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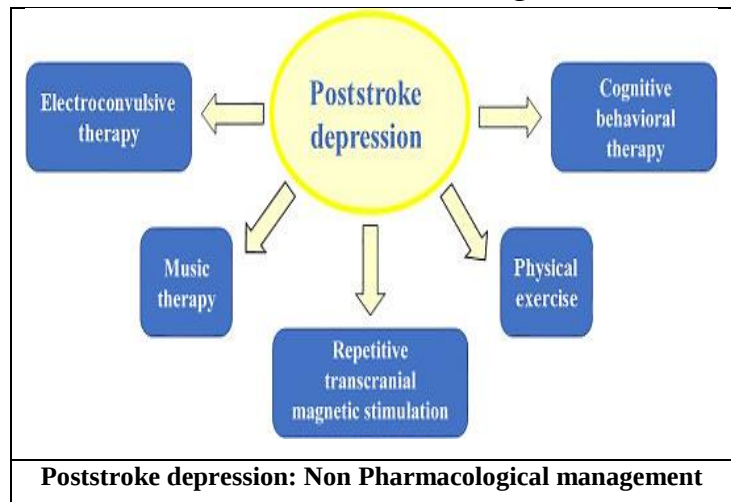
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1. Correlation between post-stroke depression and thyroid hormone levels during the acute phase of a stroke

Introduction

Post-stroke depression (PSD), which often occurs in the weeks and months following an acute stroke, is a typical neuropsychiatric outcome. The etiology of PSD was multifactorial and involved a variety of pathophysiological abnormalities in addition to psychosocial factors. These abnormalities include reduced levels of monoamines, increased inflammation, altered neurotrophic response, and dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis. Depression has also been shown to be influenced by inflammation in the brain.¹ The formation of the brain and the maintenance of brain function throughout life were significantly influenced by thyroid hormones. The pituitary gland's secretion of thyroid-stimulating hormone (TSH) controls the production and release of triiodothyronine (T3), a bioactive hormone, and thyroxine (T4), the prohormone produced by the thyroid gland. A fraction of T4 in the bloodstream was converted to T3 by a process known as deiodination. In the peripheral blood, T4 and T3 exist in both bound and free forms. The bound form became biologically inert when it connected to proteins, but the free form was also active since it entered relevant bodily tissues and performed biological tasks. At least during the acute period, a stroke may contribute to alterations in thyroid function in non-thyroidal disease. Thyroid hormones have been linked to depression, although their relationship with post-stroke depression remains unclear.² So, the purpose of this meta-analysis was to investigate the relationship between thyroid hormone levels in acute stroke and PSD.



Methods

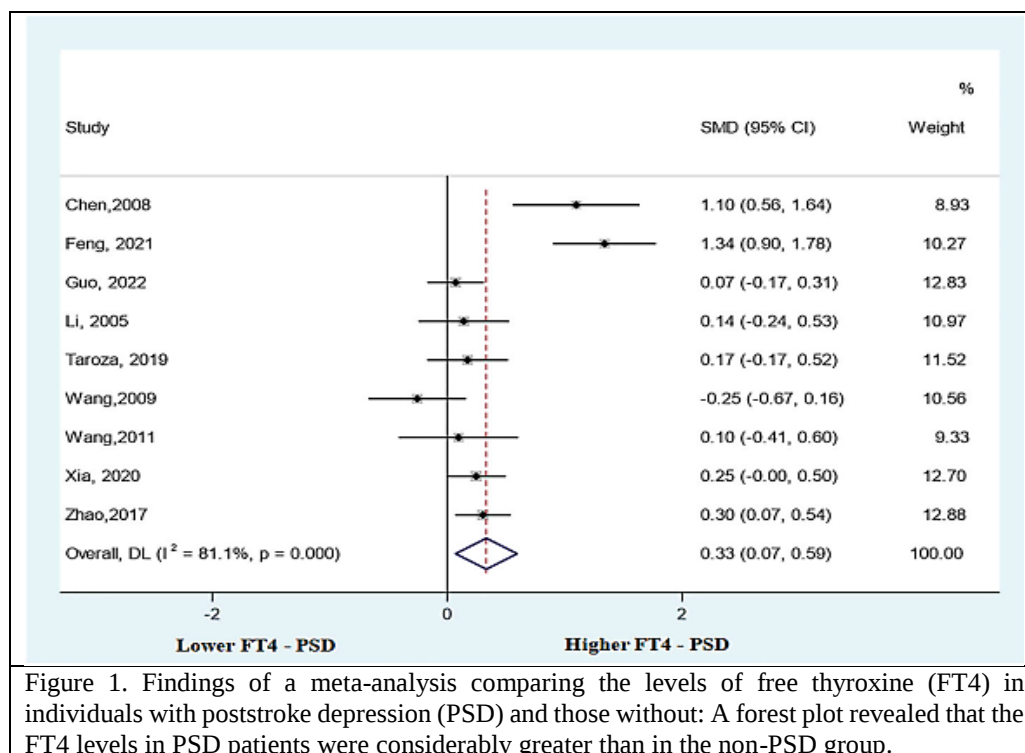
After searching through databases for studies, 13 studies were found and included in this meta-analysis. The relationship between thyroid hormone levels, such as free triiodothyronine (FT3), free



