BRIEF REPORT

Neuropsychological correlates of early grief in bereaved older adults

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ABSTRACT

Prolonged grief disorder (PGD) is associated with impairments in cognitive functioning, but the neuropsychological correlates of early grief in older adults are poorly understood. This preliminary study cross-sectionally examined neuropsychological functioning in bereaved adults with high and low grief symptoms and a non-bereaved comparison sample and further explored the relationship between multidomain cognitive measures and grief severity. A total of ninety-three nondemented older adults (high grief: $n = 44$; low grief: $n = 49$) within 12 months post-bereavement and non-bereaved comparison participants ($n = 43$) completed neuropsychological battery including global and multiple domain-specific cognitive functioning. Linear regression models were used to analyze differences in multidomain cognitive measures between the groups and specifically examine the associations between cognitive performance and grief severity in the bereaved, after covariate adjustment, including depressive symptoms. Bereaved older adults with higher grief symptoms performed worse than those with lower symptoms and non-bereaved participants on executive functioning and attention and processing speed measures. In the bereaved, poorer executive functioning, attention and processing speed correlated with higher grief severity. Attention/processing speed–grief severity correlation was seen in those with time since loss $\leq$ 6 months, but not > 6 months. Intense early grief is characterised by poorer executive functioning, attention, and processing speed, resembling findings in PGD. The putative role of poorer cognitive functioning during early grief on the transition to integrated grief or the development of PGD remains to be elucidated.

Key words: bereavement, grief, older adults, aging, cognition

Introduction

Most bereaved older adults successfully navigate grief and transition to integrated grief by 12 months post-loss, but approximately 7%–10% develop prolonged grief disorder (PGD). It is less clear who in the early grieving period will progress to integrated grief versus PGD. Grief theories implicate various loss- and deceased-related cognitive processes that contribute to PGD development. Grieving is also considered a form of learning through experience over time, facilitating integration of loss, and restoring purpose and meaning in life (O'Connor & Seeley, 2022). These processes likely rely heavily on neuropsychological functioning during early grief, and poor neurocognitive performance may disrupt them, potentially leading to PGD. Given the higher frequency of bereavement and variable cognitive abilities in older adults, understanding the neuropsychological functioning during early grief in this population is vital.

Prior evidence suggests that PGD patients exhibit lower global cognition, and worse multidomain cognitive performance compared to non-PGD bereaved and non-bereaved participants, independent of depression status (Hall *et al.*, 2014; O'Connor & Arizmendi, 2014). In contrast, research on the neurocognitive aspects of early grief has produced conflicting results. Some studies have revealed bereaved older adults to perform poorly on memory, attention, processing speed, and verbal fluency tests than their non-bereaved counterparts

(Ward *et al.*, 2007; Xavier *et al.*, 2002). However, these results, in part, were driven by demographic characteristics or cognitive impairment in the bereaved samples, or from neglecting to account for depression severity (Ward *et al.*, 2007; Xavier *et al.*, 2002). It is crucial to account for depressive symptoms when characterizing neuropsychological functioning in older adults with early grief, since depression that is highly prevalent during the first year after loss is associated with cognitive impairments across multiple domains. Moreover, bereaved adults with more intense symptoms during the first year after a loved one's death appear to confer a higher risk of developing PGD (Bonanno & Malgaroli, 2020). Therefore, to gain a better understanding, we explored neurocognitive differences across multiple domains in a well-defined predominantly older bereaved sample with high and low grief symptoms who were within 12 months post-loss and non-bereaved comparison (Comp) participants, while accounting for demographics and depression severity. We further explored how these global and multidomain cognitive measures correlated with grief severity in the bereaved.

Methods

Study participants

136 adults between the ages of 50 and 87 years completed cross-sectional clinical and multidomain cognitive assessments. The bereaved ($n = 93$), except for one participant, were within 12 months following the death of a loved one. One participant was enrolled over 12 months post-loss (i.e., 384 days) to avoid transient symptom increases that can occur around the death anniversary. These participants were separated into two early grief groups based on the 19-item Inventory of Complicated Grief (ICG) scores: (1) high ($n = 44$): score $\geq$ 30 and (2) low ($n = 49$): score < 30. Forty-three Comp participants who were comparable in age, gender, and education and had not experienced bereavement within 18 months were also enrolled.

