Posttraumatic Distress Symptoms and Their Response to Treatment in Adults With Prolonged Grief Disorder

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ABSTRACT

Objective: Posttraumatic stress disorder and prolonged grief disorder (PGD) arise following major life stressors and may share some overlapping symptomatology. This study aimed to examine the presence and response to treatment of posttraumatic stress symptoms (PTSS) in bereaved adults with a primary diagnosis of PGD.

Methods: A randomized controlled trial of 395 adults with PGD (defined as an Inventory of Complicated Grief score ≥ 30 plus confirmation on structured clinical interview) randomly assigned participants to either complicated grief treatment (CGT) with citalopram, CGT plus placebo, citalopram, or placebo between March 2010 and September 2014. This secondary analysis examined the presence of PTSS (per the Davidson Trauma Scale) at baseline and change in PTSS with treatment using longitudinal mixed-effects regression and examined the role of violent compared to nonviolent deaths (loss type).

Results: High levels of PTSS were present at baseline, regardless of loss type, and were associated with increased functional impairment ($P < .001$). CGT with placebo demonstrated efficacy for PTSS compared to placebo in both threshold (OR = 2.71; 95% CI, 1.13–6.52; $P = .026$) and continuous ($P < .001$; effect size $d = 0.47$) analyses, and analyses were suggestive of a greater effect for CGT plus citalopram compared to citalopram alone (threshold analysis: OR = 2.84; 95% CI, 1.20–6.70; $P = .017$; continuous analysis: $P = .053$; $d = 0.25$). In contrast, citalopram did not differ from placebo, and CGT plus citalopram did not differ from CGT plus placebo.

Conclusions: Bereavement-related PTSS are common in bereaved adults with PGD in the context of both violent and nonviolent death and are associated with poorer functioning. CGT shows efficacy for PTSS, while the antidepressant citalopram does not.

Trial Registration: ClinicalTrials.gov identifier: NCT01179568

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The death of a loved one is among the most stressful and traumatic life experiences. While acute grief is a natural experience that most people navigate with time, a notable minority do not adapt to the death and instead develop a persistent grief condition. A diagnostic guideline for prolonged grief disorder (PGD) was recently included in *ICD-11*,[1] and a condition with the same name is being considered for inclusion in a revision of *DSM-5*.[2,3] *Complicated grief* was the term used for this condition in the parent study from which this article's data were derived[4]; however, to align with current nomenclature, this article utilizes the term *prolonged grief disorder*.

PGD is characterized by persistent, impairing grief, including emotional pain and social disruption.[5–9] Available data suggest PGD affects approximately 10% of adults bereaved by natural causes[10] and may be more prevalent with violent deaths such as homicide or suicide.[5,8,11,12] Posttraumatic stress disorder (PTSD) is a relevant construct to PGD, as both occur in response to a major stressor. Both PTSD and PGD are characterized by intense distressing emotions that may be heightened by specific situations or reminders, excessive avoidance of reminders of the loss/trauma, and functional impairment. However, there are also some differences. In PTSD, the primary emotion is fear, while in PGD it is yearning and longing.[13] Further, avoidance is based on an exaggerated or overgeneralized fear of trauma recurrence in PTSD, while it may be driven by difficulty accepting that the deceased is no longer present in PGD.[14]

The handling of the relationship between death of a close attachment and a PTSD-qualifying traumatic event in *DSM* to date has varied. *DSM-5* currently defines loss as a qualifying trauma for PTSD only if the death was violent or accidental, while other types of death do not qualify.[3] Previous studies of loss of a loved one as an inciting event for both PTSD and PGD have reported high comorbidity[13,15] and positive correlation of their symptom severities.[5,16,17]

Clinical Points

- Posttraumatic distress symptoms in bereaved people with prolonged grief disorder, and how they respond to different types of treatments, are poorly understood.
- Patients with prolonged grief disorder should be assessed for the common presence of bereavement-related posttraumatic distress symptoms, which respond better to an evidence-based psychotherapy, complicated grief treatment, than to the antidepressant medication citalopram.

