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NEWSLETTER

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Best Regards,

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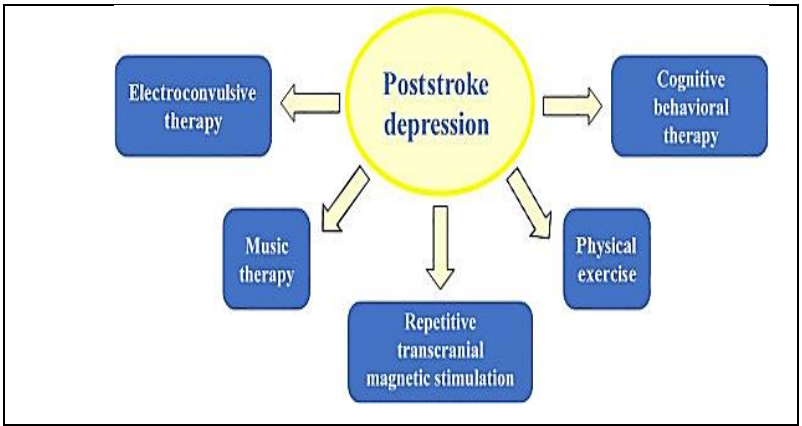
1. The impact of hyperbaric oxygen therapy on post-stroke depression

Introduction:

Post-stroke depression (PSD) is a common consequence in stroke patients that can impact those in the acute stage as well as those in the convalescence stage, with an estimated 33% frequency. Many people with PSD, however, may go undetected and untreated. Gender, a history of mental illness, the size and location of the stroke, a lack of social support, and the degree of physical disability are all risk factors for PSD [(1)]. Antidepressants (AD) are mostly used to treat PSD. However, AD has a poor treatment impact and a higher prevalence of adverse events (AEs) [(2)]. Hyperbaric oxygen treatment(HBOT) can enhance tissue oxygen content and alleviate ischemia and hypoxia, therefore protecting brain cells and restoring brain cell function [(1)]. HBOT was given to patients suffering from hemorrhagic stroke, acute ischemic stroke, suspected stroke, and post-stroke, and the growing scientific research on the issue showed both effective and failed outcomes [(3)]. The study's goal was to assess the clinical effects of HBO treatment on individuals with diabetes with PSD.



Post stroke Depression: Treatment Strategies

Methods:

A total of 190 diabetic patients with PSD were randomly allocated to one of two groups: observation or control (95 patients in each group). For eight weeks, the control group was given escitalopram oxalate 10mg once a day. In addition, during eight weeks, the observation group received HBO treatment once a day, five times a week. The primary outcome of this study was to compared the Montgomery Depression Rating Scale (MADRS), National Institutes of Health Stroke Scale (NIHSS), hypersensitive C-reactive protein, tumour necrosis factor (TNF)-α, and fasting glucose levels.

Results:

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There were no significant differences between the groups in terms of age, gender, or depression course (P > 0.05). MADRS scores in both groups reduced considerably after HBO therapy (14.3±5.2), and were significantly lower in the control group (18.1± 3.5). After HBO therapy, NIHSS scores in both groups declined considerably, with the observation group (12.2±4.0) decreasing more than the control group (16.1±3.4) (table 1), a statistically significant difference (P< 0.001). The levels of hypersensitive C-reactive protein and TNF-α were considerably reduced in both groups, with the observation group much lower than the control group (P< 0.001). Fasting blood glucose levels decreased considerably in both groups, with the observation group declining more (8.02 ±1.10) than the control group (9.26± 1.04), with statistical significance (t = -7.994, P< 0.001).

Table 1: Depressive state and neurological function scores of the two groups

Group	Patients	MADRS scores		NIHSS scores	
		Before treatment	After treatment	Before treatment	After treatment
Observation group	95	33.7 ± 5.0	14.3 ± 5.2[a,d]	21.9 ± 4.1	12.2 ± 4.0[a,d]
Control group	95	33.0 ± 4.0	18.1 ± 3.5[a]	21.0 ± 3.9	16.1 ± 3.4[a]

a P < 0.001 vs before treatment in this group. d P < 0.001 vs control group after treatment.

