**Psychiatric Case Series**

**Vol 6 | Issue 27 |2025**

**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. **Case report of Depression in an older patient with dysgeusia as the first symptom**
2. **Case report of Fahr's syndrome accompanied by hypoparathyroidism, schizophrenia and iron deficiency anemia**
3. **Case Report on Manic Episode After Dengue Fever**
4. **Case Report on Psychosis in Laurence-Moon Syndrome**

**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,**

## 1. Case report of Depression in an older patient with dysgeusia as the first symptom

**Introduction:**

**Dysgeusia symptoms, which are easily mistaken for oral disease, are uncommon in geriatric depression, which frequently presents with a variety of physical symptoms.**

Depression is one of the most common psychiatric disorders and the major cause of disability globally. Researchers and the general public have given depression a lot of attention, especially because of its connection to suicide. Depression affects an estimated 280 million individuals of all ages globally, including 5 % of adults.[1] Clinical signs include melancholy, grief, and even numbness. Approximately one-third of older adult patients experience physical discomfort as their first symptom; however, dysgeusia as the initial symptom is uncommon in clinical practice. Dysgeusia is a clinical symptom with no distinct objective sign, which increases the risk of misdiagnosis and missed diagnosis.[2] The purpose of this case report was to explore the clinical and pathological condition, as well as the rate of depression diagnosis and treatment in older adults. Also, this case shows the digestive system as the initial symptom of geriatric depression, resulting in fewer misdiagnosis and treatment.

Dysgeusia

**Case Presentation:**

A 60-year-old female homemaker with elementary school as her highest level of education reported to the outpatient department due to dysgeusia, poor sleep, and low mood that had persisted for more than one year. The issues intensified in the two weeks before her hospital visit. The patient had chronic hiccups, a rush of gas inside the body, persistent dysgeusia that was not relieved by consuming sweet foods, a poor appetite, and a depressing feeling. Digestive pills, domperidone, and omeprazole was not effective. Further, the patient described feeling tired and weak, having a decreased ability to function, and continuously informing those around her about the dysgeusia. The patient also had difficulties sleeping and often urged her relatives to take her to the hospital to have the dysgeusia treated. The patient stated two weeks before to her hospital admission that she felt bitterness in her heart and had repeated crying episodes.

She also had suicidal thoughts on occasion, did not feel capable of caring for her children, rested at home, and lost significant weight (from 61 kg pre-illness to 52 kg). The patient had no prior history of a similar condition and denied a family history of mental illness in the prior two generations. She exhibited stable vital signs. The psychiatric examination revealed clear consciousness; the patient was clean and appropriately dressed, and she indicated self-care behaviors; yet she expressed worry. The patient took the initiative to explain the dysgeusia, particularly on the right side of the mouth and saw a transient relief after eating sweets. The patient provided direct responses to inquiries, and had a calm voice, and a decent orientation, but had symptoms of hypochondria, depression , and anxiety. She also had low energy, memory, appetite, and weight, as well as feelings of hopelessness, suicidal ideation, sleep problems, and a lack of self-awareness. Examination indicated taste anomalies. In terms of psychological assessment, the 32-item hypomania symptoms checklist yielded a total score of 6, indicating no previous hypomanic episodes. The Hamilton Depression Scale-24 (HAMD-24) (Figure 1) score was 36, suggesting significant depression; the Hamilton Anxiety Scale (HAMA) score was 17, showing anxiety. The Coping Style Questionnaire score of 151 points suggests an immature coping style.

