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ORIGINAL ARTICLE

Tissue Plasminogen Activator (Cardiovascular Risk Marker): Interventional Studies on Periodontitis

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ABSTRACT

The association between periodontal disease and cardiovascular disease has received considerable attention, although it is not clear whether these is a casual component. We used a treatment intervention model to study the relationship between periodontitis and tissue plasminogen activator (t-PA) cardiovascular indicator of risk. We studied ten adults with periodontitis required scaling and root planning. Blood samples were obtained (1) at immediately prior to treatment; (2) one week after scaling and root planning and (3) two weeks after scaling and root planning. The ten patient (control) without periodontitis and systemic disease selected for control. After scaling and root planning, there was a significant decrease in t-PA, although higher as compared to control. This study shows that elimination of periodontitis by scaling and root planning reduces the t-PA cardiovascular risk marker. Analysis of the data supports the hypothesis that treatment of periodontal disease may lower cardiovascular risk.

Keywords: Periodontal disease, tissue plasminogen activator, cardiovascular disease

Introduction

Epidemiological associations between periodontitis and cardiovascular disease have been reported.[1] Artherosclerosis and periodontitis have complex aetiologies, genetic and gender predispositions, and may share pathogenic mechanism as well as common risk factors. This could be directly due to periodontal pathogens or their products on endothelial cell via transient bactermia or indirectly due to products of the inflammatory response.[2] Also, increased levels of serum tissue plasminogen activator (t-PA) have been related to increased cardiovascular risk.[3] The effect of treatment on tPA has not been reported in the literature. The purpose of this study was to investigate whether elimination of periodontitis by scaling and root planning reduces level of serum tPA as compared with control.

Materials and Methods

Systemic normal forty patients (non-smoker, non-alcoholic) (M:F, 20:20 in age group of 32-40 years) having at least a minimum of seven sites periodontal involved, 9 mm (9.0 $\pm$ 1.2 mm) of clinical loss of attachment, the 20 number of teeth (21 $\pm$ 1.2) in the dentition were selected for the study. In all these cases, the peripheral blood were drawn before starting any treatment, one week later after scaling & root planning and two week after scaling and root planning for tPA estimation. Non-smoker, non-alcoholic healthy control without any periodontal (without clinical loss of attachment, 28 number of teeth) disease (n=5, age 37-40) were selected and peripheral blood were drawn for t-PA. Plasma was obtained after centrifugation at 1500 g for 10 min and stored at -4^0 C until analysis. t-PA concentrations were determined by ELISA (Imulgse tPA, Bioipool International). The data was analyzed by SPSS 11.0.

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Table 1: Effect of scaling and root planning on t-PA [pretreatment (T_1)], one week later scaling & root planning (T_2) two week later scaling & root planning (T_3) and normal healthy controls (T_0).

Groups	t-PA (mg/dl)
T_1 (pretreatment)	13.7 (7.2 – 21.7)
T_2 (one week after scaling and root planning)	12.5 (6.8 – 20.3)
T_3 (two week after scaling and root planning)	11.7 (5.6 – 17.5)
T_4 (normal healthy)	9.9 (4.2 – 11.5)

The tPA values were (13.7 mg/dl) followed treatment one week after scaling and root planning. It fall to 12.5 mg/dl and after two week after scaling and root planning levels were 11.7 mg/dl. These values were still higher as compared to normal healthy. tPA levels were significantly raised in periodontal patients as compared to controls ($p<0.001$).

Results and discussions

Results

Discussion and Conclusion

Increased plasma t-PA has been reported to be marker of endothelial dysfunction and has been associated with increased risk of CHD.[4,5] To the best of our knowledge this is first study reporting effect of scaling and root planning in periodontitis to decrease t-PA levels. In the present study, high plasma tPA levels were observed in periodontitis patients as compared to controls and the levels fall significantly following treatment though the levels were significantly higher than controls.

Since the number of patients in this study were relatively small, these results need to be interpreted with caution. Nevertheless, these results are compatible with the view that only susceptible individual react to periodontitis with increase tPA and that these individuals were not necessarily the ones with most severe disease. Treating periodontitis by scaling and root planning lower tPA levels in these individuals, possibly also reducing their CHD risk. Our findings support the view that periodontitis patients are at increased risk of CVS disease.

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