Discussion:

Current study showed that when compared to the control group, HBO treatment reduced depressed symptoms and neurological impairment in patients with PSD and lowered levels of hypersensitive C-reactive protein and TNF-α. Patients with diabetes and PSD who had HBO treatment showed reduced fasting glucose levels [(1)].

Zhang Z et al and Liang ZH et al in their study showed diabetes, a chronic disease characterised mostly by increased blood glucose levels, may increase the risk of stroke and PSD [(4,5)]. Furthermore, the combination of PSD and diabetes may raise the risk of recurrent stroke, exacerbate depressed symptoms, and potentially increase patient death.

Currently, escitalopram is the most often used first-line medicine for the treatment of PSD, and its usage can relieve patients' emotional symptoms. But Starkstein SE et al showed after taking escitalopram, some individuals with PSD still exhibit depressive symptoms, indicating that the medication is ineffective [(6)].

Conclusion:

Current study concluded that HBO treatment can considerably reduce depressed symptoms and neurological dysfunction in PSD patients, as well as lower levels of hypersensitive C-reactive protein, TNF-α, and fasting blood glucose.

In individuals with Post-stroke depression (PSD), Hyperbaric oxygen (HBO) therapy improved depressive symptoms and neurological impairment while also lowering levels of hypersensitive C-reactive protein and TNF-. Patients with diabetes and PSD who had HBO therapy exhibited lower fasting glucose levels.

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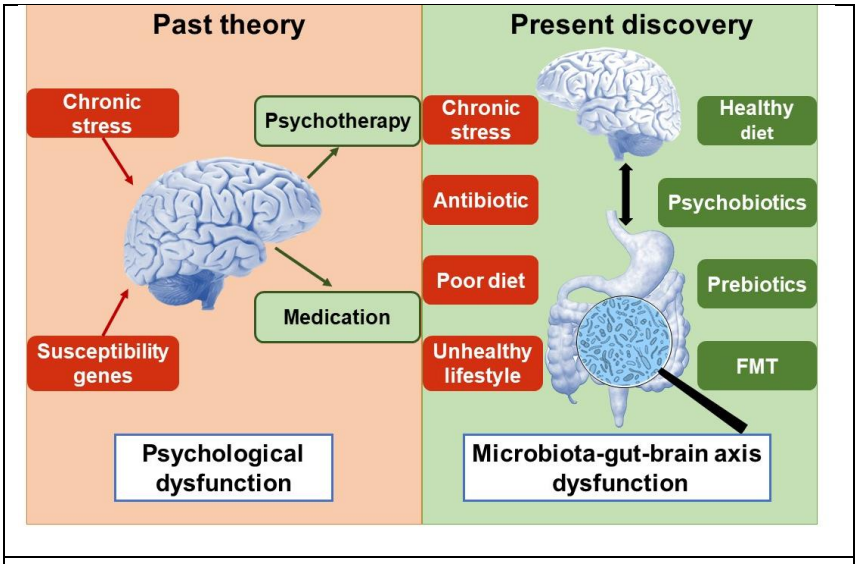
2. Modified gut microbiota profile in perimenopausal panic disorder patients

Introduction:

Perimenopausal panic disorder(PPD) is defined by sudden and frequent panic episodes, intense feelings of approaching death, and autonomic nervous system dysfunction, including palpitations, tremors, and sweating [(1)]. During the menopause transition, women often experience a steady decrease in ovarian activity as well as a physiologic worsening of hypothalamic-pituitary-ovarian axis function in association with fluctuating hormone levels, resulting in menopausal syndrome (MPS) [(2)]. The microbiome-gut brain axis findings suggested a complicated multiorgan bidirectional signalling mechanism between the gut microbiota and the brain. As a result, the gut microbiota has the ability to influence brain function and, as a result, mental health [(3)]. The gut microbiota has been shown to influence the central nervous system (CNS), including the amygdala, hippocampus, and prefrontal cortex. These brain regions constitute the neuroanatomical basis for panic. Furthermore, oestrogen deficiency changes the gut microbiome balance during perimenopause and produces anxiety-like behaviour. As a result, current study hypothised that perimenopausal gut microbial imbalances may trigger panic episodes [(1)]. The purpose of this study was to identify unique microbiota in PPD patients and the inherent link between them.



