

NON-CONVULSIVE STATUS EPILEPTICUS WITH PSYCHIATRIC PRESENTATION: A CASE REPORT

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INTRODUCTION

Non-convulsive status epilepticus (NCSE) can be defined as a change in behaviour and/or mental processes associated with continuous epileptiform discharges in the EEG (Holtkamp & Meierkord 2011), generally persisting for at least a 30-minute period (Kinney et al. 2017).

NCSE can be classified into with or without coma, and the latter can be separated into generalised or focal (Kinney et al. 2017). Aetiology can be categorised into: NCSE due to epilepsy and other neurologic conditions (cerebrovascular, central nervous system infections, trauma, progressive neurodegenerative states and malignancy - primary, metastatic or paraneoplastic) or medical disorders (metabolic, endocrine, sepsis, toxic, illicit drugs and certain medications) (Maganti et al 2008, Kinney et al. 2017).

Semiology is varied and the more noticeable features include variable degrees of impaired consciousness, cognitive dysfunction, disturbances of language, subtle motor signs (myoclonus, automatisms and nystagmus), catatonia, perceptual and psychiatric disturbances (Kinney et al. 2018). The latter are more common in focal non-convulsive seizures arising in temporal or frontal areas (Kinney et al. 2018), and include mood disturbances, anxiety, impulsivity, psychosis (Kinney et al. 2017, 2018), complex automatisms and wandering behaviour (Kinney et al. 2017). Duration of symptoms may range from hours to weeks and episodes may be recurrent (Kinney et al. 2018).

CASE REPORT

To illustrate the diagnostic challenges of NCSE, we describe the case of a 37-year-old female patient admitted to our psychiatric inpatient unit. She had no personal nor family history of psychiatric disorders.

Recently she had undergone neurosurgery for a ruptured aneurysm of the anterior cerebral artery and associated subarachnoid haemorrhage. Data shows NCSE is a frequent complication of microsurgical clipping of ruptured intracranial aneurysms, occurring in 15% of patients (Kikuta et al. 2021). After hospital discharge she was stabilised under seizure prophylaxis with Levetiracetam 1000 mg qd.

One month later she presented with a rapid onset of hypoactive delirium with catatonic and psychotic features. Staring, mutism and echopraxia alternated with volatile non-systematized persecutory and mystical delusions, as well as olfactory and visual hallucinations.

She was admitted to the emergency department observation unit to undergo investigation. Laboratory findings were unremarkable. Brain CT-Scan and MRI showed only squarer lesions of the aneurysm rupture in the paramedian frontal lobe. Lumbar puncture revealed normal cerebrospinal fluid parameters. The electroencephalogram showed slow activity localised to the right temporal region, compatible with the lesion, with no paroxysmal activity.

With the clinical hypothesis of a psychotic episode with catatonic features, the patient was transferred to the psychiatry in-patient unit, but psychopharmacological treatment with Lorazepam up to 2.5 mg/day and Aripiprazole 15 mg/day, failed.

Due to the atypical presentation and previous treatment failure, even though consecutive EEGs showed no pathological activity, NCSE was considered and Levetiracetam was increased to 2000 mg/day. She made a complete recovery within 3 days with full symptom remission and presented complete amnesia for the previous 2 weeks of the symptomatic episode.

One month later, due to medication non-adherence, the symptoms recurred and the patient was brought to the emergency department. Levetiracetam was switched to Sodium Valproate 1500 mg/day p.o., since blood levels of the latter were easier to monitor in our hospital's setting. Full recovery was observed within a few days. At follow-up, the patient remains stable undergoing anticonvulsant treatment.

DISCUSSION

The main challenge regarding NCSE management is early identification and treatment (Baeker et al. 2019). The diagnosis of NCSE is based on clinical suspicion and on the existence of epileptiform on the EEG, but it often goes underdiagnosed due to several factors (Kinney et al. 2017). Symptoms are heterogeneous and may be discrete (Meierkord & Holtkamp 2007, Kinney et al. 2017). Additionally behavioural/psychiatric symptoms may be prominent, with NCSE often being misdiagnosed

primarily as a psychiatric disorder (Taniguchi et al. 2020). Despite EEG being essential for a definitive diagnosis, there is no pathognomonic pattern (Kinney et al. 2017, Goselink et al. 2019). Also, false-negative results occur, partially due to the low sensitivity of isolated scalp EEGs particularly with focal seizures without impairment of consciousness (Kinney et al. 2018).

Without proper treatment NCSE can be associated with significant morbidity and mortality (Shneker & Fountain 2003). Thus, in case of diagnostic doubt, serial or continuous EEG should be performed if available, and in selected cases foramen ovale or sphenoidal electrodes may offer extra diagnostic information (Kinney et al. 2018).

CONCLUSIONS

This clinical case illustrates the importance of considering NCSE in rapid onset psychiatric conditions in patients with no personal or family psychiatric history, but with known risk factors for seizures. Efforts to increase availability of EEG testing and monitoring, as well as more efficient communication between different specialties in the emergency department, may lead to timely identification of psychiatric symptoms as a commonly overlooked presentation of NCSE, avoiding delays in treatment and improving outcomes.

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Contribution of individual authors:

All authors made equal contribution to this case report in terms of drafting, writing, obtaining the patient's consent, revising the paper and approved the final version of manuscript.

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