MICRO LABS SYNAPSE

Vol 4|Issue 19|2023

Interesting Cases PSYCHIATRY

Psychiatric Case Series

Vol 4 | Issue 20 |2023

Dear Doctor,

Greetings from Micro Labs Limited.

We are happy to bring you the "PSYCHIATRY Case series", which contains the latest and most interesting cases in the field of Psychiatry.

This issue contains

Case report on Auditory Charles Bonnet syndrome and psychosis

1. **Case report on Auditory Charles Bonnet syndrome and psychosis**
2. **Case report on posterior reversible encephalopathy syndrome in a chronic alcoholic patient with alcohol withdrawal**
3. **Case report on Cushing's Disease Presenting with Functional Neurological (Conversion) Disorder**
4. **Case report of a patient with acute hypoxia and patent foramen ovale initially presented with symptoms of depression and anxiety**

Should you require any specific article or medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback

Best Regards,

Medical Team,

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1. Case report on Auditory Charles Bonnet syndrome and psychosis

Introduction:

The limited response to antipsychotic therapy and the emergence of Musical hallucinations (MH) some years after the beginning of psychosis raised the possibility of a co-existing disease. An accurate otorhinolaryngologic exam revealed underlying acquired hearing loss, which was effectively corrected with surgery.

Charles Bonnet syndrome (CBS) is a clinical symptom in which visually challenged patients experience intense visual hallucinations. Musical hallucinations (MHs) found in elderly people with hearing loss have recently been termed as auditory CBS (aCBS). Brain disease, sensory impairment, and aging were all established risk factors for the disorder.[1] MH can be easily tolerated, but they were frequently debilitating, resulted in a worse quality of life, significant distress, concomitant anxiety or depression, and difficulty in concentrating and falling asleep. In 67% of cases, MH has been linked to hearing impairment. The lifetime prevalence of MH in patients with psychiatric disorders has been reported to be as high as 20%, particularly in persons with obsessive-compulsive disorder, schizophrenia, depression, or alcoholism.[2] This case study described a patient whose clinical course illustrated the frequently disregarded correlation between hearing loss and psychotic symptoms.

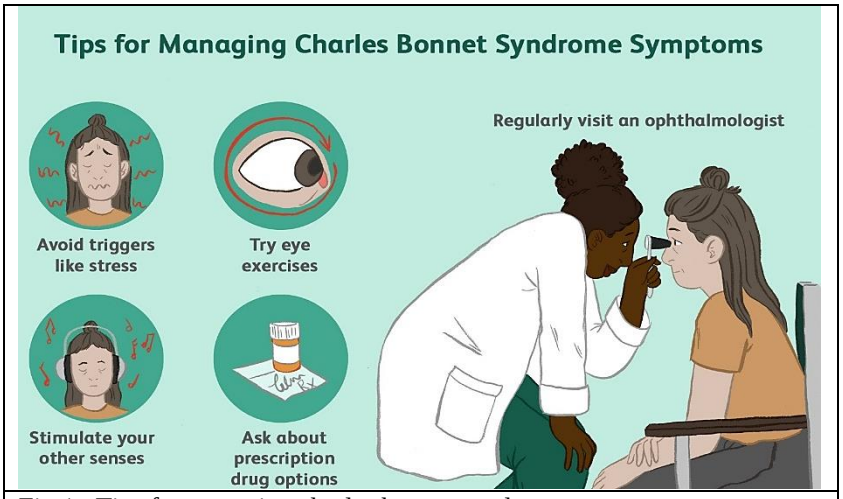

Fig 1: Tips for managing charles bonnet syndrome symptoms

Case Presentation:

A 37-year-old woman who had attempted suicide and had been suffering from delusional-hallucinatory illness for two years, along with significant behavioural involvement, presented to the emergency room. She stated that she had no history of psychiatric disease, substance abuse, traumatic brain damage, or any general medical comorbidities. Her look and attitude were both proper when examined. She was kind and polite. She had no signs of cognitive decline. She complained of prejudice, control, mystic delusions, and sensory-perceptual changes that interfered with her routine. She described feeling uncomfortable and depressed, as well as a progressive lack of interest in everyday activities and a significant risk of self-neglect. She was no longer able to carry out her job. She had a few friends, but she had ignored her social life since experiencing psychotic symptoms. She also complained of sleep issues and

