The Effect of Omega-3 Fatty Acids on Chronic Periodontitis in Postmenopausal Osteoporotic Females

Mohamed A. Galal[1], Basma M. Zaki[2] and Nermine Nassif[3]

[1] Researcher Assistant at Surgery and Oral Medicine Dep., National Research Center, Cairo, Egypt.
[2] Assistant Professor at Surgery and Oral Medicine Dep., National Research Center, Cairo, Egypt.
[3] Researcher at Surgery and Oral Medicine Dep., National Research Center, Cairo, Egypt.
boshta@hotmail.com

Abstract: The aim of the present study is to assess the effect of omega-3 fatty acids, as an adjunctive therapy in management of chronic periodontitis in postmenopausal osteoporotic females. Forty postmenopausal females having osteoporosis and chronic periodontitis were included in this study. These patients were divided into two groups: the first group (n=20) were given rutin and vitamin C (50 mg rutin + 100 mg vitamin C) once daily. The second group (the omega-3 group, n=20) were given 1gm omega-3 daily in addition to 50 mg rutin + 100 mg vitamin C once daily too. These medications were given for 6 months. Scaling and root planing were done to all patients before starting the medical treatment. Periodontal indices measured before and 6 months after treatment were: pocket depth (PD) and clinical attachment loss (CAL). Radiographic evaluation was also done. The results showed that the periodontal indices and radiographic measurements were improved in both groups after 6 months intervals favoring the omega-3 group. It can be concluded that the use of omega-3 is a beneficial nutritional supplementation in management of chronic periodontitis.

[Mohamed A. Galal, Basma M. Zaki and Nermine Nassif. **The Effect of Omega-3 Fatty Acids on Chronic Periodontitis in Postmenopausal Osteoporotic Females.** *Life Sci J* 2014;11(11):1104-1112]. (ISSN: 1097-8135).http://www.lifesciencesite.com. 188

Keywords: osteoporosis, periodontitis, omega-3, rutin, vitamin C

1. Introduction

Periodontal disease is recognized as a major public health problem worldwide and is the most common cause of tooth loss among adults. Periodontal disease is a general term used to describe several pathological conditions that affect the supporting structures/tissues of the teeth **(Abdellouni *et al.*, 2000)**.

Pain, discomfort and cosmetic considerations are some of the factors that demonstrate severity of the problems associated with dental diseases and hence, it is of utmost importance to minimize and control these diseases.

Periodontal treatment aims to cure inflamed tissue, reduce the number of pathogenic bacteria and eliminate the diseased pockets. Conventional therapy includes scaling- removal of the calculus and the plaque, curettage clearing the inflamed soft tissues, and root planing- removal of necrotic tissues on the root surface. In addition to the mechanical therapy, chemotherapy and systemic administration of antibiotics are some of the clinical methods that have been also utilized **(Friedman and Golomb, 1982)**.

Osteopenia and Osteoporosis are systemic skeletal disorders characterized by compromised bone strength and mass, with a consequent increase in bone fragility and susceptibility to fracture **(Koduganti *et al.*, 2009).**

Osteoporosis occurs mainly in postmenopausal females; although younger women and men can be also affected. It was estimated that one in four women during menopause and one in three women > 65 years of age are affected by osteoporosis **(Passos *et al.*, 2010).**

The evaluation of the relationship between osteoporosis and periodontitis is complicated by the fact that both diseases are multifactorial in the etiology. Multiple systemic factors influence the progression of osteoporosis, including age, race, diet, gender, hormone therapy, smoking, genetic factors, exercise, and body weight. Several of these factors are also risk factors for periodontal disease **(Al Habashneh *et al.*, 2010).**

Several researches support the idea that osteoporosis independently influences alveolar bone loss. Strategies for reducing osteoporosis risk also may help retard alveolar bone loss, which can be easily fulfilled by meeting dietary intake recommendations **(Kaye, 2007; Koduganti *et al.*, 2009 and Otomo-Corgel *et al.*, 2012).**

Omega-3 fatty acids (FA) are a subset of polyunsaturated fatty acids found in marine sources as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) and in some leafy vegetables, nuts, and oils as α-linolenic acid (ALA) **(DeFilippis & Sperling, 2006).**

Other nutritional supplements are also beneficial to periodontal health and affects bone density as well. Rutin and vitamin C are a common and low-priced combination. Rutin (RT), a quercetin-3-rutinosid or vitamin-P, is considered as one of flavonoid

glycosides, which is found in onions, apples, tea and red wine. Rutin is well known to exhibit multiple pharmacological activities including antibacterial, antitumor, anti-inflammatory, anti-diarrheal, antiulcer, anti-mutagenic, vasodilator and immunomodulatory **(Alsaif, 2009).** Furthermore, rutin showed an inhibitory effect against membrane lipid peroxidation, as well as antioxidant activities which suggest its protective role in oxidative stress-mediated diseases. Vitamin C is a water-soluble enzyme, abundantly present in different plants and some animals. Ascorbic acid is the most predominant form of vitamin C in the human body and is involved in tissue growth and repair. Ascorbic acid has a potent antioxidant activity and is well known to protect tissues from oxidative injury through efficiently quenching the damaging free radicals produced by many biological processes **(Al-Rejaie *et al.*, 2012 and Zhu *et al.*, 2012).**

