

Research Article

Effect of melatonin supplementation on the gingival health and lipid profiles in obese periodontitis patients

Hussam Sami Ismail ¹, Maha Sh. Mahmood ^{2,*}

¹ Master student, B.D.S, Department of Periodontics, College of Dentistry, University of Baghdad

² Professor, B.D.S., M.Sc. Department of Periodontics, College of Dentistry, University of Baghdad.

* Correspondence: maha.shukri@yahoo.com

Received date :1-11-2021

Accepted date :7-12-2021

Published date: 15-3-2022



Copyright: © 2022 by the authors. Submitted for possible open access publication under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

<https://doi.org/10.26477/jbcd.v34i1.3092>

Abstract: Background: Obesity increases the host's susceptibility by modulating the immune and inflammatory systems in a manner that predisposes to inflammatory tissue destruction and leaves an individual at greater risk of periodontitis. Melatonin is a pineal secretory product involved in numerous actions, such as regulation of internal biological clocks and energy metabolism, and it functions as an antioxidant and anti-inflammatory agent. There exists a substantial amount of evidence supporting the beneficial effect of melatonin supplementation on obesity and its complications. Aim of the study: To investigate the effects of systemic melatonin intake on periodontal health status and lipid profiles in obese periodontitis patients. Subjects and methods: Subjects included in the study were distributed into the following groups: Group I, 20 subjects with normal weight and healthy periodontium (controls) not subjected to any treatment. Group II: 30 obese periodontitis patients subjected to scaling and root planing (SRP) only. Group III: 30 obese periodontitis patients subjected to SRP and supplemented with 5mg melatonin tablets for 1 month. Study groups subjected to estimation of plaque index (PLI), bleeding on probing (BOP), cholesterol (chol), triglycerides (TG), high-density lipoprotein (HDL) and low-density lipoprotein (LDL) at baseline and after 4 weeks recall visit. Results: Regarding the clinical parameters, the second visit exhibited decreasing in all parameters in both study groups except BOP score 0 were it increased significantly. Regarding lipid profiles, the second visit showed decreasing in all profiles except HDL where it increased in both study groups with a significant difference. All correlations between lipid profiles in recall visit in both study groups exhibited a positive significant correlation between chol and TG, chol and LDL, LDL and TG in group III. In group II all results exhibited a positive significant correlation, whereas the only strong negative correlation was found between chol and HDL, HDL and LDL. Conclusion: Daily supplementation with 5mg melatonin tab significantly improved periodontal health and reduced chol, TG, LDL with increasing HDL.

Keywords: melatonin, lipid profile, obese periodontitis patients.

Introduction

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth. The inflammatory condition is induced by microbial dysbiosis, resulting in progressive destruction of the periodontal ligament and alveolar bone ⁽¹⁾. Obesity increases the host's susceptibility by modulating the immune and inflammatory systems in a manner that predisposes to inflammatory tissue destruction and leaves an individual at greater risk of periodontitis ⁽²⁾. Obesity has been postulated to reduce blood flow to the periodontal tissues and promoting the development of periodontal disease ⁽³⁾. Periodontal blood vessels

in obese persons show a thickening in their innermost membrane, which indicates diminished blood flow in the periodontium ⁽⁴⁾. One of the reasons for the association between obesity and periodontal disease include the social stigma associated with obesity in younger adults. A greater source of chronic stress found in obese young individuals than in older adults is considered more acceptable. Stress and how an individual copes with stress have been shown to increase a person's risk for periodontal disease ⁽⁵⁾.

Using the classification and regression tree (CART) method, it has been suggested that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction ⁽⁶⁾. A study of the Fourth Korean National Health and Nutrition Examination Survey found that abdominal obesity is significantly correlated with periodontitis ⁽⁷⁾. Despite these common inflammatory-mediated mechanisms, the specific underlying biological pathways linking both diseases are not well characterized. Some authors have suggested the possible influence of alterations in the circadian cycle since significant reductions in the melatonin hormone levels were reported in experimental studies in both, obesity and periodontitis ⁽⁸⁾. In fact, when melatonin was administered in obese subjects, significant reductions in systemic pro-inflammatory biomarkers together with reductions in body weight and adipose tissue deposits were reported ⁽⁹⁾. Melatonin participated in homeostasis and metabolism of energy through activation of brown adipose tissue and enhance energy expenditure ⁽¹⁰⁾. Multiple studies reported that melatonin significantly increases HDL level and decrease TG and HDL level in addition to increased cholesterol catabolism ⁽¹¹⁾.