All participants scored > 21 on the Montreal Cognitive Assessment or > 26 on Mini-Mental State Examination, intact basic and instrumental activities of daily living, and had at least 10 years of education. Exclusion criteria included lifetime history of bipolar, psychotic, or neurological disorders, acute suicidality, substance use disorders during the past 5 years, or delirium/unstable medical conditions. Additional exclusion criteria for the Comp participants included any lifetime psychiatric history, death of a loved one within 18-months, 17-item Hamilton Depression Rating Scale (HAM-D) score $\geq$ 7 or taking psychotropic medications (see supplement).

Participants provided informed consent according to the IRB-approved protocols.

Study procedures

Bereaved participants ($n = 93$) completed the ICG scale. Time since loss (TSL) and relationship of the deceased were documented in the bereaved.

Global and multiple neurocognitive domains, including memory, language, executive function, and attention and processing speed, were assessed in all participants (see supplement). Structured Clinical Interview for DSM-5 Research Version (SCID-5-RV), and assessments measuring depressive and anxiety (i.e., Hamilton Anxiety Rating Scale [HAM-A]) symptoms, and medical illness burden were completed. Psychotropic medication use was documented.

Statistical analysis

Generalized estimating equations (GEE) were used to fit the linear regression models because the normality assumption for the cognitive measures was not justified. GEE-estimated regression coefficients (Coeff) are derived without relying specifically on the distribution of the observations. GEE-fitted models examined the neurocognitive differences between high grief, low grief, and non-bereaved Comp groups, after adjusting for age, gender, education, with and without HAM-D. The Wald Chi-Square (W) test was used as the test of significance and contrasts were calculated to compare the groups, with high grief group as the reference group. GEE-fitted linear models also separately explored the relationship between global and multidomain cognitive measures and ICG scores across all bereaved participants, after adjusting for age, gender, education, TSL, with and without HAM-D.

GEE-fitted models were repeated with added interaction terms to explore if the significant neurocognitive measures-grief symptom associations were moderated by age (dichotomized into age groups < 65, $n = 47$; $\geq$ 65, $n = 46$), TSL (dichotomized into $\leq$ 6 months, $n = 53$; > 6 months, $n = 40$), or HAM-D (dichotomized into score < 16, $n = 54$; $\geq$ 16, $n = 39$) while adjusting for covariates.

The overall significance was determined by $p < 0.05$. Analyses were conducted using SPSS Version 28.0.

