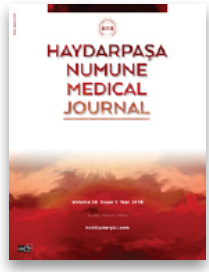
HAYDARPAŞA NUMUNE MEDICAL JOURNAL

DOI: 10.14744/hnhj.2018.42104
Haydarpasa Numune Med J 2018;58(2):102–105



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REVIEW

Diffuse Alopecia

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Abstract

Hair loss is a common health problem. It leads to serious anxiety and emotional stress in patients. Changes in hair cycle are responsible for the pathogenesis of diffuse alopecia. Diffuse alopecia is categorized into two groups, namely anagen effluvium and telogen effluvium, according to affected hair. Detailed history, dermatological examination, and laboratory investigations, if needed, are important for diagnosis. In this review, diffuse alopecia has been discussed, along with its diagnosis and treatment.

Keywords: Anagen effluvium; diffuse alopecia; telogen effluvium.

Hair loss is a frequently encountered health problem and occurs in more than half of the total men and women once during their life time. Relevant cosmetic concerns lead to serious anxiety and emotional stress. Alopecia is generally divided into cicatricial (leading to permanent hair loss) and non-cicatricial (leading to transient hair loss). It can be also classified as local and diffuse alopecia [1]. In this review, diffuse non-cicatricial alopecia will be discussed.

Changes in the hair cycle are responsible for the pathogenesis of diffuse alopecia [2]. Hair cycle comprises the phases of anagen, catagen, and telogen. In normal healthy individuals, anagen (active growth phase) lasts for 2–6 years, catagen (resting stage) for 2-3 weeks, and telogen (thinning, and shedding hair) for approximately 3 months. Generally, the anagen phase lasts longer in women [3].

The average number of hair cycles during one's lifetime ranges between 10 and 20. Every day, approximately 100–200 hairs are lost, and this may differ based on individual and seasonal changes. Hormones, cytokines, growth factors, and environmental factors (toxins, deficient nutrition, vitamin, and energy deficiency) affect the hair cycle [2]. Hair follicle is the fastest developing tissue after bone marrow in our body. Hair follicles need calories, proteins, trace elements, and vitamins for intensive synthesis. In case of deficiency of the required essential constituents, hair loss can be seen, hair disorders may be triggered, and the diseases may become refractory to treatment. Hair follicle provides the required energy for the synthesis from carbohydrates. In individuals on a low-calorie diet, hair follicles try to provide the required energy from amino acids, leading to a decrease in both energy and essential constituents. Thus, the hair become thin and easily break [4].

Pathological Dynamics of Hair Loss

Many factors may cause pathological hair loss. The pathological process involving the hair cycle that leads to diffuse alopecia is categorized into two groups: dystrophic anagen effluvium and functional telogen effluvium [2].

Dystrophic Anagen Effluvium: It is characterized by the sud-

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Submitted Date (Başvuru Tarihi): 07.03.2018 **Accepted Date (Kabul Tarihi):** 08.03.2018

den arrest of mitotic activity in rapidly dividing matrix cells, leading to hair loss in the anagen phase [2]. *Functional Telogen Effluvium:* It is characterized by increased hair loss during the telogen phase. It has five different types:

i: Immediaye anagen release: It is observed rather frequently and is characterized by passing of premature anagen hair follicule into the telogen phase. It is observed following physiological stress associated with high fever. Pyrogens and released cytokines initially induce apoptosis of hair follicles. It is observed 3–4 months after the onset of physiological stress.

ii: Delayed anagen release: As a classical entity, hair follicles that grow during pregnancy pass into the telogen phase during the postpartum period and are later shed (Fig. 1).

iii. Immediate telogen release: Hair normally falls at the end of 100 days; in conditions of rapid passage into the anagen phase, it may be shed prematurely. Loss of telogen hair filaments rapidly passing into the anagen phase after minoxidil treatment is an example of this type of hair loss.

iv: Delayed telogen release (hair loss): Although it resembles molting (skin replacement) in mammalians, it is characterized by prolonged telogen phase. Seasonal hair loss is an example of this type of hair loss.

v. Short anagen phase: It is defined as an increase in hair loss due to abnormally short scalp hair. It can be observed in hereditary hypotrichosis, ectodermal dysplasias, or even healthy children [2].