thyroxine (FT4), and thyroid-stimulating hormone (TSH), in acute stroke patients and the risk of PSD was assessed using standard mean differences (SMD) and 95% confidence intervals (CI). A quality assessment of the study was conducted using the Newcastle-Ottawa scale (NOS) criteria. The NOS assessed the study's quality based on three criteria: outcome or exposure, comparability, and selection. A study was considered good quality if it had a total score of at least five out of nine possible points.

Results

The serum levels of TSH and FT3 (TSH: SMD = -0.59 , FT3: SMD = -0.40) and FT4 (SMD = 0.33) were significantly lower in PSD patients than in non-PSD patients (Figure 1). To evaluate the impact of every study on overall SMD, a sensitivity analysis was carried out. The results of this analysis were stable, as evidenced by the fact that the aggregate SMD did not significantly change when any study was excluded. Compared to TSH and FT4, subgroup analysis suggested that there may be a stronger statistically significant correlation between FT3 and the risk of PSD. A meta-regression analysis revealed that age and gender did not account for study heterogeneity.





Discussion

According to the current meta-analysis, stroke patients with PSD showed lower TSH and FT3 levels, as well as greater FT4 levels, in comparison to patients without PSD. The findings provided evidence that blood thyroid hormone levels could be used as possible indicators to forecast a stroke survivor's probability of developing PSD.² The current study's PSD prevalence of 34.68% was comparable to that of Chen et al.³ The significant levels of variability were not fully explained by the subgroup analyses that were currently conducted based on the kind of stroke, study location, and timing of depression evaluation.² Karaahmet OZ et al. revealed age and gender have also been found to be important determinants of PSD risk.⁴

Conclusion

The current study concluded that patients in the acute stage of stroke who have higher blood FT4 levels and lower serum TSH and FT3 levels may be more prone to PSD.

Post-stroke depression (PSD) in stroke survivors was probably predicted via thyroid hormones, the most commonly investigated endocrine index.

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2. Investigation of the relationship between depression, anxiety, and quality of life with chest pain origin

Introduction

Chest pain is one of the most prevalent reasons for admitting patients to emergency medical and cardiovascular clinics. Both non-cardiac and cardiac (coronary or non-coronary) conditions can be the cause of chest pain. A new or untreated medical or psychological issue was indicated when a patient with known coronary artery disease experiences chest pain.¹ Patients who have persistent and recurrent chest pain often seek medical attention for non-cardiac chest pain (NCCP), which is a common condition in clinical cardiology. Many individuals have their chest pain diagnosed as having no cardiac etiology, and they are discharged without a definitive diagnosis. Psychological distress, mainly anxiety, despair, and terror, has been linked to NCCP. Furthermore, compared to cardiac patients, patients with NCCP report comparable or greater levels of psychological distress. Numerous investigations have verified the adverse effect psychological distress has on patients' health-related quality of life (HRQoL).² There were some evidence suggesting that people experiencing chest pain frequently have mental health issues. Patients with chest discomfort frequently have anxiety disorders (including panic disorder and phobia), sleep issues, depressive disorders, low quality of life (QOL), somatization, and alexithymia. In addition to being linked to NCCP, psychological issues, including anxiety and depression, also stand alone as risk factors for coronary artery disease (CAD). Many times, psychological variables were disregarded as possible causes of cardiovascular disease.¹ The purpose of this study was to look at the connection between quality of life, anxiety, and depression and the cause of chest pain.

