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Alopecia Areata Associated with Generalized Mild Chronic Periodontitis: A Case Report and Review of the Literature.

Kazem Fatemi¹, Hamideh Sadat Mohammadipour² and Ali Forouzanfar^{3*}.

¹Oral & Maxillofacial Diseases Research Center, School of Dentistry, Mashhad University of Medical Sciences, Mashhad, Iran.

²Dental Materials Research Center, Department of Operative Dentistry, School of Dentistry, Mashhad University of Medical Sciences, Mashhad, Iran.

³Research Center for Patient Safety, Mashhad University of medical sciences, Mashhad, Iran.

ABSTRACT

Alopecia areata is described by the patch like hair loss on the scalp or elsewhere on the body with the prevalence rate of less than 1% in the general population. Although the etiology and pathogenesis of alopecia areata is unclear, recent studies have suggested the role of genetics, environment, infections and psychological stresses in the development of the disease. Alopecia areata occurs in both males and females with a mild predilection in younger adults who are apparently healthy and have no other skin disorder. Since the hair loss usually involves the face and scalp, esthetic considerations play an important role in the management of the disease. This article reports a case of alopecia areata associated with generalized mild chronic periodontitis.

Keywords: Alopecia areata, Hair loss, Chronic Periodontitis, Focal infection.

**Corresponding author*

INTRODUCTION

Alopecia areata (AA) is a common dermatologic condition in which hair is lost from some or all areas of the body specifically from the scalp without skin inflammation or scarring. The prevalence of Alopecia Areata is estimated to be about 0.1–0.2% of the general population, but the exact cause of the disease is still unknown [1,2]. Historically the first clinical report of alopecia areata dates back to Celsus (14 to 37 B.C). Celsus described two forms of alopecia. The first form that he described was complete hair loss occurring in people of all ages and second form occurring in the children. For acknowledgement to Cornelius Celsus, Alopecia areata is sometimes named as "area celsi"[3]. Throughout history Alopecia areata has been presented by different names, but the actual term "alopecia areata" was first used by Sauvages in 1760 [4]. Willan and Gruby in 1843 proposed the role of fungi for the etiology of alopecia areata and Jacquet hypothesized the disease could cause by focal infections such as oral or periodontal lesions [5]. Recent studies suggested several etiologic factors such as autoimmune disturbances, hereditary and genetic susceptibility, environmental status, psychological stress and microcirculation interruptions for the etiology and pathogenesis of Alopecia Areata [6, 7].

Case Report

A young man was referred to the periodontist in Mashhad special dental clinic for scaling. His past medical history and general physical examinations were unremarkable. During extra-oral examination a localized circular area of hair loss at his forehead that measured approximately 2x2 cm in diameter discovered (Fig 1). No other sign of alopecia or any other skin lesions in the whole body was present. The patient's chief complaint was gum bleeding during brushing. The gingiva was red, swollen and non-scalloped. There was a significant amount of supra-gingival and sub-gingival calculus and dental plaque around teeth (Fig 2). Clinical examination revealed mild attachment loss, moderate probing depth and bleeding on probing in almost all parts of the mouth. Orthopantomogram revealed mild bone loss at the interproximal alveolar bone crest particularly on the anterior part of the mandible (Fig 3). On the basis of dermatologic and intraoral clinical and radiographic findings the diagnosis of localized mild chronic periodontitis associated with Alopecia Areata was given and for treating of periodontal inflammation meticulous scaling and root planning, antibiotic (amoxicillin 500 mg, three times daily for 8 days) and chlorhexidine (0.2%) as a mouthwash prescribed [8, 9]. After periodontal therapy the patient reported that the alopecia was controlled and he was satisfied for the treatment. This case report represents the theoretical possibility of periodontitis as a risk indicator for the occurrence or progression of alopecia areata.

Figure 1: Photographs showing alopecia areata at the right forehead area of the scalp.



Figure 2: Photographs showing dental plaque, calculus and gingival inflammation.



Figure 3: Orthopantomogram showing mild bone loss at the interproximal alveolar bone.



DISCUSSION

Periodontal disease is an inflammatory condition of tooth supporting structures initiated by bacterial pathogens. The interactions between these pathogens and host inflammatory cells lead to the production of destructive enzymes that are responsible for damaging the epithelial and connective tissue cells. The resulting inflammatory processes are usually presented as gingival inflammation and redness, bleeding on probing, alveolar bone loss and tooth mobility. Several factors that affect periodontal disease development and progression through several pathways include environment, genetic susceptibility, nutrition and stress which could be also considered in the etiology of alopecia areata. On the other hand systemic conditions such as endocrine, hematologic and genetic disorders can alter the initiation and progression of gingivitis and periodontitis by affecting neutrophils, monocytes, macrophages and lymphocytes function. These alterations may manifest clinically as destructive periodontal disorders [10-13]. In 1900, British physician William Hunter first developed the concept of focal infection that oral and periodontal pathogens can play an important role in the etiology of many systemic diseases. Although Hunter's theory of focal infection has changed during past decades, today's evidence-based medicine and dentistry has confirmed the possible relationships between periodontal infection and systemic disorders. A similar immune-inflammatory mechanism may be responsible for the progression of alopecia areata during periodontitis [14-17].

Recent studies have revealed the potential effects of inflammatory periodontal diseases on several organ systems and human health. The presence of complex gram negative anaerobic pathogens in periodontal structures of patients suffering from periodontitis results in a distinctive and persistent bacterial challenge that is responsible for a general immune-inflammatory response. In addition periodontal pathogens and their products, such as *lipopolysaccharide* (LPSs), penetrate to the blood circulation through the sulcular epithelium of periodontal pocket, which is frequently ulcerated and discontinuous. This phenomenon is called bacteremia, which often occurs in patients with periodontitis during normal daily function, oral hygiene procedures and even after mechanical periodontal therapy [18, 19]. This bacteremia and the immune-inflammatory host response explain the mechanisms for the interactions between periodontal infection and a variety of systemic disorders like alopecia areata. For this reason several antimicrobial strategies have been applied for the treatment of periodontal diseases and related disorders [20, 21].

CONCLUSION

Alopecia areata is a rare dermatologic condition but with significant psychosocial considerations for the patients due to the esthetic consequences which can affect both men and women at any age. Although the exact etiology and the mechanisms of its development are not yet understood, the association of alopecia areata and focal infections such as dental or periodontal may be explained by the autoimmune nature of the disorder or other joint risk factors. It could be also suggested that inflammatory cytokines (Interleukin-1 alpha and beta or Tumor necrosis factor) which are produced during periodontitis may inhibit the proliferation of cells in the pilar follicle, by this means interrupting hair synthesis or destruction. However, more longitudinal

case-controlled trials and cross sectional studies are needed to investigate the exact interrelationships of both diseases.

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