International Journal of Pharmacy and Pharmaceutical Studies **ISSN: 2457-0419**

Vol. 5, Issue I, Jan-Mar, 2021 http://www.bharatpublication.com/current-issue.php?jID=33/IJPPS

Overview of Drug-Inducing Hair-Loss[1]

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Received: 02 December 2020; Accepted: 07 January 2021; Published: 10 January 2021

ABSTRACT

Drugs and chemicals can cause a wide range of hair loss, from hardly discernible shedding to complete, irreversible baldness. Agents may operate directly or indirectly on the follicle, although diagnosing mild alopecia can be challenging in either situation. A wide range of medications can disrupt the cycle of hair, resulting in hair loss. Drugs can impact anagen follicles in two ways: (i) by causing an unexpected termination of mitotic activity in rapidly dividing hair matrix cells (anagen effluvium) or (ii) by causing the follicles to enter a state of premature rest [telogen effluvium]. Hair loss normally develops within days to weeks of commencing therapy for anagen effluvium, whereas hair loss appears two to four months after treatment starting for telogen effluvium. Antineoplastic drugs cause acute damage to the rapidly dividing matrix of hair cells, resulting in anagen effluvium, a common side effect. Anticoagulants, retinol (vitamin A) and its derivatives, interferon, and antihyperlipidaemic medications are among the pharmaceuticals that might cause telogen effluvium. Hair loss caused by medications is usually reversible after the medication is stopped. Alopecia's prevalence and severity are determined by the drug used as well as an individual's predisposition genetically. Some medicines cause hair loss in the majority of patients who are given the correct dosages, while others cause hair abnormalities only on rare occasions. Both hirsutism and hypertrichosis may be linked to the use of certain medications. Metyrapone, Corticotrophin (ACTH), Glucocorticoids, danazol, anabolic steroids, and Testosterone are the most prevalent drugs that cause hirsutism. Cyclosporine, minoxidil, and diazoxide are all known to cause hypertrichosis.

Keywords: *hair loss, hair growth, alopecia, hypertrichosis.*

1. INTRODUCTION

In the practice, the identification and management of drug-induced hair and nail abnormalities provides a unique difficulty for the physician. The physician must not only identify the triggering and address the symptoms, but also assess the impact these conditions have on the patients' quality of life. This article examines the clinical identification, pathogenesis, and possible management options for drug-induced hair and nail abnormalities [1].

1.1. Hair Growth Physiology

Alopecia is a disorder that is commonly seen in clinical practice. Medical diseases, nutritional deficits, physical forces, infections, medicines, and other factors can all contribute to alopecia. Drugs are one of the most common causes of alopecia, and anti-mitotic medicines are frequently linked to the condition. Other medicines, including a range of pharmaceuticals, can cause alopecia in rare cases. Substance-induced alopecia is frequently reversible after the offending drug is stopped [1, 2].

Except for the palms and soles of the feet, human skin has a vast number of hair follicles. Hair follicles create two forms of hair after birth, depending on the location of the body: terminal hair and Vellus hair. Vellus hair, which covers the whole body except the soles and palms, is silky, short (typically less than 2cm), and often colourless [1]. The terminal hair is coarse and long. It has a medulla and is

[1] *How to cite the article:* Mahmood M.D., Overview of Drug-Inducing Hair-Loss, *IJPPS*, Jan-Mar 2021, Vol 5, Issue 1, 1-7

generally colored. It is found in the scalp, brows, and eyelashes during birth. All follicles may generate Vellus hair first, followed by terminal hair, or the other way around. Under the influence of sexual hormones, follicles in the axillae and pubes that generate Vellus hair before puberty begin to create terminal hair after puberty [1,3]. Under the influence of androgens, the scalp follicles of people with androgenic alopecia change from terminal to Vellus follicles.

