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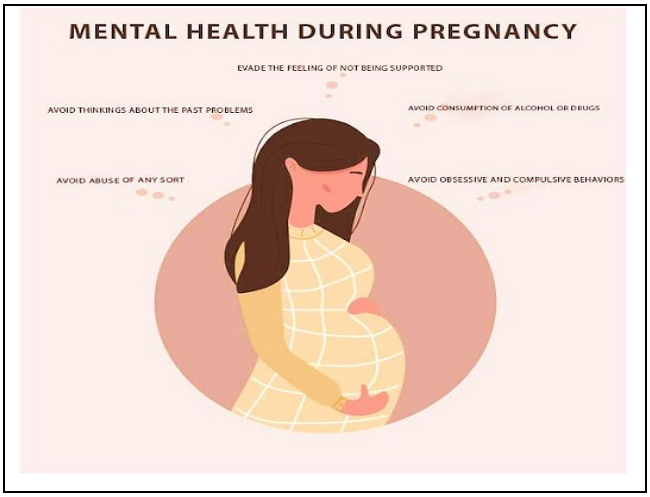
1. Relationship between perinatal depression and diabetes mellitus in pregnancy

Introduction

Depression is a prevalent condition globally, and women are approximately twice as likely as men to acquire depression during their lives. Pregnancy is unquestionably a significant life event that was frequently accompanied by hormonal changes and a period of greatly elevated susceptibility to antepartum depression. According to studies, antepartum depression was more common in the second and third trimesters (12.0%–12.8%) than it was in the first trimester (7.4%).[1] A severe psychiatric disorder that can manifest during pregnancy and in the first year following birth, perinatal depression (PND) can include feelings of worthlessness, irritability, energy loss, a lack of interest in activities, and suicidal thoughts. It is possible for parents who are not biological to have this problem. It is a serious global health concern that places a heavy weight on society because it negatively affects every member of the family. During the perinatal period, diabetes mellitus in pregnancy (DMP) was the most prevalent metabolic disease. Pre-gestational diabetes mellitus, which includes type 1 and type 2 diabetes, or gestational diabetes mellitus were the two possible diagnoses for women with DMP. Depression and diabetes were two common pregnancy problems that were commonly linked to one another. However, there has been contradictory evidence on the connection between postnatal depression (PND) and diabetes mellitus in pregnancy (DMP).[2] The purpose of this study was to investigate the relationship between DMP and PND.



Methods

A total of 4459 women between the ages of 18 and 48 were selected from the Biology, Affect, Stress, Imaging, and Cognition study to participate in this prospective cohort study. DMP was diagnosed as pre-gestational, gestational, or unexplained diabetes based on International Classification of Diseases

code O24 from medical records. Antepartum and postpartum depression were identified based on the results of psychometric tests, clinical interviews, and/or registration data used in the assessment of PND. The relationships between DMP and antepartum and postpartum depression were investigated using multivariable logistic regressions. Multivariable linear regressions were used to examine the relationship between DMP and continuous depression levels, both antepartum and postpartum.

Results

In this study, 1123 women experienced postpartum depression (25%), while 949 women had antepartum depression (21.2%). Only 1.2% of people were affected by DMP. Compared to women without DMP, women with DMP had two times more chances of experiencing postpartum depression. DMP was linked to greater antepartum depression scores, even though there was no correlation between DMP and antepartum depression. In both the unadjusted and adjusted models, DMP was not statistically significant (Table 1). Among the subgroups, antepartum depression was four times more common in women with unspecified diabetes (OR = 4.34; 95% CI = 1.45–12.9; p= 0.008) than in those without diabetes mellitus. However, after controlling for variables, this did not remain significant (OR = 2.86; 95% CI = 0.89–9.20; p = 0.08).