Materials and methods

This study was conducted in AL-Diwanyah dental specialized center at the Department of Periodontics, all subjects were collected from December 2019 to March 2020. This study approved by the ethical committee at the College of Dentistry, University of Baghdad, follow the roles of Tokyo and Helsinki for human (Reference no.128619 in 28/11/2019). The study sample consisted of 80 males and females individuals with age ranged between 24-55 years, all subjects enrolled voluntarily to the study after signing an informed consent sheet to participate in research. All subjects were submitted to a questionnaire including their name, age, total medical history, dental history, their BMI, if they were subjected to any diet regimen, sleep nature, smoking, drinking alcohol, followed by a complete examination of clinical periodontal parameters (PLI, BOP). The sample was divided into 3 groups according to their BMI, periodontal health status, and melatonin supplementation. Group I (20 subjects) (controls) was the normal weight group and healthy periodontium, their BMI was (18.50 - 24.99) this group presents at the baseline only for examination of clinical periodontal parameters (PLI, BOP), lipid profile (chol, TG, HDL, LDL), don't exposed to any periodontal therapy., Group II (30 patients) have obesity, (BMI was ≥ 30.00) ⁽¹²⁾ designed to have latest sleep, generalized stage III periodontitis don't follow any diet regimen. This group presents at baseline and after a one-month recall visit for determination of clinical periodontal parameters (PLI, BOP), lipid profiles (chol, TG, HDL, and LDL) in pre and post periodontal treatment with scaling and root planing only without melatonin supplementation. The third group (30 patients) have obesity (BMI was ≥ 30.00) designed to have latest sleep, generalized stage III periodontitis don't follow any diet regimen. This group presents at baseline and after a one-month recall visit for determination of clinical periodontal parameters (PLI, BOP), lipid profiles (chol, TG, HDL, LDL) in pre and post periodontal treatment with scaling and root planing with melatonin supplementation 5mg tablet for 1 month (NOW, USA). All individuals were instructed not to eat for at least 12 hr. before taking the sample. Blood samples (8ml) were drowned from antecubital vein by using disposable syringe size 10mm and pushed directly into the gel tube. The tube was centrifuged at 3000 rpm for 10min to obtain serum. The extracted serum was divided by using micropipette into plain tubes and put in Colling box to send into the laboratory were stored in - 80c in a deep freeze in AL-Diwanyah general medical laboratory. Following one month, all individuals (except controls) were returned to perform the same manner of collection. The second visit was determined after one month and involved only group II and group III. Clinical periodontal parameters involving (PLI, BOP,) was recorded and the same manner for blood collection and storing was performed for all patients.

After completion of the sample, the frozen serum was thawed at room temperature before analysis for determination of lipid profiles by using a spectrophotometer (Cecil instrument limited CE 7200, England). The participants were examined intraorally to determine the plaque index (PLI) and ⁽¹³⁾ Bleeding on probing (BOP) ⁽¹⁴⁾.

Results

All variables including clinical periodontal parameters (PLI, BOP) were tested for normality using the Shapiro-Wilk test at P value greater than 0.05 and they were normally distributed

Table 1: Intra- and Inter-comparisons of PLI among groups and visits using one way Analysis of Variance ANOVA, Paired sample t-test and Independent Sample t-test.

