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Hair Pain (Trichodynia): Frequency and Relationship to Hair Loss and Patient Gender

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Key Words

Hair pain \cdot Female gender \cdot Scalp telangiectasia \cdot Neuropeptide substance P

quantitative parameters of hair loss or specific morphologic changes of the scalp, management remains empiric and tailored to the individual.

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Abstract

Background: Patients complaining of hair loss frequently claim that their hair has become painful. Objective and Methods: The aim of the study was to evaluate the frequency of this phenomenon and its relationship to hair loss. Patients seeking advice for hair loss either spontaneously reported or were questioned about painful sensations of the scalp. Hair loss activity was quantified by a hair pull, daily count and wash test. Telogen percentage was obtained by a hair pluck. The scalp surface was examined by dermatoscopy. Results: Of 403 examined patients, 20% of women and 9% of men reported hair pain, irrespective of the cause and activity of hair loss. A minority presented scalp telangiectasia. This strongly correlated with hair pain. Conclusions: Hair pain (trichodynia) affects a significant proportion of patients complaining of hair loss and may increase the anxiety. The symptom neither allows discrimination of the cause nor correlates with the activity of hair loss. A higher prevalence of female patients might be connected to genderrelated differences in pain perception in relation to anxiety. The role of vasoactive neuropeptides in the interaction between the central nervous system and skin reactivity is discussed. In the absence of any correlation with

The term 'trichodynia' has recently been proposed for discomfort, pain or paresthesia of the scalp related to the complaint of hair loss [1]. Rebora et al. [1] found that 34.2% of female patients, who had their hair consultation because of hair loss, complained of this phenomenon. In a subsequent survey, Grimalt et al. [2] claimed that 14% of their patients reported trichodynia. The cause of trichodynia is not understood, though it has been proposed that it is probably polyetiologic [3, 4]. The most prevalent speculations with respect to the pathogenesis of trichodynia are perifollicular inflammation [5], increased expression of the neuropeptide substance P localized in the vicinity of hair follicles [6] and underlying psychiatric disorders [7]. An interesting observation made by Hua et al. [8] was scalp tingling in 2 of 30 patients on labetolol for arterial hypertension. So far, trichodynia has been reported to be more prevalent in female patients with chronic telogen effluvium (CTE) and to a lesser extent in patients with androgenetic alopecia (AGA) [1, 2]. Rebora [9] proposed the symptom to be distinctive for CTE. This observation has recently been challenged [4].

We present our observations on 403 patients (311 females, 92 male) whose main complaint was hair loss.

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The aim of the study was to assess the frequency of trichodynia in patients complaining of hair loss and its correlation with gender, age, cause and activity of hair loss.

Patients and Methods

In patients seeking advice for hair loss in the hair consultation clinic between 1997 and 1999, a study of the following data was systematically performed: (1) gender and age, (2) cause of hair loss, (3) complaint of painful sensations of the scalp and (4) activity of hair loss. Patients either spontaneously reported or were questioned about painful sensations of the scalp. Pruritus was not considered, and specific dermatologic diseases of the scalp were excluded. Hair loss was quantified by a hair pull, daily count and wash test, and telogen percentage was obtained by a hair pluck (trichogram), as previously described [10]. The scalp surface was examined by dermatoscopy (×10 Heine optics).

Results

Of 403 patients aged between 13 and 87 years (mean: 42 years), 311 were female (age range: 13–87 years; mean age: 46 years) and 92 were male (age range: 14–71; mean age: 31 years). Seventy of 403 patients (17%) suffered from trichodynia.

There was a significant relationship between the gender of patients and the complaint of trichodynia. Trichodynia preferably occurred in female patients with 62 of 311 female (20%) and only 8 of 92 male patients (9%), respectively (p = 0.0119 in Fisher's exact test).

Overall there was also a significant correlation between the age of the patient and the occurrence of trichodynia. The mean age \pm SD of patients with trichodynia was 46 \pm 15 years, while that of those without trichodynia was 41 \pm 15 years (p = 0.0106, Mann-Whitney test). This was due to gender-dependent differences in the frequency of trichodynia, and the younger age of men complaining of hair loss: the mean age \pm SD of female patients with trichodynia was 47 \pm 14 years, while without trichodynia it was 45 \pm 15 years (p = 0.1488, Mann-Whitney test, nonsignificant). The mean age of male patients with trichodynia was 35 \pm 17 years, while without trichodynia it was 30 \pm 11 years (p = 0.4799, Mann-Whitney test, nonsignificant).