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anorexia with quantifiable weight loss. All of the blood results were within normal ranges. No indication of acute lesions or brain atrophy was seen in imaging investigations, including cranial computed tomography. She claimed no memory or cognitive issues, and no such issues were seen throughout the interview. According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, she was diagnosed with paranoid schizophrenia and was treated with psychotropic medications. After one month, she showed significant improvement in various psychiatric symptoms including encapsulation of delusional ideas and partial criticize of them, as well as remission of depressed symptoms. The patient shared that visual hallucinations had disappeared and auditory hallucinations had decreased, while they remained present. She was discharged from the inpatient facility after being determined to be in partial remission. During follow-up, the patient described the recurrence of referential delusions as well as the continuation of intrapsychic voices that got more strong and intrusive with time. Clozapine was begun and raised to 600 mg/day after several failed antipsychotic attempts (Figure 2). Following that, the patient reported that she was feeling well and appeared to be in a good mood, with a reactive affect and no delusions or paranoid thoughts. She did, however, continue to report experiencing some altered sensory perception, such as whispers, thought echoes, and voices making comments about her actions. Importantly, at the age of 44, after 7 years of therapy and 9 years after the onset of her initial symptoms of schizophrenia, the patient characterized the appearance of MH as a new sort of hallucination. She reported hearing music in her mind, which she identified as a continuous, repeated melody that she recognized, while not being able to identify a specific vocalist. Since the hallucinations were highly disturbing and interfered with her social, occupational, and everyday functioning, the patient expressed worry. The emergence of MH and the poor response of auditory hallucinations to medicine prompted additional investigation into the case. An otorhinolaryngologic and audiometric examination revealed a typical picture of otosclerosis, including bilateral air conduction hearing loss. In the following months, a left stapedectomy was performed, and a right stapedectomy was conducted a year later. Her hearing had returned to normal some months after the previous operation to treat her otosclerosis, and her auditory hallucinations were reduced to sounds like cracks and whistles. She had completely recovered from her MH and auditory hallucinations one year after the operation, and she had not experienced any further hallucinations. Throughout the subsequent ten years

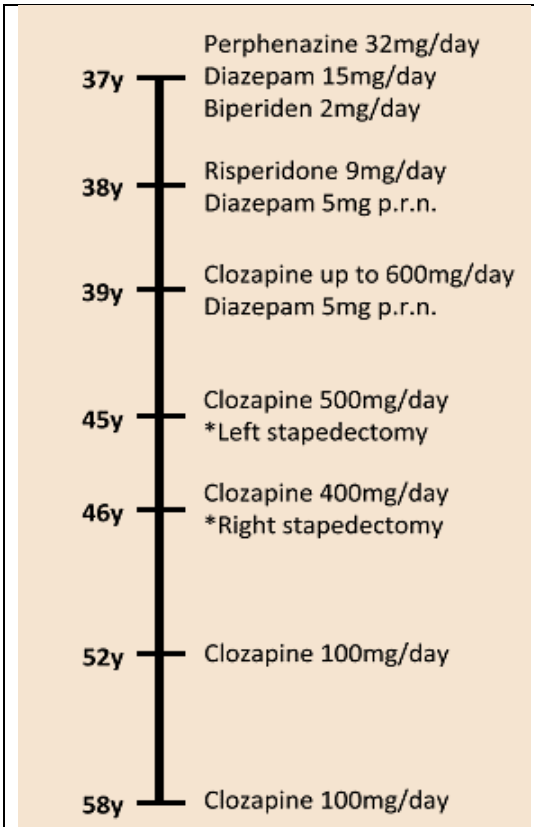

Figure 2: Timeline of the patient's age as well as pharmacological and surgical therapies.

of observation, the patient has maintained complete symptom remission and a high degree of functioning. The patient has been able to reduce her consumption of psychiatric medication due to improvements in her symptoms.