| | | PLI | | | | |
|----------------------|------|-------------------|-----------------|-------------|----------------|-------|
| Groups | | Base line 1 visit | Recall visit | Paired test | P value | ES |
| Control | Mean | 0.455 | | | | |
| | ±SD | 0.048 | | | | |
| Group II | Mean | 2.491 | 1.777 | 25.890 | 0.000** | 3.342 |
| | ±SD | 0.237 | 0.229 | | | |
| Group III | Mean | 2.556 | 1.564 | 30.233 | 0.000** | 3.903 |
| | ±SD | 0.171 | 0.173 | | | |
| Statistics (F or T) | | 978.536 | 4.074 | | | |
| df | | 2 | 58 | | | |
| P value | | 0.000 HS | 0.000 HS | | | |
| ES | | 0.962 | 1.050 | | | |

Table 2: Multiple Comparisons of PLI in the baseline visits between groups using Games-Howell.

| Dependent Variable | (I) groups | (J) groups | Mean Difference (I-J) | Sig. |
|--------------------|-------------------|-------------------|-----------------------|----------------|
| PLI | control | Without melatonin | -2.036 | .000 ** |
| | | With melatonin | -2.101 | .000 ** |
| | Without melatonin | With melatonin | -.065 | 0.449 |

The findings in tables 1 and 2 illustrated that PLI was found to be higher in group III than other groups followed by group II with least in the control one. Further analysis of multiple comparisons indicated that there is no significant difference between the two study groups while when compared each one with control, results were found to be a highly significant difference. Regarding the second visit, PLI appeared to be higher in group II than that of group III. There was a decrease in PLI (1.564±0.1) and (1.777±0.2) in group III and in group II respectively with a highly significant difference with more effect size and variability in group III than that of group II.

Table 3: Statistical test of BOP among groups and in each visit using independent sample T.

| Groups | | BOP1, baseline | BOP1, recall visit | BOP1 pre-BOP1 post , paired t-test | df | ES |
|-------------------|------|----------------|--------------------|------------------------------------|----|-------|
| Without melatonin | Mean | 63.267 | 41.60 | 12.619 | 29 | 1.629 |
| | ±SD | 6.443 | 8.27 | | | |
| With melatonin | Mean | 65.467 | 28.267 | 30.564 | 29 | 3.946 |
| | ±SD | 6.822 | 5.564 | | | |
| T | | 1.284 | 7.327 | | | |
| P value | | 0.204 | 0.000 HS | | | |

Findings in table 3 illustrated that in baseline visits BOP was found to be higher in group III than that of group II with no significant difference. Regarding the second visit, all results demonstrated highly significant differences between the two groups. The changes of BOP from baseline to recall visits indicated that there was decrease for BOP score 1 with more effect size and variability for group III (BOP1: 3.946) than that group II (BOP1: 1.629).

Table 4: Statistical test of lipid profile among groups and visits (Inter and Intra comparisons) using One Way ANOVA, Independent and dependent sample t-test.

| | HDL | | Paired t-test | P value | LDL | | Paired t-test | P value |
|--|-----------------|-----------------|---------------|--------------------------|-----------------|-----------------|---------------|--------------------------|
| | pre | post | | | pre | post | | |
| | 70.475 | | | | 113.055 | | | |
| | 8.095 | | | | 6.833 | | | |
| | 22.000 | 24.0 | | | 135.0 | 132.4 | | |
| | 35.000 | 36.0 | 6.440 | 0.000 ES=0.831 | 162.0 | 162.0 | 6.142 | 0.000 ES=0.793 |
| | 29.127 | 30.167 | | | 142.64 | 140.930 | | |
| | 4.320 | 4.134 | | | 6.525 | 6.863 | | |
| | 20.500 | 35.2 | | | 133.2 | 111.6 | | |
| | 36.000 | 57.6 | 28.008 | 0.000 HS ES=3.616 | 167.0 | 146.2 | 21.976 | 0.000 HS ES=2.837 |
| | 29.547 | 43.94 | | | 145.72 | 126.96 | | |
| | 4.219 | 5.364 | | | 9.372 | 7.41 | | |
| | 2 | 58 | | | 2 | 58 | | |
| | 0.000 HS | 0.000 HS | | | 0.000 HS | 0.000 HS | | |
| | 0.917 | 2.88 | | | 0.551 | 1.956 | | |

