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Analysing Glutathione Peroxidase and Reductase in the Serum of Alopecia Areata Patients: A Case-Control Investigation

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To The Editor

One of the autoimmune illnesses in which hereditary and environmental variables are important is alopecia areata (AA).1. The aetiology of this illness has been the subject of numerous theories. The imbalance between antioxidants and oxidants is one of these theories. In emotional, environmental, and autoimmune stressors, an imbalance in the ratio of oxidant and antioxidant agents frequently arises.2. Skin has a vast range of antioxidants available. An enzymatic and non-enzymatic antioxidant network makes up this system. Enzymatic antioxidants include glutathione peroxidase, catalase, and superoxide dismutase; intracellular nonenzymatic antioxidants include a-tocopherol, ubiquinone, b-carotene, ascorbate, and glutathione.3. The balance of redox within cells plays a crucial part in maintaining skin health. In skin disorders, it maintains a balance between oxidant and antioxidant stimulation.

Strong antioxidant glutathione shields cell constituents against peroxides and free radicals.5. Usually, glutathione is paired with harmful free-radical molecules, which are then eliminated from the body. When glutathione reductase is eliminated by lowering glutathione's active form, free radicals build up and lipid oxidation causes metabolic damage.

Adverse effects, like the interchange of ionic permeability and enzymatic function, follow lipid peroxidation.Six Another enzyme with peroxidase activity is glutathione peroxidase, which can neutralise a variety of peroxides and whose primary biological function is to shield organisms from oxidative damage. There are conflicting findings from the few research on oxidative stress in AA that are accessible.

The purpose of our study was to compare the activity of glutathione reductase and glutathione peroxidase in AA patients with that of healthy controls. Our study included fifty-six patients who gave their consent. Nineteen healthy people who were the same age and gender made up the control group. The diagnostic kits (Zell Bio GmbH, Germany) were used to measure the activity of glutathione reductase and glutathione peroxidase in serum. The manufacturer's instructions were followed when conducting the ELISA tests. The Tehran University of Medical Sciences Ethics Committee has reviewed and approved this study (IR.TUMS.VCR.REC.1397437), and it was carried out in accordance with the Declaration of Helsinki. Thirteen girls and six males in the control group were in good health, while the patients consisted of 32 females and 24 males with AA. Nine patients—six females and three males—had alopecia totalis (AT), twenty-eight patients—seven females and eleven males—had AA, and nine patients—nine females and ten males—had alopecia universalis (AU). The average age of the patients was 31.05±13.72 years, while the healthy controls' mean age was 29.63±6.30 years (p value~0.05).

The activities of glutathione reductase and glutathione peroxidase in patients were measured and the mean and standard deviation were 267.22±217.21 and 71.46±54.42, respectively, whereas in healthy individuals the values were 180.24±89 and 50.99 ±36.9, respectively. There were no significant differences (p value <0.05 for both).

The glutathione peroxidase activity level mean and standard deviation in patients with AA, AT, and AU were 246.18±203.82, 322.46±233.75, and 272.05±235.21, respectively (p value~0.05). The mean and standard deviation of glutathione reductase

activity were 83.54±59.98, 85.73±68.79, and 46.89±24.20, respectively, for patients with AA, AT, and AU (p value~0.05). For both male and female patients, the mean glutathione reductase activity level was 64.98±54.69 and 76.32±54.58, respectively (p value~0.05). For both male and female patients, the mean glutathione peroxidase activity level was 191.43±172.60 and 324.06±232, respectively (p value~0.05).

In this investigation, there was no discernible difference in the serum concentrations of glutathione reductase or glutathione peroxidase between the AA patients and the control cohort. However, there is a great deal of disagreement among researches on the part antioxidant defence plays in the aetiology of AA. Superoxide dismutase and glutathione peroxidase activity rose dramatically in the scalp skin of AA active individuals, according to several investigations (Akar et al. It was proposed that AA does not compromise antioxidant defence.7. Some have documented reduced levels of certain antioxidants in AA. In the Bakry et al. study supporting AA, it was demonstrated that the mean serum total antioxidant capacity (TAC) value was lower in AA cases than in the healthy control group.

Similarly, Kim et al. found that patients' TAC levels were significantly lower than those of controls.10 According to Naziroglu and Kockam, patients with AA had significantly decreased glutathione peroxidase activity in their erythrocytes and plasma compared to the control group. Patients with alopecia were shown to have a notably reduced plasma b-carotene level in comparison to healthy persons.

In the serum samples of patients with AA and normal participants, Amirnia et al. discovered a substantial variation in the levels of zinc, copper, superoxide dismutase (SOD), and malondialdehyde (MDA). When compared to the healthy controls, these patients' levels of glutathione peroxidase were considerably lower. (p = 0.001).11 In contrast to the healthy controls, Gungor et al. found that patients with AA had higher serum lipid peroxidation and lower red blood cell levels of SOD and glutathione peroxidase.12 Our investigation did not find a significant difference between the two groups, in contrast to the Gungor and Amirnia study, which found that patients with AA had lower levels of glutathione peroxidase than the control group.

To sum up, there is a great deal of disagreement between the various studies. Examination of different tissues could be a possible cause. It has been shown that the activity of antioxidant enzymes varies throughout cell types. The short sample size is the primary limitation of our study. An additional constraint on our research was assessing the blood level of antioxidant enzymes without the evaluation of these skin tissue enzymes. Another drawback was not using the severity of alopecia tool (SALT) score to assess the degree of AA. To evaluate the role of antioxidant defence in the pathogenesis of AA, more research with larger sample sizes is required to assess the anti-oxidative enzymes and other related items of redox systems from various tissues related to AA.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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