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Pain as a predictor of depression treatment outcomes in women with childhood sexual abuse

Ellen L. Poleshuck[1,2], **Nancy L. Talbot**[1], **Haiyan Su**[3], **Xin Tu**[1,3], **Linda Chaudron**[1,2], **Stephanie Gamble**[1], and **Donna E. Giles**[1]

[1] Department of Psychiatry, University of Rochester Medical Center, Rochester, NY 14642

[2] Department of Obstetrics/Gynecology, University of Rochester Medical Center, Rochester, NY 14642

[3] Department of Biostatistics, University of Rochester Medical Center, Rochester, NY 14642

1. Introduction

Women with histories of childhood sexual abuse are at greatly increased risk for depression and chronic pain. Adult depression is 1.5 to 4 times more common among women with childhood sexual abuse (CSA) histories compared to those who do not report CSA [1,2]. Further, CSA is associated with increased risk for many pain syndromes, including pelvic pain [3], headache [4], gastrointestinal symptoms [5], and musculoskeletal pain [6]. Despite increased risks, no studies to date have examined the role of pain in depression treatment outcomes among women with CSA.

Studies evaluating depression treatments for individuals with pain generally find improvements for depression. In the IMPACT trial, for example, depressed older adults with arthritis who received antidepressant medications and/or Problem-Solving Treatment showed improvement in depression and function [7]. Yet secondary analyses of state-of-the-art depression treatment studies suggest patients with pain may have a less robust treatment response to standardized depression treatments [8,9]. Among 405 depressed primary care patients who received usual care or enhanced care management for depression in the RESPECT trial, pain interference was associated with less depression improvement [10]. Karp and colleagues conducted two studies examining the contribution of pain to depression outcomes in trials of Interpersonal Psychotherapy (IPT) and antidepressant medication [11,12]. In both studies, pretreatment depression and pain were significantly correlated. Further, patients entering treatment with more pain had poorer outcomes and took longer to reach remission.

Given the frequency of comorbid pain and depression among women with CSA, it is important to examine the role of pain in their depression treatment outcomes. Furthermore, little is known about the role of pain on treatment outcomes specifically in younger, predominantly minority women. In a preliminary attempt to consider these questions, we compared women with major depression and histories of CSA with and without clinically significant pain in a study of IPT in a community mental health center (CMHC). We hypothesized that depressed women with

Ellen L. Poleshuck, Ph.D., University of Rochester School of Medicine, 300 Crittenden Boulevard, Rochester, NY 14642, Phone: 585-275-3138, Fax: 585-276-2065, E-mail: Ellen_Poleshuck@urmc.rochester.edu.

CSA who reported clinically significant pain would have greater depressive severity and poorer outcomes compared to women without clinically significant pain.

2. Method

2.1. Design

The participants were 66 women aged 18 and older presenting to a CMHC for psychotherapy. Written informed consent was obtained. Inclusion criteria were major depression based on the Structured Clinical Interview for the DSM-IV (SCID) [13], and sexual abuse history obtained from a structured clinical interview (Talbot et al., 1999). Childhood sexual abuse was defined as unwanted sexual contact prior to age 18, or sexual contact with a family member 5 years or older than the patient. Sexual contact was defined as physical contact of a sexual nature, ranging from fondling to sexual intercourse. Exclusion criteria included psychosis, schizophrenia, bipolar disorder, mental retardation, and active substance abuse.

The sample is derived from two closely related studies: an uncontrolled pilot study of IPT in 36 depressed women with CSA presenting for treatment to a CMHC [14], and a subsequent randomized controlled trial comparing IPT to treatment as usual in 70 depressed women with CSA in the same setting. During intake interviews, 1080 women were screened for eligibility based on depressive symptoms and self-report of CSA. Of 163 eligible women, 133 (81.6%) agreed to be contacted for an evaluation. Twenty-three (17.3%) did not meet inclusion criteria, 4 (3.0%) did not complete the baseline evaluation, 106 (79.7%) enrolled in the study, and 73 received IPT. The first seven IPT patients were not assessed for baseline pain, leaving 66 subjects. Participants in the treatment as usual group received individual therapy from master's level prepared community mental health center psychotherapists who were not trained in IPT. There were no significant differences between the treatment group and the comparison group for baseline depression severity, age, race, or education. To ensure all subjects in the sample received a rigorous, empirically validated treatment for depression, only participants in the IPT group were included.

The IPT protocol included 14 individual weekly sessions followed by two biweekly sessions. Pain was not an identified target for treatment. To be considered trained in IPT, research therapists were required to complete didactic instruction in IPT, successfully complete three training cases, and achieve satisfactory scores on the Therapist Strategy Rating Form [15], which was completed monthly on randomly selected sessions by the PI. Seven master's level community mental health therapists practicing for an average of 13 (SD = 9) years delivered IPT. They continued to receive weekly supervision for their IPT cases after completing their training cases.

