Background: Idiopathic granulomatous mastitis (IGM) is a non-infectious inflammatory disorder of the breast characterized by non-caseous granulomas. It is a chronic granulomatous inflammatory disease frequently seen in young fertile women, the cause of which has not been clearly understood. Immunosuppressive agents and surgical interventions are used in the treatment. LL-37 is a cathelicidin-derived antimicrobial peptide with immunomodulatory properties that are effective in innate immunity. In addition, IL-36, galectin-3, TLR-3 are effective in autoimmunity with proinflammatory properties.

Objectives: With this study, we aimed to investigate the potential alterations of LL-37, IL-36, Galectin-3 and TLR-3 levels in IGM.

Methods: 35 female patients with biopsy-confirmed IGM and 35 healthy controls were included in the study. The serum samples of the subjects LL-37, IL-36, Galectin 3 and TLR-3 levels were studied using the Elisa method. While studying LL-37 and Galectin 3 levels in the tissue, samples of 10 patients who underwent mammoplasty for cosmetic reasons were used for the control group. Ten patients whose paraffin biopsy samples

Results: When the patient and control groups included in the study were compared, there was no significant difference in age. In serum samples, LL-37, IL-36, Galectin 3 and TLR-3 levels were statistically significantly lower in IGM group compared to the control group (p < 0.001 for each) (Table 1). In biopsy samples, LL-37 level was found to be significantly lower in IGM group compared to the control group (p < 0.001). However, no significant difference was detected in Galectin 3 levels in tissue studies (Table 2).

Conclusion: In our study, we found that the levels of LL-37, IL-36, Galectin 3 and TLR-3 decreased in serum samples in IGM disease whose etiology was not clearly understood. In addition, we showed that in patients with IGM, LL-37 levels decreased at the tissue level. Studies have shown that in cases of severe sarcoidosis, LL-37 deficiency is reduced both in level and gene expression. So they thought, deficiency of cathelicidin LL-37 may impede resolution of inflammation in the tissue of patients with severe form disease.

Disclosure of Interests: None declared.

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