Chronic stress as a trigger for telogen effluvium: a psychophysiological aspect in the practice of a trichologist

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Abstract. Nowadays, the concept of "stress" is an integral part of our lives. We use it to describe our state of being out of our comfort zone too often, without thinking about the actual content of this term, the scale of morpho-functional, psycho-somatic manifestations and consequences. It is impossible to consider the effects of stress locally, because the mechanisms of its occurrence are not isolated processes, but parts of long functional chains that operate according to the "domino" principle, when one component, falling, pushes another onto the next and starts a chain reaction of falling. In the human body, which is a dynamic system and is represented by a complex of other dynamic systems, a similar principle of mutual activation operates, which can also return in a circle to the initiator and restart pathogenesis with new force. Using the example of stress-induced telogen effluvium, some components of the psychosomatic component in the development of alopecia are considered, the main axes and circles of interaction of the mechanism of hair loss due to the prolongation of the telogen phase of the hair follicle under stress, which occurs indirectly through the action of a number of hormones, such as, for example, the relationship between elevated cortisol levels and impaired microcirculation in the scalp; the direct effect of the nervous system on the hair follicle, impaired expression of biological substances (neurotransmitters) necessary for maintaining the microenvironment and the hair's own homeostasis, and many others. Attention is focused on the ways of correction, not only surgical and medicinal, but also non-medicinal by using methods of stabilizing the human condition (gentle care rituals, relaxation techniques, work with trichological ampoules, massage), which can be provided to the patient as a client of the salon practice.

Keywords: psycho-emotional stress, functional system, mental illness, telogen effluvium, hair loss, hair thinning, anagen, telogen, hair cycle.

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Introduction

Why has the topic of post-stress hair loss been, is, and will continue to be relevant for a long time? Because stress-induced hair loss, most often referred to as telogen alopecia, is not only an organic hair problem, but also a reflection of internal problems of the body, economic problems of the individual, social problems, and even socio-economic problems of society. A specialist who is approached by a person with a cosmetic defect should be prepared for this in order to correct it quickly, rather than solving all the problems that caused it to appear. A trichologist is a specialist with medical education who deals with the treatment, diagnosis, and prevention of skin and scalp diseases. For maximum effectiveness, a trichologist's practice should combine medical and hardware methods, as it exists at the intersection of several specialties: cosmetology, dermatology, and even psychology. The object of study and influence in trichology is the hair and scalp.

According to the classification, the following types of hair are distinguished on the human body: long, bristle, and vellus. Hair is considered a skin appendage and is associated with it not only embryonically and locally, but also functionally. Morphologically, a hair consists of a shaft and a root. The root and part of the shaft are embedded quite deeply in the skin and surrounded by external and internal epithelial sheaths, a hyaline membrane, and a dermal root sheath, which create a favorable microenvironment for hair growth and regeneration. The growth and nutrition of the hair is directly provided by the hair bulb, which consists of a matrix and a hair dermal papilla [1]. Let us consider the structure and function of the latter to emphasize its importance for the normal physiological state of the hair. The hair dermal papilla is represented in classical morphology as an ingrowth of loose connective tissue into the basal membrane of the matrix, from which the hair root grows. A distinctive feature of the structure of loose connective tissue is that it contains a capillary network and is a source of nutrition for the epithelial cells that lie on it, separated by the basement membrane. Nutrition is provided by the drainage of nutrients through the basement membrane. What does hair have to do with it? The fact is that it is mainly formed by epithelial cells at different stages of their differentiation, called “keratinocytes,” and the matrix cells are also undifferentiated epithelial cells. Since hair growth and regeneration depend on the morpho-functional state of the matrix cells, which in turn depend on the components of the hair dermal papilla, the question of its morpho-functional changes under the influence of external and internal factors becomes obviously important and relevant.

