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Psychopathology and alexithymia in severe mental illness: the impact of trauma and posttraumatic stress symptoms

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Abstract *Objective* To empirically investigate whether or not symptoms of posttraumatic stress disorder (PTSD) are essential for transferring the negative effects of trauma on the severity of severe mental illness (SMI) as recently suggested by an interactive model. *Methods* About 122 inpatients with either schizophrenia or major affective disorder were administered the Posttraumatic Diagnostic Scale, the Symptom Checklist and the Toronto Alexithymia Scale. *Results* At least one trauma was reported by 83 participants (68%) and 28 patients (23%) reported symptoms of current PTSD. Those SMI subjects with current PTSD symptoms had significantly more psychopathological distress and alexithymic features than those with a trauma exposure but without PTSD symptoms and those patients without any traumatic experiences. *Discussion* In line with prior research, our data indicate that patients with SMI have frequently been exposed to traumatic events and that a third suffers from current posttraumatic stress symptoms. Despite some methodological limitations our findings support the interactive model, which posits that a comorbid PTSD increases the symptom severity of SMI. More attention should be directed at assessing trauma and PTSD in SMI patients and at developing therapeutic interventions.

Key words severe mental illness · trauma · posttraumatic stress disorder · psychopathology · alexithymia

Introduction

Patients with schizophrenia and other types of severe mental illness (SMI) such as bipolar or major depressive disorder represent a high-risk group for trauma and the subsequent development of posttraumatic stress disorder (PTSD) [16, 18, 26, 28, 30, 31, 33]. It was reported that the lifetime prevalence of trauma exposure in general ranged between 74% and 98% [16, 27, 33]. Certain types of trauma, e.g. interpersonal ones including physical and sexual aggression, seem to be particularly frequent in individuals with SMI [10, 17–19, 22–19, 29]. As a consequence, the prevalence of PTSD in patients with SMI is markedly elevated compared to that of the general population with rates ranging between 13% and 43% [10, 23, 27, 32, 33, 40]. These high rates in subjects with SMI are not only consistent with their increased exposure to trauma, but also indicate an elevated risk for developing PTSD given exposure to a traumatic event compared to the general population [28].

Most recently, an interactive model was proposed in which PTSD is given a key role in directly and indirectly mediating the negative effects of trauma on the severity and course of SMI [28]. While there is ample evidence of the adverse effects of trauma exposure, particularly of interpersonal trauma, on mental health [reviewed by 1, 42], the proposed model [28] has a slightly different implication: Although, the trauma itself may partially affect the symptom severity and prognosis of SMI, it is rather the PTSD, which predominantly mediates the negative effects.

It was argued that PTSD can directly and indirectly influence the course and severity of SMI [28]. The indirect effects relate to common correlates of PTSD such as substance abuse, re-traumatization and a poor alliance with treatment providers. The direct effects of PTSD on SMI are theoretically mediated by the core symptom clusters that define PTSD [2], i.e. avoidance of trauma-related stimuli,

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distress due to re-experiencing the trauma, and neuroendocrine reactions including mesolimbic dopamine release due to hyperarousal [28]. However, the relationship between specific PTSD symptoms and symptom severity of SMI has not yet been studied [28].

Taking these considerations into account, our study addressed the following hypotheses and questions: (i) If it was true that rather the PTSD than the trauma alone plays the key role in increasing the symptom severity in SMI, SMI patients with post-traumatic stress symptoms might be hypothesized to have more psychopathological impairment than those with a trauma history but without PTSD symptoms and those without traumatic experiences at all. This might be true not only for psychopathological distress, but also for dissociative symptoms and alexithymia, which are frequent phenomena in PTSD patients [7, 20, 25, 37]; (ii) The relationship between the PTSD symptom clusters of intrusions, avoidance and arousal and the symptom severity of SMI remains to be determined.

