Management of Aggressive Periodontitis Using Ozonized Water

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B*ackground*: Aggressive peridontal disease characterized by loss of bone around special teeth in adolescents and young adults.

Objective: The objective of this study was to assess the clinical and antimicrobial effect of ozonized water in management of aggressive periodontitis.

Subjects and Methods: Twenty patients suffering from localized and generalized aggressive periodontitis were selected, age range from 13 to 25 years. They were clinically and radiographically examined. Scaling, root planning, oral hygiene measures with or without ozone application were followed.

Results: Different treatment plans were evaluated regarding plaque and gingival indices, pocket depth, attachment loss and mobility scores. Antibacterial effect of ozone was investigated.

Conclusion and Recommendation: We recommend the use of oral hygiene instructions, scaling and root planning together with subgingival irrigation with ozonized water in the management of aggressive periodontitis. Up to our knowledge this is the first Egyptian study using subgingival irrigation in combination with the routine treatment plans for Aggressive periodontitis.

Keywords: Aggressive periodontitis - Ozone

Introduction

Periodontal disease is a group of inflammatory disorders, the pathophysiology of which is related to tooth accumulated microbial plaque and the host response to those accumulations (Miller *et al*., 1984).

Listgarten (1987) stated that major forms of periodontal disease are considered to represent bacterial infections in which certain bacteria appear to play a significant role in inducing and maintaining the inflammatory process. The health of periodontal tissues is maintained in a relatively stable state through the establishment of host – parasite equilibrium compatible with minimal tissue destruction

and repair of damaged structures.

On the other hand, some clinical entities such as juvenile periodontitis & other rapidly progressive forms of periodontitis as described by Watanabe, (1990) appear to be associated with potential periodontal pathogens, which include: Actionobacillus actinomycetem comitans (Aa) and certain black pigmented bacteria, as Bacteroides gingivalis, Bacteroids intermedius, Capnocytophaga sputigena and Eikenella corrodens.

The term "juvenile periodontitis" or "early onset periodontitis", "prepupertal periodontitis" and "rapidly progressive periodontitis" is now classified as "aggressive periodontitis": localized or generalized (Armitage, 1999).

Aggressive periodontitis is an autosomal dominant triad with reduced penetrance. Parents, offspring and siblings of patients affected with aggressive periodontitis have a 50% risk of this disease also (Kinane and Hart, 2004).

Localized aggressive periodontitis (LAP) previously known as localized juvenile periodontitis is one of the rapidly progressive periodontal diseases. Certain forms of familial LAP show a simple Mendelian pattern of transmission. However, no gene mutation has been identified to be responsible for the LAP phenotype (Li *et al*., 2004). LAP is linked to human chromosome 1q25 or 11q14 (Hart *et al*., 2000).

Juvenile periodontitis as described by Stabholz *et al*., (1998) which is a relatively rare aggressive early onset form of periodontal disease, characterized by pattern of rapid vertical loss of alveolar bone around more than one permanent tooth mainly the permanent first molars & incisors. The severity of the destruction is not proportional to the mass of plaque or calculus present.

Clinical picture of aggressive periodontitis characterized by an insidious onset during the circumpubertal period. The most striking feature is that during the early stage, there is lack of clinical signs of inflammation. Moreover, the severity of periodontal destruction, which is evidenced by deep periodontal pocket formation, tooth mobility and migration, is out of proportion to the magnitude of local initiating factors. In advanced cases diastemata are formed as well as rotation and elongation of individual teeth. At this stage clinical

inflammation starts to appear as a result of plaque accumulation on the elongated clinical crowns. Regional lymph nodes enlargement has been also reported (Lehner *et al*., 1974; Grant *et al*., 1988 and Schwartz, 1990). Despite rapid progression of periodontal destruction, the condition is absolutely painless (Schwartz, 1990). As the disease progresses, other symptoms may arise. Denuded root surfaces become sensitive to thermal and tactile stimuli. Deep dull, radiating pain may occur with mastication due to irritation of the supporting structures and food impaction (Lehner *et al*., 1974).

