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First Large-Scale Cohort Study of Organic Catatonia: Demographics, Comorbidities, and Long-Term Survival Outcomes

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September 22, 2025

LAIDLAW SCHOLARS

Contents

1 Introduction

Catatonia is a neuropsychiatric disorder that can present with a wide range of clinical signs, including stupor, mutism, and echophenomena. Signs can be categorised as motor, affective, and cognitive-behavioural. Motor signs, such as posturing, catalepsy, stereotypies, and mannerisms, can be classified as hyperactivity or hypoactivity (Hirjak et al., 2024; Rasmussen et al., 2016; Walther and Strik, 2016). On the other hand, affective signs impact body language (mannerisms) and emotional expression (grimacing). Lastly, cognitive-behavioural signs refer to changes in an individual's behaviour, for example, echolalia and echopraxia (Hirjak et al., 2024). Catatonia can have many different clinical presentations, some examples being retarded catatonia, excited catatonia, and malignant catatonia (Rasmussen et al., 2016; Rogers et al., 2019). Due to the diverse nature of the disorder, it is useful to explore it independently to gain a better understanding of the underlying causes and the potential pathophysiology behind it.

Both the International Classification of Diseases 11th revision (ICD-11) and the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5-TR) recognise catatonia as an independent diagnosis that can be comorbid with psychiatric and medical conditions (American Psychiatric Association, 2022; Hirjak et al., 2024; "ICD-11," n.d.; Wilson et al., 2025). This has been the case since the release of the ICD-10, which introduces organic catatonic disorder separate from catatonic schizophrenia ("ICD-10 Version:2019," n.d.). Catatonia that is secondary to a medical condition that is not psychiatric in nature is also known as organic catatonia. Previous studies have identified that a significant proportion of organic catatonia cases were caused by inflammatory disorders that affected the central nervous system (CNS). (Heckers and Walther, 2023; Oldham, 2018) A systematic review conducted by Oldham et al. identified that inflammation of the CNS was the medical cause of catatonia in 87 out of 302 patients (28.8%). Of these 87 patients, 80 were due to cases of encephalitis. These included autoimmune, infection, and paraneoplastic encephalitis. The following largest categories identified were neural injury (19.2%) and substance-induced catatonia (11.6%) (Oldham, 2018). It is interesting to note that previous studies have shown that there is an association between N-methyl-D-aspartate receptor (NMDAR) encephalitis and catatonia (Beach et al., 2024; Heckers and Walther, 2023; Rogers et al., 2019). Infectious diseases and autoimmune conditions, such as systemic lupus erythematosus (SLE), have been linked to catatonia, and it is theorised that this is because they affect neuronal functioning in the CNS or periphery (Beach et al., 2024; Rogers et al., 2019).

Our current understanding of organic catatonia is limited as the systematic reviews of it are based on case reports. Case reports are subjective due to reporting bias, and as each case is unique to each individual, they might not be representative of most organic catatonia cases. As such, patient demographics, common comorbidities, and mortality rates are not reliably known (Oldham, 2018). By learning more about these details on a population level, clinical care of organic catatonia can be improved and better understood.

Overall, organic catatonia is a severe neuropsychiatric disorder that is worth exploring to aid in future clinical diagnosis and treatment. As it may arise from a range of psychological and medical conditions, or have no known cause, it is difficult to investigate, and the pathophysiology is poorly understood (Rogers et al., 2019). In this paper, we focus on the epidemiology of organic catatonia in Swedish patients. Our primary aim is to identify any key patterns that will further our understanding of this disorder, which could be used to improve future clinical practice and treatment. To do this, we look at the demographics and underlying medical causes of Swedish patients who had a diagnosis of organic catatonic disorder. Alongside this, we observe how organic catatonia tends to present clinically by looking at the number of catatonic episodes per patient and the common comorbidities that manifest compared to a reference group.

2 Methods

2.1 Data sources

In this retrospective descriptive cohort study, data from the Swedish National Patient Register (NPR) were acquired to be used for analysis with approval from the UCL Research Ethics Committee (21029/001). This contains information about patient hospital records, such as dates of inpatient and outpatient visits and diagnoses until the year 2017. It also includes data about general patient demographics (e.g. age, sex, ethnicity, etc.). The data from the Swedish death register and migration register were also used in our analyses. There were multiple files from these registries across different time spans that we combined for analysis. The data files were also stripped of excess variables.

2.2 Cohort

Two patient cohorts were analysed – the organic catatonia (OC) group and the catatonic schizophrenia (CS) group. Catatonic schizophrenia was selected as the reference group due to its shared catatonic symptomatology with OC, providing a clinically relevant comparator. This allowed for the examination of differences attributable to underlying organic versus psychiatric aetiology while accounting for the baseline presence of catatonic features, thereby facilitating a clearer interpretation of demographic patterns, comorbidities, and outcomes across the two cohorts. The diagnosis of organic catatonic disorder and catatonic schizophrenia was identified by searching for their specific ICD-10 codes – F061 and F202, respectively. Due to organic catatonic disorder only first being introduced in ICD-10, the analysed patients were limited to those diagnosed from 1997 onwards. This reduces the impact of confounding variables due to inaccurate diagnosis or conditions that have a similar presentation. The catatonic schizophrenia group excluded patients with a diagnosis of organic catatonia to ensure a clean comparison against cases where catatonia arises independently of a discernible organic cause.

