MICRO LABS SYNAPSE

Interesting cases PSYCHIATRY

Vol 1|Issue 3|2020

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.

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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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Vitamin D Treatment for a Patient with Major Depressive Disorder- A case report.

Introduction:

Major depressive disorder is one of the common disorder prevalent in India. Lower recovery rates are seen during certain circumstances, including prominent anxiety, personality disorders, high symptom severity, and psychotic features. Diagnostic and statistical manual of mental disorders, fifth edition (DSM-5) indicates that psychotic features like delusions and/or hallucinations are present at any time within the depressive episode and discusses differentiation between the mood congruencies of these symptoms. Auditory hallucinations are most prevalent in psychotic disorders such as schizophrenia and schizoaffective disorder. Current is a case of major depressive disorder with psychotic features treated with vitamin D.

Case presentation:

Patient was a 40 year-old married male disabled veteran with major depressive disorder with severe mood incongruent psychotic features. Patient had struggled with depression for 20 years. During the initial 15 years of depression, the patient's symptoms had shown improvements with both psychopharmacologic and psychotherapeutic interventions. But for the past five years the symptoms had been more difficult to manage as he began to experience psychotic symptoms.

In context of worsening depression symptoms and auditory hallucinations, initial laboratory workup from the complete blood count, comprehensive metabolic panel, thyroid panel, lipid panel, folate, vitamin B12, and vitamin D. Results were within normal limits with the exception of mixed hypercholesterolemia and low vitamin D concentrations (23.8 ng/mL; reference level ≥ 28:9 ng/mL). CT of head which was normal.

Patient had demonstrated significant depressive symptoms with anhedonia, but the most prominent symptoms included lack of motivation, very low energy level, fatigue, and psychomotor retardation. Patient was also screened for bipolar disorder, anxiety disorders, obsessive compulsive disorder, posttraumatic stress disorder, and personality disorders, all of which were negative.

His disability compensation was secondary to a lower back injury from his previous civilian job. The patient was able to clearly recollect his first experience of auditory hallucinations at the age of 44 years after the deaths of multiple family members and losing his job. His depression was severe at that time, and he began isolating himself from the outside world. He described hearing indistinct muffled voices, as if a group of people were talking in a different room. He also stated that these voices were very difficult to distinguish and low enough that he could not understand their words. This occurred predominantly at night, though it did not have any hypnagogic or hypnopompic correlation.

Diagnosis of MDDPF was made, and the patient was started on duloxetine 20 mg/day and quetiapine 50 mg/day. Patient lost to follow-up with primary care, and the psychiatrist did not address his vitamin D level

His depression symptoms fluctuated throughout the years often linked to stressors, such as financial difficulties, marital conflict, and deaths in the family. However, with each antidepressant dosing optimization, these symptoms appeared to steadily improve without any direct correlation of change in his psychotic symptoms.

On the other hand, there was a separate encounter where he had an excellent response to his depression treatment, but his voices were more prominent, i.e., not louder or more frequent, but more clear and understandable (described as brief, infrequent, and attention seeking exclamations). The patient's duloxetine dose was gradually increased over time to 90 mg/day. During the aforementioned hospitalization, the patient was augmented with bupropion, which was eventually optimized to 300 mg/day over time. With this doses, the patient sustained a level of stability with his depression symptoms despite continued stressors and despite continued auditory hallucinations. The patient's auditory hallucinations over these years ranged from the originally described muffled voices to more complex and clear hallucinations. At times, he heard doorbells when no one was at his door and faint sounds similar to footsteps in his hallway when he could clearly see no one in the hallway.

Most disturbing hallucinations included multiple instances of a clear single, nonrecognizable, male voice using one-word exclamations like, "hey," "look," "what?" and the patient's name, typically occurring when the patient was alone or not actively engaged in a conversation. Quetiapine dose was gradually optimized to 600 mg/day before cross titrating to risperidone due to lack of effect. The risperidone dose was pushed up to 8 mg/day with little to no effect on his auditory hallucinations.

Patient was cross titrated to aripiprazole, and it was eventually dosed up to 20 mg/day. Each antipsychotic trial lasted between 9 and 18 months before switching due to little or no change in his auditory hallucinations. All the laboratory results were unremarkable with the exception of hypercholesterolemia and low 25-hydroxyvitamin D level at 22.3 ng/mL (reference level ≥ 28:9 ng/mL). Patient was not interested in changing his antidepressant medications due to his current symptomatic stability, but was willing to start vitamin D replenishment. He was started on 2000 IU/day of cholecalciferol with behavioral activation discussions, particularly referring to getting out into the sunlight more and/or obtaining a light box. Patient had had complete resolution of the psychotic features. At 15 months of follow up patient had complete absence of psychotic features for 1.5 years with duloxetine, bupropion, and cholecalciferol.

