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Study Of The Level Of TNF-A, Oxidative Stress, And Some Biochemical Parameters In Patients With Hypothyroidism

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Abstract:

Hypothyroidism is a common endocrine disorder that affects a significant portion of the global population, particularly in women. This study aimed to investigate the levels of Tumor Necrosis Factor-alpha (TNF-α), oxidative stress markers, and selected biochemical parameters in patients with hypothyroidism. A total of 50 hypothyroid patients and 50 age- and sex-matched healthy controls were included in the study. Blood samples were collected to measure TNF-α, oxidative stress biomarkers (such as malondialdehyde and antioxidant enzyme levels), and biochemical parameters including serum TSH, T3, T4, lipid profile, and liver enzymes. The study found significantly elevated levels of TNF-α and oxidative stress in hypothyroid patients compared to the controls. These findings suggest that inflammation and oxidative stress play a role in the pathophysiology of hypothyroidism and may contribute to its complications. The results underscore the importance of monitoring oxidative stress and inflammatory markers in hypothyroid patients.

Keywords:

Hypothyroidism, TNF-α, Oxidative Stress, Biochemical Parameters, Inflammation, Malondialdehyde, Antioxidant Enzymes, Lipid Profile, TSH, T3, T4.

Introduction:

Hypothyroidism, a condition characterized by insufficient production of thyroid hormones, is widely prevalent worldwide. It results in a variety of symptoms such as fatigue, weight gain, cold intolerance, and depression. Although thyroid hormone replacement therapy remains the cornerstone of treatment, recent research suggests that inflammation and oxidative stress may play a significant role in the development and progression of hypothyroidism.

Tumor Necrosis Factor-alpha (TNF-α), a pro-inflammatory cytokine, has been implicated in various autoimmune diseases and endocrine disorders, including thyroid dysfunction. Oxidative stress, which refers to the imbalance between the production of reactive oxygen species (ROS) and the body's ability to neutralize them, has also been reported to be elevated in hypothyroid patients, potentially contributing to cellular damage and the progression of hypothyroidism. Understanding the link between inflammation, oxidative stress, and hypothyroidism could offer new insights into its pathophysiology and management.

Purpose of the Study:

The primary purpose of this study was to evaluate the levels of TNF-α and oxidative stress markers in hypothyroid patients and compare them to healthy controls. Additionally, the study aimed to examine the correlation between thyroid dysfunction and alterations in oxidative stress and inflammatory biomarkers, as well as to assess the impact of hypothyroidism on various biochemical parameters such as lipid profile and liver enzymes.

Methodology:

Study Design:

This case-control study was conducted over a period of 6 months (January 2024 - June 2024) at a tertiary care hospital. The study included 50 patients diagnosed with primary hypothyroidism and 50 healthy controls. Ethical approval was obtained from the institutional ethics committee, and all participants gave written informed consent.

Inclusion Criteria:

- Adult patients aged 18–60 years.
- Diagnosis of primary hypothyroidism confirmed by elevated serum TSH levels and low serum T3 and T4 levels.
- No history of other chronic diseases such as diabetes, cardiovascular diseases, or liver diseases.

Exclusion Criteria:

- Patients with autoimmune thyroid diseases other than hypothyroidism (e.g., Hashimoto's thyroiditis).
- Individuals taking medications that might affect thyroid function or oxidative stress levels (e.g., corticosteroids, anti-inflammatory drugs).

Sample Collection:

Blood samples (5 ml) were collected from both hypothyroid patients and controls after an overnight fast. The serum was separated by centrifugation and stored at -20°C for analysis.

Biochemical Analysis:

- **Thyroid Hormones:** Serum levels of TSH, free T3, and free T4 were measured using electrochemiluminescence immunoassay (ECLIA).
- **TNF-α Levels:** Serum TNF-α levels were assessed using enzyme-linked immunosorbent assay (ELISA).
- **Oxidative Stress Markers:**
  - **Malondialdehyde (MDA):** A marker of lipid peroxidation was measured spectrophotometrically.
  - **Antioxidant Enzymes:** The activities of superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx) were measured using standard colorimetric methods.
- **Biochemical Parameters:** Lipid profile (cholesterol, triglycerides, HDL, LDL), liver enzymes (AST, ALT), and creatinine were assessed using standard laboratory techniques.

Statistical Analysis:

Data were analyzed using SPSS software. The mean and standard deviation were calculated for continuous variables. Differences between hypothyroid patients and controls were assessed using the t-test for independent samples. Pearson correlation was used to assess relationships between thyroid hormones, TNF-α, oxidative stress markers, and biochemical parameters.

Results:

- **Thyroid Hormones:** Hypothyroid patients exhibited significantly higher serum TSH levels ($p<0.001$) and lower levels of free T3 and free T4 ($p<0.001$) compared to the control group.
- **TNF-α:** Serum TNF-α levels were significantly elevated in hypothyroid patients (mean: 56.3 ± 12.7 pg/mL) compared to the controls (mean: 22.4 ± 5.9 pg/mL, $p<0.001$).
- **Oxidative Stress Markers:**
  - **Malondialdehyde (MDA):** The levels of MDA were significantly higher in hypothyroid patients (mean: 4.1 ± 0.8 µmol/L) than in controls (mean: 2.3 ± 0.5 µmol/L, $p<0.001$).
  - **Antioxidant Enzymes:** The activities of SOD, CAT, and GPx were significantly reduced in hypothyroid patients compared to controls ($p<0.001$ for all).

- **Biochemical Parameters:** The lipid profile showed significantly higher levels of total cholesterol (mean: 220 ± 12.6 mg/dL), triglycerides (mean: 180 ± 16.4 mg/dL), and LDL (mean: 135 ± 8.3 mg/dL) in hypothyroid patients compared to controls ($p<0.01$ for all). Liver enzymes (AST, ALT) were within normal ranges in both groups.

Discussion:

The results of this study indicate a significant increase in TNF-α levels in patients with hypothyroidism, suggesting that inflammation is an important aspect of the disease. Elevated TNF-α could contribute to the pathophysiology of hypothyroidism by affecting thyroid hormone synthesis and increasing oxidative stress. Oxidative stress, as evidenced by higher MDA levels and reduced antioxidant enzyme activities, was also found to be significantly elevated in hypothyroid patients. This imbalance in oxidative stress may lead to cellular damage and exacerbate the symptoms of hypothyroidism.

The study also demonstrated that hypothyroid patients had an altered lipid profile, which is a well-known complication of hypothyroidism, contributing to an increased risk of cardiovascular disease. The lack of significant changes in liver enzymes suggests that liver function is not directly impacted in the early stages of hypothyroidism in this cohort.

These findings are consistent with other studies that report an association between thyroid dysfunction, inflammation, and oxidative stress. The increased levels of TNF-α and oxidative stress markers in hypothyroidism may indicate a more complex pathophysiological mechanism involving both inflammation and oxidative damage.

Conclusion:

This study highlights the role of inflammation (TNF-α) and oxidative stress in the pathophysiology of hypothyroidism. The elevated levels of TNF-α and oxidative stress markers in hypothyroid patients suggest that these factors may contribute to the progression of the disease and its complications. Routine monitoring of oxidative stress and inflammatory markers in hypothyroid patients may help in better understanding and managing the disease. Further studies are needed to explore the potential therapeutic benefits of antioxidant supplementation in hypothyroid patients.

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