http://dx.doi.org/10.5272/jimab.2014205.669

Journal of IMAB
ISSN: 1312-773X
http://www.journal-imab-bg.org

OPEN ACCESS

Journal of IMAB - *Annual Proceeding (Scientific Papers)* 2014, vol. 20, issue 5

CASE OF ALOPECIA AREATA ORIGINATED FROM DENTAL FOCUS

Atanaska Dinkova, Donka Kirova, Galina Gavasova, Martin Drangov, Dimitar L. Gospodinov

Department of Oral surgery, Faculty of Dental Medicine, Medical University – Plovdiv, Bulgaria

SUMMARY:

For many years the oral infection, especially periodontitis, is considered as a potential contributing factor to a variety of clinically important systemic diseases.

Alopecia areata (AA) is an autoimmune disease with unclear etiology and pathogenesis, rarely associated with dental foci. This disease has a strong psychological impact on the patient, because it presents with hair loss and can affect any hair-bearing area, but usually involves the face and scalp, where esthetic considerations play an important role in self perception.

In this article is presented a case of alopecia areata resulting from dental foci that was effectively resolved by eliminating a focalized dental infection via dental extraction.

In this sense, patients with AA should be subjected to careful exploration of the oral cavity in search of possible dental infections. Close collaboration between dentist, dermatologist, endocrinologist and other medical specialists in the interdisciplinary approach of diagnosis and treatment is needed.

Key words: Alopecia areata, Dental focus, Dental focal infection, Periodontitis

INTRODUCTION:

Focal infection

In 1891 W Miller published his Theory of Focal Infection that postulates a myriad of diseases caused by bacteria, fungi, viruses or their toxins that arise endogenously from a focus of infection.

A focal infection is a confined area that contains pathogenic microorganisms, can occur anywhere in the body and usually causes no clinical manifestations. It leads to localized or generalized infection caused by the dissemination of microorganisms or toxic products from a focus of infection.[1]

Primary foci can arise from the tonsils, adenoids, sinuses and oral cavity with less common from the prostate, appendix, gallbladder and kidney.

Secondary foci are the direct result of infections from other foci through contiguous tissues, or at a distance through the blood stream or lymph channels resulting in development of arthritis, neuritis, myalgias, nephritis, osteomyelitis, endocarditis, brain abscesses, prosthetic joint infections, skin abscesses, pneumonia, asthma, anemia, alopecia, gastritis, pancreatitis, colitis, diabetes, emphysema, goiter, thyroiditis, Hodgkin's disease, fever of unknown origin, stupidity and 'nervous diseases of all kinds'. Currently, diseases postulated to be caused by microorganisms include cancer, sarcoidosis, multiple sclerosis, amyotrophic lateral sclerosis, autism, Guillain–Barre´ syndrome, pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections. [2, 3, 4, 5]

Oral primary foci have traditionally been ascribed to pyorrhea alveolaris (periodontitis), alveolar abscesses and cellulitis, pulpless teeth, apical periodontitis, general oral sepsis and endodontically treated teeth with viridans group streptococci (VGS) being the principal metastatic microbial culprits. [2, 3, 6]

Nowadays oral focus is most commonly connected with chronic periodontitis and systemic diseases. In July 1998, the American Academy of Periodontology stated that infections in the mouth may play an important role in disorders involving other parts of the body. At present it is generally agreed on that oral status is connected with systemic health, since poor oral health may occur concomitantly with more serious underlying diseases and/or it may predispose to other systemic diseases. [1, 4, 7, 8, 9, 10, 11]

In **Chronic periodontitis p**eriodontal pathogens, which are gram negative predominantly, and their toxins, such as cytolitic enzymes and lypopolisaccharide may access the blood stream through the compromised and/or ulcerated epithelium of the periodontal pocket. Within the inflamed gingival tissue are produced inflammatory mediators as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1â, prostaglandin E2 (PGE2), and ã-interferon which can access the blood stream and contribute to the global inflammatory burden. Thus, the systemic exposure to periodontal pathogens, their toxins, and periodontal derived/elicited inflammatory mediators may determine pathologic consequences in different organ or systems. [12, 13, 14, 15]

