

ORIGINAL ARTICLE

The Impact of Group-Based Cognitive Hypnotherapy on Somatic Symptoms and Neuroendocrine Levels in Patients with Anxiety Disorder: A Randomized Clinical Trial

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ABSTRACT

Introduction: The prevalence of anxiety disorders is rising, negatively affecting quality of life. Cognitive Behavioral Therapy (CBT) is the most common psychological treatment but has limitations. Hypnosis, often overlooked, can enhance CBT by reducing psychological defenses and increasing plasticity. This study investigates the impact of group cognitive hypnotherapy (GCH) on somatic symptoms and neuroendocrine levels in patients with anxiety disorders. **Methods:** A randomized controlled trial was conducted with 84 participants meeting inclusion criteria. They were randomly assigned to an intervention group or control group using a computer-generated method. Both groups underwent baseline assessments, including the Hamilton Anxiety Scale (HAMA), Self-Rating Anxiety Scale (SAS), Somatization Symptom Checklist (SSS), and blood tests measuring cortisol (COR), triiodothyronine (T3), thyroxine (T4), thyroid-stimulating hormone (TSH), and adrenocorticotropic hormone (ACTH). The intervention group received weekly GCH for six weeks, while the control group received standard care. Post-treatment, both groups were reassessed using the same measures. **Result:** This study indicated significant improvements in the intervention group: HAMA scores decreased from 24.79 ± 5.48 to 7.52 ± 3.194 , SAS from 59.07 ± 3.047 to 28.14 ± 3.00 , and SSS from 56.12 ± 3.262 to 27.50 ± 1.824 . Blood levels of COR (581.66 ± 79.24 vs 343.05 ± 50.38 nmol/L), ACTH (14.37 ± 1.48 vs 12.67 ± 0.78 pmol/L), TSH (3.54 ± 0.66 vs 2.84 ± 0.76 mIU/L), T3 (1.23 ± 0.82 vs 0.94 ± 0.42 ng/ml), and T4 (8.33 ± 0.71 vs 6.74 ± 0.64 ng/ml) all decreased significantly ($P < 0.05$). The control group also showed significant reductions in HAMA and SAS scores ($P < 0.05$). **Conclusion:** GCH effectively alleviates somatic symptoms and positively influences neuroendocrine levels in anxiety disorder patients, supporting its use in clinical practice.

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INTRODUCTION

Nowadays, the pace of life is uncontrollable and accelerating; competition is becoming more intense, and personal emotional imbalance is becoming more evident, which has led to a yearly increase in anxiety

disorders(1). Bareeqa (2) surveyed the prevalence of emotional disorders in China during the pandemic and found that anxiety disorders had risen to 21.8%, with 48.1% feeling stressed due to COVID-19. Compared to studies from different countries, China's prevalence of anxiety is much higher than in India, Singapore, and other countries (3). According to a study by Parker et al. (4), when Chinese people face anxiety and stress, it is often denied and usually manifests physically. Dong and Ronjuan (5) reported that nearly half of Chinese patients with anxiety disorders primarily present with somatic symptoms when visiting outpatient clinics,

a phenomenon known as “Chinese somatization.” This could be related to our cultural and customary backgrounds as Chinese people are accustomed to focusing on physical symptoms and rarely discussing psychological feelings (6). As anxiety disorders are increasingly recognized (7), many scholars have begun to pay attention to physiological changes in addition to psychological characteristics. The hypothalamic-pituitary-adrenal (HPA) axis and the hypothalamic-pituitary-thyroid (HPT) axis are the most studied. Steimer (8) found that hormones of the HPA axis, such as cortisol (COR) and adrenocorticotrophic hormone (ACTH), typically increase in situations of anxiety and fear. Hilbert (9) also suggested that cortisol is associated with negative emotions, especially hypercortisolism, as an indicator of HPA axis activity changes in generalized anxiety disorder (GAD). Unfortunately, the results are inconsistent, whereby some studies suggest that in stressful situations, the function of the hypothalamic-pituitary-adrenal cortex (HAP) in patients with anxiety disorders increases and patients’ plasma cortisol levels significantly increase (10). Whereas other studies suggest no significant changes in cortisol levels (11, 12). Some people think patients’ cortisol levels increase in the afternoon (13), but not in the morning (14).