Aggressive periodontitis seems to be restricted to permanent dentition, whereas deciduous teeth are not affected or prematurely lost. This distinguishes aggressive periodontitis from periodontal changes, which are caused by some intrinsic disease such as; cyclic neutropenia, lazy leukocyte syndrome , Papillon Lefvre syndrome, Down syndrome, Diabetes mellitus, hypophosphatasia, hypothyroidism and hypoadrenocorticoidism (Budunelli *et al*., 2000).

Radiographic findings in aggressive periodontitis show bilateral, usually symmetrical bone resorption is seen in relation to the first molars and /or incisors. The extent of bone loss depends upon the stage of the disease at the time of diagnosis, whether early or advanced. Bone loss starts usually on the mesial aspects of molars, while buccal and lingual or palatal plates resorbe last, leading to furaction involvement only in advanced cases. Periapical radiographs may show a cupped out (arc shaped) bony defect extending from the distal surface of the second premolar to the mesial aspect of the second molar (Grant *et al*., 1988). The rate of bone destruction is very rapid and radiographic evidence of a three-fourths loss of bony support of involved teeth can be achieved in a 5-years interval or even less. This progression rate is about four times as much as for adult periodontitis (Bonta *et al*., 2003).

Haraszthy *et al*., (2000) have been postulated that the bilateral symmetry in the pattern of bone loss resulted from a genetically determined developmental defect. However, another contradicting point of view was that this symmetry was caused by the pattern of eruption of the affected teeth.

It is important to determine the association between aggressive periodontitis and bacteria implicated in its etiology. It has been found

that actinobacillus actinomycetemcomitans is frequently found in higher numbers in localized aggressive periodontitis lesions than in healthy sites in the same individuals, and it has been implicated as an etiologic agent of aggressive periodontitis (Haraszthy *et al*., 2000). Other bacteria involved in the etiology of aggressive periodontitis are Eikenella corrodens (Mandell, 1984) and Capnocytophaga species (Newmann *et al*., 1976; and Savitt and Socransky, 1984).

It has been found that the composition of the subgingival microflora in localized aggressive periodontitis appears to be different from that in generalized aggressive periodontitis in which there is a higher number of Bacteroids gingivalis (Loesche *et al*., 1985; Tanner *et al*., 1987 and Dzink *et al*., 1988).

The problem of periodontal inflammatory disease treatment is one of the urgent problems in stomatology. In spite of an abundance of existing remedies & methods of treatment, this problem is far from solution. The importance of searching new remedies becomes evident. The use of ozonized solutions for complex treatment of inflammatory diseases of paradontium is of great interest (Sorokina and Zaslavskaja, 1997).

The effect of ozonized water on oral microorganisms and dental plaque was studied by Nagayoshi *et al*., (2002). They found that ozonized water should be useful in reducing the infections caused by oral microorganisms in dental plaque.

Subjects and Methods

Twenty patients suffering from aggressive periodontitis (age range from 13 to 25 years) were selected from the outpatient clinic of the Oral Medicine and Periodontology Department, Faculty of Oral and Dental Medicine, Cairo University as well as from the outpatient dental clinic of the Oro-dental Genetic Department of the National Research Center, Cairo. 15 of these patients were suffering from localized aggressive periodontitis and the remaining 5 were suffering from generalized aggressive periodontitis.

Clinical procedures:

Before starting the treatment the following clinical parameters were measured:

a) *Plaque Index* (according to Silness and Loe, 1964).

b) *Gingival Index* (according to Silness and Loe, 1964).

c) *Periodontal Pocket* Depth (6 measurements), (according to Carranza, 2002).

d) *Periapical radiographic examination* to confirm the diagnosis.

