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Non-Convulsive Status Epilepticus in the Presence of Catatonia: A Clinically Focused Review

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Abstract

Introduction: Catatonia is classically associated with psychiatric conditions but may occur in medical and neurologic disorders. Status epilepticus (SE) is a seizure lasting more than five minutes or two or more seizures within a five-minute period without interictal recovery of consciousness. Non-convulsive status epilepticus (NCSE) is SE without prominent motor activity that may present with catatonic symptoms. The relevance of NCSE as a potential etiology for catatonia is not clear in the literature.

Methods: A systematic review was completed to evaluate the literature on NCSE presenting with catatonia. PubMed and PsycInfo databases were searched and articles were reviewed for the presence of catatonia and NCSE.

Results: 15 articles describing 27 cases meeting inclusion criteria were identified. The authors add 1 case to the literature. The most common catatonic symptoms identified in NCSE were mutism and stupor. Clinical features frequent in NCSE presenting with catatonia included new catatonic symptoms, age over 50 years, comorbid neurological conditions, or a change in medications that affect seizure threshold. A documented psychiatric history was also common and may contribute to delayed diagnosis.

Discussion/Conclusion: It is important to consider NCSE in the differential diagnosis of new catatonic symptoms. A suggested approach to diagnostic evaluation is provided.

Keywords

Catatonia; Status epilepticus; Seizure; Altered mental status

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Declaration of Competing Interest

None.

1. Introduction

Catatonia, first described by K.L. Kahlbaum in his 1874 publication “Die Katatonie oder das Spannungsirresein” [1], is a syndrome characterized by psychomotor abnormalities including stupor, mutism, negativism, posturing, and stereotypy that may occur in multiple psychiatric, neurological, and general medical conditions. When first describing the syndrome, Kahlbaum emphasized the possible association with seizure disorders and other neurological conditions but, in part due to the legacy of Kraepelin and Bleuler, catatonia became strongly associated with psychiatric conditions, particularly schizophrenia [2]. The differential diagnosis of catatonia is extensive, but here we consider a specific condition that may be initially misdiagnosed as catatonia of psychiatric origin.

Status epilepticus (SE) is defined as a seizure lasting more than five minutes or two or more seizures occurring within a five-minute period without complete inter-ictal recovery of consciousness [3]. Non-convulsive status epilepticus (NCSE) is SE without prominent motor activity that often presents with stupor, staring, and unresponsiveness [4]. NCSE is further classified as absence status epilepticus or simple—/complex-partial status epilepticus based on clinical signs and electro-encephalogram (EEG) activity [4]. Complex-partial status epilepticus can present with complex motor movements, such as mannerisms and stereotypies, that may confound diagnosis [4]. NCSE is often not diagnosed promptly among patients with altered mental status [5–8].

Reports of catatonia due to NCSE were documented sporadically as early as 1960, but it is likely that many cases of NCSE (and other neurological and medical conditions) presenting with catatonia were not described as such because it was not until the publication of the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV) in 1994 that catatonia due to a general medical condition was recognized as a diagnostic entity [9]. Much like delirium, catatonia can be conceptualized as the endpoint of many different pathophysiological processes, and it is important to aggressively investigate for the underlying etiology. NCSE in particular can present with catatonia and due to similar presenting signs and symptoms, these two conditions can be impossible to differentiate by clinical examination alone [8,10]. NCSE may in fact be mistaken for “primary” or “psychiatric” catatonia or other psychiatric conditions, in some cases for many years [6,11,12].

Since NCSE can lead to permanent neurological sequelae [5], rapid identification and treatment is essential. To this end, the authors present an illustrative case in which catatonia was attributed a mental disorder, rather than its actual culprit, NCSE. We then systematically review the literature on NCSE presenting with catatonia, with the aims to identify common themes among cases of NCSE presenting with catatonia, elucidate risk factors for occult NCSE in the setting of catatonic symptoms, and recommend an approach to diagnostic evaluation of this challenging clinical presentation.

2. Case presentation

A 65-year-old man was admitted to the neurology service at a tertiary care center following a witnessed generalized tonic-clonic seizure (GTCS) at home and another witnessed GTCS in the emergency department. His psychiatric history was significant for obsessive-compulsive disorder (OCD) and suspected cluster A personality traits or autism spectrum disorder (ASD). His medical history was significant for one seizure five years prior, provoked by electrolyte abnormalities in the setting of poor nutrition.

Seizures were again suspected to be provoked by poor nutrition subsequent to a recent upper respiratory tract infection. He was treated with 2 mg of intravenous lorazepam in the emergency department and was started on 1 g of intravenous levetiracetam twice daily by the neurology service. Psychiatry was consulted on hospital day 1 due to persistently altered mental status, poor eye contact, withdrawal, and disorganized thought process, despite apparent resolution of seizure activity on examination and routine EEG. The primary team raised concern for catatonia in the setting of known past psychiatric illness.

Upon psychiatric evaluation, patient was somnolent and lethargic but intermittently arousable to loud voice. His speech was abnormal with prolonged latency, diminished prosody, and poor articulation. He was largely non-participatory in the examination, did not follow commands, and could not recall events leading to his hospitalization. He was oriented to person but not place or time. He could not participate in digit span testing as he would not repeat any digits. Motor exam was significant for psychomotor retardation throughout with the exception of repetitive, purposeless movements of the bilateral lower extremities. No other motor signs of catatonia were present. The patient's BFCRS score was 5 (+1 immobility, +1 relative mutism, +1 relative staring given poor eye contact, +2 stereotypy given repetitive bilateral lower limb movements) and he met Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) criterion A for catatonia, with mutism, negativism, and stereotypy [13].

