Periodontal Tissue Condition on Systemic Lupus Erythematosus Patients: A Clinical Study

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Abstract

Objective: To evaluate periodontal tissue condition on systemic lupus erythematosus (SLE) patients and its characteristics. Material and Methods: This descriptive and cross-sectional study involved 61 SLE patients. Clinical examination of the oral cavity was performed using periodontal index (PI), gingival index (GI), clinical attachment loss (CAL) and number of loose teeth. Also, we evaluated SLE duration, treatment duration, ethnics, marital status, educational background, family income, and occupation. Results: In the evaluation of periodontal tissue, 93.4% had bleeding on probing, 80.3% clinical attachment loss, and 16.3% loose teeth. A total of 54 patients (88.5%) with SLE had periodontitis. Seven subjects had no periodontitis, 11 mild periodontitis, 29 moderate periodontitis and 14 severe periodontitis. Mean Periodontal Index score, Gingival Index, Clinical Attachment Loss (mm), and the number of mobility teeth, Plaque Index and Calculus Index respectively were 2.66 ± 1.20, 1.95 ± 1.02, 0.75 ± 0.59 mm, 1.49 ± 1.77. There was a significant difference in periodontal index score, shown periodontitis between employment and unemployment subjects (p=0.004) and a moderate correlation between periodontitis and occupation. Conclusion: Periodontitis found as manifestations SLE patients, followed by bleeding on probing and loose teeth. Its characteristics is playing a role in periodontitis in SLE patients.

Keywords: Lupus Erythematosus, Systemic; Periodontal Diseases; Periodontitis.
Introduction

Periodontal tissue consists of the gingiva, periodontal ligaments, cementum, and alveolar bone. The presence of bacterial infections could be inducing inflammation in periodontal tissue, resulting in progressive attachment bone loss and aggravated if systemic manifestations are present [1]. Periodontal disease begins with inflammation of gums and could develop into periodontitis. Gingivitis is found only in gingival tissue around the teeth, while periodontitis is inflammation of periodontal tissue, which could spread into periodontal tissues, associated with bacteria supplied by biofilms on teeth. As a result of periodontal disease can damage the structure of the jawbone, causing pain, disruption of activity at a more severe level, while bacterial infections continue to develop can cause systemic disease to death [2].

The main cause of periodontal disease is the presence of dental plaque. Dental plaque is a soft, yellow layer that attaches to the teeth surface. The content of dental plaque is various types of microorganisms, especially bacteria. Plaques containing pathogenic microorganisms play an important role in causing and developing periodontal destructions. Periodontal breakdown occurs because of an increasing number of gram-negative bacteria in gingival plaques, such as Porphyromonas gingivalis and Actinobacillus actinomycetemcomitans initiate periodontal infection [3].

An increasing periodontal infection could be caused by several reasons, because of chronic process lasts long in a local site or systemic conditions. Systemic diseases that play a role in this condition are the presence of blood disorders, diabetes, and rarely discussed systemic lupus erythematosus (SLE). SLE is an autoimmune disease with many clinical manifestations. SLE patients have immune system hyperactivity, which causes a decrease in the immune system’s ability to fight bacteria and the elimination of foreign antigens decreases and susceptible to infection. High autoantibodies cause hyperactivity of the immune system against the body’s own cells resulting in large tissue damage [4].

SLE has various clinical manifestations, such as atherosclerosis, malignancy, infection, and manifestation in oral cavity. The manifestation of SLE in the oral cavity can be ulcerated lesions on the mucosa, cavities, and damage to periodontal tissue, periodontitis. Xerostomia is found 75%, ulcerated lesions 15-40%, cavities (33-60%) and periodontitis (60-93%) [5]. Whereas in Indonesia, there has never been a research that shows periodontal tissue conditions in SLE patients. This study aimed to evaluate periodontal tissue condition on systemic lupus erythematosus patients and their characteristics.

Material and Methods

Study Design and Sample

The design of this study was a descriptive study with a cross-sectional approach. The sample consisted of 61 SLE patients recruited from the Department of Rheumatology, Saiful Anwar Hospital, Malang, Indonesia, from June 2017- June 2018. In all SLE patients’ clinical examination of the oral cavity to examine periodontal tissue using a periodontal index (PI), gingival index (GI), clinical attachment loss (CAL), and numbers of loose teeth. Also, we evaluated SLE duration, treatment duration, ethnics, marital status, educational background, family income, and occupation.

