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Comparative Evaluation Of Serum Troponin Levels In Patients With Chronic Generelised Periodontitis And Chronic Generalised Gingivitis Before And After Non-Surgical Periodontal Therapy- A Randomized Controlled Clinical Trail.

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Abstract

Introduction : Chronic periodontitis is a chronic inflammation associated with elevation of several inflammatory and cardiac markers like troponin I studies implicated that chronic periodontitis is one of the etiologies in coronary heart disease.

Aims & objectives : This study evaluates and compares the level of troponin in chronic periodontitis patients and in gingivitis patients. Thereby,explaining the quantitative relationship of periodontal status and coronary heart disease.

Materials and methods: Group A: Ten patients diagnosed with chronic periodontitis, received scaling and root planning with mean pocket depth of 6mm, peripheral venous blood from each group was taken for measurement, blood was drawn at baseline and 3 months after SRP

Group B: ten patients diagnosed with chronic generalized gingivitis were recruited from the department of periodontics st.joseph dental college and hospital .blood was collected at base line and 3 months after scaling.

Summary of results: study was statistically significant (p<0.5).Group A showed mean decrease in follow up, in Group B being the control group showed no much difference, as p value is <0.001 it is significant.

Keywords : Troponin, Troponin I ,Chronic Gingivitis, Chronic Periodontitis, Cardiovascular disease, Atherosclerosis

Introduction

Gingivitis may lead to periodontitis without proper treatment ,the inflammatory periodontal tissues will progress with age and may lead to pathological destruction of the tooth supporting tissues, tooth loosening and potential tooth loss.[1] Periodontal disease is defined as chronic inflammation of gum tissue, including the ligaments and bony structures that hold the tooth in place. [2] Chronic periodontitis (CP) is a chronic inflammation associated with elevations of several inflammatory and cardiac markers.[1]Cardiovascular disease affects millions of individuals.[3] Cardiac markers are biomarkers which can evaluate the heart function by identifying the blood chemicals that are associated with damage of heart muscle cells.[1] Bacteria are an important causative agent in periodontitis. Bacteremia occurs when bacteria enter the bloodstream from the inflammatory activity of periodontitis. The bacteria infects epithelial integrity in

periodontal pocket ,and vascular endothelium of the arterial wall. Chronic infection has been reported to be a contribution to atherosclerosis.[1] Chronic periodontitis (CP) was associated with increased serum levels troponin I.[1] Cardiac troponins are highly specific markers for detecting myocardial injury.[4] The troponin complex consists of three subunits—troponin C, troponin I, and troponin T. The cardiac isoforms troponin T and I are only expressed in cardiac muscle. Hence, cardiac troponin are more specific for myocardial injury, because of their high sensitivity, they are elevated when other concentrations are not.[5] Periodontitis and atherosclerosis have many potential pathogenic mechanisms in common. As mentioned previously, monocyte infiltration into the subintima is a crucial pathogenic process in plaque development, and clearly the monocyte is crucial as both an infiltrating cell and a cell that can initiate the process by releasing cytokines, upregulating adhesion molecules and binding to them. The monocyte can also produce IL-8, a chemotactic factor that will aid in the recruitment of more leukocytes to this area.[6] The aim of this study was to evaluate the association of periodontal disease with CHD by examining the cardiac biomarker (troponin I) in subjects with severe chronic periodontitis and chronic generalized gingivitis before and after Scaling and Root planing [SRP].

Materials and Methods

Study population included a total of 20 patients who attended the department of periodontics,st joseph dental college with chronic periodontitis of age group 35 to 55 years . Patients were categorized into 2 groups based on the history of inflammation :

Group A included 10 patients with periodontitis.

Group B included 10 patients with gingivitis.

The patients were otherwise healthy, with no history of major illness and consumption of antioxidants, antibiotics, anti-inflammatory or any other drugs and had not received any periodontal therapy for at least 3 months prior to the study. Ethical committee clearance from research committee and Informed consent from the patients were taken for all the patients before commencement of the study.

All the individuals selected for this study underwent clinical examination of the oral cavity and periodontal indices such as plaque index (PI) ,gingival index(GI), probing depth (PD) and clinical attachment level(CAL).5ml of blood was collected from the anticubital fossa by venipuncture using 20 gauge needle. The collected serum was eavaluated for troponin I.

All the patients including group A and group B underwent clinical examination,serum evaluation, periodontal therapy(scaling /scaling and root planning) and recalled after 3 months for clinical and serum evaluation.