Regarding HPT research, Fischer and Ehlert (15) found that thyroid disease is significantly comorbid with anxiety and the symptoms associated with anxiety are prevalent in hyperthyroidism. Chinese scholars (16) found that thyroid hormone levels well reflect the functional status of the HPT axis. There is a close relationship between anxiety disorders and thyroid function. Thyroid hormone disorders can affect patients’ brain tissue, thus leading to abnormalities in neurological and mental states (17). The relationship between anxiety disorders and the level of neuroendocrine function in patients with anxiety disorders is still a topic of much debate and requires further exploration.

Over the past two decades, researchers have extensively researched cognitive-behavioral therapy for various anxiety disorders, proving that cognitive-behavioral therapy and hypnosis can effectively treat various anxiety disorders (15). However, no therapy is perfect, and each will have some deficiencies in certain aspects (18). Although hypnosis was initially used as an independent psychological treatment method in clinical treatment, hypnotherapy lacks the core theoretical basis of psychotherapy and behavior change. It includes numerous techniques, making it unlikely to become a mainstream psychological treatment method (1). Cognitive-behavioral therapy (CBT) has a relatively complete theoretical basis and related treatment techniques. However, it cannot directly restructure the negative beliefs in the patient’s subconscious and mainly achieves the purpose of cognitive restructuring through reasoning (19). Therefore, Alladin proposed cognitive hypnotherapy, which adds hypnotherapy to

cognitive-behavioral therapy by focusing on individual thinking and behaviors in specific situations and enhancing the therapeutic effect while overcoming emotional and behavioral problems (20). It can pay attention to both the conscious and unconscious realms of the patient and directly restructure the patient’s cognition at both conscious and unconscious levels. In a study intervening in pain, Jensen (21) found that using cognitive hypnotherapy can effectively reduce pain intensity by 39%. Baharvand et al (22) in their research also found that GCH is better than CBT and eye movement desensitization and reprocessing (EMDR) in relieving women’s anxiety levels and improving autobiographical memory, thus making it more positive. Ramondo (23) found in meta-analysis of cognitive hypnotherapy that many pieces of literature indicate that cognitive hypnotherapy is more effective than cognitive-behavioral therapy alone. However, there are currently no reports in China on using cognitive hypnotherapy to alleviate the somatic symptoms of patients with anxiety disorders and improve their neuroendocrine levels through group therapy.

This study used a randomized control method to compare the changes in somatic symptoms and neuroendocrine levels of the two groups of patients (control and intervention) after GCH. The aim is to explore the impact of GCH on the somatic symptoms and neuroendocrine levels of patients with anxiety disorders.

MATERIALS AND METHODS

Study design and setting

This study employed a randomized control trial design. The study population consisted of patients diagnosed with anxiety disorders receiving treatment from the Department of Medical Psychology, Affiliated Brain Hospital of Nanjing Medical University. This study has also been registered with ISRCTN (ISRCTN45667531). The inclusion and exclusion criteria of the patients are shown in Table I.

This study was approved by the Ethics Committee of Universiti Sains Malaysia (Approval number: USM/JEPeM/22120758) and the Institutional Review Board (IRB) of Nanjing Brain Hospital and Nanjing Medical University (Approval number: NMUB20210325). This study has also been registered with ISRCTN; the number is ISRCTN45667531. Informed consent was obtained from all participants. Patients admitted to the Department of Psychology at Nanjing Brain Hospital from October 2022 to March 2023 who met the DSM-5 diagnostic criteria for anxiety disorders were invited to participate in this study.

This study followed a structured process involving six core steps: determining the topic, forming the team, providing systematic training, developing an

Table 1: Inclusion and Exclusion Criteria for Participants.