Microbiota-gut-brain axis dysfunction

Methods:

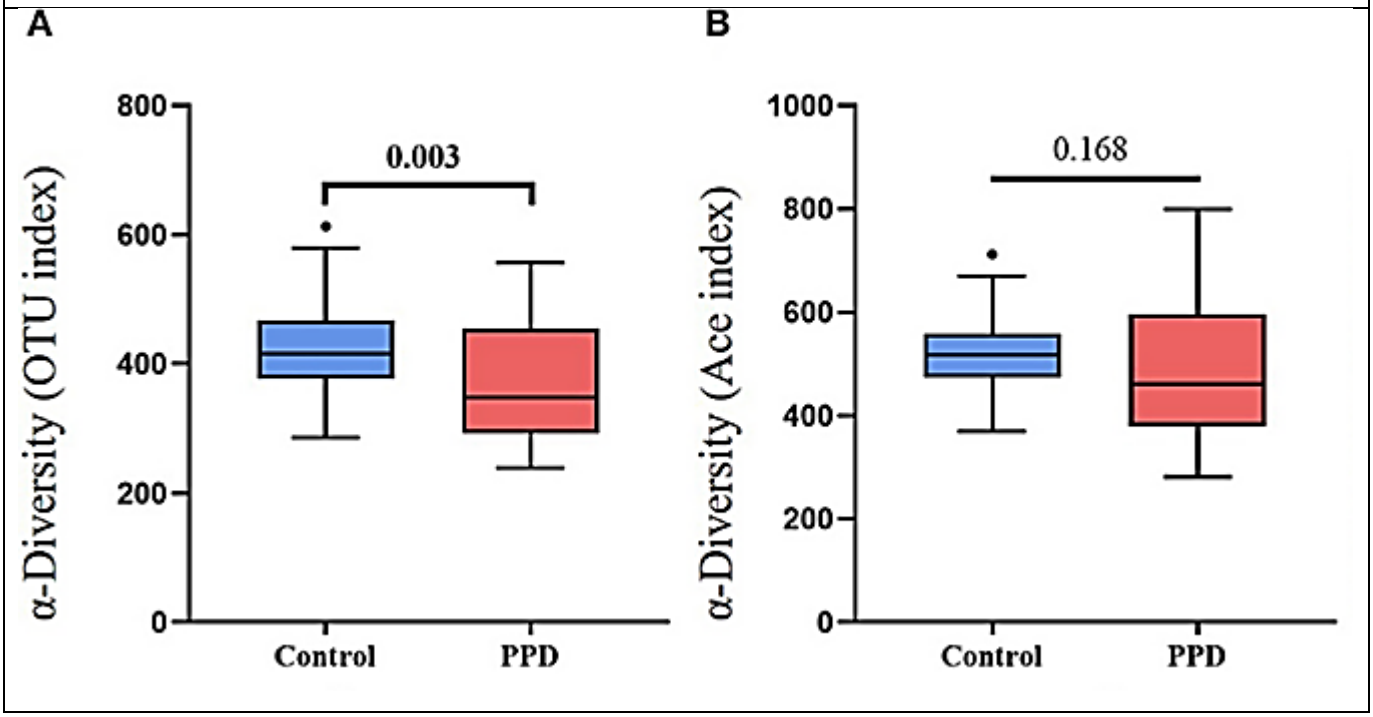
Patients with PPD were selected for this study. All PD patients aged 41 to 60 years were diagnosed using the DSM-5, and their Hamilton Anxiety Scale (HAMA) score was ≥14. The microbial genomic DNA was isolated from faeces using the QIAamp R DNA Stool Mini Kit. The DNA content and purity were determined using an OD-1000 spectrophotometer and 1% agarose gel electrophoresis. As

a template, diluted genomic DNA was employed. The gene was amplified efficiently and precisely using GoTaq R Hot Start Colourless Master Mix. The Illumina MiSeq was utilised in this study to calculate the levels of diversity within and between communities. MetaboAnalyst software was used to show and evaluate differences in a microbial community using principal component analysis (PCA) and orthogonal partial least squares-discriminant analysis (OPLS-DA).

