

Hair Follicles of Anterior Parietal Region are More Vulnerable than Occipital Region in Classic Acute Telogen Effluvium

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Background: The well known clinical feature of the classic acute telogen effluvium is diffuse hair loss from all over the scalp and the diagnosis is confirmed by a trichogram showing an increased telogen count. While the telogen hair shedding continues, newly cycled back young anagen hairs develop in the involved scalp.

Objective: To see if there is a regional difference in the numbers of the involved hair follicles in that seemingly diffuse hair loss.

Methods: In 7 cases of the telogen effluvium, the telogen count was done at two sites, anterior parietal and occipital. During the count, the short tapered anagen hairs(<1cm) which developed during the course of the telogen effluvium were counted together and compared according to the regions.

Results: The sum of the short anagen hairs and the telogen hairs was 50.3% in anterior parietal area, whereas it was 31.6% in occipital area($p<0.01$).

Conclusion: Hair follicles in anterior scalp appear to be more vulnerable than occipital scalp in the acute telogen effluvium. (Ann Dermatol 15(4) 144~148, 2003).

Key Words : Classic acute telogen effluvium. Regional difference

Classic acute telogen effluvium is caused by premature entering of excessive numbers of anagen follicles to telogen follicles by a causative stress. The well known clinical feature of the disease is diffuse hair loss, which are all club hairs, from all over the scalp with noticeable onset. The excessive hair loss resolves within 3 - 6 months and the

hair density returns to normal in almost all cases. With all of these characteristics the differential diagnosis from other non-scarring alopecia is not difficult. Including its original paper, most of the publications about the telogen effluvium has described it as a diffuse hair loss "throughout the scalp"¹, or "all over the scalp"². The diagnosis is confirmed by a trichogram showing an excessive number of telogen count. The telogen values of the patients may be greater than 50%¹ but usually between 15 and 30%³.

For the trichogram in suspected cases of telogen effluvium, there is no standard that a specific scalp region or a specific time should be selected. It is usually made once on frontoparietal area at the time of examination but the telogen effluvium is a process occurring through several months, during which time a lot of telogen follicles have already cycled back to anagen stage. If the cycled back new anagen hairs are counted together with the telogen hairs, a

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Table 1. Regional trichogram of 7 cases of telogen effluvium

Pts	Sex	Age	Cause	site	①Anagen hairs <1cm(%)	②Telogen hairs(%)	Total telogen hairs from the onset to the time of examination (%) (①+②)
1	F	18	Fever	P	29.0	19.0	48.0
				O	9.0	23.0	32.0
2	F	26	Postpartum	P	62.0	6.0	68.0
				O	3.0	24.0	27.0
3	F	14	Fever	P	15.0	11.0	26.0
				O	3.0	28.0	31.0
4	F	38	Drug	P	3.0	40.0	43.0
				O	0.0	22.0	22.0
5	F	33	Weight loss	P	90.5	2.4	92.9
				O	27.5	52.5	80.0
6	F	39	Postpartum	P	3.6	39.3	42.9
				O	0.0	3.0	3.0
7	F	28	Postpartum	P	8.0	23.0	31.0
				O	0.0	27.0	27.0
Average				P	30.2	20.1	*50.3
				O	6.0	25.6	*31.6

P : (anterior) parietal, O : occipital

* The difference between parietal and occipital : $p < 0.01$

better calculation could be made about how many follicles have actually been involved during the telogen effluvium.

By adding the newly developed extra anagen hairs in usual telogen count and by comparing the result between parietal area and occipital area, we wanted to know if the telogen effluvium is strictly diffuse over the scalp. The telogen effluvium in this study is acute self-limited classic telogen effluvium first described by Kligman in 1961¹. The recently discussed chronic telogen effluvium was not included⁴.

