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Significance Of Tsh & Serum Cortisolin Patients Of Anxiety Disorder

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Abstract

Background: Anxiety disorders are among the most common psychiatric disorders and can significantly affect daily functioning and quality of life. Dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and alterations in thyroid function have been implicated in anxiety. Serum cortisol and thyroid-stimulating hormone (TSH) may therefore serve as useful biochemical markers associated with anxiety symptoms.

Aim: To evaluate the association of serum cortisol and TSH levels with anxiety symptoms.

Materials and Methods: A cross-sectional observational study was conducted in the Department of Biochemistry, Pacific Institute of Medical Sciences (PIMS), Umarda, Udaipur, Rajasthan. A total of 100 participants aged 18–60 years were included and divided into an anxiety group (n=50) and a control group (n=50). Anxiety status was assessed using a standardized anxiety assessment scale. Serum cortisol and TSH were estimated using standard immunoassay methods. Data were expressed as mean ± SD and appropriate statistical tests were applied. A p-value <0.05 was considered statistically significant.

Results: The mean age was comparable between the anxiety and control groups (34.6 ± 8.2 vs. 35.1 ± 7.9 years; p=0.75). Serum cortisol levels were significantly higher in the anxiety group compared with controls (18.6 ± 5.4 vs. 13.2 ± 4.1 µg/dL; p<0.001). Similarly, TSH levels were significantly higher in the anxiety group (3.42 ± 1.48 vs. 2.51 ± 1.12 µIU/mL; p=0.001). Anxiety scores showed a moderate positive correlation with serum cortisol (r=0.48, p<0.001) and a weak positive correlation with TSH (r=0.29, p=0.003).

Conclusion: The study suggests that anxiety symptoms are associated with increased serum cortisol and TSH levels. Alterations in HPA-axis activity and thyroid function may therefore contribute to the biological mechanisms underlying anxiety. Further longitudinal studies with larger sample sizes are required to establish causal relationships and determine the clinical significance of these biomarkers.

Keywords: Anxiety, Cortisol, Thyroid-stimulating hormone, HPA axis, Thyroid function, Biochemical markers.

Introduction

Anxiety disorders are a group of mental disorders characterized by persistent feelings of anxiety, fear, tension and discomfort. They include generalized anxiety disorder (GAD), panic disorder, phobias, social anxiety disorder, obsessive-compulsive disorder (OCD) and post-traumatic stress disorder (PTSD). Symptoms may range from mild to severe and are often chronic rather than episodic.[1] Anxiety disorders are among the most common psychiatric disorders worldwide and contribute substantially to disability.[2],[3]

Anxiety may occur as a normal response to stressful situations; however, when excessive anxiety persists and significantly interferes with daily functioning, it may constitute an anxiety disorder. Common

symptoms include palpitations, breathlessness, sweating, trembling, dizziness, nausea, abdominal discomfort, feelings of loss of control and impending doom. Anxiety disorders may occur at any age, are more common in women, and frequently begin during early adulthood.[4]

Anxiety disorders accounted for approximately 24.6 million years lived with disability (YLD) globally in 2015 and ranked among the major contributors to non-fatal health loss.[1] They are reported more frequently in females, with approximately 273 million people estimated to have an anxiety disorder globally in 2010.[5] In India, the National Mental Health Survey (2015–2016), conducted by NIMHANS, reported a prevalence of anxiety disorders of approximately 3.1% in the population.[6]

Cortisol

The hypothalamic-pituitary-adrenal (HPA) axis is a major component of the physiological stress-response system and plays an important role in maintaining homeostasis during physical and psychological stress. Corticotropin-releasing hormone (CRH), released from the hypothalamus, stimulates adrenocorticotropic hormone (ACTH) secretion from the pituitary, which subsequently promotes cortisol release from the adrenal cortex.[7],[8] Dysregulation of the HPA axis, either hypo- or hyperactivity, has been implicated in the development and maintenance of stress-related psychiatric disorders, including anxiety disorders.[9–11]

Cortisol follows a characteristic diurnal rhythm, with a rapid rise after awakening, known as the cortisol awakening response (CAR), followed by a gradual decline throughout the day. Cortisol levels are generally highest during the early morning and progressively decrease to low levels at bedtime. Alterations in the CAR and overall daily cortisol secretion have been associated with anxiety and other psychiatric disorders.[12–14]

Thyroid-Stimulating Hormone (TSH)

Thyroid hormones are essential for normal brain development and function, including neurogenesis, myelination, dendritic development and synapse formation. Stress can also influence the hypothalamic-pituitary-thyroid (HPT) axis.[15],[16]

The association between thyroid dysfunction and mood or anxiety disorders is well established. Patients with thyroid dysfunction have a higher prevalence of mood and anxiety disorders, and thyroid hormone receptors are present in brain regions involved in mood regulation.[17–19] Anxiety symptoms may occur in both hyperthyroidism and hypothyroidism, as well as in thyroiditis and other endocrine disorders. Thyroid dysfunction may therefore mimic or exacerbate primary anxiety disorders and may sometimes remain unrecognized.[20–22]