Statistical Analysis

All data was expressed as mean, standard division (SD) at baseline, 3 months. Data were entered in MS-Excel and analysed by using software SPSS V22. Descriptive statistics were represented with mean and SD. Paired t-test and Independent t-test were applied to find significance in follow-up and between the groups. $P<0.05$ was considered as statistically significant.

Results

The mean values of Plaque Index, Gingival Index, Clinical Attachment Level, Probing Pocket Depth showed significance in Group A. The mean values of troponin I showed significance in groups A with P values [<0.005] ,and group B being control group showed no much difference . Group A showed decrease in troponin I post operatively than group B which is non significant(>0.005), group A showed more and significant decrease in troponin I levels post operatively than Group B which is control group as shown in tables1 and 2.

Comparison in follow-up (within the group)

TABLE 1: Comparison of mean, standard deviation and P values of PI, GI, PPD, CAL,troponin I in group A(periodontitis).

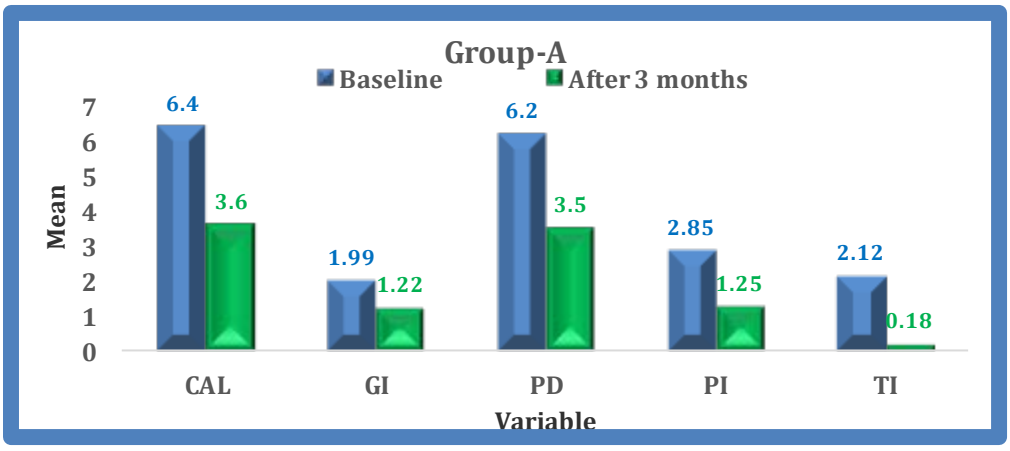
Table 2: Comparison of mean, standard deviation and P values of PI, GI, PPD,CAL, troponin I in group B (gingivitis) control group.

Group	Variable	Base					After 3 months					P-value
		N	Minimum	Maximum	Mean	SD	N	Minimum	Maximum	Mean	SD	
B	CAL	10	2.0	3.0	2.20	0.42	10	2.00	3.00	2.20	0.42	1
	GI	10	1.0	1.8	1.35	0.24	10	1.00	1.40	1.22	0.13	0.07
	PD	10	2.0	3.0	2.20	0.42	10	2.00	3.00	2.20	0.42	1
	PI	10	1.0	1.8	1.36	0.24	10	.90	1.30	1.06	0.15	**0.005**
	TI	10	0.0	.1	0.01	0.03	10	0.00	.20	0.06	0.08	0.102

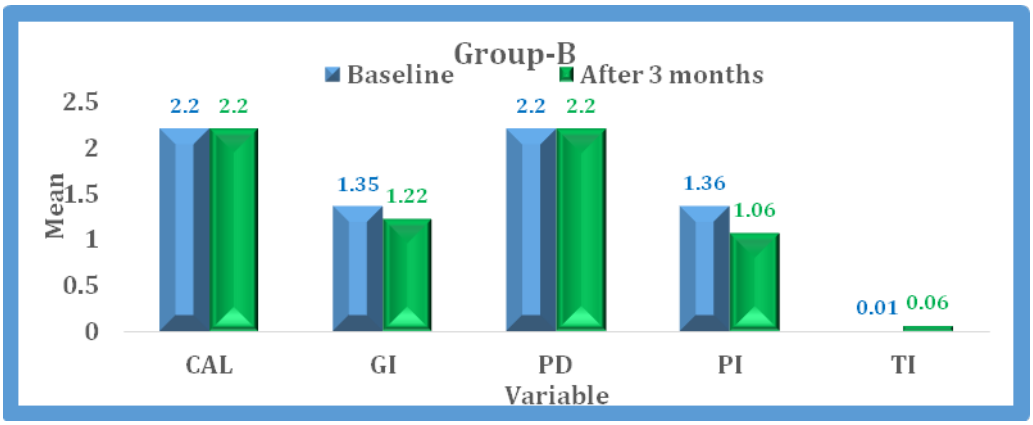
Table 3: Intergroup comparison of mean, standard deviation and P values of PI, GI, PPD, CAL,troponin I in group A (periodontitis) and group B (gingivitis)