The differential diagnosis of altered mental status for this patient was broad, as there are many conditions that can present with catatonic symptoms. While the patient had a purported history of OCD and ASD, both of which can present with catatonic symptoms [14,15], given the atypical presentation in this case, particularly with recent seizures and with the presence of repetitive, purposeless movements in the absence of other motor signs of catatonia, primary catatonia or other primary psychiatric pathology was considered unlikely to explain the patient's acute alteration in mental status. Recent seizure activity raised concerns for possible NCSE, despite resolution of seizure activity on routine EEG. Further work-up to identify the underlying medical or neurological etiology was recommended. Continuous video EEG subsequently demonstrated multiple sub-clinical seizures consistent with a diagnosis of NCSE. The patient's mental status improved significantly with IV lorazepam and he returned to his cognitive baseline after seizure activity was controlled with levetiracetam. He was discharged from the hospital to home. No follow-up data were available.

3. Methods

A systematic literature search was conducted in October 2020 by authors DCV and KGM in order to identify cases of catatonic symptoms arising in the setting of NCSE. Before the search process, the authors defined and agreed upon inclusion criteria. Included studies would: be published in English, report original cases in which authors suspected catatonia or used the terms “catatonia” or “catatonic” in at least one case, and report EEG results. Case studies in which adults met DSM-5 criterion A for catatonia, as independently verified by two authors, and had EEG results consistent with NCSE, would be selected for inclusion.

Searches were conducted independently in an identical manner based on predetermined search criteria. The electronic research databases “PubMed” and “PsycInfo” were searched with the keyword combination ((“status epilepticus“ AND ‘catatonic’) OR (status epilepticus” AND “catatonia“)) OR (“ictal catatonia”). In order to identify the maximum quantity of relevant cases, the search terms were applied to all fields. Output was limited to English-language articles.

Two authors (DCV, KGM) independently completed an initial screen of the search results by examining the abstracts for relevance (potential cases of NCSE presenting with catatonia). If at least one author deemed an abstract potentially relevant, the text was reviewed in full by both authors. As a secondary measure, reference lists for relevant abstracts were also examined by both authors independently for additional relevant citations. Each citation identified in this manner was screened by both authors for relevance before inclusion for full-text review.

Both authors independently completed full-text review to examine reports with respect to the previously determined inclusion criteria. Presence of catatonic symptoms was independently verified by authors DCV and KGM by applying DSM-5 criterion A to each suspected case before inclusion; any cases that did not clearly meet DSM-5 criterion A were excluded. Diagnosis of NCSE was independently verified by authors DCV and KGM by reviewing full text for EEG results consistent with NCSE, as reported by study authors. In papers with multiple cases, authors DCV and KGM independently evaluated each case for inclusion criteria.

Author KGM abstracted data for each relevant case in selected studies. Data collected included patient age and gender, specific catatonic symptoms as reported by the authors, underlying etiology of catatonia or NCSE (if reported), prior history of seizure disorder, prior history of other neurological disorder, psychiatric history including any history of catatonia, history of substance use, and clinical outcome. Common patterns and themes among included cases were elucidated, synthesized in a narrative format, and contextualized in the extant literature.

4. Results

Using the strategy outlined in the methods section, the search of the PubMed database resulted in 32 citations and the search of the PsycInfo database resulted in 20 additional citations, 9 of which were duplicates from the PubMed search, and 1 of which was a citation

that appeared twice and was excluded. Authors independently screened abstracts for relevance. After this initial screening, 22 citations (19 from PubMed, and 3 additional unique citations from PsycInfo) were included for full-text review. Review of references for relevant articles yielded an additional 36 potentially relevant unique citations. Abstracts for each of these citations were reviewed by both authors independently, resulting in an additional 25 unique citations. Thus, 78 citations were selected for full-text review.

After full-text review, 32 studies were excluded for reasons including lack of original data ($N = 4$), lack of patient level EEG data ($N = 8$), no evidence of NCSE on EEG ($N = 3$), no reported cases of catatonia in the article (N = 8), insufficient patient-level data to allow application of DSM-5 criterion A for catatonia ($N = 1$), or failure of described cases of catatonia to meet DSM-5 criterion A ($N = 5$). Three of these citations were excluded as the authors were unable to obtain a full-text copy of the article for review [16–18]. Altogether, 15 citations describing 27 unique cases of interest were included for analysis and discussion. The authors add the case reported herein to the existing literature, for a total of 28 cases of interest. For details of the screening and selection process, see PRISMA Flow Diagram in Fig. 1.

Twenty-eight cases of NCSE presenting with catatonia are summarized in Table 1, with mean age 54.9 years (SD = 15.4); 64% were males. The most common catatonic symptoms described in cases of NCSE were mutism (93%; 26/28 cases) and stupor (89%; 25/28 cases), followed by negativism (64%; 18/28 cases), catalepsy (36%; 10/28 cases), waxy flexibility (29%; 8/28 cases), and stereotypy or mannerisms (29%; 8/28 cases). Echolalia was less commonly described (14%; 4/28 cases) as was posturing, grimacing, or echopraxia (each noted in 11%; 3/28 cases). Most patients had new onset of catatonic symptoms (i.e. no known history of past catatonic symptoms; 89%; 25/28 cases). Many patients had a history of a neurological condition (57%; 16/28), including a seizure disorder (39%; 11/28) and/or head trauma (14%; 4/28). Prior documented psychiatric history was also common (43%; 12/28). Recent withdrawal or change in dosing of sedative-hypnotics, alcohol, or antiepileptics was noted in several cases (18%; 5/28), as was recent initiation or increase of a medication known to lower seizure threshold (11%; 3/28; bupropion, nortriptyline, thioridazine). Most patients (71%; 20/28), and particularly most patients with a psychiatric history (75%; 9/12), were given an initial diagnosis of a psychiatric condition or un-specified catatonia.

