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University of Baghdad
College of Dentistry

INVOLVEMENT OF IL-17A GENE POLYMORPHISM AND IL-23, IL-12 LEVELS IN THE DEVELOPMENT OF PERI–IMPLANTITIS

A thesis
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ABSTRACT

Background: Dental implantation has been practiced since ancient times and has gone through several stages. Dentists use dental implants to support dental prostheses such as crowns, bridges, dentures, face prostheses, or as an orthodontic anchor.

Material and Methods:

This cross-sectional study included 80 subjects (15 peri-implantitis patients, 35 successful implants and 30 healthy controls); their mean age was 43.91±11.33 years). Peri-implant sulcus fluid samples was collected from all subjects (patients with peri-implantitits, successful implants and healthy controls) attending the department of Oral and Maxillofacial Surgery in the Dental College Teaching Hospital/ Baghdad University, and Shahid Ghazi Al-Hariri Hospital/ Medical City Baghdad, Iraq. The period between March 2021 to November 2021. Enzyme-linked immunosorbent assay was carried out to estimate the Peri-implant sulcus fluid levels of interleukin-23 and interleukin-12. While interleukin-17A gene polymorphism in blood samples was conducted by a polymerase chain reaction.

Results:

The current study revealed an obvious significant elevation in the mean level of interleukin-23 in the peri-implantitis patient's group more than its level in the successful implant and control groups ($P< 0.05$). The increased level of this inflammatory cytokine (interleukin-23) might add to the systemic inflammatory burden a predisposing factor, which may lead to an impaired osseointegration and subsequent bone loss or implant failure. It is well known that interleukin-23 its pro-inflammatory properties. Regarding

interleukin-12, which is considered a pro-inflammatory mediator involved in the inflammatory response, its increase, alone, does not appear to affect the osseointegration of dental implants or worsen the peri-implantitis condition. This means that elevated levels of IL-12 have no direct or strong effect on inflammation-induced bone loss.

Conclusion: These results suggest that IL-17A gene polymorphism may play a role in peri-implant disease susceptibility, especially in persons carrying the rs2275913 A allele at a higher risk of developing peri-implantitis as compared with those carrying the G allele. Furthermore, the risk of peri-implantitis was observed among people who had IL-17A /AA or GA genotypes, namely A allele-containing genotypes. The increased level of IL-23 that could be causing devastating bone loss. Thus, IL-23 can act as a biomarker to predict peri-implant diseases and treatment efficacy.

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