Short Communication

Role of Interleukin-17A in Endodontic Disease

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Abstract

Background: Endodontic disease is a complicated condition marked by inflammation of the apical tissues and is influenced by microbial, immunological, and environmental variables. Inflammatory cells, proinflammatory, and immunoregulatory cytokines are all recruited as part of the intricate immunological host response that occurs here; one of these cytokines is interleukin-17A (IL-17A). **Objectives:** This study aimed to determine the concentration of IL-17A by Enzyme Linked ImmunoSorbent Assay (ELISA) in the saliva of patients with endodontic disease to find the correlation between IL-17A and endodontic disease. **Materials and Methods:** The samples are collected from 60 patients (n = 60) with an age range of 20–50 years. The samples are collected from specialized dental centers and private clinics throughout the time period (December 2021–February 2022). The control samples are 20 (n = 20). The rate of samples was divided between males and females with ratios of 43.33% and 56.66%, respectively. **Results:** The result showed that the concentration of IL-17A was increased in patients compared with the control P-value (0.000). The difference in age showed no effect on the results of this study. **Conclusion:** This study showed that proinflammatory cytokines start immune responses in pulp tissues after bacterial infections and imply IL-17A endodontic illness.

Keywords: Endodontic disease, IL-17A, immune response, proinflammatory cytokines

Introduction

The most common causes of endodontic inflammation are severe caries lesions or leaking fillings when bacteria and/or its product reach the pulp (pulpitis).[1] Endodontic infections are polymicrobial, including a combination of Gram-positive bacteria such as *Streptococci*, *Parvimonas micra*, *Actinomyces* species, *Propionibacterium* species, *Lactobacilli*, *Enterococci* and Gram-negative pathogenic groups such as *Bacteroides*, *Porphyromonas*, *Fusobacterium*, and Campylobacter are invading the necrotic pulp tissue.[2-4]

Enterococcus faecalis has shown to be the utmost communal kinds detected in human oral infections, they are facultatively anaerobic, Gram-positive organisms that are thought to be transient elements of the oral microbiome, and it may contribute to a variety of oral and systemic illnesses, although it has been seen in conditions like periodontitis, dental caries, and tooth root infection.[5] The bacteria enter the dentin tubuli and directly communicate with the pulp through the metabolic byproducts of the bacteria, like acids; innate immunity is instantly activated by host cells following their detection of microbial components.[6] Phagocyte activity and cytokine synthesis are both components of the innate immune response. This creates a feedback loop that amplifies innate processes and connects them to the later emergence of adaptive immunity; these alterations in the pulp tissues may result in severe pain that could last for several days.[7]

The etiology of the periapical lesion depends on proinflammatory and immunoregulatory cytokine,[8] Th17 cells perform critical roles in the establishment of autoimmunity and the progression of inflammation by producing IL-17.[9]

IL-17 is thought to be an essential connecting molecule between the adaptive and innate immune systems because it is noticeably a proinflammatory cytokine with significant

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effects on many cells of the innate immunological network, mainly the granulocyte lineage.[10,11] A member of the IL-17 cytokines family, interleukin 17A (IL-17A), IL-17A might show a crucial role in the exacerbation of inflammation in a periapical lesion, in response to IL-17A exposure, fibroblasts, epithelial cells, osteoblasts, and endothelial cells, release proinflammatory cytokines such IL-6 and IL-8, matrix metalloproteinase and granulocyte colony-stimulating factor.[12]

Materials and Methods

Patient characteristics

This work includes 60 patients exposed to endodontic disease and 20 control with good oral hygiene to compare the concentration of IL-17A. In the discussed groups, most of the patients were female (56.6%), and the respondent's age ranged from 20 to 50 years.

Saliva sampling

Samples from each participant were collected in a contaminant-free environment. Each subject had their saliva sampled, which was around 5 mL. Each individual was inquired to prevent eating, drinking, smoking, and using the oral hygiene product for at most 60 min before sampling saliva. All patients' mouths were rinsed for 30–60 s with purified water (10 mL) to verify that any debris was removed. Nonstimulated clean saliva was collected in sterile laboratory cups Samples were centrifuged at (3000 rpm) for 10 min to separate the unwanted free salivary particles. The supernatants were collected and cryopreserved at -20°C till the measurements.[13]

Estimating the concentration of salivary cytokines

The salivary concentration of IL-17A was assessed with utilize of commercially accessible ELISA kit (Elabscience/USA). This ELISA kits use sandwich-ELISA as the method. The ELISA microplate was already precoated within an antibody that recognizes human IL-17A. The specific antibody and stands or samples are placed in the ELISA microplate wells. Then, a biotinylated detection antibody specific for human IL-17A and an avidin-horseradish peroxidase (HRP) mixture is added gradually to and incubated in each microplate well. Free elements are distant by washing. To each well, the substrate solution is applied. The only wells that will be blue in color contained IL-17A of human, biotinylated recognition antibody, and avidin-HRP conjugate. After adding stop solution, the enzyme-substrate reaction is stopped, and the color changes to yellow. The optical density (OD) is determined spectrophotometrically at 450 ± 2 nm wavelength; the OD values are related to the human IL-17A concentration. It was determined the concentrations of human IL-17A in the sample via comparing the OD of the sample with that of standard curve.

