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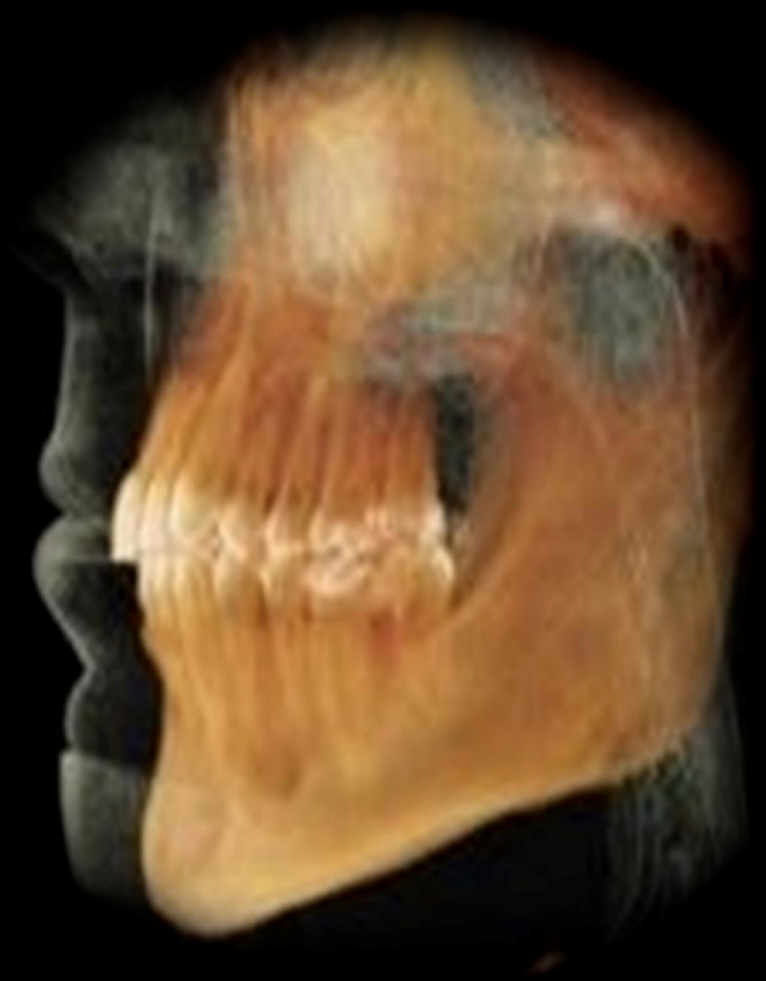

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Editorial

Dear Researcher,

We have the great pleasure to entry the eleventh edition, volume one, of the ***Journal of Surgical and Clinical Dentistry - JSCD****.*

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Mário dos Anjos Neto Filho
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Summary

LITERATURE REVIEW

ORIGINAL

HISTOLOGY OF THE ORTHODONTIC MOVEMENT

MARIA PITTNER[1*], RICARDO CESAR GOBBI DE **OLIVEIRA**[2]

1. Undergraduate student in Dentistry by UNINGÁ; **2.** PhD in Orthodontics, Faculty of Dentistry of Bauru - USP, Professor of Dentistry, UNINGÁ.

* Quintino Bocaiúva Street, 1154, Ap. 34, Zone 07, Maringa, Parana, Brazil. ZIP CODE: 87020-160, mariapittner@hotmail.com

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ABSTRACT

The orthodontic movement occurs by reorganization of periodontal tissue in response to application of a mechanical force, and various biochemical substances are synthesized and released for initiation and maintenance. Bone cells recognize the application of the mechanical load generating coordinated bone remodeling in bone resorption which is adjacent to areas of compression of the periodontal ligament and bone formation adjacent to areas voltage of the periodontal ligament, with subsequent adaptive changes ligament. Therefore, cells and tissues using sensitivity and transduction in response to application of mechanical force, with subsequent movement of the tooth.

KEYWORDS: Bone remodeling, fibroblasts, osteoclasts, neoformation.

1. INTRODUCTION

To better understand orthodontic movement, we need to understand the tissue phenomena that occur at each stage of tooth movement. Thus minimizing the risks of root resorption, optimizing the treatment and comfort of the patient.

