CLINICAL IMAGE

# Patchy Hair Loss

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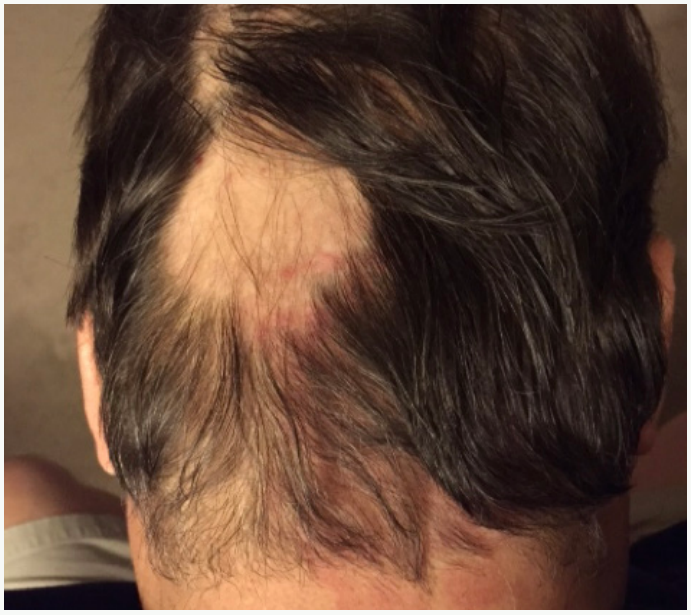
A 64-year-old male presented with a small patch of hair loss on his left occipital scalp. He stated that it started about one-month prior as a small quarter sized patch, which was initially recognized by a barber. There was no scaling, itching, or other associated symptoms. The patch progressively enlarged in size *(Figure 1)* and more patches developed on the scalp. The hair loss stopped after three months with no apparent regrowth. The patient was referred to a dermatologist by his PCP for further evaluation. Prior to these symptoms, the patient had typical male pattern baldness, which he stated runs in the family. Patient denied previous episodes of similar, or other hair loss. He denied pain or pruritis to the area, rash, fevers, chills, or arthralgias.

FIGURE 1:

Hair loss on the left occipital scalp



## QUESTIONS

### 1. What is the most likely diagnosis?

A. Alopecia areata
B. Alopecia neoplastica
C. Lichen planopilaris
D. Tinea capitis
E. Trichotillomania

### 2. Which of the following statements is most correct?

A. Intralesional corticosteroids are usually the first line treatment.
B. This condition is often a side effect of treatment with Janus kinase inhibitors.
C. This condition results from an underlying malignancy.
D. This condition will occasionally respond to behavior modification therapy.
E. Wood's light examination sometimes shows florescence of surrounding hairs.

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## ANSWERS:

### 1. What is the most likely diagnosis?

Correct Answer:
*A) Alopecia areata*

This sharply demarcated patch of non-scarring hair loss is most consistent with a diagnosis of alopecia areata. This may be difficult to distinguish from trichotillomania, which results from habitual pulling of hairs. However, in trichotillomania, patients are often not able to pull all hairs completely, broken hairs are commonly seen, and the distribution is usually in the frontotemporal scalp.[1,2] In tinea capitis, there is inflammation that is usually evident by scaling in some types or frank abscesses (kerion) in others.[3] Scarring, never seen in alopecia areata, is evident in lichen planopilaris, where the follicular orifices are obliterated.[4] Alopecia neoplastica is alopecia secondary to an underlying malignancy, usually metastatic, and is also a scarring process.[5] Lesions may be single or multiple plaques having a red-pink color and may be smooth or uneven and are often associated with telangiectasia.[5]

### 2. Which of the following statements is most correct?

Correct Answer:
*A) Intralesional corticosteroids are usually the first line treatment.*

Unless the alopecia is very extensive or total, intralesional steroids are usually the first option for treating alopecia areata.[6,7] Recent reports have shown promise in the treatment of alopecia areata with oral or topical Janus kinase inhibitors, but they have not been thought to be implicated in triggering the disease.[8] Behavior modification would be a more appropriate approach to the management of trichotillomania but is not likely to be very effective for alopecia areata.[6] Alopecia areata is not a scarring response to a malignancy, although there have been cases of concomitant malignancies such as lymphomas reported in association.[9,10] Wood's light examination would not be expected to reveal fluorescence in alopecia areata but would yield a green fluorescence with a dermatophyte infection with Microsporum canis.[3]