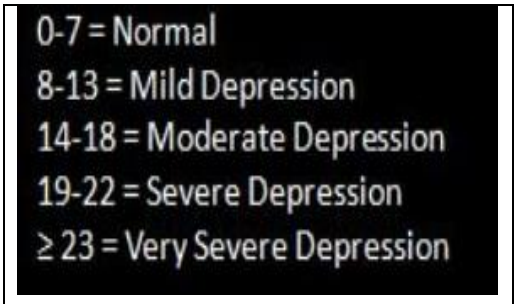


Figure 1: Hamilton Depression Rating Scale (HAM-D) scores

Based on the patient's history, psychological evaluation results, and psychiatric examination results, a diagnosis of major depressive episode with psychotic symptoms was made using the International Classification of Diseases (10$^{th}$ edition) diagnostic criteria. Treatment involved starting with 5 mg of escitalopram per day and progressively increasing it to 10 mg per day. Alprazolam (0.4 mg/night) was also administered. Simultaneously, psychological treatment was initiated, and the patient was informed about the association between physical discomfort, such as dysgeusia, and a bad mood. The family also received psychological education to help them understand and support each other. Two weeks later, the patient returned to the clinic and reported that her sleep had substantially improved, her dysgeusia had decreased, her mood was less irritable, her diet had improved, and her HAMD-24 and HAMA scores were 27 and 10, respectively. Four weeks later, the symptoms had greatly improved: dysgeusia was mostly absent, sleep was normal, mood and appetite had improved, and the patient was able to conduct tasks and care for her children. At this point, the HAMD-24 and HAMA scores were 14 and 7, respectively.

Alprazolam was gradually withdrawn, while escitalopram was maintained at a daily dose of 10 mg. By the 8th and 16th weeks of treatment, the HAMD-24 scores had dropped to 8 and 5 respectively. The patient has been followed up in the outpatient department for more than two years, takes his prescription on time, has a stable mood, and gets enough sleep. There have been no additional complaints of dysgeusia, and the patient can complete home activities and care for her children. The patient is still taking escitalopram at a dose of 10 mg per day and has no other difficulties.

**Discussion:**

Depression is the most frequent mental disorder among elderly persons. The patient in this study experienced dysgeusia as her initial symptom, which is extremely uncommon in clinical practice. In the absence of salty foods, dysgeusia can be regarded as a phantom taste symptom. Approximately one-third of patients over 60 years old suffer physical discomfort as the initial symptom, which frequently involves the digestive system and manifests as belching, acid reflux, and bloating.[2] A previous study showed that when depression was severe, some elderly individuals were more likely to develop hypochondria and report physical discomfort, which can lead to hypochondriac hallucination.[3]

**Conclusion:**

Physical discomfort is a common complaint among elderly individuals with depression. If physical symptoms persist despite medical treatment, it may be necessary to analyze the patient's mental condition or refer them to a psychiatrist to diagnose geriatric depression. For older adults, prompt pharmaceutical and psychological therapy is the recommended course of treatment.

**References:**

1. Chang L, Wei Y, Hashimoto K. Brain-gut-microbiota axis in depression: A historical overview and future directions. Brain Res Bull. 2022 May;182:44-56.
2. Guo L, He Z. Case report: Depression in an older patient with dysgeusia as the initial symptom. Front Psychiatry. 2024 Dec 10;15:1478359.
3. Ferraris C et al. Liking of salt is associated with depression, anxiety, and stress. Chem Senses. 2023 Jan 1; 48:1-13.

## 2. Case report of Fahr's syndrome accompanied by hypoparathyroidism, schizophrenia and iron deficiency anemia

**Introduction:**

**Clinicians must compare Fahr's syndrome with psychiatric conditions to increase their understanding and provide patients with the appropriate diagnosis and treatment regimens.**

Fahr's syndrome is a rare neurological condition, with an incidence of less than one per million people. It is primarily distinguished by aberrant intracranial calcifications in areas such as the basal ganglia, dentate nucleus, and cerebral cortex. Fahr's syndrome has a complicated etiology, with both hereditary and secondary causes, including calcium and vitamin D metabolism disorders, parathyroid dysfunction, and toxic exposure. Also, one of the most prevalent secondary causes is parathyroid-related illnesses, such as idiopathic hypoparathyroidism (IHP) (low serum calcium and PTH deficiency), autoimmune hypoparathyroidism, pseudohypoparathyroidism (PHP), pseudopseudohypoparathyroidism (PPHP), and hyperthyroidism.[1] Chronic hypocalcemia causes ectopic calcification in the brain, worsening neurological and mental disorders. Fahr's syndrome rarely coexists with mental illnesses, but its neuropsychiatric changes might mimic primary psychiatric disorders like schizophrenia, creating diagnostic challenges.[2] Here, a 45-year-old woman with schizophrenia was diagnosed with Fahr's syndrome, which can be difficult to diagnose due to accompanying neurological conditions.

