MICRO LABS SYNAPSE

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Interesting Cases PSYCHIATRY

Psychiatric Case Series

Vol 4 | Issue 17 |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

1. **A case report of acute mania in Fanconi-Bickel syndrome.**
2. **A Case Study of a Woman with Charles Bonnet Syndrome and a Pituitary Adenoma.**
3. **A case report on Cotard's syndrome.**
4. **A case report on Anti-leucine-rich glioma-inactivated 1 encephalitis discovered by a manic episode.**

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. A case report of acute mania in Fanconi-Bickel syndrome.

Introduction:

Fanconi-Bickel Syndrome (FBS) is characterised by glycogen accumulation in the kidney and liver. It is an autosomal recessive carbohydrate metabolism disorder caused by SLC2A2 gene mutations.SLC2A2 encodes for glucose transporter 2 (GLUT2), a low affinity facilitative glucose transporter found mostly in tissues involved in glucose homoeostasis such as renal tubular cells, enterocytes, pancreatic -cells, hepatocytes, and discrete brain areas. FBS is caused by aberrant glycogen accumulation in the liver and kidneys, which impairs glucose and galactose utilisation. The traditional presentation of FBS is characterised by dysglycemia and its multiple repercussions however there is no research on neuropsychiatric features of FBS.[1] Researchers believe that this is the first report on the detection and management of acute mania in an FBS patient. This presentation is expanded upon, as are the neurological repercussions of the FBS metabolic disturbance. The researchers also described this presentation and the neurological implications of the FBS metabolic disturbance.[2]

This is the first case study of a potential neuropsychiatric presentations in Fanconi-Bickel Syndrome.

Case Presentation:

Mr. C, a 28-year-old young man with consanguineous parents, was diagnosed with Fanconi Bickel syndrome as an adult after presenting to a mental emergency room with severe acute mania. As a relatively uncommon genetic disorder, this is the first publication according the researchers to address the detection and treatment of psychological symptoms in Fanconi Bickel syndrome. He has chronic kidney disease, heart problems, hepatomegaly, postprandial hyperglycemia, and fasting hypoglycemia, all of which necessitate frequent meals. He was living at home with his parents and younger brother at the time of the psychological evaluations at institution, and he was divorced. He rarely attended college before joining his father in the family construction business. There is no history of mood disorders or other mental illnesses in the family. Mr. C began acting impulsively and aggressively in his early adolescence. His first psychiatric hospitalisation occurred when he was 21, due to a severe major depressive episode. He has been hospitalised several times since then owing to repeated manic episodes marked by intense anger, impulsivity, and violent behaviour. The patient had a history of intermittent cocaine, benzodiazepine, and cannabis use, although he was not using any drugs during his hospitalisations. Mr. C was admitted twice to the psychiatric unit. The initial admission was followed by an incident in which the patient repeatedly hit his father in the head and then chased him while threatening to murder him. He attacked other patients and personnel at the psychiatric emergency room repeatedly and without provocation. Throughout the emergency department,he was given haloperidol,