Results:

Alpha diversity, including the OTU index, ACE, Chao index, Shannon, and Simpson, suggested the abundance and variety of gut microbiota. The alpha diversity in this research was greater and Gut bacteria species diversity is greater in healthy individuals than in PPD patients (Figure 1). PPD and healthy controls showed distinct gut microbiota compositions, according to β-diversity. At the genus level, the abundance of 30 microbiota species differed substantially between the PPD and healthy controls (fig 1). HAMA, Panic Disorder Severity Scale (PDSS), and Panic-associated Symptoms Scale (PASS) measures have been collected in two groups. Bacteroides and Alistipes were found to be positively linked with PASS, PDSS, and HAMA.

Figure 1. Box plots of α-diversity between PPD patients and healthy controls. (A) OTU index, p = 0.003. (B) ACE index, p = 0.168.



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Discussion:

Present study is the first study to find substantial differences in the gut microbiota of perimenopausal panic disorder (PPD) patients and healthy women (healthy controls) [(1)]. The gut microbiome has been widely studied in the context of mood disorders. In a similar researches by Wagner-Skacel J et al and Lach G et al found that gut microbial imbalance is one of the aetiologies of psychiatric illnesses [(4,5)]. The β-diversity revealed distinct microbiome communities in PPD patients compared to healthy controls, and the microbial composition of PPD changed, as previously documented in the study by Chen Y-H et al [(6)].

Conclusion:

This study concludes that PPD group patient have different intestinal flora compare to healthy women. Bacteroides and Alistipes dysbiosis are the most common unbalanced microbes in PPD. PPD can be influenced by the gut microbiota via immunological inflammation and tryptophan-kynurenine pathway metabolism. This microbial change might be an aetiology and physiopathological aspect of PPD. The outcomes of this study will be used to help the clinical diagnosis and treatment of PPD.

The gut microbiota may affect Perimenopausal panic disorder (PPD) through immunological inflammation and tryptophan-kynurenine pathway metabolism.

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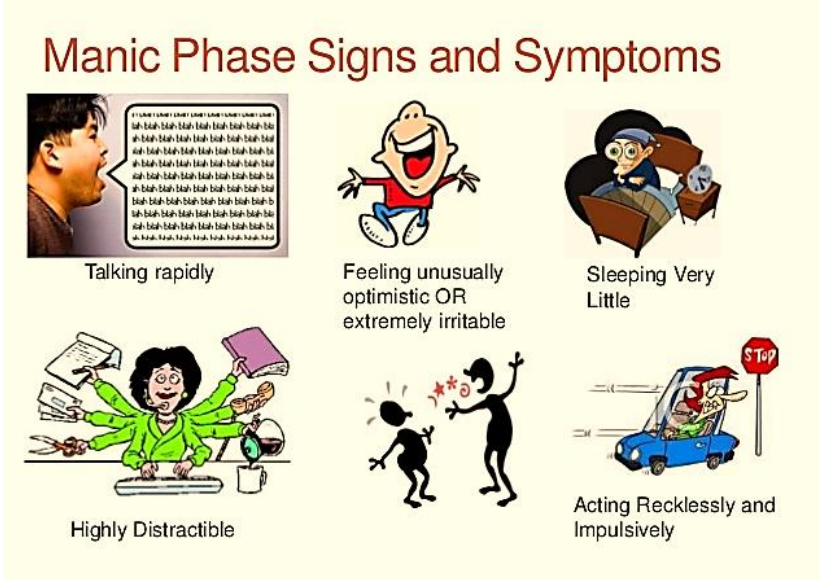
3. Physical Violence Clinical Markers in Bipolar Disorder Patients in Manic States