Two processes are fundamental for orthodontic tooth movement to occur, and one of them is reabsorbed in the areas of pressure, while in the areas of tension a new bone is formed. This is possible because bone is a dynamic tissue that allows a constant remodeling, thus, a process that requires interactions between different cell types, regulated by biochemical factors or mechanical factors[1].

Pressure-tension theory explains the process of bone remodeling, where this process is slower on the pressure side, than on the tension side of the fibers belonging to the periodontal ligament. On the pressure side, it occurs to the compression of the tissues, periodontal, generating a process of degeneration, evolving to necrosis with aseptic characteristic, exhibiting hyaline appearance[2]. The collagen fibers constituting the periodontal ligament suffer a disaggregation, accompanied by hemodynamic changes, increased vascularization and, consequently, migration of inflammatory cells to the site, mainly neutrophils. Concomitantly, on the tension side, where the fibers of the periodontal ligament are stretched, the number of fibroblasts and osteoblasts increases, resulting in the production of collagen and bone matrix, promoting bone neoformation of the wall adjacent to the tractioned tooth[3,4,5].

It was observed that the repair of the tissue by the orthodontic movement occurs after the reduction of the force applied[6], requiring the use of intermittent forces, which requires gradual reduction and constant reactivation is required. However, with the development of super-elastic nickel-titanium (NiTi) yarns, it was possible to apply continuous forces during the period of orthodontic movement, analyzing the possible responses of the periodontal tissue of sustentation against mechanical stimuli.

In this context of the histology of the orthodontic movement, it is necessary to understand what precedes this, that is, how these tissues are formed and how they behave, as we activate them. The fact that there is no resorption in cement occurs due to the absence of receptors on the cementoblasts membrane for mediators of bone resorption, since the mechanism of orthodontic movement can be explained by molecular actions[7].

The inflammation is responsible for the orthodontic movement, and the orthodontic force is the cause of the trauma, which is characterized biologically by the formation of free protein in the body. Proteins are bound together, and when a protein becomes free, it becomes unstable, and undergoes degradation by enzymes. The proteins that are free generate a series of events fundamental for orthodontic movement, which will be discussed later[2].

Thus, as the orthodontic movement occurs through the restructuring of the periodontal tissues in response to the application of a mechanical force, and several chemical substances are synthesized and released for their initiation and maintenance, we will address these cellular and molecular reactions that allow the activation of the orthodontic movement[1,2].

2. MATERIAL AND METHODS

To carry out this study we following steps: 1) identification of the research question, followed by a search of the descriptors or keywords; 2) determining the

criteria for inclusion or exclusion of research in online databases; 3) categorization of studies, summarizing and organizing relevant information; 4) assessment of studies for critical analysis of the extracted data; 5) discussion and interpretation of the examination results, contextualizing theoretical knowledge and evaluating their applicability as; 6) presentation of the integrative review and synthesis of knowledge of each article reviewed briefly and systematic way.

In the present study the guiding question of the integrative review was: such as histological modification due to orthodontic movement

Bases (Latin American and Caribbean Literature on Health Sciences) LILACS, SciELO (Scientific Electronic Library on Line) and PubMed (NCBI US National Library of Medicine National Center for Biotechnology Information) were consulted. Studies that have addressed the thematic, published from 1972 to 2016, regardless of the languages of publication were included. The following controlled for the search and also used as keywords descriptors were used: Bone remodeling, fibroblasts, osteoclasts, neoformation.

3. LITERATURA REVIEW

Since inflammation is required for orthodontic movement, Eltman *et al*. (2001)[8] have schematized the phases following inflammation, which are caused by the mechanical forces involved. Continuous to the trauma, we obtain free proteins, free phospholipids, which undergo the enzymatic action of phospholipase, which transforms into arachidonic acid, the molecules of phospholipids. This substrate will undergo two enzymatic pathways: one is the enzyme cyclooxygenase, generating the formation of prostaglandins and thromboxanes; And the other is the enzyme lipoxygenase, resulting in the formation of hydroxyethoxytetraenoic (Hete) and leukotrienes[9].

The substances generated from arachidonic acid are called eicosanoids. Among these, as already mentioned we have prostaglandins (PG), and prostaglandin E (PGE), which play an important role in orthodontic movement. PGE is responsible for vasodilation, increased cellular permeability, regulation and synthesis of osteoblasts and fibroblasts, and alteration of cellular immune functions. PGE, basically speaking, alters the metabolism of fibroblasts and osteoblasts, which secrete collagenase and also PGE, altering its morphology[2].