Although not consistently,[18] the violent death of a loved one has been reported to result in higher risk to develop both PGD and PTSD related to the death.[19–21] For example, in a study[22] of 496 bereaved adults, the violent death of a loved one (eg, homicide, suicide, accident) was associated with significantly higher PGD and PTSD severity compared to loss due to illness.

In our randomized controlled trial (RCT)[4] of 395 adults with PGD comparing the selective serotonin reuptake inhibitor (SSRI) citalopram to placebo, with or without complicated grief treatment (CGT), CGT in combination with citalopram or placebo was more efficacious for PGD (treatment response measured with the Complicated Grief–anchored Clinical Global Impression Scale) than citalopram alone. However, CGT with citalopram was superior to CGT with placebo in reducing comorbid depression symptoms. Similar to depression, among pharmacotherapies, SSRIs are first-line evidence-based medications for PTSD.[23] The present study, a secondary analysis of the aforementioned RCT, aimed to compare baseline posttraumatic stress symptoms (PTSS) among violent versus nonviolent deaths and to determine PTSS response to CGT versus citalopram. Further, we aimed to better understand whether PTSS response to treatment is impacted by loss type and whether PTSS are independently associated with functional outcomes in adults with a primary PGD diagnosis. We hypothesized that high PTSS would be present in patients with PGD at baseline, and PTSS would be significantly higher with violent death. Further, we hypothesized that CGT would be more effective in reducing PTSS compared to medication, consistent with the primary study results; however, we also hypothesized that citalopram might be more effective than placebo for PTSS given the established efficacy of SSRIs for PTSD. Lastly, we anticipated that PTSS would have an additive negative impact on work and social functioning, above and beyond the effects of Inventory of Complicated Grief (ICG) scores.

METHODS

Participants

Data originated from HEAL (Healing Emotions After Loss; ClinicalTrials.gov identifier: NCT01179568), a 20-week multicenter RCT of adults with PGD conducted between March 2010 and September 2014. Full study methods and primary results are available.[4,24] Briefly, 395 adults with a primary diagnosis of PGD were randomized to CGT plus pill placebo (CGT + PLA, n = 96), CGT plus citalopram (CGT + CIT, n = 99), citalopram (CIT, n = 101), or placebo (PLA, n = 99).[4] Primary diagnosis of PGD was determined by a score of 30 or higher on the ICG[25] and grief as the primary problem based on a structured clinical interview.[26] Active *DSM-IV* substance use disorder, lifetime psychotic disorder, bipolar I disorder, active suicidal plans, Montreal Cognitive Assessment[27] score less than 21, death-related lawsuit or disability claim, concurrent psychotherapy, and antidepressants were exclusionary. The institutional review boards of each site approved the study. All participants provided written informed consent.

Assessments

Self-report and clinician-rated assessments occurred at baseline and weeks 4, 8, 12, 16, and 20. The Structured Clinical Interview for *DSM-IV*[28] and the Structured Clinical Interview for Complicated Grief,[26] a 31-item clinical interview assessing persistent grief-related symptoms, were administered at baseline. Clinician raters assigned cause of death categories (illness < 1 month, illness ≥ 1 month, accident, murder, suicide, or other). Accident, murder and suicide were categorized as violent death, and illness (both < 1 and ≥ 1 month) was categorized as nonviolent.

The Davidson Trauma Scale (DTS),[29] a 17-item self-report questionnaire that measures *DSM-IV* PTSD symptom severity, was used to assess PTSS. Responses are rated on a 5-point frequency and severity scale (range, 0–136 points). As an anchor, participants identified their "most disturbing trauma" just prior to completing the DTS. A score of 40 has been proposed as a cutoff for PTSD diagnosis[29–31] and was labeled as threshold in our study. The suggested DTS cut-score of 18 for remission of PTSD[32] was labeled as asymptomatic/remission level. Scores from 19 through 39 were considered subthreshold.

The ICG,[25] a 19-item self-report questionnaire, assessed current PGD symptoms. Responses are rated on a 5-point frequency scale with higher scores indicating greater severity (range, 0–76 points).

The Work and Social Adjustment Scale (WSAS)[33] is a 5-item self-report questionnaire measuring impairment in functioning due to grief. Responses are rated on a 9-point severity scale (range, 0–40 points), with higher scores indicating more severe impairment.

