

# Comparative assessment of the prevalence of periodontal disease in subjects with and without systemic autoimmune diseases: A case–control study

S. G. RAMESH KUMAR, M. B. ASWATH NARAYANAN, D. JAYANTHI

## Abstract

**Background:** Immune mechanism shares a common pathway both for systemic autoimmune diseases and periodontal diseases. Scientific exploration of literature revealed limited studies on the association between systemic autoimmune diseases and periodontal diseases in India. **Aim:** The aim of the study is to find whether the presence of systemic autoimmune diseases in an individual is a risk factor for the development of periodontal disease. **Settings and Design:** This was a hospital-based case–control study. **Materials and Methods:** A sample of 253 patients with systemic autoimmune diseases, attending the Rheumatology Department of Government General Hospital, Chennai-3, and 262 patients without systemic autoimmune diseases, attending the outpatient department of the Tamil Nadu Government Dental College and Hospital, Chennai-3, constituted the case and control groups, respectively. Age, gender, and oral hygiene status matching was done. Oral hygiene status was assessed using oral hygiene index (OHI) and periodontal status was assessed using community periodontal index (CPI) and loss of attachment (LOA) index. **Statistical Analysis:** Statistical analysis was done using SPSS version 15 (SPSS Inc, 2006, Chicago). **Results:** Results showed 99.2% and 73.9% prevalence of gingivitis and periodontitis, respectively, in the case group as compared to 85.5% and 14.9%, respectively, in the control group. There is no linear relationship between OHI scores and prevalence of periodontitis (CPI and LOA scores) in the case group. Patients suffering from systemic autoimmune diseases showed more prevalence of periodontal diseases irrespective of oral hygiene scores. **Conclusion:** It is postulated that the presence of systemic autoimmune diseases may pose a risk for the development of periodontal diseases.

**Keywords:** Non-organ specific autoimmune diseases, Periodontal disease, systemic autoimmune disease

## Introduction

The immune system protects the host against infection specifically by recognizing and eliminating foreign agents from the body. The immune system does not normally respond to self-antigens.<sup>[1]</sup> The ability to discriminate between self-antigens and non-self-antigens is tolerance.<sup>[2,3]</sup> Immunologic tolerance is a state in which the individual is incapable of developing an immune response to a specific antigen, which can be achieved by various routes.<sup>[4]</sup>


Autoimmune diseases comprise a group of disorders where there is nothing apparently in common other than

an exaggerated immune response to one or more of the self-antigens.<sup>[5]</sup> The term autoimmune diseases refer to a disorder in which there is evidence of an immune response against self. The functional abnormality in immunologic self-tolerance directly leads to the development of autoimmune diseases.<sup>[6]</sup> A common feature of all autoimmune diseases is the presence of autoantibodies and inflammation including mononuclear phagocytes, autoreactive T-lymphocytes, and plasma cells (autoantibody-producing B-cells). Autoimmune diseases are classified into<sup>[7]</sup> organ-specific autoimmune diseases and nonorgan-specific (systemic) autoimmune diseases.

Major pathogenic role in many autoimmune diseases lies behind the damage induced by cells of the immune system against own body cells. The predominant infiltrating cells in autoimmune diseases include phagocytic macrophages, neutrophils, self-reactive cluster of differentiation (CD) 4<sup>+</sup> T-helper cells, and self-reactive CD8<sup>+</sup> cytolytic T-cells,

*Department of Public Health Dentistry, Tamil Nadu Government Dental College and Hospital, Chennai, Tamil Nadu, India*

**Correspondence:** Dr. S. G. Ramesh Kumar,  
G-1, Ram Arun Flats, Plot No. 22, 7<sup>th</sup> Street, Balaji Nagar,  
Valasaravakkam, Chennai - 600 087, Tamil Nadu, India.  
E-mail: ramesh17200@gmail.com

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with smaller number of natural killer cells, mast cells, and dendritic cells. Immune cells damage tissues directly by killing cells or indirectly by releasing cytotoxic cytokines, prostaglandins (PGE), reactive nitrogen, or oxygen intermediates.<sup>[7,8]</sup> Pathogenic mechanism common to autoimmune diseases is the increased production of the cytokines, such as tumor necrosis factor (TNF) and interleukin factor (IL)-1b.<sup>[7]</sup>

Periodontal disease is characterized by a chronic infection and inflammation in the periodontal tissue leading to the destruction of the bone surrounding the teeth and ultimately to dental loss.<sup>[9]</sup> Periodontal disease, which includes gingivitis and periodontitis,<sup>[10]</sup> is one of the most common chronic disorders of infectious origin known in humans with a high prevalence in adults. The paradigm of immune responses is consistent with the specific plaque hypothesis, current concepts in immunology, and the classic clinical and histologic observations.