The PGE synthesized by these cells attracts the osteoclasts to the site of the trauma. The collagen matrix may be degraded by collagenase enzymes, because these matrices are not mineralized, this being possible because they form channels between the osteoblasts[10,11]. Degradations of the periosteum expose the area of hydroxyapatite, which results in the chemotaxis of osteoclasts. Then, migration of the osteoblasts to the periphery of the bone occurs, and osteoclasts move to the hydroxyapatite region (PAH)[10].

Concomitantly, PGE performs vasodilation, allowing the migration of macrophages, and other cells. Macrophages release interleukins, which is a type of cytokine, essential in the inflammatory process. In addition, cytokines induce the production of PGE and collagenase by osteoblasts and fibroblasts, as well as can contribute to cell division and minimize bone formation in vitro[12].

Osteoblasts and macrophages together are called the hard tissue resorption unit (10). Metabolism and also migration of osteoclasts can be controlled by both osteoblasts. In the process of bone remodeling, due to the fact that odontoblasts and cementoblasts do not have cellular receptors for PGE, this chemical mediator is not involved in this mechanism.

The undifferentiated and perivascular cells of the mesenchyme may be attracted by the reabsorption process, due to this, releasing growth factors (peptides). These mesenchymal cells differentiate into osteoblasts and begin to synthesize and secrete constituents of the bone tissue, such as matrix components, proteoglycans, type I collagen, osteonectin, osteocalcin and osteopontin, chemical mediators that control: and synthesis as the Bone resorption; The presence of other constituents of the region; And actions of a systemic character, that is, that go beyond the region that is being manipulatedAmong the systemic factors, the main ones are growth hormones, thyroid hormones, parathyroid hormone, glucocorticoids, calcitonin, and vitamin D3. Among the most important local factors, such as prostaglandins E2 (PGE2), skeletal growth factor (SGF), fibroblast growth (FGF), platelet-derived growth (PDGF), epidermal growth (EGF) Beta transformer (TGF), gamainterferon (INF) and interleukin (IL-1)[2,5,10].

Tissue reaction when in orthodontic movement

Faced with a mechanical stimulus obtained through orthodontic movement, tissue reactions occur in the periodontal tissues, mainly in the periodontium of sustentation. Studies have established that, generally, tooth movement occurs through two processes: by resorption in pressure areas and by formation in tension areas. The bone allows us this movement due to the dynamics of its fabric, which allows a constant remodeling. This change in bone structure is only achieved through biochemical reactions, allowing cellular interactions, and mechanical factors, thus allowing a tissue response. Several histological studies evaluating the movement through the application of intermittent forces, where the intensity of force gradually declines, requiring recurrent activations in biomechanics, proceeded the classic concepts on tissue reactions against orthodontic movement[13,14].

Several studies have documented that in areas

where there was pressure there was an increase in the number of osteoclasts and consequently bone resorption. In the periodontal ligament, changes occur in vascularization, collagen disruption and hyalinization[13,15].

The hyalinization of an aseptic necrotic air, due to the fact that it does not have cells in this region of the periodontal ligament, a limitation for orthodontic movement occurs. Thus, the process of hyalinization should be eliminated for continuity of the movement process, with the formation of new periodontal structures[13]. If this necrotic process is not deconstitution, permanent lesions such as root resorption can be generated[16]. Much research indicates that hyalinization of the periodontal ligament during the initial phase of orthodontic treatment is almost inevitable.

This repair process occurs when pressure is reduced and cells migrate to the site. In addition, new capillary vessels are formed, being an important factor in this repair process. However, root resorption may still occur where these areas of hyalinization are formed[9].

On the tissue changes in the areas of tension, there is an increase in the spaces between the structures of the periodontal ligament. The cells change shape, becoming longer, with their elongated axis, toward the traction. The osteoblasts that migrate and distribute in the alveolar surface region, characterize the first response of these cells to the orthodontic stimulus[21]. Then, there is an increase in the number of fibers and vascular changes, such as vasodilation. Bone deposition occurs over the entire surface of the alveolus, progressing with increased movement time[14].