	Antepartum Depression	No Antepartum Depression	Unadjusted OR (95% CI)	*p*	Adjusted[a] OR (95% CI)	*p*
DMP						
No DMP	933	3471	Ref		Ref	
DMP	16	39	1.53 (0.85–2.74)	.2	1.04 (0.56–1.92)	>.9
Subtypes						
No diabetes	933	3471	Ref		Ref	
Pregestational diabetes mellitus	3	7	1.59 (0.41–6.18)	.5	0.80 (0.20–3.23)	.8
Gestational diabetes mellitus	6	26	0.86 (0.35–2.09)	.7	0.66 (0.26–1.66)	.4
Unspecified	7	6	**4.34 (1.45–12.9)**	**.008**	2.86 (0.89–9.20)	.076

DMP = diabetes mellitus in pregnancy; OR = odds ratio; CI = confidence interval.
Bolded text signifies significant *p* value.
[a] Adjusted for age, prepregnancy body mass index, parity, depression history, and pregnancy complications.

Table 1: Relationship among antepartum depression, DMP, and subtypes.

Discussion

The individual and combined effects of DMP-antepartum depression co-morbidity on postpartum depression were investigated in this study. Women who also experienced prepartum depression and gestational diabetes had seven times higher risks of postpartum depression, but with a greater effect size.[2] Shuffrey et al. discovered that antepartum depression without DMP was associated with five times the probability of postpartum depression.[3] According to Lee KW et al., there was a substantial increase in the risk of antepartum depression when gestational diabetes mellitus (GDM) was present. Pregnant women with pre-existing DM and GDM should pay greater attention to their mental health state due to the significant relative risk of antepartum depression among those with DMP.[1]

Conclusion

The current study concluded that DMP raises the risk of PND. Diabetes mellitus may be a risk factor to consider when identifying PND high-risk populations.

Diabetes mellitus in pregnancy (DMP) was associated with perinatal depression (PND), which may be considered a risk factor when screening for high-risk populations.

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2. Comparison of cognitive function between individuals having comorbid schizophrenia and type 2 diabetes mellitus and those having schizophrenia alone

Introduction

Schizophrenia is a disease that reduces life expectancy. Sufferers have a 20% lower life expectancy and twice the death rate of the general population. Accidents and suicide account for only a minor percentage of the increased mortality, with more than two-thirds explained by "natural causes" such as physical disorders like diabetes mellitus.[1] Type 2 diabetes mellitus (T2DM) is widespread in patients suffering from schizophrenia, with meta-analyses estimating a prevalence incidence of roughly 11%. T2DM and schizophrenia both cause a variety of cognitive deficiencies that impair a person's functioning and ability to manage their illness. Most patients with schizophrenia have cognitive abnormalities ranging from mild to severe intensity that affect several domains such as verbal and visual learning, processing speed, reasoning and problem-solving, working memory, and both in the early and chronic stages. Similarly, people with T2DM have reported cognitive dysfunctions ranging in severity from subtle cognitive deficits in domains such as verbal and visual memory, attention and concentration, processing speed, executive function, and motor control to severe cognitive impairment meeting the criteria for mild cognitive disorder or dementia. There hasn't been much study done on how schizophrenia patients' cognitive function was affected by prediabetes (PD) or T2DM.[2] This study aimed to assess the cognitive performance of patients with schizophrenia, prediabetes, or type 2 diabetes and compare them to individuals with schizophrenia who had normal blood sugar levels.

Methods

In this retrospective analysis, 181 patients with schizophrenia or schizoaffective disorder were admitted to an inpatient rehabilitation facility. Retrospectively, sociodemographic details such as age and gender, as well as clinical information such as length of admission, principal diagnosis, comorbidities, current substance abuse, duration of psychosis, fasting blood glucose (FBG), and

whether the patient met the criteria for a clinical diagnosis of treatment-resistant schizophrenia and was treated with clozapine, were gathered. The Brief Assessment of Cognition in Schizophrenia (BACS) was used to assess cognitive function. Parametric and non-parametric analyses were utilized to investigate the study's objectives.