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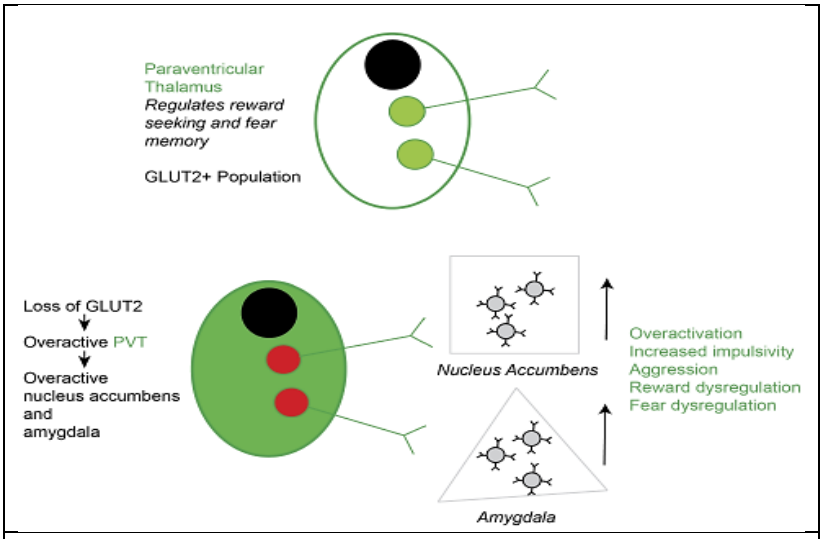
lorazepam, fluphenazine, chlorpromazine, and diphenhydramine. On admission, the mental status evaluation revealed extreme irritability, grandiosity, recurring aggressive behaviours, psychomotor agitation, racing thoughts, and paranoia. Fluphenazine 10 mg twice daily and valproic acid 500 mg in the morning and 1,000 mg at night were used to begin treatment. Fluphenazine decanoate was also given. After 10 days, the patient returned to euthymia and was discharged. Six days after being discharged, the patient returned to the mental emergency room. He was not taking the fluphenazine and valproic acid after discharge, according to his father. He then stopped sleeping and relapsed into a similar manic state marked by acute irritability, grandiosity, impulsivity, psychomotor agitation, violent behaviour, and paranoia. An episode occurred in the psychiatric emergency room during which the patient appeared calm, asked a nurse a question, and then began striking the nurse and other personnel without reason. He resumed treatment on fluphenazine 10 mg twice day and valproic acid 500 mg in the morning and 1,000 mg at night during the second inpatient stay. After 12 days, the patient returned to euthymia and was discharged.



A loss of GLUT2 induces and overactivation of the PVT-to-Nucleus accumbens and PVT-to-amygdala circuit, contributing to acute mania and impulsivity in FBS

Discussion:

FBS, or glycogen Storage Disease XI, is a rare glucose metabolic condition characterised by many known loss-of-function mutations in SLC2A2, the glucose transporter 2 (GLUT2) gene.[1] FBS is extremely rare, having been diagnosed in less than 200 cases globally. This case report covers a patient with FBS who was hospitalised to a psychiatric facility for manic episodes resembling bipolar I disease.[2] Researchers investigate how FBS mutation and glucose imbalance may contribute to bipolar illness. Researchers speculate that the loss of GLUT2 function in FBS may explain this unique neuropsychiatric presentation of acute mania and impulsivity. Based on studies using human, non-human primate, and rodent models, loss of GLUT2 function may result in overactivity of the paraventricular thalamus (PVT) and downstream brain structures, and consequently impulsivity and manic manifestations.[1,2,3]

Conclusion:

This case report explains a patient with FBS who was hospitalised to psychiatric inpatient facility for treatment of an intense manic episode mimicking those often observed in bipolar I illness. This is the first case study of a possible neuropsychiatric FBS presentation. The amygdala and nucleus accumbens, which are known to be abnormally engaged during mania, may get more excitatory input as a result of the PVT's overactivity as a result of impaired GLUT2 function, according to the FBS hypothesis for how mania

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might be explained. This singular example emphasises a possible connection between mood and metabolism, indicating a part for glucose metabolism in psychological symptoms.

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2. A Case Study of a Woman with Charles Bonnet Syndrome and a Pituitary Adenoma.

Introduction:

Complex visual hallucinations that happen in conjunction with other visual abnormalities, such as visual field loss or visual acuity loss, are known as Charles Bonnet syndrome (CBS). The pathways that result in CBS include any dysfunction in the visual pathway, including the optic nerve, brain, or eyes, as the condition develops in people with visual impairment or vision loss.[1] Rarely are reports of CBS linked to pituitary macroadenomas found in the literature. Here is a case of CBS in a 44-year-old woman who had a pituitary macroadenoma. After having a transsphenoidal adenomectomy, the patient continued to have visual hallucinations.[2]

Case Presentation:

It is the first Charles Bonnet syndrome report to be reported and this peculiar condition is under-recognized. Clinicians need to be aware that even people without mental illnesses may have visual hallucinations.