Three mechanisms by which periodontal infection may influence systemic health have been described: 1. metastatic infection caused by translocation of Gram-negative bacteria from the periodontal pocket to the bloodstream; 2. metastatic injury, such as vascular lesions from the effects of circulating microbial toxins and pro-inflammatory mediators; 3. metastatic inflammation due to the immunological response to the periodontal pathogens and their toxins. [3, 16, 17]

On the basis of epidemiological studies the association between chronic periodontitis, and cardiovascular disease, respiratory diseases, diabetes, osteoporosis, chronic kidney disease, rheumatoid arthritis, Alzheimer's disease, Alopecia areata, Alopecia totalis and other diseases have been proposed. [4]

Alopecia

Hippocrates first used the term alopecia but the characteristics of the hair loss disease were first described by Cornelius Celsus in 30 A.D. with two forms: first - complete baldness occurring in people of all ages; second he called "ophiasis", which literally translates as "snake", due to the winding way the bald region spread across the skin only seen in children. Alopecia areata is sometimes known as "area celsi" in tribute to Cornelius Celsus.

The actual term "alopecia areata" was first used by Sauvages in his "Nosologica Medica", published in 1760 in Lyons, France.

From the 1800's onwards there was debate about the cause of alopecia areata and two main hypotheses were put forward: one based on parasitic infection (Gruby 1843, Radcliffe-Crocker 1903); the other based on a nervous disorder (Von Barensrung 1858).

One of the more unusual variations on the neuropathic origin of alopecia areata was put forward by Jacquet (1902) who suggested alopecia areata was initiated by sources of nerve irritation such as defective and diseased teeth. Jacquet's hypothesis was apparently confirmed by others (Decelle 1909).

With the start of the 20th century alopecia areata was associated with disorders of the endocrine glands (Sabouraud 1913), particularly the thyroid, due to a hormone dysfunction and toxic agents and syphilis which often manifested itself by sudden, rapid loss of hair in well defined patches, just like alopecia areata (Ormsby 1948, Roxburgh 1950), before progressing to development of lesions and sores. [18, 19, 20]

Alopecia areata (AA) is an autoimmune disease with unclear etiology and pathogenesis which presents with hair loss and can affect any hair-bearing area. Attacks of alopecia are often associated with other autoimmune conditions such as lupus and allergies. In about 20% of cases, the patient is related to someone who has or has had the disease. [21]

It often presents as well demarcated patches of alopecia on skin of overtly normal appearance.

Usually starts out as smooth, small, round or oval patches of baldness that rapidly form on one side of the head. The edges of the patches are usually studded with "exclamation point hairs." Erythema of the skin can be present early on. [22, 23]

Alopecia areata appears as one or two bald patches on one side of the head, but also causes thinning all over the scalp. In the areas of baldness, the hairs are very short, broken, and narrow. Alopecia areata has a variable course. In some cases, the bald patches regrow within a few months or a year, but sometimes, extensive patchiness develops. Extensive alopecia has a poor prognosis. There is about a 25% recurrence rate.

ILCSs are the preferred method of treatment. Newer topical and systemic agents (eg, biologics) have been tried, but the outcomes have been unattractive. Various therapeutic agents have been described for the treatment of alopecia areata (AA), but none are curative or preventive. [23, 24, 25]

CASE REPORT:

A 55-year-old female presented at the Oral surgery department, Medical university, Plovdiv, referred from a dermatologist for focal infection diagnostic in association with limited hair loss.

The patient was referred from dermatologist for focal diagnosis in connection with restricted hair loss on the parietal area.

Complaints of the patient were tooth roots that interfered with eating and caused bad breath.

In late February 2012 during a visit to the hairdresser was first noticed hair loss of two restricted areas of the parietal area of the head. Patient related the onset of the hair loss with stress.

A month later the patient was referred from her GP to the dermatologist and to the gynecologist, but from hormone and blood tests results no cause of alopecia was found.

Dermatologist diagnosed Alopecia areata (AA) and prescribed treatment with Revalid (1 Capsula containing: 100 mg methionine, 50 mg cysteine, 50 mg calcium pantothenate, 1.5 mg tiamin hidrohlorid, 10 mg piridoksin hidrohlorid (vitamin B6), 20 mg of parabens, 50 mg extract of millet, 50 g wheat germ extract, 50 mg of brewer's yeast, 2 mg of iron, zinc and 2 mg 0.5 mg copper) and special shampoo.