It was proven that periodontitis is associated with a low concentration of vitamin C in plasma **(Pussinen *et al.*, 2003).**

Few strategies have been developed linking dietary intake and periodontitis. Dietary influences are important avenues for research because of the wide variability in dietary practices, vast research linking diet to periodontitis and osteoporosis, and the broad public health implications. The aim of the present study is to assess the effect of omega-3 fatty acids in management of osteoporotic females with chronic periodontitis.

2. Subjects and Methods

Study Design

The research was conducted on a selected group of postmenopausal women (≥ 50 years of age) who attended the outpatient clinic in the medical unit of the National Research Center (NRC), Cairo, Egypt for bone densitometry testing in the routine yearly check up. Only women who experienced natural menopause (no menstruation for at least one year) were chosen and invited to participate in this study. They received systemic bone mineral density (BMD) assessment as an initial screening using dual energy x-ray absorptiometry (DXA) of the hip (Norland XR46 version 3.9.6). Systemic BMD was classified according to the WHO criteria, where osteoporosis was defined as BMD ≥ 2.5 SDs (standard deviation) below the optimal mean BMD of young healthy individuals of the same race and gender. Only postmenopausal females with BMD T-score less than -2.5 SD, where T-score is the expression of BMD values in terms of standard deviations from the normal value of a female young adult mean were included in this study. Women with a history of a systemic condition or medication intake that might influence the BMD or periodontal disease severity were excluded (i.e., women with a history of diabetes mellitus, thyroid diseases, chronic renal problems, and connective tissue diseases). Postmenopausal females on corticosteroids, chemotherapy, recent peptic or esophageal disorders were also excluded.

Exclusion criteria also included postmenopausal females treated with drugs that inhibit gastric acid secretion for more than 2 weeks in the last 6 months; chronic treatment with non-steroidal anti-inflammatory drugs (NSAIDs), hormone replacement therapy or any other drug known to alter bone calcium metabolism were not included. Smoking females were not allowed to participate in the study. All patients were systemically reviewed. The participants were chosen to be of the same socio-economic level. The postmenopausal osteoporotic females who accepted the participation in the study were then referred to the dental clinic at the NRC medical services unit and to the dental clinic at the Faculty of Oral and Dental Medicine, Department of Oral Medicine and Periodontology, Cairo University after making prior appointments for evaluation of their oral condition. All participants received further information about the study protocol and objectives at the dental clinics.

Participants diagnosed as having chronic periodontitis according to the criteria of the **American Academy of Periodontology (2000)** were chosen. Each patient presented with probing depth (PD) ≥ 5 mm in at least three teeth or periodontal attachment level (PAL) ≥ 4 to 6 mm and vertical bone loss ≥ 3 mm with no history of periodontal therapy or use of antibiotics in the preceding 6 months was selected to be part of the study.

The study included forty non-smoking females, 50-65 years old, who were at least one year postmenopausal, osteoporotic and have not undergone hysterectomy or ovariectomy. All patients were also diagnosed as having chronic periodontitis. Subjects were age-matched into 2 groups of 20 patients each; Group (1) which included 20 osteoporotic postmenopausal females diagnosed as having chronic periodontitis. The patients in this group received rutin and vitamin C (50 mg rutin + 100 mg vitamin C) once daily for 6 months for the treatment of osteoporosis as described in accordance with their physician. The second group (the omega-3 group, n=20) were given 1gm omega-3 daily in addition to 50 mg rutin + 100 mg vitamin C once daily too.

For both groups detailed oral hygiene instructions were given and full mouth scaling and root planing using ultrasonic scalers and periodontal curettes under local anesthesia was completed. Scaling and root planing was performed for each patient in two sessions, one session for each half and completed over one week. Occlusal adjustment was done whenever indicated. Patients were seen every 3

weeks for 6 months at which time the teeth were polished and the oral hygiene instructions were reviewed. The participating females were informed about the nature, objectives, and possible risks of the study, and they signed informed consent statement that authorized their inclusion in the study.

Clinical Study

Medication compliance was assessed at each visit by counting the tablets remaining in the blister packs. During each visit, bacterial plaque (BP) was assessed using plaque detection tablets and flossing technique was reviewed. During the study period, all patients within the same group received identical periodontal assessment and treatment.