| Groups | Cholesterol | | | Paired t-test | P value | Triglyceride | | Paired t-test | P value |
|-------------------|-----------------|----------|---------|---------------|--------------------------|-----------------|----------|---------------|--------------------------|
| | Mean | pre | post | | | Mean | pre | | |
| Control | Mean | 165.935 | | | | 166.520 | | | |
| | ±SD | 14.553 | | | | 11.486 | | | |
| Without melatonin | | | | 0.751 | 0.459 ES=0.097 | 245.0 | 240.0 | 4.946 | .000 ES=0.639 |
| | Mean | 280.923 | 280.44 | | | 377.0 | 375.0 | | |
| | ±SD | 36.623 | 36.761 | | | 36.775 | 37.042 | | |
| With melatonin | | | | 7.804 | 0.000 HS ES=1.007 | 254.0 | 243.8 | 15.140 | 0.000 HS ES=1.955 |
| | Mean | 293.843 | 287.777 | | | 399.0 | 381.0 | | |
| | ±SD | 39.961 | 40.115 | | | 314.82 | 297.617 | | |
| Df | 2 | 58 | | | | 2 | 58 | | |
| P value | 0.000 HS | 0.463 NS | | | | 0.000 HS | 0.579 NS | | |
| ES | 0.715 | | | | | 0.785 | | | |

The results in table 4 demonstrated that in baseline visit, all lipid profiles were found to be highest in group III followed by group II with the least value in the control group. HDL level showed an exception of that, in which the control group has the highest value other than other groups followed by group III and the least value found in group II. In the second visit, the cholesterol and HDL levels were found to be lower in group II than in group III. The opposite findings in Triglyceride and LDL levels were found to be higher in group II (302.973)-than in group III (297.617). All lipid profiles were found to be lowered between visits in each group with significant differences except for HDL were it showed to be higher with significant differences in each group with greater effect size and variability for group III. Table 5 clarified further analysis in the multiple comparisons of lipid profile in the baseline between groups. There was no significant difference in lipid profile between group III and II while when compared each one with the control group, the results were highly significant difference.

Table 5: Multiple comparisons of lipid profile in the baseline between groups by using Games-Howell test

| Dependent Variable | Groups | Groups | Mean Difference | P value | |
|--------------------|--------------|-------------------|-------------------|----------|----------------|
| cholesterol | Games-Howell | Control | Without melatonin | -114.988 | 0.000** |
| | | Without melatonin | With melatonin | -127.908 | 0.000** |
| Triglyceride | Games-Howell | Control | Without melatonin | -139.110 | 0.000** |
| | | Without melatonin | With melatonin | -148.300 | 0.000** |
| HDL | Games-Howell | Control | Without melatonin | -12.920 | 0.398 |
| | | Without melatonin | With melatonin | -9.190 | 0.616 |
| LDL | Games-Howell | Control | Without melatonin | 41.348 | 0.000** |
| | | Without melatonin | With melatonin | 40.928 | 0.000** |
| | | Control | Without melatonin | -420 | 0.923 |
| | | Without melatonin | With melatonin | -29.585 | 0.000** |
| | | Control | Without melatonin | -32.665 | 0.000** |
| | | Without melatonin | With melatonin | -3.080 | 0.338 |

Table 6: Correlation between lipid profiles in recall visit in each group

| Groups | | Trig | HDL | LDL | |
|-------------------|-------------|---------|-------------|--------------|--------------|
| Without melatonin | Cholesterol | r | .925 | -.274 | .668 |
| | | p value | .000 | .143 | .000 |
| | Trig | r | | -.150 | .702 |
| | | p value | | .430 | .000 |
| | HDL | r | | | -.253 |
| | | p value | | | .177 |
| With melatonin | Cholesterol | r | .972 | -.696 | .765 |
| | | p value | .000 | .000- | .000 |
| | Trig | r | | -.700 | .757 |
| | | p value | | -.000 | .000 |
| | HDL | r | | | -.478 |
| | | p value | | | -.008 |

Results of table 6 showed that correlations between lipid profiles in group II are the same as for baseline visits in the same group. There were significant correlations between cholesterol/triglyceride, cholesterol/LDL and between triglyceride and LDL while others had no correlations. The same results were found in group III, in which all results were significantly correlated with each other. Negative correlations were found between cholesterol/HDL, HDL/LDL, and Triglyceride/ HDL.