Results

In this investigation, 91.4% (n = 171) of the participants were diagnosed with schizophrenia, while 8.6% (n = 16) had schizoaffective disorder (Figure 1). Nine percent had T2DM, 25% had PD, and 66% had normal blood sugar. The BACS composite score demonstrated an increasing gradient of cognitive losses ranging from mild to severe in the normal, PD, and T2DM groups. The T2DM group showed significantly lower composite scores compared to the PD ($p = 0.026$) and normal groups ($p < 0.001$). The BACS subtest results showed no significant difference in the scores of T2DM and PD patients, with the exception of the token motor task, where the T2DM group scored considerably lower ($p < 0.001$). T2DM patients also scored lower on BACS subtests, with the exception of memory tests, when compared to those with normal blood sugar. The composite and subtest cognition scores showed no significant difference between the PD and normal groups.

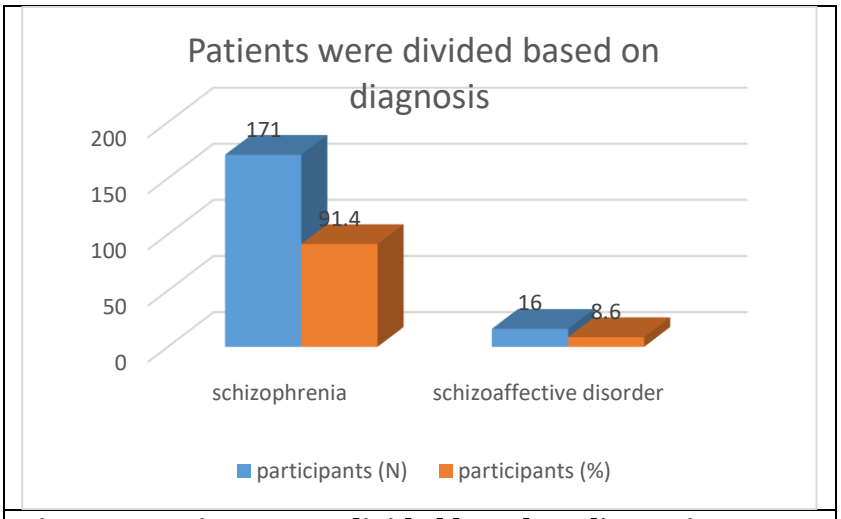

Figure 1: Patients were divided based on diagnosis

Discussion

According to Li et al., patients with schizophrenia and concomitant diabetes outperformed those with schizophrenia alone in terms of delayed memory.[3] Hagi et al., showed that patients with schizophrenia with co-morbid T2DM performed worse on the composite cognitive score and all subtests except memory tasks than those with schizophrenia who did not have diabetes.[4]

Conclusion

The current investigation found more severe cognitive abnormalities among patients with schizophrenia and dysglycemia, particularly those with T2DM, than among those with schizophrenia and normal blood sugar levels. Clinical practice should routinely assess cognitive deficits in individuals with schizophrenia and dysglycemia and their effect on functioning. Appropriate pharmacological, psychosocial, and environmental interventions to lessen the impairments should be more aggressively implemented in clinical settings.

Global cognition impairment was substantially higher in patients with co-morbid schizophrenia and type 2 diabetes mellitus than in patients with schizophrenia alone.

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3. Association between cognitive impairments and the length of untreated prodromal psychosis

Introduction

Schizophrenia causes significant cognitive impairment. The degree of functioning in people with schizophrenia was significantly impacted by cognitive impairment. Cognitive impairments were observed in both first-episode and persistent patients with schizophrenia. According to the neurodevelopmental hypothesis, difficulties in the development of cognitive abilities during development are primarily responsible for cognitive abnormalities in schizophrenia. It was generally agreed upon that a major contributing element to schizophrenia's cognitive impairment was neurodevelopmental issues. In addition, the onset of psychotic symptoms may impede the typical development of advanced cognitive talents, which do not fully mature until late adolescence or early adulthood. The period of time that passes between the development of initial psychotic symptoms and the start of the first successful intervention was known as the duration of untreated psychosis (DUP).[1] Since their positive symptoms have not yet escalated to the point of a full-blown psychotic episode, people at clinical high risk (CHR) for psychosis display diminished positive symptoms while retaining some understanding of their illness. In this context, the time interval known as the duration of untreated prodromal symptoms (DUPrS) refers to the relatively understudied yet critical phase that occurs between the onset of moderate symptoms and seeking professional assistance in the development of psychosis. It was still unclear whether there was a connection between cognitive performance and the length of untreated prodromal symptoms (DUPrS) in people who are clinically high-risk (CHR) for psychosis.[2] This study sought to investigate the complex interactions between DUPrS, cognitive performance, and conversion outcomes in order to shed insight on the potential involvement of DUPrS in influencing cognitive trajectories and psychosis risk in people at CHR for psychosis.

