Catatonia and Psychosis Related to Epilepsy: A Case Report

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Abstract

Although a variety of metabolic, toxic, psychiatric, and neurologic conditions can produce catatonic syndromes, it is less widely recognized that this state may be caused by epilepsy. We present the case of a woman with catatonic behavior, which she could not recall. She also exhibited olfactory, auditory and visual hallucinations. An EEG demonstrated diffuse abnormal electrical activity, mainly on left temporal and frontal areas. Treatment with anticonvulsant drugs yielded excellent response.

Key Words: Temporal Lobe Epilepsy, Catatonic Syndrome, Epileptic Psychosis

Introduction

Catatonia is a syndrome characterized by motor dysregulation and speech disorders (1). A variety of conditions can produce catatonic states. Although the association between epilepsy and catatonia has been pointed out in a few review articles (2, 3), little recognition and research have been devoted to the association. In every case of catatonic syndrome, a process of clarification of the etiological diagnosis must be performed systematically.

Epilepsy and psychosis coexist more frequently than chance would predict. Typically, psychotic symptoms closely resemble schizophrenia, with paranoid ideas, ideas of influence and auditory hallucinations. The diverting points from classic schizophrenia are the common religious coloring of paranoid ideas, tendency of patient affect to remain warm and appropriate, and no premorbid personality (4).

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Submitted: January 11, 2016; Revised: May 6, 2016; Accepted: May 23, 2016

To illustrate the importance of diagnosing catatonic syndromes properly, we report the case of a patient with catatonic behavior, auditory, visual and olfactory hallucinations associated to epilepsy.

Case Study

Mrs. F's symptoms were first noticed by her husband when she was 49-years old. He reported that sometimes the patient would suddenly stop talking, turn her head and neck a little to the right, stare motionless and not interact with anyone. The episodes lasted from seconds to a few minutes. She would start to speak again as if nothing had happened. When she was asked about those moments, the patient said that she could not recall any. Apparently, Mrs. F had already had similar episodes of motionless stare that lasted for a few minutes during childhood, but with no treatment. These episodes kept going daily over the years. She was not hospitalized at any time during the period.

Mrs. F presented sudden and abrupt behavior changes at 58 years of age, along one single day. The patient initially experienced a dreamy state. She had complex visual hallucinations, heard voices and felt a smell of incense. She went out of her house, leaving all the doors opened, stood still on the

street and stared at her home without moving for a few minutes. Mrs. F could not remember the episode. The patient's husband brought her to the hospital. According to his report, it was the first time that Mrs. F had experienced hallucinations.

At the time of admission Mrs. F was fully conscious and oriented, denying any delusions or hallucinations. There was no previous report of infectious diseases or fever prior to the admission. The patient was afebrile and the findings in both the general physical and the neurological examination were normal. Mrs. F had mild verbal memory impairment and no premorbid personality. She did not have any relevant past medical history except for febrile convulsions. There was no family history of psychiatric disorders.

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During hospitalization, Mrs. F shifted between a lucid, oriented, pleasant mental status with warm and appropriate affect and a state in which she had hallucinations and catatonic behavior. She had auditory and olfactory hallucinations right before the catatonic syndrome. It was common to find her in unusual postures. For instance, one day she was found standing with her right arm flexed, holding an apple, as if she wanted to take the fruit to her mouth but without finishing the action. Occasionally, she was also found in waxy flexibility postures. In such moments, she became unresponsive to external environment for a few minutes. At that time she had no recall of her catatonic behavior.

Initial extensive laboratory investigations, including cerebrospinal fluid, liver function tests, C-reactive protein, autoimmune screen, thyroid antibodies, copper studies, and HIV serology, were all negative. EEG was applied. The findings showed diffuse abnormal electrical activity, although mainly on left temporal and frontal areas. The patient did not present convulsive symptoms or neurological focus. A brain MRI was performed, revealing no remarkable findings, except a smaller hippocampal volume on the left.

Anticonvulsant therapy was prescribed. Three weeks after treatment with oxcarbazepine 1,500 mg/day, with a blood concentration of 27 µg/ml, all the symptoms disappeared simultaneously. Mrs. F was discharged 28 days after her admission with no more catatonic behavior and with an organized and cooperative attitude.

Discussion

Our patient presented auditory, visual and olfactory hallucinations, mutism, akinesia and waxy flexibility. As she did not have any memory of her catatonic episodes (lacunar amnesia), which is not common on typical catatonic states, epilepsy was suspected. In addition, her EEG showed alterations. She progressed well on antiepileptic drugs. Thus, this case illustrates the difficulty to distinguish a psychiatric disorder due to epilepsy from other psychiatric conditions. Absence of memory for the event and alteration of consciousness favor organic etiology. In doubtful cases, it is necessary to weigh the clinical data to exclude the conditions that may produce a catatonic picture and investigate appropriately to establish an etiological diagnosis.

The visual, auditory and olfactory hallucinations experienced by the patient could be characterized as a simple partial seizure (5). Such ictal psychiatric phenomena preceded a complex partial seizure, which was associated to catatonic behavior and restriction of consciousness.

The moment when Mrs. F holds an apple with her right arm flexed is presumably an ictal dystonia. This phenomenon is well explained by ictal hyperperfusion in the basal ganglia on the ipsilateral side to the seizure onset zone and opposite to the dystonic extremity (6).

Thus, this case illustrates the difficulty to distinguish a psychiatric disorder due to epilepsy from other psychiatric conditions.

Dreamy states may be associated with electrical discharges on the mesial temporal lobe (7), while complex visual hallucinations have been described with both medial temporal lobe foci (8) and lateral temporal lobe foci (9). Ictal auditory hallucinations—such as complex auditory phenomena—seem to relate to auditory association cortices (10). Olfactory hallucinations have been reported as being closely related to the anterior mesolimbic cortex, including the amygdala (8). All the symptomatology exhibited by the patient suggest focus on the temporal lobe. In association, the EEG at the time of admission showed diffuse abnormal activity predominating on left temporal and frontal areas. The MRI exhibited a smaller hippocampal volume on the left, probably associated to chronic seizures activity.

In conclusion, it is important to consider epilepsy as a differential diagnosis, especially when the case presents characteristics such as absence of memory for the event (lacunar amnesia) and alteration of consciousness. We would like to emphasize that it is necessary to exclude the conditions that may mimic a psychiatric picture and investigate appropriately to establish an etiological diagnosis.

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