Discussion:

Auditory CBS was a rare and sometimes misunderstood syndrome characterized by the presence of MH in individuals with sensorineural hearing loss and etiology was not clearly due to psychiatric conditions. In the present case, patient was treated with clozapine and increased up to 600 mg/day. Her hearing had returned to normal many months after the latest surgery to cure otosclerosis, and her auditory hallucinations were reduced to noise, crackles, and whistles.[2] In previous study by J Rebol found that the patient communicated effectively two months following surgery. His persecutory and reference delusions, thought insertions and withdrawals, and auditory hallucinations diminished to noise, crackles, whistles, and faint human voices. In this case, patient was treated with olanzapine 20 mg, quetiapine 200 mg and antidepressant duloxetine 60 mg, without a remission.[3]

Conclusion:

The goal of this case study was to raise awareness of hearing loss as a possibly reversible factor to, or comorbidity with, psychosis. Prompt diagnosis and treatment can have a significant impact on patients' mental health, functioning, and quality of life. The emergence of MH some years after the beginning of psychosis, as well as the partial response to antipsychotic therapy, highlighted the possibility of a co-occurring disease. An accurate otorhinolaryngologic exam revealed underlying acquired hearing loss, which was effectively corrected with surgery.

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2. Case report on posterior reversible encephalopathy syndrome in a chronic alcoholic patient with alcohol withdrawal

Introduction:

Posterior reversible encephalopathy syndrome (PRES) is an uncommon radiological and clinical condition characterized by typical brain edema and a variety of symptoms including high blood pressure, encephalopathy, headache, visual abnormalities, focal neurological impairments, seizures, and status epilepticus.[1] Generalized tonic-clonic seizures were the most frequent initial symptom. PRES was diagnosed based on certain clinical and radiographic patterns, which were frequently observed in association with a risk factor. The proper management of PRES include recognizing and resolving the underlying cause, as well as treating the symptoms. Numerous PRES cases have been described in correlation with chronic drinking.[2] This was a case study of a 60-year-old man with a history of chronic drinking who acquired PRES during an episode of acute hypertensive crisis.

Since there are no specific, proven diagnostic criteria for PRES, the key to diagnosing the disease and avoiding harmful work-ups, particularly in patients with atypical presentations, is identifying the distinctive radiologic abnormalities.

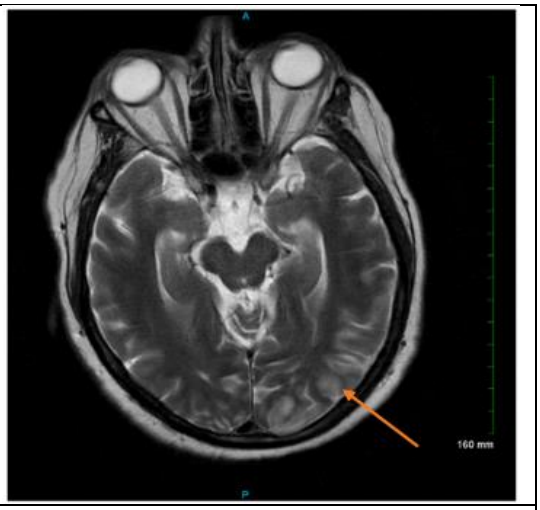

Figure 1: MRI of Brain T2 Hyperintensity in the occipital lobe demonstrating PRES following acute alcohol withdrawal.

Case Presentation:

A 60-year-old male patient presented to the emergency department in an altered mental state. He had a medical history of essential hypertension, peripheral artery disease, hyperlipidaemia, hypothyroidism, chronic kidney injury, and vitamin D deficiency in addition to a psychiatric history of bipolar disorder and chronic alcohol use disorder. Moreover, he consumed alcohol before making his admission. The patient's physical examination revealed tachycardia of pulse rate 124 per minute and hypertension of blood pressure 180/110 mm Hg. As a result, he was transferred to the critical care unit and his numerous comorbidities were managed while he was kept under observation for delirium tremens. Neuroimaging results confirmed the existence of posterior reversible encephalopathy syndrome (PRES) as well as total chronic occlusion of the left common carotid artery and left internal carotid artery. Furthermore, diffusion-weighted imaging revealed

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bilateral cerebral lesions and punctate lesions, which most likely indicated to a hypertensive crises connected to PRES. The patient's medical complications necessitated the use of intravenous lorazepam for sedation and intubation, as well as dexmedetomidine on occasion to control his agitation during his hospital stay. While lowering his sedatives, he suffered agitation, tachycardia, and hypertension. The patient was diagnosed with delirium, most likely as a result of pre-existing medical issues that were worsened by alcohol withdrawal.