Discussion

Regarding periodontal parameters, baseline visit showed a significant difference between control and study groups due to the fact that control subjects had healthy periodontium and good plaque control, which may attribute to their performance of good oral hygiene measures, and also by the fact of selection criteria of subjects in all groups.

There was a highly significant difference of PLI between the study groups at recall visit when compared with a baseline visit (p value ≤0.05) with more variability and effect size in group III than group II. Instructions and motivation with the aid of scaling with root planing may results in a significant reduction in the means of PLI in both study groups. In addition, when melatonin was used, it showed greater variability and significance when compared with the groups that were treated with scaling and root planning only. These findings agreed with the study confirmed by Cutando et. al (15). Similar finding agreed with these results confirmed by Almughrabi et. al 2013 which showed that consumption of melatonin reduces the formation of bacterial biofilm, but this reduction was not significant compared with the control

group. Similarly, Syrinath et. al showed that melatonin had the activity against *Streptococcus mutans*, *Prevotella intermedia* and *Porphyromonas gingivalis*, which play a key role for biofilm formation and progression of periodontal diseases ⁽¹⁶⁾. BOP score 1 was decreased from baseline to the recall visit. SRP alone (without melatonin group) was effective in decreasing BOP score 1, which agreed with previous studies ^(17, 18). While melatonin group exhibited more increasing in BOP 0, indicating there was an increase in the percentage of gingival health.

Similar findings were reported by Cutando et.al that topical application of melatonin in diabetic patients will significantly reduce bleeding and probing in active periodontitis through the down-regulation in pro-inflammatory mediators, and decreasing the rate of bone loss ⁽¹⁹⁾. Another study by Montero et.al ⁽²⁰⁾ where agreed with the present study, they found that topically applied melatonin (1% orabase cream formula) for 20 days will significantly reduce clinical periodontal parameters involving BOP score 1. The free radicals scavenging action of melatonin decrease gingival inflammation by its antioxidant effect. In addition, its efficacy on reduction of lipid uptake of microorganisms, regulation of duplication of bacteria and its effect to bind with iron, may explain this effect.

Regarding lipid profiles; at baseline visit there was highly significant differences between the control and study groups, which may attributed to their chosen criteria as obese subjects compared to systemically healthy control counterpart.

According to F family test of statistical test (ANOVA. Repeated measures, between factors) of the G-power program, the complete sample size determined to be taken was 78 at 0.80 power on α error probability 0.05. So, 100 subjects decided to be included in this study to compensate for any anticipated dropout that described study sample that consisted of 80 males and females individuals with age ranged between 24-55 years, all subjects enrolled voluntarily to the study after signed informed consent sheet to participate in research.

Regarding the comparison between both study groups from baseline visit to recall visit, it demonstrated that there was a highly significant difference and variability about changes in lipid profiles in melatonin group than without melatonin group. The findings of the present study results were agreed with the previous study that showed administration of 5mg of melatonin for two months significantly decrease the LDL level in patient don't exposed for a hypolipidemic diet for 3 months ⁽¹¹⁾. Another study reported that supplementation of melatonin significantly increase HDL level in peri-and postmenopausal women ⁽²⁷⁾. The mechanism of promoting the effect of melatonin on lipid profile is attributed by stimulating Brown adipose tissue activity ⁽²⁸⁾. Another mechanisms of melatonin on obesity are the enhancement of cholesterol catabolism by bile acids and increase the receptors of LDL to inhibit cholesterol synthesis ^(29, 30).

Regarding the correlation of recall visit, demonstrated that group III showed a significant correlation between cholesterol/HDL, triglyceride/HDL and LDL/HDL, in which it agreed with the results of Sun H et. al ^{(32) (33)}—that reported the significant association of melatonin on reducing the level of cholesterol, triglyceride and LDL and increasing the level of HDL. However, our results have disagreed with others that showed no significant correlation of melatonin with HDL ^{(28) (29)}. This discrepancy may be due to the diversity of dose and duration of melatonin therapy and the study population. Therefore, more research is needed on larger study groups to investigate the precise effect of melatonin on anthropometric indices. According to the current research design, certain limitations have been addressed which need to be determined and recommended for future work.