**Case Presentation:**

A 45-year-old female with a history of iron deficiency anemia and schizophrenia presented to the emergency department after experiencing loss of consciousness while walking. The patient fell and sustained a 1-inch full-thickness laceration in the occipital region. According to the patient's history, the patient also experienced a tonic-clonic seizure episode of undetermined duration following the fall. The patient was not on any medications during the episode. The patient's blood pressure was 139/80 mmHg, and pulse rate was 80 beats per minute, respiration rate was 17 beats per minute and SpO2 was 98% on room air. The patient denied experiencing convulsions, dizziness, or blurred vision prior to the occurrence. There was no evidence of tongue biting, feces, or urine incontinence. The patient's neurological assessment showed a Glasgow Coma Scale (GCS) score of 15/15, indicating alertness and complete consciousness. Somatosensory function was intact, with normal motor strength (5/5) and reflexes (2+). Coordination and gait were normal, suggesting no neurological issues. The patient had

hypocalcemia (5.0 mg/dL) and a positive toxicological test for cannabis. Echocardiography revealed an ejection fraction of 55%-60% and normal left ventricular systolic function. The electrocardiogram (ECG) exhibited diffuse T-wave inversion and no acute events on telemetry. On admission, her calcium level was 5.0 mg/dL (Table 1). Her PTH level was 9.4 pg/mL, with a vitamin D 25-OH level of 17.7 ng/mL. Phosphorus levels were elevated, at 6. The patient's hemoglobin level was 7.2 g/dL at admission. Fecal occult blood was found to be negative. The fall was connected to an isolated seizure induced by severe hypocalcemia rather than Fahr's syndrome or anemia. The patient was admitted for further treatment of hypocalcemia and anemia. Intravenous calcium gluconate was administered, resulting in blood calcium levels reaching 7.0 mg/dL within 2 days. To treat iron deficiency anemia, intravenous iron (ferrous sulfate 400 mg/day) was administered, and two units of packed red blood cells were transfused, resulting in hemoglobin levels of 9.7 g/dL. The hemoglobin level stabilized at 9.7 g/dL. A brain CT scan revealed bilateral basal ganglia calcifications as hyperdense regions (Figure 1). A brain MRI revealed basal ganglia mineralization, sparing the caudate head and primarily involving the pallidum (Figure 2). There were also small areas of white matter mineralization in the deep white matter of the frontal lobes in the centrum semiovale and the cerebellar hemispheres bilaterally. A cardiology consultation recommended using an extended event monitor or loop recorder. Routine outpatient follow-ups were advised, along with avoidance of drugs that could prolong the QT interval. The patient was discharged with recommendations to consult with an endocrinologist, cardiologist, and primary care physician regarding her findings.

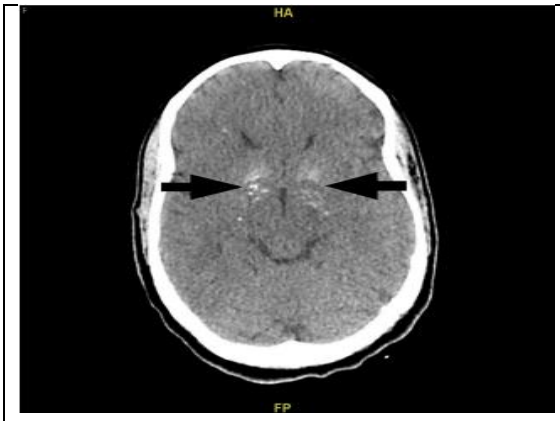


Figure 1. Brain CT scan reveals bilateral basal ganglia calcification, resulting in hyperdense regions.

