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CLINICAL ASSESSMENT OF PERIODONTAL STATUS IN PATIENTS ON LONG TERM CORTICOSTEROID THERAPY

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ABSTRACT: Corticosteroids (Cs) are used widely for their anti-inflammatory and immunosuppressive properties. However their long-term administration may lead to impaired periodontal health. The aim of the present study was to clinically assess the periodontal status in patients on long-term corticosteroid therapy. Periodontal health of 100 patients under long-term corticosteroid therapy for a minimum of 6 months duration was compared with sex- and age-matched 100 healthy controls. The periodontal examination included measuring oral hygiene index-simplified (OHI-S), gingival index (GI), sulcus bleeding index (SBI), probing pocket depth (PPD) and clinical attachment loss (CAL). The results showed that mean values of OHI-S, GI and SBI did not differ significantly ($p>0.05$) between cases and controls. Mean PPD and CAL was significantly higher in cases when compared to the controls ($p = 0.0003$). Within the limitations of the study, it can be concluded that there is a positive correlation between periodontal status and long term steroid therapy.

KEYWORDS: Corticosteroids, collagen synthesis, bone mineral density, periodontal status

INTRODUCTION

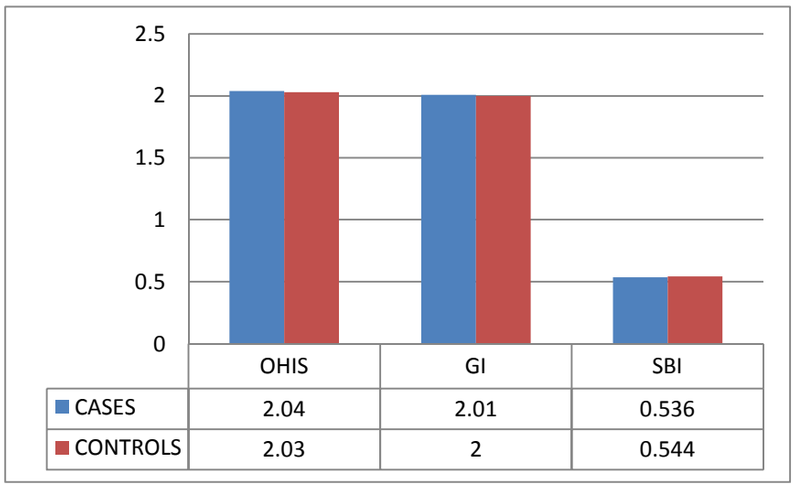
Corticosteroids are hormones either prepared endogenously by adrenal gland or synthetically prepared for therapeutic use. Most of the diseases for which steroids are used are characterized by inflammation, which appears secondary to a hypersensitivity reaction against auto components.[1] Corticosteroids do not interfere with the primary disease mechanisms but they are used because of their anti-inflammatory and immunosuppressive effects. Steroids are used as palliatives during the acute phase of various diseases and/or as long-term suppressors of the general host defense.[2] An important anti-inflammatory action of corticosteroids is their ability to inhibit the migration of neutrophils, monocytes and macrophages to the site of inflammation (Ward 1966, Parrillo and Fauci 1979). Corticosteroids may block the effects of certain lymphokines (macrophage inhibition factor) on their target cells thereby preventing local accumulation at the inflammatory site (Balow and Rosenthal 1973, Weston et al. 1973). They also interfere with the cytotoxic potential of macrophages (Dimitriu 1976).[3]

The pharmacological properties of the corticosteroids ensure their common therapeutic use in the treatment and palliation of disorders such as rheumatoid arthritis, osteoarthritis, collagen disorders, allergic and hypersensitivity problems, ocular, skin and renal disease and disorders of the gastrointestinal tract. For the treatment of such conditions, long term administration of corticosteroids may be necessary and under these circumstances the anti-inflammatory and immune properties of this group of drugs may extend to inhibit or infiuence the progression of plaque-associated periodontal disease (Waterhouse 1969).[4] By interfering with the synthesis of new protein and accelerating the catabolism of existing bone matrix (Jenkins 1978) prolonged therapy with corticosteroids may favour osteoporosis of bone, including that of the maxilla and mandible. Long term use of corticosteroid has a deleterious effect on soft tissue healing by inhibiting blood flow to the injured area, new blood vessel formation, immune cells like leukocytes and macrophages, protein synthesis, fibroblast proliferation and ultimately collagen formation. Cortisone weakens collagen and therefore soft tissue such as ligaments and tendons.[5]

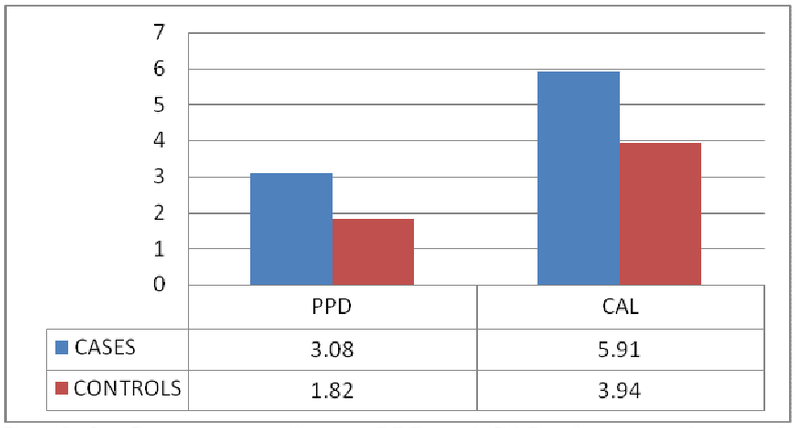
This present study was conducted to assess the periodontal status in patients with or without long term corticosteroid therapy.