Inclusion criteria were female subjects, age 18-55 with a confirmed diagnose of SLE, willing to become the subject of study, could read and write and had full consciousness. Exclusion criteria were smoking, pregnancy, diabetes, and another systemic disease, no past or present history of malignant disease, particularly hematological malignancies, and patients who had hemolytic anemia or defined deficiency of vitamin B12.
Data Analysis

Data were analyzed using IBM SPSS Software, version 20 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to calculate the absolute and relative frequencies, mean and standard deviation.

Ethical Aspects

The research received ethical approval from the UB Medical Ethics Committee from the Faculty of Medical, Brawijaya University, Malang, East Java, Indonesia. All patients included in this study were required to sign informed consent.

Results

Regarding the characteristics of SLE, 23% had been diagnosed over 5 years and 18% were being treated for less than 5 years. Ethnic Java was found dominant (80.3%) and 57.3 were married. Subjects with a bachelor’s degree or more were 32.7% and 73.7% were unemployed (Table 1).

| Variables                        | N   | %   |
|----------------------------------|-----|-----|
| Duration of Illness              |     |     |
| > 5 Years                        | 14  | 23.0|
| < 5 Years                        | 47  | 77.0|
| Duration of Treatment            |     |     |
| > 5 Years                        | 11  | 18.0|
| < 5 Years                        | 50  | 82.0|
| Ethnics                          |     |     |
| Java                             | 49  | 80.3|
| Others                           | 12  | 19.7|
| Marital Status                   |     |     |
| Marriage                         | 35  | 57.3|
| Single                           | 26  | 42.7|
| Educational Background           |     |     |
| Bachelor                         | 16  | 26.3|
| Undergraduate                    | 45  | 73.7|
| Occupation                       |     |     |
| Employed                         | 20  | 32.7|
| Unemployed                       | 41  | 68.3|
| Family Income Salary per Months* |     |     |
| > Standard                       | 36  | 59.1|
| < Standard                       | 25  | 40.9|

*Standard Household income in Malang is $200.

In the evaluation of periodontal tissue in SLE patients, 93.4% had bleeding on probing, 80.3% clinical attachment loss, and 16.3% loose teeth. A total of 54 patients (88.5%) with SLE had periodontitis. Seven subjects had no periodontitis, 11 mild periodontitis, 29 moderate periodontitis and 14 severe periodontitis. Mean Periodontal Index score, Gingival Index, Clinical Attachment Loss (mm), and the number of mobility teeth, Plaque Index and Calculus Index respectively were 2.66 ± 1.20, 1.95 ± 1.02, 0.75 ± 0.59 mm, 1.49 ± 1.77 (Figure 1).

There was a significant difference in periodontal index score, shown periodontitis between employment and unemployment subjects (p=0.004). The other variables did not show statistical significance (Table 2).
Figure 1. Periodontal tissue conditions on SLE patients.

Table 2. Association between periodontal index and clinical and demographic variables.

| Variables            | Periodontal Index (Mean ± SD) | p-value |
|----------------------|-------------------------------|---------|
| Duration of Illness  |                                |         |
| > 5 Years            | 2.91 ± 0.63                   | < 5 Years | 2.58 ± 1.32 | 0.37 |
| Duration of Treatment|                                |         |
| > 5 Years            | 3 ± 0.58                      | < 5 Years | 2.58 ± 1.29 | 0.29 |
| Ethnics              |                                |         |
| Java                 | 2.66 ± 1.29                   | Others  | 2.63 ± 0.78 | 0.92 |
| Marital Status       |                                |         |
| Marriage             | 2.83 ± 1.21                   | Single  | 2.44 ± 1.18 | 0.20 |
| Educational Background|                              |         |
| Bachelor             | 2.55 ± 1.34                   | Undergraduate | 2.7 ± 1.66 | 0.67 |
| Occupation           |                                |         |
| Employed             | 3.28 ± 1.07                   | Unemployed | 2.35 ± 1.66 | 0.004 |
| Family Income        |                                |         |
| > Standard           | 2.58 ± 1.08                   | < Standard | 2.76 ± 1.37 | 0.57 |

There was a significant and moderate correlation between periodontitis and occupation. There is no correlation between periodontal index and duration of illness, duration of treatment, ethnics, marital status, educational background and family income with periodontitis (Table 3).