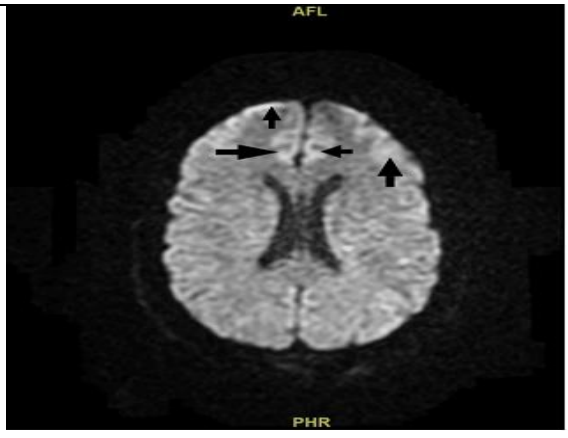


Figure 2. Brain MRI reveals tiny mineralizations in the frontal lobe white matter, centrum semiovale, and cerebellar hemispheres, as well as bilateral basal ganglia mineralization, primarily in the pallidum, with the caudate head spared.

**Discussion:**

Fahr's syndrome, also known as PFBC, and schizophrenia are both difficult to diagnose and manage due to similar clinical symptoms and involvement of multiple factor pathways. Fahr's syndrome is characterized by calcification in brain tissue, particularly in the basal ganglia, thalamus, and cerebellum.[2] Calcification is linked to genes involved in phosphate control and vascular tropism, such as SLC20A2, PDGFB, PDGFRB, and XPR1.[3] Treatment includes symptomatic support and the identification of causes, but there is no specific treatment for preventing the growth of calcification in the basal ganglia. Early treatment, particularly in hypoparathyroidism, can help prevent calcification and neurophysiological problems.[4]

**Conclusion:**

There is no known treatment for Fahr's syndrome, and the course of the condition is determined by the intensity and course of symptoms. Early intervention is crucial in managing metabolic alterations that would otherwise result in neurological impairment. Treatment for relapsing-remitting psychiatric diseases, such as schizophrenia, tries to reduce symptoms and disability in the long term. Comparing Fahr's syndrome to mental disorders helps physicians diagnose and treat patients with these conditions more accurately.

**References:**

1. Wang X et al. Fahr's Syndrome with Pseudohypoparathyroidism: Oral Features and Genetic Insights. Int J Mol Sci. 2024 Oct 29;25(21):11611.
2. Haider A et al. Fahr's Syndrome With Hypoparathyroidism in a Patient With Schizophrenia and Iron Deficiency Anemia: A Case Report. Clin Case Rep. 2025 Jan 6;13(1):e70093.
3. Balck A et al. Genotype-Phenotype Relations in Primary Familial Brain Calcification: Systematic MDSGene Review. Mov Disord. 2021 Nov;36(11):2468-2480.
4. Arruda ACG et al. Hypoparathyroidism and Fahr's syndrome: case series. J Bras Nefrol. 2022 Oct-Dec;44(4):592-596.

## 3. Case Report on Manic Episode After Dengue Fever

**Given the rising global incidence of dengue, integrating psychiatric assessments into post-dengue management could aid in early diagnosis and management of neuropsychiatric complications.**

**Introduction:**

Dengue is the second most frequent mosquito-borne disease affecting humans, following malaria. Clinical signs range from mild to severe hemorrhagic fever, which is transmitted by mosquitos of the Aedes genus. A change in the range of clinical manifestations has recently been observed, with neurological signs being recognized more frequently. The most prevalent neurological consequences of dengue are encephalopathy and encephalitis, which have an estimated prevalence of 0.5 to 6.2%.[1] Less commonly, Reye's syndrome, meningoencephalitis, paresis, seizures, and stiff necks may occur. Dengue fever can cause unusual psychiatric symptoms such as excessive talkativeness, euphoria, increased activity, grandiosity, decreased sleep and food intake, irritability, restlessness, and aggression. Post-infection symptoms may include hallucinations, delusions, amnesia, and dementia. Estimating the prevalence of neurological and psychiatric symptoms in adults with dengue fever is challenging due to limited documentation in case series and reports.[2] In this case report, a 22-year-old man experienced manic and psychotic symptoms after recovering from dengue fever, including sleeplessness, irritability, grandiosity, and auditory hallucinations.

