



Epileptic psychosis: A case report

Fatma Fariha Cengiz^a, Betül Özdilek^{b*}, Esra Aydın Sünbül^a, Mustafa Bilici^a

^aDepartment of Psychiatry, Erenkoy Research and Training Hospital for Neurologic and Psychiatric Disorders, Istanbul, Turkey

^bDepartment of Neurology, Erenkoy Research and Training Hospital for Neurologic and Psychiatric Disorders, Istanbul, Turkey

ARTICLE INFO

ABSTRACT

Article History

Received 29 / 10 / 2013

Accepted 01 / 12 / 2013

* Correspondence to:

Betül Özdilek

Department of Neurology

Erenkoy Research and Training Hospital for

Neurologic and Psychiatric Disorders,
Istanbul, Turkey

e-mail: betulozdilek@yahoo.com

Keywords:

Epileptic psychosis

Forced normalization

Psychosis

Temporal lobe epilepsy

Epilepsy is a neurological disorder and frequently observed with psychiatric disorders. Epileptic people seem particularly liable to certain major psychiatric disorders. Even though epilepsy may be a risk factor for the development of the schizophrenia, it is clear that the majority of those with epilepsy do not become psychotic. Most forms of epileptic psychosis occur commonly in the partial epilepsies. Clinical presentation of epileptic psychosis can vary from paranoid hallucinatory states to anxiety and conversion phenomena, and that it may occur in both generalized and focal epilepsies. Incidence of schizophrenia-like psychosis especially increases in temporal lobe epilepsy. An antagonistic relationship is described between psychosis and seizures in some patients. Both electrical and pharmacologic kindling have been used in attempts to develop models of the antagonism between epilepsy and psychosis. A possible hypothetical relationship between psychosis and epilepsy regarding the mesolimbic dopaminergic system and kindling of this system with epileptic discharge in temporolimbic circuits could induce a florid psychotic state in some patients. The case is about the patient who mostly was referred to the hospital as catatonic psychosis, and was treated after having a seizure spontaneously.

J. Exp. Clin. Med., 2014; 31:245-246

© 2014 OMU

1. Introduction

Epilepsy is a neurological disorder that affects the quality of life of the patients and frequently observed with psychiatric disorders. In epileptic patients, psychosis has been variously involved, sometimes referring to a distinct entity, epileptic psychosis, often episodic, but at other times to chronic psychosis such as schizophrenia and affective disorders. Slater et al. (1963), introduced the term of schizophrenia-like psychosis as symptomatology included prominent paranoid ideation, often with mystical and religious coloring. Marchetti et al. (2001) reports that incidence of schizophrenia-like psychosis shows an increase in temporal lobe epilepsy. A retrospective study in patients with temporal lobe epilepsy estimates that around 10% of patients with postictal psychosis become chronically psychotic (Falip et al, 2009).

Both electrical and pharmacologic kindling have been used in attempts to develop the models of the antagonism between epilepsy and psychosis that seems to exist, at least in a proportion of patients. The pharmacological model is dopamine release in the striatum which was regulated by

presynaptic and postsynaptic receptors in the caudate nucleus and nucleus accumbens and by GABA in the substantia nigra and ventral tegmental area (Stevens and Livermore, 1978).

Prognosis for epilepsy is better when it is combined with schizophrenia than when it presented alone (Meduna, 1985). Also an antagonistic relationship between psychosis and seizures has been described in some patients. Meduna (1935) promoted that affective symptoms of schizophrenia were more responsive to induced seizures than catatonic symptoms. A possible hypothetical relationship between psychosis and epilepsy regarding the mesolimbic dopaminergic system and kindling of this system with epileptic discharge in temporolimbic circuits could induce a florid psychotic state in some patients.

2. Case report

A 31-year old, high school educated, married for one year housewife was admitted to our psychiatry outpatient clinic with the complaints of decreased eating, social withdrawal, inability to work and slowing down of movements.

In her mental state examination self-hygiene was decreased, there was restless appearance, mood was flat, affect was depressed, and her communication was limited. She did not answer the questions in time and had no eye contact. She could not express herself. Her speech tempo was reduced and monotonous. Her thoughts were of mystical content. She had delusions of persecution and auditory hallucinations. She had no insight about her mental health problem and the judgement was poor.