Inclusion criteria	<ol style="list-style-type: none"> 1. ICD-10 diagnosis of generalized anxiety disorder, social anxiety disorder and panic disorder 2. Diagnose with anxiety disorder lasting more than 3 months but not more than 3 years 3. Ability to express and understand simple Chinese characters 4. Give informed consent and voluntary participation in the study
Exclusion criteria	<ol style="list-style-type: none"> 1. Patients with other co-morbid psychiatric disorders (e.g., depression bipolar disorder) in combination, or patients at risk for suicide and self-injury 2. Patients with thyroid-related disease 3. Patients with serious cardiovascular diseases such as severe hypertension, diabetes, and hyperlipidemia 4. Patients who have had substance dependence or abuse, such as alcohol and drug abuse and drug dependence. 5. Pregnant women

intervention manual, implementing the intervention, and analyzing the data. First, we focused on determining the topic by assessing the current status of anxiety disorder patients both domestically and internationally. We used questionnaires to evaluate their anxiety levels and the severity and frequency of their physical symptoms. Additionally, blood samples were collected to analyze their neuroendocrine levels. Second, we formed a specialized CBH team, including a psychological department doctor as an expert supervisor, a head nurse, three nurses from the medical psychology department, and five statisticians.

Third, the team received comprehensive training in GCH from an expert in cognitive behavioral therapy and hypnotherapy. This training consisted of six systematic sessions to ensure all members had a consistent and thorough understanding of GCH principles. Their knowledge was evaluated at the end of the training to confirm uniform competency. Fourth, we collaboratively developed a standardized GCH intervention manual. This manual was designed to guide the treatment process effectively and consistently for patients with anxiety disorders.

Fifth, we implemented the intervention, conducting weekly sessions of GCH over six weeks, with each session lasting 1.5 hours. During this phase, the team received regular group supervision from an expert to ensure the treatment's homogeneity and safety. Finally, the sixth step involved organizing and analyzing the data collected during the intervention to assess its effectiveness. This comprehensive, step-by-step approach ensured a rigorous evaluation of group cognitive hypnotherapy's impact on patients with anxiety disorders.

Sampling method

Sample size calculation for this study was based on the formula for t-test.

$$N = 2 \left(\frac{t_{\alpha/2} + t_{\beta}}{\delta / \sigma} \right)^2$$

In this study, standard deviation (σ) was estimated, and the mean difference (δ) of two continuous variables was calculated based on the change in the number of qualified samples. The test level was set at a two-sided $\alpha = 0.05$ and a type II error probability $\beta = 0.2$ (one-sided), where $t_{\alpha/2} = 1.96$ and $t_{\beta} = 0.84$. According to the experimental results of the preliminary trial, σ was calculated to be 3.14 and δ was 2.017. After considering a possible 10% dropout, the final sample size for the experimental and control groups was determined to be 42 cases each.

This study used a computer-generated randomization method (Random Allocation Software, version 1.0) to generate random numbers and allocate participants to different groups based on these numbers. This ensures the impartiality and randomness of the assignment to the intervention and control groups, eliminating potential systematic biases in the study. By assigning a unique code to each participant and associating it with the generated random number, fair and random allocation to intervention and control groups can be effectively achieved. This method helps ensure that groups have similar characteristics at the start of the experiment, thereby enhancing the internal validity of the study.

The contamination of the control and experimental groups was controlled using three methods. Firstly, the intervention activities for the experimental and control groups were set in different locations and periods to avoid any exchange of information between participants from the two groups. Secondly, confidentiality agreements were signed before the intervention began, and participants were asked not to discuss the intervention during the process, thereby emphasizing the importance of maintaining confidentiality. Lastly, the intervention process was monitored regularly by the researcher to ensure adherence to the predefined intervention plan and to prevent any influence between the groups. Randomized interviews were conducted to check if participants adhered to the confidentiality agreement.