His laboratory test results showed no additional anomalies that would have indicated a septic or renal damage. He continued to take his home prescription regimen of risperidone, gabapentin, escitalopram, sodium valproate, and mirtazapine. After discussions and consensus among the medical staff, a low-dose benzodiazepine (0.5 mg oral Lorazepam) was started three times a day to treat anxiety and possible alcohol withdrawal, which was indicated by the recurrence of tachycardia and hypertension during the sedative reduction process. The patient's delirium reduced, and he was finally discharged with his prescribed home medications.

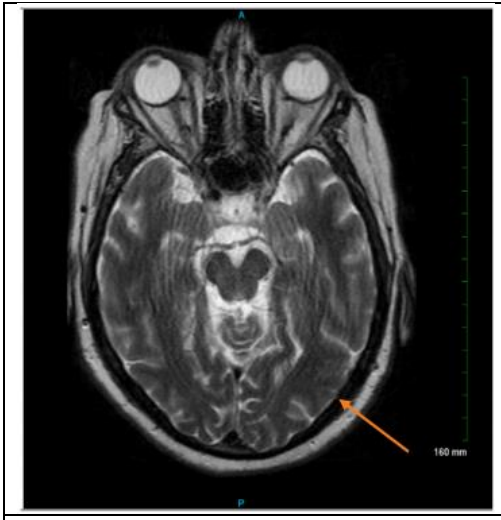

Figure 2: Repeat MRI Brain 3 months later demonstrating PRES reversal.

Discussion:

The exact etiology of posterior reversible encephalopathy syndrome (PRES) was unknown. Numerous vascular and metabolic risk factors, including hypertension, renal disease, pre-eclampsia, eclampsia, infections, substance abuse, autoimmune diseases, haematological disorders, hypomagnesemia, hypercalcemia, and hypercholesterolemia, were frequently linked to it.[2] Largeau et al. found that uncontrolled hypertension was a possible predisposing factor in over 75% of cases.[3] According to the radiographic evidence supporting vasoconstriction, hypoperfusion and ischemia, which cause secondary edema, may be the main abnormality in PRES (Figure 1 and 2).[2] Similarly, Pilato et al. found that the cortical and subcortical T2 Flair hyperintensities seen on magnetic resonance imaging correlated with endothelial damage and vascular dysregulation-induced cortical edema, which was consistent with the clinical presentation of PRES.[4]

Conclusion:

When patients have acute neurological signs and risk factors, the diagnosis of posterior reversible encephalopathy syndrome (PRES) should always be considered. Since there were no specific, proven diagnostic criteria for PRES, the key to diagnosing the disease and avoiding harmful work-ups, particularly in patients with atypical presentations, was identifying the distinctive radiologic abnormalities. Reversing or eliminating the causing factor was the first step in treating posterior reversible encephalopathy syndrome (PRES).

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3. Case report on Cushing's Disease Presenting with Functional Neurological (Conversion) Disorder

Introduction:

Cushing's syndrome (CS) is a rare illness caused by continuous exposure to high amounts of circulating cortisol. Exogenous glucocorticoid (GC) medication was the most prevalent cause of Cushing's syndrome(CS); GCs are mostly used to treat chronic pulmonary diseases, rheumatoid arthritis, and asthma. Endogenous CS pathogenetic processes were classified as adrenocorticotropic hormone (ACTH)-dependent (80% of cases, from a pituitary or other ectopic tumor) or ACTH-independent.[1] Additionally, CS was commonly attributed to a range of cognitive and neuropsychological abnormalities. Psychiatric disease may be the presenting symptom in certain people. Anxiety, depression, and irritability were few of the most prevalent psychiatric symptoms of CS. Functional neurological disorder (FND), often called conversion disorder, is a neuropsychiatric illness in which there was no neurological disease present but instead a person feels neurological symptoms such as motor dysfunctions, sensory abnormalities, speech difficulties, or nonepileptic seizures. Although the exact cause of FND was unknown, it was frequently linked to psychological stressors. Additionally, individuals with FND have aberrant stress responses and higher levels of stress biomarkers, such as cortisol.[2] This case report presented a young man with Cushing's syndrome who reported to the hospital with catatonia and multiple functional symptoms, including functional motor dysfunction and psychogenic nonepileptic seizures.