5. Discussion

The results of our systematic review demonstrate that mistaken or delayed diagnosis of NCSE in the setting of catatonic symptoms is an important clinical problem. Major themes that emerged among identified cases of NCSE with catatonic symptoms included older age, absent past history of catatonic symptoms, presence of co-morbid neurologic illness (especially seizures and/or head trauma), and recent changes in use of substances (e.g. alcohol) or medications (e.g. antiepileptics and psychotropics) that increase or decrease seizure threshold. These themes largely serve to bolster clinical intuition and wisdom: it is important to advocate for thorough diagnostic evaluation in the setting of new onset catatonic symptoms, especially when the degree of suspicion for NCSE or another

neuromedical etiology of catatonic symptoms is elevated due to atypical clinical characteristics or circumstances associated with higher risk of seizure. In light of these identifiable themes, and to better guide clinical decision making in these complicated patients, we have outlined a suggested diagnostic approach for assessing possible NCSE in the setting of catatonic symptoms (Fig. 2).

It is clear from the literature that catatonia and NCSE can each present with motor signs (slowing, posturing, and rigidity), repetitive behavior (grimacing and stereotypies), and psychosocial withdrawal (mutism and staring), and can be impossible to distinguish from one another by clinical examination alone [2,10,25,26,30,33–36]. Our review also highlights that while the most common catatonic symptoms described in cases of NCSE are mutism, stupor, and negativism, other classic symptoms of catatonia can also readily occur in the setting of NCSE, including catalepsy, waxy flexibility, stereotypy or mannerisms, echolalia, and posturing, grimacing, and echopraxia. Thus, though catatonic symptoms may have variable presentations depending upon underlying disorder – for example, motor symptoms of catatonia in autism spectrum disorders most commonly include slowness in initiating movements [15,37–39] – particular catatonia symptom profiles do not appear to be of use in narrowing the differential diagnosis to exclude NCSE. Rather, these findings emphasize that it is critical to maintain a high index of suspicion for NCSE in particular, and neuromedical etiologies more generally, even in the presence of signs highly characteristic of catatonia, keeping in mind the very broad differential diagnosis of catatonia, ranging from primary psychiatric conditions to NCSE to very rare diagnoses such as Nodding syndrome in children [77] and beyond. For reference in considering a broad differential diagnosis for catatonic symptoms, Table 2 provides a summary of potential underlying neuromedical etiologies of catatonia. Conditions in Table 2 previously demonstrated to also cause NCSE are marked with an asterisk.

In some cases, NCSE cannot be distinguished from “primary” or “psychiatric” catatonia without the aid of electroencephalography, and it is essential to consider EEG in the setting of catatonia when there is suspicion for NCSE. It also bears emphasizing that routine EEG is not sufficiently sensitive to detect all cases, as seizure discharges visible on scalp leads may be intermittent, with persisting altered mental status interictally [40]. In addition, low-voltage or subcortical electrical discharges may be missed [41].

In our case, psychiatry was consulted when the patient’s altered mental status persisted despite a routine EEG demonstrating no epileptiform activity; NCSE was confirmed as the etiology of the patient’s neuropsychiatric findings only after continuous video EEG monitoring was performed. Generally, 24 hours of monitoring suffices to exclude seizures as a cause of altered mental status in most cases. It is critically important for monitoring to occur with video accompanying EEG, so as to permit review of clinical event semiology. The recordings can be accomplished in a dedicated epilepsy monitoring unit, or in a psychiatry unit, with remote epileptologist review of video and EEG recordings. In settings where video EEG is unavailable, multiple EEG recordings should be obtained in the context of close patient observation.

In another case, repeated routine EEGs were normal during periods of catatonia, until a sleep-deprived EEG led to the diagnosis of frontal lobe epilepsy [28]. With frontal lobe epilepsy in particular, EEG findings are normal or nonspecific in more than one third of patients [42]. It is therefore critical to pursue additional workup, such as continuous video EEG monitoring, sleep-deprived EEG, or rarely EEG using nasopharyngeal leads, if the index of suspicion for NCSE is high, as outlined in Fig. 2.

Of additional importance, in the cases identified and summarized in this review, patients frequently had a documented psychiatric history. This finding accords with and extends the existing literature. In one study of 22 NCSE patients, nearly one third had a significant psychiatric history [43]. Given that a number of mental health disorders are more prevalent in epilepsy than in the general population [44–46] clinicians must exercise vigilance in guarding against bias and premature diagnostic closure for patients with known mental health conditions presenting with new catatonic symptoms. In the reviewed cases of NCSE and catatonic symptoms, most patients with a psychiatric history were initially diagnosed with a psychiatric etiology for their alteration in mental status. Presence of a psychiatric history may have contributed to an initial focus on primary catatonia, without adequate consideration of alternative diagnoses [47]. Maintaining a broad differential diagnosis (Table 2) and consciously monitoring for diagnostic biases is particularly critical when evaluating patients with an established psychiatric history.