Statistical Analysis

Baseline participant characteristics were summarized using frequency and proportion for categorical variables and mean and standard deviation for continuous variables. Baseline differences across treatment groups were compared using analysis of variance (ANOVA) or χ^2 test, as appropriate. Baseline differences in rates of DTS score thresholds (threshold: score ≥ 40, subthreshold: score = 19–39, asymptomatic/remission: score ≤ 18) across loss type were assessed using χ^2 test. Mean DTS score across the 20-week period was compared using paired *t* tests.

Table 1. Baseline Characteristics by Treatment Groups

Characteristic	Total (n = 395)	CGT + PLA (n = 96)	CGT + CIT (n = 99)	CIT (n = 101)	PLA (n = 99)
Age, mean (SD), y	53.0 (14.5)	53.5 (16.0)	52.1 (15.3)	52.4 (13.1)	53.9 (13.8)
Years since loss, mean (SD)	4.7 (7.2)	4.3 (6.7)	4.7 (7.5)	4.6 (5.8)	5.3 (8.7)
Davidson Trauma Scale (DTS) score,[28] mean (SD)	63.2 (27.2)	63.6 (27.8)	63.7 (27.2)	61.5 (27.6)	63.9 (26.5)
PTSS threshold, n (%)[a]					
Asymptomatic/ remission level (DTS score ≤ 18)	14 (3.6)	4 (4.2)	3 (3.1)	5 (5.0)	2 (2.0)
Subthreshold (18 < DTS score < 40)	70 (17.9)	17 (17.9)	21 (21.4)	17 (17.0)	15 (15.3)
Threshold (DTS score ≥ 40)	307 (78.5)	74 (77.9)	74 (75.5)	78 (78.0)	81 (82.7)
Structured Clinical Interview for Complicated Grief[25] score, mean (SD)	42.77 (8.9)	43.00 (8.3)	42.63 (9.4)	43.23 (8.5)	42.23 (9.4)
Work and Social Adjustment Scale[32] score, mean (SD)	22.28 (9.8)	22.17 (9.9)	21.79 (9.6)	21.95 (9.6)	23.21 (10.1)
Cause of death, n (%)[b]					
Nonviolent death	255 (64.6)	61 (63.5)	63 (63.6)	61 (60.4)	70 (70.7)
Violent death	132 (33.4)	34 (35.4)	32 (32.3)	38 (37.6)	28 (28.3)

[a]Assessment with the DTS was not done for 4 of the 395 participants at baseline.
[b]Cause of death due to "other" (n = 8) omitted.
Abbreviations: CGT = complicated grief treatment, CIT = citalopram, PLA = placebo, PTSS = posttraumatic stress symptoms.

For the primary analysis of PTSS changes with treatment, similar to our primary PGD RCT outcomes and primary assessment time points,[4] we used a longitudinal mixed-effects linear regression model with participant-specific random intercepts to examine mean differences in DTS score across treatment arms at week 12 for CIT versus PLA and week 20 for CGT + CIT versus CIT and CGT + PLA versus PLA after adjusting for time, interaction of treatment by time, and baseline DTS score. Time was included as a categorical variable to account for a nonlinear relationship between DTS scores and follow-up weeks.

In follow-up analyses, we investigated whether cause of death (violent vs nonviolent) moderated the relationship between treatment and DTS score by conducting a stratified analysis using the mixed-effects regression model separately for each type of death, adjusting for time and baseline DTS score. We further studied the association between DTS score threshold and treatment arm for completed week 20 DTS using multinomial logistic regression.

Finally, to test whether DTS score improves prediction of WSAS score after adjusting for ICG score at week 20, we examined a likelihood ratio test comparing a reduced mixed-effects regression model with only ICG score as a covariate and a full model with both ICG score and DTS score as covariates. Baseline pooled standard deviation was used to compute standardized mean difference (d) for effect sizes.[34] All significance tests were 2-tailed at an unadjusted significance level of .05. Data analyses used lme4 and nnet packages in R 3.6.2.[35]

RESULTS

Sample Characteristics

Table 1 presents baseline participant characteristics by treatment group. Mean ± SD age at baseline was 52.9 ± 14.5 years, 82.3% (n = 325) were white, and 78.0% (n = 308) were female. The primary loss was due to a nonviolent death for 64.6% (n = 255), while 33.4% of losses (n = 132) were due to violent death: accident (14.7%, n = 58), homicide (4.1%, n = 16), and suicide (14.7%, n = 58). At baseline, sample characteristics and DTS scores were balanced, with no significant differences among treatment groups. The sample with DTS administered was 391 at baseline, 279 at week 12, 282 at week 16, and 279 at week 20.

