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A Case Report on Schizophrenia with Autoimmune Encephalitis

Dharti Meshram a*, Sonali Wavare b, Trupti Uke b and Shabnam Sayyad a

 ^a Department of Mental Health Nursing, Florence Nightingale Training College of Nursing, Datta Meghe Institute of Medical sciences (Deemed to be University) Sawangi (M) Wardha, India.
 ^b Smt. Radhikabai Meghe Memorial College of Nursing, Datta Meghe Institute of medical Science (Deemed to be University) Sawangi (M) wardha, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Schizophrenia is a severe mental illness with a high death rate and significant societal implications. Curative treatments are not available due to a lack of understanding of its etiopathogenesis. The mild encephalitis hypothesis of schizophrenia, established primarily by Karl Bechter and Norbert Müller, is one of the new research hypotheses. According to this theory, a significant subset of schizophrenia patients suffers from a mild but persistent form of encephalitis caused by a variety of etiology ranging from viral infections to traumas to autoimmune illnesses. This inflammatory method is believed to occur in the start or during the course of the disease.

The authors present case of a 65-year-old female got admitted in female psychiatric ward AVBR Hospital Sawangi Meghe, Wardha Maharashtra with chief complaint of forgetfulness, interest in environment decline, unable to communicate, poor performance at work, muttering to self, sleep disturbance, seeing people which are not seen other, fearfulness. all necessary investigation done, in mental status examination founded impairment in memory, disorientation cognitive function impairment, RBC count 3.82, WBC count 5300, Hb% 12, calcium 8.1, urea 26, creatinine 0.6, sodium 142, potassium 4.0. Alkaline phosphate 89. HIV, HBSAG non-reactive, A large number of white blood cells in the CSF An MRI that reveals evidence of brain inflammation. There was a slight increase in antinuclear antibody (1: 40 titer). Blood and CSF were positive for oligoclonal bands. The patient was received symptomatic treatment antianxiety, antipsychotic drug alleviates hallucinations and delusion.

*Corresponding author: E-mail: dhartimeshram9@gmail.com;

Disturbances of consciousness and orientation, catatonia, speech dysfunction, focal neurological signs, epileptic seizures/EEG abnormalities or autonomic dysfunction are warning signs in psychiatric patients which should always induce cerebrospinal fluid analysis with determination of antineuronal autoantibodies. Currently established immunotherapy strategies are summarised, taking into account international expert advice. Guided by clinical warning signs, our qualitative review enables rapid and reliable diagnosis of definite autoimmune encephalitis. This is of high relevance for the affected individuals, since early and sufficiently intense immunotherapy often leads to a good prognosis despite severe illness.

Keywords: Narcolepsy; anti-NMDAR receptor encephalitis; schizophrenia; hallucination; seizures; flat affect.

1. INTRODUCTION

Schizophrenia is a serious psychiatric condition that marks around 1% of the global people [1]. People suffering from schizophrenia may appear to have lost touch with reality, causing severe anguish for the individual, their family members, and friends. Schizophrenia changes how a thinks. feels. and behaves delusions, Hallucinations. disorganisation of cognition and behaviour, depression, flattened problems. and affect. cognitive social disengagement are all symptoms. that influences how a person thinks, feels, and acts [3]. "Flat affect" is one of the negative symptoms. Autoimmune encephalitis can swiftly worsen if left untreated [4]. It could result in a coma or severe brain damage. It can be lethal in rare Autoimmune encephalitis situations. formerly thought to be uncommon, but doctors are discovering more instances as their ability to detect it improves. According to a 2018 study, there are 13.7 incidences per 100,000 persons [5].

A complicated combination of various genes and environmental factors causes schizophreniform psychoses. Over 100 different gene locations have been found in large genome-wide investigations that contribute to the relative risk of psychotic symptoms. Secondary forms are based on clearly recognised reasons, either in terms of aetiology or pathophysiology. Anti-N-Methyl-D-aspartate receptor [NMDA-R] encephalitis is one example of a secondary form that has been connected to autoantibody (Ab)associated autoimmune process. With the first description of anti-NMDA-R encephalitis, the "autoimmune encephalitis" (AE) redefined. Since then, a vast variety of additional antineuronal Abs against cell surface antigens, as well as the disorders associated with them, have been discovered. Because these illnesses might be accompanied with polymorphic