Pathogenic mechanism common to autoimmune<sup>[7]</sup> and periodontal diseases<sup>[11]</sup> is the increased production of the cytokines TNF and IL-1b. Susceptible patients exhibit an abnormal immune-mediated inflammatory response.<sup>[12]</sup> In rheumatoid arthritis, there may be increased propensity to overproduce these inflammatory mediators which could possibly lead to increased periodontal destruction.<sup>[13]</sup> Rheumatoid arthritis (a type of autoimmune diseases) and periodontal diseases are associated with destruction of bone, mediated by inflammatory cytokines such as IL-1, TNF, and PGE2.<sup>[13]</sup>

Immune mechanism shares a common pathway both for systemic autoimmune diseases and periodontal diseases. Scientific exploration of literature revealed limited studies on the association between systemic autoimmune diseases and periodontal diseases in India. Thus, the aim of the study is to find whether the presence of systemic autoimmune diseases in an individual is a risk factor for the development of periodontal diseases.

## Materials and Methods

### Study design

This was a case–control study.

#### Case group

Case group constituted 253 patients with already diagnosed systemic autoimmune diseases based on diagnostic criteria attending the Rheumatology department of Government General Hospital, Chennai-3.

#### Control group

Control group constituted 262 patients without systemic autoimmune diseases matched for age, gender, and oral hygiene index (OHI) scores attending the outpatient

department of the Tamil Nadu Government Dental College and Hospital, Chennai-3.

### Inclusion criteria

- Subjects with nonorgan-specific autoimmune diseases for case group
- Subjects without systemic autoimmune diseases for control group.

### Exclusion criteria

- Subjects with any other systemic diseases for case and control groups.

### Ethical clearance

The ethical clearance of this study was obtained from the Institutional Review Board of Tamil Nadu Government Dental College and Hospital, Chennai.

### Procedure

A complete case history was recorded for both the groups. Written informed consent was obtained. Age (subgroups: 15–24, 25–34, 35–44, 45–54, 55–64, and 65–74 years), gender (subgroups: male and female), and oral hygiene scores (subgroups: OHI-original score 0–4.0, 4.1–8.0, and 8.1–12.0) matching was performed between case and control groups. Patients already diagnosed with systemic autoimmune diseases at Government General Hospital, Chennai-3 (after history, clinical examination, diagnostic tests, and markers) constituted the case group. Patients without systemic autoimmune diseases attending the outpatient department of Tamil Nadu Govt. Dental College and Hospital, Chennai-3 constituted the control group matched for age, gender, and OHI scores. Oral hygiene status of patients with and without systemic autoimmune diseases (case group and control group) was assessed using OHI by Greene and Vermillion in 1960 criteria.<sup>[14]</sup> Periodontal condition of patients was assessed using community periodontal index (CPI) with loss of attachment (LOA) by the WHO 1997 criteria.<sup>[15]</sup>

Values were tabulated and analyzed using Statistical Package for Social Sciences (SPSS) version 15 (SPSS Inc, 2006, Chicago). Categorical variables between case group and control group were analyzed using Pearson's Chi-square test. Pearson's correlation test was done to find correlation among OHI score, CPI score, and LOA score.

## Results

Female patients were more than male patients in both groups (case group: male - 61, female - 192; control group: male - 69, female - 193). The distribution of patients among case group was rheumatoid arthritis - 98, systemic lupus erythematosus - 60, spondyloarthropathy - 35, systemic sclerosis - 27, inflammatory myopathies - 11, Sjogren's syndrome - 7, vasculitis - 9, mixed connective-tissue disease - 2, antiphospholipid syndrome - 2, and adult onset

Still's disease - 2. None of the patients had OHI score of 8.1–12.