Diagnostic methods in Cases with Diffuse Alopecia

Diagnosis of diffuse alopecia primarily requires detailed anamnesis, examination of the scalp, and determination of hair loss pattern [5]. Methods used in the diagnosis of hair loss are divided into three groups: noninvasive, semi-invasive, and invasive. The noninvasive diagnostic method, called the pull test, is mostly sufficient for diagnosis. In diffuse alopecia, the pull test is positive on the vertex and hair line. Rarely, semi-invasive methods, such as trichogram analysis, and invasive methods, such as scalp biopsy, are used to finalize the diagnosis [6]. If needed, various blood tests can be performed [5].

Figure 1. Marked thinning and shedding of the scalp hair at postpartum 4 months (rapid loss of telogen hair following prolonged anagen phase).

Clinical Characteristics of Diffuse Alopecia

Diffuse alopecia is divided into two groups based on the type of hair: anagen effluvium and telogen effluvium [2]. Although not classified in this category, androgenetic alopecia (female-type hair loss) may lead to diffuse alopecia. In women with genetic predisposition, it induces noncicatricial alopecia, characterized by typical distribution pattern with miniaturization of hair follicles. Contrary to male baldness, androgens do not definitely play a role in its pathogenesis; therefore, the term "female-type hair loss" is found to be appropriate for describing this type of hair loss. It may appear without hyperandrogenemia. Hair follicles are hypersensitive to normal levels of androgens. Its onset may occur at any age, but it manifests most frequently in the 40–50-year age group. Slowly progressing hair loss and thinning constitute its clinical findings. Most frequently, vertex, upper parietal region, and frontoparietal region are affected [7].

1. Anagen Effluvium: It is divided into two categories: dystrophic anagen effluvium and loose anagen hair syndrome.

i. Dystrophic Anagen Effluvium: It may be observed in alopecia cases following the use of antineoplastic drugs (chemotherapy-induced alopecia), exposure to radiation (radiation-induced alopecia), and environmental or occupational exposure to toxins (toxic alopecia) and in patients with alopecia areata. Chemotherapy-induced hair loss manifests 1–3 weeks after the treatment onset and peaks at 1 and 2 months post-chemotherapy. Because there are more number of scalp hair in the anagen phase, they are more frequently affected (Figs. 2, 3). However, eyebrows, eyelashes, and terminal hair in the axillary and pubic regions may also be affected. It is generally reversible; however, it has also been reported in patients receiving busulphan chemotherapy]. Radiation-induced alopecia may be reversible or permanent depending on the depth of the applied radiation. Following exposure to toxic metals, toxic alopecia may be observed. Many heavy metals may impair the structure of the hair shaft by forming covalent bonds

