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Electroconvulsive therapy in the intensive care unit for the treatment of catatonia: a case series and review of the literature☆,☆☆,★

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ABSTRACT

Objective: Catatonia is an underdiagnosed syndrome that may occur in severely ill patients. The malignant subtype, consisting of motor symptoms, autonomic instability and fever, is associated with high mortality rates, though exact current mortality rates are unknown. This subtype requires a fast detection and treatment with high doses of a benzodiazepine or electroconvulsive therapy (ECT), preferably in an intensive care unit (ICU) setting.
Method: Case series and qualitative literature review.
Results: This paper presents four patients admitted to the ICU of an academic hospital diagnosed with malignant catatonia. All patients received ECT after an ineffective trial of high-dose intravenous benzodiazepine treatment. The duration of ECT ranged from 6 to 23 treatments after which the catatonic features partially or fully remitted. In addition, we have reviewed the diagnostic challenges, neurobiology, possible causes, differential diagnosis and treatment options of catatonia, focusing on the treatment with ECT and the importance of detection and multidisciplinary collaboration.
Conclusion: Malignant catatonia is an underdiagnosed, potentially life-threatening syndrome that requires fast recognition and prompt treatment, preferably in an ICU setting.

1. Introduction

Catatonia is a neuropsychiatric syndrome marked by altered mental status and characteristic motor changes [1]. It is generally associated with affective disorders and schizophrenia but also related with other psychiatric, neurological and medical conditions [2]. Pathway dysregulations of γ-aminobutyric acid (GABA), glutamate and dopamine are presumed to be underlying causes, though the exact neurobiological mechanism is unknown [3]. More than 40 symptoms of catatonia have been described, categorized in (terms of) motor signs, psychosocial withdrawal, excitement and bizarre, repetitious behavior [2,4]. Four subtypes of catatonia can be distinguished: the withdrawn type, the excited type, malignant catatonia and periodic catatonia [4]. The malignant subtype, often difficult to differentiate from neuroleptic malignant syndrome (NMS), is characterized by fever and increasing autonomic instability with reported mortality rates of up to 9% despite treatment, though exact current mortality rates are not known [3,5].

The prevalence of catatonia among hospitalized psychiatric patients ranges from 7.6% to 38% in several surveys, depending on the population and treatment setting [6]. The prevalence of catatonia secondary to a severe medical condition ranges from 7% to 45% according to studies in various clinical settings [7]. One review for example poses the frequency of catatonia secondary to a medical condition to be 20%–25% on psychiatric units, based on 65 cases [3]. There is, to our knowledge, only one published study on the prevalence of catatonia on the intensive care unit (ICU), even though catatonia is likely to be common in ICUs as a result of severe acute illness [8,9]. One case series reported an incidence rate of 3.8%, with 7 out of 186 patients meeting the Fink and Taylor's criteria [6] for catatonic disorder over a 1-year period in a six-bed general ICU in Greece [9]. Because catatonic signs and symptoms are heavily underrecognized by physicians, as is notified by a Dutch study discussed elsewhere in this paper [10], catatonia is likely an underdiagnosed syndrome, resulting in suboptimal treatment and possibly higher morbidity and mortality [11].

In this paper, we present four patients diagnosed with malignant catatonia, treated with electroconvulsive therapy (ECT) on the ICU. In addition, we provide an overview of the literature on catatonia and

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ECT and highlight the importance of multidisciplinary collaboration of critical care physicians and psychiatrists in diagnosing and treating severely ill patients with catatonia.

2. Cases

2.1. Case 1

Mr. V., a 51-year-old man with a history of recurrent psychosis, mild retardation and autism spectrum disorder was admitted with the delusion of being already dead. At presentation, dehydration was present, and shortly after admission, a malignant catatonia with mutism and muscle rigidity developed. Furthermore, parkinsonism with oral dystonia and dyskinesia of the tongue and a tachycardia of 114 bpm were present. Laboratory evaluation showed a Creatine phosphokinase (CPK) of 1114 U/l, a C-reactive protein (CRP) of 102 mg/l and a creatinin of 47 umol/l. He developed fever as a result of a hospital-acquired pneumonia, and due to hemodynamic instability, he was admitted to the ICU, where cardiorespiratory monitoring, airway support by nasal high flow oxygen therapy, intravenous (iv) lorazepam titrated up to 1 mg/h, ceftazidim and clindamycin were initiated. The consulting neurologist performed an MRI scan and a lumbar puncture, excluding a neurological cause provoking the catatonic symptoms. After excluding other metabolic causes, the psychosis in combination with the pneumonia was assumed to have caused the malignant catatonia. Though muscle rigidity improved slightly and the patient started to make eye contact after titrating lorazepam up to 24 mg/day, muscle rigidity did not improve further. It was then, that the patient was transferred to the ICU of our academic hospital, and after lorazepam was discontinued, daily bilateral ECT was started 6 days after admission on the ICU. After 14 days of ECT the malignant catatonia was in complete remission, and transfer to the psychiatric unit was possible. There, two more bilateral ECTs were administered, and clozapine was initiated. After 17 more days at the psychiatric unit of our academic hospital, the patient was discharged to a psychiatric hospital for further recovery and resocialization. At discharge, the patient had fully recovered from pneumonia, psychosis and catatonia.

