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Research Paper

Neurocognitive function and mortality in patients with schizophrenia spectrum disorders

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ABSTRACT

Individuals with schizophrenia spectrum disorders (SSD) have significantly lower life-expectancy than healthy people. Previously, we have identified baseline neurocognitive function in general and verbal memory and executive function in particular as related to mortality nearly two decades later. In this study, we aim to replicate these findings with a larger and age-matched sample. The patient group consisted of 252 individuals, 44 of whom were deceased and 206 alive. Neurocognition was assessed with a comprehensive battery. Results showed that the deceased group, compared to the living group, had significantly more severe neurocognitive deficits across nearly all domains. There were no differences in sex, remission status, psychosis symptoms, or function level between the groups. Immediate verbal memory and executive function were the strongest predictors of survival status. These results were nearly identical to our previous studies, and we conclude that baseline neurocognitive function is an important predictor for mortality in SSD. Clinicians should be mindful of this relationship in patients with significant cognitive deficits.

1. Introduction

Individuals with schizophrenia spectrum disorders (SSD) have significantly lower life-expectancy than healthy people (Hjorthøj et al., 2017), with death occurring up to 20 years earlier than in the general population (Laursen et al., 2014). Suicide and comorbid substance abuse are obvious causes of shortened life expectancy (Correll et al., 2022; Lawrence et al., 2013), as are side effects of antipsychotic treatment (Højlund et al., 2022; Mitchell et al., 2013). Somatic illnesses being leading causes of death in the general population, such as cancer and cardiovascular disease, also are more prevalent in the SSD population (Correll et al., 2022; Lawrence et al., 2013; Tanskanen et al., 2018; Zhuo et al., 2017).

One possible explanation for higher frequencies of somatic illness may be sedentary behavior and comparative lack of physical exercise in SSD patients (Strassnig et al., 2021; Vancampfort et al., 2017). Previously, we have found that reduced life expectancy 18 years after study inclusion was predicted by lowered levels of physical and social activity in SSD (Moradi et al., 2018). Moreover, the link between activity level and life expectancy was the strongest in the patient group with severe neurocognitive deficits. These findings replicated results from a 15-year prospective follow-up study, where verbal memory and executive function were identified as particularly strong predictors of survival status (Helldin et al., 2015). In neither of these studies, psychosis symptom severity, gender, age at onset of illness or sub-diagnosis within the psychosis spectrum emerged as important explanatory factors for mortality (Helldin et al., 2015; Moradi et al., 2018).

Neurocognitive dysfunction is a hallmark of schizophrenia spectrum disorders (SSD) (Kahn and Keefe, 2013), is already present in the prodromal phase (Mollon and Reichenberg, 2018) as well as in adult patients (Mesholam-Gately et al., 2009). The level of impairment seems relatively stable across time (Rund et al., 2016) and tends to persist even though classical psychosis symptoms, e.g., hallucinations and delusions, have lifted. Given that neurocognitive deficits may influence somatic illness and mortality, they should be identified as early as possible in order to alert the clinical community to patients in need of close monitoring of general health. However, most studies of mortality in SSD are retrospective and based on information from deceased patients' medical records. Through the prospective Clinical Long-term Investigation of Psychosis in Sweden (CLIPS) project we are able to perform more thorough studies with stricter methodology by assessing patients annually across 20 years, identifying mental and somatic predictors of

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illness progression and treatment outcome. In the present study, we aim to extend our previous reports of neurocognitive predictors of mortality in SSD patients (Helldin et al., 2015; Moradi et al., 2018) in a larger sample and with stricter control for possible age-related cognitive confounders. We will (1) compare the baseline neurocognitive function of SSD patients who are deceased with those who are still alive, and (2) identify specific cognitive domains that are relevant for survival status.

2. Methods

This study is part of the CLIPS project, which has been described in detail elsewhere (e.g., Helldin et al., 2015). Briefly, men and women suffering from SSD were recruited from the outpatient clinics in the NU Health Hospital region in South-Western Sweden from November 2000. Two thirds of all eligible patients on this geographic catchment area were included at baseline. The participants were required to be in a stable clinical condition. Exclusion criteria were age below 18, mental retardation precluding the ability to give informed consent, past or present neurological illness, and significant past or present use of alcohol or illegal substances. Each participant gave their written informed consent to participation. The project was approved by the Research Ethics Committee in Gothenburg, Sweden, (# Ö537–99, 507–04, 438–10, 423–14, 811–16) and carried out according to the Helsinki Declaration.