Posttraumatic Stress Symptoms at Baseline

On the lead-in trauma question, to which the DTS questions are then anchored, all participants identified the death of their loved one as their most disturbing trauma. Fully 78.5% (n = 307) of the PGD sample had a DTS score of 40 or higher. In contrast to our expectations, at baseline there were no significant differences by cause of death in mean ± SD DTS score (violent: 63.2 ± 25.6 vs nonviolent: 62.9 ± 27.7; $P = .623$) or in the proportions of those who met PTSS threshold (violent: 80.2% vs nonviolent: 77.8%; $P = .802$: see Table 1).

Changes in PTSS With Treatment

In the full sample, there was a general trend of decreasing mean DTS score over the 20 weeks, with reductions of 29.8 ($t_{276} = 18.95$, $P < .001$, $d = 1.09$), 35.3 ($t_{279} = 21.39$, $P < .001$, $d = 1.29$), and 37.5 ($t_{276} = 23.56$, $P < .001$, $d = 1.37$) from baseline to weeks 12, 16, and 20, respectively. Figure 1 presents the model-adjusted DTS mean score trajectories over time by treatment arm. Across 20 weeks of treatment, there were mean DTS score reductions of 41.4 for CGT + CIT ($t_{75} = 12.55$, $P < .001$, $d = 1.52$), 43.4 for CGT + PLA ($t_{72} = 12.95$, $P < .001$, $d = 1.59$), 32.3 for CIT ($t_{67} = 10.90$, $P < .001$, $d = 1.18$), and 31.3 for PLA ($t_{59} = 11.93$, $P < .001$, $d = 1.15$). Table 2 presents the model-adjusted differences in DTS score over time by treatment group, which were similar to the primary study's PGD outcomes.[4] At week 12, there was no significant difference in DTS score for CIT versus PLA, nor was there a DTS score difference at week 20 for

Figure 1. Model-Adjusted Mean Davidson Trauma Scale Total Scores Over Time by Treatment Arm

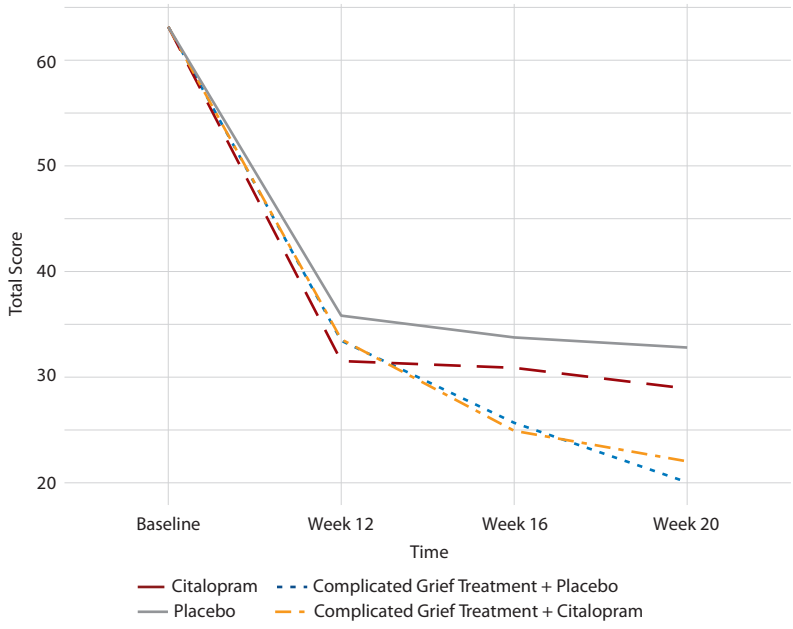


Table 2. Estimates of Model Based Difference in Traumatic Distress as Measured by Davidson Trauma Scale Total Score Reduction by Treatment Group