Root resorption

The lesion leading to root shortening or root resorption may be correlated with the force model applied, with the magnitude and type of this force, with the genetics, the type of malocclusion, some systemic condition, with sex, Age, with several factors that may result in root resorption[22,23].

In ideal conditions, orthodontic movement should involve alveolar bone and ligament. However, root resorption is an undesirable effect that occurs in some cases. It is believed that this resorption occurs adjacent to the areas of hyalinization, in the stage of removal. The elimination of this hyalinized area by macrophages and giant cells causes removal of the cementoid layer, leaving the root surface exposed[18,19] With the late occurrence of root resorption, at which time the "clasts" that resorb root structure (cementum and dentin) appear, and this reabsorption continues until all areas of hyalinization are removed[9,20].

The cement has the capacity of neoformation, but it can not be compared with the regenerative capacity of the bone. This allows the cement to regrow new layers at the affected site after resorption. However, when the dentin is reabsorbed, the cement in most cases will cover it with a thin layer, but without recovering its initial root shape. Because of this, root resorptions are considered irreversible when they reach the dentin. The periodontal tissue change in relation to the orthodontic movement depends on several factors, especially the mechanical factors that are divided into: force magnitude, force direction, type of force, period of incidence of the quantum and also the magnitude of the force of movement[9,20].

4. DISCUSSION

As previously shown, prostaglandins play a key role in orthodontic movement. But in addition to prostlaglandines, we have osteopontin (OPN), which is a non-collagenous protein found in large numbers in the matrix of mineralized tissues. Some authors suggest that this protein is produced by osteoblasts, others suggest that it is produced by osteoclasts. Osteopontin has great importance in the mechanism of bone remodeling suggesting that it participates in the phases of reabsorption and neoformation. Studies suggest that OPN synthesis increases in bone remodeling versus mechanical stimulation[24,25,26].

There are signs that OPN has an important role in relation to cement, since its absence leads to a decrease in the number of osteoclasts causing a decrease in root resorption when the mechanical force is applied to the bone tissue. In addition, it was observed in rats that root resorption was observed after approximately 10 days; Even so it did not cause any irreversible damage. However, it is possible that root resorption occurs after the period of application of force[27].

Krishnan & Davidovitch (2006)[28] in their studies report that during the early stages of tooth movement the application of force generates an acute inflammation, in which there is continuous flow of fluids in the periodontal ligament and in the bone tissue, which lead to cell and matrix distortion. As a consequence of the absence of bacterial invasion this reaction is considered as an aseptic inflammatory reaction, which involves a complex cellular response and is reintroduced with each activation of the orthodontic appliance and, because of this, the release of arachidonic acid from the cell surfaces and its metabolism through the lipoxygenase or cyclooxygenase pathway lead to the release of the first messengers (prostaglandins and leukotrienes), which activate or stimulate the release of the second messengers, The cyclic AMP. These second messengers provoke cellular reactions, leading to the remodeling of the bone and the periodontal ligament, where there is also intense angiogenesis and remodeling of the pre-existing blood vessels, through which the release of inflammatory mediators occurs. Therefore, the synthesis and local release of sev-

eral chemical mediators, such as interleukin-1 alpha (IL-1), interleukin-1 beta (IL-1), tumor necrosis factor alpha (TNF), interleukin- 6), and nitric oxide (NO), play an important role in the beginning and maintenance of the bone remodeling process, resulting in the clinical effect of tooth movement[29,30,31,32,33,34,37,38].

In order to verify which cells were involved in the removal of hyalinized tissue and in the reabsorption of root structures, after the application of orthodontic force, Brudvik & Rygh (1994)[6] moved the first right upper molar of 12 Wistar rats and, through the electron microscopy of Scanning, they observed that multinucleated giant cells as well as macrophage-like mononuclear cells were responsible for the elimination of formed hyalinized tissue as well as root resorption. Osteoclasts were only found in association with bone tissue. As a result, degraded fibrous tissue and cell debris appeared to be eliminated primarily by macrophages. Fibroblasts can also act as tissue eliminating cells[34,35,38].

Roberts & Chase (1981) 36 observed the presence of pre-osteoblasts that differentiated into osteoblasts in the periodontal ligament after orthodontic movement in rats. From its results, it could be concluded that osteoblasts could be derived from cells present in the periodontal ligament (mesenchymal cells). The production and mineralization of the matrix of any hard tissue are phasic processes that are characterized by periods of activity interspersed with periods of rest, and are morphologically reflected by perceptible incremental lines[28,29,30,31,37,38].