Data were presented at both patient-level and episode-level to account for different dimensions of the disease burden. Patient-level data included demographic information (sex, year of birth, age at diagnosis, country of birth, etc), death-related information (cause of death, age of death, etc), and summaries of episode-level information (total number of episodes, number of healthcare contacts, etc). In comparison, the episode-level data gathered each inpatient/outpatient admission as a distinct clinical event, along with its associated information, including admission year, age at the episode, and time to readmission between events.

An OC or CS episode is defined as any inpatient or outpatient admission that includes a diagnosis of OC or CS, respectively.

2.3 Outcomes

This study analyses two outcomes – comorbidities and survival time.

Comorbidities were examined at each episode of OC or CS. The top 10 comorbidities at the OC episode were identified using 3-digit codes from ICD-10. OC and CS groups were assigned a binary variable (0 or 1) for the presence or absence of these conditions.

Survival time was calculated as the difference between the date of first diagnosis and the date of death. The first diagnosis date was assumed to be the first admission with an OC or CS diagnosis. Censoring occurred if the study end date or emigration date came before the date of death.

2.4 Statistical analyses

Descriptive statistics were conducted for outcomes and covariates, with frequency and percentage calculated for discrete variables. For continuous variables that were approximately normally distributed (e.g. age), mean and standard deviation were reported; where they were not normally distributed (e.g. number of episodes), median and interquartile range were reported.

A generalised linear model was fitted to explore the relationship between comorbidities in organic catatonia and catatonic schizophrenia groups. The model was adjusted for sex, age at episode, and the year of admission. Odds ratios (ORs) with 95% confidence intervals were used for comparison to determine if any covariates were significantly more associated with organic catatonia.

Survival analyses were done by plotting Kaplan-Meier curves of time from the first catatonic episode to the time of death. Multivariate Cox proportional hazards regression analysis was also conducted for both groups, adjusting for covariates (sex, year of diagnosis, and age at diagnosis). Schoenfeld residual tests were used to test for proportional hazard assumptions. Alongside this, assumption testing was carried out on the Cox regression model for proportional hazards, DFBETA, and deviance.

All analyses on the dataset were performed in R (4.5.1). Associations were considered statistically significant if the p-value was below 0.05.

2.5 Sensitivity analyses

To test the robustness of our findings, we conducted sensitivity analyses restricting the sample to (i) patients with at least two recorded episodes of organic catatonia (n = 112), and (ii) patients with at least one inpatient episode of organic catatonia (n = 136). These subgroups were chosen to account for potential misclassification of diagnosis codes and to capture patients with potentially more clinically significant presentations of organic catatonia.

3 Results

3.1 Descriptive data

Patient Characteristics

A total of 251 patients were included from the Swedish NPR between 1997 and 2017. Of these patients, 126 (50.3%) were male and 125 (49.7%) female, with a majority of them (n = 221, 88.0%) being born in Sweden. The mean follow-up period was 11 years, with follow-up censored at the time of their last outward emigration for the 10 (4.0%) patients who left Sweden during the study period.

Patients had a median of 2 episodes of OC, with 112 patients having at least two episodes and 136 having at least one inpatient hospital admission involving organic catatonia. Patients had a median of 23 healthcare contacts. A lifetime diagnosis of catatonic schizophrenia was identified in 96 (38.3%) patients. The mean age at first diagnosis was 50 (SD=27), and 102 patients (41%) died during follow-up, with the mean age at death being 54 (SD=25). The most common category for the primary cause of death was diseases of the circulatory system (ICD-10 chapter I, n=31, 29%).

Table 1: Patient description for the OC and CS groups.

Characteristic	CS N = 10[1]	OC N = 251[1]
Sex		
Male	6 (60%)	136 (54%)
Female	4 (40%)	115 (46%)
Birth Country		
Sweden	9 (90%)	220 (88%)
Europe Other	0 (0%)	16 (6.4%)
Other	1 (10%)	15 (6.0%)
Co-diagnosis		
No	10 (100%)	179 (71%)
Yes	0 (0%)	72 (29%)
Died During Follow-Up		
No	4 (40%)	149 (59%)
Yes	6 (60%)	102 (41%)
Primary Cause of Death		
Diseases of the circulatory system	1 (17%)	31 (29%)
Mental, Behavioral and Neurodevelopmental disorders	2 (33%)	17 (16%)
External causes of morbidity	2 (33%)	13 (12%)
Neoplasms	0 (0%)	15 (14%)
Other	1 (17%)	30 (28%)
Alive	4	145
Age at First Diagnosis	50 (27)	50 (27)
Alive	0	89
Age of Death	50 (15)	54 (25)
Alive	4	145
Follow-up Period (Years)	6 (8)	11 (14)
Alive	0	89
Year of First Diagnosis	2,008 (2,003-2,011)	2,006 (1,994-2,012)
Alive	0	89
Total Number of Episodes	1 (1-1)	2 (1-4)
Alive	0	89
Number of Healthcare Contacts	16 (9-18)	23 (13-50)

[1] n (%); Mean (SD); Median (Q1-Q3)

Table 2: Episode descriptions for the OC and CS groups.