Results

The groups did not differ in age, gender, race, education, and medical illness burden. The grief groups did not differ in TSL but significantly
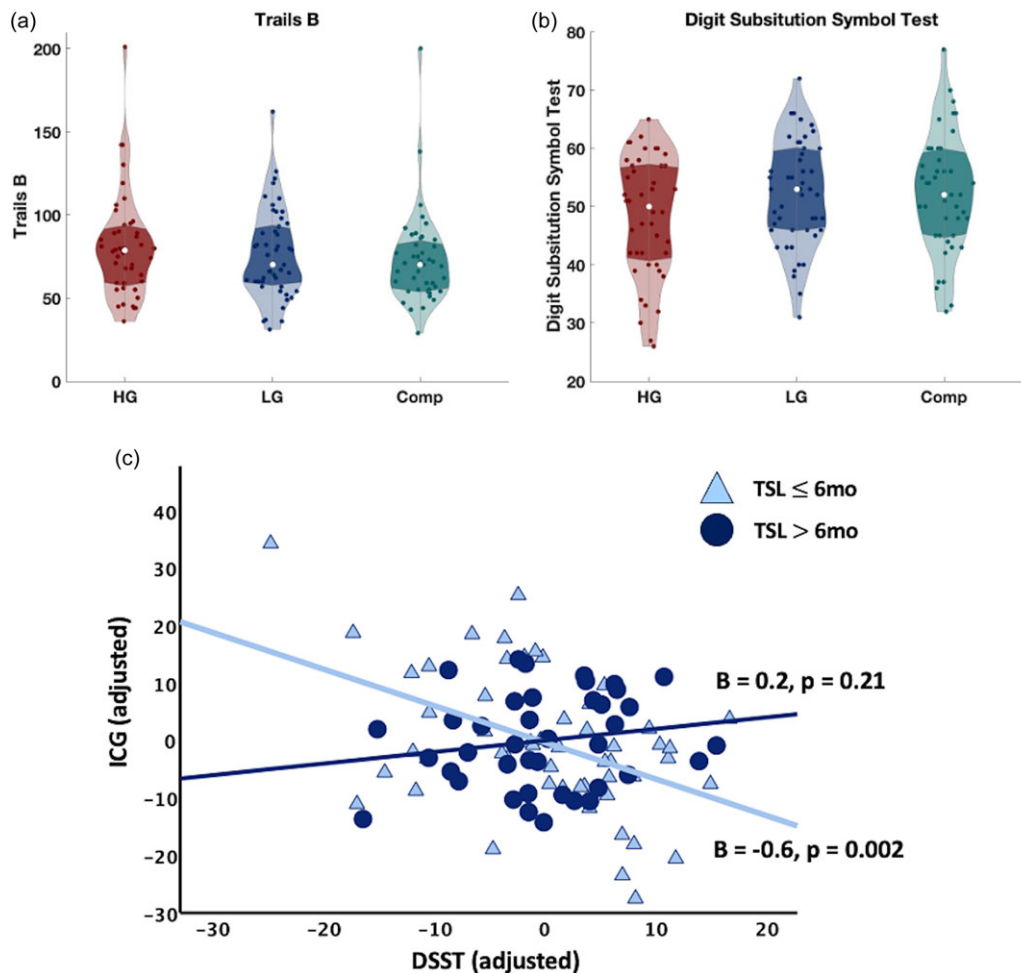
Figure 1. Violin plots illustrate the distribution of raw (unadjusted) (*a*) trails B, (*b*) digit symbol substitution test scores in high and low grief, and comparison participants; and Figure (*c*) shows time since loss as a moderator of DSST-ICG association, adjusting for covariates. HG, high grief; LG, low grief; comp, non-bereaved comparison; DSST, digit symbol substitution test; ICG, inventory of complicated grief; TSL, time since loss; B, beta coefficient; mo, months.

differed in relationship of the deceased. The high grief participants had worse HAM-D and HAM-A scores than the other groups. More high grief participants had current psychiatric disorder and endorsed antidepressant use compared to those in the low grief group (Table S1).

Controlling for age, gender, and education, GEE-fitted models showed significant differences between groups in global cognition, language, and attention/processing speed measures, with high grief participants performing poorly than the other groups (Table S2). While adjusting for demographics and HAM-D, GEE models showed significant differences in executive function (Trails B: $W = 13.79$, $p = 0.001$), and attention/processing speed (Digit Symbol Substitution Test [DSST]: $W = 7.78$, $p = 0.02$) measures. Specifically, high grief participants showed poorer executive function (i.e., higher Trails B scores), and attention/processing speed (i.e., lower

DSST scores) performance than the other groups (Table S3). Violin plots illustrate the distribution of significant (Figure 1a, b) and nonsignificant (Figure S1) neuropsychological measures in the groups without covariate adjustment.

In the bereaved (n = 93), while adjusting for age, gender, education, and TSL, global cognition, language, and attention/processing speed measures negatively correlated with ICG scores (Table S4). Extending the model to also include HAM-D as a covariate, analyses revealed that Trails B scores positively (Coeff = 0.2, $p = 0.003$) and DSST negatively (Coeff = −0.4, $p = 0.04$) correlated with ICG scores.

The DSST-ICG association was moderated by TSL (W = 9.90, $p = 0.002$), with TSL ≤ 6 months being significant: Coeff [95% CI] = −0.6 [−1.0 to −0.23], $p = 0.002$ (Table 1, Figure 1c). None of the other interaction analyses were significant.