Krishnan & Davidovitch (2006)[28] in their studies report that during the early stages of tooth movement the application of force generates an acute inflammation, in which there is continuous flow of fluids in the periodontal ligament and in the bone tissue, which lead to cell and matrix distortion. As a consequence of the absence of bacterial invasion this reaction is considered as an aseptic inflammatory reaction, which involves a complex cellular response and is reintroduced with each activation of the apparatus. Thus, the synthesis and local release of several chemical mediators plays an important role in the initiation and maintenance of the bone remodeling process, resulting in the clinical effect of tooth movement[43,44,45,46].

5. CONCLUSION

The orthodontic movement occurs as a consequence of mechanical factors applied in the bone tissue and as a consequence of these, a biochemical cascade occurs, with actions of several enzymes in several cells and as a result cellular alterations arise and finally changes in the tissue itself, but all these mechanisms can Be affected by issues of applied force, but also due to physiological factors of each individual.

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ACCIDENTAL EXTRUSION OF SODIUM HYPOCHLORITE DURING ENDODONTIC TREATMENT: A CASE REPORT

CAROLINE AMADOR **PIZZI**[1], GABRIELA LIMA **FABRI**[1], VANESSA RODRIGUES DO **NASCIMENTO**[2], LUIZ FERNANDO **TOMAZINHO**[3*]

1. Aluna do curso de graduação em Odontologia da Universidade Paranaense (UNIPAR); **2.** Especialista em Endodontia, Mestre em Radiologia, Professora do Curso de Odontologia na Universidade Paranaense-UNIPAR; **3.** Especialista em Endodontia, Doutor USP-SP, Professor do Curso de Odontologia da Universidade Paranaesnse – UNIPAR.

* Inaja Street, no 3560, Ap 42 Centro, Umuarama, Parana, Brazil. ZIP CODE: 87501-160. tomazinho@unipar.br

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ABSTRACT

The goal of endodontic therapy is the sanitation of the root canal system in the best possible way. At this stage of treatment is carried out chemical - mechanical preparation, comprising the action of the instruments on the walls of the ducts associated with an auxiliary chemical substance. Sodium hypochlorite is widely used as a sprinkler agent during endodontic treatment because it has a good antimicrobial activity, be solvent of organic matter present in the infected root canal. However, when this solution is injected and accidentally leaked beyond the root apex may cause complications to the patient. This article describes the characteristics of a clinical case of sodium hypochlorite leakage in the element 15 as well as the treatment given in case, for the same resolution.

KEYWORDS: Endodontics, irrigation, extravasation, sodium hypochlorite.

1. INTRODUCTION

The main goal during channel treatment is the complete debridement of connective tissue and the elimination of surplus microorganisms found in infected root canals. In addition, it aims at effective sealing, so that bacterial recolonization does not occur in the root conduits[1].

In order to effectively clean the root canals, it is essential to use irrigating solutions during the biomechanical preparation in order to eliminate or minimize the bacterial colony residing in the canals, as well as the dissolution of the tissues promoting cleansing and chelating action[2].

According Pécora & Estrela (2004)[3], when pulp necrosis during endodontic treatment is necessary, the antimicrobial effect must prevail in association with tissue dissolution capacity. As a solution of choice we can use Sodium Hypochlorite which is available in different concentrations between 0.5% and 2.5%.

We can observe that the Sodium Hypochlorite has properties that aid in a correct chemical-mechanical debridement in the root canals. It acts as a lubricant for instrumentation, neutralizes toxic products, has solvent action and detergent. And it still acts on a large scale of microorganisms[4].

The higher the concentration of sodium hypochlorite solution, the greater its ability to dissolve living or necrotic tissue, and the greater neutralizing action of the root content. However the more concentrated the more irritating it will be to the apical and periapical tissues[5].

The occurrence of accidents with sodium hypochlorite is infrequent, but when they occur they generate complications for the patient. In these cases, the patient should be informed of the possible sequelae and the duration of treatment. In the majority of cases, accidents with NaOCl have a favorable prognosis, requiring immediate and adequate treatment, and a cautious analysis of the situation[6].

The objective of the present study is to report a case of hypochlorite extravasation during endodontic treatment, as well as the conduct of choice that was performed to treat the surgical accident.