	CIT vs PLA (Week 12)		CGT + CIT vs CGT+PLA (Week 20)		CGT + CIT vs CIT (Week 20)		CGT + PLA vs PLA (Week 20)	
Outcome	AMD (SE)/*d*	*P* Value	AMD (SE)/*d*	*P* Value	AMD (SE)/*d*	*P* Value	AMD (SE)/*d*	*P* Value
DTS score	−4.3 (3.7)/0.16	.247	2.0 (3.5)/0.07	.570	−6.9 (3.6)/0.25	.053	−12.8 (3.6)/0.47	< .001

Abbreviations: AMD = adjusted mean difference, CGT = complicated grief treatment, CIT = citalopram, *d* = standardized mean difference,[33] DTS = Davidson Trauma Scale,[28] PLA = placebo.

CGT + CIT versus CGT + PLA. However, CGT + PLA versus PLA showed significantly greater reduction in DTS score at week 20, while CGT + CIT versus CIT was nonsignificant ($P = .053$) but suggestive of a potential difference at the level of a small effect size ($d = 0.25$; See Table 2).

Figure 2 presents the proportion who met PTSS threshold (DTS score ≥ 40), subthreshold (18 < DTS score < 40), and asymptomatic/remission level (DTS score ≤ 18) by treatment arm at baseline and week 20. There was no significant difference in the proportion of participants meeting PTSS threshold among treatment arms at baseline, with 82.7% (n = 81) for PLA, 78.0% (n = 78) for CIT, 77.9% (n = 74) for CGT + PLA, and 75.5% (n = 74) for CGT + CIT ($\chi^2_6 = 2.82$, $P = .831$) (See Figure 2). At week 20, the odds of being at the asymptomatic/remission level versus threshold for PTSS were significantly higher for CGT + PLA versus PLA (OR = 2.71; 95% CI, 1.13–6.52; $P = .026$; number needed to treat [NNT]: 6.10) and for CGT + CIT versus CIT (OR = 2.84; 95% CI, 1.20–6.70; $P = .017$; NNT: 7.58). However, there was no significant difference in the odds of PTSS asymptomatic/remission status for CIT versus PLA (OR = 1.12; 95% CI, 0.49–2.56; $P = .781$; NNT: 26.32) or CGT + CIT versus CGT + PLA (OR = 1.18; 95% CI, 0.48–2.92; $P = .721$; NNT: 166.67) at week 20.

