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Review Article

Alopecia areata & periodontal diseases- A connecting link: A review

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ABSTRACT

The systemic condition alopecia areata causes non-scarring hair loss on the scalp, face, or any other portion of the body. The disease's unknown aetiology and pathophysiology have a significant impact on patients' social lives, which over time causes stress and hastens the condition's progressive decline. In these patients, quick dental evaluation and treatment have improved the outlook for alopecia in addition to immunomodulators and stress-relieving exercises. This illustrates a connection between oral infection foci and baldness. Periodontitis has persisted as one of the most common types of oral focus of infection for systemic disorders during the past few decades. The current analysis sheds information on how stress and autoimmune disease play a role in determining whether alopecia.

1. Introduction

Periodontal diseases are characterized by localized infections and inflammatory conditions where anaerobic Gram- negative bacteria are mainly involved and directly affect teeth-supporting structure. Periodontal disease affects one or more of the periodontal tissues—alveolar bone, periodontal ligament, cementum, and gingiva. The pathogenesis of this disease involves immunological responses leading to tissue destruction and bone loss.[1]

Autoimmunity can be defined as breakdown of mechanism responsible for self-tolerance and induction of an immune response against components of the self. Such an immune response may not always be harmful (e.g., anti-idiotype antibodies).[2]

2. Evidence of Autoimmunity in Periodontal Disease

1. Enhanced presentation of self-antigens through increased expression of the molecule associated with antigen presentation, namely, the IgA antigen;
2. Polyclonal activation of cells which have the ability, for reasons unclear, to produce autoantibodies;
3. Altered T helper or T suppressor cell function;
4. Genetic predisposition factors.
5. Idiosyncrasies of the antigen idiotype network
6. Bacterial or viral cross reactivity with self-antigens
7. Leading to production of cross- reactive antibodies.[3]

3. Altered T-Helper and T Suppressor Cell Function

It has been proposed that periodontal disease causes a decrease in the T cell population of lymphocytes, which results in an increase in the B cell component of spontaneous lymphoblastic proliferation. This would imply that patients with periodontal disease would experience an increase in T suppressor activity.[4]

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4. Increased IgA Expression

Increased levels of antigen-presentation molecules, particularly on cells that do not typically express these molecules, are linked to an increase in autoantibodies in a number of autoimmune disorders. Autoantibodies may be produced as a result of self-antigen being presented to the immune system by these cells, which are ordinarily not engaged in antigen presentation.[5]

5. Polyclonal Expansion of B Cell Pool

In the absence of disease, natural autoantibodies (Nab) are discovered to a variety of self-components and are thought to be a natural occurrence. By one of the polyclonal B cell activators found in periodontal plaque (LPS), such a pool of Nab may be extended polyclonally, or it may be expanded selectively antigenically by the release of tissue components that expose these natural antigens to the immune system more than usual.[6]

6. Idiosyncrasies of the Antigen Idiotypic Network

According to Jerne's network hypothesis of the immune response, an antigen triggered the synthesis of an antibody with an idiotype, which in turn prompted the development of an anti- idiotype with an "internal image" of the antigen. This open- ended network resulted from the latter antibody, which in turn encouraged the creation of anti-anti-idiotype antibodies[7]

Anti-idiotype antibodies have not yet been proven to exist in periodontal disease. Humans and experimental animals with periodontal disease have antibodies to immunoglobulin that are directed towards the Fc end of the molecule rather than the idiotype region.[8]

7. Autoantigen Presentation and Autoreactive T Cells

All nucleated cells of the body possess class I MHC molecules so that gingival epithelial cell and fibroblast killing reported to occur in periodontal disease in vitro could be mediated by this mechanism of presentation of antigen, in association with class I MHC molecules, to cytotoxic T cells. However, in the lymphocytotoxicity seen in periodontal disease, both allogeneic and syngeneic target cells have been used with similar results. Increased expression of class II MHC molecules has been observed in the surface of basal and suprabasal gingival sulcular epithelial cells in inflamed tissues.[9]

8. Viral and Bacterial Infection and the Immune System

Infections caused by bacteria and viruses may function as an external trigger for an autoimmune reaction. An important stage in the pathophysiology of autoimmune disorders is the activation and clonal proliferation of autoreactive cells. Activated autoreactive T cells can spread disease in experimental models of autoimmunity, but not dormant ones, proving that activation of autoreactive T cells is necessary for the onset of autoimmune disorders. Infectious agents function as a stimulus for these reactions.[10]

As a result, the presence of such a bacterium in the periodontal pocket may induce the formation of antibodies both to the microbe and to serum components or degenerating host periodontal tissues.