2. CASE REPORT

A 39 year old female patient attended a private dental clinic in the city of Umuarama - PR, reporting a sensitivity to touch and chewing in the right maxilla region. During the anamnesis, she reported that this sensitivity had accompanied him for years and in recent months had been increasing. Clinically, a slight sensitivity to palpation was observed in the groove bottom region, near the apex of element 15, and a slight edema in the periapical region of said element. Faced with the pulp vitality test, he presented a negative response. Radiographically, a radiolucent image is observed in the pariapical region, suggestive of endodontic lesion (Figure 1).

In the first session, after prophylaxis of the involved element and absolute isolation of only the tooth 15, the coronary opening was then performed and we obtained access to the canal embouchure. The pulp chamber ceiling was removed and the position of the canal path of

the element was performed with pre-bending maneuvers endodontic files of the number 10, K type (Dentsply, Maillefer - Switzerland).

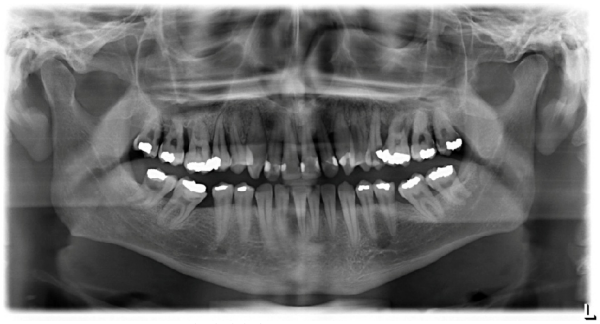

Figure 1. RX panoramic initial.

It was possible to locate and exploit any extension of the main conduit of said element. The mechanical-mechanical preparation of the canal was performed using the rotating files of the Prodesign S system (Easy, Belo Horizonte - Brazil) in the sequence proposed by the manufacturer. As an irrigating solution was 2.5% sodium hypochlorite.

At the end of the sanitization maneuver of the root canal system, the patient began to complain of a burning sensation in the region, which increased in intensity rapidly, accompanied by rapid facial edema in the region (Figure 2). It was then suspected that a possible extravasation of sodium hypochlorite in the periapical region, since the signs and symptoms are characteristic of this picture. At this moment, a saline irrigation with profuse saline was performed, in order to attenuate the irritation caused. We chose not to perform intracanal medication in this session to avoid further chemical trauma in the periapical region.

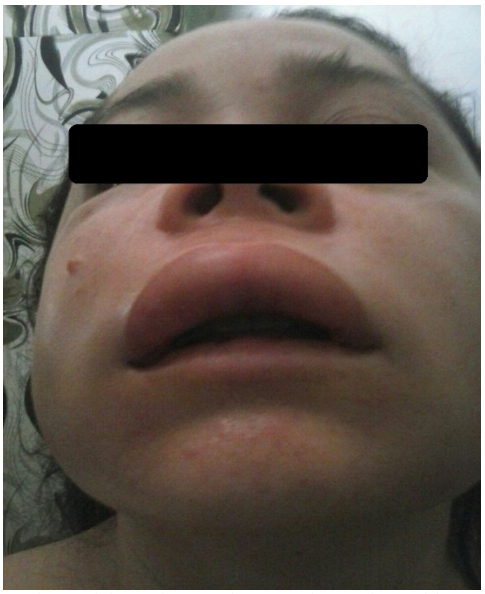

Figure 2. Photo of the patient on the same day as the accident.

Following the protocol described by Pinheiro *et al.* (1998)[7] and Witton *et al.* (2005)[8], antibiotics were prescribed (Amoxicillin 500 mg). Its use is recommended in these cases of extravasation of sodium hypochlorite, due to the presence of necrotic tissue and risk of infection. However, in more severe situations, hospitalization and support measures become prudent in order to avoid a worsening of this situation[7,8]. It was also asked to apply ice in the region in the first 24 hours, aiming to reduce edema. In order to alleviate the discomfort, it was also prescribed the use of analgesic. Aiming to facilitate oxygenation and possible drainage of maxillary sinus suppuration, a nasal nasal decongestant Rinosoro 0.9% - Sodium Chloride 9.0mg/ mL 30ml - Farmasa.