In May 2014 the dermatologist added to the treatment vitamins and retinol. After few month condition of the patient deteriorate and Dermatologist referred her to dentist for focal infection tests.

The past medical history includes 2 prior hair losses in her youth which patient connected with stress. Because of this history she postponed the dental visit for 2 months.

The patient was otherwise fit and healthy not taking any medication.

She was a non-smoker did not use alcohol.

There was no family history with similar findings.

The general physical examination revealed a moderately built patient with satisfactory vital signs. Extraoral examination revealed alopecic area on parietal area of the head with dimensions 10 x 9 cm, smooth, and well demarcated

Picture 1.

Picture 2.

Right submandibular lymph nodes with soft consistency were palpable on the extra-oral examination.

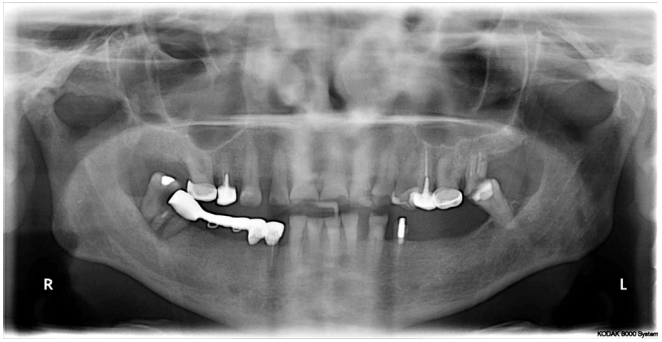
Intraorally no pathological findings on the oral mucosa were found. Artificial crowns were found on the teeth 16, 15, 25, 26, Bridge on abutment teeth 43-47 and teeth roots 27, 48, 34 which were decayed on probing, pain free on palpation and percussion.

Laboratory tests were undertaken to confirm the definite diagnosis.

Panoramic radiograph revealed root canal treatment on teeth 15, 25, 27, 34, 43,47.

Periapical lesions were observed on teeth 34, 43, 47, 48.

Picture 3.



Local reactivity test - Electro skin testing of Gelen was performed.

Due to skin reaction for active focuses were suspected teeth 43, 47 and 48. Teeth 15, 27 and 34 were defined as potential focuses.

A test for bimetallism was found negative.

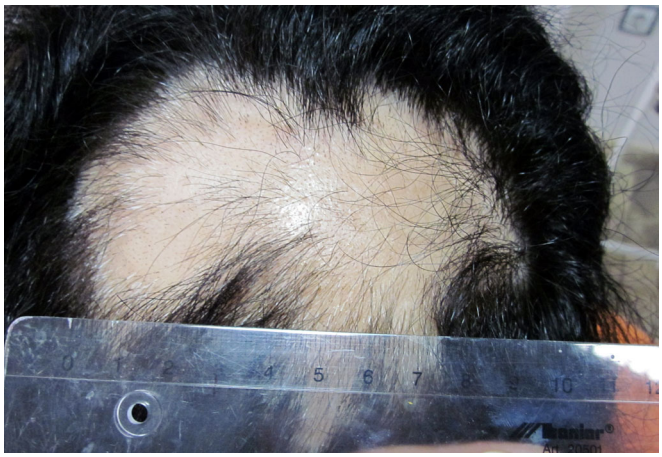
Teeth 27, 34, 43, 47, 48 were extracted.

Tooth 15 is due to root canal treatment.

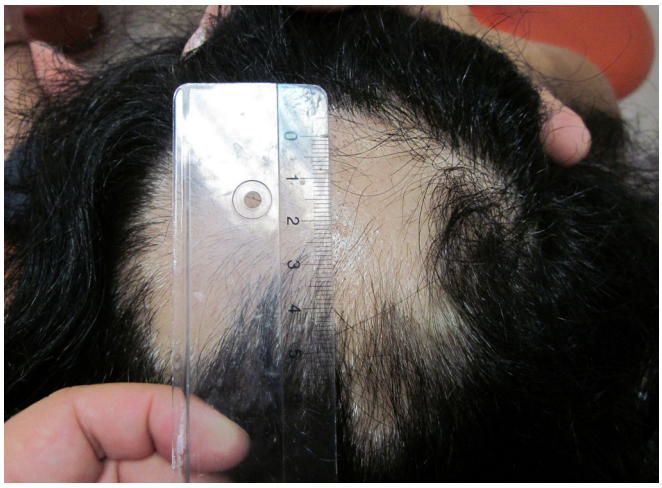
Healing process was normal without any complications.