Periodontal Assessment

Periodontal assessment was carried out at baseline before starting the initial therapy (scaling and root planing) and at the end of the study period (6 months). At the baseline evaluation, all clinical parameters were measured and mechanical treatment including removal of all supra and subgingival calcified deposits to obtain a smooth, hard tooth surface was done. Scaling and root planing was carried out by one of the investigators in two successive sessions. Patients were taught and encouraged to maintain their dental health and plaque control through brushing and flossing. All assessment measurements were taken by the same investigator. The condition of all teeth was assessed and recorded. The mean was taken for the following measurements: whole mouth probing depth and clinical attachment level. Pocket depth (PD) was measured in millimeters according to the standard procedure described by **Glavind and Loe, 1967** using a periodontal probe with Williams' calibrations at the free gingival margin and recorded at six locations (mesiobuccal, distobuccal, midbuccal, mesiolingual, distolingual and midlingual) on each tooth parallel to the long axis of the examined tooth. The distance from the cemento-enamel junction (CEJ) to the free gingival margin and the distance from the free gingival margin to the bottom of the pocket/sulcus (PD) were measured at the mesiobuccal and mid-buccal surfaces using also a calibrated probe. From these two measurements, individual subject mean attachment level (the distance from the CEJ to the bottom of the pocket or sulcus) was calculated in millimeters. All the measurements were taken pre and post treatment (after 6 months).

Radiographic parameters:

Bone mineral density (BMD) was measured for each patient by Dual-energy X-ray absorptiometry (DXA) of the hip using Norland XR46 version 3.9.6 for diagnosis of osteoporosis. Standardized intraoral periapical radiographs using the paralleling technique were taken at baseline and 6 months using Trophy x-ray machine with exposure parameters of 60 KVP, 10 mA and 0.14 sec (Trophy radiology, 94300 Vincennes, type 6510, made in France). The paralleling technique was used with Rinn XCP film holder (KKD Germany), which consists of interchangeable acrylic bite blocks, a plastic aiming ring and a metallic indicator arm. For each patient an occlusal stent was constructed to confirm reproducibility and standardization of the technique. The alveolar bone changes were measured from the radiographs using the measurement system of the Digora software (Orion Corporation, Sordex Medical System, Finland).

1- Linear Measurements calculated in millimeters (mm):

a) A line was drawn from C.E.J to the alveolar crest in each defect parallel to the long axis of the studied tooth at the most radiographically accentuated point (Distance A).

The same investigator performed the measurements twice and the mean of both trials was calculated as an attempt to eliminate intra-observer errors.

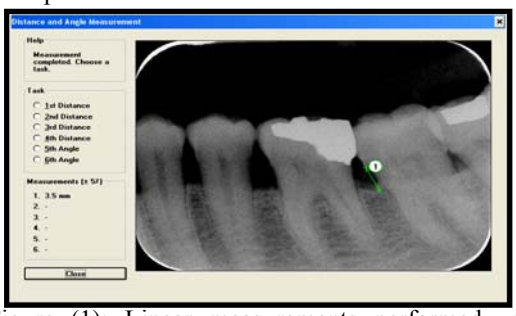

Figure (1): Linear measurements performed using Digora software.

2- Area Density Measurements (gram /cubic centimeter):

For performing standardized area density analysis, 3 successive straight lines were drawn each 1 mm apart and parallel to each other and to the root surface reaching the most radiographically accentuated points.

a) The first area was measured from C.E.J level on alveolar bone to the alveolar crest in each defect parallel to the long axis of the studied tooth (Density B).

b) The second area was measured from the alveolar crest to the apex parallel to the long axis of each studied tooth (Density C).

c) The third area was measured from C.E.J level on alveolar bone to the apex parallel to the long axis of each studied tooth (Density D). The mean value of each area was measured and their means were calculated. The same investigator performed all the measurements for each area twice and the mean of the trials was calculated as an attempt to eliminate intra-observer errors.

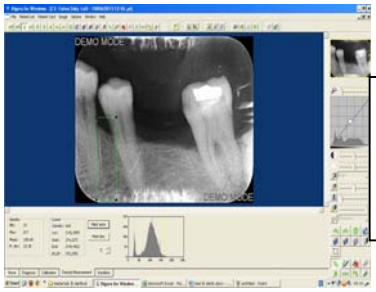

Figure (2): Area density measurement using Digora software.

3- Radiographic defect angle measurements (degree):

The radiographic defect angle was calculated and defined by two lines that represented the root surface and the bone-defect surface. The defect angle is the angle between the two lines one drawn from CEJ to the bone defect and the other one is drawn from the bone defect to the alveolar bone crest at the most radiographically accentuated point **(Fernanda *et al.*, 2011).**

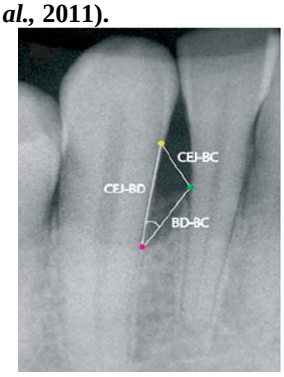

Figure (3): Anatomic landmarks used in the radiographic analyses of intrabony defects. The defect angle was defined by the two lines CEJ–BD (base of the defect) and BD–BC (coronal position of alveolar bone crest).