A 44-year-old woman with visual hallucinations was brought to the department of psychiatry by her neurosurgeon after undergoing surgery to remove a pituitary macroadenoma compressing the optic chiasm. She had a husband. She didn't have a history of mental illness. She didn't use any drugs that may cause hallucinations. The patient had a transsphenoidal adenomectomy. The patient was sent to the department of psychiatry one week after reporting persistent visual hallucinations on the first surgical day. The patient was co operative, showed no verbal delirium, and had no cognitive impairment when they arrived in the department. She described having flashes in her head. These hallucinations occurred many times every day for varying lengths of time, lasting only a few minutes. They contributed to sleeplessness and anxiety. The patient claimed that these hallucinations made her feel ashamed. She observed strange human faces without any accompanying signals. The patient was aware that the hallucinations were false since the images did not talk to him or her. There were no anomalies found during the neurologic

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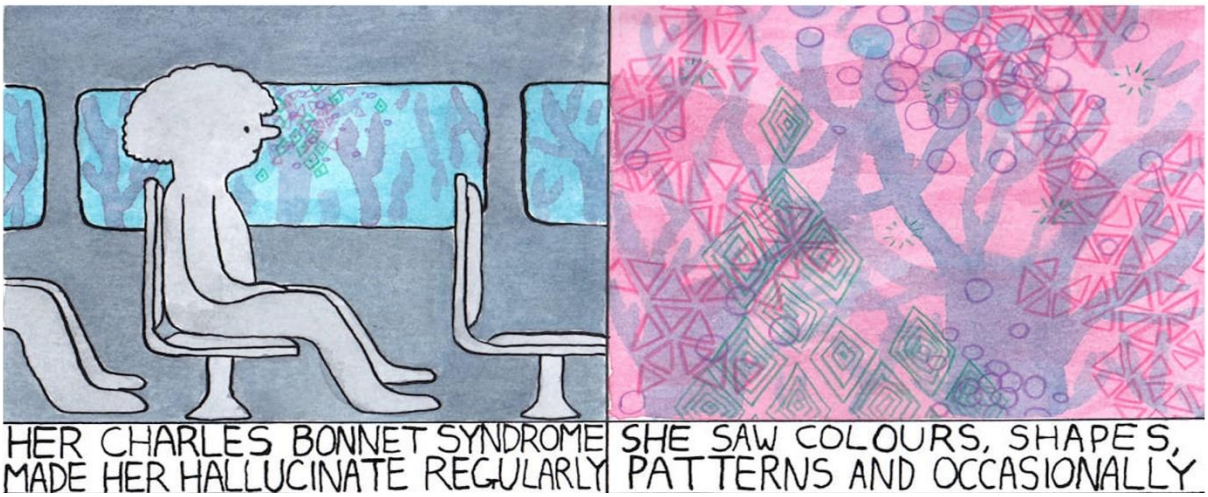
evaluation. She scored 28 on the Mini-Mental State Examination (MMSE). The biological evaluations were normal. All pituitary hormone levels were within normal range. There were no toxic substances found in the blood or urine after investigation. Apart from the anticipated postoperative modifications of a cavity in the sella turcica, the brain magnetic resonance imaging revealed no abnormalities. The patient in the present instance complied with all CBS criteria. Following the interview, no more psychological conditions were suspected. mental and neurological examinations ruled out other diagnoses such neurologic illnesses, mental disorders, toxic causes, metabolic disorders, auditory and sleep deprivation, and hypnopompic or hypnagogic hallucinations. Olanzapine 5 mg/day was prescribed to the patient. After receiving medication for two weeks, hallucinations progressively decreased and disappeared entirely. Olanzapine was continued at a dose of 5 mg per day for three months, then the dosage was progressively decreased and stopped during the next two months without a return of the symptoms. Four months after stopping the therapy, the patient had her most recent mental check-up, and was symptom free. She was satisfied with the treatment she received.



Discussion:

Although CBS is a clinical condition that is infrequently documented, the true prevalence is still unclear for a number of reasons. There are still questions about CBS's pathophysiology. It's crucial to note that the neurological processes behind the start of CBS in people with pituitary adenoma remain unknown.[2] It's vital to keep in mind that CBS has also been documented in patients with retained central vision and visual field abnormalities.[3]

Conclusion:

In conclusion, it is the first instance of CBS that has been reported in English. There seems to be a lack of awareness of this particular ailment. Clinicians need to be aware that even people without mental illnesses may have visual hallucinations. In fact, since

CBS has a different prognosis and requires different care, it is crucial to distinguish it from other mental conditions that may result in visual hallucinations.

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3. A case report on Cotard's syndrome.