Characteristic	CS N = 11[1]	OC N = 533[1]
Type of Admission		
Inpatient	9 (82%)	387 (73%)
Outpatient	2 (18%)	146 (27%)
Admission Year	2,007 (2,003-2,011)	2,012 (2,003-2,014)
Years to Readmission	NA (NA-NA)	2 (0-10)
Non-applicable episodes (n)	11	335
Age at Episode	51 (26)	56 (26)

[1] n (%); Median (Q1-Q3); Mean (SD)

Episode characteristics

Across all organic catatonia patients, a total of 404 episodes were recorded, of which 65% were inpatient visits and 35% were outpatient visits. The mean age at episode was 57 (SD = 25), and the median time to another inpatient readmission was 3. The most common comorbidity chapter for both OC and CS groups was mental, behavioral, and neurodevelopmental disorders.

Table 3: Comorbidity table

Characteristic	CS N = 11[1]	OC N = 533[1]
Comorbidities		
Mental, Behavioral and Neurodevelopmental disorders	5 (56%)	153 (44%)
Diseases of the circulatory system	1 (11%)	32 (9.2%)
Diseases of the nervous system	0 (0%)	30 (8.7%)
Endocrine, nutritional and metabolic diseases	1 (11%)	27 (7.8%)
Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified	1 (11%)	22 (6.4%)
Certain infectious and parasitic diseases	0 (0%)	15 (4.3%)
Diseases of the genitourinary system	0 (0%)	14 (4.0%)
Injury, poisoning and certain other consequences of external causes	0 (0%)	14 (4.0%)
Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism	0 (0%)	11 (3.2%)
Diseases of the respiratory system	0 (0%)	11 (3.2%)
Other	1 (11%)	17 (4.9%)
Episodes without comorbidities (n)	2	187

[1] n (%)

3.2 Sensitivity analysis

Across both sensitivity analyses, the overall demographic, comorbidity, and survival outcomes remained consistent with the primary analysis. Specifically, mental, behavioral, and neurodevelopmental disorders remained the most common category of comorbidities, with around 44% of patients in both analyses having said comorbidities. The elevated odds of psychiatric, neurological, and infectious comorbidities in the OC group compared to CS patients also remained present, and survival trends continued to show higher overall survival in the OC group, albeit without reaching statistical significance (p-values were 0.06 and 0.52). These findings suggest that the results derived from the observations are not driven by patients with a single, potentially less robust diagnosis or by outpatient-only cases, thereby supporting the validity of the primary analysis. Refer to Appendix B for analysis on interactions between comorbidities.

3.3 Comorbidities

Out of the top 10 common comorbidities for the OC group, OC patients were most likely to be diagnosed with pervasive developmental disorders (adjusted Odds Ratio = 217294.09, 95%CI = 0 - NA). Other psychiatric conditions, such as schizoaffective disorders and alcohol related disorders, were also more common in OC patients. But, surprisingly, the relative likelihoods of having bipolar disorder and major depressive disorder as comorbid conditions were significantly lower in OC patients. Beyond psychiatric conditions, the GLM analysis revealed that OC patients were more likely to have movement disorders such as Parkinson's, metabolic conditions such as hypothyroidism, and disorders of the immune system, such as chronic viral hepatitis and sepsis. In contrast, there was no significant difference between the odds ratios for having type 2 diabetes mellitus (adjusted OR = 0.41, 95%CI = 0.07 - 7.85) and atrial fibrillation/flutter (adjusted OR = 0.10, 95%CI = 0.01 - 2.14) for OC and CS groups in both adjusted and unadjusted models, suggesting that its high frequency in both cohorts reflects the overall prevalence of the condition in the Swedish population rather than a specific association with OC.

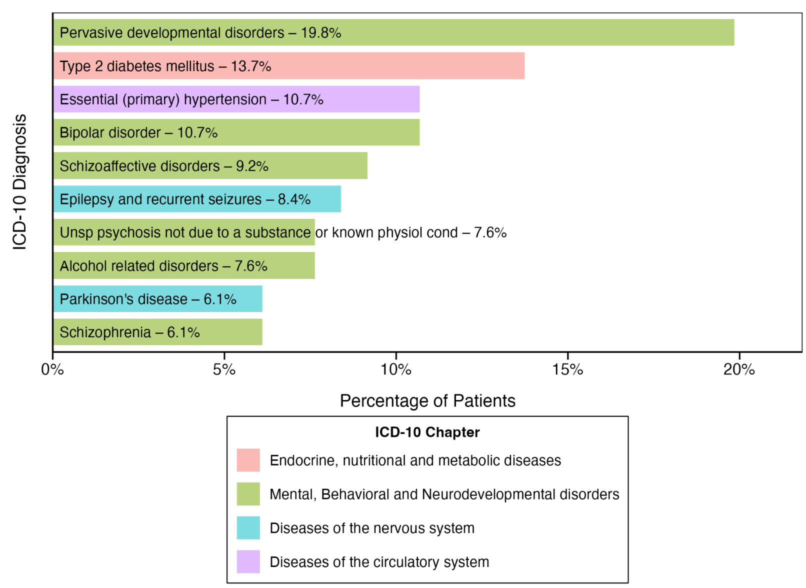
Table 4: Odds ratios and p-values for the top 10 common comorbidities between OC & CS

