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CAUSES OF DIFFUSE ALOPECIA IN WOMEN

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Fifty female patients ranging in age between 14-45 years, complaining of a diffuse loss of hair for periods varying from 1-22 months were thoroughly interrogated to look for the factors leading to the hair loss. Laboratory investigations undertaken included a routine estimation of haemoglobin, ESR, urinalysis, examination of the stools, and any other test indicated by the history or examination. The hair loss was quantitated by asking the patient to collect all the fallen hairs during a 24 hours period for 7 consecutive days, and pack them into appropriately labelled polythene bags. Each collection was weighed to calculate the average daily loss. Such collections were repeated once a month for a period of 4-5 months to monitor the variations in the hair loss and study the effect of treatment. Ten hairs randomly picked from each collection were examined microscopically to look for any shaft abnormalities in addition to the examination of the root end and the distal end. The probable causes of hair loss in these patients were found to be psychologic stress in 21 cases, fever in 11 cases, child-birth/abortion in 5 cases, and a surgical operation in 3 cases. Some of these cases had more than one factor, while in 15 cases there was no clinical evidence of any of the known causes of hair loss. Routine laboratory investigations however revealed a haemoglobin level of less than 12 gm % in 25 cases, an ESR higher than 20 mm in 18 cases, asymptomatic urinary tract infection in 13 cases and gastro-intestinal parasites in stools in 12 cases. Thus every patient had at least one of the causative factors, while several patients had more than one factor which could be responsible for the hair loss. Almost all the cases in this group seemed to have telogen effluvium. Examination of the root end in most of the hairs showed a club hair, while the hair shaft revealed trichorrhexis in some of the hairs in 10 cases and trichoptilosis in 3 cases. Appropriate treatment normalised the hair loss which was reflected in the collections of the fallen hair.

Key Words : Hair loss, Causes, Assessment

Introduction

Hair is a marvellous structure designed to perform a variety of functions in different regions of the body.[1] These include conservation of heat (body hair), prevention of friction between two skin surfaces (axillary hair), filtration of the in-coming air (nostrils, eye-lashes and moustache), mechanical protection of vital organs (scalp hair and beard), and sexual arousal and tactile function (pubic and general body hair). In the humans however, the hair has no vital function,[2] the only function which the hair perform is the cosmetic function.[3] Every one therefore, wishes to have the perfect amount of hair at the right places, and hair loss thus is one of the most common complaints in the dermatologic patients.

Most normal individuals are expected to lose 50-100 hairs from the scalp every day,[1-4] though the exact number of hairs lost per day varies from day to day. This is the physiologic hair loss and is confined to the hairs which have completed their telogen phase. It generally remains unnoticed except in the individuals who keep long hair. During summer and rainy season however, most Indians even normally, lose a larger number of hair[3] which seems to be akin to the seasonal loss of hair in some animals.

An excessive loss of hair (diffuse alopecia) can occur due to a variety of causes.

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There have been a few studies[5,6] carried out to enlist the causes of diffuse hair loss but none among the Indians. We are reporting the results of a study undertaken to analyse the causes in our patients.

Materials and Methods

Fifty female patients having diffuse hair loss were included in the study. Each case was thoroughly interrogated to look for the history of febrile episodes, child-birth/abortion, accidents/physical trauma/surgical operations, nutritional deficiencies/crash dieting, psychological stress, intake of drugs, and any systemic or dermatologic diseases which could be considered responsible for the hair loss. The history was extended to cover the entire duration of the hair loss as well as a period of at least 3 months before the onset of the hair loss. In addition, all patients were subjected to routine laboratory investigations which included estimation of haemoglobin, ESR, urinalysis and examination of the stools for parasites. Patinets showing clinical evidence of an endocrine abnormality were evaluated for the endocrine functions.