2.1. Participants

The patient group consisted of 252 individuals, 44 of whom were deceased per September 2022 and 206 who were alive (Table 1). Survival status was verified by the Swedish National Board of Health and Welfare. The upper age limit was set at 65 years for both groups, in order to prevent biased results due to age-related cognitive effects. The lower age limit was 27 years in the currently living group, as that was the lowest baseline age of a patients that later died. Sub-diagnoses in the deceased group were schizophrenia 65.7 %, schizoaffective disorder 23.5 %, and delusional disorder 10.8 %. Sub-diagnoses among the living were schizophrenia 67.4 %, schizoaffective disorder 25.6 %, and delusional disorder 7.4 %. The vast majority had Swedish as their native tongue, and 3 participants among those who are still alive required the assistance of an interpreter during the assessments. Most of the participants were medicated with second-generation antipsychotics alone or in combination with a first-generation psychotic.

Table 1
Baseline demographic and clinical characteristics of the participants.

	Deceased patients ($n = 44$)	Living patients ($n = 206$)	Test statistics
Gender	28 males (63.6 %) 16 females (36.4 %)	126 males (61.2 %) 80 females (38.8 %)	0.94
Age	46.6 (8.3) min-max 27–61	43.9 (9.8) min-max 27–64	1.84
Highest level of education (missing data n = 2)			14.45**
Elementary school	26 (61.9 %)	66 (32.0 %)	
Senior high school	12 (28.6 %)	101 (49.0 %)	
College level	4 (9.5 %)	38 (18.4 %)	
Marital status (missing data $n = 3$)			0.53
Single (never married)	26 (60.5 %)	122 (59.5 %)	
Married/partnered	8 (18.6 %)	47 (22.9 %)	
Divorced/widowed	9 (20.9 %)	36 (17.6 %)	
Duration of illness in years	22.0 (9.9)	18.8 (10.2)	1.96
In remission (missing data n = 3)	16 (36.4 %)	86 (42.4 %)	0.54
PANSS			
Positive	11.4 (3.9)	12.4 (5.1)	−0.82
Negative	17.3 (5.1)	15.9 (5.2)	1.65
General	32.4 (6.7)	30.8 (6.8)	1.61
GAF			
Symptom	47.6 (7.9)	48.2 (8.8)	−0.09
Function	49.2 (7.5)	50.9 (8.8)	−1.38

Age, duration of illness, PANSS, and GAF in mean (SD). Some of the percentages do not add up to 100, as there are missing data for a small number of participants. Test statistics: Mann-Whitney *U* test or Pearson's Chi square-test.
** $p < .01$.

2.2. Assessments

Diagnosis was determined according to the DSM-IV and ISD-10 criteria (APA, 1994; WHO, 1993). Functional level was determined according to the Global Assessment of Function (GAF), and the total score was split into a symptom and function sub score (Pedersen et al., 2007). Positive, negative, and general symptoms of schizophrenia were assessed with the Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987). Remission status was established according to the guidelines of Andreasen et al. (2005).

Subsequent to the clinical assessments, the participants performed the following neurocognitive tests in their Swedish versions:

The *Trail Making Test A (TMT-A) and B (TMT-B)* 23 (Reitan, 1958) were used as measures of visuomotor speed (part A) and cognitive flexibility (part B).
The *Letter Number Span test (LNS)* (Gold et al., 1997) was used as a measure of auditory working memory.
The *Rey Auditory Verbal Learning Test (RAVLT)* (Schmidt, 1996) consists of a 15-item word list that is read aloud to the participant five times. We used the number of words correctly recalled in trial 1 (RAVLT 1) as an indication of immediate memory, trial 1 to trial 5 (RAVLT 1–5 sum) as an indication of learning, and the number of words correctly recalled after 20 min (RAVLT 7) as an indication of retention memory.
The *Continuous Performance Test–Identical Pairs (CPT-IP)* (Cornblatt et al., 1988) is a computerized test assessing attention and vigilance. In this study, only the numbers part was used.
The *Wisconsin Card Sorting Test (WCST)* (Heaton et al., 1993) is a computerized test of executive function. We used the 6-category WCST version, and the number of completed categories, total trials, and total errors as measures of executive function.
The *Vocabulary* subtest from the revised Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1997) was used as an estimate of premorbid intelligence.

