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# Evaluation of Heat Shock Protein-70 Level in Patients with Periodontitis & Periodontitis With Diabetes Mellitus Type-II Before and After Srp: A Clinico- Biochemical Study

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***Abstract : Introduction :*** *- Periodontitis is an inflammatory disease of periodontium which is caused by periodontopathic bacteria. Moreover, various cytokines such as Interleukin-1β (IL-1β), Tumor necrosis factor-α (TNF-α), and IL-6 are expressed in the inflamed periodontium. Heat shock proteins (HSPs) protect cells from abnormal conditions including inflammation, microbial infection and diseases.* ***Aim:*** *To evaluate the levels of HSP 70 in healthy, periodontitis and periodontitis with diabetic mellitus type 2 before and after SRP.* ***Methodology:*** *A total of 24 patients between the age range of 30-60 years were selected for the study and divided equally into 3 groups with each group consisting of 8 patients. HSP70 levels were evaluated at baseline and after 6 weeks in GCF and serum.* ***Result:*** *There was decrease in the levels of HSP70 in GCF and serum of periodontitis with diabetic mellitus type 2 patients as compared to healthy patients after SRP and the decrease of HSP70 was seen to be more in GCF as compared to that in serum. Clinical parameters (Plaque index, Gingival bleeding index and probing pocket depth) also reduced significantly after SRP.* ***Discussion:*** *The levels of HSP70 in all the three groups were reduced significantly after doing scaling and root planing.* ***Conclusion:*** *HSP 70 can be used as prognostic measure of periodontitis and diabetic patients. It produces more accurate results.*

**Keywords**– *Heat shock protein-70, Periodontitis, Diabetes mellitus Type II, Indices, Biochemical study, SRP, GCF*

## I. INTRODUCTION

Periodontitis is a condition marked by inflammation that is both microbially induced and host-mediated, ultimately resulting in the loss of periodontal attachment[1]. Its pathophysiology involves well-characterized molecular pathways that lead to the activation of host-derived proteinases. These enzymes contribute to the degradation of marginal periodontal ligament fibers, facilitate the apical migration of the junctional epithelium, and permit the downward extension of the bacterial biofilm along the root surface[2]. The formation of the bacterial biofilm initiates gingival inflammation, leading to gingivitis[3]. If left untreated, gingivitis can progress to periodontitis, causing irreversible loss of periodontal support[3].The onset and progression of periodontitis are driven by dysbiotic shifts within the microbial community, which occur in response to nutrients derived from

inflammatory exudates and the breakdown of periodontal tissues[4] . These conditions favor the enrichment of specific microbial species and stimulate host antibacterial responses aimed at confining the microbial challenge to the gingival sulcus[5] .

Many studies have reported significant positive associations between diabetes and periodontitis (Firatli 1997, Hugoson *et al*. 1989, Papapanou 1996, Taylor *et al*. 1996).
Furthermore, diabetes has been identified as one of the major risk factors for periodontitis (Grossi *et al*. 1995, Grossi *et al*. 1994)[6].
Mechanical loading of the periodontal ligament (PDL) leads to ischemic conditions of the local microenvironment followed by a cascade of signaling events which eventually result in enhanced periodontal remodeling allowing for orthodontic tooth movement.[7]

Within this complex process, circulatory disturbances and cell stress evoke necrosis of a certain proportion of cells with subsequent initiation of a host immune response. This response is characterized by a chemoattraction of immune competent cells and their differentiation along the monocyte/macrophage lineage to clear the cellular debris and facilitate a structural reorganization of the periodontium in the first phase and tooth movement in a later stage (Jäger et al., 1993; Kim et al., 2010; Wolf et al., 2016).[8]

Heat shock proteins (HSPs) are conserved proteins activated by cellular stress, such as heat, inflammation, or environmental challenges. They act as molecular chaperones, aiding in protein folding, assembly and transport.
Periodontitis induces cellular stress, triggering HSP synthesis. HSPs play a protective role in periodontal health by assisting in the repair and maintenance of damaged proteins within the cells. HSPs are involved in the function of immune cells. These proteins are divided into five major classes, one of which is HSP70.[9]
Heat shock proteins (HSP) are released into the cytoplasm upon thermal stimulation, ischemic or hypoxic conditions and support protein folding and stabilization, facilitate protein transportation across cell membranes, and unfold denatured proteins and, thereby, contribute to cell survival under stress conditions (Wong et al., 1997; Wong et al., 1997).[10]

## II. MATERIALS AND METHOD

**Aim of the study**: To evaluate the HSP-70 in Serum and GCF of patients with Periodontitis and Periodontitis with Diabetes Mellitus Type - II

**Study design**: study includes a total of 24 patients (8 in each group), between age range of 30-60 years

Group A: subjects with healthy periodontium
Group B: subjects with Periodontitis
Group C: subjects with Periodontitis and Diabetes Mellitus Type – II

**Methodology:**

- Clinical parameters recorded:
  1. Plaque index (Silness J. &Loe H. 1964) using explorer #23
  2. Gingival bleeding index (Ainamo & Bay 1975) using williams Graduated periodontal probe
  3. Probing pocket depth (PPD) using UNC-15 probe

At first visit, clinical parameters were recorded and the next day GCF and serum sample was collected and SRP was done.