To minimize bias and control for information contamination, a blinding procedure was implemented. Upon enrollment, participants were informed that they would be part of a study, but the specific details of their group (experimental or control) were kept confidential. Standardized instructions were given to all participants to ensure a consistent understanding of the study. The data collection and intervention implementation was

carried out by different members of the research team. During data analysis, the researcher responsible was kept unaware of the participants' group assignments. To ensure this, data were anonymized before analysis so that analysts were only exposed to data without group identification. The summary of the data collection process is depicted in Figure 1.

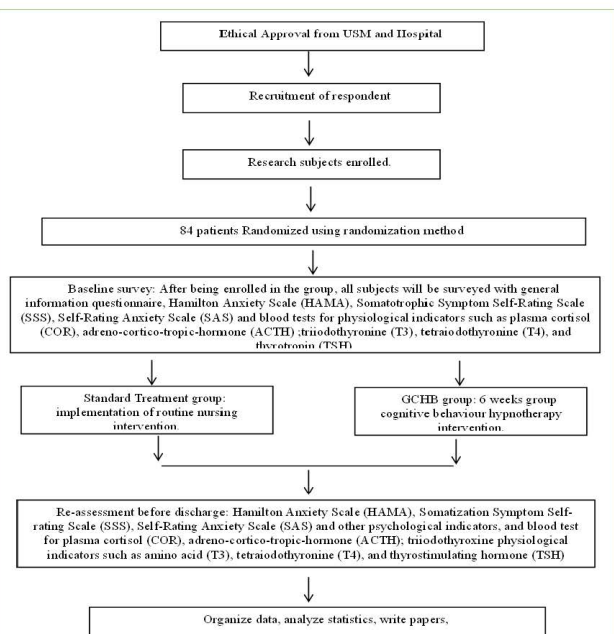


Figure 1: Study flowchart

Study Instruments & Data Analysis

This study used a sociodemographic survey questionnaire that includes the Hamilton Anxiety Scale (HAMA), Self-Rating Anxiety Scale (SAS), and Somatization Symptom Checklist (SSS) to measure the severity of symptoms and somatization symptoms in patients with anxiety disorders (24–26). The HAMA scale was developed by Hamilton and translated by Chinese scholar Yuhua Tang (24, 27). The HAMA score is 0–4 and is divided into five levels: (0) no symptoms, (1) mild, (2) moderate, (3) severe, and (4) very severe. Shanghai Mental Health Center tested the reliability and validity of the scale and the results showed that the reliability coefficient r was 0.93, while the reliability coefficient of each symptom score was 0.83–1.00. The validity coefficient was 0.36 ($P < 0.05$). The cut-off points of HAMA are as follows: more than 29 points is severe anxiety; more than 21 points is obvious anxiety; more than 14 points is must be anxiety; more than 7 points is anxiety; and less than 6 points is no anxiety symptoms. (24).

The SAS was used as a self-rating scale for participants to measure their own level of anxiety. It was developed by Zung and translated by a Chinese scholar Wu Wenyuan (25). The SAS has 20 items, and the main rating is based on the frequency of the symptoms defined by the items with four levels: a score of “1” indicates no or little time; a score of “2”. The main statistical indicator of the SAS was the total score. The correlation coefficient

of the total scores of the scale was 0.365 and 0.341, indicating good validity of the SAS. The cut-off value of the SAS standard score was 50 according to the Chinese frequent touch results. The score levels are described as follows: 1- the normal state with a standard score below 50; 2- mild anxiety with a standard score of 50–59; 3- moderate anxiety with a standard score of 60–69; and 4- severe anxiety with a standard score of 70 or more.(25). The SSS scale was developed by Chinese scholars Zhuang Qi et al. to assess somatic symptoms (26). This scale has 20 items including 9 somatization symptom factors, 5 anxiety symptom factors, 4 depressive symptom factors, and 2 anxiety-depression factors. The scale is scored on a 4-point scale with no reverse scoring for each item where a score of “1” indicates no symptoms, “2” indicates a moderate degree, “3” indicates a reasonable degree. The Cronbach’s coefficient for the SSS scale is 0.89, indicating good internal consistency. The retest reliability is 0.96, and the testability of each subscale ranges from 0.87 to 0.88, indicating that the scale has good stability. The correlation coefficients between the factors and the total score range from 0.76 to 0.88, and the correlation coefficients between the factors of each scale range from 0.56 to 0.70, indicating that the scale has good structural validity. The scale’s total score was 80, with scores ≤ 29 basically normal, mild 30–39, moderate 40–59, and severe ≥ 60 (26).