Methods

In total, 210 participants were included in this cross-sectional study. The subjects were split up into three groups: non-cardiac origin (NCCP) (n = 73), cardiac origin (CCP) (n = 70), and healthy control (n = 67). The Beck Depression and Anxiety Scales as well as the Short-Form Health Survey were used to gather data. For the statistical analysis, the exact test, t-test, Kruskal-Wallis, logistic regression, and chi-square models were utilized. SPSS software of version 26 was used for every analysis.



Results

The NCCP group had significantly higher mean depression and anxiety scores (depression = 17.03 ± 11.93 , anxiety = 17.18 ± 11.37) compared to the CCP (depression = 9.73 ± 5.76 , anxiety = 8.77 ± 5.96) and healthy (depression = 7.00 ± 7.61 , anxiety = 6.18). The NCCP group's mean quality of life score (54.87 ± 12.66) was considerably lower than the CCP group's (76.31 ± 12.49) and healthy group's (80.94 ± 15.78) scores ($p < 0.05$) (Figure 1). Compared to the CCP and health groups, patients with NCCP had lower odds of having mental quality of life ratings (adjusted OR = 0.90, 95% CI: 0.87, 0.94) and a higher possibility of having depression (adjusted OR = 4.39, 95% CI: 1.25, 15.35).

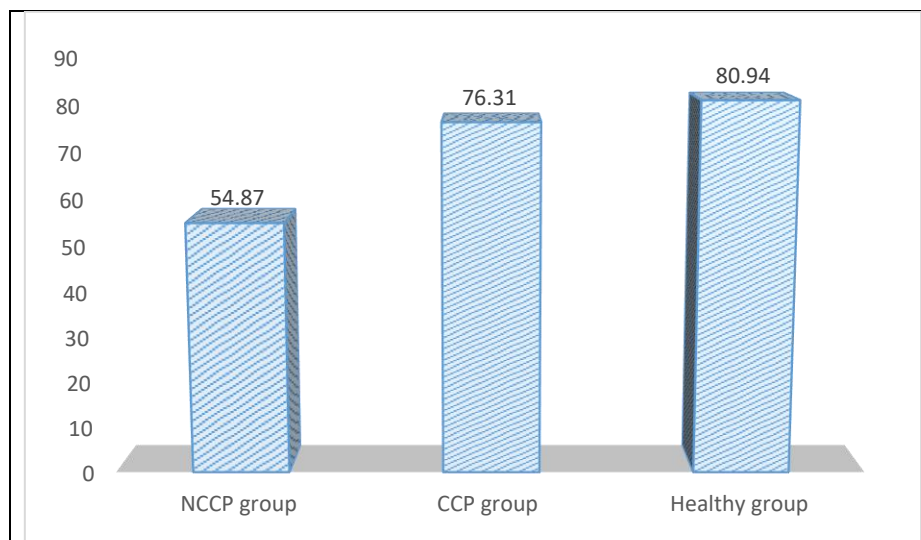


Figure 1: Mean score of quality of life in study participants

Discussion

According to the current study, compared to CCP or healthy individuals, patients with NCCP were more likely to experience moderate-to-severe depression and a poorer quality of life. Compared to patients with CCP, those with NCCP had higher median scores for anxiety and depression.¹ Low HRQoL levels were observed by patients with NCCP, especially those with a history of CD, and this was linked to psychological distress.² Patients with NCCP may experience more severe chest discomfort and a lower quality of life connected to their health if they also have comorbid panic



disorder (PD) or generalized anxiety disorder (GAD). GAD was more strongly correlated with the mental aspect of quality of life, whereas PD seems to be primarily related to the physical aspect.³

