INTRODUCTION

Periodontitis, a chronic infectious inflammatory disease of the tissue surrounding and supporting the teeth, is accompanied by a deficiency of antioxidants to balance increased oxidative stress. Melatonin, an antioxidant, might be involved in periodontal tissue destruction. In the oral cavity it acts as an anti-oxidative, free-radical scavenging, anti-inflammatory and bone-preserving agent. Decreased plasma and salivary melatonin levels in periodontitis patients were described by several studies [1-4]. In this study the effect of conservative periodontal therapy on salivary melatonin levels was investigated.

MATERIALS AND METHODS

Stimulated salivary samples (Saliva Collection System, Greiner Bio One, Austria) and serum samples of 43 participants (19 patients with generalized periodontitis and 24 periodontally healthy controls - matched for age, sex, smoking status and time point of saliva sampling) were collected. Salivary melatonin levels were determined using ELISA technique (IBL, Germany) and serum levels of C-reactive protein (CRP) were measured on a Chemistry Immuno-System Olympus AU640. Probing pocket depth (PD), clinical attachment level (CAL), and bleeding on probing (BoP) were assessed. After non-surgical periodontal therapy recording of periodontal status and saliva sampling were repeated. For statistical analysis a linear regression model and Pearson’s correlations coefficient was applied. The significance level was set to 0.05.

RESULTS

The clinical parameters in healthy individuals and periodontitis patients before and after periodontal therapy are given in Table 1. Serum CRP levels of periodontitis patients were marginally but not significantly increased (figure 1; p=0.055). The effectiveness of periodontal therapy was indicated by significantly decreased periodontal parameters (Table 1) and serum CRP levels were slightly but not significantly decreased after periodontal therapy (figure 1; p=0.2).

Further, a significantly negative correlation was present between the change of salivary melatonin and BoP after treatment (figure 3; rho=−0.53, p=0.02). Other tested parameters (PD, CAL, and serum CRP) presented a negative correlation as well, yet not statistically significant.

CONCLUSION

To the best of our knowledge, this is the first study to re-evaluate salivary melatonin levels after non-surgical periodontal therapy. Before therapy, we found similar to previous reports [2-4] somewhat decreased salivary melatonin levels in periodontitis patients compared to healthy controls (figure 2). The significant decrease of clinical parameters after non-surgical periodontal therapy was accompanied by a significant increase of salivary melatonin levels (figure 2). Further, we found a significantly negative correlation between the change of salivary melatonin and BoP after treatment (figure 3). Altogether, reducing the inflammation seems to recover salivary melatonin levels and to allow a re-establishment of the homeostasis of the oxidant/antioxidant system.

REFERENCES