Prevalence of periodontal disease was more in case group (gingivitis 99.2% and periodontitis 73.9%) than control group (gingivitis 85.5% and periodontitis 14.9%) with  $P < 0.05$ . Prevalence of periodontal disease in male and female patients of case group was more than male and female patients of control group with significant  $P$  value [Table 1]. In all OHI groups, prevalence of periodontitis was more in case group than control group with significant  $P < 0.05$ . In OHI group, with a score of 4.1–8.0, there was no statistical difference in the prevalence of gingivitis between case

**Table 1: Comparison of periodontal disease prevalence among case group and control group within sex subgroup**

Periodontal disease	Sex group	Count (%)		$\chi^2$	$P$
		Case group	Control group		
Gingivitis	Male				
	Present	61 (100)	64 (92.8)	4.597	0.032*
	Total	61 (100)	69 (100)		
	Female				
	Present	190 (99)	160 (82.9)	30.026	0.000**
	Total	192 (100)	193 (100)		
Periodontitis	Male				
	Present	46 (75.4)	13 (18.8)	41.800	0.000**
	Total	61 (100)	69 (100)		
	Female				
	Present	141 (73.4)	26 (13.5)	140.915	0.000**
	Total	192 (100)	193 (100)		

\*Significant; \*\*Highly significant

**Table 2: Comparison of periodontal disease prevalence among case group and control group within oral hygiene scores subgroups**

Periodontal disease	OHI group	Count (%)		$\chi^2$	$P$
		Case group	Control group		
Gingivitis	0-4.0				
	Present	186 (98.9)	156 (80.8)	33.982	0.000**
	Total	188 (100)	193 (100)		
	4.1-8				
	Present	65 (100)	68 (98.6)	0.949	0.330 <sup>§</sup>
	Total	65 (100)	69 (100)		
Periodontitis	0-4.0				
	Present	139 (73.9)	14 (7.3)	176.212	0.000**
	Total	188 (100)	193 (100)		
	4.1-8				
	Present	48 (73.8)	25 (36.2)	19.095	0.000**
	Total	65 (100)	69 (100)		

\*\*Highly significant; <sup>§</sup>Not significant. OHI: Oral hygiene index

and control groups [Table 2]. Prevalence of gingivitis was more in case group than control group in all age groups except in 55–65 years and 65–74 years with statistical difference ( $P < 0.05$ ) [Table 3]. Prevalence of periodontitis was more in case group than control group in all age groups except in 65–74 years with statistical difference ( $P < 0.05$ ) [Table 3].

Prevalence of periodontitis was more in male and female patients of case group than control group in all OHI groups with statistical significant value ( $P < 0.05$ ). In the OHI group, with a score of 4.1–8, the prevalence of gingivitis among case and control groups showed no statistical difference. Patients with OHI score of 0–4 showed more prevalence of periodontitis in case group than control group in all age groups except in the age of 65–74 years, with  $P < 0.05$ . There is not much difference in the prevalence of gingivitis among groups in the age groups of 55–64 years and 65–74 years. Patients with OHI score of 4.1–8 showed more prevalence of periodontitis in case group than control group in the age groups of 15–24 years, 25–34 years, and 35–44 years, with  $P < 0.05$ . There is no statistically significant difference in the prevalence of gingivitis between groups.

Higher scores of CPI and LOA were more prevalent in all the OHI groups of case group, irrespective of OHI scores. In contrast, higher scores were seen only in higher OHI scores among control group and it reduced when OHI scores reduced [Table 4]. In case group, there was no correlation among OHI scores, CPI scores, and LOA scores, with  $P > 0.01$ . In control group, there was correlation among OHI scores, CPI scores, and LOA scores, with significant value of  $P < 0.01$  [Table 5].