Six weeks after SRP, clinical parameters were recorded, GCF and serum samples were collected for the estimation of HSP70 using ELISA.

## III. RESULT

In subjects with healthy periodontium, there was 36.2% reduction of HSP -70 in serum and 36% reduction in GCF from the baseline. In Periodontitis subjects,13% and 35.2% reduction of HSP 70 in serum and GCF levels were seen respectively from base line after six weeks of SRP. In Periodontitis with Diabetes Mellitus type 2 subjects, 18.8% and 47.4% reduction of HSP 70 in serum and GCF levels respectively from base line and after 6 weeks of SRP.

In clinical parameters, Plaque index was reduced by 67% in subjects with healthy periodontium, 33% in Periodontitis subjects and 37% in Periodontitis with Diabetes Mellitus type 2 subjects. Gingival bleeding index reduced by 31% in Periodontitis subjects and 33% in Periodontitis with Diabetes Mellitus type 2 subjects. Probing pocket depth reduced by 43% in Periodontitis subjects and 46% in Periodontitis with Diabetes Mellitus type 2 subjects.

## IV. TABLES

| **SUBJECT** | **BASE LINE** | | **6 Weeks AFTER SRP** | |
|---|---|---|---|---|
| | HSP 70 IN SERUM | HSP 70 IN GCF | HSP 70 IN SERUM | HSP 70 IN GCF |
| SUBJECTS WITH HEALTHY PERIODONTIUM | 1218.3 | 358.9 | 867.2 | 122.3 |
| | 333.4 | 318 | 151.4 | 253.6 |
| | 77.8 | 505.5 | 38 | 344.9 |
| | 52.3 | 100.5 | 25.5 | 85.1 |
| | 221.8 | 113.3 | 132.2 | 89.9 |
| | 380.7 | 279.2 | 242.8 | 179.1 |
| | 328.7 | 178.7 | 217.3 | 94 |
| | 367.3 | 260.6 | 181.2 | 237.8 |

| **SUBJECT** | **BASE LINE** | | **6 Weeks AFTER SRP** | |
|---|---|---|---|---|
| | HSP 70 IN SERUM | HSP 70 IN GCF | HSP 70 IN SERUM | HSP 70 IN GCF |
| PERIODONTITS PATIENTS | 168.2 | 131.2 | 76.5 | 344.1 |
| | 294 | 962.2 | 257.2 | 234.6 |
| | 80.9 | 131.1 | 275.4 | 181.4 |
| | 133.7 | 247.2 | 99.2 | 163.2 |
| | 234.6 | 217.6 | 87.9 | 173.4 |
| | 182.28 | 211.1 | 99.5 | 137 |
| | 176.73 | 205.55 | 93.95 | 123.45 |
| | 187.83 | 216.65 | 102.57 | 167.89 |

| SUBJECT | BASE LINE | | 6 Weeks AFTER SRP | |
|---|---|---|---|---|
| | HSP 70 IN SERUM | HSP 70 IN GCF | HSP 70 IN SERUM | HSP 70 IN GCF |
| PERIODONTITS PATIENTS WITH DIABETES MELLITUS TYPE 2 | 77.3 | 304.3 | 137.2 | 354.8 |
| | 223.9 | 1085.7 | 358.8 | 651.8 |
| | 308.1 | 574.3 | 43.8 | 56.8 |
| | 412.8 | 1723.1 | 273.1 | 870.8 |
| | 152 | 422.3 | 141.6 | 233.2 |
| | 110.9 | 772.4 | 192.2 | 332.7 |
| | 137.6 | 672.2 | 201.4 | 423.9 |
| | 153.2 | 810.9 | 214.5 | 395.6 |

## V. CONCLUSION

The present study evaluated changes in Heat Shock Protein 70 (HSP70) level in both serum and gingival crevicular fluid (GCF) following non-surgical periodontal therapy (SRP) across different patient groups. In individuals with a healthy periodontium, SRP resulted in a minimal and statistically insignificant decrease in HSP70 levels in both serum and GCF. Conversely, patients diagnosed with periodontitis exhibited a significant reduction in HSP70 levels post-SRP, indicating a correlation between periodontal inflammation and HSP70 expression.

Notably, the most substantial decrease in HSP70 was observed in patients with both periodontitis and diabetes mellitus type 2. In this group, SRP led to a marked reduction in HSP70 levels in both serum and GCF, with the decrease being more pronounced in GCF. This suggests a greater local inflammatory response and therapeutic impact within the periodontal environment, especially in the presence of systemic disease[5].

Clinical parameters, including Plaque Index (PI), Gingival Bleeding Index (GBI), and Probing Pocket Depth (PPD), showed measurable improvement after SRP in all the three groups. The improvements were more significant in patients with periodontitis and diabetes mellitus type 2. Among these parameters, the most notable reduction was observed in PPD, reflecting effective resolution of periodontal inflammation[6].

The data indicate that SRP plays a key role in reducing both clinical signs of periodontal disease and associated inflammatory biomarkers such as HSP70. Given its role in cellular stress response and protein stabilization, HSP70 may serve as a valuable prognostic indicator for periodontal disease progression and treatment response, particularly in patients with systemic conditions like diabetes.

While these findings support the potential utility of HSP70 as a prognostic biomarker in periodontitis and diabetes, further longitudinal studies are required to explore its diagnostic value and therapeutic targeting in periodontal care.

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