It is well known that hair grows at an average rate of 0.35 mm per day, and this process is part of the cyclical changes in the condition of hair as an extremely dynamic structure. About 80% of human hair follicles are usually simultaneously in the active growth phase, called “anagen,” which can last up to 10 years. The next phase of the hair life cycle, characterized by a halt in growth and proliferation of keratinocytes, is called “catagen” and can last several weeks. The final phase, “telogen,” or resting phase, can last about 100 days and is characterized by follicle involution followed by hair shaft removal at the end of the phase [2]. Hair growth is influenced by the following internal factors: 1) hormonal status (androgens can enhance hair growth, but with prolonged exposure and high concentrations, they can cause premature follicular atrophy, leading to androgenic alopecia; estrogens can slow down hair growth by prolonging the anagen phase; iodine-containing thyroid hormones, such as thyroxine, on the contrary, stimulate the onset of the anagen phase, thereby shortening the overall hair life cycle; Cortisol can inhibit the onset of the anagen phase, thereby delaying or, with prolonged exposure and high concentrations, causing irreversible atrophy of the hair follicles with subsequent alopecia.

Results

Alopecia is hair loss (thinning), which leads to a reduction in hair volume or complete loss (baldness). The most common types of alopecia include scarring, patchy, diffuse, androgenic,

and others. Alopecia caused by prolongation of the telogen phase with suppression of the anagen phase is called telogen alopecia and can be caused, for example, by hormonal changes in the body, such as a prolonged increase in the concentration of cortisol in the blood due to stress. It should be noted that the loss of about 100-150 hairs per day that were in the telogen phase is considered normal [3]. The rest of the hairs, which are currently in the anagen and catagen phases, provide hair volume. Hair length depends on the duration of the anagen phase, which can vary for different hair types (1-10 years for long hair on the head and 2-3 months for short eyebrow hair). Scientific sources refer to the anagen-telogen balance as a prerequisite for healthy hair without signs of alopecia, which is expressed in a ratio of 12:1 to 14:1 and refers to the number of hair follicles in the corresponding stages of the hair cycle. A significant increase in the proportion of the telogen phase in relation to the anagen phase has been noted in alopecia of various origins. The pathological prolongation of the telogen phase with the onset of alopecia has been well studied in experiments and in humans [4]. Morphologically, in biopsy samples of the scalp of patients suffering from alopecia, with various types of inflammation, perivascular infiltration by histiocytes, lymphocytes, and other immunocytes was detected, which was ultimately interpreted as folliculitis, accompanied by a surge in serum immunoglobulin E (IgE) levels. Such a massive immune attack primarily prevented the proper blood supply to the poorly differentiated cells of the matrix and led to degenerative changes in the very source of hair growth and regeneration, thereby prolonging the telogen phase to the state of follicle involution.

Similar morphological changes in the form of perivascular infiltration in the area of the dermal papillae were found in patients with stress-induced alopecia. Research into the effects of stress on the human body depending on gender, age, and concomitant diseases has been supplemented by the findings of scientists who have found a direct link between severe nervous shock, hormonal imbalance, and the acceleration of the transition of hair follicles from the anagen to the telogen phase with the onset of alopecia [5]. As for the hormonal factor, one of the well-known stress hormones, cortisol, has a significant effect on the growth and regeneration of hair follicle cells, regulating the synthesis of glycosaminoglycans, such as hyaluronic acid, and proteoglycans of the skin and its derivatives.

It has been proven that a prolonged increase in cortisol concentration inhibits the synthesis of the above substances by 40% [6] and can slow down the transition of hair into the active growth phase. Thus, the use of proteoglycan replacement therapy in the complex treatment of post-stress alopecia and postpartum alopecia in women who have given birth as a result of psycho-hormonal stress is relevant and predictably effective [7].

Let's return to cortisol and trace possible parallel effects of a non-metabolic nature on the hair follicle cycle. Cortisol is a steroid hormone of the adrenal cortex, produced by endocrinocytes of its fascicular zone under the stimulating influence of the pituitary adrenocorticotropic hormone and the controlling action of hypothalamic releasing hormones. Cortisol belongs to the group of glucocorticoids and increases significantly in concentration under stress conditions, triggering a chain reaction of neurohumoral changes in the human body designed to prepare it physically and psychologically for actions and changes aimed at preserving life in dangerous conditions. The main functions of cortisol include: increasing blood glucose levels by stimulating the breakdown of polysaccharides, stimulating lipogenesis with the accumulation of white adipose tissue in certain areas, stimulating lymphocyte apoptosis, and powerful anti-inflammatory and anti-allergic effects, which are used in clinics to stop the development of anaphylactic shock. There are a number of important points regarding the effect of cortisol on the cardiovascular system, namely: it increases the sensitivity of receptors to catecholamines (hormones of the adrenal medulla), thereby enhancing their effect; it also enhances the pressor effect of angiotensin II, one of the main factors of the renin-angiotensin-aldosterone system; it increases the tone of arterioles and the contractile capacity of the myocardium. In excessive amounts, cortisone can cause arterioles