PATIENTS AND METHODS

14 cases(13 females and one male) of classic acute telogen effluvium were examined for their acute hair shedding. There were 6 cases of post febrile, 5 cases of postpartum, 1 case of crash diet, and 2 cases of probable drug reactions. On average, the duration between the causative stress and the onset of effluvium was about 2.5 months and the interval between the onset of the effluvium and

the examination of the hair shedding was about 1.5 months. The hair shedding lasted about 2.5 months after the beginning of excessive hair shedding. All of the patients did not have any clinical history of previous hair thinning before the development of the present illness and the hairs shed were all normal club hairs. By the overall clinical findings of the patients, the diagnosis of acute classic telogen effluvium was evident. Among the 14 patients, in 7 cases(2 postfebrile, 3 postpartum, 1 crash diet, and 1 drug reaction), all females, trichogram was permitted from two sites, anterior parietal area and occipital area respectively. During the trichogram we used a magnifying glass and a hemostat not covered with rubber tube, by which very short tapered hairs with a few millimeters long could be extracted. We found the hemostat covered with rubber tube, which is recommended for usual trichogram, was not good to take very short hairs. About 50 hairs were examined in each trichogram. During the trichogram the tapered new anagen hairs shorter than 1cm were calculated separately.

RESULTS

The trichogram showed as follows (Table 1): The average telogen count in anterior parietal area was 20.1%. It was 25.6% in occipital area. The average count of the tapered new anagen hairs shorter than 1cm was 30.2% in anterior parietal area, whereas it was 6.0% in occipital area. The sum of the short anagen hairs and the telogen hairs was 50.3% in anterior parietal area, whereas it was 31.6% in occipital area ($p < 0.01$).

DISCUSSION

Although it was already known that some cases of postpartum telogen effluvium showed prominent bitemporal or frontal hair thinning², it has been well accepted that the characteristic clinical feature of classic acute telogen effluvium including the postpartum effluvium is a diffuse hair loss from all over the scalp. But, to our knowledge, the degree of the hair loss in the acute telogen effluvium has not been strictly compared by the regions of the scalp.

Before the discussion on the result of the trichogram of the patients, it would be necessary to see normal regional trichogram and the incidence of the new short anagen hairs in normal scalp. There are several reports on the regional trichogram in normal scalp. According to those reports there is no significant difference between parietal and occipital areas in normal females. The different telogen counts found in males according to the regions were also in the range between 15 and 20%^{3,5,6}.

Human hair follicles cycle continuously in a mosaic pattern and, therefore, should always be different lengths' anagen hairs in human scalp. The length of anagen period and the number of daily hairs shed are related to each other. In dermatological literatures there are different figures about length of normal anagen period and number of normal daily hairs shed. We observed that in 90% of normal adult men in the age group of 3rd and 4th decades the number of daily hairs shed was less than 60⁷. Kligman himself found that daily telogen loss in white adult women was 47 and in white adult men 44 in the same age group¹. Stroud also mentioned that normal scalp hair loss more than 50 hairs/day when it is not shampooed is unusual⁸. With all these figures shown above, we thought it would be acceptable that the normal

daily shed is about 50, at least in young and middle age group.

It has been generally assumed that the number of total scalp hairs is about 100,000. Accordingly the average longevity of the hairs in days would be 2,000 days (100,000/50) or about 5 years. Actually we often see adult women who grow hairs longer than 60 cm down to the mid back, which indicate the long hairs have grown for about 5 years (normal hair growing rate 1cm/a month $\times 12 \times 5$). With the norms of the anagen period (longevity) and hair growing rate, we can figure out that the hairs shorter than 1 cm should comprise only about 1.7% of total hairs in normal scalp. Even though the number of the scalp hairs is generally regarded as about 100,000 in dermatological literatures, they say the number may be about 60,000 with about 30 daily shed hairs in Korean. Even if the Korean figures are dependable and we should follow the Korean figures because the present patients are Koreans, the hairs shorter than 1cm also should be only 1.7% of the total scalp hairs as well.

Considering the rate of hair growing, 1cm/ a month, and the time of trichogram in the patients, 1.5 months in average after the beginning of the excessive hair shedding, it would be safe that anagen hairs shorter than 1cm with tapered distal tip are regarded as the hairs newly cycled back into anagen growth phase after the initiation of the telogen effluvium. In other words they are the follicles involved by the telogen effluvium.