GLM Analysis Results

	Organic Catatonia Cases (n)	Catatonic Schizophrenia Cases (n)	Unadjusted OR (95% CI)	Adjusted OR (95% CI)
Pervasive developmental disorders	26	0	2918641.49 (0.00-NA)	2712974.09 (0.00-NA)
Type 2 diabetes mellitus	17	1	0.39 (0.07-7.48)	0.41 (0.07-7.85)
Bipolar disorder	13	2	0.13 (0.03-0.94)	0.10 (0.02-0.78)
Essential (primary) hypertension	14	0	1523343.66 (0.00-NA)	1410038.22 (0.00-NA)
Schizoaffective disorders	12	0	1299078.24 (0.00-NA)	1015120.11 (0.00-NA)
Epilepsy and recurrent seizures	11	0	1187799.34 (0.00-NA)	1006745.92 (0.00-NA)
Alcohol related disorders	9	0	966927.55 (0.00-NA)	2353080.86 (0.00-NA)
Unsp psychosis not due to a substance or known physiol cond	9	0	966927.56 (0.00-NA)	893283.28 (0.00-NA)
Schizophrenia	8	0	2330454.31 (0.00-NA)	2118910.72 (0.00-NA)
Parkinson's disease	8	0	2330454.27 (0.00-NA)	2403640.10 (0.00-NA)

(refer to Appendix C for the extended version)

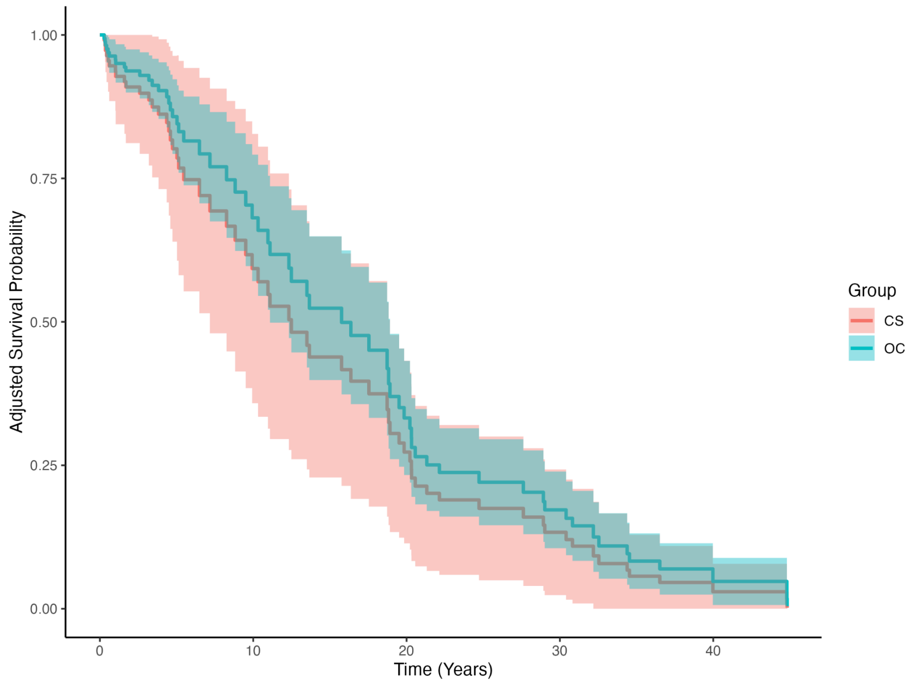
Figure 1: Top 10 most common comorbidities in cohort



3.4 Survival analysis

Survival outcomes of OC and CS groups following first diagnosis were estimated (figure 1), which showed that the CS group had a significantly worse overall survival than the OC group, with a median survival time of 8.82 years (95% CI: 1.03 - 18.32) compared to 22.14 years (95% CI: 19.84 - 32.18 years), and log-rank test corroborating a p-value below 0.05, when unadjusted for confounding variables.

Figure 2: Comparison of overall survival between OC and CS groups



Cox proportional hazard analysis revealed that an individual with OC is 53% less likely to die than someone with CS (HR = 0.47, 95%CI = 0.21-1.04), adjusting for covariates. The Kaplan-Meier curve shows that the OC curve (blue) is consistently above the CS curve (red) with early divergence in the first few years, suggesting that OC may carry a more favourable prognosis. However, when accounting for covariates, the difference didn't quite reach statistical significance (p = 0.06). As expected with the significant developments made in organic catatonia treatment, the Cox model indicated diagnosis year to be the single most important indicator of survival.