to spasm and reduce the permeability of the capillary wall, which has a negative effect on tissue trophism, including the hair follicle matrix, which is nourished by the vessels of the hemicirculatory bed of the dermal papilla.

Separately, it should be noted that cortisol has an antiproliferative effect, which is due to the inhibition of fibroblast proliferation, as the main producers of connective tissue components of the dermis and hypodermis of the skin, which, with a prolonged increase in cortisol levels in the blood, leads to atrophic changes in the hypodermis and microenvironment structures of the hair follicle, on the condition of which the normal physiological course of the hair cycle depends.

If signs of alopecia induced by high cortisol levels due to stress appear, it is justified to use cortisol-blocking agents in the treatment regimen, such as 2% ketoconazole in hair care products, and those that improve the permeability of the capillary wall, increase blood flow to the problem area, and thus improve its nutrition. Good results in these areas in professional trichology have been obtained with the use of external preparations containing 2% minoxidil (trichological ampoules) [8]. Among the methods that can increase blood flow to areas of skin with impaired trophism, the use of physiotherapeutic methods such as darsonvalization of the scalp, ozonation of the scalp, massage, microcurrents, and myostimulation has been proven to be effective. An extremely successful solution in the treatment of telogen stress-induced alopecia is a combination of correction with methods of stabilizing the condition of the client or patient, such as gentle care rituals, relaxation techniques, and relaxing massage, aimed at gently suppressing psycho-emotional arousal and at the same time reducing the level of cortisol in the blood.

An important aspect of human life is the correspondence and internal synchronization of various cycles of existence and functioning of organs and systems that are subject to the influence of general circadian rhythms controlled by neurohumoral mechanisms. The main center for controlling the circadian rhythms of the human body is considered to be the pineal gland, which produces the well-known hormones serotonin and melatonin, triggering a chain reaction of sequential hormone secretion by various endocrine glands according to the time of day, followed by the launch of reactive responses from the working organs. This harmonious structured scheme of interaction can be disrupted and even destroyed by simply not adhering to a sleep regimen. Again, this problem is closely related to cortisol and a high probability of telogen alopecia [9]. The physiological norm is the release of cortisol by the adrenal cortex at around 6-8 a.m., reaching its peak at around 9 a.m. and beginning to affect the above-mentioned organs and systems that have the corresponding receptors. After 12:00, cortisol levels begin to decline, falling to their lowest point in the evening and thus ceasing to stimulate cardiovascular activity, metabolism, etc. Before bedtime, our body must slow down active processes in all areas in order to transition to a state of relative calm and accumulate reserves for the next day's active functioning. However, under certain conditions and for various reasons, this balanced and coordinated internal schedule can be disrupted, forcing the regulatory systems, nervous and endocrine, to adjust, trying to compensate for the negative effects of changes with some overtime activities, such as continuing intense mental work at a time when the brain is usually accustomed to transitioning to a state of relaxation; interrupted sleep leading to a spike in cortisol levels to bring the body's systems into tone. In fact, there are many such examples, and all of them, in terms of their mechanism of development and effect, fall into the category of stressful situations: something suddenly happens that forces the body to mobilize to the maximum in order to avoid danger. This opens up two possible options for the development of post-stress chain reactions: fast - after stressful activation of organs and systems by releasing catecholamines (adrenaline and noradrenaline) into the bloodstream through direct stimulation by the central nervous system; and a slow path - through the release of glucocorticosteroids (cortisone, cortisone) through the hypothalamic-pituitary-adrenal stimulation axis. Fast and slow mobilization

mechanisms work in parallel but have different durations. If the stressor is too strong or lasts too long, it can lead to different levels of stress (acute, subacute, chronic).