Methods

■ Participants

A total of 122 inpatients of our university department with ICD-10 diagnoses of either schizophrenia ($N = 64$), schizoaffective disorder ($N = 11$), bipolar disorder ($N = 20$) or major depression ($N = 27$) were studied. Their mean age was 39.6 years ($SD = 13.0$). The sample comprised 55 women (45.1%) and 67 men (54.9%). A detailed description of the number of participants in each diagnostic subgroup, their mean age and the respective gender distribution is given in Table 1. All participants were either in a post-acute state, i.e. they had just been transferred from the closed ward to the open one after partial recovery from the acute episode, or they had initially been admitted to the open ward due to a milder presentation of their psychopathology. They all gave written informed consent and were above the age of 18 years.

■ Measures

The *Posttraumatic Diagnostic Scale* (PDS) represents a 49-item self-report instrument for the assessment of PTSD [13]. The items correspond to the criteria A–F of the DSM-IV [2], and a diagnosis is very likely if all six criteria are met. Criterion A1 is covered by a checklist of 12 traumatic events. In case of more than one trauma, individuals are asked to refer to the most distressing event when completing the subsequent sections including criterion A2, B (intrusions: 5 re-experiencing symptoms), C (7 avoidance symptoms) and D (5 arousal symptoms). The frequency of each of the 17 symptoms in the past month is rated on a 4-point scale (0 = not at all or only one time; 3 = five or more times a week/almost always). The duration of the PTSD symptoms and the subsequent impairment in different life areas are finally assessed. Additionally, the scale allows quantifying the symptom severity by summing the individual's responses corresponding to the PTSD symptom clusters of intrusions, avoidance and arousal.

The revised version of the *Symptom Check List-90* (SCL-90-R) is a 90-item, self-report clinical rating scale widely used to measure current psychopathology [12]. In addition to a global rating (*Global Severity Index*, GSI), it comprises nine subscales: somatization, obsessional compulsion, interpersonal sensitivity, depression, anxiety, anger-hostility, phobic anxiety, paranoid ideation, and psychoticism. The reliability and validity of the German version of the SCL-90-R is similar to the original version [14].

The *Dissociative Experiences Scale* (DES) is a 28-item, self-administered questionnaire with good reliability and validity, which is based on the DSM definition of dissociation [6]. Factor analyses yielded three subscales representing dissociative amnesia, absorption/imaginative involvement and derealization/depersonalization [9]. The psychometric properties of the German adaptation are almost identical to the original version [38].

The 20-item *Toronto Alexithymia Scale* (TAS-20) is the most widely used and validated alexithymia self-report measure [4, 5, 41]. Factor analyses have suggested that the TAS-20 has three subfactors indicating different facets of alexithymia: difficulty identifying feelings (DIF), difficulty describing feelings (DDF), and externally orientated thinking (EOT). Its German version has similarly good psychometric properties as the original scale [3].

■ Statistical analysis

The data analyses were computed using the 'Statistical Package for the Social Sciences' (SPSS, version 11.5). We applied χ^2 -tests and analyses of variance (ANOVA) followed by post hoc pairwise

Table 1 Clinical and demographic characteristics of the diagnostic samples

ICD-10	Diagnostic subgroups	Total		Age		Women	
		N	%	M	SD	N	%
F20	Schizophrenia	64	52	34.6	12.6	22	34
F20.0	Paranoid schizophrenia	61	49	34.0	12.7	22	36
F20.1	Hebephrenia	3	2	22.5	5.0	0	0
F25	Schizoaffective disorder	11	9	38.0	12.2	8	73
F25.0	Manic type	9	7	41.6	10.6	7	78
F25.1	Depressive type	1	1	28.0	–	1	100
F25.2	Mixed type	1	1	19.0	–	0	0
F31	Bipolar affective disorders	20	16	44.1	11.7	8	40
F31.0	Hypomanic	4	3	42.5	6.1	0	0
F31.1	Manic w/o psychosis	7	6	49.9	10.4	2	29
31.3.	Moderate depression	7	6	41.1	13.2	4	57
31.6	Current episode mixed	2	2			2	100
F32/F33	Depressive disorders	27	22	48.7	8.9	17	63
F3x.2	Severe, w/o psychosis	22	18	48.4	8.9	13	59
F3x.3	Severe, with psychosis	5	4	50.0	9.8	4	80
Total		122	100	39.6	13.0	55	45

comparisons according to Bonferroni's least-significant difference method (LSD). Correlation coefficients (Pearson) were calculated. Finally, to assess the impact of the different PTSD symptom clusters on the general psychopathological distress, we performed a linear regression with the GSI of the SCL-90 as the dependent variable. Significance level was established at $P < 0.05$.