2.3. Statistics

All statistical analyses were performed with IBM SPSS version 26. Non-parametric group comparisons were chosen due to large differences in sample size. Identification of the cognitive domains that contributed most to survival status was done with forward conditional stepwise logistic regression analyses.

3. Results

There were no statistically significant differences in demographic variables or PANSS or GAF scores (Table 1). We recruited out-patients in order to ascertain their ability to undergo a lengthy and cognitively demanding assessment procedure. Moreover, we attempted to assess the participants in a relatively stable phase as close to their premorbid state as possible. Therefore, the psychosis symptom levels were relatively low.

At baseline, the neurocognitive function was significantly lower in the group that was deceased 20 years later compared to those who were alive. The only exception was the score of the Attention/Vigilance domain, which showed no statistically significant group difference (Table 2).

Next, stepwise logistic regression analyses were performed with

Table 2
Neurocognitive function of the participants.

Test	Deceased (n = 44)	Living (n = 206)	Mann-Whitney U test
TMT-A (*Processing speed*)	61.6 (29.4)	49.6 (29.8)	−3.00**
TMT-B (*Flexibility*)	193.6 (112.4)	132.5 (82.8)	−3.38***
LNS (*Working memory*)	7.7 (3.1)	8.9 (2.7)	−2.38*
RAVLT 1 (*Immediate memory*)	3.6 (1.5)	4.7 (1.9)	−3.86***
RAVLT-sum 1–5 (*Learning*)	33.0 (10.1)	40.2 (11.7)	−3.90***
RAVLT-7 (*Retention memory*)	6.1 (2.9)	7.9 (3.5)	−2.97**
WCST Categories compl. (*Exec. f.*)	1.7 (1.7)	3.1 (2.2)	−3.87***
WCST Total trials (*Ecex. f.*)	126.6 (7.4)	118.2 (18.8)	−3.06**
WCST Total errors (*Exec. f.*)	67.4 (20.0)	52.4 (25.3)	−3.52***
CPT-IP Total d'prime (*Att./Vig.*)	0.4 (0.7)	0.5 (0.5)	−1.14
WAIS Vocabulary (*IQ*)	33.9 (12.2)	39.9 (11.2)	−3.02**

Neurocognitive test results in mean (SD) raw scores. *U*: significance test.
*** p < .001.
** p < .01.
* p .05.

survival status as the dichotomous dependent variable and each cognitive test entered in a forward conditional manner as independent variables. The final significant model consisted of RAVLT-1, representing immediate memory and WCST Categories completed, representing executive function (Table 3). This result is almost identical to that from a smaller sample from the same project (Helldin et al., 2015).

4. Discussion

In this study of the relationship between neurocognitive function at baseline and survival status in SSD patients 20 years later, we replicated previous findings from the same project (Helldin et al., 2015; Moradi et al., 2018) using larger samples and age control. The neurocognitive function at baseline was significantly lower in those participants who were deceased 20 years later compared to those who were not. This applied to all cognitive domains except for Attention/Vigilance. Moreover, immediate verbal memory and executive function each contributed significantly to predicting survival status, and with the combined level of explained variance in mortality reaching 17 %. Importantly, survival status was not associated with sex, remission status, psychosis symptoms, or function level.