Discussion:

Vitamin D deficiency is associated with an 8% to 14% increase in depression. With Vitamin D deficiency, an increase in dopamine transporter density in the caudate putamen and increased

dopamine binding affinity in the nucleus accumbens. studies showing correlations with vitamin D and psychotic symptoms where low vitamin D levels was associated with Schizophrenia.

Conclusion:

A case of psychotic symptoms with major depressive disorder was successfully controlled with correction of vitamin D levels.

Reference:

Fipps DC et al. Psychotic Features Abated with Vitamin D Treatment in a Patient with Major Depressive Disorder. Case Reports in Psychiatry. 2020; 2046403: 1-6 pages

A case of Bipolar disorder seen with megalencephalic leukoencephalopathy with subcortical cysts.

Van der Knaap disease is a rare spongiform leukodystrophy that is characterized by macrocephaly, progressive motor dysfunction, and mild mental retardation. It is very rare for mental illness such as psychotic disorders, affective disorders and anxiety disorders to occur in MLC.

Case presentation:

A 18 year old patient was admitted to our hospital with catatonia. He was the first child of healthy parents. His grandmother was diagnosed with major depression, but no manic state, hypomanic state, epileptic attack, or catatonia. Also she had no signs of enlarged head circumference, neurological symptoms, or intellectual disability.

Pregnancy, delivery, and the neonatal period were uneventful. Patient began to walk without support at 15 months, but with a waddling gait. At the age of 10 years he had an episode of seizures and was hospitalized. Patient was then diagnosed with Megalencephalic leukoencephalopathy with subcortical cysts (MLC) based on characteristic brain imaging findings and clinical features including macrocephaly, impaired motor function with ataxia and spasticity, and mild mental retardation.

Analysis of gene revealed an S93L mutation of the MLC1 gene. He completed primary education, attending school until 16 years of age, and became depressed at 17 years of age with no precipitating factors. He then quit senior high school. Subsequently, he presented with negativism.

On admission, height was 157 cm and weight 47.4 kg. Neurological examination revealed macrocephaly with a head circumference of 58 cm and evaluated as being within the 75th percentile. He had spastic paralysis of the left leg and the Babinski reflex was present bilaterally. Gait was unstable with spasticity and ataxia. Routine blood analyses were normal. Experienced psychiatrist observed catatonia with diminished interest, insomnia, loss of energy, negativism, mutism, refusal of food, atypical mannerisms, and psychomotor agitation, not able to interview

because of his negativism. His catatonia with negativism persisted for a month after hospitalization. Present history showed depressive episode with loss of interest, insomnia, loss of energy, refusal of food, and psychomotor excitation.

Magnetic resonance imaging (MRI) showed diffuse cerebral white matter hyperintensity on T2-weighted and FLAIR images, predominantly in the frontal lobes including the subcortical U-fibers. Subcortical cysts were seen in both temporal poles. Deep white matter areas, such as corpus callosum, external capsule, and the posterior limb of the internal capsule, also involved.

In Comparison with images obtained at age 10 years, the subcortical cysts were slightly larger and cerebral atrophy had progressed slightly, although diffuse T2 hyperintensity in the cerebral white matter was unchanged. The easy Z-score (eZIS) analysis for 99mTc-ethyl cysteinate dimer-SPECT (99mTc-ECD-SPECT) revealed decreased regional cerebral blood flow in the medial and lateral prefrontal and anterior temporalcortices.

Electroencephalographic (EEG) studies showed a 2 to 3 Hz delta wave independently in the bilateral frontal area during waking and sleep. Performance IQ was 49, verbal IQ was 62, and total IQ was 52 on the Wechsler Adult Intelligence Scale-III. It was performed after remission of depression with catatonia so that the timing of the examination did not affect the results.

Olanzapine 20 mg administration was done after hospitalization. Sertraline 50 mg was added and reduced olanzapine to 5 mg because the symptoms did not improve after 30 days' admission. Catatonia significantly improved after 90 days' admission and he was discharged 4 months later.

After month, he developed hypomania with increased energy, decreased need for sleep, excessive talkativeness, and psychomotor agitation. He was diagnosed as having bipolar disorder. carbamazepine 300 mg was started and quitted olanzapine and sertraline although his depression and hypomanic states recurred about every two months. These symptoms were controlled after six months.

Discussion:

Patient's phenotype was characteristic of MLC with the MLC1 mutation until adolescence, and diagnosis had been made based on characteristic brain MRI findings and clinical features, such as macrocephaly, motor impairment with ataxia and spasticity, and mild mental retardation.