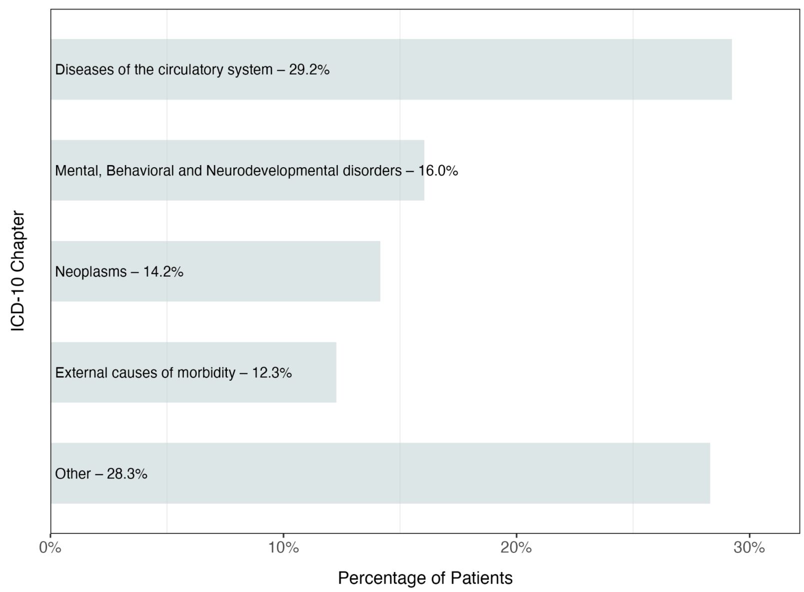
Table 5: Multivariate Cox P-H

Cox Proportional Hazards Model

Variable	Hazard Ratio	95% CI	p-value
GroupOC	0.47	0.21–1.04	0.06
SexMale	1.17	0.73–1.86	0.52
Diagnosis_Year	1.13	1.09–1.18	<0.001
Diagnosis_Age	1.00	0.99–1.01	0.34

Of the 40.6% patients who were deceased during the follow-up, the three leading causes of death were due to diseases of the circulatory system (29.2%), mental, behavioral, and neurodevelopmental disorders (16.0%), and neoplasms (14.2%), respectively.

Figure 3: Leading causes of death in patients with organic catatonia



4 Discussion

Organic catatonia is recognized to have a wide range of possible aetiology, encompassing autoinflammatory, infective, metabolic, and neurological causes. However, the scientific literature is characterized by a scarcity of high-quality evidence, with current understandings mostly derived from anecdotal case series/reports, which introduce biases that skew the results. The challenges in diagnosis and a lack of comprehensive investigation into patients with organic catatonia may have also exacerbated publication bias; many cases get underreported, while the more niche yet dramatic cases are prioritized for publication. This large-scale retrospective analysis of the Swedish cohort aims to mitigate these limitations by providing a robust, population-level perspective.

4.1 Summary of results

The analysis of this present study identified epilepsy and recurrent seizures, movement disorders, and substance-related causes, respectively, as the three most common causes of organic catatonia in the cohort. Despite overlapping symptomatology, OC and CS showed distinct comorbidity profiles, with psychiatric comorbidities, such as pervasive developmental disorders, being significantly more common in OC patients. Concordant with this finding, the descriptive statistics revealed that 38.3% of OC patients had a lifetime catatonic schizophrenia diagnosis at some point in their lives, corroborating the novel findings on the close dynamics between psychiatric and medical aetiology.

Moreover, the analysis provided the first insights into the long-term outcomes of organic catatonia. Organic catatonia patients were shown to have more favorable survival outcomes compared to those with catatonic schizophrenia, with a median survival of over 22 years compared to 8.8 years. However, this difference did not remain statistically significant after adjusting for covariates. Sensitivity analyses restricted to patients with multiple or inpatient OC episodes yielded consistent findings, supporting the robustness of the primary results. Although the results didn't reach statistical significance, this finding prompts more research to be conducted to further investigate the prognostic implications of organic catatonia and to clarify the significance of the observed effects with greater statistical power. The primary cause of death of these patients was due to the diseases of the circulatory system – likely due to complications associated with long-term immobility.

4.2 Limitations

Despite these findings, several unaddressed constraints may limit the generalizability of the results. For one, as expected from a cohort from the Swedish registry, the population is highly ethnically homogeneous, with 88.0% of patients being of Swedish descent. Of the remaining patients, half were also of European descent. This homogeneity may limit the applicability of these findings to more diverse populations.

Moreover, the retrospective design prevents an understanding of how the assessment of the diagnostic process and labeling was conducted. There is no external validation of the diagnostic codes, and reporting standards may vary across hospitals and clinics, introducing potential inconsistencies in the data that cannot be accounted for. Such limitations may also introduce selection bias, as only patients with officially documented diagnoses in the dataset are included. If, for instance, patients die before a formal diagnosis can be concluded, this may result in the underestimation of mortality risks for organic catatonia.

Although this study represents the most considerable scope of organic catatonia research to date, the relatively modest sample size limited statistical power and precluded the use of propensity score matching to construct cohorts with comparable baseline characteristics. As a result, residual confounding cannot be excluded, and differences between OC and CS patients should be interpreted with caution. The lack of data on factors such as socioeconomic indicators in the dataset further risks biases that limit the ability to establish causation.

Most significantly, the research in this report is limited by the data in use. The current results are preliminary in nature, and as this report is being written, data analysis on a larger catatonic schizophrenia sample is being conducted to enable improved comparisons in the primary analysis.

4.3 Integration with existing literature

The present findings both corroborate and extend the existing literature on organic catatonia. Previous evidence, based on case reports/series, identified inflammation of the CNS, neural injury, infectious diseases, metabolic, and substance-related conditions as some of the most frequent underlying causes. (Heckers and Walther, 2023; Oldham, 2018) In alignment with this, this study found epilepsy and recurrent seizures, movement disorders, and substance-related conditions (mostly alcohol but also psychoactive substances) to be amongst the three most common aetiological categories, respectively, in the cohort. Relative to the CS cohort, OC patients also had greater odds of having hypothyroidism, sepsis, and chronic viral hepatitis, thereby confirming the observations in the literature at a population level.

