International Journal of PharmTech Research
CODEN (USA): IJPRIF, ISSN: 0974-4304, ISSN(Online): 2455-9563
Vol.14, No.01, pp 15-19, 2021

Alopecia totalis in 14 years old boy: A Rare case

Roveline Anissa[1*], Satya Wydya Yenny[2]

[1]Department of Dermatology-Venereology, School of Medicine, Medical Faculty of Andalas University, Padang, West Sumatera, Indonesia
[2]Department of Dermatology-Venereology, Head of Dermatology Cosmetic division, Medical Faculty of Andalas University, Padang, West Sumatera, Indonesia

Abstract : Alopecia totalis (AT) means a total lost of scalp hair. There are some etiologic factors contributing to AT that difficult to know. It is hypothesized to be an organ-specific autoimmune disease with genetic predisposition and an environmental triger. Approximately 0,5 % of the total number of alopecia totalis cases occur in children. This is the third case of AT in children at Dr.M.Djamil hospital Padang since the last five years. A 14 years old boy with alopecia totalis was reported. He complain hair loss accompanied by baldness on the scalp and eyebrow which is increasing since 2 months ago The alopecia was begin on the top of scalp then extending to all over the scalp. On dermatologic state: there was found effluvium, vellus hair, with localized distribution and size in plaquet. On trichoscopy were found black dots, yellow dots, exclamation hair. The problem of this case are the rare case, and treatment for AT. Based on literature, availability of modality therapy and considering young age of patient, he was treated with minoxidil 5 % solution twice a day and topical potent corticosteroid twice a day. After four weeks therapy, there was improvement with new vellus hair.
Keywords : *Alopecia totalis, rare case*

Introduction

Alopecia areata (AA) is a chronic, an autoimmune disease mediated by autoreactive CD8+ Tcells, presenting with patches of nonscarring hair loss, most commonly on the scalp. This disease affects both adults and children. The severity of AA ranges from alopecia focalis (AF), which presents with circular patches of nonscarring hair loss over the scalp, to alopecia totalis (AT), a total loss of scalp hair, and universalis (AU), a total loss of scalp and body hair.[1]

DOI= http://dx.doi.org/10.20902/IJPTR.2021.140102

Alopecia areata is the most common form of alopecia seen in children. Approximately 10% of the total number of cases occur in children. The familial occurance is around 15% but expression of the disorder is variable among different family members. 5 % of patient suffering from alopecia areata develop hair loss of their entire scalp hair (alopecia totalis), and 1 % of patients develop lost of total body hair (alopecia universalis) at some points.[2] Alopecia totalis is rare case, but many literature did not mention the incidences, so the real incidence of AT is not known. In Dr.M.Djamil hospital Padang this is the third case of alopecia totalis since the last five years.

Alopecia totalis (AT) and alopecia universalis (AU) are the two most severe forms of alopecia areata (AA). Alopecia totalis results in the loss of the entire scalp hair and may occur suddenly or follow partial alopecia. Partial alopecia may be observed in other areas of the body as well. Loss of total body hair is called alopecia universalis and may also occur suddenly or follow of long-standing partial alopecia.[3]

The risk a high frequency of a positive family history of alopecia totalis in affected individuals, ranging from 10% up to 42% of cases, and a much higher incidence of a positive family history in early onset alopecia totalis. Many patients report the experiene of major emotional stress prior to the onset of alopecia. A genome-wide association studies recently showed several loci linked to alopecia totalis containing genes controlling both inate and scquired imunity, as well as genes expressed in the hair follicular it self[1,2]

A careful history often such as the underlying cause of alopecia totalis. Crucial factors include the duration and pattern of hair lost, whether the hair is broken orsheed at the roots, and whether shading or thining has increase. The patient diet, medication, reason and past medical condition, and family history of alopecia are other important factors.[4] The disease that can be associated with AT such as thyroid disease, syphilis, iron deficiency, sistemic lupus eritematosus, hereditary syndrome. Beside the condition of androgen excess, trauma also can be predisposing factors in AT. [1,4]