Each hair follicle goes through a cyclic activity with three distinct phases: anagen, catagen, and telogen. The follicle produces hair continually during the anagen phase, which can last anywhere from a few months to several years. The infundibulum, isthmus, and bulb are the three parts of an anagen follicle, with the latter holding the dermal papilla and the hair matrix that generates the hair shaft [1,4]. The majority of the time, normal hair follicles are in anagen. Anagen lasts a variable amount of time in different parts of the body and different people. The length of each hair is determined by the duration of anagen (scalp hair, axillary hair, eyebrows or other hair). The anagen phase lasts 4 to 8 years on an adult's scalp. Anagen follicles are characterized by high levels of mitotic activity, making them particularly vulnerable to toxic events [1].

Catagen is a phase of transition that lasts about two weeks. Hair follicles undergo regressive modifications during catagen, which precede the period of resting. Telogen is the hair cycle's resting phase, which lasts roughly 100 days. The hair remains fixed inside the follicle during telogen by an enlarged base known as the club [1]. Because their metabolic and mitotic activity is uneven, catagen and telogen follicles, unlike anagen follicles, are not responsive to noxious events. The ratio of anagen to telogen hair on a typical scalp is roughly 90:10. The number of scalp hair shed daily varies from 30 and 100 [1,2]. Human hair growth, on the other hand, is seasonal, with hair loss peaking in the late summer.

Only when follicles are actively proliferating are they vulnerable to pathogens. The hair matrix's mitotic activity is so high during the anagen phase that it can be likened to the body's most actively replacing tissues, such as bone marrow and mucous membranes. As a result, the anagen matrix of hair is extremely vulnerable to harmful events, whereas the catagen and telogen follicles of hair are generally safe because their mitotic activity has been halted. As a result, exogenous insults, including medicines, are more severe in body parts where anagen follicles are high in percentages, such as the beard or scalp, than in body parts where anagen follicles are low in percentages, such as the eyelashes and eyebrows. Exogenous assaults cause hair follicles to behave predictably. The severity and duration of exposure to the noxious event, rather than the properties of the agent[s] involved, determine the outcome [1,2,5].

Hair Loss Pathophysiology

A wide range of medications can disrupt the hair cycle, resulting in hair loss. Hair loss caused by medications is usually reversible after the medication is stopped. Alopecia's prevalence and severity are determined by the drug used as well as an individual's predisposition genetically. Some medicines cause loss of hair in the majority of patients who are given the correct dosages, while others cause hair abnormalities only on rare occasions [1,6,7].

Drugs can impact anagen follicles in two ways: (i) by causing an abrupt termination of mitotic activity in quickly dividing matrix of hair cells (anagen effluvium) or (ii) by causing the follicles to enter a state of premature rest (telogen effluvium). Because adjacent follicles are in various stages of the cycle of hair and follicular response to noxious events is strongly linked to anagen sub phase and mitotic activity, anagen and telogen effluvium can occur concurrently on rare occasions [1,2,7,8].

Hair loss happens in anagen effluvium between days to weeks after starting the medication. Alopecia is usually severe and sudden, affecting the majority of the hair on the scalp at the same time. Dystrophic anagen hairs are shown by hair plucking. Although anagen loss is virtually always reversible, it often takes several weeks for regrowth to occur [1,2,7,8]. Because scalp hair only grows about 1 cm each month, the patient should be warned that regaining a full head of hair will take several months. Hair loss appears 2 to 4 months after commencing treatment for telogen effluvium. Depending on the percentage of follicles affected, alopecia may not be evident. Despite this, many report a rise in hair loss. Telogen hair loss is a harmless, transitory disorder that does not require treatment. Chronic telogen loss, on the other hand, may trigger or accelerate androgenic alopecia in genetically susceptible patients if the medicine that causes telogen effluvium cannot be stopped.

In such circumstances, topical minoxidil therapy may be beneficial [9]. Anagen effluvium is a well-known side effect of anticancer drugs that causes abrupt damage to rapidly dividing hair matrix cells [9].

2. DRUG-INDUCED ALOPECIA

2.1. Antineoplastic Agents [10-12]

Loss of Hair is the most prevalent cutaneous side effect of anticancer drugs, with alopecia affecting nearly every patient. Combination chemotherapy is more prevalent and severe than single-drug chemotherapy. The medications' toxic effect on the hair matrix causes either complete hair halting or weakening of the hair shaft, which easily breaks at the base with minor trauma [10-12]. Moderate telogen effluvium is typically accompanied by anagen loss. Alopecia appears 1 to 2 months after the initiation of cytostatic therapy, and hair shedding commences within 7 to 14 days following the first administration of the medications [11]. The severity of alopecia is linked to the medicine used as well as the dosage. Hair loss can be nearly complete in some situations, particularly in people who have undergone numerous cycles of chemotherapy.