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Methods

This cohort study, which comprises 506 individuals at CHR for psychosis, typically shows attenuated positive symptoms. The participants were identified using the Structured Interview for Prodromal Syndromes, had baseline neuropsychological testing performed, and had a 3-year clinical follow-up evaluation. Psychosis conversion was the main result of this investigation. Based on percentiles, the DUPrS was divided into three groups: short [3 months], middle [4–9 months], and long [10 months]. Using quantile regression and Cox proportional hazards regression analyses, the DUPrS, cognitive factors, and the probability of transitioning into psychosis were examined.

Results

The Category Fluency Test (CFT) and the Brief Visuospatial Memory Test-Revised (BVMT-R) showed that the long DUPrS group performed better cognitively than the short and medium DUPrS groups (Figure 1). DUPrS rank and BVMT-R scores showed favorable relationships, according to quantile regression analysis. In the BVMT-R, participants in the short DUPrS group performed worse than those in the medium and long groups, as evidenced by their lower scores. In a three-year period, 20.8% (95% CI: 17.4%–24.5%) of the 506 subjects advanced to psychosis. The Cox proportional hazards regression analysis revealed that characteristics linked to conversion included a lower level of education, noticeable negative symptoms, and poorer performance on the BVMT-R and Neuropsychological Assessment Battery: Mazes tests.

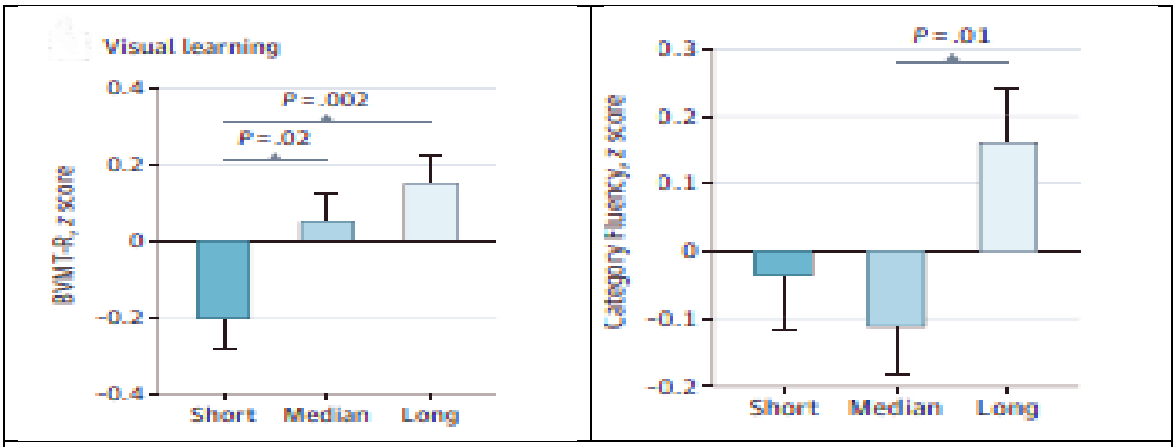

Figure 1: The distribution of cognitive variables was in patients with short, median, and long durations of untreated prodromal symptoms who were at clinically high risk for psychosis.