psychotic symptoms, immunological conceptions of schizophreniform psychoses have received a lot of interest in recent years. In a German case series of 100 patients with various forms of AEs and Abs against antineuronal antigens, more than half (60%) exhibited with psychotic symptoms. In the majority of cases when antineuronal Abs are positive, patients develop apparent neurological symptoms along the course of the disease, such as dystonic movement abnormalities or epileptic seizures. Recently, the name "autoimmune psychosis" (AP) was proposed for AE with predominant psychotic symptoms. Encephalopathy is the evolving term for autoimmune neuropsychiatric disorders. Traditionally, this word has referred to long-lasting brain injury. When secondary brain injury was presumed, but the actual mechanism of the disease remained unknown, the term was employed (e.g., hepatic or epileptic also encephalopathy). **Because** antineuronal autoantibodies (Abs) can now be discovered, cases of encephalopathy that were previously classified as neuroinflammatory may now meet the criteria for autoimmune encephalitis [6].

Autoimmune encephalitis is a group of linked disorders in which the immune system of the body assaults the brain, causing inflammation. Antibodies are molecules produced by the immune system that mistakenly assault brain cells. The disease, like multiple sclerosis, can be progressive (worsening over time) or relapsing-remitting (with alternating flare-ups and periods of recovery). There are numerous subtypes of autoimmune encephalitis based on the antibodies present [7].

The cause of autoimmune encephalitis is uncertain in many cases. However, researchers believe it can be caused by exposure to specific bacteria and viruses, such as streptococcus and the herpes simplex virus. A teratoma is a form of tumour that stimulates the immune system to

manufacture specific antibodies. Teratomas are most commonly found in the ovaries. Some tumours, on rare occasions, can cause an autoimmune response (when the immune system targets the body's own tissues) [8].

ADEM, Anti-NMDAR receptor encephalitis, Hashimoto's encephalopathy, limbic encephalitis, and Rasmussen's encephalitis are all examples of autoimmune encephalitis. Over the course of a day and weeks, signs and symptoms are covered. Symptoms of the flu, such as headache, fever, nausea, and muscle discomfort. Psychiatric symptoms may emerge, go, then reappearance [9]. Later symptoms, such as decreased consciousness and potential coma, may be more severe [10].

Impaired memory and comprehension, Unusual and uncontrollable movements, Involuntary facial movements (facial dyskinesia), Difficulties with balance, speech, or vision, to name a few. Insomnia, Feelings of weakness or numbness Seizures, Anxiety or panic episodes that are severe, Obsessive behaviours, Changes in sexual behaviour, Changes in behaviour such as anxiety, fear, or euphoria Inhibition loss, Hallucinations, ideas of paranoia, Coma or loss of awareness [11].

2. PRESENTATION OF CASE

This case selected from AVBR Hospital Sawangi Meghe wardha. The authors present case of a 65-year-old female patient referred to female psychiatric ward AVBR Hospital Sawangi Meghe, Wardha, Maharashtra with chief complaint memory loss, of forgetfulness, restlessness, behaviour. confusion. environment decline, unable to communicate, poor performance at work, muttering to self, sleep disturbance, and hallucinations seeing people which are not seen other, fearfulness in the last 2 year. there was no any history of mental illness in her family. all necessary investigation done, such as history collection, mental status examination founded impairment in disorientation, cognitive impairment, mini mental status examination score of 13/30, verbal fluency poor, her general physical examination was unremarkable without evidence of cataracts. RBC count 3.82, WBC count LP was performed, and cerebrospinal fluid (CSF) showed white blood cell count, 8 mm neutrophils, Hb% 12, calcium 8.1, urea 26, creatinine 0.6, sodium 142, potassium 4.0. Alkaline phosphate 89. 1 mm; lymphocytes, HIV,

HBSAG non-reactive, A high white blood cell count in the cerebrospinal fluid An MRI that shows signs of brain inflammation. A mild elevation of antinuclear antibody (1:40 titter) was noted. Blood and CSF were positive for oligoclonal bands. CSF testing for herpes simplex virus 1 and 2 DNA, varicella zoster PCR, VDRL, FTA ABS, enterovirus, and viral cultures were negative. Her past medical history was relevant for vitamin B OR D deficiency. There was no any past or present surgical history. She had no history of smoking, alcohol, or other drug misuse.