Bleomycin, Hydroxycarbamide, daunorubicin, Vincristine, Methotrexate, Cyclophosphamide, Doxorubicin, chlormethine, fluorouracil are the most prevalent drugs that cause alopecia. Hydroxyurea. thiotepa, Chlorambucil, vinblastine, dactinomycin and cytarabine [cytosine arabinoside] are some of the drugs that can worsen alopecia when taken in combination with chemotherapy (actinomycin D) [10-12]. The use of a cooling turban on the scalp for 10 minutes before to 30 minutes after the medicine is administered may help to partially reduce hair loss [10-12].

2.2. Anticoagulant

Anticoagulants of any kind might cause hair loss. heparinoids, indandiones, coumarins, dextran, and Heparin are among them. Telogen effluvium affects around half of all patients, and it appears to be linked to treatment dosage. Women are more likely to experience it [13].

2.3. Antithyroid agent

During the treatment of thyrotoxicosis Iatrogenic hypothyroidism arises is characterized by reversible baldness. It can occur in persons who do not develop iatrogenic hypothyroidism on rare occasions. Hair brittleness and dryness are common symptoms of telogen effluvium. Iodine, thiouracils, and carbimazole are antithyroid medications that can cause telogen effluvium [14].

2.4. Contraceptives used orally

After discontinuing oral contraceptive therapy, telogen hair loss is common for 2 to 3 months afterwards. Increased telogen loss is solely a result of the higher percentage of anagen hair retained during pregnancy, comparable to the pathogenesis of postpartum hair loss. Because estrogens cause the anagen phase to be prolonged, the use of contraceptives containing modest concentrations of estrogens is only sometimes linked to this impact [6, 15, 16].

2.5. Lithium

Loss of Hair is a common side effect of lithium carbonate therapy, and it can appear as early as a few months after commencing treatment. Telogen effluvium, which affects roughly 10% of patients, does not appear to be dose-related [17-21].

2.6. Interferons

Twenty to thirty per cent of patients treated with interferons develop telogen effluvium (IFNs). Human leucocyte IFN, as well as IFNa, IFNa-2a, and recombinant IFNa-2b, have been used to characterize it. There is no link between IFN dosage and the severity or onset loss of hair in our experience [22, 23]. Despite continued treatment, telogen loss diminishes in some patients.

2.7. Retinoid

Although retinol (vitamin A) is regularly given for the treatment of a variety of hair diseases, telogen effluvium is frequently caused by chronic administration of large doses of this vitamin. Diffusely, hair loss can affect the scalp, pubic, axillary, and vellus hair [24-27]. Hair brittleness is also a possibility.

Diffuse hair loss is frequent after etretinate treatment, but only approximately 20% of patients develop noticeable alopecia. It is more common among women who have been given excessive medication dosages. Alopecia can be reversed if the medicine is stopped or the dosage is reduced. Hair kinking has also been reported as a side effect of etretinate administration. Kinking was seen with regrowth after etretinate-induced generalized alopecia in certain individuals [17, 28]. Although alopecia is listed as an uncommon side effect on the isotretinoin product datasheet, no incidences of hair loss have been reported in the literature. After taking four times the authorized amount of isotretinoin, one patient experienced practically total but reversible hair loss [29].

2.8. Antihyperlipidaemic

Drugs Patients receiving trimaran, which has been pulled from the market due to cataract induction, have complained of loss of colour, ichthyosis and hair loss, and. Clofibrate can cause hair loss and brittleness in certain people [30, 31].

2.9. Immunosuppressant

Loss of Hair is a side effect of several immune-suppressing medicines used to treat autoimmune diseases like lupus and rheumatoid arthritis. Methotrexate, leflunomide, cyclophosphamide, and etanercept are a few of them [32-36].