Moreover, corroborative of the literature distinguishing between organic and psychiatric causes of organic catatonia, the descriptive statistics of the Swedish cohort demonstrate that male patients are slightly more likely to have organic catatonia compared to female patients. (Smith et al., 2012) This may reflect sex-specific differences in underlying medical risk factors, though further investigation is needed.

A novel finding of this study was the high prevalence of psychiatric comorbidities in patients with organic catatonia. While Oldham's systematic review noted instances of overlap between psychiatric and medical aetiologies, quantification was limited to isolated reports. (Oldham, 2018) This analysis quantifies this phenomenon, showing that nearly 40% of OC patients had a lifetime diagnosis of catatonic schizophrenia, and over 40% carried other psychiatric comorbidities. This extends previous case-based reports that speculated on the blurred boundaries between psychiatric and medical aetiologies (Rogers et al., 2019; Beach et al., 2024), and suggests that the current nosological distinction may not be reflective of the clinical reality. Notably, Smith et al. (2012) have similarly reported that 47.4% of the OC patients had a history of psychiatric illness, supporting the broader conclusion that the overlap between psychiatric and medical causes is much more common than previously anticipated.

Moreover, although prior reports have focused on short-term outcomes, such as response to benzodiazepines or ECT, acute illness focus (e.g. malignant catatonia), and functional recovery, long-term follow-up across broad cohorts has been absent. (Luchini et al., 2015; Haroche et al., 2020; V et al., 2022; Zingela et al., 2022) The results from the present analysis, showing a trend toward better long-term survival for OC patients (though not statistically significant following adjustment for covariates) compared to CS patients, are the first of their kind to our knowledge.

Overall, this study supports the existing literature on organic catatonia in identifying neurological, inflammatory, and substance-related pathologies as the central underlying medical causes, while contributing new insights into the field by providing large-scale data on comorbidity profiles and survival outcomes. Subsequently, it challenges the current understanding of organic catatonia as a purely secondary, medical condition, completely distinct from psychiatric causes, and underscores the complex relationship between psychiatric and organic factors.

4.4 Future directions

Further subgroup analyses stratified by primary aetiology could help to identify prognostic factors in organic catatonia. Although our sensitivity analyses did not reveal differences in significance or overall survival trends, the wide heterogeneity in clinical presentation and severity means it remains uncertain whether the observed outcomes are consistent across all patient subgroups. The current clinical guidelines for OC suggest that the underlying condition must also be treated, such as providing immunosuppressants for inflammatory disorders or even surgical interventions for cases like paraneoplastic syndrome. Given the differences in treatment based on aetiology, addressing this limitation can enhance the current analysis and provide more value to clinicians in guiding clinical management. The stratified data analysis can also provide insights into. However, for this to occur, an even bigger cohort of patients would likely be required to ensure sufficient statistical power.

Along the same lines, the findings of this present study should be validated in a multi-ethnic, international cohort. This would help determine whether the demographic patterns and comorbidity profiles identified in this Swedish population are generalisable to more diverse healthcare systems and patient backgrounds.

Furthermore, new clinical guidelines can be considered based on the novel findings of this study to improve the early diagnosis and management of organic catatonia, especially in non-psychiatric settings, given the frequency of misdiagnoses. In particular, the significant likelihood of having psychiatric comorbidities with organic catatonia must be taken into greater account. What superficially seems like a classical psychiatric presentation cannot completely rule out an organic catatonia diagnosis if there are other underlying medical conditions, such as movement disorders, metabolic diseases, and inflammatory disorders associated with the manifestation of catatonic symptomology. Such guidelines could also integrate stratified treatment recommendations informed by underlying aetiology, thereby aiding clinicians in selecting timely and effective interventions.