A week after the teeth was extracted the patient reported cessation of the hair loss. Month after treatment there were new hair grow observed.

Picture 4.

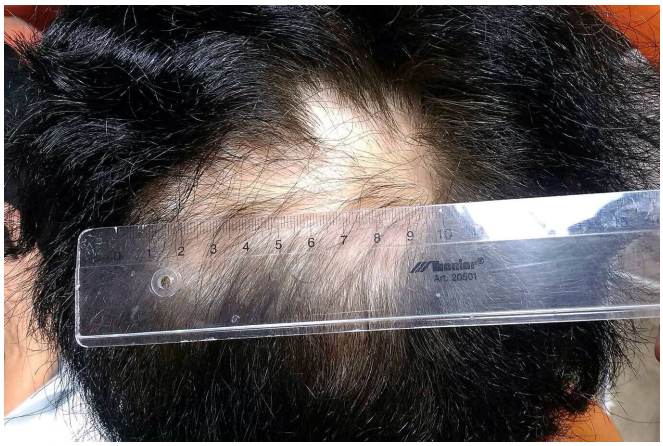


Picture 5.



3 months after dental treatment new hair growth was observed.

Picture 6.



Picture 7.

DISCUSSION:

Myriads of less or more serious local and systematic diseases are believed to originate from dental focal infection. Some of them are associated with serious disability of the patient, while others affect the appearance and additionally have a serious psychological impact.

Alopecia areata (AA) usually appears on the head and face and has a great impact on the appearance and psyche of the afflicted individual.

In many cases of AA the etiology factor is difficult to be revealed, and usually the standard treatment doesn't lead to desired healing process.

There is no general agreement on its etiology and pathogenesis, although the immunologic hypothesis is most frequently put forward. An emotional stress is also very frequently associated with the start of an alopecic disease. Dental focus associated with AA is relatively rare, but should not be underestimated.

Consequently in AA a global approach is necessary, because unscarred restricted alopecia is most often of multi factorial origin, and so the treatment must be based on a multidisciplinary approach and close collaboration between dentists, and other medical specialists.

In this case after the focus was eradicated with extraction of the affected teeth, only complete regrowth can corroborate, a posteriori, whether dental factors are the sole cause.

CONCLUSION:

This case of alopecia areata shows the relationship between the oral diseases and other local or systemic disorders. Medical community should be aware of the potential negative effects of periodontal infections on systemic health. First of all, periodontal infections must be recognized and treated, and then a regular oral care must be maintained. Treatment of focal infections promotes a strong collaboration between dental and medical professionals: combination of dental and internal medicine implicates a better communication among the stake-holders and an effective team approach in the clinical practice.

REFERENCES:

1. Indramohan J, Karthika B, Mohiddin G. Myth of Endodontics in Oral Focal Infection. *Indian J of Multidiscip Dentistry*. Nov 2011 - Jan 2012; 2(1):380-382.

2. Pallasch TJ, Wahl MJ. Focal infection: new age or ancient history? *Endodontic Topics.* 2003 Sep;4(1):32–45. [CrossRef]

3. Gammal R. Dead teeth can affect your health. Focal infection is real. November 2012, p.1-132.

4. Pizzo G, Guiglia R, Lo Russo L, Campisi G. Dentistry and internal medicine: from the focal infection theory to the periodontal medicine concept. *Eur J Intern Med*. 2010 Dec;21(6):496-502. [PubMed] [CrossRef]

5. Wisher FP. Focal Infection, A Medico-Dental Problem. *Cal West Med.* 1925 Aug;23(8):977-980. [PMC]

6. Miller WD. The human mouth as a focus of infection. *The Lancet.* Volume 138, Issue 3546, Pages 340 - 342, 15 August 1891; Available online 28 September 2003. [CrossRef]

7. Zivkovic S. [Endodontic treatment in the therapy of alopecia areata.] [in Croatian] *Stomatol Glas Srb.* 1990 Jun;37(3):299-305. [PubMed]