Using split mouth design, the patient's mouth was divided into four quadrants according to the following fixed scheme:

a) *1st Quadrant* (Upper Right Quadrant): Scaling and root planning were performed.

b) *2nd Quadrant* (Lower Right Quadrant): Scaling and root planning were done together with ozonized water application.

c) *3rd Quadrant* (Upper Left Quadrant): Oral hygiene measures were followed.

d) *4th Quadrant* (Lower Left Quadrant): Oral hygiene instructions were followed together with ozonized water application.

Irrigation of the pockets by ozonized water is done by using a blunt tipped sterile plastic syringe. Periodontal pockets were irrigated with 150 ml of ozonized water over 5 to 10 minutes once weekly, for a clinical four weeks study.

The selected ozone concentrations (2000 - 3000 μg per treatment) have a positive rather than destructive effect according to the selective reactivity of ozone (Ziad Fahmy, 1992).

Bacteriological studies:

For each patient the following samples were taken before, during (not all cases) and at the end of the treatment (4 weeks period).

1- A random sample was taken from the area containing the deep pathological pocket (5mm depth or more) .

2- Scaling and root planning were done in the 1st and 2nd quadrant:

- In the 1st quadrant only scaling and root planning were performed alone without ozone application and a sample was taken before & after 4 weeks.

- In the 2rd quadrant ozone was applied and a sample was taken before & after the first ozone application and another one was taken at the end of the 4 weeks treatment.

3- Oral hygiene measures were instructed in the 3rd and 4th quadrant:

- In the 3rd quadrant oral hygiene instructions were followed

alone without ozone application and a sample was taken before & after 4 weeks.

- In the 4th quadrant ozone was applied and a sample was taken before and after the first ozone application and another one was taken at the end of the 4 weeks treatment.

Each sample was taken by insertion of a small sized paper point in the pocket depth and was left for about 30 seconds and then removed and inserted in a glass tube containing 3 ml of thioglycolate broth.

Bacterial count:

The indirect plate count: The plate count is based upon the assumption that each organism trapped in or on nutrient agar, medium will multiply and produce a visible colony. The number of colonies therefore are the same as the number of viable cells introduced into the medium.

Procedure: (according to standard methods for bacterial counting)

Set up 6 tubes containing 0.9 ml sterile water. Aseptically, transfer 0.1 ml of the bacterial suspension into the first tube (10^{-1} dilution). Mix well and using a fresh pipette tip, transfer 0.1 ml to the next dilution tube (10^{-2} dilution). Proceed in this way with dilution down to 10^{-6}. The dilution should be mixed thoroughly with a vortex before each dilution to ensure that bacterial clumps are dispersed. Different pipettes should be used for each dilution. 10μ sample of the last dilution were transferred to two empty sterile dishes and blood agar cooled to 50C was added. The plates were swirled to mix the inoculum and the agar. Once dry, one plate was incubated anaerobically for 3 days in an anaerobic jar and the other was incubated aerobically at 37C. Colony count was done from both plates and the number of colonies/ml broth was calculated to obtain the total bacterial count.

Results

From the results present in table (1) there is a significant statistical difference concerning the changes in both plaque index score and gingival index score before and after the different treatment plans.

The results obtained in table (2) represent the effect on pocket depth before and after the different treatments. There is a significant improvement between the pocket depths obtained before treatment and those obtained at the end of the treatment but only in the mesial measurements (average of mesiobuccal and mesiolingual measurements) and distal measurements (average of distobuccal and distolingual measurements) related to quadrants treated by scaling and root planing together with ozone application for 4 weeks with P-value < 0.001.and also related to areas treated by scaling and root planing only with P-value < 0.005. While there is no significant difference related to both buccal and lingual measurements in the mentioned two quadrants. There is no significant difference related to all obtained measurements related to quadrants treated by oral hygiene instructions only or together with application of ozonized water.