Statistical Analysis

Descriptive statistics including the mean ± SD (standard deviation) were calculated. Also the p-value was measured. The paired t- test was used to compare pre and post treatment values. The independent t- test was used to compare between the two groups. The significant level was set at $p \leq 0.05$ **(Dawson and Trapp 2001).** Statistical analysis was performed with SPSS 16.0 (Statistical Package for Scientific Studies) for Windows (SPSS, Inc., Chicago, IL, USA).

3. Results

A) Periodontal Assessment:

1- Pocket depth (PD) measurements revealed that there was no statistically significant difference between the two groups before treatment. After treatment both groups showed statistically significant reduction of the PD measurements as presented in table (1).

2- Clinical attachment level (CAL) measurements revealed that there was no statistically significant difference between the two groups before treatment. After treatment there was statistically significant gain of the clinical attachment level after treatment as also shown in table (1).

Table (1) mean ± SD of PD and CAL measurements of both groups I and II before and after treatment (in mm).

PD measurements				CAL measurements			
Group I		Group II		Group I		Group II	
Mean ± SD		Mean ± SD		Mean ± SD		Mean ± SD	
Before	After	Before	After	Before	After	Before	After
6.40 ± 1.3	5.20 ± 1.4	6.44 ± 1.4	4.50 ± 1.2*	5.60 ± 1.2	4.50 ± 1.3	5.62 ± 0.8	4.22 ± 0.7*

*Statistically significant results as *p*-value 0.001. PD: pocket depth; CAL: clinical attachment level

B) Radiographic assessment:

1) Linear measurements of group I and II before and after treatment:

Table (2) demonstrates the linear measurements (Distance A) before and after treatment for both groups I and II. There was a statistically significant difference in both groups before and after treatment with a *p*-value of (0.0001) for both measurements.

Table (2) mean ± SD of radiographic linear measurements before and after treatment for both groups (in mm).

Group I		Group II	
(Distance A)		(Distance A)	
Before	After	Before	After
4 ± 0.94	3.62 ± 0.95*	4.18 ± 1.15	3.02 ± 0.78*

*Statistically significant results as *p*-value 0.001.

2) Comparison between group I and II in the linear measurements (Distance A) revealed that there was no significant difference in pre treatment values as the *p*-value was (0.59), while there was a significant difference in the post treatment values where *p*-value was (0.03) in group I. While for group II there was also no significant difference in pre treatment values as the *p*-value was (0.74), while there was a significant difference in the post treatment values where *p*-value was (0.01).

3) Density measurements of groups I and II:

In group I there was no significant difference between pre and post treatment values as *p*-value was (0.96) for the (B) measurements. Also for the (C) measurements as *p*-value was (0.62). In addition there was no significant difference between before and post treatment values as *p*-value (0.78) for the (D) measurements.

There was a significant difference between before and after treatment values in group II as *p*-value was (0.0001) for all the measurements (B), (C) and (D).

Tables (3) mean ± SD of alveolar bone density measurements at areas (B, C and D) before and after treatment for groups I and II.

	Alveolar Bone Density Measurements			
	Group I		Group II	
	Before	After	Before	After
Areas				
B	38.3 ± 13.41	38.47 ± 16.21	34.33 ± 16.06	51.24 ± 17.12*
C	122.45 ± 31.0	126.24 ± 30.75	119.07 ± 16.91	143.21 ± 14.57*
D	108.2 ± 22.8	109.98 ± 26.75	107.3 ± 9.82	125.14 ± 14.19*

*Statistically significant results as *p*-value 0.0001.

4- Comparison between the density measurements of groups I and II that there was no significant difference in pre treatment values where *p*-value was 0.67, while there was a significant difference in the post treatment values of (B), (C) and (D) measurements where p-value was 0.03.

5- Defect angle measurements (in degrees):

There was a significant difference between pre and post treatment values as p-value was (0.04) in group I and p-value was (0.00001) in group II as shown in table (4).

Table (4): mean ± SD of defect angles measurements before and after treatment for groups I and II.

Group I		Group II	
Mean ± SD		Mean ± SD	
Before	After	Before	After
59.79 ± 12.53	65.44 ± 12.63*	58.4 ± 9.01	74.36 ± 9.21*

*Statistically significant results.

6- Comparison between the defect angle measurements in groups I and II reveals that there was no significant difference in the before treatment values where the *p*-value was (0.68), while there was a significant difference in the post treatment values where *p*-value was (0.01).

Table (5) percentages of improvement of all the measurements in both groups after treatment.