Histologically, alopecia totalis is characterized by an inflamatory infiltrate, comprised many of T-cells, in and around the bulbs of anagen hair folicles (swam of bees). Sometime mast cells, plasma cells and eosinophils can also be seen. However, the classic inflamatory infiltrate maybe missing in subacute or chronic forms. Laboratory test to rule out thyroid disfunction should be performed.[5]

Very little evidence-based data is available for the treatment of alopecia. Recomendation are mainly based on case series and clinical experience. In alopecia totalis and universalis, treatments have a high failure rate.fter the discussion of posible risk and benefit of all options, "no treatment" maybe a legitimate option for some patient below shows and algorithm for treating alopesia areata based on age and scalp involvement.[1]

Minoxidil shows little efficacy in alopecia totalis and universalis. Diphenylcyclopropenone (DPCP) and squaric acid dibutyl ester (SADBE) are the most commonly used contact sensitizers. The DPCP and SADBE are compounded in an acetone base and stored in opaque bottles to protect the solution fro photodegradation. Applying a small amount of a 2% solution to a small scalp or other area (often the arm) on week prior to treatment initiation sensitizes the patient. The DPCP or SADBE solution is then applied weekly to the scalp, starting at a concentration of 0.0001%. The scalp should not be washed for 48h post treatment and should be protected from UV radiation. Every week the concentration is carefully increased until the patient develops a mild erythema and mild itching. The treatment is continued with this concentration; the highest concentration used is 2%. Success rates vary from 17%-75% with the lowest success rates in patient with alopecia totalis and universalis.[1]

Case Report

A 14 year old boy with hair loss accompanied by baldness on the scalp and eyebrow which is increasing since 2 months ago. The alopecia was begin on the top of scalp then extending to all over the scalp. The hair scalp was starting to loss since 8 months ago and became baldness since 2 months ago. Physical examination from dermatologic states were found effluvium in all of hair scalp and eyebrow, vellus hair, no sicatrics (**Figure.1**). Results of laboratory examinations including complete blood count, serum chemistries, ferritine and iron total blood examination, testosterone hormone examination, zinc serum level, and vitamin D serum level, and potassium hiroxide examination, were normal. Trichoscopy examination were found exclamation mark hair, black dots, yellow dots, and broken hairs. The patient was treated with minoxidil

solution 5 %, 40 mg, topical corticosteroids clobetasole propionate cream 0,05 % on the scalp. There was improvement after four weeks therapy with new vellus hair.

Figure 1. Alopecia from the front hairline and then on the top of scalp then extending to all over the scalpwithout sicatric and atrophic scar.

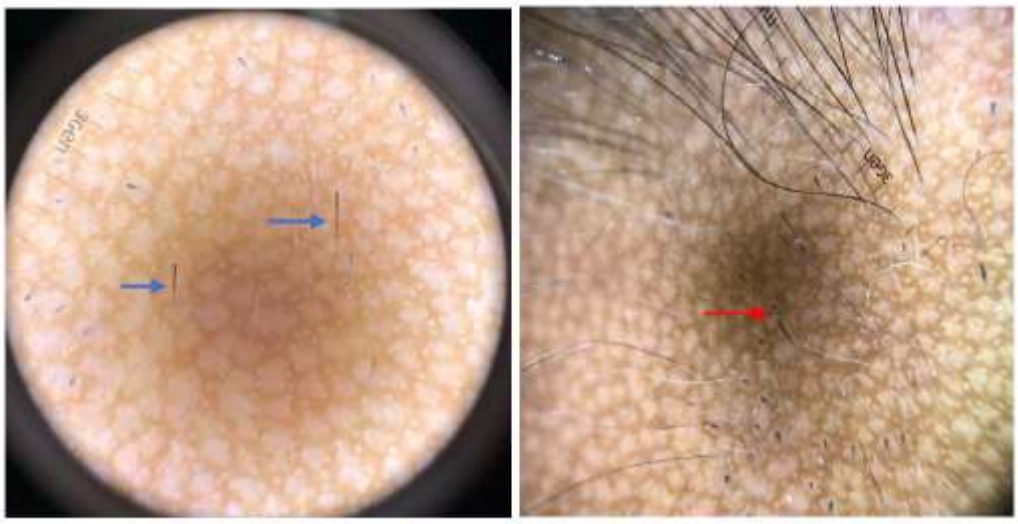

Figure 2. Trichoscopy feature A. Exclamation point hair B.Broken hair(red arrow), Yellow dot (yellow arrow),