The loss of hair was quantitated by the method described by one of us (JSP). This method consists of asking the patients to collect all the hairs which fall during the 24-hours period for 7 consecutive days and pack them into small polythene bags labelled with the particulars of the patient and the date of collection. Such collections are repeated at approximately 1 month intervals during the follow up. The mass of hair collected each day is weighed to calculate the average daily loss of hair during the week. These values are used to evaluate objectively the variations in the hair loss during the follow up. The hair collections in this study were continued for a period of 4-5 months or till the hair loss seemed to have become normal.

Ten hairs randomly selected from each of the collections were also examined microscopically from one end to the other to look for any structural abnormalities of the shaft in addition to examination of the root end and the distal end of the hair.

All the patients were advised to wash their hair with a simple shampoo and avoid applying any hair oil, hair dye or other physical or chemical cosmetic procedures. Patients found to have some concomitant disease were given appropriate treatment, including the treatment for anaemia, gastro-intestinal infestation or the systemic infection.

Result

Of the 50 female patients included in the study, 13 patients were less than 20 years in age, 35 patients were between 20-40 years while two were more than 40 years, the age range being 14-45 years. The duration of hair loss had been less than 6 months in 25 cases, 6-12 months in 22 patients and more than 12 months in 3; the minimum duration being 1 month and the maximum 22 months.

The probable aetiologic factors as recorded from the history were : (1) Psychologic stress in 21 cases which included prolonged marital difficulties (10 patients), tension due to the examination or work at office (4 cases), a hyperirritable personality (1 case), depression (1 case), financial problems (1 case), tense relationship with a parent (1 case), anxiety about the future of her only son (1 case), tension having to look-after a mentally retarded sister-in-law (1case), and safety of the relative in Afghanistan during the war (1 case); (2) Fever in 11 cases which included viral fever (6 cases), malaria (2 cases), typhoid (2 cases), and pyrexia of unknown origin (1 case), (3) Child-birth (4 cases) and abortion (1 case); and (4) Surgical operation in

3 patients, the operations being appendicectomy, skin grafting and lower segment caesarian section in one case each. While some of the patients had more than one factor which could be responsible for the hair loss, in 15 patients there was no clinical evidence of any of the factors known to cause diffuse alopecia. The routine laboratory investigations revealed a haemoglobin level of less than 12 gm% in 25 cases, a raised ESR (higher than 20 mm 1st hour) in 18 cases, asymptomatic urinary tract infection in 13 cases, and gastro-intestinal infection in 12 cases, the parasites being *Entamoeba coli* in 6 cases, *Giardia lamblia* in 4, and *Entamoeba histolytica* and *Ascaris lumbricoides* in 1 case each.

Thus in the final analysis, even if *Entamoeba coli* and *Giardia lamblia* are considered to be non-pathogenic commensals, each one of the patients had at least one of the factors which could be considered responsible for the hair loss. In 25 patients, only one factor was considered to be responsible, and these were, psychological stress in 8 patients (3 of whom also had a raised ESR), raised ESR without any detectable cause in 4 patients (one each of whom had *Entamoeba coli* and *Giardia lamblia* respectively in their stools), asymptomatic urinary tract infection in 3 cases (two of whom also had *Entamoeba coli* in their stools), and gastro-intestinal infestation with *Entamoeba histolytica* in 1 case. In the remaining 25 patients, there was evidence for more than one of the causative factors responsible for the hair loss. None of the patients in this group was taking treatment with drugs known to cause diffuse alopecia and there was no clinical evidence of any of the endocrinopathies. A family history of androgenetic alopecia in the male members was available in 26 patients, but in only 2 patients, the pattern of alopecia seemed to be of the androgenetic type. The levels of androgens in both these patients were within the normal range.

Microscopic examination of the root end revealed club hairs (Telogen phase) in 40 cases, while 10 cases had catagen hair roots as well. Anagen hair roots were not seen in any case. the distal end of the hair had been trimmed with scissors in 34 patients, while 14

Table I. Correlation of the variations in the hair loss with the different aetiologic factor(s) in selected representative cases.