A multidisciplinary approach was necessary for the successful treatment of Cushing's syndrome with FND. Depending on the severity of the patient's illness and the involvement of various organ systems, this approach may include internal medicine, psychiatry, neurology, endocrinology, neurosurgery, psychology, physical/occupational therapy, and other services.

Case Presentation:

A 28-year-old man reported to the hospital from a nursing facility with a medical history of paraplegia of unknown origin for 6 months, resulting in bedbound status, unspecified seizure condition, untreated Cushing's disease, and hypertension. The patient was afebrile, hypertensive (160/101 mm Hg), tachycardic (107 beats/minute), tachypneic (22 breaths/minute), and had a 98% oxygen saturation level on room air when he first arrived. The patient was not reacted to verbal stimuli but would retreat from discomfort. Dark striae on the abdomen, a loss in upper extremity strength, and an inability to move the lower limbs against gravity were seen during the physical examination. Laboratory investigations revealed hypokalemia (3.5 mEq/L) and thrombocytopenia (78,000/mL), but otherwise were normal. There were no acute intracranial findings on the head CT scan. He was able to follow simple orders and talk again after administering 2 mg lorazepam, but with substantial speech latency

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and hypoverbality. Based on motions made by both lower limbs, the medical staff questioned the paraplegia diagnosis. An MRI of the brain was carried out, although motion artifacts restricted the results and the patient showed anxiety and seizure-like behavior. The patient was monitored with an electroencephalogram (EEG) for three days and experienced repeated bouts of body, limb, and head shaking without epileptiform discharge. Because the patient expressed suicidal ideas, psychiatry was consulted. The patient had a Bush-Francis catatonia score of 12 at the initial psychiatric examination, indicating catatonia symptoms. Catatonia was treated with intravenous (IV) lorazepam 2 mg four times a day, which improved speech quantity to complete sentences.

The patient was switched from intravenous lorazepam to oral clonazepam 2 mg twice daily when his Bush-Francis catatonia score improved. The patient was started on sertraline 200 mg daily and aripiprazole 25 mg daily after confirming that he had depressive and psychotic symptoms, such as poor appetite, anhedonia, suicidal thoughts, auditory hallucinations of talking or screaming voices, and visual hallucinations of people or "dark shadows." The seizure-like occurrences continued but were less frequent. Despite the psychiatric medications being optimized, the patient still experienced residual symptoms of depression, such as persistent auditory and visual hallucinations, suicidal thoughts, and a depressed mood. Additionally, the patient started stuttering when speaking. Following further inquiry, the psychiatric team known that the patient's family had seen physical changes, such as weight increase and striae, two years previous to admission. The patient looked to have developing limb weakness and loss of control over the lower extremities a year before admission. The patient was admitted to a medical-psychiatric hospital for five months after being diagnosed with Cushing's disease in an outpatient setting. Despite this, the patient did not receive treatment because of the concurrent onset and quick worsening of psychiatric symptoms, which included suicidal ideation, poor oral intake, memory loss, and depression with anhedonia. The patient also started experiencing seizures and was given 1000 mg of valproic acid three times a day and 1500 mg of levetiracetam twice a day, but no official examination for epilepsy was performed. The patient was finally transferred to a nursing facility without any psychiatric medications and returned three weeks later because the seizure-like events were becoming more frequent. Further investigations were led by an endocrinology consultation. The patient had a significantly high urine cortisol level over the course of 24 hours (1767.4 mcg/24 hr; normal: <50 mcg/24 hr), which supported the diagnosis of CS. Furthermore, there was an increase in serum cortisol levels to 31.6 mcg/dL in the morning and 23.1 mcg/dL in the evening. Additional investigation showed that the amount of plasma ACTH had also grown to