8. Balcheva M, Abadjiev M. A case of alopecia areata, associated with a focus of dental origin. *J of IMAB.* 2009; 15(2):73-74, [CrossRef]

9. Atanasov D. (editor), Oral Surgery. *Plovdiv*. 2011. pp.232-233 [in Bulgarian]

10. Botushanov P. Diagnosis and treatment of diseases of oral mucosa. *Plovdiv: Auto Spectar.* 1997; p.56-58. [in Bulgarian]

11. Botushanov P, Vladimirov St. Endidontics. Theory and practice. *Plovdiv; Auto Spectar.* 1998; p. 532-572. [in Bulgarian]

12. Teng Y-TA, Taylor GW, Scannapieco F, Kinane DF, Curtis M, Beck JD, et al. Periodontal Health and Systemic Disorders. *J Can Dent Assoc*. 2002 Mar;68(3):188-92 [PubMed]

13. Scannapieco FA. Position paper of The American Academy of Periodontology: periodontal disease as a potential risk factor for systemic diseases. *J Periodontol*. 1998 Jul;69(7):841-50. [PubMed]

14. Jacinto RC, Gomes BP, Shah HN, Ferraz CC, Zaia AA, Souza-Filho FJ. Quantification of endotoxins in necrotic root canals from symptomatic and asymptomatic teeth. *J Med Microbiology.* 2005 Aug;54(Pt 8):777–783. [PubMed] [CrossRef]

15. Morita T, Yamazaki Y, Mita A, Takada K, Seto M, Nishinoue N, et al. A cohort study on the association between periodontal disease and the development of metabolic syndrome. *J Periodontol.* 2010 Apr;81(4):512–9. [PubMed] [CrossRef]

16. Li X, Kolltveit KM, Tronstad L, Olsen I. Systemic diseases caused by oral infection. *Clin Microbiol Rev.*

2000 Oct;13(4):547–58. [PubMed] [CrossRef]

17. Thoden van Velzen SK, Abraham-Inpijn L, Moorer WR. Plaque and systemic disease: a reappraisal of the focal infection concept. *J Clin. Periodontol*. 1984 Apr;11(4):209–220. [PubMed]

18. 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 Feb;62(2):177-188. [PubMed] [CrossRef]

19. Grossman L. Pulpless teeth and focal infection. *J Endod.* 1982 Jan;8(Suppl):S18-S24. [CrossRef].

20. Bonjour J. Observations on alopecia areata. *Urological and Cutaneous Review*. 1932; 36: 674-82.

21. Lew BL, Shin MK, Sim WY. Acute diffuse and total alopecia: A new subtype of alopecia areata with a favorable prognosis. *J Am Acad Dermatol*. 2009 Jan;60(1):85-93. [PubMed] [CrossRef]

22. Amin SS, Sachdeva S. Alopecia areata: A review. *J Saudi Society of Derm & Derm Surgery.* 2013 Jul;17(2): 37-45. [CrossRef]

23. Tosti A, Bellavista S, Iorizzo M. Alopecia areata: A long term follow-up study of 191 patients. *J Am Acad Dermatol.* 2006 Sep;55(3):438- 441. [PubMed] [CrossRef]

24. Alkhalifah A, Alsantali A, Wang E, McElwee KJ, Shapiro J. Alopecia areata update Part II. Treatment. *J Am Acad Dermatol*. 2010 Feb;62(2):191-202. [PubMed] [CrossRef]

25. Lesclous P, Maman L. An unusual case of alopecia areata of dental origin. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 1997 Sep; 84(3):290-292. [PubMed] [CrossRef]

Please cite this article as: Dinkova A, Kirova D, Gavasova G, Drangov M, Gospodinov DL. CASE OF ALOPECIA AREATA ORIGINATED FROM DENTAL FOCUS. *J of IMAB.* 2014 Oct-Dec;20(5):669-673.
doi: http://dx.doi.org/10.5272/jimab.2014205.669

Received: 22/09/2014; Published online: 09/12/2014

CC BY NC SA

Address for correspondence:
Atanaska Dinkova, DMD
3 Ledenuka str., Plovdiv 4002 , Bulgaria
tel: +359886711031
E-mail: dinkova_asia@yahoo.com