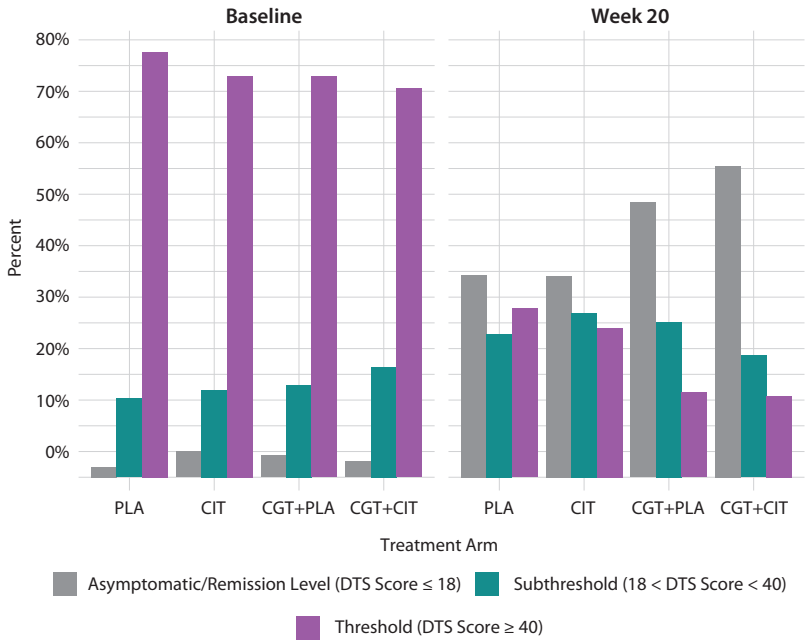
PTSS Over Time Based on Loss Type

DTS score reduction over 20 weeks of treatment did not vary by loss type, with a nonsignificant difference of 4.21 points (b = 4.21, SE = 2.44, $P = .085$, $d = 0.15$). Figure 3 presents the model-adjusted mean trajectories of DTS score for violent versus nonviolent deaths over time by treatment arm. Longitudinal models were next stratified by type of loss. For violent death, the mean difference in DTS score over 20 weeks of treatment was significantly different from PLA for CGT + PLA (b = −16.84, SE = 5.60), $P = .003$, $d = 0.61$) and CGT + CIT (b = −19.10, SE = 5.78, $P = .001$, $d = 0.69$), but not for CIT (b = −10.53, SE = 5.90, $P = .078$, $d = 0.39$). In contrast, for nonviolent losses, there was no significant difference in DTS score change for any treatment arm compared to PLA over 20 weeks.

Association Between Endpoint PTSS and Functional Impairment

At baseline, with adjustment for ICG score, there was a significant association between DTS score and functional impairment (WSAS: b = 0.21, SE = 0.02, $P < .001$). This association remained at week 20 after adjusting for ICG score, treatment group, and time (b = 0.19, SE = 0.016, $P < .001$). Further, the significant likelihood ratio test comparing 2

Figure 2. Davidson Trauma Scale (DTS) Thresholds by Treatment Arm at Baseline and Week 20



Abbreviations: CGT = complicated grief treatment, CIT = citalopram, PLA = placebo.

models (with ICG alone and with ICG and DTS) at week 20 provides evidence that DTS score explains additional variance in WSAS score after controlling for ICG score ($\chi^2_1 = 126.12$, $P < .001$).