Assessments were conducted at baseline, and at 10, 24, and 36 weeks. A single interviewer trained on the SCID conducted all interviews to ensure consistency across participants and time. A consensus model was used to verify subjects' DSM-IV diagnosis on the SCID. Following acquisition of all available data from interviews and chart reviews, Consensus Diagnostic Conferences were attended by a psychologist, a psychiatrist, and the master's level interviewer. The interviewer made a detailed case presentation and consensus multi-axial DSM-IV diagnoses were made. The psychiatrist also assessed the possible role of medical illness in the psychiatric presentation, specifically whether depression may be secondary to medical illness. All procedures were approved by the University of Rochester Institutional Review Board.

2.2. Measures

2.2.1. Pain—Pain severity and interference were evaluated with the bodily pain subscale of the Medical Outcomes Study 36-Item Short Form (SF-36) [16]. Pain severity was assessed with the question, "How much bodily pain have you had during the past four weeks?" Participants responded using a 6-point Likert scale ranging from none to very severe. Pain interference was evaluated with the question, "During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?" Patients responded using a 5-point Likert scale ranging from not at all to extremely. The SF-36 bodily pain scale has been shown to be reliable, with internal consistency ranging from .78–.88. It is associated with ability to work, and has moderate to high (.41–.93) correlations with other standardized pain measures [17]. To identify patients experiencing clinically significant levels of pain, they were classified as high-pain if they reported at study entry both: 1) moderate, severe, or very severe bodily pain, 2) that interfered with normal work moderately, quite a bit, or extremely. Patients who did not report moderate or greater pain intensity and interference at baseline were classified as low-pain. Pain duration and medical diagnosis were not obtained.

2.2.2. Outcomes—*Depression severity* was evaluated with the 17-item Hamilton Rating Scale for Depression (HRSD) [18] and the Beck Depression Inventory (BDI) [19]. These measures are standard in studies of depressed populations, and have good psychometric properties, including sensitivity to detect changes in depression severity. Depression severity on the BDI is interpreted as: 0–13 = minimal depression; 14–19 = mild depression; 20–28 = moderate depression; and 29–63 = severe depression. Depression severity on the HRSD is interpreted as: 0–6 = normal; 7–17 = mild depression; 18–24 = moderate depression; and > 24 = severe depression. *Level of functioning* was assessed by the role-emotional and role-physical subscales on the SF-36 [15]. Convergent and discriminant validity and internal consistency have been demonstrated with the SF-36.

2.3. Data analysis

T-tests and chi-squares were used to compare the participants with high- and low-pain on the descriptive variables. Weighted Generalized Estimating Equations (WGEE) and Generalized Estimating Equations (GEE) methods were used to assess change robustly over time in intent-to-treat analyses [20]. Pain (high or low) was a categorical predictor, and age (continuous) and race (White or African American) were controlled in all analyses. To control for the possible effect of depressive symptoms on SF-36 role-physical functioning, baseline depression scores (HRSD; BDI) were included as covariates in the role-physical functioning analysis.

The impact of missing data was characterized by model estimates through two well-established missing data mechanisms: missing completely at random assumption (MCAR) and missing at random assumption (MAR) [21]. We tested the MCAR assumption by modeling the missingness of patient's response as a function of observed responses using logistic regression. If the results of logistic modeling showed that missingness depended on observed responses, we would proceed using WGEE with weights estimated from the logistic model for missing data. If data were MCAR, GEE methods would then be used. Session attendance did not follow the MCAR assumption for participants with more severe depressed mood (BDI; HRSD), lower emotional role functioning (SF-36), worse physical role functioning (SF-36) and greater bodily pain (SF-36) at treatment entry; each was associated with increased likelihood of remaining in treatment. WGEE methods were therefore used for all models. Omnibus tests are reported.

3. Results

3.1. Study Sample

Our sample of 66 participants had a mean (SD) age of 35.8 (10.7) years; 34 (53.1%) were African American and 30 (46.9%) were White (Table 1). Average household annual income was $17,093 (14,349). Most women were not living with a partner (n = 52, 78.8 %) and had a high school diploma or greater (n = 55, 83.4 %). The women reported extensive childhood sexual abuse, with 30.3 % (n = 20) stating they were abused by a parental figure, and 74.2 % (n = 49) reporting their abuse included penetration. Thirty-five women (53.0%) met study criteria for high-pain at baseline. The women in the high pain group were older (39.3 years, SD = 10.5) than women in the low-pain group (31.8 years, SD = 9.5); there were no differences by pain group for race, marital status, CSA severity, frequency of co-morbid psychiatric diagnosis, or baseline anti-depressant medication use. The average number of IPT sessions completed by 36 weeks was 12.0 (6.9); there was no significant difference between the low- (11.4 (6.4)) and high- (12.6 (7.3)) pain groups in session attendance.

