MICRO LABS SYNAPSE

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Interesting Cases PSYCHIATRY

Psychiatric Case Series

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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 of Charles Bonnet Syndrome**
2. **A case of 66-year-old male patient with a pacemaker who received 13 months of maintenance ECT therapy.**
3. **A case of schizophrenia psychosis clinically diagnosed as Rubinstein-Taybi syndrome but resolved as carrying a 22q11.2 deletion.**
4. **Diagnostic challenges in the identification of elevated acute phase protein levels in a drug addicted female adolescent**

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 of Charles Bonnet Syndrome

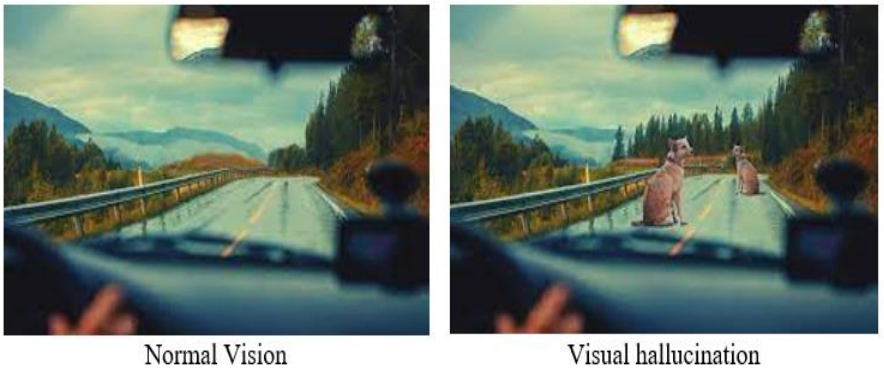
Introduction:

Charles Bonnet syndrome (CBS), also known as Visual hallucinations ordischarge hallucinations, can occur in nonpsychotic people with vision impairment. Vivid and complex visual hallucinations are a hallmark of the otherwise cognitively healthy but visually impaired Charles Bonnet syndrome.[1]The majority of the time, patients are persons who have eye disease, optic nerve damage, or vision impairment brought on by ageing. Only the visual sense is disturbed; the other senses are unaffected. The inferotemporal cortex also experiences transient hyperactivity.[2] In order to highlight diagnostic and therapeutic concerns and to share their experience, Researchers publish the case of a 65-year-old woman below.

Management suggestions are limited in Charles Bonnet syndrome, which explains this clinical case's significance and the necessity for research in this field.

Case Presentation:

A 65-year-old widowed patient who had a posterior cerebral fossa tumour surgically removed 20 years prior and had permanent sequelae blindness underwent routine follow-ups with her neurosurgeon and ophthalmologist up until there was a change in her condition. After an uneventful evaluation at age one, the patient's ophthalmologist decided to refer her for a psychiatric appointment since she was experiencing escalating visual hallucinations. The patient was treated with two different antipsychotics (AMISULPRIDE 400 mg/day and OLANZAPINE 10 mg/day) while being monitored by a psychiatrist for a year, but there was no change. The patient was seen in consultation in our department and reported complex visual hallucinations of the kind of small characters that annoy her and occasionally drive her to act, something that may cause damage or occasionally injury. The patient was calm, cooperative, well-oriented in time and space, coherent, and able to speak clearly. The request for a neurological opinion or a clinical and para-clinical evaluation that revealed no abnormalities was made. A regimen based on CARBAMAZEPINE 200 mg twice day plus RISPERIDONE 1 mg daily was initiated. When control was achieved one month later, the patient's and her



Example of Charles Bonnet syndrome

family's reported hallucinations and accompanying behaviours had almost entirely disappeared. The patient was stable on the same dosages during the follow-up.

Discussion:

Visual illusions in conscious people with decreased visual acuity or visual field loss and no underlying psychiatric disorder are consistent with Charles Bonnet syndrome. Afferents in the visual cortex are lacking when a person loses their vision, and this results in hyperexcitability of the neurons in the visual cortex, according to a commonly accepted theory. Outside of the visual system, deafferentation or denervation-induced hyperexcitability is a phenomena. Thus, spontaneous neuronal discharges could be assessed in vitro and in brain regions that had been isolated at the neuronal level. The involvement of abnormal spontaneous activity in Charles Bonnet syndrome is uncertain.[2] The therapeutic method is based on case studies. There is insufficient evidence to support the use of antipsychotics (for example, olanzapine, quetiapine), cholinesterase inhibitors (for example, donepezil), serotonin reuptake inhibitors (for example, escitalopram), and antiepileptics (e.g. carbamazepine, clonazepam, valproate). Some patients are able to resist the illusions by blinking or moving their eyes quickly. However, this patient responded effectively to CARBAMAZEPINE PLUS RISPERIDONE. Therapeutic data are inadequate, possibly due to the rarity of complex and serious types, as well as the difficulty of making a series of cases given the dispersion of patients among different specialists (ophthalmologist, neurologist, and psychiatrist), as well as the majority of cases being simple.