9. Alopecia

Alopecia refers to a medical condition that causes hair loss, typically on the scalp but sometimes affecting other parts of the body as well. It is not a single disease but rather an umbrella term for various forms of hair loss. The most common type is called alopecia areata, which is an autoimmune condition where the immune system mistakenly attacks the hair follicles, resulting in hair loss.

Apart from alopecia areata, there are other types such as alopecia totalis (total loss of scalp hair), alopecia universalis (loss of all body hair), and androgenetic alopecia (patterned hair loss commonly known as male or female pattern baldness). The exact cause of alopecia is unknown, but it is believed to be a combination of genetic and environmental factors. Autoimmune disorders, hormonal changes, certain medications, and high levels of stress may contribute to the development of alopecia.[11] The primary symptom of alopecia is hair loss. In alopecia areata, hair loss usually occurs in small, round patches on the scalp. In more severe cases, it can progress to total hair loss on the scalp (alopecia totalis) or complete loss of body hair (alopecia universalis). A dermatologist can diagnose alopecia based on the pattern of hair loss and by ruling out other possible causes. Sometimes, a scalp biopsy or blood tests may be recommended to confirm the diagnosis.[12] There is no known cure for alopecia, but there are treatment options available to manage the condition. The choice of treatment depends on the type and extent of hair loss. Some common approaches include topical medications, injections of corticosteroids into the affected areas, oral medications, and in certain cases, hair transplantation. Hair loss can have a significant psychological and emotional impact on individuals, affecting their self-esteem and quality of life. Support groups, counseling, and therapies such as cognitive-behavioral therapy (CBT) can help individuals cope with the emotional challenges associated with alopecia.[13]

Autoimmunity is a double-edged sword in the sense that its exaggerated response leads to the destruction of the cell, at the same time, it has a protective role too (anti-idiotype antibodies). Alopecia areata (AA) is one of the most common autoimmune conditions.

10. Correlation of Alopecia Areata with Oral Foci of Infection

In their report from 2014, Dinkova et al.[14] focused on an AA case that had a dental focus. A 55-year-old female was investigated in that study to learn more about the potential causes of alopecia. It was noted that the patient's alopecic areas didn't exhibit evidence of hair regrowth until after the excision of decaying and defective root canal treated teeth. This finding supports the idea that localised infections contribute significantly to immunologic disorders.

A case of AA in a 37-year-old male patient with an oral fistula apical to a central incisor with pulpal necrosis was described by Gil Montoya et al.[15] Following root canal therapy, the bald areas totally vanished, pointing to the importance of understanding localised infections in alopecic individuals.

Lesclous and Maman.[16] in 1997 observed a mechanical irritant (rather than bacterial) to be a triggering factor in AA. It was observed that a 35-year-old male patient who reported bilateral dull retromandibular pain in the jaws due to impacted mandibular third molars, had a complete remission of AA subsequent to the extraction of both the wisdom teeth.

Fatemi et al. in 2016 published a case report of a young male patient with a diagnosis of localized mild chronic periodontitis with AA and observed hair regrowth in the focal spot after complete periodontal treatment. This finding suggests a plausible role of periodontitis as an essential factor to be considered in the course and development of AA.

Jaya, Nand and Rameshwari 2023 discussed in detail about interrelationship between Alopecia and Periodontitis. They observed the stress is the common causative factors for causing Alopecia and Periodontitis in patients. They observed increased stress leads to increased level of P,VIP,CRH.[17]

11. Discussion

Although the precise aetiology of AA has not yet been fully defined, numerous possibilities are being investigated to reach the closest conclusion. While the majority of the available research focuses on an autoimmune cause illustrating circulating autoantibodies and inflammatory infiltrates near the HFs, dental origin or foci of infection connected to the origin of AA cannot be ignored. Endogenic immunoinflammatory agents like cytokines, kinins, complement fragments, neuropeptides, lysosomal enzymes, and fibrinolytic peptides emerge as a result of the complex interactions between oral irritants (mechanical, chemical, or microbial) and the host protector cells, dividing immune responses into cell-mediated and antigen-antibody-mediated immune responses. Given that several studies revealed the presence of immune complexes that were widely distributed in the systemic circulation (mostly in acute oral infections).