Results

At least one trauma was reported by 83 of the 122 patients (68.0%). In those with a trauma exposure, the mean number of traumatic experiences was 2.5 (SD = 1.8; range 1–8). According to the PDS 28 patients (23.0% of all participants and 33.7% of those exposed to a trauma) met the DSM-IV criteria for a PTSD. Table 2 presents a detailed description of the traumatic events reported and indicates which traumatic experiences led to posttraumatic stress symptoms.

Based on these results, the patients were assigned to the following groups: (1) SMI without trauma exposure ($N = 39$; 32.0%); (2) SMI with trauma exposure but without PTSD symptoms ($N = 55$; 45.0%); and (3) SMI with PTSD symptoms ($N = 28$; 23.0%). These three groups did not differ with respect to gender ($\chi^2 = 0.54$, $P = 0.765$) nor to age ($F = 1.12$, $P = 0.330$).

As shown in Table 3, there were significant differences between the three groups with respect to psychopathological distress (SCL-90): Traumatized individuals reporting posttraumatic stress symptoms scored significantly higher on all SCL-90 scales than the traumatized patients without PTSD symptoms and those without a trauma exposure. The later two groups did not differ in their SCL-90 scores apart from the subscales *anger-hostility* and *paranoid ideation*.

The same was true for the dissociative symptoms: While there were no significant differences between the non-traumatized patients and the traumatized ones without PTSD symptoms, these two groups scored significantly lower than the traumatized SMI

patients reporting posttraumatic stress symptoms. This pattern was also seen with respect to alexithymia apart from the subscale externally orientated thinking (EOT; cf. Table 3).

The associations between the severity of the PTSD symptom clusters of *intrusions*, *avoidance* and *arousal* with the various psychopathological syndromes as measured by the SCL-90 were analyzed in the 83 traumatized patients by calculating the respective correlation coefficients. The results are depicted in Table 4 and indicate moderate to high correlations with significant coefficients ranging between 0.291 and 0.691. The symptom cluster of *arousal* showed the highest correlation with the SCL-90 subscales, while the *intrusion* and *avoidance* cluster hardly differed. To explore whether or not the three PTSD symptom clusters are associated with the symptom severity, we performed a linear regression analyses with the GSI of the SCL-90 as dependent variable and the three PTSD symptom clusters as independent variables: Only the PDS subscale *arousal* significantly predicted the general psychopathological distress (standardized $\beta = 0.582$, t -value = 4.94; $P = 0.001$), but the PDS subscales *intrusions* (standardized $\beta = 0.125$, t -value = 0.871; $P = 0.387$) and *avoidance* (standardized $\beta = 0.021$; t -value = 0.141; $P = 0.889$) did not.

Discussion and conclusions

In line with previous results [10, 16, 18, 22, 27, 29, 30, 33] we found that the majority of patients with SMI (68%) have been exposed to at least one trauma in their life time. Particularly high rates were found for interpersonal trauma, i.e. both violent and sexual assault as well as childhood sexual abuse ($N = 99$; 81%), which corresponds to other investigations reporting interpersonal trauma to be frequent in patients with SMI [10, 17, 18, 22, 29]. The overall rate of subjects reporting posttraumatic stress symptoms in our sample was 23.0% and 33.7% of those exposed to a trauma. Again, this finding is in good keeping with