Group	Group I	Group II
Measurements		
PD	18.75 %	30.12 %
CAL	19.64 %	24.91 %
Linear (Distance A)	9.25 %	27.75 %
Alveolar Bone Density (B)	0.41 %	49.22 %
Alveolar Bone Density (C)	3.09 %	20.26 %
Alveolar bone Density (D)	1.63 %	21.41 %
Defect angle	9.43 %	27.32 %

From table (5) all the clinical and radiographic measurements proved that group II showed higher percentages of improvement than group I

4. Discussion

Osteoporosis is often called the "silent disease" because bone loss occurs without symptoms. People are not aware that they have osteoporosis until their bones become so weak that a sudden strain, bump or fall causes a fracture or a vertebra to collapse

(Ghozlani *et al.*, 2009). Osteoporosis is one of the risk factors that have been implicated in the progression of periodontitis **(Graves & Cochran, 2003).** A number of studies showed that there is a relationship between oral and systemic bone loss as well as an association of osteoporosis with periodontal diseases **(Brennan *et al.*, 2007 and Nackaerts *et al.*, 2008).**

Understanding the association between periodontal disease and osteoporosis and the mechanisms underlying this association may aid health care professionals in the prevention, early detection, and treatment of these common diseases **(Shum *et al.*, 2010).**

The use of different nutritional supplements was discussed in management of both diseases. This study was conducted in an attempt to throw some light on easy, affordable and economic nutritional supplementations as omega-3, rutin and vitamin C as an adjunctive treatment modality in management of chronic periodontitis in postmenopausal osteoporotic females. The dose used in this study was selected in accordance with the recommendation of **Saravanan *et al.*, 2010** in a large intervention trial of secondary prevention after myocardial infarction. 1 g per day of polyunsaturated fatty acids was used with the results revealing reduction in all cause and cardiovascular mortality.

Salari *et al.*, 2010 and McMahon, 2012 evaluated the effect of omega-3 FA on bone biomarkers in osteoporotic postmenopausal women ingesting 900 mg of omega-3 FA per day for 6 months showing positive results recommending the use of omega-3. Also **Tartibian *et al.*, 2011** examining the effects of long-term aerobic exercise and omega-3 supplementation on serum inflammatory markers, bone mineral density (BMD), and bone biomarkers in postmenopausal women recommended the use of 1000 mg/day of omega-3 FA for 24 weeks. This is in similarity to the dose used in our current study. Several researchers also suggested the increase of this dose especially in chronic periodontitis; as **Martinez *et al.*, 2013** who constructed their study by selecting a test group composed of 10 patients with generalized chronic periodontitis (mean age 44 ± 6.4 years) treated with scaling and root planing associated with 4 months of omega-3 supplementation eicosapentaenoic acid (EPA) + docosahexaenoic acid (DHA), 3 g/d. Our study proved the effectiveness of improving the PD and CAL in both groups especially in group II recording the highest improvements.

Rosenstein *et al.*, 2003 on the other hand made a clinical trial on thirty adult human subjects with periodontitis who were administered either fish oil 3000 mg daily; borage oil 3000 mg daily; fish oil 1500 and borage oil 1500 mg daily, or placebo. The gingival index (GI), the plaque index (PI), periodontal probing depths (PD) and beta-glucuronidase levels in gingival crevicular fluid were measured at baseline and after 12 weeks of treatment proving the effectiveness of using these supplementations which in line with our study.

Rutin-C (rutin 50 mg + vitamin C 100 mg) was applied in our work for the control group. Rutin is a common medication for decreasing capillaries fragility and gingivitis in the dental field. Ascorbic acid is the active form of vitamin C with good properties on improving gingival tissue health. **McAnulty *et al.*, 2011** recommended its use in a study which examined the effects of 1,000 mg quercetin + 1,000 mg vitamin C, and 400 mg omega-3 FA; taken each day for 2 weeks.

The measurement of pocket depth and clinical attachment level are established in the literature for diagnosing existing periodontal disease, determine the prognosis of individual teeth, and monitor disease progression that tends to be episodic and specific to the tooth and site **(Snider, 2010).**

Hence, in our study we performed a comprehensive clinical examination in addition to radiographic evaluation to assure accuracy of results in proving the outcome of the proposed medication.

Many experimental studies were done to detect Omega-3 effect on periodontitis and alveolar bone loss. Dietary supplementation with high dose docosahexanoic acid tuna oil caused a marked elevation of Omega-3 FA levels in oral soft tissues. This diet reduced the amount of alveolar bone loss in a murine experimental periodontitis model compared to a control diet containing no Omega-3 FAs (by an average of 54-72% less alveolar bone resorption in response to the different bacterial infections was detected) **(Bendyk, 2008).**

Indahyani *et al.*, 2008, tested the hypothesis that fish oil alters lipopolysaccharide (LPS)-induced hydroxyapatite loss in rat alveolar bone, and found that the hydroxyapatite contents of alveolar bone in rats treated with fish oil at the same day with or before LPS injection were significantly higher than those in rats injected with LPS alone, but still lower than those in untreated animals .

Kesavalu *et al.*, 2006 have done a study on rats. Rats were fed fish oil (Omega-3 FA) or corn oil diets for 22 weeks and were infected with Pg (*Porphyromonas Gingivalis*). Rats on the Omega-3 FA diet exhibited elevated serum levels of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), documenting diet-induced changes. Pg-infected rats treated with Omega-3 FA had significantly less alveolar bone resorption. These results demonstrated the effectiveness of an Omega-3 FA-supplemented diet in modulating alveolar bone

resorption following Pg infection, and supported that Omega-3 FA may be a useful adjunct in the treatment of periodontal disease which is in accordance with our study .