As other studies in this field are wanting, we have no solid empirical base for our explanation of the relationship between neurocognitive function and mortality. Based in circumstantial evidence, we propose several, and not mutually exclusive, suggestions. First, there is some evidence of a direct link between low neurocognitive function and low cardiorespiratory fitness in SSD patients, possibly part of the neurodevelopmental aberration in those who later present with psychosis (Holmen et al., 2019). Second, we have previously reported low physical and social activity levels in SSD patients with low cognitive function (Moradi et al., 2018). The implication is that information regarding the importance of exercise may go amiss in individuals with compromised cognitive function, or they may lack the practical knowledge necessary for life style changes (Stubbs et al., 2017). Moreover, cognitive deficits may preclude academic or vocational success, causing isolation and loneliness, which are well-known risk factors for premature death (Naito et al., 2021; Strassnig et al., 2017, 2018). Third, symptoms of life-threatening illnesses may not be detected to the same degree in SSD patients as in healthy groups because of their systematic under-use of general health care services (Laursen et al., 2014). Although SSD patients are in frequent contact with mental health institutions, their somatic health is often ignored during regular check-ups with clinicians (Haussleiter et al., 2021). In SSD patients with cognitive dysfunction, communicating concerns over somatic symptoms may be especially difficult unless explicitly questioned by a health care provider. This explanation is compelling because the most common causes of excess mortality in SSD patients are cardiovascular disease, cancer, diabetes, i.e., illnesses that are common causes of death also in the general population, but that seem to be detected earlier and thus have better prognosis in the latter group (Correll et al., 2022; Lawrence et al., 2013; Tanskanen et al., 2018; Zhuo et al., 2017).

Table 3
Stepwise logistic regression analysis of the relationship between baseline neurocognitive function and mortality status (N = 250).

Model	Independent variables	B	Wald	Nagelkerke R^2
Model 1	WCST Cat. compl.	−0.37	12.56***	0.13
Model 2	RAVLT-1	−0.30	5.58*	0.17
	WCST Cat. compl.	−0.31	8.26**	

*** p < .001.
** p < .01.
* p .05.

4.1. Clinical implications

Our findings suggest that cognitive dysfunction has severe consequences for the somatic morbidity and mortality in SSD patients. Care providers must be aware of the extra burden that cognitively compromised patients carry in terms of increased vulnerability to ill health. Standardized somatic examinations should be performed regularly as part of the mental health check-ups that these patients are offered, and the clinicians should be particularly mindful of communication difficulties in these individuals. Several clinicians find obtaining valid and reliable self-assessed health information from severely mentally ill patients difficult (Haussleiter et al., 2021). In a study from the CLIPS project the patient's self-rating ability regarding functional performance relates to neurocognitive performance and real-world functional performance was investigated. The results showed that 37 % of patients overestimate their functional performance and also that clinicians seem to have greater difficulty assessing patients who overestimate their functioning (Olsson et al., 2019). Similar results of overestimation of competence has been obtained by other research groups (Bowie et al., 2007; Gohari et al., 2022), suggesting a general problem of overconfidence in this patient population. However, in a 4-year follow-up study of the same patients (Olsson et al., 2019), we found that with repeated assessments, they became increasingly better at rating, via semi-structured interviews, their own mental and somatic symptoms and communicating them to their case workers (Olsson-Tall et al., 2019).

4.2. Strengths and limitations

Apart from our previous studies (Helldin et al., 2015; Moradi et al., 2018) we are unaware of any other reports of cognitive function predicting mortality in SSD patients. The novelty of this research, and the consistency of results across study samples, are two of the strengths of this study. Other strengths are the prospective design, the long follow-up period spanning 20 years, age-matched samples, and naturalistic, population based samples that are characteristic for Swedish long-term psychosis patients.

Our main limitations are first that our relatively low N, particularly in the deceased group, precluded us from controlling for causes of death, possible side effects of medication, number of psychosis episodes, or other variables that may have contributed to ill health. Second, we had no clinical or healthy control group. There is some indication that cognitive deficits is related to somatic illness in other neurodevelopmental disorders (Li et al., 2022), suggesting that the cognition-morbidity association is not unique to SSD. Third, our

neuropsychological battery contained no tests of visual learning or memory or social cognitive function. Therefore, our results may not generalized to those domains.

CRediT authorship contribution statement

Christine Mohn: Writing, analysis, interpretation of results.

Anna-Karin Olsson: Clinical assessment, interpretation of results, review of paper.

Iris van Dijk Härd: Clinical assessment, interpretation of results, review of paper.

Lars Helldin: Design of study, funding, analysis, interpretation of results, review of paper.

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Declaration of competing interest

The authors report no conflicts of interest.

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