For physiological indicators, 5 ml of fasting venous blood is collected from the experimental and control groups at 8 a.m, the day before and the second day after the end of the intervention. Venous blood is centrifuged for 10 minutes in a 4000 (r/min) centrifuge, and the serum is extracted after centrifugation. The levels of cortisol (COR), serum triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) in the plasma of all participants are measured by radioimmunoassay.

This study utilized Microsoft Excel 2007 spreadsheet software to organize the data and SPSS 26.0 (Chinese version) to analyze data. The level of significance is set as p -value < 0.05 . All data were analyzed using an independent sample t -test. Q-Q plots and t -tests were used to ensure the normality of data distribution and the success of randomization. Numerical variables were normally distributed and were analyzed using t -test to examine the differences in treatment response for symptom severity (HAMA, SAS) and somatization symptoms (SSS) based on group (GCH, ST), time (pre-treatment, post-treatment), and the interaction between group and time.

RESULTS

The study recruited 84 patients: 26 males (31%) and 58 females (69%). During the six-week cognitive and hypnotherapy intervention period, the dropout rate was 0%. The 42 patients in the experimental group received GCH interventions in stages over 6 weeks, once a

week, with each group consisting of approximately 6-8 people and each session lasting around 45-60 minutes. The experimental group consisted of 12 males (28.6%) and 30 females (71.4%) with an average age of 45.86 ± 8.78 years old (range: 29-59 years old). The control group consisted of 14 males (33.33%) and 28 females (66.67%) with an average age of 44.57 ± 9.58 years old (range: 20-60 years old).

In terms of anxiety levels and somatic symptoms, no significant differences between the two groups before treatment ($P > 0.05$) indicate similar characteristics between the control and experimental groups (Table II). After the intervention, the total scores of HAMA, SSS, and SAS in the experimental group were significantly reduced compared to before treatment ($P < 0.05$), and the full scores of HAMA, and SAS in the control group were significantly decreased. After treatment, the scores of HAMA, SSS, and SAS in the experimental group were significantly lower than those in the control group ($P < 0.05$) (Table II).

Table II: Comparison HAMA, SSS, SAS, COR, ACTH, TSH, T3 and T4 scores before treatment in both groups

Variable	Experimental Group (GCH) (mean \pm SD)	Control Group (mean \pm SD)	t	P value ^a
Hamilton Anxiety Scale (HAMA)	24.79 \pm 5.48	26.88 \pm 4.76	-1.805	0.065
Somatization Symptom Checklist (SSS)	56.12 \pm 3.26	56.43 \pm 3.91	-2.149	0.695
Self-Rating Anxiety Scale (SAS)	59.07 \pm 3.48	60.57 \pm 3.34	-0.394	0.055
Cor (nom/L)	581.65 \pm 78.241	580.58 \pm 49.653	0.074	0.941
ACTH (pmol/L)	14.37 \pm 1.481	14.39 \pm 0.698	-0.075	0.940
TSH (mIU/L)	3.53 \pm 0.657	3.49 \pm 0.356	0.347	0.730
T3 (ng/ml)	1.22 \pm 0.081	1.23 \pm 0.079	-0.297	0.767
T4 (ng/ml)	8.32 \pm 0.711	8.29 \pm 0.516	0.263	0.793

^a Independent t-test, P is significant at < 0.05

There were no significant differences between the experimental group and the control group in the indicators of COR, ACTH, TSH, T3, and T4 before the intervention. (Table II). After the intervention, the levels of COR, ACTH, TSH, T3, and T4 in the experimental group were significantly reduced compared to before the intervention ($P < 0.05$). The levels of COR and ACTH in the control group also decreased after 6 weeks ($P < 0.05$), but there were no statistically significant changes in TSH, T3, and T4 in the control group after the 6 weeks (Table III).