These experimental animal studies are in accordance with our results that proved the improvement that occurred both clinically and radiographically especially in group II after using omega-3 FA in chronic periodontitis patients.

Dietary human surveys were done to confirm this relation. To date, the treatment of periodontitis has primarily involved mechanical cleaning and local antibiotic application. Thus, a dietary therapy, if effective, might be a less expensive and safer method for the prevention and treatment of periodontitis **(Naqvi *et al.*, 2010).**

Using data from the National Health and Nutrition Examination Survey (NHANES), investigators from Harvard Medical School and Harvard School of Public Health, Boston, found that dietary intake of docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) was associated with a decreased prevalence of periodontitis (by 20%), but linolenic acid (LNA) did not exhibit this association. The cross-sectional study involved more than 9,000 adults between 1999 and 2004 **(Snider, 2010).**

Iwasaki *et al.*, 2011 studied the longitudinal relationship between two types of Omega-3 dietary intake in presence of periodontal disease in 235 Japanese subjects for whom data were available for the years 2003–2006. Omega-3 intake was assessed at baseline with a brief-type self-administered diet history questionnaire. Full-mouth periodontal status, measured as the clinical attachment level (CAL), was recorded at baseline and once a year for 3 years showing the importance of this nutritional supplementation.

On the other hand in a very recent study by **Martinez *et al.*, 2013** who constructed the methodology of their study that the test group was composed of 10 patients with generalized chronic periodontitis (mean age 44 ± 6.4 years) treated with scaling and root planing associated with 4 months of Omega-3 supplementation as eicosapetaenoic acid (EPA) plus docosahexaenoic acid (DHA), 3 g/d. The placebo group was composed of 11 patients (47.9 ± 10.5 years) that received scaling and root planing plus placebo. The periodontal examination included probing depth, clinical attachment level, bleeding on probing and plaque index. They presented that the Omega-3 dietary supplementation had no effect on clinical outcome of treatment.

In our study there was a significant improvement in both clinically and radiographically measured parameters favoring the omega-3 group after the 6 months study period.

Also in contrary to our results, **Rosenstein *et al.*, 2003** performed a clinical trial on thirty adult human subjects with periodontitis who were administered either fish oil 3000 mg daily; borage oil 3000 mg daily; fish oil 1500 and borage oil 1500 mg daily, or placebo. The modified gingival index (GI), the plaque index (PI), periodontal PD and beta-glucuronidase levels in gingival crevicular fluid were measured at baseline and after 12 weeks of treatment. Improvement in gingival inflammation was observed in subjects treated with borage oil, with a trend apparent in subjects treated with fish oil or a combination of both. There was no statistically significant improvement in PI, although a trend was apparent in those receiving borage oil. Improvement in PD was seen in those subjects treated with either fish oil alone or borage oil alone, but statistical significance was only seen for the comparison of borage oil and placebo.

Other study combined omega-3 with other NSAIDs, for antagonism as the study done by **El-Sharkawy *et al.*, 2010** who tried to test their innovative strategy for periodontal treatment using Omega-3 and low dose aspirin. Eighty healthy subjects with advanced chronic periodontitis were enrolled in Mansoura, Egypt, in a parallel-design, double-masked clinical study. The control group was treated with (SRP) and a placebo, whereas the Omega-3 group was treated with SRP followed by dietary supplementation of fish oil (900 mg EPA + DHA) and 81 mg aspirin daily. Statistical analysis demonstrated a significant reduction in PD and a significant attachment gain after 3 and 6 months in the Omega-3 group compared to baseline and the control group. The results of this preliminary clinical study suggested that dietary supplementation with Omega-3 FAs and 81 mg aspirin may provide a sustainable, low-cost intervention to augment periodontal therapy.

Vardar *et al.*, 2005 used another NSAID; i.e. celecoxib. The results of the that study indicated that celecoxib and omega-3 fatty acid, when used individually, show a rather partial effect on the control of the analyzed mediators, but when combined they show a synergic effect and provide significant reductions in the gingival tissue levels of prostaglandin E2 (PGE2), prostaglandin F2α (PGF2α), leukotriene B4 (LTB4), and platelet activating factor (PAF) in LPS-induced experimental periodontitis (Experimental periodontitis was induced by repeated injection of Escherichia coli endotoxin). These findings may pioneer further clinical human studies investigating the possible place of celecoxib and omega-3 fatty acid in periodontal treatment.

Still we need confirmation about the effect of these dietary supplementations from human clinical

trials which are very little in the literature; up to our knowledge .

Conclusion

Our study results proved that omega-3 supplemental medications have a positive impact on management of chronic periodontitis.