Statistical analysis

Statistical analyses were achieved utilizing the statistical package SPSS. Results are stated as (mean ± SD) Independent sample *t*-test was utilized for comparing patient and control systemic. The quantitative variables are presented as mean and standard deviation (mean ± SD) values. *P*-value was used for comparisons between two groups and later used to compare three age groups, $P < 0:05$ was considered to be an indication of statistical significance.[14]

Ethical approval

This study was carried out in conformity with the ethical standards outlined in the Helsinki Declaration. Before taking the sample, the patient's verbal and analytical consent were obtained. To obtain this permission, a local ethics committee evaluated and approved the study protocol, subject information, and consent form using document number 566 (containing the number and date on January 30, 2022).

Results

The result shows that the concentration of IL-17A was significantly higher in endodontic disease with M and SD (48.434 ± 20.91) for patients, and in control group, M and SD were (22.011 ± 5.826) and *P*-value (0.000*) found in Table 1.

The age range from 20 to 50 years was distributed into groups; the first age group was 20–30 years with M and SD of age (46.862 ± 18.475) in patients, while in control M and SD (23.536 ± 7.535) and *P*-value (0.000*), the second group of age was 31–40 years with M and SD of age (42.792 ± 14.032) in patients, while in control M and SD (23.536 ± 7.535) and *P*-value (0.001*), the last age group 41–50 years with mean and SD of age (47.071 ± 22.527) in patient, while in control M and SD (19.532 ± 4.046) and *P*-value (0.001*). There is a large noteworthy difference between the control and patient in the whole age group ($P \leq 0.05$), and our results revealed that was no positive significant correlation between the age group and IL-17A shown in Table 2.

Discussion

IL-17A is induced via numerous extracellular bacterial infections, and the induction at innate immunity levels was detected via several of the bacteria. The result of this study

Table 1: The mean and standard deviation for IL-17 concentration in endodontic patients and control group ($P \leq 0.05$)

Parameter	M ± SD		*P* value
	Patients	Control	
IL-17A	48.434 ± 20.91	22.011 ± 5.826	0.000***

***High significant

Table 2: The mean and standard deviation for IL-17A concentration in endodontic patients and control group according ($P \leq 0.05$)

Parameter (years)	M ± SD		*P* value
	Patients	Control	
20–30	46.862 ± 18.475	23.536 ± 7.535	0.000****
31–40	42.792 ± 14.032	23.536 ± 7.535	0.001**
41–50	47.071 ± 22.527	19.532 ± 4.046	0.001*

* significant at $P \leq 0.05$

** significant at $P \leq 0.01$

***High significant at $P \leq 0.001$

shows the correlations between salivary concentrations of examined cytokine and endodontic disease. In agreement with other studies of[15-17] a positive statistically noteworthy correlations among a salivary concentration of IL-17A in the groups of the whole patient.

The results showed that the production of IL-17A was significantly higher in endodontic disease ($P < 0.05$). In previous work, the researchers determined IL-17A expression and its correlations within neutrophil infiltrations besides the type of T lymphocyte effector (CD4+ or CD8+) in periapical abscesses and Periapical granulomas. The findings show that there are a considerably higher number of CD4+/IL17A positive cells compared within CD8+/IL-17A cells both in the periapical abscesses and granulomas. The chief source of IL-17A in the abscesses and granulomas was shown CD4+ cells.[18]

However, the existence of IL-17A was related to an exacerbations of inflammatory responses, a raised number of neutrophils, and bone-resorption.[19,20] These findings are in comparison to the fact that effector Th17 cells are mainly localized in inflamed tissues. The study also showed that there was no significant effect of age on the concentration of interleukin. The results showed that the *P*-value was close in all age groups ($P \leq 0.05$), and the concentration of interleukin increased in all age groups of patients compared to control.

Conclusion

Result of this study improved that proinflammatory cytokine play an significant role in the start of immune responses in the pulp tissues following bacterial infections and suggests the role of IL-17A endodontic disease.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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