5 Conclusion

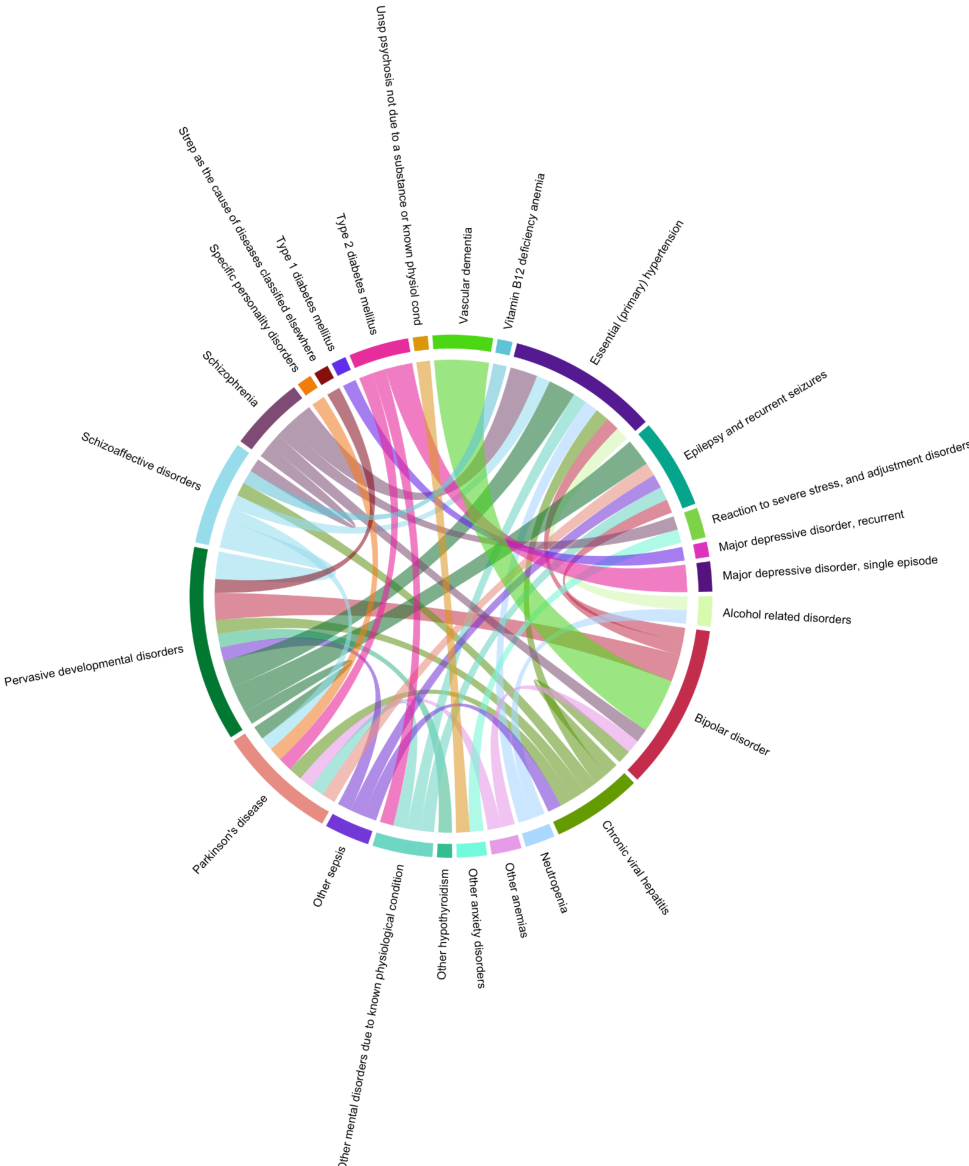
This study represents the first large-scale, population-level analysis of organic catatonia, providing novel insights into its causes, comorbidities, and long-term outcomes. Epilepsy and recurrent seizures, movement disorders, and substance-related conditions emerged as the three most common aetiologies, aligning with findings from prior literature while providing statistically robust confirmation at a national level. Interestingly, the findings emphasize a substantial overlap between medical and psychiatric causes, with nearly two in five patients carrying a lifetime diagnosis of CS and over two in five presenting with other psychiatric comorbidities, challenging the current nosological distinctions. Survival analyses suggested that OC may be associated with more favorable long-term outcomes when compared to CS. Although this trend did not remain statistically significant after adjusting for covariates and remains preliminary in nature, the results represent the first insight into long-term prognosis and necessitate the need for further investigation in larger, multi-ethnic, and prospective cohorts. By quantifying its demographic patterns, comorbidities, and outcomes at scale, this work lays a foundation for future research and has potential implications for refining clinical guidelines, enhancing diagnostic accuracy, and informing more targeted management strategies for improved long-term survival.

6 Appendix

A: Methods

- Swedish national patient registry (NPR):
    - 1x masterfile file - contains info on:
        - ID
        - Sex
        - Country of Birth
        - Date of Birth
    - 3x outpatient visits files - contains info on:
        - ID
        - Date of visit
        - Diagnoses
    - 2x inpatient visits files - contains info on:
        - ID
        - Date of admission
        - Date of discharge
        - Diagnosis
- Death data (2x files):
    - 1961-2011
    - 2011-2016
- Total population register (1x file about migrations)

B: Chord diagram



C: Extended GLM analysis results

GLM Analysis Results

	Organic Catatonia Cases (n)	Catatonic Schizophrenia Cases (n)	Unadjusted OR (95% CI)	Adjusted OR (95% CI)
Pervasive developmental disorders	26	0	2918641.49 (0.00-NA)	2712974.09 (0.00-NA)
Type 2 diabetes mellitus	17	1	0.39 (0.07-7.48)	0.41 (0.07-7.85)
Bipolar disorder	13	2	0.13 (0.03-0.94)	0.10 (0.02-0.78)
Essential (primary) hypertension	14	0	1523343.66 (0.00-NA)	1410038.22 (0.00-NA)
Schizoaffective disorders	12	0	1299078.24 (0.00-NA)	1015120.11 (0.00-NA)
Epilepsy and recurrent seizures	11	0	1187799.34 (0.00-NA)	1006745.92 (0.00-NA)
Alcohol related disorders	9	0	966927.55 (0.00-NA)	2353080.86 (0.00-NA)
Unsp psychosis not due to a substance or known physiol cond	9	0	966927.56 (0.00-NA)	893283.28 (0.00-NA)
Schizophrenia	8	0	2330454.31 (0.00-NA)	2118910.72 (0.00-NA)
Parkinson's disease	8	0	2330454.27 (0.00-NA)	2403640.10 (0.00-NA)
Vascular dementia	6	1	0.14 (0.02-2.69)	0.10 (0.01-2.01)
Major depressive disorder, single episode	7	1	0.16 (0.02-3.12)	0.16 (0.02-3.10)
Major depressive disorder, recurrent	7	1	0.16 (0.02-3.12)	0.16 (0.02-3.26)
Other anxiety disorders	7	0	2034023.95 (0.00-NA)	40799215.79 (0.00-NA)
Other anemias	6	0	1739079.61 (0.00-NA)	1848011.73 (0.00-NA)
Other psychoactive substance related disorders	6	0	1739079.58 (0.00-NA)	1698338.78 (0.00-NA)
Other sepsis	5	0	1445609.91 (0.00-NA)	1103052.67 (0.00-NA)
Chronic viral hepatitis	5	0	1445609.90 (0.00-NA)	956079.48 (0.00-NA)
Other hypothyroidism	5	0	1445609.91 (0.00-NA)	1593541.76 (0.00-NA)
Atrial fibrillation and flutter	5	1	0.11 (0.02-2.27)	0.10 (0.01-2.14)