Table 1. The relationship between neurocognitive measures and grief severity (ICG) in bereaved participants, while adjusting for covariates

INDEPENDENT VARIABLE	COEFFICIENT (SE) [95% CI]	WALD CHI-SQUARE	*P*-VALUE
Global Cognition			
MoCA[*]	−0.7 (0.6) [−1.8 – 0.5]	1.36	0.24
DRS-2 total	−0.7 (0.5) [−1.8 – 0.4]	1.64	0.20
Memory			
Immediate paragraph recall	0 (0.3) [−0.6 – 0.6]	0.001	0.98
Delayed paragraph recall	−0.1 (0.3) [−0.7 – 0.5]	0.16	0.69
Language			
Boston naming test	−0.7 (0.5) [−1.6 – 0.3]	2.01	0.16
Category fluency	−0.3 (0.2) [−0.8 – 0.2]	1.57	0.21
Executive function			
Trails B	0.2 (0.1) [0.1 – 0.3]	9.08	**0.003**
Attention/processing speed			
DSST	−0.4 (0.2) [−0.7 – 0]	4.13	**0.04**
Trails A	0.1 (0.2) [−0.2 – 0.5]	0.48	0.49
TSL as a moderator[**]			
TSL [*] Trails B		1.28	0.26
TSL [*] DSST		9.90	**0.002**
TSL ≤ 6 mo [*] DSST	−0.6 (0.2) [−1.0 to −0.2]	9.52	**0.002**
TSL > 6 mo [*] DSST	0.2 (0.2) [−0.1 – 0.5]	1.56	0.21

Statistical analyses conducted using generalized estimating equations−fitted linear models, while adjusting for demographics, time since loss (TSL), and HAM−D.

df = 1. *p* < 0.05 highlighted in bold. ICG, Inventory of Complicated Grief; SE, standard error; CI, confidence interval; MoCA, Montreal Cognitive Assessment; DRS-2, Dementia Rating Scale−2; DSST, Digit Symbol Substitution Test.

[*]*n* = 91 (*n* = 2 low grief participants missing MoCA).

[**]This interaction model was adjusted for age, gender, education, TSL, DSST, and HAM-D.

Discussion

This exploratory study comprised a well-characterized sample of nondemented older adults who had lost a loved one in the past year. Individuals with more intense early grief had weaker executive function, attention, and processing speed than those with milder symptoms and non-bereaved comparison participants. In the bereaved, poorer performance in these neurocognitive measures correlated with greater grief symptom severity, with attention/processing speed-grief symptom association found primarily in acute grievers. These associations remained after accounting for demographics and depression severity. The coefficients and CIs from Table 1 suggest that the effect sizes observed here appear small. While the mean cognitive scores of all three groups fell within the normal range for nondemented individuals, the DSST and Trails B scores in the high grief group trended towards the lower end of the normal range. Our results partly align with the neurocognitive substrates of PGD, urging future longitudinal studies to examine the role of cognitive functioning during early grief in PGD development.

Existing literature on the neurocognitive functioning of early grief lacks clarity due to poorly defined samples and inconsistent results. Previous studies included individuals with cognitive impairment (Xavier *et al.*, 2002), or those who had experienced loss within 12–18 months (Ward *et al.*, 2007). In line with our findings, an earlier study found that bereaved older adults had poorer performance in global cognition, processing speed, and language measures. However, these differences disappeared when accounting for mood-related variance, prompting the authors to suggest that a poorer affective state is the primary contributor to cognitive variance after bereavement (Ward *et al.*, 2007). This notion has merit, as late-life depression is characterized by impairments in multiple cognitive domains. In our study, we observed similar trends in global cognition and language measures; yet, the significant association of poorer executive function, attention, and processing speed with grief severity persisted even while adjusting for depressive symptoms. Further analyzing HAM-D as a moderator did not reveal significant interactions. Our preliminary results provide valuable insights into the neurocognitive substrates of early grief.