Patient received the treatment of empirical treatment with vancomycin, ceftriaxone, and acyclovir for possible infectious causes of encephalitis and intravenous immunoglobulin (IVIG) 400 mg/kg/day for 5 days and methylprednisolone 1 g/day for 5 days. Plasma exchange therapy also administered. Injectable Tacrolimus. azathioprine. and sirolimus clozapine. haloperidol. also given, patient improved. condition she observed the conversation between family member and improvement in speeches also finds.

Generally, the treatment of this illness includes Surgery to remove a teratoma. Steroids to reduce brain inflammation and the immune system's response. Plasma exchange (removal and replacement of the liquid part of the blood) to take out harmful antibodies. Intravenous immunoglobulin (IVIG), given in an IV drip, to introduce antibodies from the plasma of healthy donors. IVIG removes harmful antibodies and reduces inflammation. Immunosuppressant medications, if other treatments are not effective [12].

3. DATA EXTRACTION

Sources of data collection included Cochran, Medline, PubMed, libraries, hand book.

4. DISCUSSION

The occurrence of at least four of the following symptoms within three months is required for the diagnosis of probable anti-NMDAR encephalitis: abnormal behaviour or cognitive dysfunction, speech dysfunction, movement disorder, dyskinesia, rigidity/abnormal posture, decreased state of consciousness, autonomic dysfunction, or central hypoventilation are all examples of abnormal behaviour or cognitive dysfunction. In

addition, patients must have an abnormal electroencephalogram or CSF with pleocytosis or oligoclonal bands, as well as a fair exclusion of other illnesses [13].

Autoimmune pathways generating a variety of mental disorders are becoming more widely understood, resulting in a paradigm change in neuropsychiatry. The discovery of underlying antibodies against brain ion channels or receptors raised the possibility that many patients were misdiagnosed with a primary psychiatric disorder. However, there is no clear agreement on which clinical symptoms in psychiatric patients should urge additional examination, including anti-neuronal autoantibody testing. As a result, we set out to investigate the presenting symptoms in patients with autoimmune encephalitis, as well as the period between symptom onset and the antibody commencement of diagnostics. Between May and October 2016, we recruited 100 patients from the Charité Centre for Autoimmune Encephalitis, representing all kinds autoimmune encephalitis. **Psvchiatric** disorders were the most common clinical signs, accounting for 60% of all cases. One-third of the patients were initially admitted to a psychiatric unit. All patients who tested positive for antibodies to the N-methyl-d-aspartate receptor experienced behavioural abnormalities. hallucinations, memory impairments, catatonia, or delusions. Patients with antibodies to other cell surface or intracellular antigens were regularly admitted to the hospital with a psychosomatic diagnosis. The period between the onset of first symptoms and antibody testing was frequently disturbingly protracted. The average delay in diagnosis and treatment was 74 days in patients with symptom start between 2013 and 2016, and 483 days in cases identified between 2007 and 2012, indicating that improved knowledge of this emerging disease group helped to hasten accurate diagnosis and treatment. We found clinical indicators that may aid in earlier identification by thoroughly reviewing the medical records, such as seizures, catatonia, autonomic instability, or hyperkinesia. Indeed, reanalysing the entire cohort with these "red flags" resulted in a 58 percent reduction in the period between symptom onset diagnosis. We conclude that the application of the stated clinical warning indicators can aid in timely diagnosis of an autoimmune psychiatric disorder, potentially allowing for earlier immunotherapy and a better prognosis. Furthermore, the cut-off point for cerebrospinal

fluid analysis and autoantibody testing should be low [14].

One study discovered that 10 out of 61 cases of typical encephalitis and cases of a broader spectrum of psychiatric diseases, including narcolepsy and schizophrenia, were anti-NMDAR antibody positive. In addition to the three typical cases, we discovered seven cases with anti-NMDAR antibody associated with various psychotic and sleep symptoms, but no obvious clinical signs of encephalitis (seizures and autonomic symptoms) throughout the course of the disease episodes; this finding suggests that research into the nosology autoimmune-mediated pathophysiology of atypical psychosis and sleep disorders is needed

5. CONCLUSION

Anti-NMDAR encephalitis is extremely unusual in male clients., specially those who do not have any fundamental malignancies. If a young client presents by unsolved neuropsychiatric signs, clinicians should reflect this finding as a possibility. Younger clients who existing with an early event of strange behaviour should be evaluated to instruction out severe organic reasons, as delayed diagnosis results in poor outcomes of clients.

CONSENT

As per international standard or university standard, patient written consent had been taken.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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