Table 2 Traumatic events as assessed by the PDS in patients with SMI

	All traumatic events		Event leading to PTSD	
	<i>N</i>	%*	<i>N</i>	%**
Combat or war zone experience	3	2.5	–	–
Natural disaster	11	9.0	–	–
Violent assault (non-sexual)	52	42.6	10	12.0
Sexual assault	23	18.9	1	1.2
Childhood sexual abuse	24	19.7	5	6.0
Torture	7	5.7	1	1.2
Imprisonment	8	6.6	1	1.2
Serious/ near fatal accident	37	30.3	3	3.6
Life-threatening illness	25	20.5	3	3.6
Other qualifying trauma	17	13.9	4	4.8

*Percentages refer to the total number of participants ($N = 122$)

**Percentages refer to those exposed to a trauma ($N = 83$)

Table 3 Comparison of psychopathology (SCL-90), dissociative experiences (DES) and alexithymia (TAS) in SMI patients without trauma (group 1; $N = 39$), traumatized SMI patients without PTSD (group 2; $N = 55$) and traumatized SMI patients with PTSD (group 3; $N = 28$)

	Patients w/o trauma (= 1)		Patients with trauma but w/o PTSD (= 2)		Patients with PTSD (= 3)		ANOVA		
	M	SD	M	SD	M	SD	<i>F</i>	<i>P</i> ≤	LSD
SCL-90									
Somatization	0.6	0.6	0.5	0.5	1.1	0.8	8.8	0.001	3 > 1; 3 > 2
Obsessive compulsion	0.9	0.7	1.0	0.8	2.0	0.8	19.6	0.001	3 > 1; 3 > 2
Interpersonal sensitivity	0.8	0.8	0.9	0.8	1.8	0.9	16.6	0.001	3 > 1; 3 > 2
Depression	0.9	0.6	1.0	0.8	1.9	0.9	18.3	0.001	3 > 1; 3 > 2
Anxiety	0.7	0.7	0.8	0.6	1.6	0.8	18.4	0.001	3 > 1; 3 > 2
Anger-hostility	0.5	0.4	0.7	0.6	1.0	0.6	8.2	0.001	3 > 1; 3 > 2; 2 > 1
Phobic Anxiety	0.5	0.7	0.4	0.5	1.5	1.1	22.9	0.001	3 > 1; 3 > 2
Paranoid ideation	0.7	0.5	1.0	0.8	1.5	1.0	10.2	0.001	3 > 1; 3 > 2; 2 > 1
Psychoticism	0.5	0.5	0.7	0.6	1.3	0.7	12.7	0.001	3 > 1; 3 > 2
GSI	0.7	0.5	0.8	0.5	1.6	0.6	24.9	0.001	3 > 1; 3 > 2
DES	9.3	10.9	13.1	9.1	25.0	18.4	13.2	0.001	3 > 1; 3 > 2
Amnesia	6.6	10.3	8.7	8.0	17.9	18.3	7.7	0.001	3 > 1; 3 > 2
Absorption	12.9	13.1	16.2	10.3	31.4	21.2	14.0	0.001	3 > 1; 3 > 2
Derealisation	7.6	13.2	8.8	11.1	23.5	21.2	11.2	0.001	3 > 1; 3 > 2
TAS total	46.3	9.7	47.3	11.1	55.0	12.1	6.0	0.003	3 > 1; 3 > 2
DIF	11.1	4.6	12.4	4.8	16.1	6.3	8.0	0.001	3 > 1; 3 > 2
DDF	11.9	3.3	12.5	4.1	15.4	4.8	6.9	0.002	3 > 1; 3 > 2
EOT	21.1	4.2	20.4	4.7	20.7	4.3	0.3	0.780	

Table 4 Correlations of the PTSD symptoms of intrusion, avoidance and arousal (PDS) with different psychopathological syndromes (SCL-90) ($N = 83$ traumatized patients with a SMI)

	Intrusion	Avoidance	Arousal
SCL-90			
Somatization	0.419*	0.332*	0.550*
Obsessive compulsion	0.479*	0.447*	0.691*
Interpersonal sensitivity	0.414*	0.482*	0.566*
Depression	0.427*	0.440*	0.548*
Anxiety	0.526*	0.439*	0.588*
Anger-hostility	0.300*	0.409*	0.561*
Phobic anxiety	0.547*	0.501*	0.662*
Paranoid ideation	0.291*	0.356*	0.404*
Psychoticism	0.393*	0.380*	0.468*
GSI	0.518*	0.526*	0.677*

* $P < 0.01$

prior research indicating a PTSD frequency in SMI ranging between 13% and 43% [10, 16, 26, 27, 29–31, 33–31, 40].