## DISCUSSION

Alopecia areata is estimated to affect 6.8 million people in the United States and has a lifetime incidence of 2.1% worldwide.[11,12] While not a particularly severe health threat, it can be cosmetically disturbing as well as psychologically stressful for the patient.[6] Alopecia areata is a type of non-scarring hair loss which usually involves well circumscribed patches of nearly complete hair loss, but which can be more diffuse involving the entire scalp (alopecia totalis) or the entire scalp and body (alopecia universalis).[6] The affected skin can commonly present with slight redness but otherwise has no other abnormalities. Frequently, "exclamation mark" hairs, or short, broken hairs that are thicker at its damaged section and thinner proximally as it enters the scalp, can be seen around the margins of alopecia areata, specifically where it is expanding.[6,13] Nail changes, such as grid-like nail pitting, can also be seen in patients with alopecia areata.[13] It can be seen in all ages and there is no known prevention, race, or sex dominance and also has a variable occurrence of relapse.[6] A common practice in diagnosis involves the use of dermoscopy, a noninvasive handheld instrument with a transilluminating light source and magnifier, where one can see the presence of "round yellow dots" around the areas of hair loss which implies progression of alopecia areata.[6]

Roughly 20% of people with alopecia areata have a family history of the disease, therefore, it is believed that there is a genetic predisposition to the disease.[14] The direct cause of alopecia is unknown. Theories suggest that there is an autoimmune destruction of hair follicles due to chronic inflammation as evidenced by histopathologic examination, which reveals a dense lymphocytic infiltrate of T lymphocytes around the anagen follicular bulb referred to as a "swarm of bees" appearance.[15,16] There have been multifactorial associations with a variety of genes, including major histocompatibility complex (MHC) and cytokine genes.[14,17] Melanocytes, which are active during the anagen phase, could be responsible for the immune response against the hair follicles by expressing an autoantigen.[18] Since alopecia areata is associated with many autoimmune diseases, such as autoimmune thyroid disease, it is further believed that alopecia areata may be autoimmune in nature.[19]

The differential diagnosis of localized hair loss is vast. Compared to trichotillomania, an impulse-control disorder which can be mistaken for alopecia areata, the broken hairs are firmly attached to the scalp and remain in the growing phase (unlike exclamation mark hairs).[6] Unlike alopecia areata, the scalp in tinea capitis is often inflamed, erythematous, and scaly where hair loss is present.[20] Androgenetic alopecia, commonly known as male or female-pattern baldness, is distinguished by diffuse thinning of hair with either the frontal hairline still intact (female pattern) or an M pattern of hair loss (male) with both displaying a negative pull test away from hair loss.[20] A pull test is used to diagnose hair loss and involves grasping 40-60 hairs at the base using the thumb, index, and middle fingers and gently applying traction away from the scalp while lightly pulling on the hairs.[21] This process is usually repeated in two other areas of the scalp for confirmation.[21] A positive pull test results when more than 10% of hairs (4-6 hairs) are pulled from the scalp, indicating that the hair is actively shedding.[21] Telogen effluvium is a nonscarring and noninflammatory alopecia that presents suddenly but subtly involving hair loss on the entire scalp and primarily affects women ages 30-60 years.[18] Telogen effluvium is most commonly caused by physiologic changes in health status (infection, chronic illness, medication exposure, surgery, pregnancy, hypothyroidism) or even emotional stress that, once removed, hair typically regrows.[18,20,22] Trichorrhexis nodosa occurs when hairs break due to trauma, i.e. traction with tight braids, straightening, or hair product overuse.[20] Lastly, anagen effluvium is a diffuse hair loss caused most commonly by chemotherapy, which disrupts the mitotic activity of hair follicles.[20]

## TREATMENT/PREVENTION

Patients may initially present to the family practitioner once they start to experience hair loss. It is recommended that a dermatologist evaluate more advanced hair loss cases or refractory