**Case Presentation:**

A 22-year-old healthy male presented to the Emergency Department of a general medical facility with a five-day history of fever, nausea, non-bilious vomiting, diffuse stomach discomfort, headache, body pains, and hemoptysis. He denied having epistaxis, diarrhoea or melena. The patient occasionally smokes cigarettes and drinks alcohol. He denied having any recent travel history. There is no prior psychiatric history or family history of psychiatric disorders. On physical examination, the patient's Glasgow Coma Scale (GCS) score was 15/15. He had no pallor, jaundice, or lymphadenopathy, but did show minor indications of dehydration. The patient's chest was clear, with equal bilateral air entry and normal heart sounds (S1 and S2). The patient's belly was soft with mild discomfort, no organomegaly, and no lower limb edema. The neurological examination revealed no significant impairments. Blood laboratory tests revealed a positive dengue virus IgM result. The patient had a white blood count of 4.99 $x10^3$ /mcL and thrombocytopenia with a platelet count of 40 $x10^3$ /mcL. The patient underwent symptomatic treatment, including IV fluids, acetaminophen 1 gram once daily, pantoprazole 40 mg once daily, and ondansetron 4 mg if necessary (Table 1). After six

days, he was discharged as afebrile with a platelet count of 104 x $10^3$ /mcL. Within a week of being discharged from the hospital, the patient admitted to the Emergency Department with a noticeable shift in his behavior. The patient's escorts reported that a day after discharge, he experienced significant insomnia, irritability, and violence toward relatives and roommates. Symptoms immediately worsened, including social disinhibition, decreased hunger, talkativeness, rapid speech, high self-esteem, and increased smoking. The patient exhibited impulsive conduct, including excessive spending outside of his regular routine. On presentation, his Mental Status Examination (MSE) revealed substantial euphoric affect, which contrasted his claimed bad mood and grandiose attitude and ideations. He demonstrated poor judgment and understanding by expressing homicidal thoughts, including the intention to use a knife on an undefined individual. A non-contrast CT scan results were normal for intracranial pathology. He was transported to a tertiary psychiatric facility. To sedate him before transfer, he was given IM haloperidol 5 mg and IV diazepam 10 mg. To address his manic symptoms, he was prescribed olanzapine at a dose of 5 mg once day at bedtime (Table 1). His Young Mania Rating Scale (YMRS) score at admission was 54. The individual scored 95 on the Positive and Negative Syndrome Scale (PANSS), with positive subscales of 37, negative subscales of 12, and a general psychopathology subscale of 46. According to the DSM-5-TR, the individual was diagnosed with bipolar disorder because of another medical illness. He claimed having commanding auditory hallucinations while in the inpatient unit. He had persecutory delusions, believing he was being watched, plotted against, and his safety was at risk. In addition, he had grandiose beliefs, claiming immense wealth and the ability to buy large assets like skyscrapers. He was noted to be very familiar with hospital staff and other patients. He was noted to be agitated, animated, and exuberant. As a result, olanzapine was increased to 10 mg at bedtime and bromazepam to 1.5 mg twice daily (Table 1). His symptoms entirely resolved after ten days in the inpatient setting. His sleep pattern improved, appetite restored, manic and psychotic symptoms subsided, and murderous ideas stopped. He was discharged with olanzapine 10 mg once daily at bedtime. His Young Mania Rating Scale (YMRS) score at discharge was 2. During a follow-up appointment one week after discharge, he was found to be doing well, with no signs of manic or psychotic symptoms. He was eating and sleeping well, with no unusual behavior seen. He returned to work and resumed normal functioning.

| Medication | Drug Class | Route | Dosage | Frequency | Duration | Notes |
|---|---|---|---|---|---|---|
| Ondansetron | 5-HT3 antagonist (for nausea and vomiting) | Intravenous | 4 mg | OD | 6 days | Administered during hospitalization at the field hospital. |
| Pantoprazole | Proton-pump inhibitor | Intravenous | 40 mg | OD | | |
| Acetaminophen | Analgesic and antipyretic | Intravenous | 1,000 mg | OD | | |
| Olanzapine | Second-generation antipsychotic | Oral | Initially 5 mg daily at bedtime, then titrated to 10 mg daily at bedtime | HS | 10 days | Administered during hospitalization at the tertiary psychiatric facility. |
| Bromazepam | Benzodiazepine | Oral | 1.5 mg | BID | | |