The effects of cortisol on the body as a whole have already been described, but it is worth adding information about its long-term blocking effect on the endocrine glands: suppression of pancreatic function, leading to an increase in glucose levels; inhibition of thyrotropic hormone secretion with a subsequent decrease in metabolic activity; suppression of sex hormone production, depletion of adrenal cortex endocrinocytes with a high probability of hypocorticism in the future. The issue of cortisol's effect on the pancreas is extremely relevant at present, as statistics show an increase in the percentage of stress-induced hyperglycemia, especially in young and middle-aged people. During acute stress, neuropeptides are rapidly released, which inhibit the production of adrenocorticotropic hormone by the pituitary gland, thereby reducing the synthetic activity of adrenal cortex endocrinocytes and, at the same time, the level of cortisol. Among these neuropeptides, glucagon-like peptide 1 (GLP-1) deserves special attention. It is produced by endocrine cells of the small intestine mucosa and stimulates the B cells of the islets of Langerhans to secrete insulin. It reduces appetite by blocking the hunger center in the brain and slows down gastric emptying by inhibiting the motility of its muscular membrane through the blocking of the contractile ability of smooth myocytes [10]. The next neuropeptide, Y (NPY), a powerful appetite stimulant, is produced by neurons in the brain stem, limbic system, and hypothalamus; [11] signals hunger and, under conditions of acute stress, ceases its action, indirectly leading to loss of appetite, but under chronic stress, it is activated and signals intensely, causing the need to “eat away stress.”

Considering the mechanisms of stress development at the morpho-functional level, affecting changes in all body systems without exception, we often observe the formation not only of chains of reactive changes, better known as axes, but also the formation of functional closed loops that represent a complex of processes that generate processes, stimulating the onset of subsequent ones, intensifying and moving to a new level of development, moving in a circle. An example of this is the self-stimulating development of chain reactions in the nervous system, which move in a circle during chronic stress, self-activating, continuously increasing in strength and cortisol concentration, driving themselves to complete exhaustion. Often, external and internal psychological factors act as additional catalysts for chronic stress, depriving it of the chance to self-terminate. Such factors may include, in addition to the already described circadian rhythms and sleep disturbances, emotional burnout, multitasking, perfectionism, family conflicts, conflicts at work, career pressure, financial instability [12], and repeated traumatic experiences. Early clinical manifestations of stress appear within two months in the form of infectious diseases due to decreased immunity, systemic inflammatory processes, neuroses, tremors, tremors, and disorders of the digestive, cardiovascular, and other systems. These are quickly joined by changes in appearance, which gradually turn into cosmetic defects over time, becoming particularly traumatic factors. These include increased sweating, specifically non-congenital hyperhidrosis, anabuty, due to hyperactive sympathetic and parasympathetic nervous systems, mediated by adrenaline and noradrenaline; the appearance of acne due to the hyperproduction of sebum by the exocrine cells of the sebaceous glands of the skin, stimulated by androgens, the level of which increases under conditions of high cortisol levels, and directly by cortisol itself; the occurrence of skin inflammation due to a decrease in local immunity, disruption of the skin microbiome, and the addition of bacterial flora; various types of alopecia as a result of pathological changes in the skin and directly in the hair structure.

Let's return to the issue of pathological changes in hair under stress conditions that lead to alopecia. As mentioned earlier, each hair grows from a hair follicle, receiving nutrition and innervation from the dermal papilla, which contains capillary and nerve networks. The morpho-functional state of the follicle determines the phase of hair development and depends

on the state of the dermal papilla and the correct interaction of the matrix and root components with it.