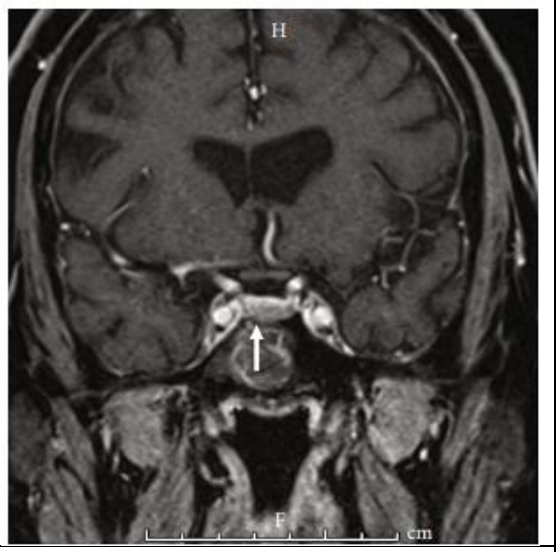

Figure 1: Pituitary microadenoma shown on MRI was consistent with Cushing's syndrome . The anterior inferior pituitary lesion, which measures around 6x6x3 mm, was seen to the right of the midline on the pituitary gland's coronal T1-weighted enhanced MRI.

119 pg/mL. Cushing's syndrome was diagnosed after a sella turcica MRI revealed a 6x6x3 mm pituitary microadenoma suggestive of an ACTH-secreting tumor (Figure 1). Serum cortisol levels were almost normal after starting treatment with 2.5 mg of cabergoline three times a day on weekdays. In view of treatment resistance, 250 mg of ketoconazole three times a day was administered. The pituitary microadenoma will be resected through neurosurgery, which will be the definitive therapy. The patient restored hand function, which included the capacity to write with a pen and eat on his own. The patient's functional symptoms improved along with a decrease in suicidal thoughts, anhedonia, and depression.

Discussion:

Chronic glucocorticoid exposure causes CS, which causes a variety of physical and psychological abnormalities. In this instance, the patient had a variety of symptoms rather than just one, such as PNES, bilateral lower limb paralysis that left them bedridden for an extended period of time, impaired finger flexibility that limited their hand activity, and new-onset stuttering.[2] A previous study on 100 individuals with functional movement disorder by C. Delgado et al. found that 62% exhibited changes in the severity of their symptoms.[3] N. Matin et al. found mixed functional symptoms were seen in 28 out of 100 FND patients.[4]

Conclusion:

This case study demonstrated an unusual psychiatric manifestation of Cushing's syndrome that presented as mixed symptoms FND or conversion disorder. Patient in this case had restricted finger flexion, speech problems, bilateral lower extremities paralysis, and PNES, among other FND symptoms that changed in intensity over time. A multidisciplinary approach was necessary for the successful treatment of Cushing's syndrome with FND. Depending on the severity of the patient's illness and the involvement of various organ systems, this approach may include internal medicine, psychiatry, neurology, endocrinology, neurosurgery, psychology, physical/occupational therapy, and other services.

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4. Case report of a patient with acute hypoxia and patent foramen ovale initially presented with symptoms of depression and anxiety

Introduction:

Anxiety disorders are the most prevalent kind of mental illness. The WHO ranked anxiety disorders as the ninth most common cause of impairment among health-related conditions due to their high incidence, comorbidity, and chronicity. Notably, the risk of anxiety disorders was influenced by both hereditary and environmental factors. Numerous investigations on brain imaging have also revealed structural changes in the medial temporal, prefrontal cortex, and cingulate areas associated with anxiety disorders.[1] Patent foramen ovale (PFO) is the most common fetal cardiac defect, affecting around 25% of the world's population. Although PFO may not often present with obvious clinical symptoms, clinical studies suggest that it was commonly detected in individuals with obstructive sleep apnea, migraine, stroke, and other central nervous system (CNS) disorders.[2] Most persons with PFO were absolutely asymptomatic throughout their lives. The left atrial pressure was often greater than the right, prohibiting blood flow against the gradient. The role of right-to-left shunting through the PFO, anxiety, depression, and hypoxia in systemic circulation was yet unknown.[1] This was a case report of a 52-year-old woman with no heart or lung illness who was hospitalized for anxiety for 5 months and had symptom aggravation with dizziness for 4 days before presenting with cyanosis.