These may include: using different doses and duration of intake of melatonin supplements, larger sample size for statistical values to avoid the outlier effect, depending on more objective and reliable method for melatonin detection close to periodontal tissue other than serum such as gingival crevicular fluid, and using other designs such as parallel blinding or crossover clinical trial with local delivery to overcome the bias concerns.

Conclusion

Daily supplementation with 5mg melatonin tab may significantly improve periodontal health and reduced chol, TG, LDL with increasing HDL. This may be designed to use as an adjunctive in the treatment of periodontal diseases.

Conflict of interest: None.

References

1. Carranza FA, Newman MG. Clinical periodontology 8th edition. W B Saunders Company Philadelphia. 1996.
2. Iacopino AM, Cutler CW. Pathophysiological relationships between periodontitis and systemic disease: recent concepts involving serum lipids. *J. Periodontol.* 2000;71(8):1375-84.
3. Shuldiner AR, Yang R, Gong D-W. Resistin, obesity, and insulin resistance—the emerging role of the adipocyte as an endocrine organ. *N Engl J Med.* 2001;345(18):1345-6.
4. Saito T, Shimazaki Y, Koga T, Tsuzuki M, Ohshima A. Relationship between upper body obesity and periodontitis. *J. Dent. Res.* 2001;80(7):1631-6.
5. Al-Zahrani MS, Bissada NF, Borawski EA. Obesity and periodontal disease in young, middle-aged, and older adults. *J. Periodontol.* 2003;74(5):610-5.
6. Nishida N, Tanaka M, Hayashi N, Nagata H, Takeshita T, Nakayama K, et al. Determination of smoking and obesity as periodontitis risks using the classification and regression tree method. *J. Periodontol.* 2005;76(6):923-8.
7. Kim EJ, Jin BH, Bae KH. Periodontitis and obesity: A study of the fourth Korean national health and nutrition examination survey. *J. Periodontol.* 2011;82(4):533-42.
8. Virto L, Haugen HJ, Fernández-Mateos P, Cano P, González J, Jiménez-Ortega V, et al. Melatonin expression in periodontitis and obesity: An experimental in-vivo investigation. *J. Periodont. Res.* 2018;53(5):825-31.
9. Amini AM, Muzs K, Spencer JP, Yaqoob P. Pelargonidin-3-O-glucoside and its metabolites have modest anti-inflammatory effects in human whole blood cultures. *Nutr Res.* 2017;46:88-95.
10. Hardeland R, Poeggeler B. Melatonin and synthetic melatonergic agonists: actions and metabolism in the central nervous system. *Cent. Nerv. Syst. Agents Med. Chem. (Formerly Current Medicinal Chemistry-Central Nervous System Agents).* 2012;12(3):189-216.
11. Koziróg M, Poliwczak AR, Duchnowicz P, Koter-Michalak M, Sikora J, Broncel M. Melatonin treatment improves blood pressure, lipid profile, and parameters of oxidative stress in patients with metabolic syndrome. *J. Pineal Res.* 2011;50(3):261-6.
12. Consultation WE. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet (London, England).* 2004;363(9403):157-63.
13. Silness J, Løe H. Periodontal disease in pregnancy II. Correlation between oral hygiene and periodontal condition. *Acta. Odontol. Scand.* 1964;22(1):121-35.
14. Newbrun E. Indices to measure gingival bleeding. *J. Periodontol.* 1996;67(6):555-61.
15. Lindhe J, Lang NP, Karring T. Clinical periodontology and implant dentistry: Blackwell munksgaard Copenhagen; 2003.
16. Rai B, Kharb S, Anand S. Salivary enzymes and thiocyanate: Salivary markers of periodontitis among smokers and non-smokers; a pilot study. *Adv Med Dent Sci.* 2007;1(1):1-4.