Figure 3. After four weeks got a treatment with minoxidil 5 % and clobetasole propionate cream 0,05% there were vellus hair.

Discussion

A rare case of alopecia totalis in 14 year old boy was reported**.** The male to female ratio of alopecia totalis disease was 1:2. Approximately 10% of the total number of alopecia totalis cases occur in children. In Dr. M Djamil Hospital Padang, this is the 3rd case in 5 year.

Diagnosis of alopecia totalis was made based on anamnesis, physical examination and laboratory findings. On anamnesis and physical examination, we had found that baldness on hair scalp, eyebrow since 8 months ago. Based on literature, if there is hairloss in all of hair scalp we can diagnose with alopecia totalis.

Androgenetic alopecia, or hair loss mediated by the presence of androgen dyhidrotestosterone, is the most common form of alopecia in men and women. Almost all persons have some degree of androgenetic alopecia. The hair loss usually begins between the ages of 12 and 40 years and is frequently in suficient to be noticed. If the diagnosis is not clear based on the history and physical examination, selected laboratory test, and occasionally, punch biopsy may be indicated.[4,5]

The percentage of children with alopecia totalis and positive family history in the literature ranges from 8.4% to 51.6%. AA sinha et al (India,2012) reported one hundred and sixty-nine probands (32.9%; n = 169) reported a family history of alopecia totalis. Overall, this included a total of 231 relatives, including 124 (53.7%) first-degree (parents, siblings and children), 67 (29%) second-degree (grandparents, aunts/uncles, nieces/nephews and grandchildren) and 40 (17.3%) third-degree (including first cousins, great grandparents and great aunts/uncles).[6] In this case, were found familial history from patient's grandfather with baldness on scalp.

On trichoscopic evaluation, "exclamation-point" in which the proximal hair shaft has thinned but the distal portion remains of normal caliber hairs, black dots and yellow dots are found in alopecia totalis. In this case, from trichoscopic examination were found exclamation hair, black dots and also yellow dots.[7]

Routine screening for autoimmune disease (thyroid disease in particular) is not generally indicated because of insufficient clinical evidence. Older patients, patients with long disease duration, males, patients with persitent patchy AA (as compared to transient patchy AA), and patients with AT/AU have been found to more likely have thyroid abnormalities.[2] In this case we did'nt find sign and symptom of thyroid disease. But based on literature, AT condition and male patient we still did thyroid function examination due to possibility silent thyroid disease, and to confirm if there is any increasing of T3, T4 and TSH, and the result of thyroid function examination we found all in normal limit. [8] So, one of predisposing factor for AT in ths patient could be excluded.

Potassium hydroxide, fungal culture, lupus serology, syphilitic screenig, and a scalp biopsy may be necessary in ambiguous or difficult to diagnose cases.However, most presentations of AA are obvious, and further laboratory tests are not indicated in the majority of cases.[9]In this patient, we have done potassium hydroxide examination and there was no hyphae and groups of spores found. Patients were given 1x500 mg griseofulvin tablet, and cream ketokonazole twice daily for 2 months by the dermatovenereologist before. However, hair loss does not decrease.Therefor, we can excluded fungal infection of scalp in this patient. Syphilitic screening was not examined also in this patient because there was no sign and symptom from anamnesis and he never did sexual contact with anybody. So, the possibility of getting syphilis could be excluded.