7 References

American Psychiatric Association, issuing body, 2022. Diagnostic and statistical manual of mental disorders: DSM-5-TR, Fifth edition, text revision. ed. American Psychiatric Association Publishing, Washington, DC.

Beach, S.R., Luccarelli, J., Praschan, N., Fusunyan, M., Fricchione, G.L., 2024. Molecular and immunological origins of catatonia. Schizophr. Res., Catatonia 263, 169–177. https://doi.org/10.1016/j.schres.2023.03.013

Haroche, A., Rogers, J., Plaze, M., Gaillard, R., Williams, S.C., Thomas, P. and Amad, A. (2020). Brain imaging in catatonia: systematic review and directions for future research. *Psychological Medicine*, [online] 50(10), pp.1585–1597. doi:https://doi.org/10.1017/S0033291720001853.

Heckers, S., Walther, S., 2023. Catatonia. N. Engl. J. Med. 389, 1797–1802. https://doi.org/10.1056/NEJMra2116304

Hirjak, D., Rogers, J.P., Wolf, R.C., Kubera, K.M., Fritze, S., Wilson, J.E., Sambataro, F., Fricchione, G., Meyer-Lindenberg, A., Ungvari, G.S., Northoff, G., 2024. Catatonia. Nat. Rev. Dis. Primer 10, 49. https://doi.org/10.1038/s41572-024-00534-w

ICD-10 Version:2019 [WWW Document], n.d. URL https://icd.who.int/browse10/2019/en (accessed 7.17.25).

ICD-11 [WWW Document], n.d. URL https://icd.who.int/en/ (accessed 7.17.25).

Luchini, F., Medda, P., Mariani, M.G., Mauri, M., Toni, C. and Perugi, G. (2015). Electroconvulsive therapy in catatonic patients: Efficacy and predictors of response. *World Journal of Psychiatry*, 5(2), pp.182–192. doi:https://doi.org/10.5498/wjp.v5.i2.182.

Oldham, M.A., 2018. The Probability That Catatonia in the Hospital has a Medical Cause and the Relative Proportions of Its Causes: A Systematic Review. Psychosomatics 59, 333–340. https://doi.org/10.1016/j.psym.2018.04.001

Rasmussen, S.A., Mazurek, M.F., Rosebush, P.I., 2016. Catatonia: Our current understanding of its diagnosis, treatment and pathophysiology. World J. Psychiatry 6, 391–398. https://doi.org/10.5498/wjp.v6.i4.391

Rogers, J.P., Oldham, M.A., Fricchione, G., Northoff, G., Wilson, J.E., Mann, S.C., Francis, A., Wieck, A., Wachtel, L.E., Lewis, G., Grover, S., Hirjak, D., Ahuja, N., Zandi, M.S., Young, A.H., Fone, K., Andrews, S., Kessler, D., Saifee, T. and Gee, S. (2023). Evidence-based consensus guidelines for the management of catatonia: Recommendations from the British Association for Psychopharmacology. *Journal of Psychopharmacology*, 37(4), pp.327–369. doi:https://doi.org/10.1177/02698811231158232.

Rogers, J.P., Pollak, T.A., Blackman, G., David, A.S., 2019. Catatonia and the immune system: a review. Lancet Psychiatry 6, 620–630. https://doi.org/10.1016/S2215-0366(19)30190-7

Smith, J.H., Smith, V.D., Philbrick, K.L. and Kumar, N. (2012). Catatonic Disorder Due to a General Medical or Psychiatric Condition. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 24(2), pp.198–207. doi:https://doi.org/10.1176/appi.neuropsych.11060120.

V, B.P., Abhiram, P., Vaidyanathan, S. and Kumar, P.S. (2022). 'Clinical profile of Organic Catatonia- a case series'. *Indian Journal of Psychiatry*, 64(9), pp.557–557. doi:https://doi.org/10.4103/0019-5545.341601.

Zingela, Z., Stroud, L., Cronje, J., Fink, M. and van Wyk, S. (2022). Management and outcomes of catatonia: A prospective study in urban South Africa. *SAGE Open Medicine*, 10, p.205031212211055. doi:https://doi.org/10.1177/20503121221105579.