Introduction:

Cotard syndrome, a rare disorder marked by nihilistic delusions about the body or life, is present in a number of neuropsychiatric disorders. It often occurs with the depressed symptoms.[1] While Cotard's disease, also known as nihilistic delusions, is characterised by the symptom "I am already dead," the symptom "you are already dead" gets less attention.[2] Here, Researchers report the instance of a schizophrenic patient who, in addition to the standard nihilistic delusions, also stated the belief that "you are already dead." and then discuss various theories for this symptom's underlying processes.

Researchers describe a patient who presented with nihilistic delusions about both herself and other people as well as previous cenesthetic hallucinations

Case Presentation:

A female in her 60s who was hospitalised to hospital for acts of domestic violence brought on by her auditory hallucinations and delusions of reference was given an operational diagnosis of schizophrenia based on the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition, criteria. Even though she had a history of hypothyroidism, her psychotic symptoms were unrelated to this condition. Magnetic resonance imaging (MRI) results for structural analysis were negative. Her condition progressively deteriorated after the start of symptoms at the age of 19. Resistance to therapy and repeated hospital stays were also caused by inadequate adherence to therapy. The patient showed agitation, sadness, suicide thoughts, guilt delusions, catatonia, and Cenesthetic hallucinations. A neurological evaluation indicated lack of pain and touch sensibility. The patient's cenesthetic hallucinations developed during her hospital stay and eventually included nihilistic delusions. Her accounts of cenesthetic hallucinations and different delusions included the idea that spoons, irons, nails, rulers, bins, and coins were within her body, as well as the idea that she was burning or in danger of exploding. She also stated that she had a different perception of her own body, believing it to be bigger than usual or reversed. Interestingly, she stated that the patients in her shared room and the doctor standing in front of her had died. She also claimed that she had killed her daughter, her son would soon pass away, and another doctor had also committed suicide. She was unable to give a justification for her ideas, and occasionally

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she gave delusional explanations. Usual drugs failed to alleviate these symptoms, and mECT with implantation of bitemporal electrodes was used more than 20 times (twice per week) with only limited success. As a result, clozapine (up to 200 mg) was recommended, which significantly reduced her symptoms (positive and negative syndrome scale scores of 31→13 for the positive, 28→25 for the negative, and 27→16 for the general). In the end, the patient was feeling alive again and realised that the doctor was also alive.

Discussion:

Researchers described a patient who experienced censenic hallucinations and nihilistic illusions about both oneself and other people. Notably, the patient's numerous cenesthetic hallucinations with delusional descriptions gave rise to her nihilistic delusions. The two-factor theory of delusions may also be supported by the cenesthetic hallucinations of our patient that included delusional descriptions (secondary delusions). This idea holds that her hallucinatory claims—such as the existence of many things inside her body and the burning or explosion of her body—can be interpreted as abnormal thinking.[2] Her sense of touch may be connected to her delusional descriptions. The trajectory of this patient's symptoms from diverse cenesthetic hallucinations to nihilistic delusions may corroborate this idea given the constant underlying pathology.[3] Furthermore, atypical reasoning may be related to metacognitive deficiencies, which are well-known in schizophrenia and aid in the emergence of delusions. Beyond her current course, patient's clinical history shows a distinct lack of awareness and metacognition regarding her symptoms, which may have contributed to her current delusions.[2]

Conclusion:

Here, Researchers describe a patient who previously experienced cenesthetic hallucinations as well as nihilistic delusions about herself and others. This case lends support to theory that these two types of nihilistic delusions may be connected by unusual interoceptive processing. It is necessary to conduct further study on the pathophysiology of Cotard's syndrome. Researchers think that this case study addresses an essential component of Cotard's disease that has been neglected.

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4. A case report on Anti-leucine-rich glioma-inactivated 1 encephalitis discovered by a manic episode

Introduction:

Limbic encephalitis (LE; sometimes known as antibody-mediated encephalitis) is a category of illnesses that mostly manifest as neuropsychiatric symptoms.[1] Anti-LGI1 is an abbreviation for anti-leucine-rich glioma protein. A rare form of autoimmune encephalitis (AE), encephalitis is characterised by cognitive and mental impairment, facio-brachial dystonic seizures (FBDSs), and hyponatremia.[2] An isolated psychiatric condition is an uncommon presentation of anti-leucine-rich glioma-inactivated 1 (LGI1) encephalitis, a limbic encephalitis. The purpose of this study was to describe a case of anti-leucine-rich glioma-inactivated 1 (anti-GLI1) encephalitis with a mostly psychiatric presentation that delayed identification and treatment and accelerated cognitive deterioration.

