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To Evaluate the Indicators of Lipid Peroxidation and Enzymes of the Antioxidant System in Patients with Alopecia

Leila Rafikovna Khakimona, Sulaymanova Nilufar Ergashevna
General Practice of Family Medicine, Samarkand State Medical University, Samarkand, Uzbekistan

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ABSTRACT

this article attempts to reveal the main reasons for assessing the indicators of lipid peroxidation and enzymes of the antioxidant system in patients with alopecia areata (AA). To carry out scientific work, the author conducted a study of the venous blood of 35 patients with focal forms of HA in the progressive stage of the disease (main group; mean age 34.1±2.0 years) and 31 healthy individuals (comparison group; mean age 30.5±1 ,8 years). The problem in question is still little studied, and therefore requires more thorough research.

Introduction: Alopecia (lit. "baldness" from other Greek ἀλωπεκία through Latin alopecia "baldness, baldness") is a pathological hair loss, leading to their partial or complete disappearance in certain areas of the head or torso. The most common types of alopecia include androgenetic (androgenetic), diffuse or symptomatic (effluviums), focal or nested (areata), scarring (scarring).

Allocate alopecia:

by prevalence

- total or atrichia (loss and absence of hair on the head (including eyebrows and eyelashes) and even on the whole body);
- diffuse or hypotrichia (thinning and thinning of hair throughout the head or body, including: Unn's syndrome, anagen alopecia, telogen alopecia, with asbestos lichen);
- focal or nested (occurrence of foci of complete absence of hair, including: frontal fibrous alopecia, temporal triangular alopecia, ophiasis (alopecia areata);

scarring of the hair follicle

- cicatricial (hair does not grow on the skin of scars):

primary, for example, with pseudopelade (atrophic circular), Kenko decalving folliculitis, Pusey exfoliating cellulitis (undermining folliculitis and perifolliculitis of the head), central centrifugal cicatricial alopecia, keloid folliculitis (keloid acne);

secondary, for example, with post-traumatic scars, scleroderma, mucinous folliculitis, etc.;

- ✓ non-scarring, for example: premature (presenile, androgenic) - male pattern baldness of the scalp, associated with blood levels of male sex hormones; traction alopecia (manipulative, samurai disease) - usually caused by wearing certain hairstyles that pull hair together;
- ✓ mixed, for example: Piccardi-Lassueur-Graham-Little syndrome - scarring alopecia of the scalp and non-scarring alopecia of the axillary and inguinal regions, observed with lichen planus [de], a type of lichen planus, can be combined with vulvo-vaginal-gingival syndrome and frontal fibrosing alopecia.

In addition, alopecia can accompany some diseases - for example, syphilis, ringworm, trichotillomania, progeria, skin myxedema, Fox-Fordyce disease, Sjögren-Larssen syndrome, radiation sickness, lamellar ichthyosis, etc.

Aim: to evaluate the parameters of lipid peroxidation and enzymes of the antioxidant system in patients with alopecia areata (AA).

Materials and methods: the material for the study was the venous blood of 35 patients with focal forms of GA in the progressive stage of the disease (main group; mean age 34.1±2.0 years) and 31 healthy individuals (comparison group; mean age 30.5±1.0 years). 8 years). The serum level of malondialdehyde (MDA), the activity of superoxide dismutase (SOD) and catalase in erythrocyte hemolysate were determined. Spectrophotometry was performed on a Genesys 5 instrument (USA). The scores are described using the median and the 25th and 75th quartiles. The Mann-Whitney test was used to compare the data.

Results: in patients with GA, the SOD activity index was 202.04 [197.57; 221.94] arb. units/g, exceeded the same indicator in the comparison group, equal to 112.87 [108.91; 112.75] arb. U/g protein, by 179.0% (<0.001). At the same time, the enzymatic activity of catalase in patients with GA was lower than in the comparison group by 35.8%, and, accordingly, amounted to 73.02 [60.28; 86.38] arb. u/g protein and 113.79 [103.86; 118.17] (<0.001). This indicates that the rate of hydrogen peroxide synthesis significantly exceeds the rate of its utilization by the enzymatic route. The accumulation in the tissues of this product, which is capable of interacting with metals of variable valence (iron, copper), will lead to the formation of a highly active hydroxyl radical and exacerbate oxidative stress. Evidence of this is the increase in the level of MDA, which is the final metabolite of lipid peroxidation, in the serum of patients with GA. Thus, the median content of MDA in the main group was 1.6 times higher than the level of this metabolite in healthy individuals and amounted to 3.86 [3.14; 7.71] μmol/ml and 2.36 [1.97; 2.83] μmol/ml (p<0.001).

Conclusions: a possible relationship between the pathogenesis of GA and oxidative stress can be explained by the initiation of hydrogen peroxide in the synthesis of the chemokine CXCL16 by keratinocytes, which, along with the skin lymphocytic antigen, provides a homing effect to the skin of CD8+ T cells synthesizing IFN-γ. An increase in the intrafollicular level of the latter initiates a key event in the pathogenesis of GA - the loss of the immune privilege of the hair follicle.

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