2.2. Case 2

Ms. H., a 44-year-old woman with a history of recent initiation of steroids for facialis nerve paralysis and bipolar disorder with lithium-induced hypercalcemia and diabetes insipidus, presented with increasing incorrigible motor agitation, psychosis and fever. Symptoms started after a steroid-induced manic episode with delusional thoughts, for which several antipsychotics were administered in a psychiatric hospital. The patient was admitted at the medium care unit under suspicion of an NMS where all antipsychotic drugs were ceased. A delirium due to a somatic cause and an encephalitis were excluded by cerebral MRI, lumbar puncture, chest X-ray, laboratory tests and urine screening. After the antipsychotic drugs were eliminated under sedation, the patient stabilized and was transferred to the psychiatric unit in 9 days. There, progressive autonomic dysregulation (hypertension, hyperthermia, tachycardia and tachypnea) and excessive motor activity with stereotyped movements, counting behavior, mutism and negativism developed, suggestive for malignant catatonia. Lorazepam IV titrated up to 30 mg daily showed no effect, and admission at the ICU for propofol sedation and daily bilateral ECT was necessary. Lorazepam was ceased at initiation of ECT. During the first six bilateral ECT treatments, the need for sedation and the motoric restlessness steadily decreased. After 11 bilateral treatments, ECT was discontinued, as no further improvement was attained in the last five sessions. At that point, the patient was only able to answer questions with “yes” or “no” and showed no more motoric restlessness. Six days after the last ECT, her clinical condition further improved spontaneously, and she was moved to the psychiatric unit for recovery. Carbamazepine was initiated as a mood stabilizer, and after 24 days of hospital admission, the patient had fully recovered.

2.3. Case 3

Mr. K., a 58-year-old patient with a history of schizoaffective disorder was admitted to a psychiatric hospital with confusion and hallucinations after lithium cessation of own initiative. After administration of low-dose benzodiazepines, patient turned unresponsive with pinpoint pupils and severe hypoxia, making admission to the ICU for intubation necessary. A pneumonia was diagnosed and treated. After 1 day, the patient regained consciousness and autoextubation took place, due to aggressive behavior and restlessness. After the extubation, the oxygen saturation dropped, and he had to be resuscitated, sedated and reintubated. Lithium was restarted. During the stay on the ICU, the patient developed fever (with low CRP and leukocyte count), autonomic dysfunction (tachycardia, hypertension and tachypnea) and a CPK of 120.000 U/l. An electromyogram showed severe myopathy. An oliguric renal insufficiency developed due to biopsy-proven rhabdomyolysis, and renal replacement therapy with continuous veno-venous hemofiltration was initiated. A malignant catatonia was suspected. Discontinuation of lithium and haloperidol in case of a possible NMS did not improve his condition. Daily bilateral ECT was started, resulting in improvement of symptoms; patient could follow orders and answer questions. After six bilateral ECTs, there were no more signs of psychosis or catatonia, and olanzapine was initiated to treat the underlying psychiatric disorder. As a result of ICU-acquired weakness, recovery was slow; only after 5 weeks transfer to a general ward was possible. Two weeks later, the patient was transferred to a nursing home. Due to dehydration, acute-on-chronic kidney failure and noncompliance of the patient, a conservative treatment was initiated 3 weeks later, after which the patient passed away.

2.4. Case 4

Mr. B., a 32-year-old man, was found unresponsive in his house. The patient had a history of paranoid-type schizophrenia, posttraumatic stress disorder, drug-induced psychosis and cannabis dependency. A brain computed tomography scan showed no abnormalities. Drug screening was positive for methadone. Patient was admitted at the ICU, and intubation was necessary for his coma and hypoxia. When methadone effects diminished, patient became restless and agitated, making sedation with propofol necessary, and haloperidol was initiated. The sputum and blood culture showed a *Staphylococcus Aureus,* and because of pleura-empyema, a thorax drain was placed; thereafter, it was possible to wean patient from the respirator. After 13 days, the patient developed perspiration, tachycardia, fever, echolalia, rigidity and a posturing of the head. Neurological evaluation showed no triggering neurological cause. Malignant catatonia provoked by methadone overdosis and staphylococcus aureus infection appeared to be the most probable diagnosis. The symptoms did not improve on lorazepam, and daily bilateral ECT was initiated. Because he developed increasing anxiety and psychosis, olanzapine was started. Worsening of the pleura-empyema was present, and high fever and hypoxemia made reintubation necessary. Sedation with dexmedetomidine was initiated, and drainage of pleural cavity was improved. Olanzapine was ceased. After in total 23 bilateral ECTs, only poor improvement was observed; patient being able to communicate and being less rigid, though his mental status was fluctuating. Severe contracture of his cervical muscles was present. Amantadine, an N-methyl-D-aspartate (NMDA) antagonist, was initiated and increased up to 400 mg, with good effect on the catatonic symptoms. After 4 weeks on the ICU and 10 days after the last ECT, transfer to the psychiatric unit was possible. Clozapine was initiated, and amantadine was continued. Two weeks later, the patient could be discharged to a psychiatric hospital. The catatonia was in remission, but psychotic symptoms remained present. At follow-up 3 months later, except a