognosis of malocclusions is aggravated if the start of treatment is delayed or if inappropriate treatment is applied: the imbalance between form and function increases[22].

Asymmetric malocclusions are often complicated to correct, especially when there is an underlying skeletal component. The optimal treatment for this condition will depend on its severity and the age of the patient[22,25,26]. According to Proffit, when the patient is growing it is possible to try to control and modify this asymmetric growth[26]. A first phase of orthopaedic treatment is allowed to avoid orthognathic surgery when growth ends. In adults, however, skeletal asymmetries are usually treated by combining orthognathic surgery and orthodontics[25].

In addition to correcting the crossbite, the professional should also concentrate on rehabilitating the function. If the correction were to focus solely on the form, uncrossing the bite, and the patient continues to chew on the corrected side, the malocclusion would tend to recur[2]. To stimulate the function, the patient must chew gum on the side that does not have the crossbite to stabilize the correction. This will progressively improve the masticatory function and mandibular rest position[22].

According to Plans' minimum vertical dimension law, the side with the masticatory preference will have the lowest canine guide, minor posterior disocclusion and will present a minor Masticatory Functional Angle. Therefore, it will be necessary to perform selective reduction and apply composite tracks[2,12]. These tracks are composite resin aggregates that act as inclined planes to position the jaw and achieve a different intermaxillary relationship. The technique tries to add composite to the canine on the cross side to increase the canine guide on that side and make chewing difficult. The tracks can also be built on the occlusal faces of molars from lingual to palatine on the side of the crossbite. They are a very good option, since they do not need the patient to be collaborative and they remain active 24 hours a day and the 7 days of the week[2,27]. They are constructed in such a way that, when performing lateral movements, the Planas' Functional Masticatory Angle is smaller on the non-crossed side to functionalize it. If necessary, selective reductions could be made so that the vertical dimension remains smaller on the non-crossed side. In this way we try to change the functional side[12].

If selective reductions alone are not effective, an upper removable expansion plate can be used at an early age to expand the maxilla, thus reducing the risk of lengthening the posterior crossbite[28,29]. According to Del Pinal et al., the most used device for slow expansion is the Quad-Helix. It performs a symmetrical expansion of the arch and an increase of its vertical dimension through reciprocal forces on the teeth. In 75% of the cases it produces a discreet opening of the palatal suture. In late ages such as 10 years old, it produces effects only at the dentoalveolar level[27].

DISCUSSION

There are several causes that can make an individual to establish a non-alternating unilateral chewing as a usual masticatory pattern in their day to day. When analysing with visual control and T-Scan the chewing of 100 people, 50 with unilateral non-alternating right mastication and 50 with unilateral non-alternating left mastication, it was concluded that the participants preferred one side or another depending on the contact area between the teeth, being greater on the preference side. Other studies that use wax for the analysis of this condition report this. Haralur et al. state that wax presents poor dimensional stability, so it is not possible to analyse small occlusal contacts. The T-Scan sensors are 98 nanometres thick, while the wax is 0.5 to 0.75 mm[30].

Non-alternating unilateral mastication can also be influenced by other parameters such as laterality contacts, occlusion, cuspidal shape, posterior teeth absen-

ce, interference on the working side and the size and consistency of the ingested food. In addition, oral sensorimotor systems and pulp nociception, periodontal and articular tissue are also related to mastication behaviour[31-33]. Pond et al. state that these occlusal factors are influential when the mastication pattern is developing in the child, but when this pattern is established, only painful stimulation can change it[34].

In their study, Nissan et al. compared the preference of the mastication side with the preference in the use of the feet, hands, eyes and ears to see if it is another hemispheric lateralization such as these last conditions, this relationship being positive[31]. However, Wilding et al. state in their work that some studies reject this and that, while the preference for using mostly one of the hands, eyes or ears is centrally controlled, the preference for one mastication side is determined by peripheral factors such as masticatory efficiency or comfort[35]. The first impulse to bite and chew a food is a voluntary act, but subsequently becomes an involuntary act mediated by central and peripheral neural mechanisms[33].

According to the Moss' functional matrix hypothesis, the growth of a bone and its changing position in space are related to the growth of the muscle that is inserted into it[36]. This is reinforced by Mew, who states that changes in mandibular growth are effected by the cells of this bone, which act in response to positional information they receive from the tissues around the jaw.