2.10. Mood stabilizers and antidepressants

Loss of Hair is a side effect of some antidepressant and mood stabiliser drugs. This can be caused by a variety of medications, including amitriptyline, fluoxetine, protriptyline, paroxetine hydrochloride, and sertraline [37,38].

2.11. Medications for weight loss

Hair loss is a side effect of some weight-reduction drugs, such as phentermine, but it isn't always mentioned. This is because dieters who lose their hair are frequently malnourished or have underlying health issues that contribute to hair loss. As a result, while some persons taking weight-reduction medicines have complained of hair loss, it could be due to hunger [39, 40].

2.12. Gout medication

Hair loss has been reported with gout drugs like allopurinol [41].

2.13. Medications for Chemotherapy [3, 9, 10, 14, 17, 42-48]

Chemotherapy treatments nearly invariably result in hair loss because they target cancerous cells that divide quickly. Chemotherapy medications shut down actively generating hair follicles by quickly dividing cells in the follicle of hair that makes the shaft of the hair. Anagen effluvium is a kind of loss of hair that is characterized by a large amount of hair loss in a short period. Because approximately 90% of all follicles of hair are actively developing hairs at any given moment, with the remaining 10% resting or passively waiting to shed hairs, chemotherapy medications typically cause near-total loss of hair in a short period. Hair loss usually occurs 2 to 3 weeks after the first treatment of chemotherapy and over the next one to two months progresses. Loss of hair on the pubic area, arms, legs, underarms, and face, is common, and with continued treatment, hair loss on the face, arms, legs, underarms, and pubic area may occur. Hair normally starts to grow back three to four months after the final chemotherapy treatment, but it only grows approximately half an inch each month, so it may take many months to achieve good coverage.

Chemotherapy-induced hair loss has terrible effects psychologically on cancer patients, and numerous devices and medicines have been tried to lessen the degree of hair loss. Another option is to use various cooling devices to chill the scalp to restrict blood flow and limit the effect of the chemotherapy medications. There have also been advancements with treatments that temporarily inhibit hair follicles from growing hair, reducing the chemotherapy drugs' absorption. None of these methods is ideal, and they all have the potential to raise the likelihood of cancer cells living in hair follicles. Fluorouracil, Methotrexate, etoposide, dactinomycin, cytarabine, cyclophosphamide, Bleomycin, daunorubicin and doxorubicin are some of the most common chemotherapy medications that induce hair loss.

2.14. Anabolic Steroids

Synthetic androgens, also known as "male hormones," are anabolic steroids. Anabolic steroids have a history of being used by bodybuilders aiming to increase their muscular bulk, in addition to being prescribed for certain medical issues [25]. Excessive usage of these drugs can induce early baldness in males who have a genetic susceptibility to hair loss. Testosterone is a drug that comes in a variety of forms, including Depo-Testosterone, and methyltestosterone, danazolol, Fluoxymesterone, and stanozolol are some of the other anabolic steroid hormone drugs that can cause loss of hair [6, 49, 50].

2.15. Mercury and Thallium Poisoning

Alopecia is a common sign of thallium poisoning, and it usually appears by the second week following exposure. The eyelashes, scalp, eyebrows lateral elements and the arms and legs are all affected by hair loss. In most cases, epilation is complete after one month [1, 18,19].

Hair loss is a telltale indicator of mercury poisoning. Nail discolouration, weight loss, stomatitis, hearing and sensory loss, and mental disorders are all common symptoms [18, 19].

2.16. Other agents

In isolated case reports, a variety of different medicines have been linked to hair loss [1,2,5, 7,8,18,19].

CONCLUSION

When the link between drug intake and hair loss isn't clear, diagnosing drug-induced hair loss might be tricky. Hair loss may appear several months after starting therapy with some medicines and may resolve spontaneously even if the drug is continued. Before beginning cancer chemotherapy, patients should be aware that they may lose their hair suddenly. Special care, such as the use of soft brushes, light shampoos, and the avoidance of colours, heated curlers, and hair dryers, is recommended to reduce the severity of alopecia.

Financial Support and Sponsorship: Nil

Conflict of Interest: None

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