Conclusion

The current study concluded that the NCCP group scored lower on quality of life outcomes and had higher levels of anxiety and depression than the CCP and healthy groups. Individuals with NCCP were more likely to experience depression and have lower mental health. When compared to healthy individuals, CCP participants exhibited lower quality of life and greater levels of anxiety and sadness.

It might be essential for psychiatrists and other professionals to work together to enhance patients' quality of life and medical conditions.

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3. Evaluation of the association between biological rhythm dysregulation and suicidal ideation in major depressive disorder patients

Introduction

Circadian rhythms are periodic behavioral and physiological processes that occur inside organisms. They include the sleep-wake cycle, hormone secretion, food behaviors, and energy consumption. The evolution of biological clocks was regarded as a biological approach that has improved an organism's chances of survival by adapting to the daily changes brought about by a continually changing environment. However, the demands of modern life, such as long hours spent indoors without access to natural light and exposure to artificial light after sunset, have resulted in changes in everyday behavior that contradict natural circadian rhythms. This change in lifestyle may increase the risk of developing a number of ailments, such as metabolic disorders, cancer, and cardiovascular diseases.¹ Suicidal ideation was characterized by thoughts of suicide or wishes to die, which may be followed by plans and intents to commit suicide. According to data from the National Survey on Drug Use and Health, around 4.5 million Americans aged 18 or older—or about 4% of the population—reported having considered suicide in the previous year. This represents an assessment of the continuum of suicidality.² There was an increased risk of suicide in patients with MDD. The association between biological rhythm disruptions and the intensity of suicide ideation in MDD patients was yet unknown, despite the fact that disruption of circadian rhythms was a prominent clinical characteristic of the illness.¹ The purpose of this study was to investigate the features of several biological rhythm aspects of major depressive disorder (MDD) and their correlation with the intensity of depressive symptoms and thoughts of suicide.

Methods

In this study, a total of 100 participants were included. Among 50 MDD patients and 50 healthy controls, their demographic information was gathered. The Hamilton Depression Rating Scale, which has 17 items, was used to evaluate the intensity of depression symptoms (HDRS 17). The Beck Scale for Suicide Thoughts (BSS) was used to assess the degree of suicidal thoughts. The individuals' biological rhythm dysregulation was evaluated using the Chinese translation of the Biological Rhythms Interview of Assessment in Neuropsychiatry (BRIAN) scale. The association between



biological rhythm and MDD risk was investigated using multiple logistic regression analyses. In order to examine the connection between various biological rhythm aspects and suicidal ideation, multiple linear regression analysis was carried out in the MDD group.

Results

The total BRIAN score ($Z = 5.41$, $P < 0.001$) and the scores for each dimension showed significant differences between the MDD group and the control group (Figure 1). Multiple logistic regression analysis showed a significant correlation ($OR = 1.20$) between the total BRIAN score and the presence of MDD after correcting for confounding variables. Additionally, there was a significant correlation between scores in various BRIAN dimensions and the presence of MDD (activity: $OR = 1.47$; sleep:

BRIAN score	MDD(n = 50)	Health controls(n = 50)	Z	p-value
BRIAN total score	41.00(26.75,45.25)	23.00(20.75,26.00)	-5.41	<0.001
BRIAN-Sleep	13.00(8.50,16.00)	8.00(5.00,9.00)	-5.14	<0.001
BRIAN-Activity	11.50(6.00,14.25)	5.00(5.00,7.00)	-5.11	<0.001
BRIAN-Social	5.50(4.00,8.25)	4.00(4.00,4.25)	-4.17	<0.001
BRIAN-Eating pattern	7.00(4.00,10.00)	5.00(4.00,6.25)	-2.25	0.025

BRIAN=Biological Rhythm Interview Assessment in Neuropsychiatry;
BRIAN total score, and scores of individual scales were described as median (lower quartile, upper quartile) [M (QL, QU)], and intergroup comparisons were conducted with the Mann-Whitney U test

Table 1. BRIAN domain comparison between MDD patients and healthy controls.