Many of the studies focused on cellular factors more than clinical indices and radiographic measurements, which makes omega-3 in such ways preventive more than being therapeutic, rendering it of low grade importance in periodontitis if compared to antibiotics. Thus further investigations are needed to prove the effectiveness of omega-3 FA on alveolar bone health.

Corresponding author:
Basma Mostafa Zaki,
Assistant Professor of Oral Medicine and Periodontology, Surgery and Oral Medicine Dep., Oral and Dental Research Division, National Research Center, 33 El Bohouth Street, 12311, Cairo, Egypt.
Fax:
E-mail: boshta@hotmail.com

References

Al Habashneh R., Alchalabi H., Khader Y. S., Hazza'a A.M., Odat Z, and Johnson G.K. Association between Periodontal Disease and Osteoporosis in Postmenopausal Women in Jordan. J Periodontol, (2010); 81:1613-1621.

Al-Rejaie S.S., Abuohashish H.M., Alkhamees O.A., Aleisa A.M. and Alroujayee A.S. Gender difference following high cholesterol diet induced renal injury and the protective role of rutin and ascorbic acid combination in Wistar albino rats. Lipids in Health and Disease 2012, 11: 41-45.

Alsaif, M.A. Beneficial Effects of Rutin and Vitamin C Co-Administration in a Streptozotocin-Induced Diabetes Rat Model of Kidney Nephrotoxicity. Pakistan Journal of Nutrition, (2009), 8, 745-754.

American Academy of Periodontology, (2000). Parameter on chronic periodontitis. J Periodontol.; 71: 853-855.

Bartzela T., Türp J.C., Motschall E. and Maltha J.C. Medication effects on the rate of orthodontic tooth movement: A systematic literature review. Am J Orthod Dentofacial Orthop, (2009); 135(1):16-26.

Bendyk A. The effect of dietary Omega-3 polyunsaturated fatty acids on experimental periodontitis lesions in the mouse. Doctorate Thesis of clinical dentistry (Periodontics) submitted to the University of Adelaide. Australia. 2008.

Brennan R. M., Genco R. J., Hovey K. M., Trevisan M.and Jean Wende J.W. Clinical Attachment Loss, Systemic Bone Density, and Subgingival Calculus in Postmenopausal Women. J Periodontol, (2007); 78: 2104-2111.

Dawson B. and Trapp R.G. Basic and Clinical Biostatistics, New York: Lange Medical Books-McGraw Hill, (2001).

DeFilippis AP and Sperling LS. Understanding omega-3's.Am Heart J (2006); 151(3):564- 70.

El-Sharkawy H., Aboelsaad N., Eliwa M., Darweesh M., Alshahat M., Kantarci A., Hasturk H. and Van Dyke T.E. Adjunctive Treatment of Chronic Periodontitis with Daily Dietary Supplementation with Omega-3 Fatty Acids and Low-Dose Aspirin. Journal of Periodontol, (2010): Vol. 81, no. 11: 1635-1643.

Fernanda V. R., Renato C.V. C., Francisco H.N. J., Enilson A. S. and
Ma´ rcio Z. C., (2011). The Role of Enamel Matrix Derivative Protein in Minimally Invasive Surgery in Treating Intrabony Defects in Single-Rooted Teeth: A Randomized Clinical Trial. J Periodontol; 82:522-532.

Friedman M. and Golomb G. New sustained release dosage form of chlorhexidine for dental use. J Periodontal Res, (1982); 17: 323-328.

Ghozlani I., Ghazi M., Nouijai A., Mounach A.,Achemlal R. A., Bezza A. and El Maghraoui A., (2009). Prevalence and risk factors of osteoporosis and vertebral fractures in patients with ankylosing spondylitis. Bone, 44(5): 772-776.

Glavind L. and Löe H.. Errors in the clinical assessment of periodontal destruction. J Periodont Res., (1967); 2: 180-188.

Graves D.T. and Cochran D. The contribution of interleukin-1 and tumor necrosis factor to periodontal destruction. J Periodontol, (2003); 74: 391-401.

Hughes P. Aging, Systemic Disease and Oral Health: Implications for Women Worldwide (Part I). Crest® Oral-B® at dentalcare.com Continuing Education Course, (2010).

Indahyani D.E., Santoso A.L.S., Utoro T., Soesatyo M.H. and Sosroseno W. Effect of fish oil on lipopolysaccharideinduced hydroxyapatite loss in rat alveolar bone: A Preliminary Study. Online J Health Allied Scs, (2008); 7(4): 7-13.

Iwasaki M., Taylor G.W., Moynihan P., Yoshihara A., Muramatsu K., Watanabe R. and Miyazaki H. Dietary ratio of n-6 to n-3 polyunsaturated fatty acids and periodontal disease in community-based older Japanese: A 3-year follow-up study.

Prostaglandins, Leukotrienes and Essential Fatty Acids, (2011); 85:107–112.

Kaye EK. Bone health and oral health. J Am Dent Assoc, (2007); 138(5): 616-9.