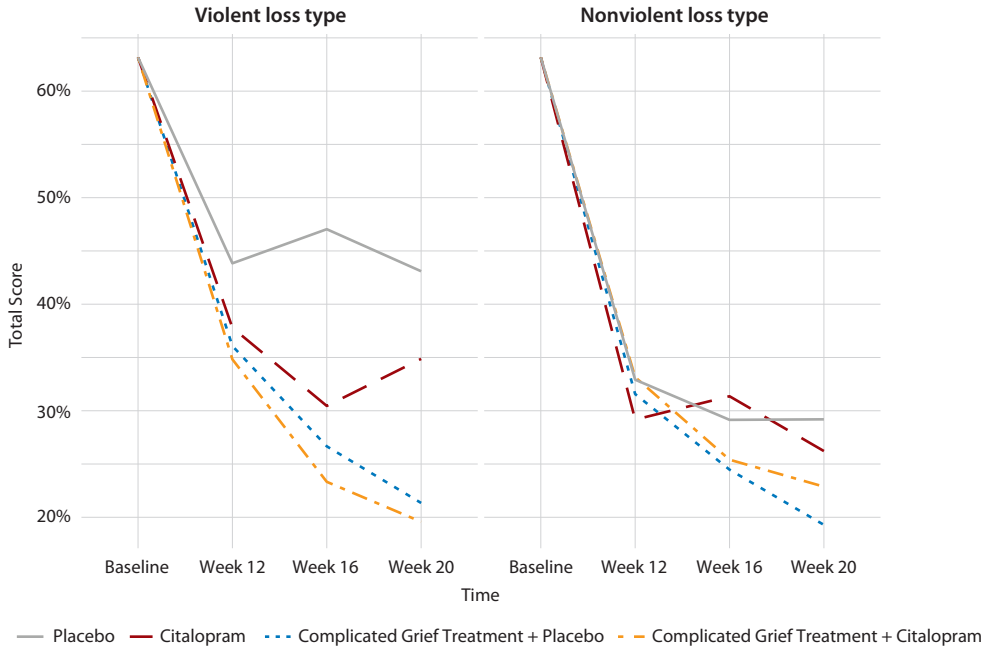
DISCUSSION

The present study investigated differences in PTSS by loss type and in the response of PTSS to treatment. We found that 78.5% of treatment-seeking adults with primary PGD at baseline had high levels of PTSS at the level recommended in the literature as a cut-score for PTSD diagnostic threshold.[32] Further, as measured by the DTS, the level of PTSS and the proportion at the recommended probable PTSD diagnostic symptom threshold level were comparable for violent and nonviolent deaths, suggesting that any type of death can cause traumatic distress symptoms in people with a primary diagnosis of PGD. These findings differ from those of some previous reports[20,36,37] of more severe PTSD symptoms in those who lost loved ones to violent deaths. Other studies,[18,38] however, have reported similar nonsignificant differences between violent and nonviolent deaths. In *DSM-5*, having experienced a nonviolent death of a family member or loved one does not meet the Criterion A definition: the death must be sudden, violent, or accidental to qualify.[3] It is noteworthy that the two-thirds of our participants (65.8%) who met the symptom severity threshold for PTSD based on DTS standards would not qualify for a *DSM-5* PTSD diagnosis, while those with a violent loss at the same level of PTSS potentially could. This finding raises questions about whether PTSS symptoms in adults with a primary PGD diagnosis represent PTSD comorbidity or rather may be inherent PGD symptoms regardless of loss type when the trauma is the same death that resulted in PGD.

Of note, participants with primary PGD assigned to all treatment groups, including placebo, evidenced a mean DTS score reduction of 30 or larger at week 20, with the CGT groups resulting in an even larger effect (mean DTS score reduction of 42 points). Regarding specific treatment effects, our overall results are similar to our prior findings for PGD symptoms[4]: the odds of being at the asymptomatic/remission level versus threshold for PTSS were significantly higher for CGT + PLA versus PLA and for CGT + CIT versus CIT. The same broad pattern of findings was observed when examining continuous PTSS scores, with significantly greater improvement in CGT + PLA versus PLA and a suggestion of potentially greater improvement in CGT + CIT versus CIT, though this latter finding was narrowly nonsignificant ($P = .053$). In contrast, there was no evidence for an effect of citalopram alone versus placebo (CIT vs PLA) or for citalopram combined with CGT (CGT + CIT vs CGT + PLA). US Food and Drug Administration approvals and clinical practice guidelines support SSRIs as a first-line pharmacotherapy for PTSD.[3,39] The lack of significant effect of an SSRI versus placebo for PTSS in patients with a primary PGD diagnosis further highlights the question of whether PTSS may be best understood as part of the condition of PGD, especially when the most significant trauma is the same death associated with the primary PGD diagnosis, as occurred in our sample.

Notably, these PTSS results differ from the parent RCT findings for depressive symptoms. Depressive symptoms

Figure 3. Model Adjusted Mean Davidson Trauma Scale Total Scores Over Time by Treatment Arm in Violent and Nonviolent Loss Groups



improved significantly more in CGT + CIT versus CGT + PLA, whereas PTSS did not differ for CGT + CIT versus CGT + PLA.[4] Furthermore, there was no significant difference in PTSS over 20 weeks with CIT versus PLA in either the nonviolently or violently bereaved subgroups, despite a nonsignificant hint of a potential difference in the violent subgroup at the level of a small effect size ($d = 0.39$, $P = .078$). In contrast, CGT showed a robust effect on PTSS. By design, because we conceptualized PGD as having elements of traumatic distress, CGT includes components informed by prolonged exposure (PE) for PTSD. Specifically, imaginal and situational revisiting exercises are core elements of the treatment that closely resemble imaginal and in vivo exposure. It is thus not surprising that CGT was efficacious in treating PTSS. Further, the lack of additive efficacy for CGT plus citalopram is similar to some findings with PE for combat-related PTSD which show that PE efficacy may not be greater when combined with an antidepressant.[40]