## Discussion

Immune mechanism and their effects are similar for both systemic autoimmune diseases and periodontal diseases. Increased production of cytokines and abnormal immune-mediated inflammatory response<sup>[12]</sup> are the pathogenic mechanisms in both the conditions.<sup>[7,11]</sup> Studies have been done to find the association between periodontal diseases and various systemic autoimmune diseases independently. There were no studies to evaluate the periodontal condition of systemic autoimmune disease as a single group. Hence, this study has been done to evaluate the periodontal condition of systemic autoimmune diseases. Studies conducted by Ishi Ede *et al.* in 2008,<sup>[13]</sup> Biyikoglu *et al.*, in 2006,<sup>[16]</sup> Anne Havemose-Poulsen *et al.*, in 2006,<sup>[17]</sup> and Mercado *et al.*, in 2001<sup>[18]</sup> showed a positive association between periodontitis and rheumatoid arthritis in contrast with the results of the study by Fatma Yesim Bozkurt *et al.* in 2000.<sup>[19]</sup> In the present study, periodontal disease prevalence (gingivitis 98.9% and periodontitis 74.5%) was more in rheumatoid arthritis patients than control patients irrespective of OHI scores when compared to other studies. The predominant reason might be due to the

**Table 3: Comparison of periodontal disease prevalence among case group and control group within age group**

	Age group	Count (%)		$\chi^2$	P
		Case group	Control group		
Gingivitis	15-24				
	Present	39 (100)	36 (81.8)	7.847	0.005**
	Total	39 (100)	44 (100)		
	25-34				
	Present	69 (100)	67 (91.8)	5.921	0.015*
	Total	69 (100)	73 (100)		
	35-44				
	Present	84 (100)	70 (83.3)	15.273	0.000**
	Total	84 (100)	84 (100)		
	45-54				
	Present	45 (97.8)	37 (80.4)	7.180	0.007**
	Total	46 (100)	46 (100)		
55-64					
Present	10 (90.9)	10 (90.9)	0.000	1.000 <sup>s</sup>	
Total	11 (100)	11 (100)			
65-74					
Present	4 (100)	4 (100)	-	-	
Total	4 (100)	4 (100)			
Periodontitis	15-24				
	Present	28 (71.8)	1 (2.3)	43.959	0.000**
	Total	39 (100)	44 (100)		
	25-34				
	Present	52 (75.4)	7 (9.6)	63.187	0.000**
	Total	69 (100)	73 (100)		
	35-44				
	Present	64 (76.2)	11 (13.1)	67.658	0.000**
	Total	84 (100)	84 (100)		
	45-54				
	Present	31 (67.4)	16 (34.8)	9.787	0.002**
	Total	46 (100)	46 (100)		
55-64					
Present	8 (72.7)	1 (9.1)	9.214	0.002**	
Total	11 (100)	11 (100)			
65-74					
Present	4 (100)	3 (75)	1.143	0.285 <sup>s</sup>	
Total	4 (100)	4 (100)			

\*\*Highly significant; <sup>s</sup>Not significant

difference in the sample size. Kobayashi *et al.* in 2003<sup>[20]</sup> and Novo *et al.* in 1999<sup>[21]</sup> showed the risk of development of periodontitis in SLE patients in their study. The present study also revealed the same. Oral hygiene scores were not similar between case group and control group in the

**Table 4: Comparison of community periodontal index score among case group and control group within oral hygiene scores group**

OHI score	Parameter Score	Count (%)		$\chi^2$	P	
		Case group	Control group			
OHI score 0-4	CPI	0	2 (4.5)	34 (73.9)	66.433	0.000**
		1	2 (4.5)	8 (17.4)		
		2	6 (13.6)	4 (8.7)		
		3	4 (9.1)	0 (0)		
		4	30 (68.2)	0 (0)		
	Total		44 (100)	46 (100)		
OHI score 4.1-8.0	CPI	0	0 (0)	0 (0)	51.855	0.000**
		1	0 (0)	29 (42)		
		2	17 (26.2)	13 (18.8)		
		3	13 (20)	21 (30.4)		
		4	35 (53.8)	6 (8.7)		
	Total		65 (100)	69 (100)		
OHI score 0-4	LOA	0	51 (27.1)	176 (91.2)	183.284	0.000**
		1	20 (10.6)	16 (8.3)		
		2	96 (51.1)	1 (0.5)		
		3	21 (11.2)	0 (0)		
		4	0 (0)	0 (0)		
	Total		188 (100)	193 (100)		
OHI score 4.1-8.0	LOA	0	17 (26.2)	42 (60.9)	38.912	0.000**
		1	8 (12.3)	20 (29)		
		2	39 (60)	7 (10.1)		
		3	1 (1.5)	0 (0)		
		4	0 (0)	0 (0)		
	Total		65 (100)	69 (100)		