17. Cutando A, Montero J, Gómez-de Diego R, Ferrera M-J, Lopez-Valverde A. Effect of topical application of melatonin on serum levels of C-reactive protein (CRP), interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- α) in patients with type 1 or type 2 diabetes and periodontal disease. *J. Clin. Exp. Dent.* 2015;7(5):e628.
18. Srinath R, Acharya AB, Thakur SL. Salivary and gingival crevicular fluid melatonin in periodontal health and disease. *J. Periodontol.* 2010;81(2):277-83.
19. Santos VR, Lima JA, De Mendonça AC, Braz Maximo MB, Favari M, Duarte PM. Effectiveness of full-mouth and partial-mouth scaling and root planing in treating chronic periodontitis in subjects with type 2 diabetes. *J. Periodontol* 2009;80(8):1237-45.
20. Wennström JL, Tomasi C, Bertelle A, Dellasega E. Full-mouth ultrasonic debridement versus quadrant scaling and root planing as an initial approach in the treatment of chronic periodontitis. *J. Clin. Periodontol.* 2005;32(8):851-9.
21. Cutando A, López-Valverde A, de Diego RG, de Vicente J, Reiter R, Fernández MH, et al. Effect of topical application of melatonin to the gingiva on salivary osteoprotegerin, RANKL and melatonin levels in patients with diabetes and periodontal disease. *Odontology.* 2014;102(2):290-6.
22. Montero J, López-Valverde N, Ferrera M-J, López-Valverde A. Changes in crevicular cytokines after application of melatonin in patients with periodontal disease. *J. Clin. Exp. Dent.* 2017;9(9):e1081.
23. Rizk NM, Yousef M. Association of lipid profile and waist circumference as cardiovascular risk factors for overweight and obesity among school children in Qatar. *Diabetes, metab. Syndr. Obes.: targets and therapy.* 2012;5:425.
24. Falaschetti E, Hingorani AD, Jones A, Charakida M, Finer N, Whincup P, et al. Adiposity and cardiovascular risk factors in a large contemporary population of pre-pubertal children. *Eur. Heart J.* 2010;31(24):3063-72.
25. Hirschler V, Aranda C, de Luján Calcagno M, Maccalini G, Jadzinsky M. Can waist circumference identify children with the metabolic syndrome? *Arch. Pediatr. Adolesc. Med.* 2005;159(8):740-4.
26. Cowin I, Emmett P. Cholesterol and triglyceride concentrations, birthweight and central obesity in pre-school children. *Int. J. Obes.* 2000;24(3):330-9.
27. Tamura H, Nakamura Y, Narimatsu A, Yamagata Y, Takasaki A, Reiter RJ, et al. Melatonin treatment in peri- and postmenopausal women elevates serum high-density lipoprotein cholesterol levels without influencing total cholesterol levels. *J. Pineal Res.* 2008;45(1):101-5.
28. Tan DX, Manchester L, Fuentes-Broto L, Paredes S, Reiter R. Significance and application of melatonin in the regulation of brown adipose tissue metabolism: relation to human obesity. *Obes Rev.* 2011;12(3):167-88.
29. Mullerwieland D, Behnke B, Koopmann K, Krone W. Melatonin inhibits LDL receptor activity and cholesterol-synthesis in freshly isolated human mononuclear leukocytes. *Biochem Biophys Res Commun* 1994;203(1):416-21.
30. Chan T, Tang P. Effect of melatonin on the maintenance of cholesterol homeostasis in the rat. *Endocr Res.* 1995;21(3):681-96.
31. Sun H, Huang F-f, Qu S. Melatonin: a potential intervention for hepatic steatosis. *Lipids Health Dis.* 2015;14(1):1-6.
32. Agil A, Navarro-Alarcón M, Ruiz R, Abuhamad S, El-Mir MY, Vázquez GF. Beneficial effects of melatonin on obesity and lipid profile in young Zucker diabetic fatty rats. *J. Pineal Res.* 2011;50(2):207-12.
33. Virto L, Cano P, Jiménez-Ortega V, Fernández-Mateos P, González J, Haugen HJ, et al. Melatonin as adjunctive therapy in the treatment of periodontitis associated with obesity. *J. Clin. Periodontol.* 2018;45(11):1336-46.