Sub Group	Group	Mean difference from Base to 3 months	SD	P-value
CAL	A	2.80	0.63	<0.001
	B	0.00	0.00	
GI	A	0.77	0.31	<0.001
	B	0.13	0.19	
PD	A	2.70	0.67	<0.001
	B	0.00	0.00	
PI	A	1.60	0.23	<0.001
	B	0.30	0.16	
TI	A	1.94	0.45	<0.001
	B	0.05	0.08	

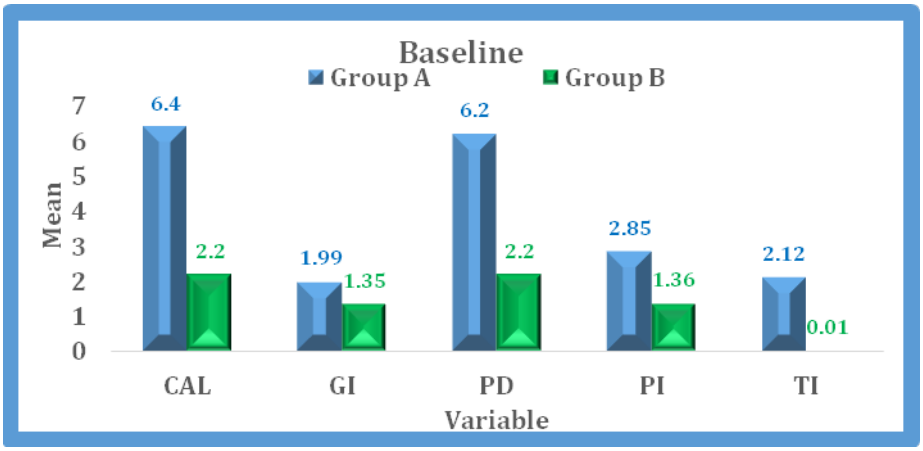
Graph 1 : comparison of mean and standard deviation and P values of PI, GI, PPD, CAL,troponin I in group A (periodontitis)at baseline [BL] & 3months

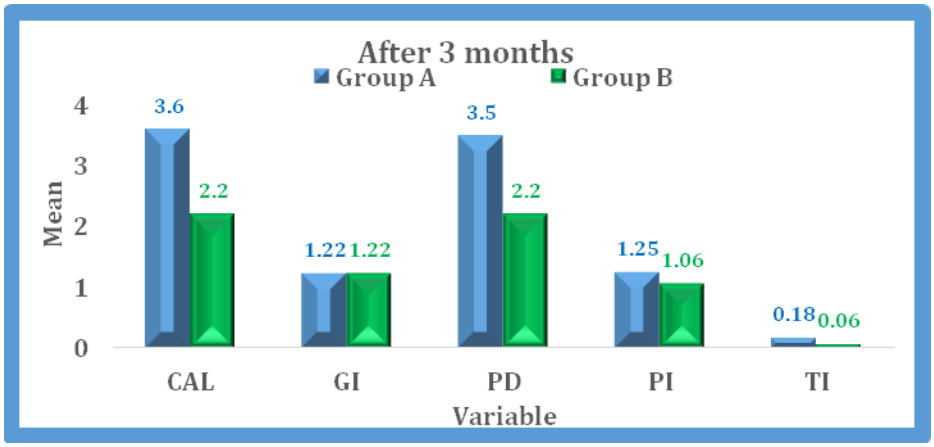


Graph 2 : comparison of mean and standard deviation of PI, GI, PPD, CAL, troponin I in group B (gingivitis) at baseline [BL] & 3months [3M]



Graph 3: Intergroup comparison of mean, standard deviation and P values of PI, GI, PPD, CAL,troponin I in group A (periodontitis) and group B (gingivitis)





Discussion

In the present study there was a significant reduction in GI & PI values in both the groups, 3 months after SRP, which could be attributed to constant reinforcement of oral hygiene and Hawthorne effect [7]. There was significant reduction in the PPD and gain in CAL in Group A compared to Group B .The reduced improvement in PPD and CAL following therapy in chronic periodontitis patients compared with chronic gingivitis patients which was in accordance to the study done by Wings TY Loo et al [1].