This study has several limitations to consider. First, while a systematic review and narrative synthesis of individual cases can provide essential insights into rare conditions and inform future research, such a design cannot elucidate information regarding prevalence of such conditions nor provide the robust and uniform data required for more rigorous analysis. Large epidemiologic studies and/or multi-center prospective studies of patients presenting with catatonic symptoms would markedly strengthen the research database in this area. The current study design is also limited by ability to detect such cases in the first place (i.e. how many cases of NCSE in the setting of catatonic symptoms are never ultimately identified as NCSE?), and also limited by publication bias (very few such cases will ultimately be written, submitted, and published in a searchable medical journal). Thus, the current review sheds light on an important clinical problem, while surely underestimating the scope. With regard to other limitations, our requirement for EEG confirmation of NCSE likely excluded additional potential cases, as EEG is often not performed until after treatment is initiated [36] or at all during symptomatic periods [39]. However, this requirement also served to ensure that included cases truly reflected catatonia in the setting of NCSE. Another limitation is that DSM-5 criterion A for catatonia was required for inclusion; some cases did not provide adequate information on which to evaluate this criterion and were thus excluded despite potentially providing important data. Reported cases generally did not include sufficient information to apply DSM-5 criteria B-E. Finally, our findings should be interpreted with caution given the relatively small number of included cases.

6. Conclusion

This review demonstrates that when presenting with catatonic symptoms, initial misdiagnosis or delayed diagnosis of NCSE is an important clinical problem. The cases

summarized in this review illustrate the need to maintain a high index of suspicion for NCSE in the setting of catatonia, especially in the setting of new catatonic symptoms, co-morbid neurologic conditions, and/or use of substances or medications that impact seizure threshold, while past psychiatric history should not reduce suspicion for NCSE.

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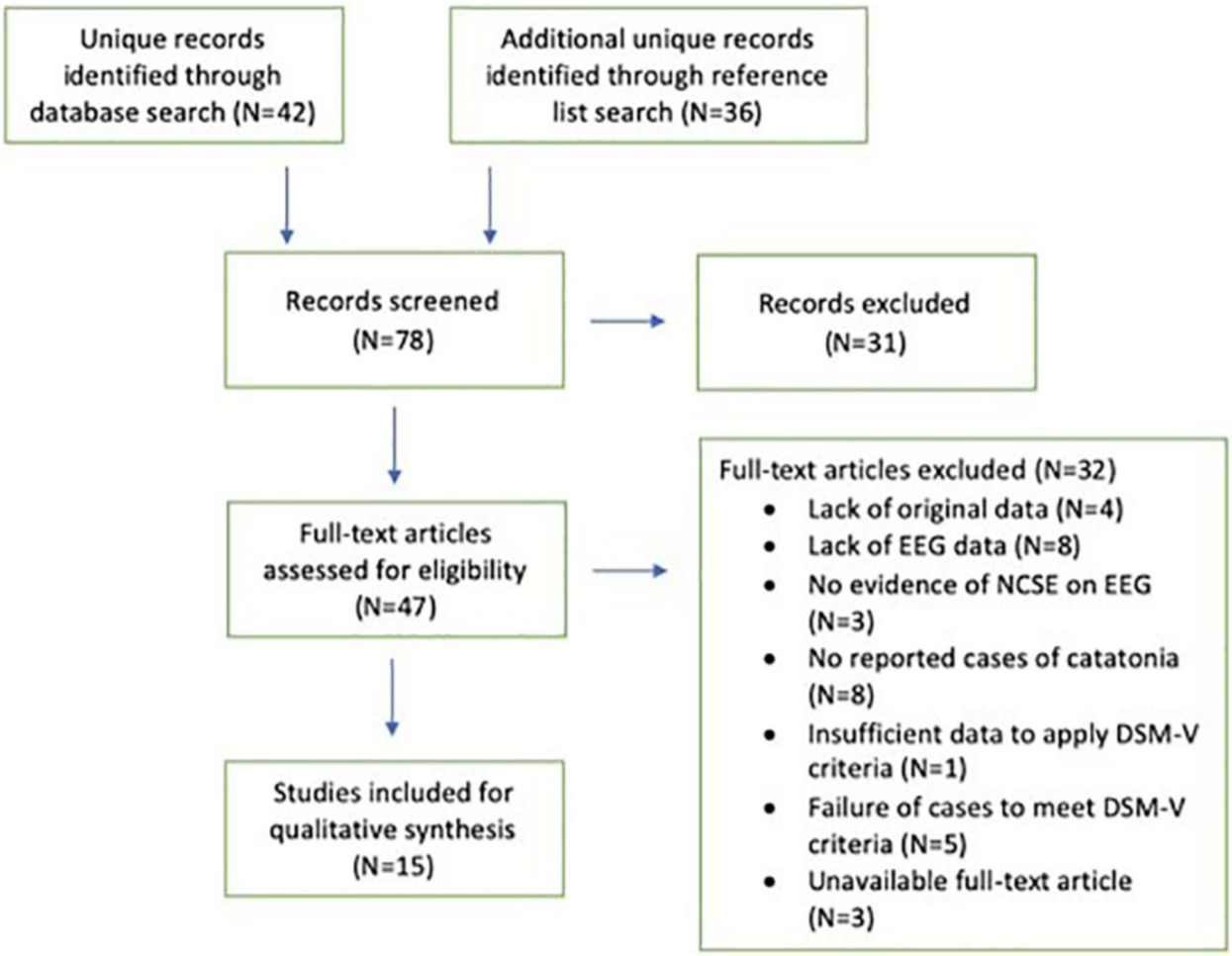