About lupus serology, we had thought about this possibility, but after did anamnesis and physical examinaion, we thought that this patient did'nt suffer lupus disease. We consulted this patient to pediatric departement to find any systemic disease that correlated with AT. The pediatric department made working diagnoses with alopecia areata et. Causa suspect autoimune disease. There was suggestion to make sure the diagnosis of SLE. From pediatric department planned to check the ANA test to rule out SLE or other autoimmune disease that can be correlated with his baldness, and the result was in normal limit. However we didnt see SLE criterias in this patient (malar rash, discoid rash, photosensitivity, oral ulcers, arthritis, serositis, renal disorders, neurologic dsorders, hematologic dsiorders). Another lupus disease that could cause hair loss is discoid lupus erythematosus (DLE). The scalp is involved in 60% of patients with DLE, irreversible, scarring alopecia resulting from such involvement has been reported in one-third patient. The irreversible, scarring alopecia resulting from DLE differs from the reversible, nonscarring alopecia thet patients with SLE often

develop during periods of systemic disesase activity. This type of hair loss, so-called lupus hair, may be telogen effluvium occuring as the result of flaring systemic disease. In this patient, the type of alopecia is nonscarring.[10] So we can excluded the possibility of SLE and DLE in this patient.

Scalp biopsy to this patient was not perfomed because the result histopatological examination also cannot support the diagnosis. The other etiologic factor that could be happened in this patient such as nutritional deficiency (malnutrition, iron, and zinc deficiency). But from the physical examination, he has good nutrition with normoweight and good appetide. There was no sign and symptom that he has acquired zinc deficiency that may result from inadequate intake, impaired absortion or increased excretion including pregnancy, lactation, extensive cutaneous burns, generalized exfoliative dernatoses, food faddism, parenteral nutrition anorexia nervosa and even excessive sweating.[11]

Conclusion:

A rare case of alopecia totalis in 14 year old boy was reported.The patient was treated with topical minoxidil 5% solution and clobetasol propionate 0,05 % ointment. After 1 month therapy there was improvement with new vellus hair.

References

1. Otberg N, Shapiro J. Hair growth disorders. In: Goldsmith LA.Katz SI, Gilchrest BA, Paller AS, Fitzpatrick's dermatology in general medicine edisi 8. New York: Mc Graw-Hill; 2012: 979-1008.
2. Safavi K. Prevalence of alopecia areata in the First National Health and Nutrition Examination Survey. Arch Dermatol 1992; 128(5): 702.
3. Mirzoyev SA, Schrum AG, Davis MDP, Torgerson RR. Lifetime incidence risk of alopecia areata estimated at 2.1% by Rochester Epidemiology Project, 1990-2009. J Invest Dermatol 2014; 134(4): 1141–1142.
4. Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in North- ern India. Int J Dermatol 1996; 35(1): 22–27
5. Shapiro J. Dermatologic therapy: Alopecia areata update. Dermatol Ther,2011; 24(3): 301.
6. Lacarrubba F, Micali G, Tosti A. Scalp dermoscopy or trichoscopy. Curr Probl Dermatol 2015; 47: 21–32.
7. Rudnicka L, Olszewska M, Rakowska A, Kowalska-Oledzka E, Slowinska M. Trichoscopy: a new method for diagnosing hair loss. J Drugs Dermatol 2008; 7(7): 651–654.
8. Tosti A, Duque-Estrada B. Dermoscopy in hair disorders. J Egypt Womens Dermatol Soc 2010; 7: 1–4.
9. Lacarrubba F, Dall'Oglio F, Rita Nasca M, Micali G. Videoder- matoscopy enhances diagnostic capability in some forms of hair loss. Am J Clin Dermatol 2004; 5(3): 205–208.
10. El-Ashmawy A, Khedr A. Some trace elements' level in alopecia areata. Egyptian Dermatol Online J 2013; 9: 6.
11. Alkhalifah A, Alsantali A, Wang E, mCeLWEE KJ, Shapiro J. Alopecia areata update.part II treatment.J Am Acad Dermatol 2010;62:191-202

*****