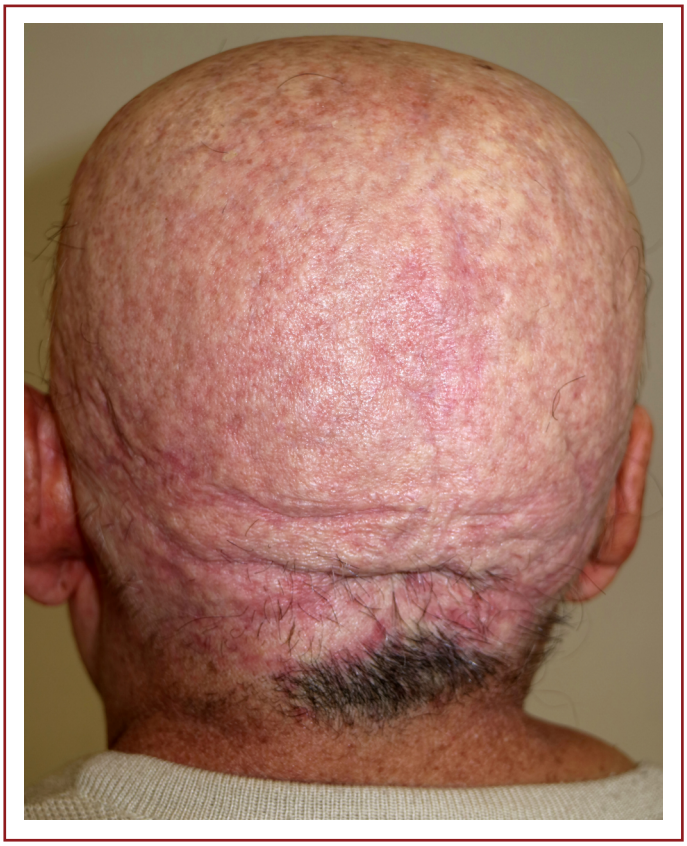

Figure 2. Nearly complete hair loss at 2 months after chemotherapy (anagen effluvium).

with sulfhydryl groups found in keratin. Tallium, mercury, arsenic, copper, cadmium, and bismuth are responsible for causing toxic alopecia. Alopecia areata is the most frequently observed type of dystrophic anagen effluvium, which is observed in otherwise healthy adults and children. Alopecia areata may be localized or generalized. It is an organ-specific autoimmune disease, characterized by peribulbar lymphocytic infiltration [2].

ii. Loose Anagen Hair: It develops as a result of nonadhesion between the hair shaft and hair follicle due to premature keratinization of the inner root sheath. Sporadic and familial cases have been reported. Mostly, the patient age at diagnosis is approximately 2 years (average, 6 years). Its onset is very rarely observed at advanced ages. The most important clinical finding is no growth of hair. Hair in the anagen phase can be easily plucked. Hair are generally thin, sparse, and short [2].

2. Telogen Effluvium: It is the most frequent cause of noncicatricial hair loss. It is more frequently observed in women. According to the duration of hair loss, it is divided into two groups: acute telogen effluvium (lasting ≤6 months) and chronic telogen effluvium (lasting >6 months). Potential triggering factors which might cause acute telogen effluvium include acute and chronic major diseases, collagen tissue diseases, major surgery, delivery, rapid weight loss, low-protein and low-carbohydrate diet, nutritional deficiencies, congenital or acquired zinc deficiency, endocrinological disorders, drugs, toxins, serious emotional stress, and inflammatory diseases of the scalp [9]. Hair loss is <50% of the total scalp hair. Thinning of the scalp hair may be observed; especially, temporal hair line may recede. Patients with reliable anamnesis with a triggering event should be reassured that they will not lose all their scalp hair and that this condition is transient. In patients without any history of triggering event, tests requested should be directed to suspect the underlying etiology and drug use, which may lead to hair loss. In patients with chronic telogen effluvium, iron deficiency, thyroid disease, other metabolic diseases (chronic renal failure, malignancy, pancreatic disease, malabsorption), systemic lupus erythematosus, other connective tissue diseases, HIV infection, and drugs that may cause hair loss should be investigated. Chronic telogen effluvium is more frequently observed in women. It is characterized by the recession of the bitemporal hair line. Pull test positivity is detected in both the vertex and occiput. It is differentiated from androgenetic alopecia by hair loss at the vertex. It has a fluctuating clinical course. Seasonal changes may be observed. Goal-targeted treatment should be administered. Because hair loss involves a circumscribed area, treatment is not required for acute telogen effluvium, mild

