Nonconvulsive status epilepticus and psychotic symptoms: case report

Terminología. El estado epiléptico no convulsivo (NCSE) es una condición, de la duración superior a 3 min, caracterizada por una actividad epiléptica continua o intermitente en el EEG asociada a una variedad de síntomas (en particular modificaciones mentales e comportamentales) en ausencia de crisis epilépticas manifiestas. Se comprende diversas formas clínicas que van de estados confusionales menores a complejos distúrbios del comportamiento, psicosis o coma. Estos síntomas psicóticos pueden ser mucho menos complejos, yendo problemáticamente una diagnóstico diferencial. Informamos el caso de un paciente de 31 años con anamnesis personal y familiar negativa para epilepsia y/o trastorno esquizofrenico, sin factores estresantes identificables, que manifestó un episodio subacuático de ansiedad con comportamiento agresivo. Inicialmente, el paciente está dimesso sin tratamiento, pero sette días después, fue llevado a urgencias por cuerpo y actividad lesiónistica. El estudio inicial EEG reveló un estado no convulsivo con un foco temporal activo. En este paciente, los síntomas psicóticos y el EEG de alteraciones consistentes con NCSE aparecieron simultáneamente. Una vez que el EEG regresó a lo normal, los síntomas solo persistían residualmente y eran compatibles con psicosis intraictal.

Palabras clave: estado epiléptico no convulsivo, psicosis ictal.

INTRODUCTION

The outbreak of an acute or subacute psychotic state does not always reflect the occurrence of a primary psychiatric disorder, since epileptic disorders leading to such symptoms have been identified. The most obvious clinical procedure is a nonconvulsive status epileptics (NCSE). We hereby introducing the case of a patient suffering from psychosis who could meet the criteria for an ictal psychosis and whose EEG diagnosis was NCSE. The NCSE, like convulsive status epilepticus, is a state of continuous or intermittent...
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seizure activity without return to baseline, lasting more than 30 min. In general, the NCSE differs from the status epilepticus by the absence of a predominant motor component (1). The characteristic of NCSE is a change in behaviour or mental state that is associated with diagnostic changes in EEG. There are two main types of NCSE: the absence status, which is a primary generalized process, and complex partial status, which is of local origin. Both are characterized by changes in the level of consciousness and behaviour. The onset may be abrupt or gradual (2). The episodes can vary in duration and intensity. In both types of NCSE, abnormal motor activity is absent or minimal.

Ictal psychosis is rare: cognitive, affective, and hallucinatory symptoms of partial epilepsy combine to produce a psychotic state (3). Most partial seizures last less than 3 min, and the psychic symptoms evoked during such transient spells rarely cause symptoms that would be considered psychotic. Ictal psychosis relates most commonly to visual or auditory illusions and hallucinations combined with affective changes, such as agitation or fear or paranoia. Other psychic phenomena of partial epilepsy include depersonalization, derealization, autoscopy, out of body experience, or a sense of “someone behind.” These ictal experiential phenomena most often localize to the temporal lobe with activation of limbic and neocortical temporal areas. Prolonged ictal psychotic states are rare and may occur as a NCSE with simple or complex partial or absent seizures (4).

**CASE REPORT**

The patient is a 31-year-old male, native of Bulgaria and living in Spain for 6 years. Neither he nor anyone in his family had any relevant surgical medical history, recorded psychiatric epileptic history or schizophrenic disorder.

In the current episode, the patient was brought to the psychiatry emergency room by his family due to symptoms consisting in one week lasting prejudice delusions, visual and auditory hallucinations (he saw himself in the mirror warped and mandatory hearing voices telling him to take off the poison on his body). He used to work as a horse keeper in a farm and for seven days he had shown a blatant change in his behaviour (at the beginning he was suspicious) resulting in his subsequent layoff the very same day he attended the emergency services (showing self-harm).

His erratic and disruptive behaviour was explained as the intense emotional resonance of a prejudice/persecution delirium. His family explained these clinical symptoms that had been evolving for one week during which the patient could not sleep for fear of being attacked.