Discussion

Overall, there was no significant association between DUP and cognitive abilities, with the exception of a weak relationship with a particular cognitive area. A very weak but significant correlation was found between a longer DUP and worse planning and problem-solving abilities.[1] Compared to both the median and long DUPrS groups, participants in the short DUPrS group had noticeably lower outcomes. This data raised the possibility that those receiving critical care for psychosis who have rapidly developed symptoms may have impaired verbal learning and visuospatial memory due to the urgency of symptom manifestation, which may result in cognitive impairment.[3]

Conclusion

The current study revealed the complex relationship between DUPrS, cognitive function, and the probability that an individual at CHR for psychosis will convert to psychosis. Lower cognitive outcomes were linked to shorter DUPrS, with varying effects on different cognitive domains across DUPrS categories.

Reduced performance on cognitive assessments, specifically the Category Fluency and Brief Visuospatial Memory Tests, was linked to shorter duration of untreated prodromal symptoms (DUPrS).

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4. Case report on postpartum psychosis caused by thyroiditis

Introduction

Postpartum thyroiditis and postpartum psychosis are frequently investigated in the differentials of individuals who report postpartum psychosis, and they become primary diagnoses only when other medical disorders are ruled out.[1] Diagnostic Standard Manual V (DSM-5) defines psychosis as abnormalities in two or more of the following five domains: hallucinations, delusions, disorganized speech, grossly disordered or abnormal behavior, and negative symptoms (such as diminished emotional expression, avolition, or alogia) plus one abnormality in the delusion, hallucination, or disorganized speech domain. Individuals with primary thought disorders like schizophrenia or schizoaffective disorders were most likely to have a complex etiology of psychosis. Less frequently, medical disorders such as thyroid problems can cause people to experience psychosis.[2] Though this presentation has been documented, the prevalence of postpartum psychosis was estimated to be between 0.1% and 2%, and it was unclear how often it was for psychosis to co-occur with thyroiditis in the postpartum phase. Patients with postpartum thyroiditis may not report concomitant psychosis to clinicians, which makes sense given the paucity of solid data on this co-occurrence.[1] This case discussed a young woman who was first diagnosed with postpartum thyroiditis but required subsequent treatment for postpartum psychosis due to persisting symptoms.

Case presentation

A 35-year-old woman came for a wellness check 10 weeks after giving birth to twins via C-section. She expressed feeling more anxious and stressed out. In order to address these concerns, the patient reported that she had been receiving professional counselling. The patient specifically described symptoms such as restlessness, anxiety, and irritability. During the visit, the patient had conversational bouncing between topics related to work status, home life anxiety, and nutrition issues. Her spouse and co-workers saw that she spoke with increased pressure and had disordered, tangential thought processes. The significant medical history of the patient included a diagnosis of major depressive disorder at the age of eighteen, which was treated for two to three years with escitalopram.

When she was 23 years old, she was diagnosed with type 1 bipolar disorder and took valproate for two years. After experiencing no symptoms for a considerable amount of time, she made the decision to stop taking medication.

After that, the patient has mentioned experiencing occasional episodes of low mood lasting one to two weeks but no other indications of a mood disorder. This was in spite of the stress of moving between nations during a pandemic, completing residency training, and becoming pregnant with twins. Significant aspects of her familial background included her mother's Crohn's condition and her parents' unexplained hypothyroidism. During her pregnancy, her reproductive endocrinologist prescribed her a prenatal vitamin supplement and 25 micrograms of levothyroxine each day. Because thyroid function has been linked to impacts on pregnancy and fetal development, levothyroxine was often prescribed to women who wish to conceive with increased TSH. She took this dosage of medication during her pregnancy and stayed euthyroid until the day of her presentation. Among the issues she faced during her pregnancy was gestational diabetes, which she managed with glyburide and nutrition.