Introduction:

Bipolar disorder (BD) is a severe chronic psychiatric disease characterised by periods of mania, hypomania, and alternating or interconnected spells of depression, with a global prevalence of 1%. Acute bipolar mania can be a medical emergency, requiring psychiatric hospitalisation to protect patients from hyperactive and impulsive behaviour [1]. The beginning and progression of BD may be linked to structural and functional brain changes, genetic factors, social and environmental variables, and metabolic pathways. Physical violence can cause a delay in treatment, which is crucial for the disease's prognosis. Physical violence is a basic characteristic of bipolar disorder in manic states (BD-M) and refers to non-accidental physical attack that threatens or damages others. Several indicators of physical aggression were found in BD-M patients. Serotonin and dopamine function, nicotinic acetylcholine receptor function, inflammatory markers, hypothalamic-pituitary-adrenal axis activity, and cortisol are among them. Furthermore, despite the great specificity of some markers, the high price of these approaches constituted a considerable hurdle [2]. As a result, the current investigation aimed to identify clinical indicators that were affordable, accessible, and reproducible by examining the association between variables and the likelihood of physical violence in patients with acute onset BD-M.



Signs and symptoms of mania

Methods:

This is a cross-sectional retrospectively study included 316 patients with BD. From 316 BD-M subjects, unidentified sociodemographic variables (sex, age, years of education, marital status) and clinical variables (weight, height, body mass index, blood pressure, Bech-Rafaelsen Mania scale (BRMS) score, number of BD episodes, psychotic symptoms, history of violence, biochemical

parameters, and blood routine parameters) were collected, and the risk of physical violence was identified using the Broset Violence Checklist (BVC). To identify clinical indicators for the risk of physical violence, difference tests, correlation analyses, and multivariate linear regression analysis were used.

Results:

The individuals were divided into three categories based on their risk of physical violence: low (49, 15.51%), medium (129, 40.82%), and high (138, 43.67%). There were significant differences in the number of BD episodes, serum uric acid (UA), free thyroxine (FT4) levels, history of violence, and monocyte-to-lymphocyte ratio (MLR) across groups (all P<0.05). The number of BD episodes (r=0.152), FT3 (r=0.131) and FT4 (r=0.132) levels, history of violence (r=0.206), and monocyte/lymphocyte ratio (MLR) (r=-0.132) levels, as well as the risk of physical violence, were all strongly linked to the risk of physical violence (all P0.05) (table 1). In patients with BD-M, the presence of a history of violence, the frequency of BD episodes, UA, FT4, and MLR were found as clinical indicators of the risk of physical violence (all P<0.05).

Table 1: Correlation Between Variables and Risk of Physical Violence

Variables	N	*r*	*P*
Episodes	316	0.152	**0.007**
Duration	316	0.025	0.653
Education	316	0.014	0.810
UA	316	0.107	0.058
TT3	316	0.082	0.146
TT4	316	0.104	0.064
FT3	316	0.131	**0.019**
FT4	316	0.132	**0.019**
BRMS	316	0.070	0.216
Age	316	0.003	0.961
BMI	316	0.048	0.391
DBP			
Blood glucose	316	0.102	0.071
TC	316	0.008	0.891
TG	316	0.033	0.562
History of violence (Yes)	316	0.206	**<0.001**
Male sex	316	0.003	0.952
Marital status	316	0.445	0.426

Abbreviations: Episodes, number of BD episodes; Duration, duration of BD; Education, years of education; UA, uric acid; TT3, total triiodothyronine; TT4, total thyroxine; TSH, thyroid stimulating hormone; FT3, free triiodothyronine; FT4, free thyroxine; BRMS, Bech-Rafaelsen Mania scale; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; HDL, high-density lipoprotein; TG, triglyceride.