Older adults during the first-year post-loss show heterogeneous grief trajectories. Notably, a larger proportion enduring intense symptoms during early grief develop PGD, as opposed to those with milder symptoms (Bonanno & Malgaroli, 2020). In the Dual Process Model of grief, confronting versus

avoiding loss and restoration stressors is a dynamic emotional regulatory process, which is considered a crucial mechanism for adaptive bereavement coping. Multiple cognitive processes, such as negative appraisals, rumination, avoidance coping, and impairments in emotional expression and autobiographical memory retrieval, are also believed to contribute to PGD development. These processes likely depend on efficient executive function, attention, and processing speed during the early, particularly in the acute grief phase. We hypothesize that cognitive dysfunction of these domains during early grief, a proxy of disrupted prefrontal-based cognitive control and attentional neural circuitries, can intensify grief-related intrusive thinking and avoidance behaviors, hindering adaptive coping and heightening the risk of PGD development.

Comparable cross-sectional deficits spanning diverse neurocognitive domains, encompassing global cognition, processing speed, and executive function measures are evident, in chronic intense grief and PGD, though with some inconsistencies (Fernandez-Alcantara *et al.*, 2016; Hall *et al.*, 2014; O'Connor & Arizmendi, 2014; Saavedra Perez *et al.*, 2015). Unlike in PGD, we did not find poorer performance in attention or memory measures in early grief. These discrepancies could be due to different cognitive measurements used across studies or suggest that deficits in these cognitive domains might emerge later in the course after PGD arises. Intriguingly, PGD is associated with future declines in global cognition and memory performance over time (Perez *et al.*, 2018). Thus, unanswered questions of great clinical relevance persist regarding the impact of specific neuropsychological impairments during early grief on the development of varying grief trajectories among bereaved older adults. Furthermore, the role of neuropsychological impediments on the chronicity, treatment response, and ensuing cognitive decline in PGD remains poorly understood. Future longitudinal investigations are crucial for unraveling these relationships.

Important limitations require mention. Our self-selected sample was predominantly white, female, and educated. This is a cross-sectional study, and causal inferences cannot be made. We measured grief severity using the ICG. Future studies should utilize the Prolonged Grief Disorder-13-Revised that aligns well with DSM-5 PGD criteria (Prigerson *et al.*, 2021). Lower scores in certain cognitive domains may have driven the observed cognitive-grief symptom associations. While the GEE-fitted models are appropriate for nonnormal distributions, extreme cases may still influence the coefficients. Notably, those with lower cognitive scores also exhibited more severe early grief. Future longitudinal studies with larger samples should disentangle the role of poorer cognitive domain performance on the development of PGD in those experiencing intense early grief. Older individuals with pre-loss poorer cognitive functioning or depression history may grieve more intensely during the acute phase and increase their vulnerability to develop PGD. These factors were not considered here. Our findings should therefore be interpreted cautiously and require replication.

This exploratory study highlights the neurocognitive substrates of early grief and underscores the importance of assessing neuropsychological functioning in bereaved older adults. Further research is needed to understand their possible role in PGD's evolution, persistence, and treatment response. Investigating the neurobiology of these cognitive impediments during early grief might offer insights into innovative therapeutic approaches aimed at preventing PGD.

Conflict of interest

None.

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Description of author(s)' roles

Brianna M. Hoffmann and Nutta-on P. Blair contributed equally to this article.

BMH, NPB, and JSG: conception and design. SAC, JSG, and AW: acquisition of data. BMH, NPB, TLM, GH, EL, CFR, and JSG: analysis and interpretation of data. BMH, NPB, TLM, EL, and JSG: drafting the original version. BMH, NPB, TLM, GH, EL, SC, AW, CFR, and JSG: revising and editing the manuscript critically for important intellectual content and gave final approval. All authors contributed to the article and approved the submitted version.

Supplementary material

For supplementary material accompanying this paper visit https://doi.org/10.1017/S1041610224000048

Data availability

The data have not been previously presented orally or by poster at scientific meetings.

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