Fig. 1.
PRISMA Flow Diagram

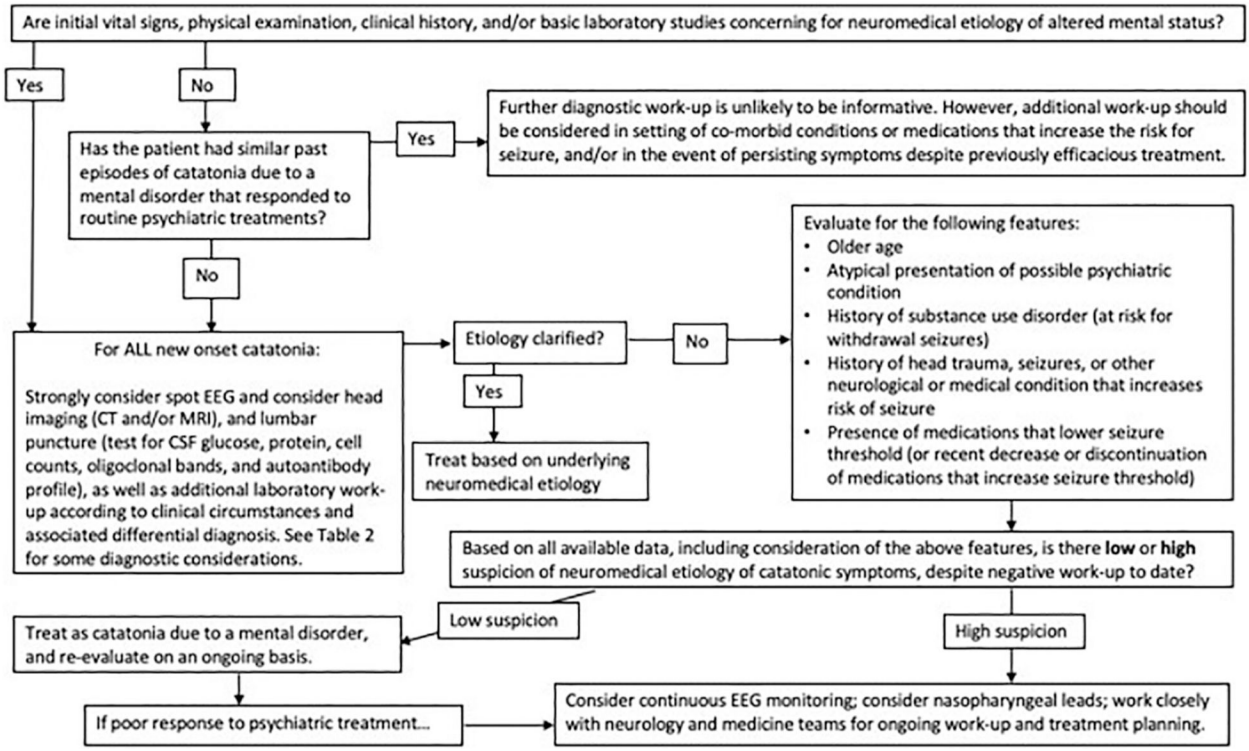

Fig. 2.

Suggested algorithm to evaluate for possible NCSE in the setting of catatonic symptoms.

Table 1

Cases of EEG-confirmed NCSE also meeting DSM-5 criteria for catatonia.

Author, publication year[a]	Case report number, EEG findings	DSM-5 criteria for catatonia present	Onset-age, sex	Known seizure history (other neurological, where applicable)	Psychiatric history (substance use, where applicable)	Initial diagnosis (if reported)	Final diagnosis	Outcome
Case Reported herein, 2020	1, near-continuous generalized periodic discharges, intermittent "spike-wave morphology with evolution into brief to intermediate duration runs of generalized rhythmic delta, consistent with electrographic seizures."	Mutism, negativism, stereotypy	65, M	1 GTCS with status epilepticus 5 years prior in the setting of infection & electrolyte abnormalities	OCD & suspected ASD vs cluster A traits. No reported history of catatonia.	Catatonia	NCSE due to infection & presumed history of epilepsy	EEG & clinical improvement with acute BZD administration & ultimate resolution with levetiracetam
Tan, 2016 [19]	1, "Episodes had a clear onset R paracentral region (P4), where arrhythmic spikes/ polyspikes became increasingly frequent & spread to the contralateral paracentral region before evolving to become generalized rhythmic discharges"	Mutism, negativism, stupor, waxy flexibility	59, M	No (a fall with head injury & small L paracentral subarachnoid hemorrhage 5 days prior)	Bipolar disorder. No history of catatonia (chronic prescribed BZDs, held on admission)	Delirium	NCSE due to recent subarachnoid hemorrhage & BZD withdrawal upon admission	Acute resolution of NCSE & improvement of catatonia with BZD; longer term resolution of both NCSE & catatonia with levetiracetam
Repchak, 2016 [20]	1, "Generalized periodic epileptiform discharges"	Mutism, stupor, waxy flexibility	44, M	Epilepsy	No	Catatonia	Catatonia due to NCSE	Lacosamide improved EEG & catatonia resolved 1 day later
	2, "Intermittent sharp & slow wave discharges, maximal over the R frontal area, with contralateral spread to the L frontal area"	Catalepsy, echopraxia, mutism, negativism, stupor	78, M	No (peripheral neuropathy)	Depression, anxiety; recent withdrawal of paroxetine & gabapentin 7 days prior. No reported history of catatonia.	Catatonia	Catatonia due to NCSE.	EEG & clinical status improved with acute BZDs & ultimately phenytoin
	3, "Continuous R frontal epileptogenic cerebral dysfunction (status epilepticus)"	Echolalia, echopraxia, mutism, negativism, stereotypy	55, F	No (subarachnoid hemorrhage due to aneurysm s/p clipping, complicated by meningitis)	Bipolar disorder. No reported history of catatonia.	Catatonia	Multi-drug-resistant epilepsia partialis continua	Clinical status & EEG did not improve despite three antiepileptics; discharged to rehabilitation facility & lost to follow up
Gélisse, 2015 [21]	1, "Continuous, irregular bilateral 5–6 Hz spike & wave discharges"	Mutism, negativism, stupor	24, F	Juvenile myoclonic epilepsy	Bipolar disorder. No reported history of catatonia (chronic prescribed BZDs with switched from clonazepam to	Confusional state	Mixed absence-myoclonic status in the setting of JME & recent change to BZD regimen	Resolution of catatonia & NCSE with BZD