S. No.	The probable clinical cause	Time interval in months between the onset of the cause and		Association abnormality in the laboratory investigation	Average weight of the hair lost per day (mg)				
		Onset of hair loss	First hair collection		1st mth.	2nd mth.	3rd mth.	4th mth.	5th mth.
1	Malaria	3	16	↓ Hb, UTI	1645	754	521	354	--
2.	Child-birth	2	7	↓ Hb, UTI	520	436	335	166	--
3.	Surgery	3	9	↓ Hb, ↑ ESR	151	82	54	--	--
4.	Psychologic stress	2	6	Nil	186	326	159	108	--
5.	Psychologic stress	3	9	SI	603	554	1145	554	957
6.	Nil	?	?	↑ ESR, UTI	475	454	238	121	--
7.	Nil	?	?	↓ Hb	524	494	184	69	--

↑ ESR = Erythrocyte sedimentation rate more than 20 mm 1st hour Westergren.
↓ Hb = Haemoglobin level less than 12 gm%.
UTI = Asymptomatic urinary tract infection discovered on urinalysis.
SI = Asymptomatic infestation in the stools.

cases had normal tapering ends and 2 patients had terminal splitting. The shaft of the hair revealed trichorrhexis nodosa 1-2 cm from the hair root in 10 cases and trichoptilosis in 3.

The amount of hair lost during the consecutive 7 days showed day to day variations, more hair being lost on the day the hair were washed, and the 7-day collection at monthly intervals reflected the trend of the hair loss. The patients showed perceptible changes visually as well as by weight (Table I) as the hair loss decreased or increased.

Following treatment of the appropriate cause, the recovery from excessive hair loss occurred within 2-3 months in all the patients having urinary tract infection, within 1-2 months in 4 of the 5 patients having anaemia, in 2-4 months in patients having gastro-intestinal infestation with *Entamoeba histolytica*. In contrast in 2 of the 3 patients having fever, and 2 out of 8 patients having psychological stress, the hair loss actually increased during the observation period before it finally decreased to the normal level, in 2 other patients having psychological stress the hair loss continued to increase, while in another 2 patients the reduction in the hair loss was slow and insignificant.

Comments

Diffuse alopecia can be broadly classified into three categories, (1) Androgenetic alopecia, (2) Telogen effluvium, and (3) Anagen defluvium. Androgenetic alopecia[1-4,6,7] occurs under the influence of androgenic hormones in genetically predisposed individuals and worsens as the age advances. It is very common among the males, but the females who have inherited the abnormal gene can also manifest androgenetic alopecia if they have an excess of the androgenic hormones or there is increased susceptibility of the hair follicles to normal levels of androgens, though this type of alopecia in the females is never as severe as in the males. Since androgenetic alopecia can only be prevented, it is necessary to obtain a detailed history of androgenetic alopecia in all the male members of the family both on the paternal as well as the maternal side and try to assess if this could be solely or partly responsible for the hair loss. Unfortunately there are no parameters to decide if the hair loss is androgenetic except that the hair loss is confined to the vertex and the frontal areas and it is almost continuous and progressive. Family history of androgenetic alopecia is usually positive in a very high percentage of the cases, as in our study as well and it may seem irrational to start treatment with anti-androgens in all the cases having a positive family history. On the other hand if one waits for a clinically confirmed diagnosis of androgenetic alopecia before starting the treatment it may already be too late because in that case it may be possible only to stop further progression of the disease or just slow down the process. A compromised approach therefore may be to exclude the other causes of diffuse hair loss, and if there is a suggestive family history of androgenetic alopecia, to give the benefit of doubt to the patient and start the treatment.

The term telogen effluvium was coined by Kligman[8] to describe an excessive loss of hair that occus under the influence of a variety of factors[9,10] all of which induce a large number of hair follicles prematurely into the catagen phase followed by the telogen phase. The hair is lost at the completion of the telogen phase when the new hair has already started growing in its place, the hairs would reveal either telogen (club) hairs or catagen hairs. The factors which lead to telogen effluvium include, (1) a febrile epiosode[1,3,4,11] such as typhoid, dengue, viral fever, malaria,