Materials and methods

The study was conducted in the department of Dermatology and Nephrology at King George Hospital, Visakhapatnam. Hundred subjects aged 20 to 60 years who were on long term corticosteroid therapy for a minimum period of six months for dermatological and nephrological problems were included in the study with a mean age group of 43±2.2 years and using steroids for 13.16 ± 4.24 months. Hundred systemically healthy



Graph 1: Comparison of mean OHI-S, GI and SBI scores of cases and controls



Graph 2 : Comparison of mean PPD and CAL of cases and controls

individuals were recruited as controls. The study population ie., both the cases and the controls belonged to the same socioeconomic status. A single center, cross-sectional, case-controlled study was conducted. Informed patient consent was obtained.

Measurement of the periodontal status was done using clinical parameters like oral hygiene index- simplified (OHI-S, John C.Greene and Jack R. Vermillion, 1964), gingival index (Loe and Silness, 1964), sulcus bleeding index (Mulhemann and Son 1971), probing depth and clinical attachment loss for all teeth present. Third molars were excluded.

Statistical analysis

Statistical analysis was done using SPSS software version. p value < 0.05 was considered statistically significant. Inter group comparison with respect to oral hygiene index-simplified (OHI-S) and gingival index (GI) was analyzed by Mann-Whitney U test where as Inter group comparison of sulcus bleeding index, probing pocket depth (PPD) and clinical attachment loss(CAL) was analyzed by paired t test. Intra group comparison of each parameter was analyzed by Karl Pearson's correlation coefficient method.

Results:

Fifty eight males and forty two females constituted the test group while fifty males and forty eighty females formed the control group. In the present study, the mean OHI-S, GI, SBI, PPD and CAL for study group were 2.04 ± 0.51, 2.013 ± 0.39, 0.536 ± 0.191, 3.08 ± 1.45 and 5.91± 1.64 respectively and for the control group 2.03 ± 0.41, 2.0 ± 0.39 and 0.544 ± 0.166, 1.82 ± 1.26 and 3.94 ± 0.82 respectively. Oral hygiene index- simplified (OHI-S), gingival Index (GI) and sulcus bleeding index (SBI) did not differ significantly ($p>0.05$) when the mean values of cases and controls were compared, indicating that the oral hygiene status was similar in both the groups **(Table 1).** Mean probing pocket depth and clinical attachment loss was significantly higher in cases when compared to the controls ($p = 0.0003$) **(Table 2).**

Discussion

Corticosteroids can be employed locally or systemically. Hargitai LI, Sherman CR et al (2001) stated that long-term use of corticosteroids may lead to adrenal suppression, immunosuppression, central obesity, hyperglycemia, increased susceptibility to infection, reduction of bone mineral density (BMD) and increased risk of osteoporosis.[6] Kallali B et al (2011) reported that long-term use of corticosteroids can result in oral changes like oropharyngeal candidiasis, periodontitis and bony changes in trabecular pattern of jaw bones.[7]

In the present study, mean values of oral hygiene index- simplified (OHI-S), gingival Index (GI) and sulcus bleeding index (SBI) did not differ significantly between the cases and controls, indicating that the oral hygiene status was similar in both the groups. The patients on long term corticosteroid therapy exhibited significantly greater clinical attachment loss (CAL) and probing pocket depth (PPD) when compared to healthy controls. The study findings are in agreement with Ronderos M et al (2000) who stated that greater clinical attachment loss in patients on long term steroid therapy was associated with lowered bone mineral density.[8]

Applebaum and Seelig (1955) observed microscopic changes in the teeth and periodontal ligaments of rats following cortisone treatment. Supporting bone was lost from around molar teeth of cortisone-supplemented rats and a detailed study at high magnification revealed abnormal changes in the alveolar bone of the rats given cortisol.[9] Glickman et al. (1952) reported alveolar bone osteoporosis in white mice following daily intramuscular injections of cortisone 0.5 mg for 43 days. Histological changes in the periodontium included a reduction in the number of osteoblasts, intercellular matrix, height of alveolar bone, number of fibroblasts and collagen fibres. The cortisone injections induced changes were due to accentuated catabolism or reduced production of periodontal protein matrix.[10] Ultrastructural study involving rats, injected daily with hydrocortisone acetate 12.5 mg, demonstrated a reduction in the area of extracellular matrix which was occupied by oxytalan fibres (Bond 1986).

Isuem et al. (1956) injected cortisol preparations directly into the gingival tissues of 5 patients with periodontal disease and showed, histoiogically, reduced capillary permeability, fewer plasma cells in the granulation tissues and inhibition of collagen synthesis.[4]

In contrast to these studies a study conducted by Krohn (1958) stated that regardless of long term steroid therapy, the inflammatory changes were more severe in those subjects with poorer oral hygiene and that gingival inflammation and periodontal destruction were more dependent upon plaque control than upon corticosteroid dosage.[11]

However, the limitation of this study is that it did not have radiographic evidence to support the clinical findings. The bone mineral density (BMD) of the patients was also not recorded.

CONCLUSION

On the basis of the present study, clinical attachment loss and probing depth were significantly higher in patients under long term corticosteroid therapy than the healthy controls. It can be suggested that there is positive correlation between periodontal status and long term steroid therapy. Further longitudinal and interventional studies are warranted to prove this association.

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