Kesavalu L., Vasudevan B., Raghu B., Browning E., Dawson D., Novak J.M., Correll M.C., Steffen M.J., Bhattacharya A., Fernandes G. and Ebersole J.L. Omega-3 Fatty Acid Effect on Alveolar Bone Loss in Rats. J Dent Res, (2006); 85(7): 648-52.

Koduganti R.R., Gorthi C., Reddy P.V. and Sandeep N. Osteoporosis: "A risk factor for periodontitis". J Indian Soc Periodontol, (2009); 13(2): 90-6.

Ling-Ling Zhu, Jay Ca, Merry Sun, Tony Yuen, Raymond Zhou , et al., (2012). Vitamin C Prevents Hypogonadal Bone Loss. PLOS ONE ; Volume 7, Issue 10 : 47-58.

Martinez G.L., Koury J.C., Brito F., Fischer R.G., Gustafsson A. and Figueredo C.M. The impact of non-surgical periodontal treatment on serum levels of long chain-polyunsaturated fatty acids: a pilot randomized clinical trial. J Periodontal Res, (2013); 31: 73-79.

McAnulty S.R., Nieman D.C., McAnulty L.S., Lynch W.S, Jin F. and Henson D.A. Effect of mixed flavonoids, n-3 fatty acids, and vitamin C on oxidative stress and antioxidant capacity before and after intense cycling. International Journal of Sport Nutrition and Exercise Metabolism, (2011), 21(4): 328-337.

McMahon M.S. Beneficial Effect of Omega-3 Fatty Acids on Bone Metabolism, (2012), Vol. 35, no. 9: 735 -6.

Nackaerts O., Frieda G., Anna-Maria S. and Reinhilde J. Is there a relation between local bone quality as assessed on panoramic radiographs and alveolar bone level? Clin Oral Invest, (2008); 12: 31–35.

Naqvi A.Z., Buettner C., Phillips R.S., Davis R.B. and Mukamal K.J. N-3 Fatty Acids and Periodontitis in US Adults. J Am Diet Assoc, (2010); 110:1669-1675.

Otomo-Corgel J., Pucher J.J., Rethman M.P., Reynolds M.A.. State of the Science: Chronic Periodontitis and Systemic Health. J Evid Base Dent Pract, (2012); S1: 20-28.

Passos J. D., Gomes-Filho I. S., Vianna M. I. P., Da Cruz S.S., Barreto M. L., Oliveira T. J., Borges L. D., and Monteiroi F.M. Outcome Measurements in Studies on the Association between Osteoporosis and Periodontal Disease. J Periodontol, (2010); 81:1773-1780.

Pussinen P.J., Laatikainen T., Alfthan G., Asikainen S. and Jousilahti P. Periodontitis Is Associated with a Low Concentration of Vitamin C in Plasma. Clinical and Diagnostic Laboratory Immunology, (2003); vol. 10, no. 5: 897–902.

Rosenstein E.D., Kushner L.J., Kramer N., Kazandjian G. Pilot study of dietary fatty acid supplementation in the treatment of adult periodontitis. Prostaglandins Leukot. Essent Fatty Acids, (2003); 68(3): 213-8.

Salari Sharif P., Asalforoush M., Ameri F., Larijani B., Abdollahi M. The effect of n-3 fatty acids on bone biomarkers in Iranian postmenopausal osteoporotic women: a randomized clinical trial. Age (Dordr), (2010); 32, (2): 179-186.

Saravanan P., Davidson N.C., Schmidt E.B., Calder P.C. Cardiovascular effects of marine omega-3 fatty acids. Lancet, (2010); 376(9740): 540-50.

Schwach-Abdellouni K., Vivien-Castoni N. and Gurny R. Local delivery of antimicrobial agents for the treatment of periodontal diseases. Eur J Pharm Biopharm,(2000); 50: 83-99.

Shum I., Leung P.C., Kwok A., Corbet E. F., Orwoll E. S., Phipps K.R., and Jin L. Periodontal Conditions in Elderly Men with and without Osteoporosis or Osteopenia. J Periodontol, (2010); 81:1396-1402.

Snider J. Polyunsaturated fatty acids may help treat, prevent periodontitis, study finds. JADA, (2010); 141(12): 1418- 22.

Tartibian B., Maleki B.H., Kanaley J., Sadeghi K. Long-term aerobic exercise and omega-3 supplementation modulate osteoporosis through inflammatory mechanisms in post-menopausal women: a randomized, repeated measures study. Nutrition & Metabolism, (2011), 8:71-7.

Vanlint S.J. and Ried K. Efficacy and tolerability of calcium, vitamin D and a plant-based omega-3 oil for osteopenia: A pilot RCT. Maturitas, (2012); 71:44-8.

Vardar S., Buduneli E., Baylas H., Berdeli A.H., Buduneli N. and Atilla G. Individual and Combined Effects of Selective Cyclooxygenase-2 Inhibitor and Omega-3 Fatty Acid on Endotoxin-Induced Periodontitis in Rats. J Periodontol, (2005); 76:99-106.

2014/10/11