Present study revealed remarkable symmetrically decreased cerebral blood flow in the bilateral prefrontal, anterior temporal, and parietal cortex on SPECT after catatonia had set in.

Conclusion:

A case of bipolar disorder of MLC was successfully managed with medications.

Reference:

Ishikawa, M et al. Bipolar disorder in megalencephalic leukoencephalopathy with subcortical cysts: a case report. BMC Psychiatry. 2020; 20 : 349.

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Atypical Presentation of the Syndrome of Irreversible Lithium-Effectuated Neurotoxicity(SILENT)- A case report.

Lithium is one of the main stay of treatment in bipolar. It has narrow therapeutic index, lithium toxicity is a common problem. Neurotoxicity from lithium can be reversible or irreversible, monitoring of lithium levels cannot be overemphasized. The syndrome of irreversible lithium-effectuated neurotoxicity (SILENT) was coined after case reports of persistent neurologic deficits after lithium toxicity despite normalization of lithium levels Current is a case of Syndrome of Irreversible Lithium-Effectuated Neurotoxicity(SILENT) syndrome.

Case presentation:

A 54-year-old hypertensive and diabetic, a known case of PTSD and bipolar disorder (type 1) presented with altered mental status. Patient is fully functional until three weeks prior when he was reported to have episodes of agitation and night time awakening reliving his experience during the war. He was also noted to have increased consumption of fluids. He was wandering at night carrying his luggage believing that he will be taking a plane to America. He was brought to a psychiatric facility where his maintenance medications—risperidone, clonazepan, biperiden, and lithium carbonate—were continued.

He still continued to have agitation episodes, and his lithium dosage was increased from 600mg to 900mg per day. He had increasing sleeping time and with no oral intake.He presented currently with generalized seizures and went on to status epilepticus. His vital signs were as follows: BP 120/60mmHg, heart rate 137/min, respiratory rate 24/min, and temperature 36.7°C with no other abnormal systemic physical exam findings. In Interictal state, the patient was obtunded with a Glasgow coma score of 8 (E2V1M4). There were no apparent cranial nerve abnormalities, no focal weakness, and no nuchal rigidity noted. Initial work-up showed hypoglycaemia (57 mg/dL), elevated lithium (2.8 mEq/L, therapeutic range 0.8-1.2 mEq/L), elevated creatinine (10.54 mg/dL), elevated potassium (5.4 mEq/L), and low sodium (130 mEq/L), and ABGs showed combined metabolic and respiratory acidosis.

A plain CT scan was unremarkable. The patient was given intravenous valproic acid and midazolam drip for seizure control. The patient was deemed to have lithium toxicity. He underwent intermittent hemodialysis to clear the lithium. All psychiatric medications including lithium were discontinued. An electroencephalogram (EEG) on following day showed a generalized slowing of the background activity with occasional spike waves in the frontal region. After 48 hours, the patient was seizure free, and midazolam was turned off.

Lithium levels were monitored , it was decreasing level down to 0.14 mEq/L after 5 days. Kidney function was also improving. Sodium was corrected with care to avoid rapid correction. However, the patient's mental status did not improve so urine benzodiazepine levels were obtained which was elevated at >10,000 ng/mL. Intermittent hemodialysis was continued and urine benzodiazepine level was monitored until it was below the threshold. Despite clearance of benzodiazepine and lithium (now at 0.01 mEq/L), the patient did not regain full consciousness.

The patient had eye opening and withdrawal to pain. Subsequent EEGs still showed a generalized theta slowing and with no more epileptiform discharges seen. Repeat cranial CT scan did not show any new findings. An MRI was not done due to a relative contraindication of the presence of a lodged bullet. He was voiding adequately no longer requiring hemodialysis. After 2 months of lithium cessation, the patient can now open his eyes and move his limbs spontaneously, but he still does not follow commands and has no purposeful activity. After 3 months, the patient still had the same mental status.

Discussion:

Lithium is distributed in most body tissues but with an uneven rate of distribution among different tissue compartments. It does not undergo liver metabolism but is renally cleared. Its half-life varies according to age and glomerular function. Toxicity of lithium may occur with excessive intake or impaired excretion seen with renal insufficiency, low sodium diet, and congestive heart failure. Long-term neurologic sequelae are considered persistent when symptoms are present for at least 2 months after the cessation of lithium.

Conclusion:

Syndrome of Irreversible Lithium-Effectuated Neurotoxicity is a disabling but preventable neurologic sequela of lithium toxicity, can cause persistent encephalopathic state.

Reference:

Senga MM et al. A Case Report on an Atypical Presentation of the Syndrome of Irreversible Lithium-Effectuated Neurotoxicity (SILENT) in a War Veteran with Bipolar Disorder and PTSD. Case Reports in Psychiatry. 2020; 5369297:1-4 pages.