It has been shown that the formation, growth, size, shape, spatial position and maintenance of the mandibular angular process are always secondary to the functional demands of the masseter and medial pterygoid muscles[9,28,29]. In the analysis of a corpse performed by Rogers, it was observed that the right masseter was one fifth the size of the well developed left masseter. The left temporal was medium in size, while the right was completely absent. In addition, the pterygoid muscles on the right side had a cross-sectional area of about one-quarter of the left cross-sectional area. When studying the skull, he observed the complete loss of the alveolar crest on the side with well-developed muscles (left) compared to the very sharp alveolar crest on the atrophied side. This led him to conclude that the individual predominantly performed the mastication on the left side[37].

The areas of muscle insertion in the mandibular ramus play an important role in the local remodelling and in the cortical displacement that accompanies the mandibular displacement downward and forward. If the masticatory activity of the muscles is asymmetric, the remodelling process will be altered causing a structural change. The strength of the masseter on the mastication side is transmitted to the jaw, which develops a greater trabecular bone on this side due to the higher mechanical requirement[11]. In experimental studies performed by Legrell, a shorter branch on the mastication side is observed with a compensation in bone growth at the mandibular base level and the goniac region[38,39].

Augusto et al., when analysing a skull whose mastication was predominant on the left side, observed several consequences in its development. On the mastication side, the jaw was anteroposterior shorter, higher and with more volume and the upper maxilla wider transversely and anteroposterior. On the opposite mastication side, in anteroposterior direction, the jaw was longer and the maxilla was shorter and less developed (Figure 2)[40].

The factor that determines the size of the dental arches are the facial muscles, the crowding or the space between the teeth. The bones have a secondary role[41].

Regarding the temporomandibular disorders, there is no clear consensus on the association between non-alternating unilateral mastication and temporomandibular disorders. Some epidemiological studies have shown that people with non-alternating unilateral mastication have a higher probability of developing temporomandibular disorders. This habit excessively loads the TMJ on the preferred mastication side with respect to the contralateral side, producing anatomical and structural changes in the cartilage, the glenoid cavity and the condyle. The significant statistical relationship between the mastication side and temporomandibular disorder symptoms is inherent[42,43]. However, in a study performed with a CBCT scan to compare the condylar

position between patients with habit and without habit, no differences were reported between them[42].

Santana-Mora et al. confirm that chronic unilateral temporomandibular disorders mainly affect the usual mastication side, with a higher condylar path and flatter anterior lateral guide angles. This allows us to conclude the hypothesis that the preferred mastication side may be a factor that leads to the development of temporomandibular disorders and a remodelling of the masticatory apparatus[44]. On the other hand, a study performed at the Dentistry Faculty of the University of Chile that analysed different types of temporomandibular disorders concluded that there were no significant differences in the presence of temporomandibular pathology between unilateral mastication patients and alternating unilateral masticating patients[14].

In a study evaluating the bone morphology of the TMJ in patients with alternating and non-alternating unilateral mastication, significant differences were observed in the joint space, joint fossa depth, the width of the condylar neck and the inclination of the articular eminence -which is more pronounced- among the opposite TMJ, increasing the potential risk of developing a temporomandibular disorder. An asymmetry of the condylar trajectory is produced as an adaptive mechanism caused by the predominant use of a side[45,46].

The relationship between the type of mastication and the presence of temporomandibular disorders is not clear, despite a high tendency of joint pathology in unilateral mastication patients[14].

Regarding mandibular asymmetry and unilateral posterior crossbite that generates this habit, it has been concluded that patients with this transversal relationship present an alteration in the glenoid-disc-condyle fossa relationship also with an asymmetric skeletal growth. With an increase in the thickness of the cartilage of the contralateral side and a decrease in the mastication side. In addition, they frequently show an anterior disc displacement in the TMJ of the deviated side due to the constant tension and stress on the back of the disc[23,47,48]. Pullinger et al. confirmed that there is an association between unilateral posterior crossbite and some signs and symptoms of temporomandibular disorder such as joint pain, clicks, muscle tension, or headaches[49].