34. Mostafavi S-A, Akhondzadeh S, Reza Mohammadi M, Keshtkar A-A, Hosseini S, Reza Eshraghian M, et al. Role of melatonin in body weight: A systematic review and meta-analysis. *Curr. Pharm. Des.* 2017;23(23):3445-52.
35. Mohammadi-Sartang M, Ghorbani M, Mazloom Z. Effects of melatonin supplementation on blood lipid concentrations: a systematic review and meta-analysis of randomized controlled trials. *Clin. Nutr.* 2018;37(6):1943-54.

العنوان: تأثير مكملات الميلاتونين على صحة اللثة وملاحح الدهون في مرضى التهاب دواعم السن والسمنة
الباحثون: حسام سامي إسماعيل , مها شكري محمود
المستخلص:

الخلفية العلمية: تزيد السمنة من قابلية المضيف للإصابة عن طريق تعديل جهاز المناعة والالتهابات بطريقة تؤدي إلى تدمير الأنسجة الالتهابية وتترك الفرد أكثر عرضة للإصابة بالتهاب دواعم السن. الميلاتونين هو منتج إفرازي صنوبر يشارك في العديد من الإجراءات ، مثل تنظيم الساعات البيولوجية الداخلية واستقلاب الطاقة ، ويعمل كمضاد للأكسدة ومضاد للالتهابات. أهداف الدراسة: هدفت الدراسة الحالية إلى التحقيق في آثار إعطاء الميلاتونين الجهازى على الحالة الصحية اللثوية وخصائص الدهون في مرضى التهاب دواعم السن المصابين بالسمنة. المواد وطرق العمل: تم توزيع الأشخاص والمرضى المشمولين في الدراسة على المجموعات التالية: المجموعة الأولى ، 20 شخصًا بوزن طبيعي ولثة صحية (المجموعة الضابطة) ولم يخضعوا لأي علاج. المجموعة الثانية: 30 مريضاً يعانون من السمنة المفرطة مع التهاب دواعم السن وتعرضوا لـ SRP وتستكمل بأقراص الميلاتونين 5 ملغ لمدة شهر. تعرضت مجموعات الدراسة لقياس مؤشر اللويحة الجرثومية (PLI) ، والنزيف عند الفحص المفرطة مع التهاب دواعم السن وتعرضوا لـ SRP وتستكمل بأقراص الميلاتونين 5 ملغ لمدة شهر. تعرضت مجموعات الدراسة لقياس مؤشر اللويحة الجرثومية (PLI) ، والنزيف عند الفحص (BOP) ، والكوليسترول (Col) ، والدهون الثلاثية (TG) ، والبروتين الدهني عالي الكثافة (HDL) والبروتين الدهني منخفض الكثافة (LDL) عند خط الأساس وبعد 4 أسابيع . النتائج: فيما يتعلق بالمعايير السريرية ، أظهرت الزيارة الثانية انخفاضًا في جميع المتغيرات في مجموعتي الدراسة باستثناء سكر صفر لمقياس BOP حيث زادت بشكل معنوي. فيما يتعلق بخصائص الدهون ، أظهرت الزيارة الثانية انخفاضًا في جميع المظاهر باستثناء HDL حيث زادت في كلتا مجموعتي الدراسة مع اختلاف معنوي. أظهرت جميع الارتباطات بين ملف الدهون في مجموعتي الدراسة ارتباطًا إيجابيًا قويًا معنويًا بين الـ COL و TG ، والـ LDL و LDL ، و LDL و TG في المجموعة الثالثة. في المجموعة الثانية ، أظهرت جميع النتائج ارتباطًا إيجابيًا قويًا مع وجود ارتباط سلبي قوي فقط بين الـ HDL و HDL و HDL و LDL. الاستنتاج: مكملات يومية مع 5 ملغ من الميلاتونين حسنت بشكل ملحوظ صحة اللثة وتقليل TG ، LDL مع زيادة HDL