A trigeminal-sympathetic reflex mechanism is the basis for an alternative theory for the dental origin of AA, which is supported by a small number of studies. In contrast to the centrifugal conduction involving the sympathetic nucleus close to the terminal branches of the trigeminal nerve, which may impel vasoconstriction of the pilosebaceous follicle and cause multiple alterations with subsequent hair loss, a distant mechanical or infectious stimulus triggers a centripetal response involving a triple-neuron system.

12. Conclusion

Although there is no known explanation for the uncommon autoimmune disease AA, several theories have been put up to try and come up with one. Some of these theories include stress, localised infections, and autoimmunity involving harmful cells like IL-1 alpha and beta and TNF-. Numerous dental illnesses, such as periodontitis, are the catalysts of this inflammatory cascade that prevents the growth of cells in a number of pilar follicles, interrupting the synthesis of hair or encouraging its elimination. This justifies the doctor sending such patients to the dentist office for consultation or referral. However, to make a definitive diagnosis, lengthy clinical studies are required.

13. Source of Funding

None.

14. Conflict of Interest

None.

References

1. Pizzo G, Guiglia R, Russo LL, Campisi G. Dentistry and internal medicine: From the focal infection theory to the periodontal medicine concept. *Eur J Intern Med*. 2010;21(6):496–502.
2. Shaju JP, Zade RM, Das M. Prevalence of periodontitis in the Indian population: A literature review. *J Indian Soc Periodontol*. 2011;15(1):29–34.
3. Tonetti MS, Jepsen S, Jin L, Otomo-Corgel J. Impact of the global burden of periodontal diseases on health, nutrition and wellbeing of mankind: A call for global action. *J Clin Periodontol*. 2017;44(5):456–62.
4. Nair S, Faizuddin M, Dharmapalan J. Role of autoimmune responses in periodontal disease. *Autoimmune Dis*. 2014;p. 596824. doi:10.1155/2014/596824.
5. Alkhalifah A, Alsantali A, Wang E, Mcelwee KJ, Shapiro J. Alopecia areata update: Part I. Clinical picture, histopathology, and pathogenesis. *J Am Acad Dermatol*. 2010;62(2):177–88.
6. Bonjour J. Observations on alopecia areata. *Urol Cutan Rev*. 1932;36:674–82.
7. Mirzoyev SA, Schrum AG, Davis MD, Torgerson RR. Lifetime incidence risk of alopecia areata estimated at 2.1% by rochester epidemiology project. *J Invest Dermatol*. 1990;134(4):1141–2.
8. Safavi K. Prevalence of alopecia areata in the First National Health and Nutrition Examination Survey. *Arch Dermatol*. 1992;128(5):702. doi:10.1001/archderm.1992.01680150136027.

9. Chu SY, Chen YJ, Tseng WC, Lin MW, Chen TJ, Hwang CY, et al. Comorbidity profiles among patients with alopecia areata: The importance of onset age, a nationwide population-based study. *J Am Acad Dermatol*. 2011;65(5):949–56.
10. Marketon JIW, Glaser R. Stress hormones and immune function. *Cell Immunol*. 2008;252(1-2):16–26.
11. Sroussi HY, Williams RL, Zhang QL, Villines D, Marucha PT. Ala42S100A8 ameliorates psychological-stress impaired cutaneous wound healing. *Brain Behav Immun*. 2009;23(6):755–9.
12. Nour M, Belkacem CR, Darej M, Touil D, Oualha L, Douki-Tunisia N, et al. Alopecia areata of dental origin. About a clinical case. *Odontostomatol Trop*. 2018;41:13–9.
13. Fatemi K, Mohammadipour H, Forouzanfar A. Alopecia areata associated with generalized mild chronic periodontitis: A case report and review of the literature. *Res J Pharm Biol Chem Sci*. 2016;7:1563–6.
14. Dinkova A, Kirova D, Gavasova G, Drangov M, Gospodinov DL. Case of alopecia areata originated from dental focus. *J IMAB*. 2014;20(5):669–73.
15. Montoya JAG, Soriano AC, Prat JJ. Alopecia areata of dental origin. *Med Oral*. 2002;7(4):303–8.
16. Lesclous P, Maman L. An unusual case of alopecia areata of dental origin. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1997;84(3):290–2.
17. Jaya N, Lal N, Singhal R. Alopecia and Periodontitis: Exploring the connecting dots. *J Indian Soc Periodontol*. 2023;27(3):233–7.

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