Regarding the treatment of this condition, Petrén et al. confirm that the treatment of choice for the correction

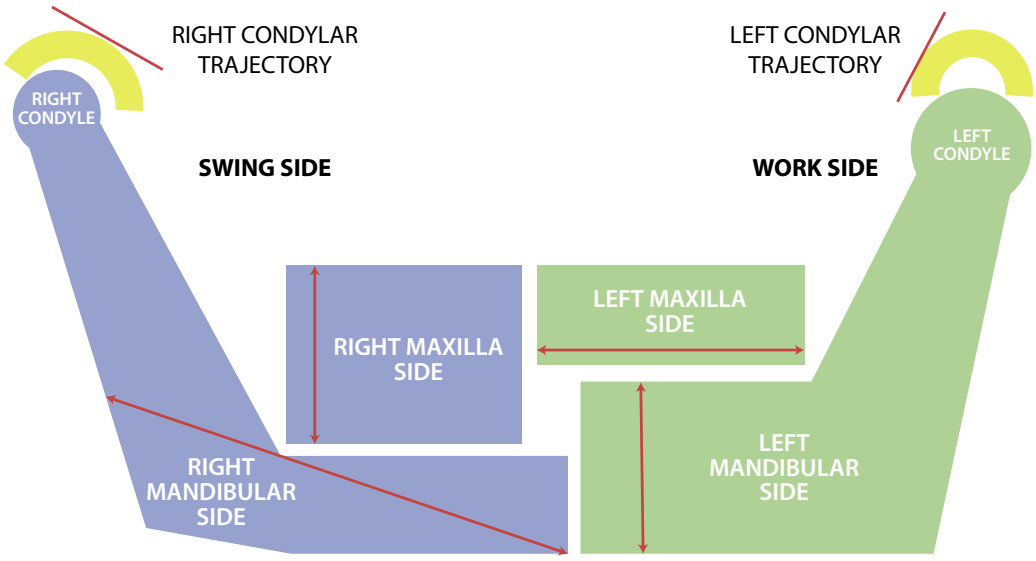

Figure 2. Own replica of the Marqués Diagram.

of a posterior crossbite in patients with primary dentition is the selective reduction of canines[50]. Facal also proposes selective reduction as the main option, decreasing the height of the canines of the non-chewing side below the height of the contralateral canines, thus performing a hypercorrection[18]. However, Planas proposes the composite tracks as the main treatment and, if necessary, perform a selective reduction of the canines[12].

De Boer and Steenks indicate in their study that selective reduction of cusps of temporary teeth can intercept the growth and development of the masticatory system of patients. Reduction can be an option only if the difference of maxillary and mandibular width is greater than 3 mm in the canine region[51]. Malandris and Mahoney claim that selective reduction changes the chewing pattern, achieving more symmetrical movements and bilateral chewing[52]. Facal proposes to add crossed elastics in the second temporary molars and in the first permanent molars, if the patient had them, to increase the vertical dimension in case the composite tracks and reduction were not enough[2].

Brin et al. confirm that the best way to treat a posterior crossbite in a mixed dentition is with slow expansion, since some degree of skeletal influence is expected at this age[53]. The most used device for this expansion is the Quad-Helix. Petrén et al. corroborate that this device is the best for the malocclusion treatment[54]. Quad-Helix can produce an expansion in the median palatine suture in 75% of cases along with orthodontic movements and dentoalveolar inclination. From 10 years old only acts at dentoalveolar level[27].

One of the concerns of orthodontists is whether, by correcting the unilateral posterior crossbite, a change in the position of the condyle in the TMJ could occur, thus reducing the adaptive capacity of some patients. Therefore, the professional should consider whether the non-surgical correction of this malocclusion can have an impact on the state of the TMJ or whether it can lead to pain or discomfort[25]. However, several studies indicate that all symptoms of temporomandibular disorders that may exist, such as joint sounds, headaches, muscle pain or weakness associated with this condition, disappear after orthodontic correction[27].

CONCLUSIONS

The non-alternating unilateral mastication habit generates a series of changes at muscle, bone and joint structures level of the TMJ, generating an asymmetric mandibular growth. This mandibular asymmetry generates a posterior crossbite on the chewing side in most cases.

This habit is determined mainly by peripheral factors such as occlusion, comfort in chewing, the contact area between the teeth, interference in lateralities, etc.

The increase in function and size of the mastication muscles on the preferred side will lead to changes in the TMJ and the jaw will be anteroposterior shorter, higher and bulkier.

At the TMJ level, alterations appear such as anterior disc displacement, lower disc thickness, more inclined articular eminence and thickened condyle. The association between non-alternating unilateral chewing, posterior crossbite and temporomandibular disorders is unclear.

The treatment of choice for the correction of non-alternating unilateral mastication with posterior crossbite during growth is the application of composite tracks in the cross-side and selective canine reduction in the non-crossed canine, if necessary, looking for the minimum vertical dimension, and therefore, mastication on this side. When it is not possible to correct it with tracks and reduction, maxillary expansion will be chosen.

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