DISCUSSION

This study aimed to evaluate the effect of GCH on the somatic symptoms and neuroendocrine levels of patients with anxiety disorders. The results show that group-based cognitive hypnotherapy can improve the somatic symptoms and neuroendocrine levels of patients with anxiety disorders. Our findings showed among the experimental group's HAMA, SAS, and SSS scores

Table III: Comparison of HAMA, SSS, SAS, COR, ACTH, TSH, T3 and T4 results within groups

Projects	Group	Pre-intervention	Post-intervention	Mean difference (95% CI)	t	p value
Hamilton Anxiety Scale (HAMA)	Experimental Group (CBH)	24.79 \pm 5.48	7.52 \pm 3.20	17.26 (15.55, 18.75)	20.39	< 0.01
	Control Group	26.88 \pm 4.76	19.21 \pm 2.46	7.66 (6.07, 9.27)	9.68	< 0.01
Somatization Symptom Checklist (SSS)	Experimental Group (CBH)	56.12 \pm 3.26	27.50 \pm 1.83	28.61 (27.38, 29.86)	46.66	< 0.01
	Control Group	56.43 \pm 3.91	55.26 \pm 4.31	1.16 (2.35, 1.99)	30.41	0.054
Self-Rating Anxiety Scale (SAS)	Experimental Group (CBH)	59.07 \pm 3.48	28.14 \pm 3.00	30.92 (29.66, 32.19)	42.02	< 0.01
	Control Group	60.57 \pm 3.34	38.55 \pm 3.12	22.02 (20.56, 23.49)	1.99	< 0.01
ACTH (pmol/L)	Experimental Group (CBH)	14.37 \pm 1.48	12.67 \pm 0.78	1.71 (1.14, 2.27)	6.10	< 0.01
	Control Group	14.39 \pm 0.70	14.09 \pm 0.60	0.30 (-8.98, -5.76)	5.09	< 0.01
Cor (nom/L)	Experimental Group (CBH)	581.66 \pm 79.24	343.05 \pm 50.38	238.60 (212.15, 265.06)	18.21	< 0.01
	Control Group	580.53 \pm 49.65	561.68 \pm 16.30	18.91 (2.09, 35.73)	2.27	0.769
TSH (mIU/L)	Experimental Group (CBH)	3.54 \pm 0.66	2.84 \pm 0.76	0.69 (0.41, 0.98)	4.94	< 0.01
	Control Group	3.50 \pm 0.36	3.38 \pm 0.39	0.11 (1.76, 4.90)	1.99	> 0.05
T3 (ng/ml)	Experimental Group (CBH)	1.23 \pm 0.82	0.94 \pm 0.42	0.29 (0.26, 0.32)	4.94	< 0.01
	Control Group	1.24 \pm 0.80	1.21 \pm 0.54	0.02 (4.15, 7.41)	1.72	> 0.05
T4 (ng/ml)	Experimental Group (CBH)	8.33 \pm 0.71	6.74 \pm 0.64	1.59 (1.26, 1.93)	9.66	< 0.01
	Control Group	8.29 \pm 0.52	8.25 \pm 0.60	0.04 (-2.89, 0.37)	0.38	> 0.05

^a Paired t-test, P is significant at < 0.05

decreased significantly after the cognitive hypnotherapy intervention. This result is consistent with previous studies (28-30). Dugas (31) proposed the anxiety uncertainty model, which pointed out that patients with anxiety disorders tend to worry and produce negative cognitive bias when the results are uncertain. The etiological model of anxiety proposed by Wolfe