The different bacterial counts at the different surfaces of treatment when applied for the first time there is a significant difference in all types of treatments except those patients who followed the oral hygiene instructions for the first time only. While after 4 weeks period there is an overall significant difference for all types of treatment in all cases as shown 1n table (3).

Table (1): Plaque index (PI) and Gingival index (GI) pre and post different treatments.

	Scaling & root planning		Oral hygiene	
	Only	With ozone	Only	With ozone
Pre treatment *(PI)*	1.605 ± 0.68	1.65 ± 0.75	1.50 ± 0.51	1.9 ± 0.76
Post treatment *(PI)*	0.30 ± 0.47	0.10 ± 0.31	0.70 ± 0.47	0.85 ± 0.49
Change (%) *(PI)*	84.2 ± 27.8	97.0 ± 10.3	55.0 ± 35.9	57.5 ± 28.3
P-Value .	< 0.000	< 0.000	< 0.001	< 0.001
Pre treatment *(GI)*	2.05 ± 0.69	2.10 ± 0.72	1.55 ± 0.51	1.95 ± 0.76
Post treatment *(GI)*	0.40 ± 0.50	0.10 ± 0.31	1.25 ± 0.44	1.40 ± 0.50
Change (%) *(GI)*	84.2 ± 10.6	96.7 ± 10.3	15.0 ± 23.5	23.3 ± 22.6
P-Value .	< 0.000	< 0.000	< 0.031	< 0.001

* P-Values ≤ 0.05 is considered significant. Values are means ± standard deviations.

Table (2): Effect on pocket depth related to 1st molar before and after the different treatments.

Upper right 1st molar *(Scaling and root planning)*	Mesial	Distal	Buccal	Lingual
Pre treatment .	3.78 ± 2.28	3.50 ± 1.97	1.70 ± 0.57	1.60 ± 0.60
Post treatment .	3.13 ± 1.82	2.90 ± 1.50	1.65 ± 0.49	1.55 ± 0.51
P-Value .	< 0.005	< 0.005	Not sign.	Not sign.
Lower right 1st molar *(Scaling and root planning + ozone)*				
Pre treatment .	3.38 ± 2.11	3.63 ± 1.97	1.60 ± 0.50	1.60 ± 0.50
Post treatment .	2.60 ± 1.37	2.65 ± 1.25	1.50 ± 0.51	1.55 ± 0.51
P-Value .	< 0.001	< 0.001	Not sign.	Not sign.
Upper left 1st molar *(Oral hygiene only)*				
Pre treatment .	1.78 ± 0.7	4.38 ± 1.68	1.50 ± 0.51	1.50 ± 0.51
Post treatment .	1.70 ± 0.61	4.30 ± 1.74	1.40 ± 0.50	1.45 ± 0.51
P-Value .	Not sign.	Not sign.	Not sign.	Not sign.
Lower left 1st molar *(Oral hygiene + ozone)*				
Pre treatment .	3.75 ± 2.04	2.48 ± 1.27	1.35 ± 0.49	1.35 ± 0.49
Post treatment .	3.68 ± 1.96	2.43 ± 1.22	1.30 ± 0.47	1.30 ± 0.47
P-Value .	Not sign.	Not sign.	Not sign.	Not sign.

* P-Values ≤ 0.05 is considered significant. Values are means ± standard deviations.

Table (3): Bacterial counts pre and post different treatments.

	Oral hygiene		Scaling and root planning	
	Only	Ozone	Only	Ozone
Pre	1272 ± 277	1275 ± 278	1293 ± 299	1355 ± 450
1st time	1249 ± 277	1188±277*	659 ± 154 *	625 ± 157*
% change	1.9 ± 4.5	6.8 ± 5.3	48.7 ± 5.4	52.6 ± 7.3
P-value*	Not sign.	< 0.005	< 0.002	< 0.001
After4weeks	1024± 269*	794 ± 248*	7.9 ± 3.5*	3.2 ± 1.7*
% change	20.0 ± 7.6	37.9 ± 12.1	99.4 ± 0.3	99.8 ± 0.1
P-value*	< 0.004	< 0.002	< 0.000	< 0.000

* P-Values ≤ 0.05 is considered significant. Values are means ± standard deviations.