Case Presentation:

A 70-year-old man was examined atthe clinic for behavioural and cognitive issues. Although he was retired at the time of the visit, he had previously worked for a heating company. He was completely self-sufficient prior to the commencement of the sickness. No prior mental or neurological disorders were identified in the patient's medical history. He was diagnosed with colorectal cancer at the age of 57, it was in remission. At the age of 67, he started to exhibit a variety of behavioural symptoms, including hyperactivity, euphoria, disinhibition, and rapid speech, and he also had several home improvement projects on the go. Additionally, he experienced extreme sleeplessness and poor cognitive function (he was unable to recognise his own house or recall dates). He first had hallucinations in October 2018, including the sense of bugs crawling on his legs, seeing people in the woods, and hearing the phone ring nonstop. Additionally, he had a persecutory delusion. His wife also noticed a number of instances of confusion and temporospatial disorientation at the same period. By December 2018, he had begun to experience memory loss, sleeplessness, and psychomotor agitation in addition to cognitive impairment. He was taken to psychiatric unit in January 2019. The majority of the symptoms were behavioural, including psychomotor agitation, sleeplessness, and persecutory delusions. Due to the unique presentation, a diagnosis of manic episode with psychotic symptoms was considered, and more research was carried out. The electroencephalogram (EEG) was normal, the magnetic resonance imaging (MRI) revealed particular white matter hyper intensities, and no neurological diagnosis was retained. Laboratory tests at this point revealed hyponatremia (132 mmol/L; reference range: 136-145 mmol/L), according to the results. Risperidone 2 mg was used to treat the patient, however he suffered from serious side effects like disorientation and hypothermia. After that, he had 1 g of sodium valproate treatment. Despite receiving neuroleptic and mood-stabilizing medications, the patient's

In this article, researchers provide an unusual instance of an anti-LGI1 encephalitis patient who primarily had a long-term psychiatric presentation.

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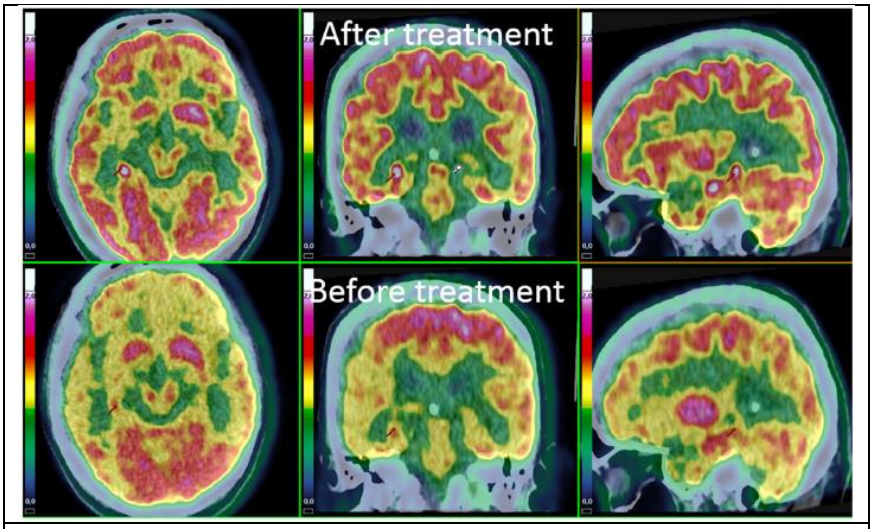
clinical appearance remained unchanged, and the cognitive symptoms grew worse. He was sent to a nursing home in August 2019 after being diagnosed with dementia and was thereafter discharged. A fresh appointment was made due to the disease's severity. One and a half years following the symptom aggravation, the patient was hospitalised to memory clinic in December 2019. The neuropsychiatric evaluation revealed sleeplessness, mood swingThe neuropsychiatric evaluation revealed sleeplessness, mood swings, apathy, and delusions with a strong Capgras syndrome. The patient maintained a regular appetite and showed no signs of hallucinations or suicidal inclinations. On the other hand, it was found that the patient had a significant attention deficit, disinhibition, episodic memory impairment, severe temporospatial disorientation, and illogical speech. A neurological examination found no evidence of ataxia, sensorimotor dysfunction, dysarthria, facio-brachial dystonic seizures, pyramidal or extrapyramidal syndrome, or dysautonomia.s, apathy, and delusions with a strong Capgras syndrome. The patient maintained a regular appetite and showed no signs of hallucinations or suicidal inclinations. On the other hand, it was found that the patient had a significant attention deficit, disinhibition, episodic memory impairment, severe temporospatial disorientation, and illogical speech. A neurological examination found no evidence of ataxia, sensorimotor dysfunction, dysarthria, facio-brachial dystonic seizures, pyramidal or extrapyramidal syndrome, or dysautonomia. Leukocytes (9/uL, normal range 0-8/uL) and protein levels (49 mg/dL, normal range 20-40 mg/dL) in the cerebrospinal fluid (CSF) were also somewhat increased. Both serum and CSF had anti-LGI1 antibodies in a positive manner. Other laboratory blood tests, including the sodium level, were all within acceptable limits. No epileptiform discharges or patterns indicative of encephalitis were observed during any of the EEGs, including one that was taken after a sleepless night. Cranial MRI showed no hyperintensities in limbic areas and only a few generalised hyperintensities on T2-weighted fluid-attenuated inversion recovery sequences. The correlation between frontal hypometabolism and temporal and striatum hypermetabolism was found during a cerebral FDG-PET scan. The results of a full-body FDGPET/CT scan were negative for structural or metabolic anomalies. Neuropsychological tests revealed frontal syndrome , significant episodic memory issues and a deficiency in the Mini Mental State Examination .Intra-venous immunoglobulins