The mechanism behind depression and anxiety may be significantly impacted by patent foramen ovale. It could open up new possibilities for investigating the connections between psychology and medicine.

Case Presentation:

A 52-year-old woman was hospitalized after suffering from anxiety and depression for 5 months and experiencing symptom exacerbation with dizziness for 4 days (Table 1). Five months prior to the onset of the symptoms, she had significant level of job stress. Distress, restlessness, anxiety, concern, apprehension, being easily startled, sadness, lack of energy, diminished interest, sleeplessness, tension headache, shortness of breath, dizziness, nausea, and poor appetite were among the symptoms that were mentioned. It was determined to be anxiety and depression. Giving the patient 225 mg of venlafaxine once a day made her feel better. However, the patient suffered dizziness, nausea, vomiting, sweating, uneasiness, panic, increasing anxiety, and gradual shortness of breath after the medicine discontinued abruptly four days earlier. Additionally, the neurological, gastrointestinal, and cardiopulmonary systems showed no encouraging indicators. Notably, the patient's mental health assessment revealed that she was anxious and had constricted eyebrows, but there was no history of delusions or hallucinations. She did, however, exhibit cyanosis and was found to be hypoxic, with a 94.48% oxygen saturation level on room air. The oxygen tension measured in arterial blood gas was 65.64 mmHg. The results of routine blood tests, biochemistry, ESR, thyroid function, cortisol, inspection of the female tumor, chest CT scan, ultrasound of the deep and intermuscular veins

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of both lower limbs, and Holter monitor were all normal. Prolactin was measured at 42.86 ng/mL, while D-dimer was determined at 0.6 mg/L (0–0.55).

Date	Treatment	Arterial blood gas analysis			HAMA	D-dimer quantitation (mg/L)	Clinical manifestations
		PaO_2 (mmHg)	SaO_2 (%)	$PaCO_2$ (mmHg)			
Day 1 (2021.9.3)	before treatment	65.64	94.48	44.22	HAMA:26 $HAMD_{24}$:31	0.6	anxiety, depression, insomnia, shortness of breath, nausea, dizziness, cyanosis, etc.
Day 3 (2021.9.5)	Oxygen, Paroxetine, oxazepam, olanzapine	89.47 (Stop oxygen inhaled)	98.29	48.06	-	normal	insomnia, shortness of breath, nausea, dizziness, cyanosis disappeared. Upset, restlessness, nervousness, squeezed headache improved
Day 11 (2021.9.13)	Paroxetine, oxazepam, olanzapine	90.42	97.88	48.42	HAMA:1 $HAMD_{24}$:1	-	anxiety, depression, and physical symptoms improved

Table 1: The patient's illness progression and timeline.

Mild tricuspid, mitral, and aortic regurgitation was seen on echocardiography. Saline contrast echocardiography that was agitated was positive (RLS grade 1; Figure 1). Transesophageal echocardiography revealed an approximately 0.9 mm wide fossa ovoid fissure (occasionally stellate shunt; Figures 1B, C). Furthermore, MRA findings were normal, and an MRI scan revealed a bilateral frontal lobe chronic minor ischemia center. E 64, L 44, N 46, P 62, and SCL-90: The overall score was 205, with 65 positive symptoms, anxiety 2.9, phobia 2.67, somatization 2.33, psychosis 1.9, obsessive-compulsive 2.4, sadness 2.38, and other symptoms 2.57. $HAMD_{24}$:31, HAMA:26. These suggested that the patient had hypoxemia along with acute anxiety and depression. According to the ICD-10, the patient was diagnosed with hypoxemia, depressive episodes, and anxiety disorder. She was therefore administered oxazepam and paroxetine to treat her sleeplessness, depression, and anxiety. Olanzapine may help with depression, anxiety, and sleeplessness, in addition to alleviating physical symptoms like nausea and vomiting. In the meanwhile, oxygen inhalation was carried out. After ruling out any other anatomical anomalies or pathological alterations in the heart and lungs, it was found that the patient had PFO (Figure 1). Remarkably, PaO2 increased to 89.47 mmHg just two days after symptoms of anxiety, nausea, dizziness, sleeplessness, and shortness of breath resolved. In addition, cyanosis disappeared and D-dimer levels recovered to normal. Consequently, the intake of oxygen stopped and the PaO2 did not fall (Table 1). Based on the findings, this study hypothesized that physical stress (nausea, shortness of breath, etc.) and emotional stress are key stressors for patients, resulting in cyanosis and acute, transitory hypoxia due to increased right-to-left shunt. After six months, the patient no longer appeared to have hypoxia or cyanosis, felt stable, and persisted on taking low oral dosages of olanzapine and paroxetine.