(32,33) also believes that anxiety disorders are related to the patient's long-term struggle with subjective experiences. The patient's anxious experience in specific situations can lead to catastrophic expectations of what is about to happen, which represents the fear of exposing the intolerable self-pain perspective at the unconscious level. This etiological theory of anxiety disorders includes both conscious and implicit or unconscious levels for patients with anxiety disorders. According to this model, it is believed that anxious emotions are usually accompanied by thoughts of potential disasters facing the self in the future (32). From this practical perspective, anxiety represents a future-oriented emotion that prevents individuals from focusing on their current emotional experience of the world (33). In anxiety disorders, thoughts about symptoms, stimuli, and situations that trigger anxiety and self-harm seem similar to negative self-hypnosis (34). Therefore, this study integrates hypnosis techniques based on cognitive-behavioral therapy and intervenes with patients in a group setting.

Firstly, in a group atmosphere where patients feel a greater sense of belonging and discover that their problems are not individual but universal, thus reducing their sense of self-harm and in a group where patients become more self-aware. When they listen to what others in the group share, they better understand their problems and motivations. Secondly, through the introduction technique of hypnosis, anxiety patients unconsciously focus their attention better on the present moment, free themselves from anxious states and situations, and apply problem-solving strategies in conjunction with CBT in the trance state to repair their catastrophic and painful perceptions and feelings, correct their negative automatized thinking and behavior, and enhance their self-esteem, thus achieving anxiety reduction (30). On the other hand, hypnosis can trigger a relaxation response; anxiety patients are neurologically excited when they are agitated and produce various somatic symptoms such as muscle tension, palpating sensations, dyspnea, and insomnia. Hypnosis can relieve patients' tense nerves and muscles and promote relaxation through stabilization techniques, paradoxical suggestion, and positive intention (29). When the patient is completely relaxed, positive subjective experiences replace negative perceptions, thus relieving anxiety.

In terms of neuroendocrine levels among the experimental group, the result of COR, ACTH, TSH, T3, and T4 indicators decreased significantly and is consistent with previous (8, 9, 14) studies. Borkovec (35) proposed a neurobiological model of anxiety disorders whereby anxiety patients overactive the central and amygdala when anticipating worries, thus leading to overactive autonomic nerves and cortisol secretion. However, as emotional regulators, these patients' vIPFC and ACC are interfered with, and personal concerns seem uncontrollable, therefore, overactive autonomic

nerves and cortisol secretion become long-term on a biological level. Due to the long-term increase in cortisol levels, serotonin uptake increases until it reaches its peak, leading to the emotional changes observed in this disease. In addition, increased cortisol secretion seems to reduce the functional connection between the amygdala and PFC and the hippocampus volume, which further increases anxiety and reduces the ability to regulate emotions. Hilbert (9) and Steimer (8) found that hormones of the HPA axis such as cortisol or corticosterone (in rodents), ACTH, and CRF usually increase in states of fear and anxiety. They also seem to regulate responses to threatening events.

Another study (15) also found that people with thyroid disease are more likely to have accompanying anxiety disorders (36)). However, according to Sareen et al (37) and Witthauer et al (38), using the age of onset data, in most cases, anxiety disorders precede the onset of thyroid diseases. This suggests that over time, subtle changes in the HPT axis in patients with anxiety disorders may develop into subclinical and/or overt thyroid disease disorders. Based on these findings, when we apply cognitive restructuring to hypnotherapy, we help patients understand their negative self-suggestions and automatic thinking while relaxing them. This activates the parasympathetic nervous system of the patient's autonomic nervous system, thus reducing the physiological response of the sympathetic nervous system, and regulates the HPA axis to balance the autonomic nervous system, thereby reducing somatic symptoms and relieving anxiety (30).