Table 1. A chronological record of the drugs given to the patient while admitted to the tertiary psychiatric health facility and the dengue field hospital (OD, once daily; HS, at bedtime; BID, twice daily)

**Discussion:**

This case report describes a manic episode in a previously healthy young male who had dengue fever. Neuropsychiatric symptoms of dengue fever have a complicated and multifactorial pathogenesis. The mechanism by which dengue fever causes psychiatric problems are not fully known. Several theories suggest that viral encephalitis, metabolic abnormalities, and neuroinflammatory processes could play an important effect.[2] A previous study showed that the dengue virus's immunological response, including the release of cytokines and inflammatory mediators can impair brain function and cause psychiatric symptoms.[3]

**Conclusion:**

This case report highlights the potential for substantial psychiatric manifestations after dengue fever. It advocates for improved clinical awareness and the necessity of psychiatric examinations in managing dengue fever, particularly in endemic locations. Further research is needed to understand the complicated pathophysiology behind dengue's neuropsychiatric consequences.

**References:**

1. Trivedi S, Chakravarty A. Neurological Complications of Dengue Fever. Curr Neurol Neurosci Rep. 2022 Aug;22(8):515-529.
2. Bokhari SA et al. Manic Episode Following Dengue Fever: A Case Report. Cureus. 2024 Sep 4;16(9):e68630.
3. Dinakaran D et al. Dengue and Psychiatry: Manifestations, Mechanisms, and Management Options. Indian J Psychol Med. 2022 Sep;44(5):429-435.

## 4. Case Report on Psychosis in Laurence-Moon Syndrome

**A multidisciplinary strategy that includes psychiatry, neurology, endocrinology, and metabolic care is necessary for effective treatment of Psychosis in Laurence-Moon Syndrome.**

**Introduction:**

Laurence-Moon syndrome (LMS) is a rare autosomal recessive genetic multisystem illness with neurological, ocular, and endocrine problems.[1] It presents as a congenital ciliopathy with a wide range of primary and secondary clinical characteristics. This disorder causes a rapid deterioration in the functionality of the brain, eyes, kidneys, hands, and feet. The syndrome's defining characteristics include mental retardation, atypical retinitis pigmentosa, hypogonadism, polydactyly, obesity, and renal dysfunction.[2] LMS has clinical similarities with other hereditary illnesses such as Bardet-Biedl syndrome (BBS) and Oliver-McFarlane syndrome (OMS). Diagnosing and managing these illnesses requires careful differentiation due to their unique clinical features. Psychiatric comorbidities in LMS patients complicate clinical management due to the intricate interplay between neurodevelopmental and psychiatric symptoms. Understanding these complexity is critical for developing better treatment strategies and patient outcomes.[1] This case report describes a 35-year-old man with LMS experienced psychological symptoms such as social isolation and limited family interaction, which increased over the past two years due to eyesight impairment.

**Case Presentation:**

A 35-year-old single Saudi male who was born from a consanguineous marriage and graduated with a bachelor's degree experienced developmental problems, including delayed speech acquisition, and pronounced his first word at the age of 4. He struggled with color recognition, item identification, and speaking clarity. He had weak eyesight, requiring regular glasses replacements, and eventually became blind at the age of 33. The patient has a medical history of LMS and had a laparoscopic cholecystectomy four years ago. His family includes his parents, both teachers aged 60, and five siblings, one of whom has LMS. Physical examination revealed slightly raised blood pressure (133/97 mmHg) and a resting tremor in the patient's right hand. He was 171 cm tall, weighs 150 kg, and has a BMI of 51, which indicates obesity. Laboratory tests revealed higher levels of total cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol, indicating a high risk of atherosclerosis (Table 1). This is particularly concerning due to the presence of additional risk factors like obesity. The patient's psychiatric symptoms began at age 15 and included social isolation and less involvement with family and friends. His illness worsened considerably two years ago, at the age of 33, when he

started experiencing distressing auditory hallucinations. He described hearing voices speaking about him and his family, especially his mother. These voices led to a subsequent delusional idea that his mother had a sexual relationship with his cousin and engaged in other sexual acts. This idea heightened his anxiety and prevented him from sleeping alone. As his hallucinations worsened, he became impatient and aggressive towards his mother.

