Posttraumatic stress disorder in eating disorder patients: The roles of psychological distress and timing of trauma

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ABSTRACT

Exposure to traumatic events may be a risk factor for subsequent development of an eating disorder (ED). In a previous study, we showed that trauma exposure impacted symptom load in ED patients. We also saw an effect of trauma on general psychological distress. The aim of the present study was to investigate the association between Posttraumatic stress disorder (PTSD) and ED severity, to focus on the mediating role of psychological distress for the association, and to assess the role of timing of trauma in relation to emergence of ED. Participants were Swedish adult ED patients with a history of traumatic exposure (N=843, Mean age 27.2, 97.3% female). One fourth (24.1%) of the participants had a lifetime diagnosis of PTSD. PTSD had an impact on ED severity, but the impact was mediated by psychological distress. When stratifying the sample based on timing of trauma a significant effect was present only in those with trauma within a year of emergence of ED. The results suggest emotion regulation as a possible underlying factor of interest in future research.

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1. Introduction

Exposure to traumatic events may be a risk factor for subsequent development of an eating disorder (ED; Smyth et al., 2008). Severe long term impairment related to the trauma in the form of posttraumatic stress disorder (PTSD) has been suggested as a possible mediator between the traumatic event and the ED (Brewerton, 2007). In a previous study (Backholm et al., 2013), we showed that trauma exposure impacted symptom load in ED patients, although trauma type did not consistently impact symptoms or ED sub-disease. We also saw an effect of trauma on psychological distress. Extending that work in light of other trauma research, the present study investigated whether psychological distress may act as a mechanism for the impact of PTSD on ED severity. Also, in our previous study we found differences between diagnostic groups in when the trauma had occurred, but we did not consider the temporal order between trauma and emergence of ED symptoms, which has been suggested as important for research in order to better understand how these constructs interrelate (Litwack et al., 2014). This is a second focus of the present study.

The symptomatology of an ED can include restrained eating, a sense of loss of control, or varying forms of concern or distress. In the fifth version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), anorexia nervosa (AN) includes preoccupation with shape and weight, and striving for thinness by restrained food intake, resulting in negative physiological consequences related to starvation and emaciation. Bulimia nervosa (BN) symptoms consist of periods of intense binge eating, followed by behavior aimed at compensating for calorie intake and avoiding weight gain, such as vomiting. Excessive eating is also a central symptom in Binge eating disorder (BED), where the food intake is followed by feelings of distress such as guilt, embarrassment or disgust, but no regular compensatory behavior. The DSM-5 further includes a residual Other specified feeding or eating disorder diagnosis (OSFED) category for clinically significant EDs that do not meet full criteria for any of the above mentioned EDs.

A traumatic event (TE) is in the DSM-5 described as a situation which involved direct or indirect exposure to actual or threatened death, serious injury, or an intrusion on physical integrity such as sexual violence (American Psychiatric Association, 2013; Friedman, 2013). Direct exposure includes being the primary
victim or witnessing the event, while indirect exposure is learning about such an event happening to a close relative or friend.

Exposure to a TE is a required stressor criterion for a PTSD diagnosis. Additional criteria include intrusive symptoms, i.e. when the trauma is re-experienced in a sudden and involuntary manner, and avoidance of reminders of the trauma. Further symptoms include negative changes in cognition and mood, e.g. loss of interest in daily activities or persistent negative thoughts about oneself or the world, and alterations in arousal/reactivity such as vigilance, aggression, or self-destructiveness (American Psychiatric Association, 2013; Friedman et al., 2011; Miller et al., 2014).

Normal responses in the direct aftermath of traumatic exposure often include varying stress-related psychological and physical reactions. Most people will be able to cope with the event, and responses will gradually diminish and finally disappear in the months following the crisis. However, in a minority of those affected, involuntary stress reactions will continue in the long run (Norris et al., 2009; Vogt et al., 2014). To be able to cope with this distress, a person may develop persistent emotion regulation strategies such as avoidance strategies or negative cognitive alterations, tension-reducing behaviors such as binge eating (Briere and Spinnazzola, 2005), and/or strict control strategies (Briere and Scott, 2007; Litwack et al., 2014).

