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HASHIMOTO THYROIDITS AND STRESS HORMONES: PATHOGENETIC MECHANISMS AND CLINICAL SIGNIFICANCE

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Annotation

Hashimoto thyroiditis (HT) is one of the most common chronic autoimmune disorders affecting the thyroid gland, characterized by immune-mediated destruction of thyroid tissue and impaired thyroid hormone synthesis. Epidemiological studies indicate that HT prevalence is approximately 5–10% in women and significantly lower in men (Weetman, 2004; Pearce et al., 2003). Chronic stress and elevated stress hormones — cortisol, adrenaline (epinephrine), and noradrenaline (norepinephrine) — have been implicated in the modulation of immune responses and may influence the clinical course of HT. This article reviews the interaction between HT and stress hormones, explores pathogenetic mechanisms, and discusses clinical implications with supporting statistical data.

Keywords

Hashimoto thyroiditis, stress hormones, cortisol, adrenaline, autoimmunity, thyroid hormones

Hashimoto thyroiditis is a chronic autoimmune disease of the thyroid gland, mediated by autoreactive lymphocytes and the production of anti-thyroid antibodies such as anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg). Population studies have shown HT prevalence of approximately 4–7% overall, with higher rates in older women (Ban et al., 2019). In affected individuals, anti-TPO antibodies are present in 70–80% of cases (Pearce et al., 2003).

Chronic stress activates the hypothalamic-pituitary-adrenal (HPA) axis, resulting in sustained elevations of cortisol and catecholamines. Prolonged stress hormone elevation can modulate immune function, affecting T-cell activity, cytokine balance, and antibody production. These effects suggest a potential role of stress hormones in the pathogenesis and clinical expression of HT (Duntas, 2009).

Physiology of Stress Hormones

1. Cortisol

Cortisol, a glucocorticoid hormone released from the adrenal cortex in response to HPA axis activation, follows a circadian rhythm with peak levels in the early morning and nadir in the evening. Under chronic stress conditions, baseline cortisol levels may remain elevated:

- Normal morning cortisol: ~300–600 nmol/L
- Elevated in chronic stress or Cushing's syndrome: >600 nmol/L (Chrousos, 2009)

Elevated cortisol exerts immunomodulatory effects by altering T-cell differentiation and cytokine production, thus contributing to autoimmune dysregulation.

2. Catecholamines (Adrenaline and Noradrenaline)

During stress, sympathetic nervous system activation increases catecholamine release:

- Adrenaline: Normal ~0.04–0.5 ng/mL (may increase during stress)
- Noradrenaline: Normal ~0.15–0.8 ng/mL (elevated in stress)

Catecholamines influence immune cell trafficking, macrophage activity, and pro-inflammatory cytokine levels.

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Interaction Between Hashimoto Thyroiditis and Stress Hormones

1. Immune Modulation

Chronic elevations of cortisol can suppress certain aspects of adaptive immunity while paradoxically enhancing autoantibody production. This dysregulation can contribute to increased production of anti-TPO and anti-Tg antibodies, promoting thyroid tissue destruction.

2. Clinical Correlations

Multiple observational studies have demonstrated that individuals with HT often exhibit significantly higher cortisol levels compared to healthy controls ($p<0.05$). Correlation analysis reveals a moderate positive relationship ($r\approx0.5–0.7$) between serum cortisol and anti-TPO antibody levels.

3. Symptomatology

Common clinical symptoms reported in HT patients include:

Symptom Prevalence (%)

Fatigue 60–80

Depressive symptoms 30–45

Weight changes 40–55

These symptoms have both endocrinological and neuropsychiatric components related to stress hormone dysregulation.

Clinical Implications and Therapeutic Approaches

1. Stress Monitoring

In clinical practice, assessment in HT patients may include:

- Circadian cortisol profiling
- Plasma catecholamine levels
- Standardized stress assessment tools (e.g., Perceived Stress Scale)

2. Stress-Reduction Strategies

Non-pharmacological interventions to reduce stress hormone levels include:

- Mindfulness meditation and breathing exercises
- Cognitive-behavioral therapy (CBT)
- Regular physical activity and structured sleep hygiene
- Dietary adjustments emphasizing omega-3 fatty acids and antioxidants

These approaches can help normalize cortisol levels and improve overall well-being.

Conclusion

There is compelling evidence that stress hormones play a significant role in the pathogenesis and clinical manifestations of Hashimoto thyroiditis. Chronic stress activates the HPA axis, leading to elevated cortisol and catecholamine levels, which in turn modulate immune responses and may exacerbate autoimmunity. Integrating stress monitoring and management into the clinical care of HT patients can improve disease outcomes and quality of life.

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