Levothyroxine was stopped once postpartum thyroiditis was identified based on the patient's clinical complaints and laboratory results. Thyroid-stimulating antibodies (TSA) and serum anti-thyroid peroxidase antibodies (TPO-Ab) were both within the normal range. The patient reported a number of new concerns at the one-week follow-up, including irrational fears that she was being followed and observed by a criminal organization, agitation with family members, and fear that a hospital staff member was giving the criminal organization information about her. The patient was admitted to the hospital due to symptoms of paranoid mania. Retest blood testing revealed somewhat lower levels of free T3 (6.42) and free T4 (2.81) upon hospital admission. Thyroid ultrasound revealed only little heterogeneity, with nodules or areas of enhanced vascularity (Figure 1).

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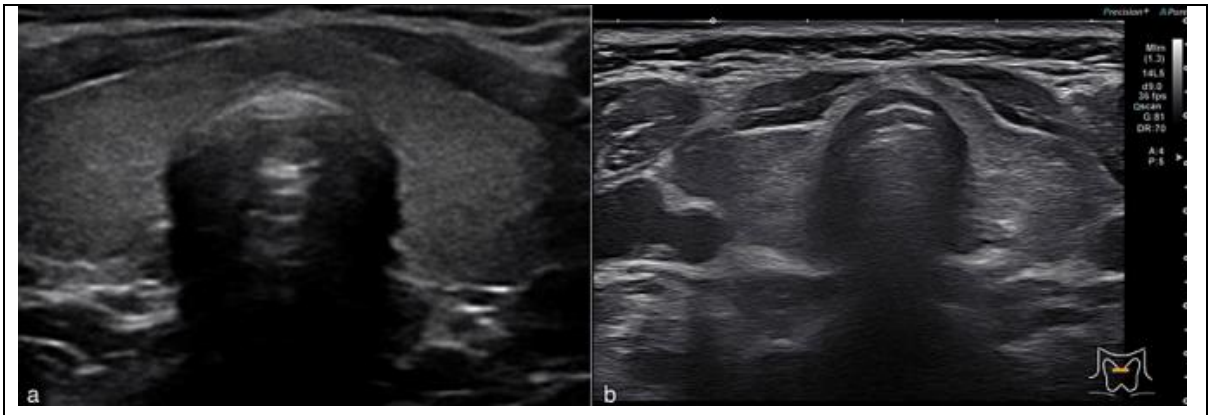

Figure 1: a) Normal thyroid gland ultrasound showing homogeneity b) A thyroid gland ultrasound of the patient revealing a small amount of heterogeneity.

The patient was breastfeeding; thus, radioactive iodine uptake was not performed. She was prescribed 5 mg of olanzapine at nighttime and 20 mg of propranolol twice daily following a psychiatric evaluation. By the third day of her hospital stay, her manic, restless, and tachycardic symptoms started to get better, and she was discharged from the hospital with a diagnosis of postpartum thyroiditis and postpartum psychosis. Two weeks after discharge, she noted that her symptoms had improved since her hospitalization. The thyroid lab results at this time indicated a hypothyroid state with decreased free T3/T4 and high TSH (14.8 mIU/mL). The patient's hypothyroid condition improved with the prescription of 100 µg of levothyroxine per day, while the patient's serum levels of anti-TPO antibodies remained normal.

Discussion

The relationship between hyperthyroidism and psychosis is still poorly understood because anti-psychotic drugs were necessary for the patient's normalization of psychotic symptoms, which persisted even after the thyroidectomy, the definitive therapy.[2] Even though postpartum psychosis was uncommon, it remained a significant clinical and social issue. Current diagnostic criteria equate it with other psychoses; it was not listed as a separate disease entity in the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders) or ICD-10 (International Statistical Classification of Diseases and Related Health Problems). This complicates classification and presents a significant legal issue.[3]

Conclusion

In the present case, there was no autoimmune thyroid dysfunction but an uncommon manifestation of postpartum thyroiditis and postpartum psychosis. In order to provide effective therapy, clinicians should take into account the probability of postpartum psychosis in patients with thyrotoxicosis, especially in high-risk patients with a history of bipolar illness and autoimmune thyroid dysfunction.

Consultation with a psychiatrist after giving birth was advised for postpartum psychosis in order to track the remission of symptoms and manage any recurrent ones.

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