He also threatened violence against his cousin, believing he had overheard him speaking with his mother. Furthermore, the patient had tactile hallucinations, as if ants were crawling on his body. For two months, he neglected basic needs including eating, drinking, and daily activities. After constant parental requests, the patient received medical attention and was diagnosed with LMS-related psychosis. Initially taken aripiprazole and eventually increased to 20 mg daily, he experienced great relief in symptoms but developed a resting tremor in his right hand. After reducing his dosage to 10 mg daily, he had a return of hallucinations. He is currently taking 15 mg of aripiprazole each day, which has helped to stabilize his condition. A trial of escitalopram 10 mg was done to decrease negative symptoms of his psychotic condition, but it did not result in significant improvement and was stopped. The patient appeared to have a normal mental condition for his age. He was helpful, but had terrible eye contact due to his blindness. There was no indication of psychomotor agitation or retardation. His speech had regular tempo, rhythm, and volume. His cognitive process was clear and goal-oriented, with no suicidal or murderous thoughts. The patient refused to cooperate during the examination, thus no formal cognitive testing was conducted. The patient's overall presentation indicated cognitive impairment, including difficulty with attention, processing speed, and executive functioning, as well as a history of developmental delays and learning challenges. This patient has a difficult case of LMS and subsequent psychosis, with physical and psychiatric issues. His medication regimen is being modified to improve symptom control while reducing negative effects. Effective management of his disease requires continuous monitoring and multidisciplinary support.

| Test Name | Result | Reference Range |
|---|---|---|
| Total cholesterol | 6.68 mmol/L | <5.17 mmol/L |
| HDL cholesterol | 1.16 mmol/L | >1.55 mmol/L |
| LDL cholesterol | 4.54 mmol/L | <2.60 mmol/L |
| Triglyceride | 2.15 mmol/L | <1.70 mmol/L |

Table 1. Lipid profile test results

**Discussion:**

LMS is a rare autosomal recessive genetic condition causing neurological, visual, and metabolic problems. Coexistence of LMS with psychiatric diseases like psychosis complicates

treatment and requires a nuanced approach. Antipsychotic medicine, notably aripiprazole, initially improved symptoms but caused a resting tremor, a known side effect. Brexpiprazole, cariprazine, ziprasidone, and aripiprazole are among the recommended metabolically sparing agents.[1] A previous study showed that adjusting aripiprazole dosage due to side effects highlights the challenge of controlling secondary psychiatric symptoms including hallucinations and delusions caused by LMS. The recurrence of psychotic symptoms after dosage reduction highlights the need to balance effective antipsychotic medication with addressing primary LMS symptoms including obesity and cognitive deficits.[3]

**Conclusion:**

This case study offers useful insights on managing psychosis in people with LMS. A multidisciplinary strategy that includes psychiatry, neurology, endocrinology, and metabolic care is necessary for effective treatment. The study suggests genetic testing and counseling for patients' families, especially during family planning, and non-invasive prenatal testing (NIPT) for siblings after marriage.

**References:**

1. Al Fareh N, Abbas M, Alosaimi FD. Psychosis in Laurence-Moon Syndrome: A Case Report. Cureus. 2024 Oct 21;16(10):e72003.
2. Munib N, Haider I, Malik SE. Laurence-Moon-Bardet-Beidl Syndrome: Case Report. Journal of Medical Sciences. 2022 Sep 30;30(3):227-9.
3. Khan OA et al. Rarity of Laurence Moon Bardet Biedl Syndrome and its Poor Management in the Pakistani Population. Cureus. 2019 Feb 21;11(2):e4114.