Conclusion:

CBS remains a condition, particularly in the elderly, with a significant visual loss and no underlying psychological disease. Management suggestions are limited, which explains this clinical case's significance and the necessity for research in this field.

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2. A case of 66-year-old male patient with a pacemaker who recieved 13 months of maintenance ECT therapy.

Introduction:

Electroconvulsive therapy (ECT) is a therapy that can be very successful for a variety of mental problems that don't respond well to medication or other psychosocial treatments.The elderly are highly susceptible to drug side effects, and ECT is increasingly being used on this patient population. Cognitive adversity with ECT, on the other hand, is a major clinical problem that is usually mild and transient, but is thought to occur more commonly in older patients.[1] Electroconvulsive treatment has the possibility of affecting cardiac electrical activity, resulting in asystole or pulseless electrical activity (PEA), especially in older individuals with irreversible structural heart disease. Pacemakers, on the other hand, have been found to have the capacity to reverse ECT-induced cardiac arrest. Furthermore, recent information shows that the length of the ECT electrical stimulus is insufficient to elicit clinically meaningful pacemaker events, particularly in advanced pacemakers.[2] Researchers provide a case study of a depressed patient with a fixed-rate dual-chamber (DOO) mode pacemaker who received maintenance ECT therapy.

A fixed-rate mode pacemaker may be effective in the long-term therapy of Electro Convulsive Therapy, even in the elderly.

Case Presentation:

This is a 66-year-old man with a history of major depressive disorder (MDD) and generalised anxiety disorder (GAD). Prior to starting ECT, he had three severe depression episodes in his life, with lingering symptoms in between, culminating in two mental hospitalizations. He had sufficient trials of eight different psychotropic drugs for depressive symptoms and therapy augmentation, including escitalopram, fluoxetine, bupropion XL, mirtazapine, levomilnacipran, asenapine, brexpiprazole, and aripiprazole. All resulted in either insufficient responses or unacceptable adverse effects. Treatment-resistant depression was detected and ECT was advised due to his history of failing many antidepressant regimens. His medical history includes asthma, obstructive sleep apnea, kidney stones, type 2 diabetes, hypertension, aortic valve replacement, and, most crucially, the installation of a cardiac pacemaker. He received a Boston Scientific L101 pacing device nineteen months before his ECT examination. The device was set to DOO mode, with a target heart rate of 60 beats per minute, following manufacturer and cardiology recommendations. Additional instructions included applying a magnet to the pacemaker during the ECT operation and waiting for Boston Scientific staff to access the pacemaker function. Due to practical problems with the availability of device representatives, the pacemaker

settings remained unchanged throughout all 13 months of ECT treatments. Before starting ECT, a chest x-ray revealed appropriate pacemaker installation and no acute pulmonary or cardiac problems. The patient reported depressive symptoms while using escitalopram 10 mg po daily and brexpiprazole 3 mg po daily at the time of the initial ECT examination. He was taking metformin 500 mg po twice a day, atorvastatin 40 mg po daily, and tamsulosin 0.4 mg po daily for his medical comorbidities. He scored a 7 on the Patient Health Questionnaire —9 (PHQ-9) at baseline, suggesting moderate depression. However, because he fit the criteria for treatment-resistant depression due to many failed antidepressant treatments, ECT was explored. The patient had 5 index and 18 maintenance bilateral-ECT treatments (ECT machine: Mecta Spectrum 5000Q). Patient received weekly ECT for 4 weeks, then gradually decreased to biweekly for 2 weeks before being maintained on monthly ECT therapy. The parameters of ECT were average seizure duration of 35.6 seconds (ranging 21-79 seconds), average stimulus train duration of 8 seconds (8 seconds for all except 3.5 second once), average energy level of 57.6 joules (ranging 22.5-101.4), pulse width of 0.789 (ranging 0.5-1), and current of 800 MA in all sessions. Prior to each session, the patient received 500 mg of IV caffeine, 30mg of ketorolac (Toradol), 150 mg of methohexital (Brevital), and 160 mg of succinylcholine. His PHQ-9 score dropped to 0 after the initial index treatments and remained there for the whole 12 months of maintenance treatment. The patient underwent 23 ECT sessions in all, and his final PHQ-9 score remained zero, suggesting effective remission of depressive symptoms. Despite the lack of pacemaker interrogation between sessions, no problems were noted and no parameter changes were required over the duration of therapy.