Table 3. Correlation between periodontal index and clinical and demographic variables.

| Variables            | p-value | r    |
|----------------------|---------|------|
| Duration of Illness  | 0.43    | 0.10 |
| Duration of Treatment| 0.31    | 0.13 |
| Ethnics              | 0.59    | 0.06 |
| Marital Status       | 0.10    | 0.20 |
| Educational Background| 0.87    | -0.02|
| Occupation           | 0.001*  | 0.41 |
| Family Income        | 0.54    | -0.07|

*Statistically Significant (p<0.05).

Discussion

Periodontal disease is common among autoimmune patients and also playing a role in the immune response. Several studies have shown there is a similar mechanism in the process of tissue damage that occurs in periodontitis and autoimmune diseases. Role of the innate and adaptive immune inducing role of
inflammatory cytokines resulting in accelerate the process of tissue destruction. Individuals with the autoimmune disease also found to have high prevalence alveolar bone \(^{[6]}\). Our result showed gum bleeding, clinical attachment loss, and loose teeth because of periodontitis. Evidence was also found there was a relationship between SLE patients and gingivitis. Recent studies in some countries were in accordance with our results \(^{[7,8]}\).

Autoantigen in SLE patients and bacterial products are inducing periodontal tissue and begin an inflammatory response. This process triggers the migration of PMN into tissues, release lysosome enzyme, which contributes to soft tissue degradation. Lymphocytes and macrophages will migrate to periodontal tissue and 60–70% of collagen be degraded at the site of the lesion, but the bone mass is still intact \(^{[9]}\).

A chronic condition in autoimmune disease prevents gingival tissue repair and further alveolar bone loss. Cytokines such as TNF\(\alpha\), IL-2, IFN\(\gamma\), IL-10 and PGE2 are found to be the dominant mediators in stimulating osteoclast activation. TNF cytokines induce via M-CSF, activate TRAF6, c-Fos and calcium signaling pathways, which are indispensable for the induction and activation of NFAT c1 as a key transcription factor for osteoclast \(^{[10]}\).

Results showed a weak correlation of occupation in SLE patients with periodontitis, and there is a difference periodontitis severity in SLE patients between employment and unemployment. These results may be influenced depending on working duration and stress in patients. Other studies also showed psychosocial disorders made patients to ignore oral hygiene and cause plaque accumulation. Poor oral hygiene could cause bacterial infections, LPS toxins originating from cell walls of gram-negative bacteria and invade periodontal tissue. This situation could increase catecholamine secretion from HPA and prevent cortisol. In addition, stress is also resulting immune function modification. Many evidence proves that emotional stress affects the severity of periodontitis \(^{[11]}\).

Ethnics, educational background, marital status, and family income also showed no correlation with periodontitis. Several genetic polymorphisms have been studied for their relationship to periodontitis, including several interleukin (IL) genes, vitamin D receptors, and Fc\(\gamma\)RIIIb-NA1 gene. However, there is still no predictive as a diagnostic or prognostic marker to identify patients in the general population who are at periodontitis risk due to population heterogeneity and different disease criteria \(^{[12,13]}\).

There were no significant correlations in the characteristics of SLE patients, which included the length of illness, duration of treatment and education. Possibilities that can affect periodontal conditions are types of drugs and knowledge in maintaining dental health. SLE is more common in ethnic Asians compared to Caucasians, and the HLA-DR gene is a risk gene in most LES patients in all ethnicities increasing 1.7 - 2.5 times risk \(^{[14]}\). SLE started from dysregulation of the immune response, causes hyperactivity of immune cells that attack the body itself. Current discovery about immune responses in SLE is still limited; thus, further research and evaluation need to be done to find an association between immune responses in SLE and periodontitis \(^{[15-17]}\).

**Conclusion**

Periodontitis found as manifestations systemic lupus erythematosus patients, followed by bleeding on probing and loose teeth. Occupancy characteristics are playing a role in periodontitis in SLE patients. Furthermore, discovery about the immune system in SLE patients is needed to find an association with periodontitis.
Authors’ Contributions

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All authors declare that they contributed to critical review of intellectual content and approval of the final version to be published.

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Conflict of Interest

The authors declare no conflicts of interest.

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