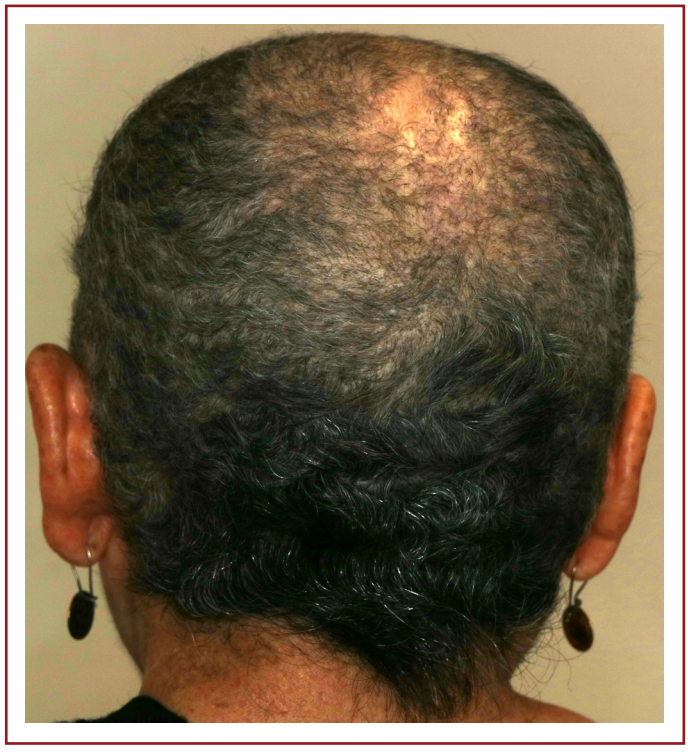

Figure 3. Nearly complete recovery of the scalp hair.

seasonal telogen effluvium, and hair loss observed at the start of minoxidil treatment. In cases with chronic telogen effluvium, if the underlying etiology can be determined, goal-targeted treatment should be employed. Especially in young women, dietary habits should be questioned, and anorexia nervosa should be considered. Although many drugs may lead to hair loss, most frequently, anticoagulants (heparin, warfarin), oral retinoids (acitretin, isoretinoin), interferon, antithyroid drugs, hypolipidemic drugs (fibrates), colchicine, and commonly used antimetabolites may lead to hair loss[2]. The role of iron in hair loss has not been definitively determined. Iron required for hemoglobin synthesis is first metabolized in hair follicles. Therefore, in cases of iron deficiency, hair loss may be observed before the onset of anemia. Although any standardization in ferritin levels for ideal hair development has not been established, ferritin levels of <40 ng/ml lead to increase in the telogen phase, those between 40 and 70 ng/ml prolong the telogen phase, and those >70 ng/ml lead to transition into the anagen phase. Although hair loss is not seen in every individual with iron deficiency, as an accepted corollary, depletion of iron stores in genetically predisposed individuals leads to hair loss. Therefore, increasing ferritin levels to >70 ng/ml is advised in individuals with hair loss. To date, any conclusive data on the association between zinc deficiency and hair loss have not been reported. In an individual with normal healthy dietary habit and without any relevant symptoms (periorificial dermatitis, angular cheilitis, and diarrhea) who has hair loss, zinc deficiency is not suspected.

Although treatment in cases with chronic telogen effluvium frequently fails, food supplements have been investigated in some studies. Studies have shown that L-cysteine is helpful in the development of hair. When cysteine is given together with B complex vitamins, an improvement in trichogram and phototrichogram findings and increase in the quality and durability of hair have been observed. In a study where L-cysteine, vitamin B complex, and medical mold were used for 6 months, a significant increase in anagen hair filaments was observed compared with the placebo. Yellow corn is a natural product containing salicylic acid, amino acids, vitamins, and minerals. As shown in many studies, yellow corn extracts together with supportive food supplements containing cysteine and calcium pantothenate increased the anagen hair. Favorable effects of biotin and niacin on the development of hair are already known. Biotin deficiency is rarely seen. Favorable effects of biotin supplements on patients without biotin deficiency have not been demonstrated. Biotin supplements are recommended for patients who consume raw eggs and use valproic acid [4].

Physicians in every field of medicine may frequently encounter diffuse alopecia cases, and diagnosis can be made based on detailed anamnesis and physical examination; laboratory examinations may be rarely required. After revelation of the potential causes of hair loss, the patient should be informed that complete hair loss is impossible, and goal-targeted treatment should be recommended.

Peer-review: Externally peer-reviewed.

Financial Disclosure: The authors declared that this study received no financial support.

Authorship Contributions: Concept: G.A.; Design: G.A., S.A.; Data Collection or Processing: G.A.; Analysis or Interpretation: G.A.; Literature Search: G.A., S.A.; Writing: G.A.

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