$OR = 1.52$; social: $OR = 1.80$; eating pattern: $OR = 1.34$). Significant positive associations were found between the HDRS17 score ($\beta = 0.56$), BRIAN eating pattern score ($\beta = 0.57$), and BSS score in MDD, according to univariate linear regression analysis with suicidal ideation severity as the dependent variable. Even after controlling for demographic variables and the severity of depression, linear regression analysis showed a strong correlation between BSS scores and BRIAN eating pattern scores ($\beta = 0.34$) in individuals with MDD.

Discussion

According to this study, MDD patients' circadian rhythms in the areas of eating, socializing, sleep, and activity were significantly altered. Moreover, there was a positive correlation between the severity of depression and the degree of circadian disturbance. In MDD patients, there was a substantial correlation between increased eating rhythm irregularity and more acute suicidal



thoughts.¹ According to Mondin TC et al., individuals with MDD who were between the ages of 18 and 24 exhibit significantly greater levels of biological rhythm abnormalities than healthy controls.³ Ozcelik et al. discovered considerably greater levels of biological rhythm abnormalities in MDD patients compared to healthy controls.⁴

Conclusion

The current study concluded that MDD patients have significantly higher degrees of dysregulation in all four biological rhythm parameters than healthy controls, and the degree of dysregulation was related to depressive severity.

Changing eating habits could be an advanced therapeutic strategy to lower suicidal thoughts and enhance patient outcomes.

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4. Case report on acute psychosis associated with primary hyperparathyroidism in a bipolar disorder patient

Introduction

Primary hyperparathyroidism (PHPT) occurs when the parathyroid glands create excess parathyroid hormone, resulting in blood calcium levels greater than 2.6 mmol/L. The reported incidence of this was 21 occurrences per 100,000 person-years, and it was often caused by a single parathyroid adenoma.¹ Hypercalcemia is a known cause of neuropsychiatric impairment. Severe hypercalcemia is characterized by changed mental status, psychosis, confusion, lethargy, and coma; in mild cases, patients may report anxiety, depression, and cognitive impairments.² While the symptoms of psychosis caused by hypercalcemia usually go away quickly after the calcium levels return to normal, this was not always the case. Changes in mood and cognition associated with hypercalcemia have been linked to its impact on dopaminergic and cholinergic metabolism as well as neurotransmission through synaptic junctions in the central nervous system, which affects monoamine metabolism.¹ The patient in this case has bipolar disorder and experienced acute psychosis along with primary hyperparathyroidism. An emergency left parathyroidectomy was performed on her after a neck ultrasound revealed a sizable tumor on the left parathyroid.

Case presentation

A female patient, 54 years old, was admitted to the hospital after exhibiting signs of agitation, delusions, hypervigilance, paranoia, and violent behavior in the previous few weeks. Due to abrupt behavioral changes, including agitation, delusions, and anger management concerns, she had previously been diagnosed with bipolar disorder at the age of 31, depression at the age of 33, and schizophreniform disorder three months earlier. Her normal prescriptions consist of 100 mg of sertraline once daily, 15 mg of aripiprazole once daily for more than 15 years, and 2 mg of risperidone twice daily, which were begun approximately three months ago because of her new symptoms. In the last three to four weeks, the nursing staff has observed increased behavioral symptoms, including somatic delusions, bewilderment, agitation, and occasionally violent behavior. She has been treated for severe psychosis for the past three months by a psychiatrist.