Discussion:

The current study evaluated the easily available indicators of physical aggression risk in individuals with BD-M, which can aid with follow-up and monitoring of these patients. In patients with BD-M who were at varying degrees of risk of physical violence, there were significant differences in the history of physical violence, the number of BD episodes, the UA, the FT3, the FT4, the TT4, and the

MLR [2]. The current study showed that a history of violence was a risk factor for physical violence in people with BD-M, which is comparable with previous research findings by Li Q et al showed that violent past, suicide history, present delusions, and excitement are risk factors for aggressiveness in general psychiatric inpatients with significant mental diseases [3].

Bartoli F et al discovered a connection between UA levels and the occurrence of physical aggressiveness in BD-M patients in their research. A further study by Chatterjee SS et al discovered that serum UA levels were associated with physical violence in people with BD-M, and that levels reduced as manic symptoms decreased. According to these data, UA is an effective predictor of physical violence in BD-M patients [4,5].

Conclusion:

Physical violence has a major influence on BD-M diagnosis and management. Physical violence history, the frequency of BD episodes, UA levels, FT4 levels, and MLR are all clinical indicators that may suggest a greater risk of physical violence in people with BD-M. In most therapeutic circumstances, this information is reliable and easily accessible. As a result, collecting these data early may help clinicians identify BD-M patients who are at high risk of physical violence, allowing them to implement successful treatment strategies in a timely manner.

Clinical indicators that may suggest a higher risk of physical violence in individuals with bipolar disorder in manic states (BD-M) include physical violence history, BD episode frequency, uric acid (UA) levels, free thyroxine (FT4) levels, and, monocyte/lymphocyte ratio (MLR).

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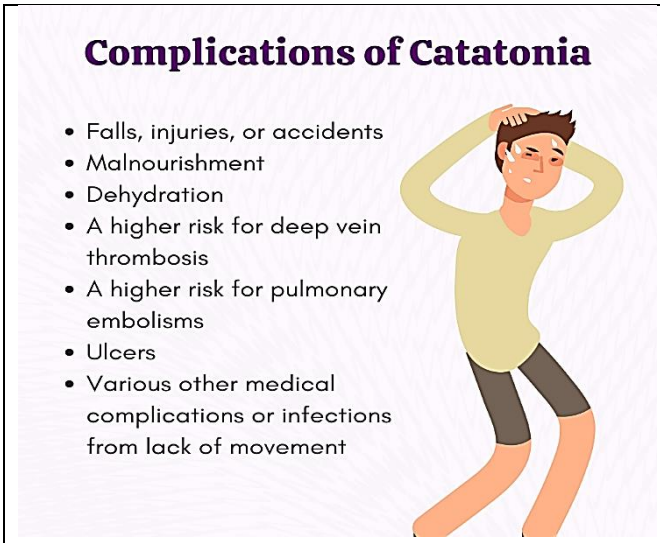
4. A case report of idiopathic catatonic syndrome in a young boy with no past psychiatric history

Introduction:

Catatonic syndrome is characterised by alterations in motor activity that can be caused by both mental and non-psychiatric disorders [(1)]. Catatonia linked with significant morbidity and mortality in infants and adolescents. Paediatric patients with untreated malignant catatonia can die within days, while adolescents with catatonia combined with psychosis and disorders of mood are 60 times more likely to die prematurely [(2)]. It has been noticed that catatonia frequently remains undiagnosed by physicians, especially in situations where there is agitation, a mix of hyperactive and hypoactive behavioural patterns, or when the signs of catatonia are overshadowed by coexisting illnesses [(3)]. This is a unique case of a 20-year-old male who presented to psychiatric with an acute catatonic illness characterised by mutism, blank staring and a lack of movement.