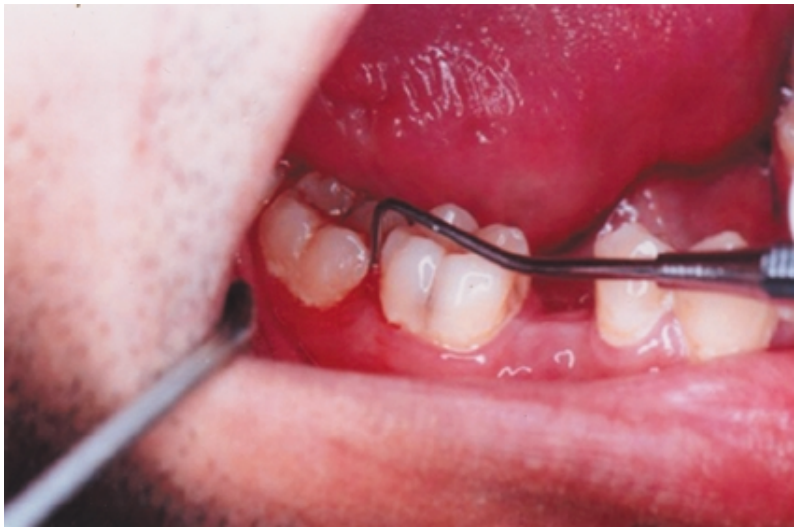

Fig. (1): Photograph for a 24 years old male patient affected by localized aggressive periodontitis (preoperative) shows very deep periodontal pocket (6mm) related to the lower right first molar with severe gingival inflammation and plaque accumulation.

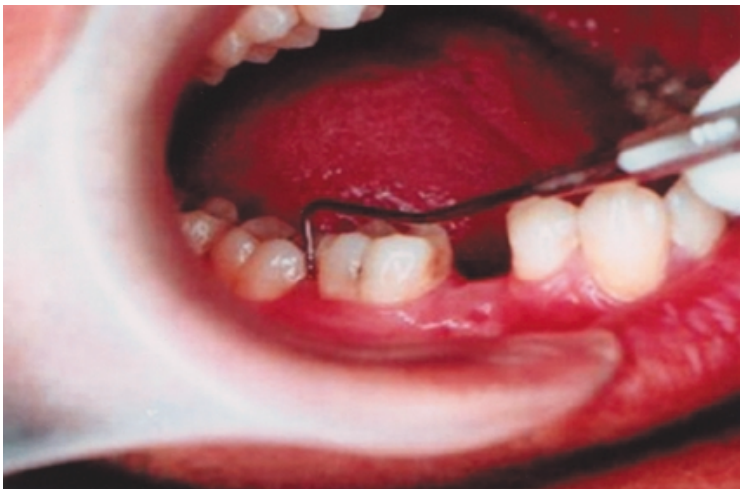

Fig. (2): Photograph for a 24 years old male patient affected by localized aggressive periodontitis (postoperative) shows improvement of periodontal pocket (5mm) related to the lower right first molar, decreased gingival inflammation and plaque accumulation after performing scaling & root planning with ozonized water application for 4 weeks.