Under conditions of acute stress, as a result of the release of catecholamines (neurotransmitters) into the blood, which disrupt trophism by possibly causing spasm of the papilla vessels, followed by a shortening of the anagen phase and the transition of the hair to the telogen phase, the situation may result in hair loss that was in or transitioned to the telogen phase, known as telogen alopecia. Depending on its duration, telogen alopecia can be acute (up to 6 months) or chronic (more than 6 months) [13]. Chronic telogen alopecia is the result of the combined pathogenic action of excess catecholamines (inducers of the acute phase of stress) and cortisol (the main catalyst for the development of chronic stress). It should be noted that prolonged hyperproduction of cortisol increases the sensitivity of receptors to catecholamines, thereby prolonging their action. In addition, it reduces the permeability of the capillary wall and induces the development of perivascular infiltrates, which together further impairs the blood supply to the hair follicles, causing prolongation of the telogen phase and alopecia. Based on the pathogenesis, the treatment regimen for telogen stress-induced alopecia includes, in addition to specific correction, the use of various physiotherapeutic methods to improve blood supply, which can be performed in beauty salons (massages, myostimulation, darsonvalization, ozone therapy). . Since the psychosomatic component in the pathogenesis of telogen alopecia has been proven, calming methods of influencing the psyche and methods of stabilizing the condition should be used. Thus, the neuroendocrine axis involving cortisol is the most significant and complex psychosomatic mechanism in the development of alopecia. Other mechanisms include the suppression of autophagy of abnormal, poorly differentiated hair follicle cells with impaired proliferation and the suppression of the production of nerve and blood vessel regeneration factors.

One of the functional antagonists of cortisol is oxytocin, a neuropeptide produced by neurosecretory cells in the anterior hypothalamus, transported along axons to the neurohypophysis, from where it enters the bloodstream through axo-vascular contacts when needed and acts as a natural relaxant, reducing anxiety, fear, and cortisol levels. It also improves blood supply to the hair follicle, promoting hair growth and completion of the telogen phase. The use of oxytocin preparations for hair stimulation is complicated by the rather large size of the molecule, but clinical studies using oxytocin agonists to stimulate hair growth in stress-induced alopecia have shown positive results [14].

Substance P is a well-known neuropeptide that acts similarly to a security alarm in the body. It is secreted by the dendrites of neurons in response to damage and signals this damage to the central nervous system [15]. In terms of its activity and significance for the skin and hair, Substance P is secreted by the dendrites of sensory neurons that innervate the thermal papilla, ensuring the release of nitric oxide by the endothelial cells of the capillary network for vasodilation and increased blood flow; increases the permeability of the vascular wall, mobilizing mast cells, and thereby improves blood circulation in the skin and hair follicle. Thus, through the action of Substance P, neurotrophic control of the hair follicle is carried out, and the production of the substance itself can be activated through local physical influence on the problem area, including by means of physiotherapy procedures, which we have already described.

It is worth focusing separately on mediators such as serotonin and melatonin and their importance for hair growth and regeneration [16]. Serotonin is a metabolic precursor of melatonin. They are produced by neurosecretory cells (pinealocytes) of the pineal gland (epiphysis). Pinealocytes are in direct contact with blood capillaries, forming axo-vascular synapses, and have numerous synaptic contacts with the ends of adrenergic sympathetic nerve fibers on the surface of the plasma membrane. The epiphysis is part of our body's