Catatonia complications

Case Report:

A 20-year-old Caucasian male college student with no known psychic history was brought to the emergency department with reduced motor activity and vocal responsiveness. He also had significant psychomotor impairment, poor speech with no spontaneous expressions, intermittent eye contact with intervals of vacant staring, muted emotion, difficulty or reluctance to obey directions, and an overall lack of cognition. The patient's medical history, collected by the patient's mother, included simple mild asthma with no history of hospitalisation, as well as anaphylactic allergy to a specific food. The patient's roommates revealed that he consumed cannabis on occasion, but not in the preceding several weeks. He was diagnosed with major depression, catatonia, and mood-related psychotic symptoms. On day of admission, lorazepam was started at 1 mg three times a day (TID), and risperidone was started the next day and continued at 0.5 mg twice a day (BID) for another 12 days. Clinical evaluation confirmed that the DSM-5 criteria for catatonia were satisfied. Based on the following characteristics,

a severity score of 24 (out of a possible total score of 69) was given using the BushFrancis catatonia rating scale: immobility, mutism, staring, posturing, stereotypy, mannerisms, negativism, waxy flexibility, withdrawal, impulsivity, automatic obedience, combativeness, and ambitendency (meeting DSM-5 criteria for catatonia; see Table 1). To avoid increasing catatonic symptoms, risperidone was stopped and substituted with quetiapine (300 mg/day).

Table 1: Definition of catatonia in DSM-V (APA, 2013):

Catatonia is diagnosed when the clinical picture is dominated by three or more of the following:
1. **Stupor:** No psychomotor activity; not actively relating to environment
2. **Catalepsy**: Passive induction of a posture held against gravity
3. **Waxy flexibility**: Slight and even resistance to positioning by examiner
4. **Mutism:** No, or very little, verbal response
(not applicable if there is an established aphasia)
5. **Negativism:** Opposing or not responding to instructions or external stimuli
6. **Posturing:** Spontaneous and active maintenance of a posture against gravity
7. **Mannerisms:** Odd caricature of normal actions
8. **Stereotypy:** Repetitive, abnormally frequent, non-goal directed movements
9. **Agitation (**Not influenced by external stimuli)
10. **Grimacing**
11. **Echolalia:** Mimicking another's speech
12. **Echopraxia:** Mimicking another's movements

There were no adverse effects recognised by staff or reported by the patient. However, because the patient's symptoms remained life-threatening due to his inability to maintain enough oral intake, decided to start the second-line treatment, electroconvulsive therapy (ECT). On hospital day 24, the patient had his first treatment with bilateral brief-pulse ECT. Immediately upon anaesthesia recovery, the patient's abilities returned sharply. On hospital day 29, the patient recovered from his third ECT treatment and had a restoration of insight, orientation, and personality. The patient had six ECT treatments, with benzodiazepines stopped 12 hours before each session. Following ECT, the patient was started on lithium for either bipolar disease, a depressive episode with catatonia, or severe depression with catatonia. On day 40, the patient was discharged from the hospital. He was able to talk easily with the treatment team on the day of release, and he no longer had any apparent symptoms of catatonia.

Discussion:

When faced with a case of idiopathic or unspecified catatonia, doctors must maintain a high level of suspicion that the underlying illness process is not psychiatric in nature. This is especially true in situations of young patients without a history of psychiatric illness, as children and adolescents with catatonia have a greater frequency of "organic" reasons [(3)].

Benzodiazepines and/or ECT are frequently used to treat catatonic symptoms, regardless of the underlying cause. In a similar study by Medda P et al showed Benzodiazepine relieves symptoms in around 80% of catatonic patients within minutes, while ECT relieves symptoms in virtually all of the remaining patients [4]. It has been suggested that ECT should preferred over benzodiazepines as a first-line treatment since the latter has a temporary effect on catatonic symptoms and does not remove every sign/symptom of an acute catatonic state

Conclusion:

The onset of acute psychomotor symptoms in the absence of a history of psychiatric conditions requires an extensive investigation to rule out medical reasons and assure effective treatment of any underlying illness. The first-line treatment for catatonic symptoms is benzodiazepines, and electroconvulsive therapy can be used to reduce symptoms in individuals who do not respond to pharmaceutical therapy.

Benzodiazepines are the first-line treatment for catatonic symptoms, and electroconvulsive therapy can be used to reduce symptoms in those who do not respond to pharmacological treatments.

Reference:

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