Forty-six of 70 (66%) patients suffering from trichodynia spontaneously reported their complaint, 24 of 70 (34%) only when directly addressed. Forty-three of 62 female patients (69%) and 3 of the 8 male patients (38%) with trichodynia spontaneously reported scalp pain. No significant correlation was found between the gender of

Table 1. Frequency of trichodynia in relation to cause of hair loss, whether reported spontaneously or upon questioning

Diagnosis	Trichodynia	Trichodynia reported spontaneously	Trichodynia reported upon questioning	
AGA CTE Combination	48/297 (16) 6/24 (25) 16/82 (20)	30/297 (10) 6/24 (25) 10/82 (12)	18/297 (6) 0/24 (0) 6/82 (8)	
Total	70/403 (17)	46 403 (11)	24 403 (6)	

Figures in parentheses indicate percentages.

patients and whether trichodynia was spontaneously reported or upon questioning (Fisher's exact test: p = 0.1125). Due to the small number of male patients, this result is not reliable.

Of the 403 patients examined, AGA was diagnosed in 297 (74%), CTE in 24 (6%) and a combination of both in 82 patients (20%). There was no correlation between trichodynia and the cause of hair loss (p = 0.4635, χ^2 test). For the frequency of trichodynia in relation to the cause of hair loss, see table 1. Also, no correlation existed between the cause of hair loss and whether trichodynia was spontaneously reported or upon questioning (χ^2 test: p = 0.1805).

Most importantly, there was no statistically significant correlation between the presence of trichodynia and parameters of hair loss activity (table 2). Also, no correlation was found with the pattern (female, male) and type (Ludwig, Hamilton-Norwood scale) of AGA.

Finally, 7 of 9 patients, in whom dermatoscopy of the scalp demonstrated telangiectasia complained of trichodynia: 2 of 3 patients with AGA, 2 of 3 with CTE, and all of 3 patients with a combination of both. Fisher's exact test calculated p < 0.0001 and herewith a strongly significant correlation between the presence of scalp telangiectasia and complaint of trichodynia, though vice versa a majority of patients with trichodynia did not show scalp telangiectasia. All patients with scalp telangiectasia and trichodynia spontaneously reported scalp dysesthesia.

Comment

Trichodynia is a commonly encountered symptom in patients concerned with hair loss. Our study confirms previously published findings in the literature that trichody-

Table 2. Trichodynia and parameters of hair loss activity (daily hair count, hair wash test, hair pull and trichogram)

Parameters of hair loss	AGA		CTE	CTE		Combination	
activity in patients with and without trichodynia	with trichodynia	without trichodynia	with trichodynia	without trichodynia	with trichodynia	without trichodynia	
Daily hair count Hair wash test	62±54 107±33	75 ± 102 321 ± 411	44±19 no values	82±80 418±489	102 ± 76 205 ± 143	87±71 293±251	0.8570 (n.s.) 0.1326 (n.s.)
Hair pull							
Pathologic hair pull (≥ 5/50 ha	airs)						
Frontal	13/25 (52)	60/110 (55)	0/3 (0)	2/16 (12.5)	4/10 (40)	14/50 (28)	
Occipital	0/25(0)	3/110 (2.5)	1/3 (33.3)	4/16 (25)	2/10 (20)	12/50 (24)	
Frontal + occipital	2/25 (8)	5/110 (4.5)	1/3 (33.3)	6/16 (37.5)	3/10 (30)	13/50 (26)	
Normal hair pull	10/25 (40)	42/110 (38)	1/3 (33.3)	4/16 (25)	1/10 (10)	11/50 (22)	0.9685 (n.s.)
Trichogram							
Pathologic trichogram (>20%	telogen)						
Frontal	15/35 (43)	91/163 (56)	0/4(0)	0/17(0)	0/12(0)	3/54 (5)	
Occipital	2/35 (6)	4/163 (2)	2/4 (50)	8/17 (47)	5/12 (42)	15/54 (28)	
Frontal + occipital	7/35 (20)	11/163 (7)	0/4(0)	5/17 (29)	6/12 (50)	28/54 (52)	
Normal trichogram	11/35 (31)	57/163	2/4 (50)	4/17 (24)	1/12 (8)	8/54 (15)	0.320 (n.s.)

n.s. = Nonsignificant; p values of daily hair count and hair wash test obtained by Mann-Whitney test, those of hair pull and trichogram by the χ^2 test. Figures in parentheses indicate percentages.

nia affects a significant proportion of patients complaining of hair loss. While Rebora et al. [1] stated a frequency of 34.2% in women attending their clinic with the complaint of hair loss, others have found in their patient collectives an overall frequency of 14.3%, i.e. 22.1% of female and 4% of male patients [2], and 30.9%, i.e. 38.5% of women and 4% of men [11]. In our series, we found that 17% of patients complaining of hair loss, i.e. 20% of female and 9% of male patients, reported 'hair pain', pain or discomfort of the scalp, not otherwise explained by the presence of a specific dermatologic disease, such as psoriasis or eczema, or a neurologic disorder, such as migraine equivalent.