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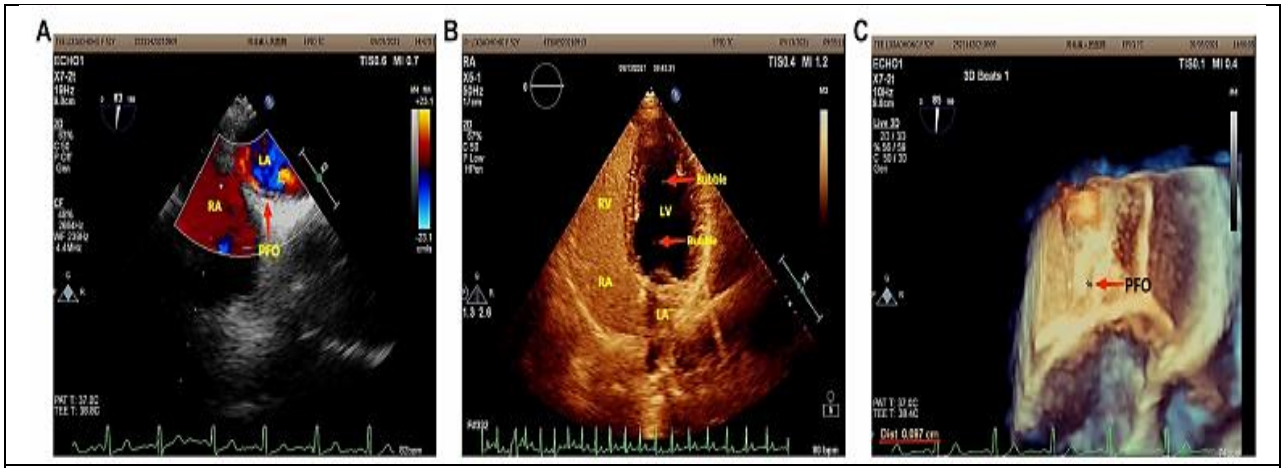

Figure 1: (A) Agitated saline contrast echocardiography revealed that the greatest number of bubbles in a single frame following the Valsalva maneuver was less than 10 bubbles (RLS 1) in three cardiac cycles. (B) A two-dimensional transesophageal heart echocardiogram revealed a 0.9 mm-wide fissure echo in the atrial septum's middle ovoid fossa. (C) Three-dimensional echocardiogram of the transesophageal heart: Following the Valsalva maneuver, the atrial septum's ovoid fossa measured 0.9 mm in width.

Discussion:

This was an uncommon and interesting instance of acute systemic hypoxemia linked with PFO that manifests as cyanosis carried on by extreme anxiety and depression. In this instance, the patient's anxiety, depression, and sleeplessness were treated with oxazepam and paroxetine. Olanzapine relieved depression, anxiety, and sleeplessness in addition to relieving physical symptoms like nausea and vomiting.[1] In a study by Kowalska M et al., demonstrated that paroxetine, a selective serotonin reuptake inhibitor, was used to treat anxiety and depression.[3] Dinis-Oliveira et al. showed that oxazepam was a benzodiazepine anxiolytic with a short half-life that was used to treat alcohol withdrawal as well as anxiety disorders and tension.[4]

Conclusion:

In individuals with acute systemic hypoxemia, right-to-left shunting, severe anxiety, and depression, the shunting mechanism and theory have never been documented. The origin and pathophysiology of anxiety and depression in PFO patients were unknown. This might have implications for pulmonologists, cardiologists, internists, psychiatrists, and experts in psychosomatic medicine. Furthermore, the mechanism behind depression and anxiety may be significantly impacted by patent foramen ovale. It could open up new possibilities for investigating the connections between psychology and medicine.

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