Differentiating anxiety secondary to thyroid dysfunction from primary anxiety disorder is clinically important because treatment directed only at anxiety may temporarily relieve symptoms while the underlying endocrine abnormality remains untreated, potentially leading to recurrence.[23] Anxiety is particularly relevant in young adults, in whom it can adversely affect academic performance, career development, social relationships and overall quality of life, even when symptoms are subclinical.[24]

Materials And Methods

Study Design and Setting

The present study was conducted as a cross-sectional observational study in the Department of Biochemistry, Pacific Institute of Medical Sciences (PIMS), Umarda, Udaipur, Rajasthan.

Study Population

The study included adult participants aged 18–60 years, recruited from the hospital and/or outpatient departments. Participants were evaluated for anxiety symptoms and relevant biochemical parameters.

Inclusion Criteria

Adults aged 18–60 years who provided written informed consent and were willing to participate in the study were included.

Exclusion Criteria

Participants with known thyroid disorders, Cushing's syndrome, major psychiatric illness other than anxiety, severe systemic illness, pregnancy, or those receiving corticosteroids or thyroid medications were excluded.

Study Parameters

Assessment included:

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1. Anxiety status using a standardized anxiety assessment scale.
2. Serum cortisol
3. Serum thyroid-stimulating hormone (TSH)

Sample Collection and Biochemical Analysis

After obtaining informed consent, approximately 3–5 mL of venous blood was collected under aseptic precautions. Serum was separated and analyzed for cortisol and TSH using standard laboratory methods on an automated immunoassay analyzer as per the manufacturer's instructions.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± SD, while categorical variables were expressed as frequencies and percentages. Comparison between groups was performed using appropriate statistical tests. A p-value <0.05 was considered statistically significant.

Result

A total of 100 participants were included in the study and divided into two groups: Anxiety group (n=50) and Control group (n=50). Serum cortisol and TSH levels were compared between the two groups.

Table 1. Comparison of demographic characteristics

Parameter	Anxiety group (n=50)	Control group (n=50)	p-value
Age (years)	34.6 ± 8.2	35.1 ± 7.9	0.75
Male, n (%)	22 (44%)	24 (48%)	0.69
Female, n (%)	28 (56%)	26 (52%)	0.69

There was no statistically significant difference in age or sex distribution between the two groups.

Table 2. Comparison of serum cortisol and TSH

Biochemical parameter	Anxiety group (n=50)	Control group (n=50)	p-value
Serum cortisol (µg/dL)	18.6 ± 5.4	13.2 ± 4.1	<0.001
Serum TSH (µIU/mL)	3.42 ± 1.48	2.51 ± 1.12	0.001

Serum cortisol levels were significantly higher in the anxiety group compared with controls (18.6 ± 5.4 vs. 13.2 ± 4.1 µg/dL, p<0.001). Similarly, TSH levels were significantly higher among participants with anxiety (3.42 ± 1.48 vs. 2.51 ± 1.12 µIU/mL, p=0.001).

Table 3. Correlation of anxiety score with biochemical parameters

Parameter	Correlation coefficient (r)	p-value
Cortisol	+0.48	<0.001
TSH	+0.29	0.003

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Anxiety scores showed a moderate positive correlation with serum cortisol (r=0.48, p<0.001) and a weak positive correlation with TSH (r=0.29, p=0.003).

Overall, the findings suggest that increased anxiety symptoms may be associated with alterations in HPA-axis activity, reflected by serum cortisol levels, and thyroid function, reflected by TSH levels.

Discussion

The present study evaluated the association of anxiety symptoms with serum cortisol and thyroid-stimulating hormone (TSH) levels. In the present study, 100 participants were included, comprising 50 participants with anxiety and 50 controls. The mean age of the anxiety group was 34.6 ± 8.2 years compared with 35.1 ± 7.9 years in controls, with no statistically significant difference between the groups. This indicates that the two groups were comparable with respect to age and sex distribution.

In the present study, serum cortisol levels were significantly higher in participants with anxiety compared with controls (18.6 ± 5.4 vs. 13.2 ± 4.1 µg/dL; p<0.001). This finding supports the involvement of the hypothalamic-pituitary-adrenal (HPA) axis in anxiety disorders. Cortisol is the principal glucocorticoid released during activation of the stress response, and persistent psychological stress may result in alterations of HPA-axis activity. A structured review by Vreeburg et al. reported alterations of HPA-axis function in different anxiety disorders, although the pattern was not completely consistent across studies.[25]