Author, publication year[a]	Case report number, EEG findings	DSM-5 criteria for catatonia present	Onset-age, sex	Known seizure history (other neurological, where applicable)	Psychiatric history (substance use, where applicable)	Initial diagnosis (if reported)	Final diagnosis	Outcome
					oxazepam 5 days prior)			
Carboncini, 2014 [22]	1, “After spectral analysis, an abnormal power peak at about 7 Hz spread all over the scalp with a high intra- & inter- hemispheric coherence.”	Mutism, negativism, stereotypy, stupor, waxy flexibility	44, M	No (severe TBI, post-traumatic minimally conscious state)	No	Minimally conscious state	Catatonia due to NCSE with atypical EEG findings	Profound “awakening” response & resolution of diffuse 7 Hz oscillations after BZD administration.
Monti, 2011 [23]	1, “Diffuse irregular 1–2 Hz spikes, polyspikes & waves”	Mutism, negativism, stupor	51, F	No	No	Catatonic stupor	NSCE due to Hashimoto’s encephalopathy	No response to BZDs, valproate, phenytoin; resolution of EEG & clinical signs 24–48 h after high dose steroids
	2, “Generalized long runs of spikes & slow waves (1–2 Hz), more evident in the frontal regions, bilaterally”	Mutism, negativism, stupor	66, M	No	No	Catatonic stupor	NSCE due to Hashimoto’s encephalopathy	No response to BZDs or valproate; complete resolution of EEG & clinical signs 24–48 h after steroids
Suzuki, 2006 [24]	1, “EEG showed subclinical seizures beginning with fast recruiting activity of low amplitude in the L temporal region & progressive spread to the L frontal region, followed by slow waves”	Catalepsy, mutism, stupor	62, F	No	Schizophrenia. Likely history of prior catatonia.	Catatonic schizophrenia	NCSE with episodic tonic clonic seizure activity	Abnormal EEG activity resolved with phenytoin; catatonia persisted for 1 month without epileptiform findings on EEG, & eventually resolved after 10 sessions of ECT
	2, “Long bursts of generalized multiple spike-&-wave activity”	Catalepsy, mutism, negativism, stupor	56, M	No	Bipolar disorder. No reported history of catatonia.	Catatonia	NCSE with intermittent clonic activity	BZD & phenytoin resolved clonic status & EEG abnormalities; catatonic stupor resolved 10 days later
	3, “Recurrent bursts of generalized spike- &-wave activity predominantly over the frontal & anterior temporal regions”	Catalepsy, mutism, negativism, stupor	67. F	No	Schizophrenia. History of catatonia.	Catatonic schizophrenia	NCSE with intermittent clonic movements	Phenytoin resolved EEG abnormalities but not catatonia; catatonia transiently resolved with BZD administration & ultimately resolved with ECT
Swartz, 2002 [25]	1, “Several sharp waves, & spike & slow wave activity that are generalized… consistent with a generalized epileptiform disturbance.”	Mutism, negativism, stupor	68, M	No (presumed TBI, reported LOC in train wreck 40 years prior)	Psychotic depression vs. schizoaffective disorder. No reported history of catatonia.	Medication-induced delirium	NCSE due to recent bupropion dose increase (likely susceptible due to history of TBI)	Resolution of catatonia with valproate