Knowledge is limited about the pathways between exposure to a TE and its association with development of an ED or ED severity, and role the timing of the traumatic exposure may have (Brewerton, 2007; Briere and Spinnazzola, 2005). Exposure to a TE may not necessarily be related to more ED impairment if this is defined as severity of symptoms (Brewerton, 2007), but has been proposed as a risk factor for developing EDs (Amstadter et al., 2013; Briere and Scott, 2007). The subtype of trauma may in turn be of importance, as interpersonal violence, and in particular sexual trauma, commonly have been associated with ED (Backholm et al., 2013; Lejonclou et al., 2014; Mitchell et al., 2012). Traumatic exposure also seems to be specifically linked to ED symptoms of a binge/purge subtype, as compared to restrictive types of EDs (Brewerton, 2007).

Is the traumatic exposure in itself or the possible post-trauma psychological distress central for the connection between ED symptomatology and trauma? The role of comorbid mediating factors is unclear and Mitchell and colleagues (2012) suggest that due to the etiological complexity of EDs, there may not be a “one size fits all” conceptualization of the link between traumatic exposure and an ED. However, scholars have suggested that the development of PTSD may partially or fully mediate the relation (Dansky et al., 1997; Mitchell et al., 2012); that is, ED symptoms may result from trauma if significant psychiatric sequelae develop from it, but not otherwise. Several other forms of psychological distress, including major depression, dissociation or substance abuse, may also be associated with trauma history in ED samples (Brewerton, 2007). It is not clear whether PTSD specifically, or psychiatric symptoms generally, are related to ED, in the context of traumatic exposure. In Fig. 1, a conceptual framework for the possible roles of traumatic exposure, PTSD, psychological distress, and ED is presented. The mediation pathway from trauma via PTSD to ED has been suggested previously (Brewerton, 2007), and general psychological distress (symptoms of depression and anxiety) associated with the PTSD may be more proximally related to ED. Thus, traumatic exposure may influence ED both directly and indirectly via PTSD, and PTSD in turn may exert an effect directly or via depressive and anxious symptoms. The present study aimed to test the second part of the model; the possible mediation of PTSD by general psychopathology, to impact ED.

In addition to the issue of mediating comorbid factors, the timing of the TE in relation to the emergence of first ED symptoms, such as body concern, weight regulation etc, needs to be taken into account. After trauma, e.g. symptoms of PTSD, problems with emotion regulation, insecure attachment patterns, or inadequate interpersonal skills may develop prior to, alongside or following the emergence of an ED, and may impact ED presentation (Fairbank et al., 2014; Pat-Horenczyk, 2008; Resick et al., 2012). Although causal links between e.g. childhood adversity and adult psychological disorders may be elusive, since complex patterns of contextual factors have contributed to personal development (Briere and Jordan, 2009), temporal relations between trauma and ED symptoms may yield insights into when and for whom trauma history is an important intervention target in ED care.

1.1. Aims

The aim of the present study was to investigate the association between PTSD and ED severity, to focus on the mediating role of psychological distress for the above-mentioned association, and to assess the role of timing of trauma in relation to emergence of ED.

2. Method

Participants were the adult ED patients who reported a trauma (N=843) in the study by Backholm and colleagues (2013). The data was extracted from the internet-based and fully computerized “Stepwise” assessment system and clinical database (Birgegård et al., 2010). Data was collected prior to treatment start when intent to treat was established, and ED and comorbid psychiatric diagnoses were assessed using semi-structured interviews, followed by clinical ratings, demographic and psychiatric history data, and questionnaires. At time of extraction 41 specialist ED units in Sweden used Stepwise. The participants (97.2% female) were M=27.2 years old (range 18–66, SD=8.67). Below we describe the measures analyzed in the present study; for a description of all measures included see Backholm et al. (2013).

2.1. Measures

2.1.1. ED diagnosis

ED diagnosis was determined by the clinician conducting the assessment based on a suggestion from a structured interview, until February 2008 the ED module of the Structured Clinical Interview for DSM-IV Axis I disorders (SCID-I; First et al., 1998) and later the Structured ED Interview (SEDI; De Man Lapidoth and Birgegård, 2010). The SEDI, developed specifically for the Stepwise system, is based on DSM-IV ED criteria and comprises 20–30 questions, depending on invoked skip-rules. Preliminary validation against the EDE interview (Fairburn, 1993) has shown good concordance, Kendall’s Tau-b = 0.69 (De Man Lapidoth and Birgegård, 2010).
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