Discussion:

Previous research reveals that using ECT with a pacemaker is usually deemed safe, but the lack of controlled trials forces the procedure's recommendations to be dependent on sporadic case reports. A substantial number of instances showed that synchronised pacing during ECT may be better than asynchronous. However, the majority of these case reports require intensive monitoring or regular inquiry by medical personnel trained in pacemaker administration, as well as subsequent reprogramming between sessions.[2]A similar case reported by Mędrala et al also concludes that the use of ECT is safe and that it is critical to offer adequate care for a patient from a person who is capable of operating pacemakers. A pacemaker must be examined on a regular basis.[3]

Conclusion:

This instance demonstrates that, in certain circumstances, a fixed-rate mode pacemaker may be effective in the long-term therapy of ECT, even in the elderly. Furthermore, precautions must be undertaken to reduce the risk of the most common cardiac problems found in pacemaker patients receiving ECT.

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4. A case of schizophrenia psychosis clinically diagnosed as Rubinstein-Taybi syndrome but resolved as carrying a 22q11.2 deletion.

Introduction:

Rubinstein-Taybi syndrome (RTS) is a rare genetic condition characterised by growth delay, phenotypic facial traits, microcephaly, developmental delay, broad thumbs, and huge toes.[1] Psychotic symptoms in RTS are uncommon, with only a few recorded examples. The most prevalent chromosomal microdeletion in humans, on the other hand, is 22q11.2 deletion syndrome. The 22q11.2 deletion has long been known to be a risk factor for schizophrenia.[2] Here is a case of schizophrenic psychosis that was clinically diagnosed as RTS but was later shown to have a 22q11.2 deletion by genetic research.

The psychological symptoms and genomic analysis might help identify 22q11.2 deletion syndrome in undiagnosed patients.

Case Presentation:

Due to psychotic symptoms, a 38-year-old Japanese male was brought to the hospital. Following paediatric growth hormone treatment, his height was 165 cm and his weight was 76 kg. Diabetes mellitus, hyperuricemia, hypothyroidism, and strabismus were all present in the past. His physical characteristics were large thumbs and toes, lowset ears, hypertelorism, bushy eyebrows, a big nose, and slight scoliosis, all of which are common in RTS. He was born as the second of two siblings, weighing 3360 g. Because of his immunological weakness, he was hospitalised three times a year from the age of three months to three years. He had epilepsy at the time, as evidenced by aberrant electroencephalography recordings. He was diagnosed with RTS based on physical traits without genetic testing when he was 9 months old. His first words came when he was 1.5 years old, but he couldn't walk without gripping onto something, so he had to use a walker until he started primary school. As he grew older, he began to exhibit developmental delays such as intellectual disabilities, social interaction abnormalities, limited communication abilities, and repetitive conduct. At the age of 22, he had his first clinical manifestations of psychotic symptoms: auditory hallucinations and persecution delusions. He had socially withdrawn, and by the age of 26, his hallucinations and delusions had worsened. Without any emotional background, he was impatient and violent. According to DSMIV criteria, he was diagnosed with schizophrenia. He attained remission on 600 mg/day of sultopride and 2 mg/day of risperidone, and sultopride was tapered down at discharge. However, the delusions persisted, and the risperidone was prolonged since he still complained of anxiety obsessively. At the age of 38, his behavioural issues revived, including roaming across his house and useless chatting. He was taken to the hospital after experiencing a return of auditory hallucinations and persecution delusions, as well as illogical thinking. Computed