The positive correlation observed between anxiety score and serum cortisol in the present study (r=0.48, p<0.001) further suggests that increasing severity of anxiety may be associated with greater HPA-axis activation. Fischer and Cleare, in a systematic review and meta-analysis, highlighted the role of cortisol and HPA-axis alterations in anxiety disorders and suggested that cortisol abnormalities may have clinical relevance.[26] Similarly, a systematic review and meta-analysis of cortisol stress reactivity across psychiatric disorders demonstrated altered cortisol responses in psychiatric conditions, supporting the importance of HPA-axis dysregulation in psychological disorders.[27] However, cortisol findings in anxiety disorders have not been uniform, which may be related to differences in anxiety subtype, duration of illness, sampling time, medication use and methodological factors.[25]

The present study also demonstrated significantly higher TSH levels in the anxiety group compared with controls (3.42 ± 1.48 vs. 2.51 ± 1.12 µIU/mL; p=0.001). A positive correlation was also observed between anxiety score and TSH (r=0.29, p=0.003). These findings suggest a possible relationship between thyroid function and anxiety symptoms.

The association between thyroid dysfunction and anxiety is biologically plausible because thyroid hormones influence brain development and neuronal function. Alterations in thyroid function may produce symptoms such as nervousness, palpitations, irritability and emotional instability, which can resemble or aggravate anxiety disorders. A systematic review by Fischer and Ehlert found evidence of an association between anxiety disorders and alterations in HPT-axis functioning, although the findings across individual studies were heterogeneous.[28]

Previous clinical studies have also reported abnormalities in thyroid function among patients with anxiety disorders. Simon et al. reviewed hypothyroidism and hyperthyroidism in anxiety disorders and emphasized the clinical importance of considering thyroid dysfunction when evaluating patients presenting with anxiety symptoms.[29] More recent evidence also suggests an increased occurrence of anxiety symptoms in individuals with autoimmune thyroid disease, even when thyroid hormone levels are within the euthyroid range.[30]

The findings of the present study therefore suggest that both HPA-axis activity, reflected by serum cortisol, and thyroid function, reflected by TSH, may be associated with anxiety symptoms. The stronger correlation observed with cortisol compared with TSH indicates that stress-system activation may have a more direct relationship with anxiety severity in the studied population.

However, the results should be interpreted cautiously because the present study is cross-sectional and therefore cannot establish a causal relationship between anxiety and hormonal alterations. Cortisol levels are influenced by several factors, including time of blood collection, sleep, acute stress, medications and other physiological conditions. Similarly, TSH

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may be influenced by age, sex, nutritional status, medications and subclinical thyroid dysfunction.

Overall, the present findings support the potential usefulness of evaluating serum cortisol and TSH in individuals presenting with significant anxiety symptoms, particularly when symptoms are persistent, atypical or associated with other endocrine manifestations. Larger longitudinal studies with standardized anxiety assessment and repeated hormonal measurements are required to establish the temporal and causal relationship between anxiety, cortisol and thyroid function.

Summary

The present cross-sectional observational study was conducted in the Department of Biochemistry, Pacific Institute of Medical Sciences (PIMS), Umarda, Udaipur, to evaluate the association of anxiety symptoms with serum cortisol and thyroid-stimulating hormone (TSH) levels.

A total of 100 participants were included and divided into an anxiety group (n=50) and a control group (n=50). Anxiety status was assessed using a standardized anxiety assessment scale, while serum cortisol and TSH were estimated using standard laboratory methods.

The mean age and sex distribution were comparable between the two groups. Serum cortisol levels were significantly higher in the anxiety group compared with controls (18.6 ± 5.4 vs. 13.2 ± 4.1 µg/dL; $p<0.001$). Similarly, TSH levels were significantly higher in the anxiety group (3.42 ± 1.48 vs. 2.51 ± 1.12 µIU/mL; $p=0.001$).

Anxiety scores showed a moderate positive correlation with serum cortisol ($r=0.48$, $p<0.001$) and a weak positive correlation with TSH ($r=0.29$, $p=0.003$). These findings suggest that anxiety may be associated with alterations in both HPA-axis activity and thyroid function.

Overall, the study indicates that serum cortisol and TSH may have a potential role in understanding the biochemical alterations associated with anxiety symptoms. However, larger longitudinal studies are required to establish causal relationships and determine the clinical utility of these biomarkers.

Conclusion

The present study concludes that anxiety symptoms are associated with alterations in serum cortisol and TSH levels. Participants with anxiety showed significantly higher cortisol and TSH levels compared with controls. Serum cortisol demonstrated a moderate positive correlation, while TSH showed a weak positive correlation with anxiety severity.

These findings suggest that dysregulation of the HPA axis and alterations in thyroid function may contribute to the biological mechanisms underlying anxiety. Assessment of cortisol and TSH may therefore provide useful biochemical information in individuals presenting with significant anxiety symptoms. However, further large-scale and longitudinal studies are required to establish the causal relationship and clinical significance of these hormonal changes.

Ithical Issues:

Research project approved by the ethics committee of Pacific Institute of Medical Sciences Udaipur-313005, Rajasthan, INDIA

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