photoneuroendocrine system, communicating with other components through these synapses. Melatonin is produced by pinealocytes at night, or in darkness, from serotonin that has already been secreted during the day (during the light period). Daylight has an inhibitory effect on pinealocytes in terms of melatonin production through a chain of interactions between structures of the nervous system. When light rays hit the retina, light-sensitive ganglion cells are activated, which carry the impulse further to the body's circadian rhythm coordinator, which is considered to be the suprachiasmatic nucleus of the hypothalamus. The excitation then travels to the neurons of the paraventricular nucleus of the hypothalamus and the upper segments of the thoracic spinal cord, where the centers of the sympathetic nervous system are located in the lateral intermediate nucleus of the lateral horn of the gray matter. then the impulse travels to the upper cervical node of the sympathetic trunk and returns to the epiphysis. Neurons of the suprachiasmatic nucleus, excited by daylight, inhibit the activity of neurons of the upper cervical ganglion, blocking the synthesis of melatonin until darkness falls. This is one of the mechanisms of circadian regulation in the human body. In addition to regulating the “sleep-wake” cycle, the hormone melatonin regulates seasonality, influences emotional state through the centers of the limbic system, and influences sexual maturation by blocking gonadotropin-releasing hormone in early childhood. In recent years, scientists have been interested in a series of studies whose results prove the positive effect of melatonin on hair growth and regeneration [17], which justifies the use of melatonin preparations in trichological practice for the treatment of alopecia of various origins. It has been proven that hair follicle matrix cells have melatonin receptors [18], stimulating their proliferation and hair growth, ensuring that the follicles remain in the anagen phase. In addition, both melatonin and serotonin have antioxidant properties, thus providing round-the-clock protection of hair follicles from aggressive free radicals and external negative factors. It should be added that since a disruption in the body's daily rhythm, brought to a state of decompensation, is a stress factor accompanied by cortisol imbalance, the addition of melatonin to the treatment regimen for stress-induced alopecia is a justified step and is widely used in trichology and medicine. However, the method of external application of melatonin preparations, the effectiveness of which is questioned by some scientists [19] and of interest to others, attracts attention. The role of locally produced melatonin in the bone marrow, spleen, intestines, and skin [20], which is controlled by the circadian rhythm, has been noted. Locally secreted melatonin participates in antitumor, antioxidant, anti-inflammatory, and pro-immune processes, having its own phase of expression. For example, the proliferation of inactive epidermal cells and hair follicles occurs mainly during the day, while DNA synthesis, activation of blood circulation, and increased permeability of the blood capillary walls occur mainly at night. In general, local melatonin in the skin increases its thickness due to the dermis and hypodermis, stimulating the synthetic activity of fibroblasts through their melatonin receptors; It regulates the physiological pH of the skin, improves the skin's barrier function by inhibiting keratinocyte apoptosis by blocking a factor associated with the mitochondrial pathway (caspase 9), and stimulates skin sebocytes to produce sebum. Melatonin has lipophilic activity, so it can penetrate the water-lipid barrier of the skin and be used for local correction of skin and hair conditions in the form of topical preparations. A reduction in skin wrinkles, manifestations of hyperkeratosis, increased hair density, and reduced hair loss have been noted with the topical application of melatonin preparations [21]. Based on the results of a series of studies, an effective dose of melatonin for topical application has been determined. According to scientists, it is 0.0033% or 0.1% solution for external application once a day for 90–180 days, compared to 1.5 mg twice a day of oral melatonin for 180 days [18]..

Conclusions

In describing the problem of hair loss, we have attempted to combine the morphological features of its structure and the surrounding environment, highlighting the nuances of its functioning and interaction with the body's dynamic control systems. The human body, being

an extremely dynamic system in an aggressive environment filled with other dynamic systems, is subject to strong external influences, which force it to change compensatorily and thereby change the subordinate internal systems. Any strong external influence that forces the body's dynamic systems to change rapidly to compensate for the effects of this influence can be considered a stress inducer. So stress is not just a mood swing or a departure from a state of comfort. It is a complex of external and internal morphological and functional changes that occur at all levels in all systems, organs, and cells of the human body. Alopecia (hair loss) is not just the loss of most of the hair cover, but a manifestation of decompensation and post-stress changes in the body. Therefore, the correction of such pathological conditions as telogen alopecia should be comprehensive and dynamic, involving a variety of possible means and methods. The latest scientific achievements offer such means of hair restoration as hair follicle transplantation and the use of stem cells [22]. They have given excellent results, are welcomed, expected, and are in high demand among patients. However, the immediate removal of the manifestation of the problem does not mean its solution and does not give a 100% guarantee of avoiding recurrence. If the cause of telogen alopecia is stress, then in parallel with cosmetic correction, the psychological factor should be worked on by referring the patient to a psychologist, salon procedures with a cosmetologist, advising them to change their rhythm and lifestyle, take a break, and visit related specialists who will add their recommendations to the plan based on the state of the body systems within their competence. This is not mandatory, but it should be effective. A specialist who deals with hair health (trichologist) must be knowledgeable about the morphological structure and physiological processes associated with all components of the hair and surrounding structures that create its environment and share its embryonic origin. This knowledge is a mandatory component of their professionalism, but deeper knowledge, which adds breadth of thinking and expands the boundaries of a trichologist's capabilities as a physician and scientist, increases their level of perception and authority in the professional environment.

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