At the baseline, the levels of troponin I are increased in Group A with inflated value which could be due to the fact that periodontal infection has been suggested as a contributing factor for functional disorders in various oragans such as heart ,liver and kidney , with Group B showing no much difference being the control group. Periodontitis could also stimulate cardiomyocytes damage that leads to higher levels of serum troponin I which was in accordance with studies done by Wings TY Loo et al[1]

A number of pathophysiological pathways have been assumed as possible links between periodontitis and CAD (Coronary Artery Disease). These pathways either indirect or direct mechanisms Wożakowska B. et al, 2013.[7]

A- Indirect Mechanism - Systemic Inflammation

The process of atherosclerosis initiates in early life years, by fat deposition on the vascular lining and such process develops through several decades. The continuing, inert atheromatous plaque may swing to a serious form that is susceptible to rupture. Plaques have unstable soft core atheroma and their break will uncover extremely atherogenic matters to plasma, with stimulation of thrombus formation and resultant ACS (Acute coronary syndrome) according to Tonetti M. 2009 [8]. The association of CAD with mediators of inflammation in circulation is well-known, meanwhile links among concentrations of inflammatory biomarkers and increment in incidents like AMI (Acute Myocardial Infarction) is well demonstrated Kaptoge S. et al, 2010[9].

B- Indirect Mechanism - Molecular Mimicry

Antigenic mimicry seems to be an increasing potential pathway correlating periodontitis and AS. Molecular mimicry is considered one sequence resemblances of both external and self-peptides yield cross stimulation of (T or B) lymphocytes to initiate autoimmunity Kohm A. et al, 2003[10]. Cross-reactive antibodies toward dental bacterial lipopolysaccharides and heat shock proteins (HSP) is well-known and upraised as a possible reason for the presumed correlation between periodontitis and CAD Lockhart P. et al, 2012 [11] . This hypothesis assumes that bacterial products or inflammatory mediators may stimulate the endothelium to harvest host defensive factors against the HSP. Such immune reaction directed against exact HSP antigens of plaques may worsen the formed

inflammation. Hence, stimulating plaque evolution and solidity (Lockhart P. et al, 2009).

C- Direct Mechanisms: Periodontal Bacteria and Vascular Infection

Adults' mouths have over and above a billion bacteria. Though the colonies differ in various mouth areas; pocket is the region of highest possible significance to atherosclerosis. Oral bacteremia is a usual incident that may ensue with mastication or teeth cleaning. Actually, it happens daily in people often with some level of gingivitis and periodontitis. According to this hypothesis the bacteria or their products directly enter the endothelial wall from the blood and disturb both the plaque initiation and/or its growth. This hypothesis is supported by many researches that have revealed existence of viable oral bacteria or their genetic parts in plaque samples from various vascular beds (Lockhart P. et al, 2012)[11].

The levels of Tn in the blood of healthy individuals is around 0.1-0.2 ng/L due to the constant microscopic damage of myocytes through normal life. These markers have late clearance[12].

The bulk of Tn is physically bound in the contractile apparatus of the myofibril, however about 7% of TnT and 3-5% of TnI is free in the cytosol. Once there is a damage in the myocyte, there will be a biphasic upsurge in serum Tn relates to the early release of free cytoplasmic Tn, followed by the steady diffusion of bound Tn (Mahajan VS. & Jarolim P. 2011).[11]

Patients with periodontitis were found to have a 25% increased risk of CHD compared to patients with minimal or no periodontal disease according to the study done by DeStefano et al. monitored subjects for 13 to 16 years after a baseline dental examination on 9,760 subjects.[14]

There was a significant reduction in serum troponin i values in group A after SRP, which can be explained by the above mentioned mechanisms. They are also measured as markers associated with cell injury and cell death and change in this enzymatic activity reflecting the metabolic changes in the gingiva and periodontium, in the inflammation. So the improvement in periodontal condition after periodontal therapy resulted in a significant reduction in troponin i levels which was in accordance with the study done by Wings TY Loo et al[1]. Periodontitis seems to influence the occurrence and severity of coronary heart disease.[13]

The main limitation of the study is small sample size.

Conclusion

There was a relation seen between the cardiac biomarker troponin I and periodontitis with high values of troponin I in periodontitis and their decreased values after SRP. The low values of troponin I in gingivitis and their constant maintenance of these values after SRP showed a inverse relationship. Within the limitations of the study, the present study shows that subjects with chronic periodontitis exhibit significantly higher levels of cardiac biomarkers than the controls. These findings suggest that CP increases the systemic levels of inflammation and potential risks for CHD. Hence, periodontal disease may represent a modifiable risk factor for CHD .However studies with larger sample size are required.

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