Alternative Psychosis – Is it a Defined Clinical Entity?

Girish H. Banwari, Chirag D. Parmar, Dhiraj D. Kandre

ABSTRACT

Following seizure control with antiepileptic drugs and normalization of electroencephalogram, behavioral problem may appear for the first time in an epileptic patient. This phenomenon has been termed 'alternative psychosis'. However, it remains poorly understood in absence of any definite diagnostic criteria, and there are no specific guidelines to treat the condition. Here we report a case of an untreated patient of epilepsy of 13 years duration, who had onset of first episode non-specific aggressive behavior within 1 week after starting treatment with sodium valproate, which responded adequately to a short course of low dose risperidone. We conclude that alternative psychosis may have a variable clinical presentation and may respond favorably to antipsychotic drugs.

Key words: Alternative psychosis, antiepileptic drugs, antipsychotic drugs, forced normalization

INTRODUCTION

The relationship between seizure control and occurrence of psychiatric symptoms was recognized as early as 1875 when terms such as 'transformed epilepsy' were used, [1] but the notion did not get prominently noticed until the 1950s when Landolt described epileptic patients who had psychotic episodes following control of their seizures and disappearance of epileptiform activity on electroencephalogram (EEG). [2] He introduced the term 'forced normalization' which is understood as the occurrence of episodic behavioral disturbance in an epilepsy patient associated with a change in EEG to relative normality compared with previous and subsequent EEG. [3] To circumvent the need for EEG,

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DOI:	98365044
10.4103/0253-7176.112213	回频2000000000000000000000000000000000000

Tellenbach coined the term 'alternative psychosis' as its clinical equivalent.^[4] Since then, many cases have been reported but its existence still continues to be a source of much debate and there are complexities inherent in reaching this diagnosis.^[5]

CASE REPORT

We encountered a 45-year-old male patient with primary complaints of easy irritability and anger outbursts, verbally and physically abusive behavior towards everyone in the family, and throwing away utensils and valuable things out of the house. Such behavior was reported to persist almost throughout the day. He had also become unusually stubborn regarding accountability of household expenses, enquiring about money spent by his wife and children, which he had never done before. When asked about this behavior, he could not give any relevant explanation. The behavioral changes were noticed since 3 months with no similar past history. However, the patient had history of untreated seizure episodes since 13 years, characterized by sudden staring look for 30 s to 60 s during which the patient did not respond to any verbal command, and had occasional

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lip smacking or chewing movements. This was followed by generalized tonic-clonic body movements with tongue bite and urinary incontinence. Post episode, the patient remained unresponsive for 5 to 10 min and manifested confused behavior for 2 to 3 min after regaining consciousness. Seizure episodes occurred once in 15 to 20 days, and his behavior remained normal inter-ictally. Three months back, he had consulted a neurologist, and was diagnosed as having Temporal Lobe Epilepsy (complex partial seizures of temporal lobe origin with secondary generalization), based upon the abnormal EEG findings and Magnetic Resonance Imaging (Brain) which showed left sided mesial temporal sclerosis. Sodium valproate 500 mg, twice a day was prescribed. Within a week of starting the antiepileptic, there was onset of behavioral complaints which had continued for 3 months till the patient was referred for a psychiatric consultation. Meanwhile, there were no seizure episodes after starting treatment. A repeat EEG done showed no abnormality. Birth history was not available. There was no past or family history of psychiatric illness. The patient smoked "bidi" since 30 years, but used no other substance of abuse. He worked as a farm laborer, but was not attending work since 3 months. His physical and neurological examination and laboratory investigations (including serum valproate and serum ammonia) were within the normal range. On mental status examination, he had an irritable mood, with thoughts preoccupied about his family's behavior and his past seizure episodes. There were no evident delusions, hallucinations, formal thought or perceptual disturbances. He performed well on tests of cognition, although his social judgment was impaired and he had no insight into his current abnormal behavior. As rated on the 18-item Brief Psychiatric Rating Scale (BPRS), his score was 52. We prescribed risperidone 1 mg twice a day to the patient, while valproate was continued. As the patient significantly improved in 2 weeks, the score on BPRS fell to 25. He was asymptomatic after I month of starting the antipsychotic. Risperidone was continued for 3 months and then gradually tapered off. It has been 6 months since risperidone was stopped, but the patient's epilepsy is adequately controlled on valproate, and he has no psychotic or abnormal behavioral symptoms.

DISCUSSION

Alternative psychosis is an ill-defined entity. Krishnamoorthy and Trimble have proposed criteria for forced normalization. [6] Although non-specific aggressive behavior has not been listed as one of the criteria, this patient's clinical course does support the possibility of alternative psychosis, as the psychiatric syndrome clearly emerged after the introduction of

an antiepileptic with good seizure control. As in this case, forced normalization is most commonly seen to occur in patients with complex partial seizures, [7] but why only a few and not all patients with complex partial epilepsy become psychotic remains unclear. Almost all anticonvulsants including valproate have been anecdotally reported in literature to induce the phenomenon. So, it probably has more to do with seizure control rather than the drug chosen to control seizures.

Although the neurophysiological basis for the phenomenon has not been fully elucidated, Wolf has proposed that it represents ongoing subcortical or mesial temporal epileptic activity with enhanced cortical inhibition. Role of kindling phenomenon and various neurotransmitters has also been implicated. In absence of any specific treatment guidelines for treatment, and as recommended to use antipsychotics in the lowest dose for the shortest time, we chose risperidone 2 mg given for 3 months, which sufficiently controlled the psychotic symptoms. It has been reported that lower doses of antipsychotics are needed to treat interictal psychosis as compared to schizophrenia.

More than a century since it was first described, forced normalization is still an enigma for clinicians. It needs to be defined more precisely to promote further research into it, which could have far-reaching clinical implications. Any abnormal behavior occurring after institution of treatment and control of seizures in a patient of epilepsy should prompt clinicians about the possibility of the condition, and a fair trial of antipsychotics may help resolve the condition.

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How to cite this article: Banwari GH, Parmar CD, Kandre DD. Alternative psychosis - Is it a defined clinical entity?. Indian J Psychol Med 2013;35:84-6.

Source of Support: Nil, Conflict of Interest: None.

FORM IV

Statement about ownership and other particulars about newspaper (Indian Journal of Psychological Medicine) to be published in the first issue every year after the last day of February as per Rule 8

1. Place of publication : Mumbai

2. Periodicity of its publication : Quarterly (January, April, July and October)

3. Printer's Name : Hemant Manjrekar

Nationality : Indian
(a) Whether a citizen of India? : Yes
(b) If a foreigner, the country of origin : N.A.

Address : B5-12, Kanara Business Center,

Off Link Rd, Ghatkopar (E), Mumbai - 400075, India

4. Publisher's Name : Hemant Manirekar

Nationality : Indian
(a) Whether a citizen of India? : Yes
(b) If a foreigner, the country of origin : N.A.

Address : B5-12, Kanara Business Center,

Off Link Rd, Ghatkopar (E), Mumbai - 400075, India Phone: 91-22-6649 1818/1816.

5. Editor's Name : Dr. M. S. Reddy

Nationality : Indian
(a) Whether a citizen of India? : Yes
(b) If a foreigner, the country of origin : N.A.

Address : Asha Hospital, 298, Road No.14,

Banjara Hills, Hyderabad-500 034, Andhra Pradesh, India

6. Names and addresses of individuals who own

the newspaper and partners or shareholders

holding More than one per cent of the total capital : Indian Psychiatric Society

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Date: 1st March 2013 Hemant Manjrekar Dr. M. S. Reddy