Patient follow-up occurred daily for a week after the accident. On the first day, the patient reported increased edema and increased burning sensation, with stabilization of the pain sensation. Clinically, the edema almost completely affected the right facial region, including the labial region (Figure 3). Already on the second and third days, the facial edema remained, but the painful sensation gradually regressed. After the fourth day, the edema was regressing and was already painless. Six days later, the patient showed a normality, with no signs and symptoms, returning to the clinic to continue the endodontic therapy.

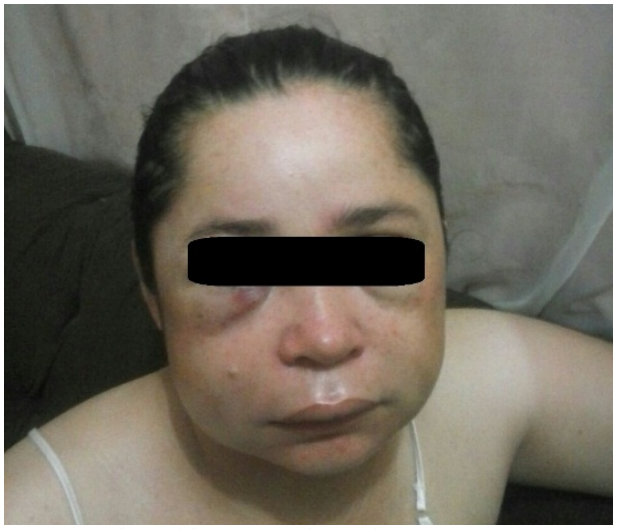

Figure 3. Photo of the patient 24 hours after.

3. DISCUSSION

The toxic effects of irritation of Sodium Hypochlorite have been described in several case reports[1,3,9], where an accident was presented with the solution. If an accident as described above occurs, a treatment protocol should be instituted to minimize possible damage. Initially a solution of saline solution should be injected into the canal of the root canal aiding in the total elimination of the hypochlorite still present. When we notice some

damage, we prescribe antibiotics to reduce the risk of a secondary infection associated with the prescription of analgesic for pain relief. It is also recommended to apply ice on edema in the first 24 hours[10].

The treatment of pain, if present, serves only as an attenuator, and it is necessary to wait for the remission of symptoms through the follow-up of the case, as explains by Rendón *et al.* (2004)[11].

To avoid accidents with hypochlorite, it is recommended to use absolute insulation during endodontic treatment, putting protective goggles on the patient and the operator. The irrigation needle should preferably be bevelled, should not be fair to the channel, and should have a safety margin of at least 2mm in relation to the working length; should be irrigated slowly without exerting excessive pressure[10].

García Zulunga *et al.* (2001)[12] report that the possibility that the disinfectant agent, used in the irrigation of the root canals, come into contact with the perriradicular tissues and other adjacent structures is high.

Ehrich *et al.* (1993)[13] Reported a clinical case of endodontic treatment in the right upper first molar, tooth 16. Where shortly after irrigation of the palatine canal with 5.25% sodium hypochlorite solution the patient complained of a bad taste in his throat. When the palatine canal was irrigated with saline solution, it was observed that the serum passed to the maxillary sinus and to the nasal cavity through the foramen along the lateral wall of the maxillary sinus. The patient complained of a mild burning sensation and an initial congestion. García Zulunga *et al*. (2001)[12] report that the possibility that the disinfectant agent, used in the irrigation of the root canals, come into contact with the perriradicular tissues and other adjacent structures is high.

Barbas *et al.* (1987)[14] reported a clinical case of fatal cerebral haemorrhage in a 52-year-old woman who underwent endodontic treatment and who, due to the extrusion of sodium hypochlorite into the maxillary sinus, reached the V cranial nerve.

Kavanag & Taylor[15] (1998) reported a clinical case similar to that described in this case report, where during endodontic treatment of the second right upper premolar the sodium hypochlorite solution was injected into the maxillary sinus. The patient presented severe pain and swelling in the region due to extravasation of the solution.

4. CONCLUSION

Based on the clinical case reported, we conclude that knowledge of the factors that can lead to this type of accident can be decisive to avoid it. However, from the moment of its occurrence, early recognition and adequate management of complications and symptoms is essential in cases of sodium hypochlorite extrusion. Where each patient should be treated according to the magnitude and type of extravasation that occurred during clinical practice.

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