The patient's perception that their upper and lower limbs were malfunctioning was one of the somatic delusional symptoms. The catatonic symptoms were echolalia, negativism, abrupt agitation, and a reluctance to eat or drink over the previous three to four weeks. The Glasgow coma scale indicated that she was hemodynamically stable and that her level of confusion was 14 (E4M6V4). Laboratory testing revealed a high calcium level of 3.54 mmol/L at the time of admission. She started receiving regular saline

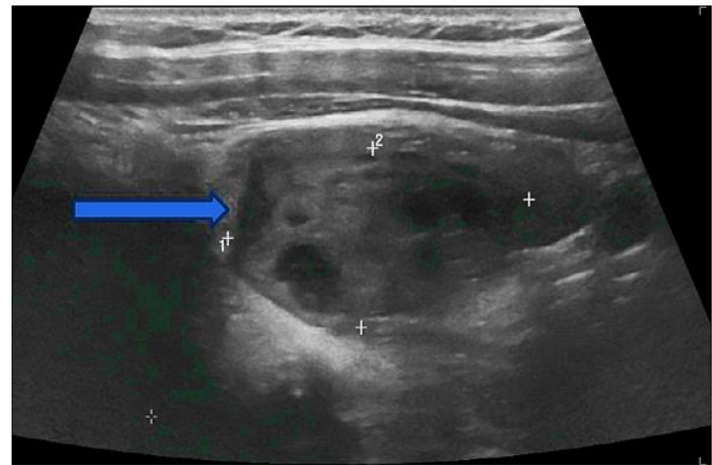


Figure 1: The pointed arrow in the ultrasound of the neck indicates parathyroid adenoma

intravenously and was given three liters in a 24-hour period. Retest blood work revealed a PTH level of 45 pg/ml and only modest improvement in hypercalcemia, from 3.54 to 3.25 mmol/L. There were no structural abnormalities of the brain found during a computerized head scan. In addition to receiving an intravenous infusion of normal saline, she received 4 mg of zoledronic acid intravenously. Following a review by the endocrinology and diabetes teams, she was instructed to start taking 30 mg of Cinacalcet twice a day before being discharged and to keep receiving IV fluids.

An ultrasound of the neck revealed a massive left parathyroid gland mass, indicating a left parathyroid adenoma with a few minor cystic components and internal color blood flow (Figure 1). In addition, the patient had technetium-99m-sestamibi scintigraphy, which revealed a high level of tracer uptake in the left parathyroid gland. After reviewing her case, psychiatrists recommended her to avoid taking regular medications, such as aripiprazole and risperidone. Her calcium level improved to 2.55 mmol/L after five days of receiving intravenous fluids for the next four to five days. The patient's level of confusion lessened, and she no longer displayed violent behavior. Additionally, her head's magnetic resonance imaging (MRI) revealed no abnormalities. She was returned to the behavior health unit on cinacalcet 30 mg twice daily, which was later raised to 60 mg twice daily. Once the patient underwent an outpatient parathyroidectomy, the patient's calcium level returned to normal. After a year of follow-up, the patient's mental health has remained stable and has been discharged.



Regular drugs such as sertraline and aripiprazole were restarted, while risperidone was discontinued permanently.

Discussion

Hyperparathyroidism should be considered in the differential diagnosis of older patients with neuropsychiatric symptoms, as treatment may result in remission. Renal damage can cause altered levels of electrolytes; therefore, using normal values cannot rule out hyperparathyroidism.³ Primary hyperparathyroidism is a frequent condition. When assessing an individual experiencing a sudden shift in their mental state, it was important to keep this diagnosis in mind, particularly for those in the older age group. This was crucial because not only was the illness treatable, but relieving mental symptoms would reduce the need for long-term antipsychotic medication and its possibly dangerous side effects.⁴

Conclusion

Hypercalcemia is a major cause of acute psychotic episodes in patients. Clinicians should take into consideration the possibility of a differential diagnosis for patients who have an underlying mental history, as they can provide diagnostic challenges. It was critical to examine acute medical problems while dealing with such difficult instances because biochemical abnormalities can lead to worsening symptoms.

A curative parathyroidectomy results in full symptom relief for the majority of individuals with primary hyperparathyroidism-related hypercalcemia. Patients receiving parathyroidectomy for primary hyperparathyroidism have a good long-term prognosis.

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