Our main finding, however, is that the non-traumatized SMI patients and the traumatized ones without PTSD symptoms did not differ in their psychopathological impairment, their dissociative symptoms and their alexithymia scores, while the sample of the traumatized SMI patients with PTSD symptoms showed significantly more psychopathological distress, dissociative and alexithymic phenomena (apart from the TAS-20 subscale EOT). This finding empirically supports the model proposed by Mueser and co-workers [28] assuming that a comorbid PTSD increases the symptom severity of SMI. According to our results, this is not only true for the core features of SMI such as depression, hostility, paranoid ideation and psychoticism, but also for associated features, e.g. somatization [34],

dissociation [8, 39] and alexithymia [11, 24]. However, with respect to alexithymia one has to discuss whether it represents a personality trait thus, being a risk-factor for the development of PTSD given trauma exposure or whether alexithymia might be a secondary state reaction to traumatic experiences and PTSD [21, 43].

Mueser and colleagues [28] assumed that PTSD is essential for transferring the negative effects of trauma on the severity and course of SMI via direct and indirect pathways. As indirect factors, three common correlates of PTSD were hypothesized including substance abuse, re-traumatization and interpersonal difficulties leading to a poor alliance with treatment providers. Meanwhile, there is empirical evidence for these considerations [29]. The direct pathways between PTSD and SMI were theoretically linked to the three core symptom clusters of PTSD [28]: The avoidance of trauma-related stimuli often extends to

personal relationships leading to social isolation and reduced opportunities for reality testing. Distress due to re-experiencing the trauma might be an intermittent or chronic stressor increasing symptom severity. Chronic hyper-arousal might impair cognitive functioning and reality testing. However, empirical support for the direct influence of PTSD on the symptom severity of SMI has been lacking. Our findings provide preliminary support that all PTSD symptom clusters, i.e. intrusions, avoidance and arousal are closely associated with the severity of psychopathology in patients with SMI. Although all three symptom clusters explain at least 8% variance in the respective SCL-90 subscale, the only significant predictor for the general psychopathological distress was the PTSD symptom cluster of arousal. Thus, we suggest that therapeutic strategies should particularly focus on the reduction of the hyperarousal in patients with SMI [15, 36].

The proposed model [28] remains silent about the temporal or even causal relationship between trauma and SMI as they are complex and interacting. However, there is evidence that early trauma predicts the later development of psychiatric illness, and that mentally ill subjects are more vulnerable to developing PTSD given trauma exposure than non-mentally ill persons [28]. Thus, psychiatric disorders, particularly SMI, might be considered as risk factor for PTSD. However, our data do not allow any conclusions on that issue because we did not analyze the chronological order of the occurrence of trauma, PTSD and SMI.

Further methodological limitations of our study also need to be acknowledged including the relatively small sample size. PTSD symptoms were assessed by a self-report screening instrument (the PDS) and not cross-validated by a structured interview. Although the scale showed good agreement (82%) with the PTSD module of the SCID [13], PTSD diagnoses based on the PDS must be considered presumptive. According to the theoretical considerations of Mueser and co-workers [28], PTSD might worsen the course of SMI; however, due to the cross-sectional design of our study we cannot comment on that issue, which is another limitation. Finally, we did not assess or even control for comorbid conditions other than PTSD symptoms; thus, it remains open whether or not posttraumatic stress symptoms mediate their complicating impact in a special way compared to other comorbid disorders.

Despite these caveats possibly limiting the generalizability of our study, the presented findings underscore the urgent need for more rigorous research on the prevalence, correlates, and course of trauma and PTSD in patients with SMI [35]. To improve the psychiatric outcome and quality of life of these patients, more attention should be directed at assessing trauma and PTSD in these samples and at developing useful therapeutic interventions [15, 36].

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