3.2. Treatment response

High-pain patients entered treatment with greater depressive severity (BDI, HRSD) and worse physical function (SF-36 role-physical subscale) and pain (SF-36 bodily pain subscale) than low-pain patients. There was no difference between groups for baseline emotional function (SF-36 role-emotional subscale). Both high- and low-pain patients experienced improvement in depression at 36 weeks (BDI; HRSD) (Table 2). There was no pain × time interaction for depression severity. Although significantly improved from pretreatment, depression scores for the high-pain group remained in the moderate range at 36 weeks compared to low-pain patients whose depression was in the minimal to mild range at study completion. A pain × time interaction was found for role-emotional functioning (SF-36). Low-pain patients had greater improvement over the study period than high-pain patients, who had minimal change in role-emotional functioning. Neither the high nor low-pain patients showed change in role-physical function (SF-36) or bodily pain (SF-36).

4. Discussion

Clinically significant pain was reported by half of the women with CSA presenting for depression treatment at a community mental health center, suggesting it is common in this population. Women with high-pain entered treatment with significantly more severe depression than those with low-pain. This gap was not bridged over the course of treatment. Although depression improved in both pain groups, women with high-pain remained moderately depressed at treatment completion. Baseline emotional functioning was comparable in the high-and low-pain groups, but women with high-pain were significantly less improved in this domain than women with low-pain.

Our goal in this study was to focus on the common and clinically significant symptom of pain, and determine its association with treatment outcomes. This was an effectiveness study in a “real life” clinic using a naturalistic design and a complex patient population. Our findings strongly suggest that clinicians should take note of pain problems among their patients with depression and CSA, carefully monitor their responses to depression treatment, and consider the need for adjunct treatments that specifically target pain. Due to the limited scope of this paper, we could not determine if comorbid physical and mental disorders, medication use, or other related variables account for some of the pain effects. We cannot assert a causal relationship between pain and depression treatment response. It is a possibility that pain is a marker, rather than a cause, of risk for poor outcomes. PTSD and borderline personality disorder, for example, when co-occurring with pain could detract from depression treatment

response, either directly or by potentiating the negative effects of pain. More research designed to elaborate our understanding of pain's role in depression treatment response is warranted.

We can only speculate as to why pain is associated with more severe depression among this sample of depressed women with CSA. In contrast, depression severity did not differ between high and low pain interference among patients in primary care [10]. One possibility is that women with CSA histories and pain experience a biologically discrete variant of depression that requires a different treatment approach [22]. For example, pain may serve as a marker for more significant depressive severity, more advanced progression of depression, or more refractory depression in women with CSA histories. Alternatively, the comorbidity of CSA, depression, and pain may occur more often among a sub-group of vulnerable patients. Another possible explanation is that depressed patients with CSA may have greater pain sensitivity. The consistent finding across self-report (BDI) and clinician-report (HRDS) suggests that this association is not simply a response bias.

A basic premise of IPT is that improving function will in turn alleviate depression. Yet for the women with high-pain, there was a lack of improvement in emotional function despite a decrease in depression severity following treatment. Several possibilities might explain this finding. IPT, like most psychosocial treatments for depression, tends to target the emotional symptoms of depression, perhaps neglecting physical symptoms and daily functioning [23]. Greater emphasis on function may be indicated in the treatment of individuals with pain and may lead to greater improvements in depression. Alternatively, among patients with CSA, depression, and pain, functional improvement may follow mood improvement or not improve to the same degree as among patients with CSA and depression only. Treatment of higher intensity or longer duration or interdisciplinary treatments may be indicated when pain is present. Perhaps pain and its sequelae distract patients from effectively engaging in treatment and following treatment recommendations that might improve function [9]. It is not uncommon for patients to receive contradictory recommendations for managing their pain and depression. For example, rest may be prescribed in response to a pain episode, but may exacerbate depressive symptoms through increased isolation and decreased pleasurable activities. It may be beneficial to design psychotherapy treatments to conceptualize and target both symptoms of pain and depression, as well as the functional contexts in which they occur.

A primary limitation of this study is the lack of comparison to another standardized depression treatment. We do not know if the findings are IPT-specific, or generalizable to other treatments. Pain is extremely challenging to measure due to its implicit subjectivity and individual variability. Additionally, we cannot report on the role of pain duration, pain location, number of pain sites, or medical diagnoses. Our relatively small sample, consisting of two related studies, and the use of retrospective data analyses, mitigate the robustness of our conclusions. The novel treatment sample, which includes minority and low-income individuals, rigorous methodology, and clinically relevant findings, however, warrant the tentative conclusions drawn based on these preliminary findings.