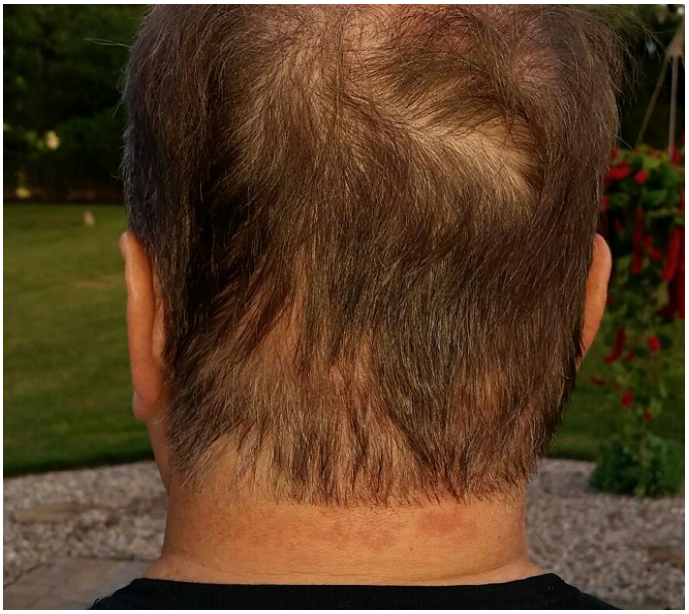
cases when necessary.[23] Since the hair follicles are preserved in nonscarring alopecia areata, recovering hair growth is possible in most cases, even in longstanding disease.[6] A study performed in Japan reported that spontaneous remission within a year was seen in 80% of patients who had small numbers of patches of hair loss.[6] Almost all patients with this disease will experience more than one episode of alopecia areata hair loss and 14-25% of these patients can progress to alopecia totalis or even alopecia universalis where treatment becomes more complex and a full recovery is difficult to achieve (< 10%).[24,25] Furthermore, the prognosis becomes even less favorable when the onset of alopecia areata develops during childhood as well as in ophiasis, or a wave-like presentation of hair loss.[24, 26-28]

The first line treatment for alopecia areata is multiple intralesional injections of corticosteroids every 4-8 weeks in the mid dermis, specifically triamcinolone acetonide (2.5-5 mg/mL) or hydrocortisone acetate (25 mg/mL)(5), which are given at monthly intervals.6,7,29 Studies report an injection of 0.05-0.1 mL will produce an area of regrowth of hair approximately 0.5 cm in diameter.6,7,29 This is most effective when treating patchy patterned hair loss with a limited extension, as well as for areas of cosmetic concern i.e. eyebrows, however, this practice is limited by patient discomfort.6 Needleless devices, such as a Dermajet™ exisit which make it more practical for diverse practitioner utilization.[6] High concentrations of corticosteroids should not be used in order to avoid excess skin atrophy at the sites of injection.[6] Systemic corticosteroids are sometimes given when the alopecia is exceptionally rapid.[6,30] A small study reported 30-47% of patients treated for 6-weeks with oral prednisolone (40 mg daily) showed 25% or more hair regrowth, but continued treatment was needed for maintaining the new growth.[30,31] Topical steroids and calcineurin inhibitors such as oral cyclosporine and topical tacrolimus are sometimes used but tend to be less beneficial as are topical retinoids and anthralin (0.5-1% cream applied daily). [6, 32-39] Reports of successful treatments with topical immunotherapeutic agents dinitrochlorobenzene (DNCB), diphenylcyclopropenone (DPCP), and squaric acid dibutylester (SADBE) have also been published.[6,40,41] Recently, there have been reports of alopecia areata responding to oral and topical Janus kinase inhibitors such as tofacitinib and ruxolitinib but relapse post discontinuation is a concern.[18,42,43] Patients can expect variable response to these treatment options. Comfort and supporting the patient is important during treatment in attempts to eliminate stress. If the patient does not retain hair growth, wigs, plugs, and distracting accessories such as eyeglasses can be recommended in concordance with the patients concern. Support groups (National Alopecia Areata Foundation) are also an option for discussion for patients suffering from psychological trauma from the hair loss.

Our Patient received intralesional triamcinolone acetonide (Kenalog – 10) injections, from 1ml to 2.5ml once a month in the patches of hair loss starting from the front (the most noticeable) then working towards the back. He received about 20 to 30 small injections around a patch each month until the full dose was administered. It took about 3 treatments (or about 3 months) in each patch site before hair started to grow and he received these injections for about a year.

## FIGURE 2:

Regrowth



**AUTHOR DISCLOSURE:**
No relevant financial affiliations

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