Nonconvulsive status epilepticus and psychotic symptoms: case report

SUMMARY. Nonconvulsive status epilepticus (NCSE) is an epileptic condition, lasting more than 30 min, characterized by continuous or recurrent epileptic activity on EEG, which is responsible for various clinical symptoms (especially in mental status or behavior) in the absence of manifest seizure activity. It includes different clinical forms, from minor confusion to complex behavioral disorders, psychosis, or coma. These psychotic symptoms can be very complex and their differential diagnosis can be difficult. We report the case of a 31-year-old male patient without previous personal or family recorded history of epilepsy and/or schizophrenic disorder, without identifiable stressors, showing a subacute episode of anxiety with aggression. Initially, he was discharged without treatment. Seven days later he went to the emergency services accompanied by his family members reporting delusions of injury. He presented a disorganized behavior with self-harm, anxiety, dysesthesia, cenestopathy and internal field hallucinations (auditory and visual). The initial EEG study revealed a nonconvulsive status with an active temporal focus. In this patient, the psychotic symptoms and EEG abnormalities consistent with NCSE appeared simultaneously. Once the EEG returned to normal, the symptoms only persisted residually and were compatible with intraictal psychosis.

KEY WORDS: Non-convulsive status epilepticus, ictal psychosis.

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 epilepticus (NCSE). We are 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...
seizure activity without return to baseline, lasting more
than 30 min. In general, the NCSE differs from the sta-
tus 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 di-
agnostic changes in EEG. There are two main types of
NCSE: the absence status, which is a primary general-
ized process, and complex partial status, which is of lo-
cal 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 du-
ration and intensity. In both types of NCSE, abnormal
motor activity is absent or minimal.

Ictal psychosis is rare: cognitive, affective, and hallu- 
cinatory symptoms of partial epilepsy combine to pro-
duce a psychotic state (3). Most partial seizures last
less than 3 min, and the psychic symptoms evoked dur-
ing such transient spells rarely cause symptoms that
would be considered psychotic. Ictal psychosis relates
most commonly to visual or auditory illusions and hal-
 lucinations combined with affective changes, such as
agitation or fear or paranoia. Other psychic pheno-
ema of partial epilepsy include depersonalization, dere-
alization, autoscopy, out of body experience, or a sense
of “someone behind.” These ictal experiential phe-
omena 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 psy-
chiatry 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 bla-
tant change in his behaviour (at the beginning he was sus-
picious) 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/persecu-
tion delirium. His family explained these clinical symp-
toms 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 neu-
rological 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. Al-
tered biorhythms along with global insomnia and anorexia
in the last days.

Owing to the psychotic episode experienced by the pa-
tient, 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 treat-
ed 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 a right tem-
poral 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 activ-
ity and irregular acute wave in the central region. A brain
MRI (1.5 T) was performed, revealing no remarkable find-
ings, normal MRI of the brain (no evidence of mass lesion,
hemorrhage, or acute ischemic injuries and no abnor-
malities in the white or grey matter). The patient was dis-
charged the following day, 15 days after his admission, un-
der treatment with haloperidol and diazepam, euthymic,
with no paranoid psychotic activity, with organized and co-
operative 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 alter-
ations in brain activity. NCSE might have been trig-
gered by risperdone treatment (5).

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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, automatisms 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