The patient lives with different people throughout her life, her epileptic story was not known. But she had four epileptic seizures in the last year. She was admitted to the emergency department of our hospital with complaints of catatonic psychosis and psychotic symptoms and she was completely recovered following the first electroconvulsive treatment. She did not use oral drugs regularly after discharge because she thinks she didn't need any drug. Hamilton depression scale was scored as 18 but was not considered as depression although her points were from agitation, insomnia, and lack of eating. Olanzapine 20 mg/day was administered. On the second day of hospitalization, she started to eat and the amount of speech increased. On the third day, she had seizure starting complex focal and generalized for one minute. Post-ictal period was about two minutes, the patient could not remember what happened, her vital signs were normal. Olanzapine was stopped due to the effect of reducing epileptic threshold and carbamazepine was administered for psychiatric findings and epilepsy. Electroencephalography (EEG) showed bioelectrical disruption of widespread slight state. Left medial temporal sclerosis was seen on brain magnetic resonance imaging. As medial temporal sclerosis originated from hypoxia in utero, congenital lesion can cause seizures. After neurology consultation, quetiapine treatment was started.

After two days of epileptic seizure, the psychosis resolved, her mood and affect was euthymic. There was no

pathologic symptom in mental status examination. Rorschach test was failed because the patient did not describe the figures on the cards. Her Alexander Intelligence Test score was 96 that is normal-average IQ. Minnesota multiphasic personality inventory reveals a possible psychotic disorder, then the patient should be followed up.

3. Discussion

The association of psychosis and temporal lobe seizure foci has long been recognized and noted to be more common than in other forms of epilepsy (Gibbs, 1982). Although our patient had temporal lobe lesion, we do not know the actual history of epilepsy. Regarding the current seizure and its results, she was diagnosed with epileptic psychosis. Because her family did not take care of her closely, seizures were unnoticed. Her each admission to the hospital was in catatonic psychosis. She was treated with electroconvulsive treatment immediately for an induced seizure and did not need any other medication.

It is possible to see this antagonistic relationship in literature. An antagonistic relationship between seizures and psychosis was suggested by von Meduna (1935). Kristensen and Sindrup (1978) studied 96 patients with complex partial seizures and found that seizure frequency was significantly lower in patients with concurrent psychosis, and they showed an inverse relationship between psychosis and seizure frequency. Also Akanuma et al. (2005) reported two temporal lobe epilepsy patients with long lasting psychosis after forced normalization of their EEG recordings.

Although the goal of treatment is to render patients seizure-free, there clearly are some patients in whom seizure control seems to provoke psychosis. In patients diagnosed with epileptic psychosis, epileptic and antipsychotic drugs should be selected carefully and decreasing the dosage of antiepileptic drugs should be thought. Thus psychiatric symptoms go to remission.

REFERENCES

- Akanuma, N., Kanemoto, K., Adachi, N., Kawasaki, J., Ito, M., Onuma, T., 2005. Prolonged postictal psychosis with forced normalization (Landedt) in temporal lobe epilepsy. *Epilepsy Behav.* 6, 456-459. doi:10.1016/j.yebeh.2005.01.013.
- Falip, M., Carreño M., Donaire, A., Maestro, I., Pintor, L., Bargalló, N., Boget, T., Raspall, A., Rumià, J., Setoain, J., 2009. Postictal psychosis: A retrospective study in patients with refractory temporal lobe epilepsy. *Seizure.* 18, 145-149. doi: 10.1016/j.seizure.2008.08.009.
- Gibbs, F.A., 1982. Ictal and non-ictal psychosis in temporal lobe epilepsy. *Ann. Neurol.* 11, 613-622. doi: 10.1016/j.seizure.2008.08.009.
- Kristensen, D., Sindrup, E.H., 1978. Psychomotor epilepsy and psychosis. *Acta Neurol. Scand.* 57, 361-370. doi: 10.1111/j.1600-0404.1978.tb02840.x.
- Marchetti, R.L., Tavares, A.G., Gronich, G., Fiore, L.A., Ferraz, R.B., 2001. Complete remission of epileptic psychosis after temporal lobectomy: Case report. *Arq. Neuropsiquiatr.* 59, 802-805. doi: 10.1590/S0004-282X2001000500028.
- Meduna, L.V., 1935. Versuche über die biologische beeinflussung des ablaufes der schizophrener. I. Campher-und Cardiazol-Krampfe. *Z ges Neurol. Psychiatr.* 152, 235-262.
- Meduna, L.V., 1985. Autobiography of LV meduna. *Convuls. Ther.* 1, 43-57.
- Slater, E., Beard, H., Glithero, E., 1963. The Schizophrenia-like psychosis of epilepsy. *Br. J. Psychiatry.* 109, 95-105. doi: 10.1192/bjp.109.458.95
- Stevens, J.R., Livermore, A. Jr., 1978. Kindling of mesolimbic dopamine system: Animal model of psychosis. *Neurology.* 28, 36-46. doi: 10.1212/WNL.28.1.36.