We and many others have conceptualized PGD as a condition comprising both separation distress and traumatic distress symptoms, which has been supported by factor models[25,41,42] and is the conceptualization we used in developing CGT.[4] A number of early studies[43,44] documented high levels of intrusion and avoidance symptoms in bereaved samples. It is possible that traumatic distress symptoms that overlap with PTSD symptoms are core symptomatology of PGD that may not have been captured by the Inventory of Complicated Grief, used to measure PGD symptoms in this study, which may help explain why DTS scores related to the death had additive association with quality of life and function above and beyond ICG scores. Previous studies have reported greater impairment with comorbid PTSD and PGD[45–47] and that PGD was associated with greater impairment than PTSD among bereaved adults.[48] In our analysis, PTSS in patients with PGD were associated with significant functional impairment, and PTSS level was associated with level of functioning after treatment independent of other PGD symptoms. The nature of traumatic distress symptoms in PGD deserves further study; however, the assessment of PTSS should be considered in studies of PGD and its response to treatment. Further, our data demonstrating high levels of traumatic distress across loss types support the classification of PGD as a stress response condition[49–51] and its placement in *DSM-5* in the trauma- and stressor-related disorders category.

Our study has several limitations. First, we did not evaluate PTSD by the gold-standard clinician-rated interview[52] and instead used a self-report scale based on *DSM-IV*, not *DSM-5* criteria. However, we followed an evidence-based threshold from validation studies, which has shown high diagnostic accuracy. Second, there was high dropout in the parent trial, which resulted in 29.4% missing week 20 DTS data.[4] Further, the parent trial was designed to examine the role of citalopram alone or combined with CGT, so all groups included pill administration for balance and blinding, thus precluding our ability to examine CGT without a pill placebo versus CIT in this secondary analysis. Lastly, while typical rules for rating PTSD symptoms based on the primary trauma were followed for the DTS, information on secondary traumas (eg, childhood/sexual trauma) that could have influenced the clinical presentation and/or treatment response was not obtained.

Notwithstanding these limitations, we found that 78.5% of a treatment-seeking study sample of adults with a primary

diagnosis of PGD endorsed DTS scores above the symptom threshold level previously reported for PTSD. Further, PTSS added significantly to functional impairment above and beyond PGD symptom severity as measured by the Inventory of Complicated Grief. These results indicate that adults with primary PGD likely experience clinically significant PTSS that contribute to functional impairment. Importantly, these clinically significant levels of PTSS occur regardless of the nature of the death. Implications of this observation for diagnosing or not diagnosing comorbid PTSD and for accurate characterization of adults presenting with primary PGD with PTSS should be studied. Regardless, our results suggest that PTSS in patients with primary PGD can be effectively treated with CGT but not with SSRIs, similar to our primary study results with PGD symptoms.

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Potential conflicts of interest: **Dr Shear** has received grant funding from the National Institutes of Health (NIH), Department of Defense (DoD), and New York Life Foundation and a contract from Guilford Press for writing a book on grief. **Dr Simon** has, in the past 3 years, received grant funding from American Foundation for Suicide Prevention (AFSP), DoD, Patient-Centered Outcomes Research Institute (PCORI), the Highland Street Foundation, NIH, and Janssen; has served on speaking/Continuing Medical Education (CME)/consulting boards for Vanda, MGH Psychiatry Academy, Axovant Sciences, Springworks, Praxis Therapeutics, Aptinyx, and Genomind; served as deputy editor for *Depression and Anxiety* (Wiley Periodicals, LLC); and received royalty from Wolters Kluwer, and her spouse has equity in G1 Therapeutics. **Dr Zisook** has received research support from COMPASS Pathways, LTD, and is a consultant to Defender Pharmaceuticals. **Dr Reynolds** has received pharmaceutical support for NIH-sponsored research studies from Bristol-Myers Squibb, Forest, Pfizer, and Lilly; has received honoraria as a speaker from Medscape/WebMD; is a paid consultant for Merck in the panel of the Ecology of Insomnia Treatment in Older Adults; and is the co-inventor (licensed intellectual property) of Psychometric Analysis of Pittsburgh Sleep Quality Index (PSQI) PRO10050447 (PI: Buysse). **Dr Bui** has received grants from the DoD, PCORI, and NIH and royalties from Springer. **Dr Adhikari** has received fellowship support from Johnson & Johnson. **Dr Na** has received fellowship support from the American Psychiatric Association Substance Abuse and Mental Health Services Administration Minority Fellowship and received royalty from Wolters Kluwer. **Drs Mauro**, **Robinaugh**, **Skritskaya**, **Szuhany**, **Lebowitz**, and **Malgaroli**; **Ms Suzuki**; and **Mr Chen** report no competing interests.

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