Author, publication year[a]	Case report number, EEG findings	DSM-5 criteria for catatonia present	Onset-age, sex	Known seizure history (other neurological, where applicable)	Psychiatric history (substance use, where applicable)	Initial diagnosis (if reported)	Final diagnosis	Outcome
Kanemoto, 1999 [26]	1, “Continuous bilateral spike & wave discharges”	Catalepsy, mutism, waxy flexibility	78, M	No	None	Psychogenic reaction	NCSE due to benzodiazepine withdrawal	Resolution of catatonia & EEG findings with BZD
Thomas, 1999 [7]	5, “Long bursts of rhythmic 1.5-Hz polyspike & slow wave activity”	Mannerisms, mutism, stupor	51, M	Partial epilepsy with secondary generalized tonic clonic seizures, noncompliance with AEDs (bifrontal meningioma s/p surgery)	No	NCSE	NCSE	NCSE did not respond to BZDs & lasted 18 h.
	9, “Continuous ictal activity over both frontotemporal areas, interrupted by R predominant frontotemporal bursts”	Agitation, mannerisms, negativism, stupor	73, M	No	No	SE	SE was the initial presentation of ethmoidal lymphoma invading the R inferior frontal area	NCSE did not respond to BZDs but responded to oral loading dose of carbamazepine 600 mg
	10, Low-voltage L predominant fast activity over F3–C3 & F4–C4, gradually replaced by generalized, L predominant, 1.5- Hz [polyspike waves]”	Echolalia, mannerisms, negativism, stupor	60, M	No	No (alcohol dependence)	Alcohol withdrawal	NCSE due to hyponatremia	NCSE responded to BZDs
Dubin, 1985 [27]	1, “Sleep deprived EEG with naso- pharyngeal leads the following morning revealed diffuse slowing & spiking in the temporal region suggesting a generalized seizure disorder.”	Mutism, stupor, waxy flexibility	24, F	No	Two prior psychiatric hospitalizations for suicidal ideation & a suicide attempt; diagnosis not specified. No reported history of catatonia.	Catatonic schizophrenia	Complex partial status epilepticus	Gradual improvement with phenytoin
Leentjens, 1998 [28]	1, Several EEGS were normal; ultimately sleep-deprived EEG showed “paroxysms of waves with durations of 0.25–0.33 s & an amplitude of up to 60 pV were recorded in the prefrontal & frontal areas, superimposed with sharp waves with durations of 0.8–0.11 s & an amplitude of up to 65 pV… strongly suggestive of temporal or frontal lobe epilepsy”	Echolalia, echopraxia, mannerisms, mutism, stupor	47, M	No	No	Catatonia	Frontal lobe epilepsy	Resolution of catatonic episodes with carbamazepine

Author, publication year[a]	Case report number, EEG findings	DSM-5 criteria for catatonia present	Onset-age, sex	Known seizure history (other neurological, where applicable)	Psychiatric history (substance use, where applicable)	Initial diagnosis (if reported)	Final diagnosis	Outcome
Walls, 1993 [29]	1, “Diffuse theta (slow wave) activity over both hemispheres”	Negativism, mutism, posturing	40, F	No	Psychotic depression with prior episodes of catatonia. Nortriptyline initiated 7 days prior.	Depression with catatonia	Complex partial status epilepticus	Rapid resolution with carbamazepine
Lim, 1986 [30]	1, “Continuous bilateral pseudoperiodic sharp waves & spike discharges”	Catalepsy, mutism, stupor, waxy flexibility	55, M	4 GTCS upon arrival to hospital, no other prior seizure history reported	Chronic psychosis treated with thioridazine. No reported history of prior catatonia	Catatonia	NCSE of unclear etiology	Resolution of catatonia with IV phenytoin
	2, “Continuous spike & wave complexes originating in the R fronto-central region with some spread to the L hemisphere.”	Agitation, echolalia, grimacing, mutism, posturing, stupor	67, M	1 seizure 15 years prior after head trauma in a motor vehicle accident	No	Severe psychotic depression	NCSE	Resolution of catatonia with IV phenytoin
	3, “Periodic lateralising epileptiform discharges in the L parieto-temporal region”	Catalepsy, mutism, negativism	59, M	1 GTCS 1 year prior after an abdominal surgery; seizure-free thereafter despite noncompliance with phenytoin	No	Not specified	NCSE	Catatonia resolved with IV phenytoin
Drake, 1983 [31]	1, “Continuous epileptiform activity was recorded from the L nasopharyngeal electrode”	Grimacing, posturing, mutism, negativism, stupor, waxy flexibility	77, F	No	No	Feigned or psychogenic unresponsiveness	NCSE (complex partial type)	Resolution with IV phenytoin
	2, “High amplitude spike & wave discharges at 3.5 Hz developed in the R posterior temporal regions & became generalized over both cerebral hemispheres”	Agitation, catalepsy, grimacing, mutism, negativism, stereotypy, waxy flexibility	30, F	Complex partial & generalized seizures since age 8	No	Feigned or psychogenic unresponsiveness	NCSE (complex partial type)	Resolution with IV phenytoin
Thompson, 1968 [32]	1, “Continuous, bilaterally synchronous & symmetrical high voltage, irregular 3 to 5 c/s slow waves, with prominent intermingled spikes”	Catalepsy, mutism, negativism, stupor	34, M	Infrequent generalized seizures since age 6	No	Catatonic schizophrenia	NCSE (absence type)	Gradual resolution with BZD & phenobarbital
	2, “Continuous, bilaterally symmetrical, & synchronous high-voltage 2 to 3 c/s waves	Catalepsy, mutism, stupor	61, F	Poorly controlled epilepsy	No	Noted catatonic “features”	NCSE (absence type)	Resolution with IV phenobarbital

Author, publication year[a]	Case report number, EEG findings	DSM-5 criteria for catatonia present	Onset-age, sex	Known seizure history (other neurological, where applicable)	Psychiatric history (substance use, where applicable)	Initial diagnosis (if reported)	Final diagnosis	Outcome
	with interspersed multiple spikes”							
	3, “Continuous, diffuse 3 to 4 c/s activity with frequent interspersed spikes”	Agitation, stupor, mutism	42, M	Epilepsy	No	“Catatonic state”	NCSE (absence type)	Resolution with phenobarbital, diphenylhydantoin, & methsuximide

AED = antiepileptic drug, ASD = autism spectrum disorder, BZD = benzodiazepine, c/s = cycles per second, EEG = electroencephalography, F = female, GTCS = generalized tonic-clonic seizure, L = left, LOC = loss of consciousness, M = male, NCSE = non-convulsive status epilepticus, OCD = Obsessive/compulsive disorder, R = right, SE = status epilepticus, TBI = traumatic brain injury.