In our observation of the present cases, the short tapered anagen hairs (<1cm) in anterior parietal area comprised 30.2% of the total sample hairs in their trichogram and 6.0% in occipital area. Those great numbers of the short tapered anagen hairs sprouted during the course of the telogen effluvium are undoubtedly the hairs emerging from the anagen follicles cycled back from the excessive telogen follicles during the telogen effluvium. In anterior parietal area it was 18 times greater than the normally expected incidence and in occipital area about 4 times greater. In short, although the telogen effluvium involves both anterior parietal and occipital area, there is a definite difference between the two areas in the degree of the involvement.

The hair loss due to non-scarring systemic influence on hair follicles is conveniently described as either diffuse or patterned. The well known example of the patterned one is the common baldness in

Sensory innervation of the human scalp

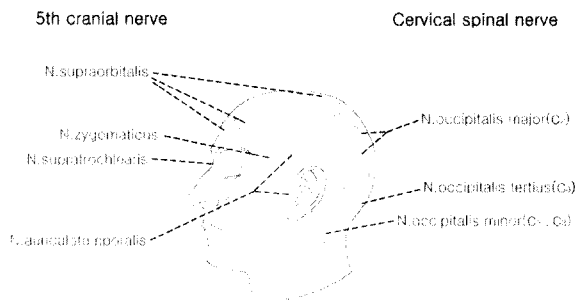


Fig. 1. Sensory Innervation of a human scalp. It shows a close regional overlap between the different sensory innervation and the different follicular behavior.

men in which the pattern is showing a prominent frontoparietal hair thinning (male pattern). In common baldness in women, though still also called diffuse alopecia in women, it has been increasingly noted that there is also recognizable frontoparietal accentuation⁹. As described in the part of Patients and Methods, the possibility of the androgenetic alopecia could be ruled out in the present cases by the apparent clinical findings.

Although the classic acute telogen effluvium is well known as an example of diffuse hair loss, we have found in the present study that the frontoparietal region shows more follicular involvement than the occipital region. Considering the involved area of the androgenetic alopecia with the result of the present study together, the frontoparietal hair follicles seem to have a common inherent vulnerability compared to occipital hair follicles. We also see, besides those of non-scarring alopecia, graying of hairs in general also show a tendency to involve anterior scalp earlier and more than occipital area. Accordingly, it appears that human scalp may be divided into two parts in relation with the durability of its hair follicles as a vulnerable region and a resistant region. The vulnerable region is the frontoparietal area while the resistant region is the occipital area.

The inherent difference between the two different regions of the scalp may be related with its difference in its embryonic origin. Ziller already found that in the quail-chick model, dermis of the frontoparietal scalp is derived from the neural crest, whereas dermis

of the occipital scalp is derived from mesoderm¹⁰ and there are authors who speculated the embryonic difference in relation with the different response of follicles in the scalp regions in androgenetic alopecia¹¹.

In addition to the embryonic developmental difference of the two scalp regions, there is also a difference in sensory nerve innervations between the two regions. Sensory innervation of the frontoparietal region is by a cranial nerve (trigeminal), whereas the sensory innervation of the occipital region is by a spinal nerve (cervical) (Fig. 1). It has been increasingly elucidated that sensory nerves function not only as an afferent but also as an efferent system able to stimulate or suppress target tissues by secreting different neuropeptides¹². Effector roles of peripheral endings of afferent neurons could include control of blood flow and vascular permeability, regulation of gene expression and control of autonomic function¹³. Studies on the role of sensory neuropeptide in hair follicle biology may contribute a novel way for managing excessive hair shedding in future^{14,15}.

In short, it appears that embryologically and neuroanatomically the frontoparietal scalp is a part of face, whereas the occipital scalp is a part of upper back. Although these coincidences are not to explain the different follicular behaviors of the different scalp regions at present time, it is very interesting to see that there are such close regional overlaps between follicular behavior, embryonic development and sensory innervation in the human scalp.

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