In this study, the control group showed a significant decrease in HAMA and SAS anxiety scores without any intervention. This was because they were still taking regular anti-anxiety medications, mainly benzodiazepines, including clonazepam, alprazolam, eszopiclone, and lorazepam. According to the patient's conditions, doctors set the time for them to take the medication, and under the monitoring of nurses, the patient was seen to take it down to ensure that the patient's medication rate was 100%. Therefore, we cannot deny the effectiveness of the medication, which alleviates the symptoms of anxiety patients to some extent. However, after the intervention, compared with the experimental group, the somatic symptoms of the patients in the control group did not see any significant changes, this is because group cognitive behavioral hypnotherapy is a comprehensive treatment method that combines cognitive therapy and hypnotherapy. It not only helps patients identify and change unhealthy thinking patterns and behavioral habits, but also helps them relax their bodies, instructs them to learn how to relax during an anxiety attack, and gives them positive suggestions when necessary to alleviate the various somatic symptoms they exhibit. Therefore, compared with the control group, the experimental group that received cognitive hypnotherapy had significantly lower levels of anxiety,

somatic symptoms, and neuroendocrine levels. In summary, we should consider diversifying the treatment of patients with anxiety disorders. Although single medication is effective, comprehensive psychotherapy is more effective.

This study has several notable strengths and limitations. Unlike previous research that primarily relied on self-assessment scales, this study ensured greater objectivity by using a combination of researcher-filled and patient-filled assessments. The Hamilton Anxiety Scale (HAMA) was administered by the researcher, while participants completed the Somatic Symptom Scale (SSS) and the Self-assessment of Anxiety (SAS). Furthermore, blood tests measuring cortisol (COR), adrenocorticotropic hormone (ACTH), thyroid-stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4) were conducted on all 84 patients, providing a comprehensive and objective set of data. This approach helped to mitigate the potential biases associated with subjective evaluations. However, the study also faced certain limitations. It was conducted in a single hospital, which may not be representative of treatment levels and plans in other hospitals. Additionally, the study lacked detailed stratification by gender and age, which are significant factors influencing hormone secretion and its variability. The absence of follow-up investigations further limits the ability to assess the long-term effects and durability of the intervention's outcomes. For future research, it is recommended to include larger samples and diverse control groups to better understand the impact of anxiety on physical symptoms and neuroendocrine levels across different genders and age groups

CONCLUSION AND IMPLICATION

This study has demonstrated that Group Cognitive Hypnotherapy (GCH) significantly improves the physical symptoms and neuroendocrine levels in patients with anxiety disorders. The broader adoption and integration of GCH in psychiatric services could enhance patient outcomes in clinical practice. By effectively addressing anxiety symptoms, GCH facilitates quicker recovery times, potentially reducing the overall economic burden on patients. This reduction in economic strain also extends to patients' families, alleviating their psychological burden and fostering better family dynamics and social harmony.

The implementation of GCH presents a cost-effective therapeutic option within psychiatric services. Its structured group format allows for the treatment of multiple patients simultaneously, optimizing resource utilization and reducing per-patient costs compared to traditional one-on-one therapy sessions. This makes GCH a viable and sustainable intervention in diverse healthcare settings.

Additionally, the results contribute to the development of a new evidence-based psychological nursing intervention model that extends beyond anxiety disorders. GCH can be applied to patients with various somatic diseases that are compounded by anxiety, providing innovative clinical care strategies. Understanding the impact of GCH on neuroendocrine function enhances our knowledge of the physiological mechanisms underlying anxiety disorders. This insight not only clarifies the relationship between anxiety and the neuroendocrine system but also guides more effective and individualized treatment strategies.

The ability to monitor and adjust treatment based on changes in patients' neuroendocrine levels allows for personalized care that aligns with each patient's unique characteristics and needs, thereby improving treatment outcomes. GCH's effectiveness in addressing both the physical and psychological aspects of anxiety paves the way for comprehensive, multidimensional treatment plans. This holistic approach ensures that patients receive care that meets their overall well-being, fostering better recovery and long-term health outcomes.

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