[a]Note: Reported cases of ictal and post-ictal catatonia occurring outside of the setting of NCSE are not included.

Table 2

Conditions presenting with catatonia; conditions also reported to cause NCSE are marked with an asterisk [8,14,15,23,37,48–77].

General cause	Specific causes
Autoimmune/inflammatory	Autoimmune encephalitis (in particular anti-NMDA receptor encephalitis)*
	Hashimoto's encephalopathy*
	Multiple sclerosis*
	Systemic lupus erythematosus*
Drugs	Adrenocorticotropic hormone
	Antibiotics (e.g. fluoroquinolones*)
	Antidepressants (e.g. tricyclic antidepressants*, monoamine oxidase inhibitors)
	Antiepileptic drugs
	Aspirin
	Baclofen*
	Clozapine withdrawal
	Cyclosporine*
	Disulfiram
	Dopamine antagonists (e.g. neuroleptics*, antiemetics)
	Dopamine depleting agents (e.g. tetrabenazine, valbenazine)
	Dopamine withdrawal (e.g. discontinuing levodopa)
	Efavirenz
	Interferon
	Lithium*
	Neuroleptic malignant syndrome*
	Opioids*
	Ribavirin
	Sedative-hypnotic withdrawal*
	Serotonin syndrome*
	Steroids
	Tacrolimus*
Endocrine/metabolic	Adrenal insufficiency
	Cushing's syndrome
	Diabetic ketoacidosis
	Homocystinuria
	Hypercalcemia
	Hyperparathyroidism
	Hyponatremia* or hypernatremia
	Idiopathic hyperadrenergic states
	Pellagra
	Pregnancy or postpartum
	Pernicious anemia
	Wernicke's encephalopathy

General cause	Specific causes
Hepatic	Hepatic dysfunction or encephalopathy*
	Liver transplantation*
	Viral hepatitis
Infectious	Acquired immunodeficiency syndrome*
	Bacterial meningoencephalitis*
	Systemic infection or sepsis*
	Malaria
	Mononucleosis
	Neurocysticercosis
	Postencephalitic states*
	Prion disease*
	Subacute sclerosing panencephalitis*
	Syphilis*[1]
	Tuberculosis*[2]
	Typhoid fever
	Viral encephalitis (e.g. herpes simplex virus encephalitis)*
Intoxication/Withdrawal	Alcohol intoxication
	Alcohol withdrawal*
	Cannabinoid intoxication
	Cannabinoid withdrawal
	Chronic amphetamine intoxication
	Hallucinogen intoxication (e.g. mescaline, phencyclidine*)
	Synthetic cannabinoid intoxication
Genetic	Acute intermittent porphyria*
	Down syndrome
	Hereditary coproporphyria
	Homocystinuria
	Glucose-6-phosphate-dehydrogenase deficiency
	Tuberous sclerosis*
	Westphal variant of Huntington's Disease
	Wilson's disease*[3]
Neurological	Alcoholic degeneration
	Cerebellar degeneration
	CNS neoplasm*
	Deep brain stimulation surgery
	Familial frontotemporal dementia
	Hydrocephalus
	Hypoxic-anoxic injury*
	Lesions of the thalamus* and globus pallidus
	Narcolepsy
	Nodding Syndrome*

General cause	Specific causes
	Parkinsonism
	Pontine and extrapontine myelinolysis
	Seizure disorders*
	Temporal lobectomy*
Poisoning	Carbon monoxide*
	Coal gas
	Manganese neurotoxicity
	Organic fluorides
	Tetraethyl lead
Psychiatric	Affective disorders (e.g. bipolar affective disorder, major depressive disorder)
	Autism spectrum disorders
	Dissociative disorders
	Functional neurological disorder
	Obsessive-compulsive disorder
	Personality disorders
	Schizophrenia spectrum disorders
Renal	Glomerulonephritis
	Kidney transplantation
	Uremia*
Traumatic	Burns
	Trauma*
	Traumatic brain injury*
Vascular	Aneurysm
	Arteriovenous malformation*
	Cerebrovascular accident*
	Intracranial hemorrhage*
	Posterior reversible encephalopathy syndrome*
	Thrombotic thrombocytopenic purpura*

Several comprehensive reviews on catatonia and NCSE were cross-referenced to determine underlying etiologies common to the two conditions to create this table. Each remaining etiology of catatonia was searched along with ("NCSE" OR "non-convulsive") in PubMed. Generated abstracts were reviewed until all either all entries were deemed irrelevant, or until a relevant citation was discovered and confirmed by review of full text.

[1] Reported to cause NCSE the setting of a Jarisch-Herxheimer reaction [68].

[2] Tuberculous meningitis reported to cause NCSE [69].

[3] Wilsons' disease reported to cause NCSE during treatment with Tetrathio-molybdate [71].