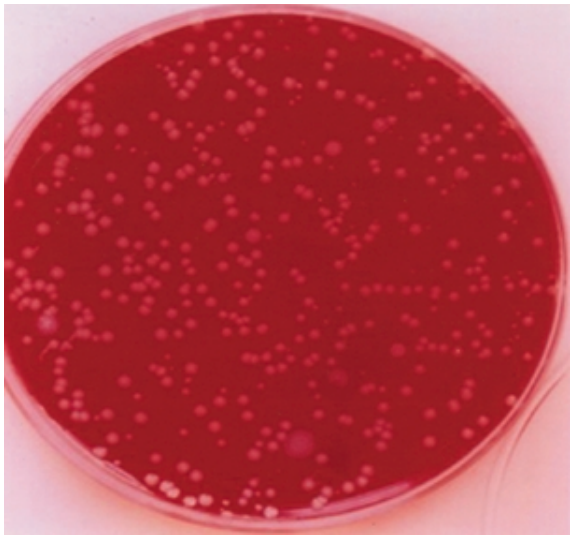

Fig. (3): Photograph showing bacterial growth on a blood agar plate of a sample taken from the depth of the pathological pocket at the lower right first molar area for the 24 years old male patient before performing scaling & root planning with ozone application.

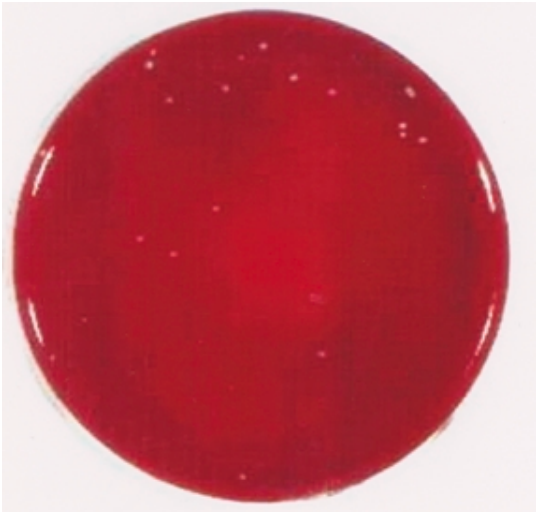

Fig. (4): Photograph showing bacterial growth on a blood agar plate of a sample taken from the depth of the pathological pocket at the lower right first molar area for the 24 years old male patient after performing scaling &root planning with ozone application at the end of 4 weeks treatment.

Discussion

Gingivitis and periodontitis are characterized by a local hypoxia of tissues and also by various microbic florae that may contain over 250 species. The presence of plaque in an increased amount causes changes in the oral cavity ecology leading to both gingivitis and periodontitis (Nobuhiro *et al*., 1999).

Aggressive periodontitis is a progressive periodontal disease characterized by loss of bone and periodontal support for special teeth in adolescents and young adults (Beaty *et al*., 1987).

The initiation and progression of periodontitis is caused by different bacterial accumulations in the subgingival pockets. Accumulations of bacterial plaque in the gingival crevice area are the cause of most, if not all, periodontal diseases. It was also proved that certain bacterial types exist in the periodontal pockets of aggressive periodontitis patients. It was also noted that these bacteria have been associated with active lesions in those patients (Nestory *et al*., 1995).

The objective of this study was to assess the clinical and antimicrobial effect of ozonized water in management of aggressive periodontitis. During this study the selected aggressive periodontitis cases show involvement of the first molar teeth and some also show involvement of the anterior incisors. At the area of involvement of the incisors it was very difficult to perform the suggested split mouth design and application of the different treatment modalities due to the close positional relationship between the anterior incisors, which may produce overlapping of the obtained results since the ozonized water with its low viscosity can easily run to the nearby teeth and thus the results obtained will not be reliable.

In the present study the high significant improvement regarding pocket depth, plaque index, gingival index and bacterial count is recorded related to quadrants treated by scaling and root planing together with ozone application, while quadrants treated by scaling and root planing show only significant improvement.