5. Conclusion

In a standardized depression treatment for women with sexual abuse histories, clinically significant pain was associated with worse depression outcomes and less improvement in function. This one variable was very powerful in its prediction about who would respond to treatment. Even if the presence of pain may not explain why certain patients do less well in treatment, it may provide an important marker for identifying those at risk for poorer outcomes. This finding is important not only because the high-pain patients report greater depression severity post-treatment, but also because individuals with clinically significant depressive symptoms after treatment completion are at increased risk for early depression relapse [24]. If

supported by future studies, this study's findings have broad clinical relevance as depression and pain are common and highly comorbid among women with abuse histories. Future studies will aim to develop and test more comprehensive explanatory models of pain, and consider potential confounds and moderators of outcomes, such as severity of abuse, psychiatric comorbidities, medication use, and chronicity of depression.

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Table 1

Sample Description

	Total sample			Low-pain			High-pain		
	Mean (SD)	**Range**	**n**	**Mean (SD)**	**Range**	**n**	**Mean (SD)**	**Range**	**n**
Age	35.8 (10.7)	19–57	66	31.8 (9.5)	20–49	31	39.3 (10.5)	19–57	35**
Education in years	12.0 (6.9)	8–16	66	12.4 (2.0)	9–16	31	12.3 (1.9)	8–16	35
	Total sample			Low-pain			High-pain		
Demographic and Clinical Distributions	%	n		%	n		%	n	
Clinically significant pain	53.0	35							
Race									
African American	45.5	36		58.8	17		45.7	19	
Caucasian	54.6	30		45.2	14		54.3	16	
Marital Status									
Married/living together	21.2	14		22.5	7		20.0	7	
Separated/Widowed/Divorce	47.0	31		45.2	14		48.6	17	
Never married	31.8	21		32.3	10		31.4	11	
Childhood Sexual Abuse									
Parental perpetrator	30.3	20		22.6	7		37.1	13	
Included penetration	74.2	49		71.0	22		77.1	27	
Comorbid Psychiatric Diagnosis									
Dysthymic Disorder	48.5	32		48.4	15		48.6	17	
Chronic Depression	65.2	43		64.5	20		65.7	23	
PTSD	66.7	44		58.1	18		74.3	26	
Panic Disorder	40.8	27		35.5	11		45.7	16	
Social Phobia	13.6	9		16.1	5		11.4	4	
Substance abuse or dependence history	53.0	35		58.1	18		48.6	17	
Borderline Personality Disorder	36.4	24		29.0	9		42.9	15	
Antidepressant use	56.1	37		45.2	14		65.7	23	

** Notes. $p < .01$ difference by pain status; no other variables distinguished between high and low-pain patients

Table 2

IPT for depressed women with sexual abuse histories: Scores at baseline, and 10, 24, and 36 weeks for women with high and low pain

	low pain					high pain				
	Pretreatment (n = 29)	**10 weeks (n = 27)**	**24 weeks (n=21)**	**36 weeks (n=19)**		**Pretreatment (n = 35)**	**10 weeks (n = 24)**	**24 weeks (n=26)**	**36 weeks (n=28)**	
	Mean (SD)	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Δ**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**Δ**
Depression										
BDI – II	27.5 (9.1)	17.5 (12.7)	17.6 (11.2)	13.6 (11.9)	15.2**	37.8 (9.6)	30.1 (14.8)	27.8 (16.3)	28.0 (14.8)	8.9**
HRSD	19.2 (4.8)	13.2 (6.1)	13.9 (6.2)	10.6 (6.5)	9.2**	24.5 (4.9)	20.9 (7.3)	18.1 (7.6)	18.0 (6.4)	14.7**
Function										
Role-Emotional	32.2 (11.4)	43.1 (13.8)	40.5 (14.4)	41.3 (13.5)	22.6**	26.1 (5.8)	30.8 (10.6)	29.4 (10.0)	27.5 (8.7)	4.7[a]
Role-Physical	47.1 (11.3)	50.6 (10.2)	46.9 (11.6)	46.4 (11.9)	2.4	30.2 (5.1)	33.3 (7.6)	32.3 (7.8)	32.5 (8.4)	1.1
Pain	48.5 (8.3)	48.1 (9.6)	47.8 (9.5)	46.6 (11.7)	2.0	29.6 (6.9)	31.6 (6.1)	32.3 (9.7)	30.1 (6.5)	2.1

** Notes. $p < .01$ between pretreatment and 36 weeks; Δ = model-based change from baseline to week 36;

[a] the high-pain group demonstrated significantly less change than the low-pain group from baseline to week 36; BDI-II, HRSD, and Role-Emotional models are calculated using Weighted Generalized Estimated Equations methods; Role-Physical and Pain models are calculated using Generalized Estimated Equation methods.