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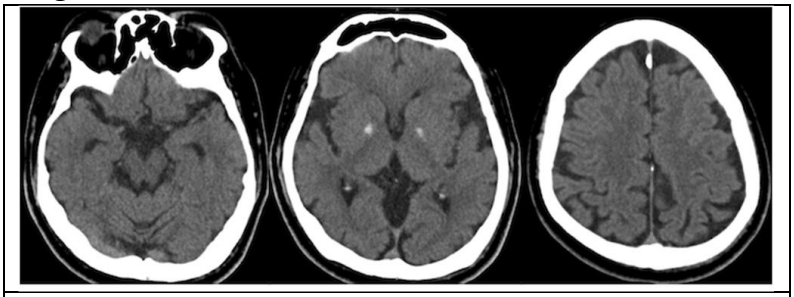
CT revealed modest temporal cortical atrophy as well as calcification of the basal ganglia and falx cerebri. A normal karyotype was discovered during cytogenetic investigation (46, XY). Whole exome sequencing (WES) revealed a potentially harmful missense mutation in CREBBP (p.Leu551Ile. As a result, researchers conducted an impartial search for variations with possible pathogenic implications. WES revealed a possible 22q.11.2 deletion, which was verified by fluorescence in situ hybridization, as well as one uncommon loss of function (LoF) variant in a LoF constrained gene (MTSS1) and three rare missense variants in missense constrained genes (CELSR3, HERC1, and TLN1). Array comparative genomic hybridization (SurePrint G3 Human CGH 1x1M Microarray [Agilent]) validated the deletion as a deletion in chr22:18,894,33921,464,119 in the GRCh37 coordinate. The patient's IQ (4th Edition Wechsler Adult Intelligence Scale) was 47. He was diagnosed with 22q11.2 DS, autism spectrum disorder, significant intellectual impairment, and schizophrenia, according to the DSM5. He was given 2mg/day of risperidone, which was eventually increased to 6mg/day. His psychotic symptoms improved two months after hospitalisation, and his Positive and Negative Syndrome Scale score dropped by 21%. However, he displayed uneasiness and fear of leaving the hospital, and his care was prolonged.



Computed tomography showed mild temporal cortical atrophy and calcification of basal ganglia and falx cerebri

Discussion:

The 22q11.2 deletion is a pleiotropy for schizophrenia symptoms and intellectual impairment in neuropsychiatry. This patient's autistic behaviour should be attributed to a 22q11.2 deletion.[2] However, the 22q11.2 deletion does not totally probe schizophrenia. Common schizophrenia-associated polymorphisms may contribute to psychotic symptoms in 22q11.2 DS patients. [3,4] Researchers hypothesise that uncommon harmful mutations in this case, whose pathogenic consequences are unknown, may potentially contribute to the mental symptoms. HERC1 is linked to intellectual disability and schizophrenia, with LoF variations playing a major role.[2] According to researcher's experience, phenotype based diagnosis is difficult, and thorough genomic study, including exome sequencing, could aid in the differential diagnosis and comprehension of the genetic basis of uncommon syndromes with mental symptoms.[2]

Conclusion:

This patient's phenotype was influenced by the 22q11.2 deletion and HERC1. However, the reason for the physical similarities between 22q11.2 deletion syndrome and RTS remains unknown. Researchers hope that this report will help with future investigations.

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4. Diagnostic challenges in the identification of elevated acutephase protein levels in a drug addicted female adolescent

Introduction:

Addiction among adolescents is a major social and health issue. A variety of neurological, gastrointestinal, and cardiovascular disorders are among the disease's symptoms. Hormonal imbalances and immune system dysregulation are also possibilities.[1]According to the literature, disturbed family relationships in adolescents, such as poor parental supervision, unstable family structure, and inappropriate interpersonal contact, are risk factors for the development of addiction.[2] Here is a case of a teenage patient with multiple diseases, in whom the elevated parameters of inflammation and high levels of antibodies caused by immune system activation were most likely related to psychoactive substance abuse.

This case illustrates the difficulty of differential diagnosis in a drug addicted adolescent patient suffering from various ailments.

Case Presentation:

A 16.5-year-old girl was hospitalised in the General Pediatric Ward of Public Hospital due to uncontrollable vomiting, severe headache, and fever. A few hours before being admitted to the ward, the patient received an intravenous injection of methamphetamine and morphine, as had been done on a regular basis in the weeks preceding the current hospitalisation. The patient had recurrent respiratory tract infections since pre-school, and ailments of hypersensitivity to grass pollen in the form of allergic rhinitis and seasonal asthma have been observed since the age of six. Aseptic necrosis of the right tibial tuberosity was confirmed at 11 years of age, and an adenoidectomy was performed at 12 years of age. Gastroenterological diagnostics were performed at the age of 13 related to chronic abdominal pain for three years. Esophagitis was diagnosed during a gastroscopic examination, and gastroprotective treatment was started immediately. Furthermore, the girl has displayed behavioural disorders and self-harm to her body since the age of 13. She was testing out various psychoactive substances (morphine, amphetamine, methamphetamine, codeine). Arypiprazol 15 mg in the morning, pregabalin 75 mg in the evening, and quetiapine XR 400 mg in the evening have been used as pharmacological treatments. She had been in the Addiction Treatment Center's outpatient treatment for a year, but the therapy was ineffective. Until recently, the patient has not agreed to treatment in an inpatient facility. Legal action has been taken to make proper treatment decisions for the adolescent.The patient was admitted with 40-degree temperature and complained of pain as well as Tachypnea was one of the abnormalities from the usual condition. Upper and lower limb fine-wave tremors, skin tremors,Tattoos around the neck, limbs, and trunk, hyperalgesia as well as countless new and old self-injury scars, Dehydration symptoms include tachycardia , abdominal skin soreness, and dry mouth mucosa were documented. Significantly, laboratory testing revealed C-reactive protein levels have risen. Procalcitonin (PCT) 50.46 ng/l, protein

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(CRP) -37.4 mg/l ml), leukopenia with lymphopenia, monocytosis, and lymphopenia Hypocapnia in capillary blood gas testing, eosinopenia increased immunoglobulin G, A (IgG, IgA) levels total immunoglobulin E (IgE), low vitamin D concentrationToxicological testing revealed the presence of amphetamines, ecstasy, and opioids in the patient's blood and urine. The patient's general condition improved significantly on the second day of her stay in the unit, and her fever dropped. During hospitalisation, psychological and psychiatric evaluations revealed behavioural abnormalities, drug misuse, and bipolar illness, and the patient was moved to a psychiatric facility for further treatment. Finally, it was determined that the increased concentration of acute-phase proteins was most likely caused by psychoactive drug intoxication. After a month, the patient returned to an allergy clinic for a routine check-up. During the patient's observation, and based on the mother's interview data regarding her daughter not receiving any therapeutic measures, a suspect of additional usage of psychoactive drugs was made, which was substantiated in a narcotic test (present—tetrahydrocannabinol, amphetamine, ecstasy). Routine laboratory tests done at the time were normal, with the exception of the high CRP (17.38 mg/l), neutrophils count to lymphocytes count ratio (NLR) (3,41 ratio), and platelets count to lymphocytes count ratio (PLR) (142,85 ratio).

Laboratory parameters	At the beginning of hospitalization	At the end of hospitalization
C-reactive protein (mg/L) (0-5)	37.4	1.84
Procalcitonin (ng/mL) (0-0.5)	50.46	0.33
Total leukocyte count (cells/l)	2.36	9.33
Differential count (%)/(cells/l)		
Neutrophils	75.9/1.77	64/5.97
Lymphocytes	12.3/0.29	26.8/2.5
Monocytes	11/1.52	6.5/5.5
Eosynophils	0.8/0.02	2.7/1.86
Platelets (cells/l)	213	337
NLR (neutrophils count to lymphocytes count ratio) (1,30+/-0,31)*	6,1	2,38
PLR (platelets count to lymphocytes count ratio) (88,95 +/-24,6)*	734,48	134,8

Discussion:

At the stage of first medical and physical examination, it was difficult to establish the diagnosis in this case. The patient's many morbidities and mental condition made it hard to get clear information on the events preceding the present sickness. The patient's symptoms, which included fever, tachycardia, tachypnea, leukopenia, and hypocapnia in blood gasometry, fit the criteria for Systemic inflammatory response syndrome(SIRS). It most commonly arises as a result of severe bacterial, viral, and fungal infections, and less frequently as a result of mechanical injuries, protracted surgical operations, burns, pancreatitis, heat stroke, and chemicals, including medications.[1] SIRS symptoms are caused by an increase in pro-inflammatory cytokines (TNF-a, Il-1, IL-6) that act both locally and systemically. These are in responsible for increasing the temperature and producing acute phase proteins (CRP, PCT, fibrinogen).[3]

Conclusion:

The case presented illustrates the difficulty of differential diagnosis in an adolescent with many disorders who has been consuming addictive drugs for several years. Many variables can

contribute to increased inflammatory markers such as Procalcitonin (PCT), C-reactive proteins(CRP), Neutrophils count to lymphocytes count ratio(NLR), and Platelets count to lymphocytes count ratio(PLR) levels. Such causes of abnormalities should be considered in adolescents who often consume psychoactive substances.

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