tuberculosis or fever due to any cause, more so if the fever is high, prolonged or recurrent, (2) an accident, severe blood loss[4,11] or a major surgical operation,[3,4,11,12] (3) child-birth,[1-5,11] abortion or cessation of oral contraceptives[1,3,11,13,14] or hormonal therapy, (4) nutritional deficiency,[11] anaemia[1,5] or crash dieting,[4,11] (5) severe or chronic systemic illness,[3,4,11,15] (6) psychiatric stress,[1,3,4,10,11,16] (7) endocrine abnormalities especially hypothyroidism,[1,4,5,10,11,17,18] hyperthyroidism,[4,5,13] or the use of antithyroid drugs,[14,19] (8) treatment with certain drugs especially anticoagulants[1,3,4,10,11,14,19] such as heparin and coumarins, hypocholesterolaemic drugs[14,19] such as triparanol and clofibrate; antihypertensive drugs such as propranolol,[4,14,19] amphetamine for weight reduction,[5,11,14] lithium,[14,20] danazol,[21] high doses of vitamin A,[4,19] and less commonly other drugs.[14,19]

Some of these factors such as viral fevers, typhoid, malaria, child-birth, surgical operation etc are short-lived and thus the hair loss due to any of these factors is expected to be brief and self limited. Such cases generally required no treatment because by the time the patient reports for consultation, the causative factor is no more operative. It is generally sufficient to ascertain the cause and reassure the patient that the hair will regrow in due course. It is however essential to ensure that the patient stops worrying about the hair loss because that worry can itself become the cause for further hair loss. It helps to instruct the patients to repeat the phrase "The new hair must have started growing in its place", whenever she notices the fallen hair.

Some other causes of telogen effluvium such as chronic systemic diseases like tuberculosis, SLE, hypothyroidism, anaemia or prolonged psychological stress on the other hand would not stop till the cause is removed. Even then the hair loss can continue for as long as three months after the causative factor has been removed because the duration of the telogen phase is 3 months and the hair is lost only after the completion of the telogen phase. In case there is recurrence of the causative factor during this period, the hair loss can increase again and /or the duration of the hair loss can get prolonged.

The term anagen defluvium[2,9,11] is applied to the situation where the causative influence slows down or stops the growth of the hair while it is in the anagen phase. The hair in such cases is lost due to weakening and breakage of the hair shaft.[9] The common causes of anagen defluvium include, radiation therapy,[4,9,11] treatment with antimitotic drugs[3,4,9,11,14,19] such as cyclophosphamide, methotrexate,[14,19] azathioprine, adriamycin,[14] vincristine,[14,19] hydroxyurea[14] and colchicine,[11,14,19] oral retinoids[11,14] and poisoning with thallium,[4,11,14,19,22] arsenic,[4,11] or gold.[11,14]

Almost all our cases belonged to the category of telogen effluvium, though a small group of cases as ours cannot be considered a true representative of the relative incidence of various factors causing diffuse hair loss. A relatively large proportion of cases who are either mild and/or recover spontaneously or those who belong to the specialised groups such as those caused by anti-mitotic drugs are aware of the cause and do not seek medical advice. Nevertheless, an important finding in our study was that a high proportion of our patients had more than one factor responsible for the hair loss and some of these factors such as anaemia, asymptomatic infections or gastro-intestinal infestations could have been missed if we had not undertaken investigations in all the cases. In cases where there are more

than one causative factor, treating only one of them will not suffice. We therefore recommend a detailed history, a thorough examination and appropriate laboratory investigations in every case to list out all the probable causative factors and treat each one of them in an appropriate manner.

The loss of hair in a female being associated with a lot of sentiment, it is essential to base one's evaluation on objective criteria rather than the patient's version. The method of collecting the fallen hairs is fairly objective and useful even though it is applicable only in those individuals who do not trim their hair too short. Each patient has to act her own control because the mass of the hair collected or its weight will be proportional to the length of the hair which a person keeps and this is so variable in different individuals.

A proportion of the hair loss can also be due to structural abnormalities of the hair shaft.[23] A microscopic examination of the hair shaft is thus useful to complete the evaluation of the patient.

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