Brain FDG-PET: axial, coronal and sagittal orientation. Before treatment: di□use frontal and temporal hypometabolism and striatum hypermetabolism. After treatment: improvement of orbito-frontal lobe and temporal hypometabolism and, to a smaller extent, of dorsolateral lobe hypometabolism. Anatomical orientation (right hemisphere on the right part of the figure). SUV ranging from 0 (blue) to 2 (red)

(IVIg) were used to treat the patient's anti-LGI1 autoimmune encephalitis at first, but there was no change in their neuropsychiatric symptoms. The patient was subsequently advised to have immunotherapy with rituximab and cyclophosphamide (rituximab on Days 1 (D1) and 15 and cyclophosphamide on Days 30 and afterwards once a month). While behavioural and delusional symptoms improved in October 2020, six months after the start of treatment, there was only a slight improvement in cognitive impairment, as seen by improvements in memory tests but persistence of frontal lobe illness. Rituximab perfusions were administered twice a year after six months of immunotherapy with no negative side effects. One year following the start of therapy, further evaluations were conducted in April 2021. Clinical assessment revealed a steady reduction in mental symptoms, normal cognition and attention, no longer experiencing mood swings or apathy, and no hallucinations or delusions. Obstructive sleep apnea (OSA) was not identified despite the continued presence of sleep disruptions. FDG-PET scans of the brain revealed a normalisation of metabolic abnormalities.

Discussion:

This case study describes a patient who presented with chronic anti-LGI1 encephalitis after a period of 1½ years of mostly manic symptoms, including unusual symptoms such cognitive impairment. After receiving immunomodulating therapy for a year, our patient clearly improved from a clinical and metabolic standpoint.[1]Three examples of individuals who presented with anti-LGI1 encephalitis and psychiatric symptoms have been documented: one with manic syndrome, one with faciobrachial convulsions, and two with psychotic symptoms and memory loss.[3,4] Contrary to two of the examples mentioned above, this patient's unique presentation delayed the diagnosis, since he had symptoms for approximately 2 years before receiving treatment.

Conclusion:

Anti-LGI1 encephalitis with a predominance of neurocognitive and manic symptoms might delay identification and treatment, resulting in a less favourable response to therapy. To effectively treat the illness and make a full recovery, it is crucial to identify warning signs of autoimmune encephalitis in individuals with unusual psychological presentations. In this article, researchers provide a rare instance of chronified anti-LGI1 encephalitis that improved clinically and radiologically after receiving second-line therapy for over a year.

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