This results were predicted due to scaling and root planing as described by Savitt *et al*., (1984) ; Pattison and Pattison, (2002) is aimed to remove the microbiologically contaminated cementum layer and to eliminate and reduce the number of pathogenic microorganisms in periodontal pocket to level below those required to induce the

disease. These changes in the microbiota are accompanied by reduction or elemination of inflamation clinically. Ozone was found to have a potent antibacterial effect explained by the fact that it causes disruption of the envelope integrity through peroxidation of phospholipeds (Unsal *et al*., 1995 and Agapov *et al*., 2001). So our results agreed with Nagayoshi *et al*., (2002) who found that ozonized water should be useful in reducing the infections caused by oral microorganisms in dental plaque.

In treatment by scaling and curettage procedures, healing and improvement of pocket depth result from the ability of the tissues to form thin junctional epithelium since the contaminated cementum layer was removed (Caton and Zander, 1979 and Vandana and Redy, 2003).

In our study quadrants treated by oral hygiene instruction together with ozonized water as well as quadrants treated by only oral hygiene instruction showed significant improvement regarding only plaque index, gingival index and bacterial count which agree with the clinical 4- weeks study done by Sorokina & Lukinych (1997) using subgingival irrigation with ozonized water in combination with professional measures of oral hygiene, plaque formation was reduced due to pronounced anti-inflammatory effects on the periodontium after using irrigation of periodontal pockets with ozone.

From the bacteriological point of view the results obtained for all types of treatments after a time period of 4 weeks proved significant differences with different percentage levels of change except after only following the oral hygiene instructions. While as after the first time of the different treatments the results obtained for all types for the first time showed significant values, except those of after following the oral hygiene instructions only ,which was found to be of no significant difference. This may be explained that mechanical tooth brushing cannot eliminate the pathological micro-organisms from the subgingival pocket area where the measurements of the study were carried especially after performing the oral hygiene instructions once.

Concerning the results obtained by Agapov *et al*., (2001) ozone can cause stimulation of body's own defenses which is in a good agreement with the present results of this study and in good conformity with the results obtained by Lukinykh and Kosiuga (1998)

who studied the efficacy of hygienic treatment of the oral cavity in combination with ozone therapy. They proved that this combination mechanically removed soft dental deposits and also decreased bacterial contamination.

It has become apparent that irrigation only is not efficient as mechanical tooth cleaning and scaling at all, but it effects the regression of the inflammation as proved by Brauner, (1991). Subgingival irrigation as the sole therapeutic method is insufficient to treat periodontitis and should not be performed in lieu of scaling and root planing (Flemmig, 2002).

Any statistical significant difference obtained in plaque index and gingival index can be explained by the fact that thorough removal of microbial plaque quickly and effectively leads to resolution of gingival inflammation (Savitt *et al*., 1984).

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علاج التهاب الأنسجة الداعمة السنية باستخدام الماء المعالج بالأوزون

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يتميز التهاب الأنسجة الداعمة السنية الشديد بفقدان عظم ما حول أسنان معينة في الصغار و البالغين . و كان الهدف من هذه الدراسة الوصول إلي التأثير الإكلينيكي لماء الأوزون في القضاء علي الميكروبات و علاج التهاب الأنسجة الداعمة السنية الشديد .

و قد تم أختيار ٢٠ مريضا يعانون من التهاب الأنسجة الداعمة السنية الموضعي و العام . و كان عمر المرضي يتراوح بين ١٣ و٢٥ عام و تم فحصهم إكلينيكيا و بالتصوير الإشعاعي و لقد اتبع نزع الرواسب الجيرية من فوق سطح الأسنان ، و تنظيف جذور الأسنان و قياسات صحة الفم مع أو بدون وضع الأوزون .

أوصينا باستخدام تعليمات صحة الفم ، و نزع الرواسب الجيرية من فوق سطح الأسنان ، و تنظيف جذور الأسنان معا مع الغسيل تحت اللثة بماء الأوزون في معالجة التهاب الأنسجة الداعمة السنية الشديد .

إلي جانب معلوماتنا هذه اول دراسة مصرية تستخدم الغسيل تحت اللثة مع الخطة العلاجية الروتينية لالتهاب الأنسجة الداعمة السنية الشديد .