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SCHIZOPHRENIA LIKE PSYCHOSIS ASSOCIATED WITH EPILEPSY IN A 14 YEAR OLD- A CASE REPORT

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ABSTRACT

Epilepsy and schizophrenia are both due to altered cerebral functioning. There had been reports of mental health disorders associated with epilepsy. Usually the psychiatric comorbidities in epilepsy is a non-negligible issue with regard to the quality of life of such patient.[1] But still Psychosis associated with epilepsy has been observed in adult and children recently. In this review, the predisposing factors, clinical features and diagnostic approach of schizophrenia like psychosis in epilepsy (SLPE) has been discussed, which is one of the most severe form of comorbidity in epilepsy in children. Here we present a case of epilepsy of generalized tonic clonic type along with schizophrenia like symptoms in a 14 year old male pediatric patient.

KEYWORDS: Psychosis, epilepsy, schizophrenia, seizure.

INTRODUCTION

According to International League Against Epilepsy (ILAE), epilepsy is defined as a brain disorder characterized predominantly by recurrent and unpredictable interruptions of normal brain function, which is called epileptic seizures.[1]

Patients with epilepsy living with sporadic seizures suffer from psychological distress, which can lead to a chronic psychotic state with psychiatric manifestations of paranoid delusions and hallucinations. Psychiatric diagnosis in people with epilepsy most frequently includes psychosis, neurosis, mood disorders (e.g. Depression), personality disorders and behavioral problems.[1]

A psychiatric symptom in epilepsy is classified on the basis of their temporal relationship with the seizure occurrence into peri-ictal symptoms and inter-ictal symptoms. A Peri-ictal symptom provides information for localization of epileptiform zone and it can be prevented with seizure control. Whereas inter-ictal symptoms substantially impairs the quality of life in such patients and requires multidisciplinary and psychopharmacologic management.[1]

Peri-ictal psychosis includes pre-ictal, ictal and post-ictal periods. Ictal psychosis represents a complex partial status of temporal lobe origin. Post-ictal psychosis significantly related with temporal as well as extratemporal structural lesions, along with complex partial seizures. Chronic inter-ictal psychosis occur more often in about 20% of the psychosis in epilepsy than in general population and approximately 7% of such patients is likely to develop chronic inter-ictal schizophrenia-like psychotic syndromes which is known as schizophrenia like psychosis (SLPE).[1] SPLE is defined as a neuropsychiatric disorder that clinically mimics schizophrenia, which is accompanied a paranoid-hallucinatory syndrome along with other psychopathological disturbances and cognitive dysfunctions in patients with epilepsy. SLPE, manifests with delusions or hallucinations associated with poor insight.[1] The characterization is complicated by the fact that anticonvulsant drugs can cause psychosis and antipsychotic drugs can lower the seizure threshold, producing seizures.[2]

CASE REPORT

A 14 year old male child from Chatra, Jharkhand, presented to the Emergency Room, Department of Pediatrics, Rajendra Institute of Medical Sciences, Ranchi with mother being a reliable informant with chief

complaints of abnormal body movement and abnormal behavior for five days. He developed abnormal body movements which started with rapid jerky movements of all the four limbs, tightening of the trunk, twitching of the face, up rolling of the eyeballs, clenching of teeth along with bowel incontinence followed by loss of consciousness for half an hour. Each episode lasted for ten minutes with five-six episodes per day. History also suggestive of third person auditory hallucinations, visual hallucinations, somatic hallucinations, bizarre delusions, wandering away from home, delusion of reference, delusion of persecution, and all these events would occur after each episode of convulsion. In the past he had history of similar illness two years back for which he had received an indigenous form of medicine. He was treated in the department of psychiatry, RIMS, Ranchi and was put on oxcarbamazepine and antiepileptic drugs which was abruptly withdrawn voluntarily without any advice or follow up. A CT scan- brain done two years back showed calcified granuloma over right parietal and temporal region. He had no history of perinatal insult, developmental delay, and non-attainment of milestones or regression of attained milestones with the onset of seizure. He had no family history of epilepsy.

On examination, patient was conscious, cooperative and oriented with no abnormality detected in general examination. In higher mental function, the patient could perform digit forward test up to four digits and digit backward test up to two digits. Recent memory was affected and was unable to recall recent events. Higher cognitive function was also affected with absent abstract thinking and unable to calculate. There was loss of spatial perception and constructional ability with loss of left orientation. All the cranial nerves were intact. Motor and sensory functions were intact. Cerebellar signs and meningeal signs were absent. Other systemic examination was within normal limit.

Mental state examination reveals instability along with poor personal hygiene. He was seated normally and was looking down towards his mother with minimal eye contact. He was partially cooperative but obeyed commands when told repeatedly. Poor staccato Speech with repetition of same words. There was absence of spontaneous speech with delayed reaction time. Language quality was also poor. Patient appeared to be depressed. His range was restricted and intensity moderate.

His baseline investigations were normal. He was diagnosed as a case of Schizophrenia like psychosis associated with epilepsy. A CT of the brain was done and was found to be normal. EEG showed epileptiform activity.

Patient was put on Phenytoin initially at the time of admission and was switched to Levetiracetam to rule out phenytoin induced psychosis. Although the seizures were under control and sensorium improved, the psychosis still persisted which ruled out phenytoin induced psychosis. Based on a psychiatric consultation the patient was put on olanzapine 5mg and clonazepam 10mg after which the patient showed improvement gradually.

DISSCUSSION

The relationship between schizophrenia and epilepsy has been of great interest. The most common predisposing factor in patients suffering with SPLE is reported to be the temporal lobe epilepsy with an increased rate of psychiatric disturbances as compared to patients with epilepsy outside the temporal lobe. The temporal lobe is the major involvement of the limbic system which regulates the affect and mood.

A neuropathology study has suggested that temporal lobe epilepsy patients with lesions such as tumors, hamartomas, granuloma, gangliogliomas, cysts and focal dysplasia have a higher risk in SPLE.

Other predisposing factors include history of febrile seizure, antiepileptic drugs, and common gene defects between epilepsy and schizophrenia.[1]

Patients with psychotic disorders with epilepsy are often overlooked and mistreated, hence early diagnosis is unanimously supported as a first step to avoid delay in treatment. As most cases of schizophrenia were reported in patients with temporal lobe epilepsy, with lesions being found in the temporal lobe in many of the cases.[3] In our study, our patient had a generalized tonic clonic seizure with a history of a calcified granuloma found in the temporal lobe of the brain in imaging which was resolved two years back, with recurrence of similar episodes with no lesion found in the second imaging. The interval between the onset of epilepsy and psychosis in our study was of 2 years. But most studies suggest an average interval ranging from 10-15 years.[3]

CONCLUSION

A long term issue of psychiatric comorbidity in patients with epilepsy significantly affecting the quality of life in such patients has been prevailing and thus cannot be ignored. Series of some recent studies suggesting a neural dyssynchrony between both psychiatric and epileptic disorders develop a disconnection hypothesis. The psychiatric and epileptic disorders often exhibiting cognitive impairments, both resting-state and task-state brain networks should elaborately be studied in future studies.

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