\*\*Highly significant. OHI: Oral hygiene index; CPI: Community periodontal index; LOA: Loss of attachment

study conducted by Novo *et al.* Studies conducted by Kuru *et al.* in 2002<sup>[22]</sup> and Boutsis *et al.* in 2000<sup>[23]</sup> showed no difference in the periodontal status of Sjogren's syndrome patients with healthy controls. Among Sjogren's syndrome patients, prevalence of periodontal disease (85.7%) was more than controls irrespective of OHI scores. The difference in results might be due to sample size. Scardina *et al.* in 2005<sup>[24]</sup> showed the presence of alteration in periodontal mucosa microcirculation in 15 systemic sclerosis patients by using periodontal capillaroscopy. In the present study, prevalence of periodontal disease had been observed instead of capillary alterations in systemic sclerosis patients, which could be a probable reason for the difference. Pischon *et al.* in 2010<sup>[25]</sup> reported that there was 6.81-fold increased risk of periodontal disease in ankylosing spondylitis patients. This study also showed similar results that spondyloarthropathy patients show an increased risk of periodontal disease development with prevalence of gingivitis 100% and periodontitis 77.1%. Márton *et al.* in 2005<sup>[26]</sup> could not



**Table 5: Correlation among oral hygiene scores score, community periodontal index score, and loss of attachment score among case and control groups**

Groups	Index and Values	OHI score	CPI score	LOA score
Case group	OHI score			
	Correlation value ( <i>r</i> )	1	-0.005	-0.100
	<i>P</i>	-	0.936 <sup>s</sup>	0.113 <sup>s</sup>
	CPI score			
	Correlation value ( <i>r</i> )	-0.005	1	0.918
	<i>P</i>	0.936 <sup>s</sup>	-	0.000**
	LOA score			
	Correlation value ( <i>r</i> )	-0.100	0.918	1
	<i>P</i>	0.113 <sup>s</sup>	0.000**	-
Control group	OHI score			
	Correlation value ( <i>r</i> )	1	0.589	0.348
	<i>P</i>	-	0.000**	0.000**
	CPI score			
	Correlation value ( <i>r</i> )	0.589	1	0.799
	<i>P</i>	0.000**	-	0.000**
	LOA score			
	Correlation value ( <i>r</i> )	0.348	0.799	1
	<i>P</i>	0.000**	0.000**	-

\*\*Highly significant; <sup>s</sup>Not significant. OHI: Oral hygiene index; CPI: Community periodontal index; LOA: Loss of attachment

demonstrate any difference in the severity of periodontal destruction between inflammatory myopathies patients and healthy controls. The present study showed more prevalence of gingivitis (100%) and periodontitis (63.6%) in case group than control patients. A study by Márton *et al.* observed patients who were affected with hyposalivation and also with minor salivary gland fibrosis. This may be a contributing factor in the variation of results. Schenkein *et al.* in 2007<sup>[27]</sup> observed elevated levels of inflammation markers in periodontitis patients with high anticardiolipin, which is an antiphospholipid antibody. The present study showed that all patients with antiphospholipid syndrome had periodontal disease, but the sample size is too small. There are no studies that evaluated the periodontal status of the patients with mixed connective tissue disorder and adult onset disease.

These observations denote that the prevalence and severity of periodontal disease were more in case group than control group. In this study, among case group, it revealed no correlation among OHI scores, CPI scores, and LOA scores. Highest score of CPI prevailed more in case group

(Score 4–58.1%) than control group (score 4–2.7%). Score 3 of LOA prevailed only in case group (8.7%).

Oral hygiene plays an important role in the development of periodontal disease, but the present results revealed that the prevalence of periodontal disease among various systemic autoimmune diseases was irrespective of OHI scores. Patients in the case group had more probing depth as well as LOA than control group. Hence, this study demonstrates that systemic autoimmune disease is one of the risk factors for the development of periodontal diseases. Conversely, in patients with periodontal diseases, it could be advisable to check for any unidentified systemic autoimmune diseases. Further studies with large sample size are warranted to prove the association between systemic autoimmune diseases and periodontal diseases.

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#### Conflicts of interest

There are no conflicts of interest.

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