In accordance with Rebora et al. [1], statistical analysis failed to demonstrate any significant correlation between trichodynia, the extent of hair thinning and hair loss activity, quantified by the hair pull, daily hair count, wash test and trichogram. It is noteworthy though that trichodynia typically increases the anxiety related to the patient's preoccupation with hair loss or fear of hair loss.

As opposed to the suggestion of Rebora et al. [12] that trichodynia would be typical of CTE, in our series the symptom did not allow any discrimination with respect to the cause of hair loss and was found with similar frequencies in association with AGA, CTE or a combination of both.

The cause of trichodynia remains obscure. Rebora et al. [1] proposed a possible role of perifollicular microin-flammation, though this is more frequently found in AGA than in CTE [5, 13]. Hoss and Segal [7] interpreted scalp dysesthesia as a cutaneous dysesthesia syndrome related to underlying psychiatric disorders, with affected individuals either suffering of depressive, generalized anxiety or somatoform disorder. We formerly proposed that trichodynia probably is polyetiologic [3, 4]. Ericson et al. [6] found localization of the neuropeptide substance P in the scalp skin of patients with painful scalp, suggesting a causal relationship between the presence of substance P and trichodynia. Substance P represents a neuropeptide involved in nociception and neurogenic inflammation.

Though only a small number of patients with trichodynia in our series showed telangiectasia of the scalp, this finding strongly correlated with the presence of trichodynia. An interesting analogy is the observation of Lonne-Rahm et al. [14], who found that patients with the telangiectatic variant of rosacea respond more frequently with stinging sensations to the topical application of 5% lactic acid on the cheeks than patients with the papulopustular type of rosacea or normal controls. On the basis of these findings, they concluded that the blood vessels are of importance in stinging sensations, and a connection exists between sensory or subjective irritation and cutaneous

vascular reactivity. Also the observation of development of cutaneous allodynia during a migraine attack provides clinical evidence for the relation of vascular changes and pain [15].

In this context, it is interesting to note that substance P not only represents an important mediator of nociception and neurogenic inflammation, but also exerts a potent vasodilatory effect. The role of substance P and related substances (neuropeptides) in the pathogenesis of trichodynia, and especially its relation to the nervous system and emotional stress need further elucidation. By the virtue of their bidirectional effects on the neuroendocrine and immune systems, substance P and other neuropeptides may well represent key players in the interaction between the central nervous system and the skin immune and microvascular system. Such mechanisms would explain the noxious effects not only of external stimuli (mechanical, thermal, chemical), but also of emotional distress on cutaneous nociception through the release of neuropeptides, such as substance P [16]. Interestingly, Arck et al. [17] have recently demonstrated that stress-induced immune changes of the hair follicles in mice could be mimicked by injection of substance P in nonstressed animals and that they were abrogated by selective substance P receptor antagonism in stressed animals.

The higher prevalence of female patients might be connected to gender-related differences in pain perception, inasmuch as an increase in pain perception in relation to

anxiety scores has been found to be more pronounced in females [18]. Trichodynia tends to affect the centroparietal area of the scalp, which is seemingly surprising since the pain threshold of the centroparietal scalp is otherwise considered to be higher [19].

In the absence of any other specific morphologic changes of the scalp or a correlation with quantitative parameters of hair loss, the management of trichodynia remains empiric and empathetic, tailored to the individual patient's needs. The therapeutic choice includes nonirritating shampoos, topical antipruritic or anesthetic agents, topical capsaicin, corticosteroids [9], tricyclic antidepressants and gabapentin [7]. The future role of antidepressants on the basis of selective substance P inhibition (MK-869) [20] for treatment of trichodynia will be interesting. As a general rule, topical overtreatment of the scalp is to be avoided. Most importantly, the patient needs to be reassured that trichodynia does not reflect hair loss activity, which may ease the patient's anxiety and in our experience may also beneficially influence cutaneous nociception.

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