**Clinical exploration**

The findings in both the general physical and the neurological examination were normal. He responded to the psychopathological examination aware and oriented in all three fields, behaving in an appropriate, cooperative and approachable manner. No perplexity. Coherent speech with certain language impairment, although able to follow a conversation during the interview. Delirious ideations of prejudice and poisoning by his colleagues. On admission he experienced visual and auditory hallucinations. He showed no active affective symptoms but did respond with secondary anxiety symptoms to the current episode. No structured autolytic ideation but with a risk is inflicting self-injury within the frame of his belief of prejudice. Altered biorhythms along with global insomnia and anorexia in the last days.

Owing to the psychotic episode experienced by the patient, it was decided to admit him to the acute in-patient guard to be examined.

**Complementary examinations**

The results of blood tests including biochemical and serology tests and a hemogram were normal. ECG and brain CT scan were also normal. EEG was applied two days after admission. The patient was already being treated with risperdone 3 ml daily, lorazepam 1.5 mg daily. The finding of this study showed brain activity compatible with non-convulsive electrical status that might have right temporal focal origin. The patient did not show convulsive symptoms or neurological focus. The previous treatment was withdrawn, and a new one consisting in diazepam 30 mg daily and haloperidol 3 mg daily was prescribed. Four days later, a new control EEG was made, showing slightly desynchronized brain activity, with theta-delta focal activity and irregular acute wave in the central region. A brain MRI (1.5 T) was performed, revealing no remarkable findings, normal MRI of the brain (no evidence of mass lesion, hemorrhage, or acute ischemic injuries and no abnormalities in the white or grey matter). The patient was discharged the following day, 15 days after his admission, under treatment with haloperidol and diazepam, euthymic, with no paranoid psychotic activity, with organized and cooperative attitude.

**DISCUSSION**

Differential diagnosis was developed for this case due to the versatility with which NCSE can appear. Three diagnostic hypotheses were proposed:

1. The patient’s psychotic clinical symptoms denote the first psychotic episode of a schizophreniform disorder, and this process is independent of alterations in brain activity. NCSE might have been triggered by risperidone treatment (5).
2. First psychotic episode is a clinical symptom of NCSE. Similar to a convulsive status epilepticus, NCSE is a state of continuous or intermittent convulsive activity with no return to the baseline situation lasting over 30 min. NCSE is usually different from convulsive status epilepticus in its lack of the major motor component (1). NCSE is a term applied to a broad range of different clinical conditions sharing as a common element a protracted state of mental disorder due to critical underlying bioelectric brain activity. Its clinical presence ranges from very slight confusion to complex behaviour disorder and even coma (6). NCSE’s clinical symptoms are varied, ranging widely from confusion symptoms with a mild alteration of the consciousness levels to coma. Sometimes a remarkable change in behaviour can occur stimulating a psychiatric disorder. Sometimes subtle but typical motor symptoms can appear and help guide the diagnosis (suckling movements, automatism or nystagmoid eye movements), but they were not detected at any time in our patient. Additional tests (CT, MRI) ruled out possible NCSE etiology such as structural injury, neoplasias or infections. EEG could have been improved: a) in a self-limited way; or b) through a diazepam treatment, irrespective of the psychotic symptoms (7).

3. The psychotic symptoms are those of an ictal psychosis. Ictal psychosis symptoms similar to schizophrenia follow the same evolution, respond to the same antipsychotic medication and, once they are set, follow a different path from epileptic crisis. The rate of epileptic patients having this psychosis ranges between 3% and 7% (the schizophrenia rate for the general population is 1%), which suggests that there exists a cause relationship. Patients usually have cognitive disorder identical to that of schizophrenia. Attention, episodic memory and executive function are affected. It also encompasses visual and auditory hallucinations, changes in affection, agitation and fear. Other features are depersonalisation, derealisation and autoscopia (4).

CONCLUSION

Mental disorders secondary to epilepsy, and particularly psychotic symptoms, must be taken into account for NCSE differential diagnosis. In this case, due to the episode’s evolution, we consider that the most likely diagnosis for the patient would be ictal psychosis and therefore we suggest that there exists a causal relationship between clinical symptoms and NCSE.

REFERENCES