A New Perspective in Post-Traumatic Stress Disorder: Which Role for Unrecognized Autism Spectrum?

Liliana Dell’Osso*, Riccardo Dalle Luche, Claudia Carmassi

Psychiatric Clinic, Department of Clinical and Experimental Medicine, University of Pisa, Pisa, Italy

Post-Traumatic Stress Disorder (PTSD) has aroused considerable interest since its first appearance in DSM-III (American Psychiatric Association, 1980) and increasing prevalence rates have been detected not only in selected samples of trauma exposed subjects but also in the general population (Kessler et al., 1995; Darves-Bornoz et al., 2008; Carmassi et al., 2014a). The number of studies conducted so far on PTSD has shed light on its clinical and neurobiological characteristics leading, for the first time in the DSM-5 (American Psychiatric Association, 2013), to encode it in a specifically devoted chapter, independent from other anxiety disorders, named Trauma and Stress Related Disorders, where it is included besides other specifically trauma related disorders. PTSD uniqueness amongst psychiatric disorders is not only due to its usual relationship to a clear time of onset but also, more specifically, to the fact that it is characterized by the failure of the normal ability to cope with traumatic events. Upon these characteristics, the identification of possible risk or protective factors is a core issue in this field (Zohar et al., 2011). There is agreement concerning the differential vulnerability to PTSD related to gender, with women being the most affected, to level of exposure, to the type of trauma and to age (Karam et al., 2014; Carmassi et al., 2014b), but it is still debated which could be the basic etiopathogenetic mechanism leading to the impairment of the adaptive response (Dell’Osso et al., 2011; 2013a,b).

Some new perspectives may derive from most recent research on Autism Spectrum Disorder (ASD). Increasing data, despite still scant, suggest that ASD subjects may be often exposed to traumatic experiences and, thus, they may be likely to develop PTSD (Hofvander et al., 2009; Storch et al., 2013). A growing body of data indicates that individuals with ASD, particularly those with moderate forms and with no cognitive or language impairment, often come to clinical attention when other mental disorders arise, leading to challenging diagnostic procedures (Kamio et al, 2013). Sometimes, their ASD remain unrecognized even after the onset of these mental disorders. Takara & Kondo (2014), for example, recently reported 16% ASD prevalence rate among first-visit depressed adult patients while Kato et al. (2013) reported rates as high as 7.3% of previously unrecognized ASD among suicide attempters hospitalized for inpatient treatment. Consistently, Storch et al., (2013) showed suicidal thoughts and behaviors to be common in youths with ASD, and associated with the presence of depression and PTSD, leading to suggest that individuals with ASD may represent a low-resilience group that could be specifically prone to develop Trauma and Stress Related Disorders. As a matter of fact, the ability to adjust has been shown to decline dramatically over time in ASD patients with respect to healthy control subjects in the aftermath of a natural disaster exposure (Valenti et al., 2012). However, it is important to notice that only a few studies explored PTSD prevalence rates among ASD patients, and those who did it reported low rates (Mayoral et al., 2010); similarly, to the best of our knowledge, no study explored ASD symptoms in PTSD subjects. This is even more surprising if we consider recent hypotheses suggesting ASD patients to be at high risk for trauma related disorders particularly because of their difficulties in expression, empathy, and understanding of shared codes of communication that would render them prone to chronic traumatization across the entire life (King et al., 2010).

PTSD symptoms can arise from multiple traumas according to the Complex PTSD model (Terr, 1991; Herman, 1992; Van der Kolk, 2005; Hofvander et al., 2009), that is to say prolonged and/or repeated trauma, such as bullying episodes or repeated sexual abuse. Complex PTSD symptomatology is often chronic and more severe than typical PTSD symptoms, including deficits in emotional regulation, negative self-perception, interpersonal problems and dissociative symptoms (King, 2010; Cloitre et al., 2013; Maercker et al., 2013). It has been observed that the symptoms of Complex PTSD, prolonged across time, lead to long-term instability in interpersonal relationships, emotional lability, unstable self-perception, as well as to maladaptive behaviors, such as substance abuse and self-injuring, such that these subjects might ultimately be labeled as Borderline Personality Disorder (BPD) patients (King et al., 2010). The vulnerability of ASD subjects to multiple traumas may reasonably lie on the severe impairment in interpersonal relationships and the inability to cope with conflicts or deep affective involvements, so that we may speculate that the low incidence of PTSD detected in previous investigations may be related to the characteristic difficulties in understanding, mentalizing and expressing traumatic events. This would prevent ASD subjects from reporting the traumatic event and would lead to overlook the diagnosis of PTSD on one hand, and to increase the probability of a diagnosis of BPD, on the other. Despite Complex PTSD never entered the official psychiatry nosography, the DSM-5 somehow recognized its relevance by adding maladaptive behaviors among core symptoms of PTSD (American Psychiatric Association, 2013; Dell’Osso et al., 2009). The inclusion of maladaptive behaviors, such as reckless or self-destructive behaviors, alcohol and drug abuse into DSM-5 criterion E (alterations in arousal and reactivity) (American Psychiatric Association, 2013; Carmassi et al., 2013) has allowed to capture the whole clinical presentation of this disorder, actually also making PTSD to overlap with some aspects of BPD (Sar, 2011). At the same time, encoding PTSD in a chapter specifically dedicated to post-traumatic stress reactions in the DSM-5 highlights the peculiarity of all these conditions that could be accounted as part of a post-traumatic stress spectrum that includes also adjustment disorder. In this regard, it is important to acknowledge that recent studies reported high rates of unrecognized adjustment disorder in outpatients seeking for treatment for a depressive symptomatology or suicidal behaviors (Cloitre et al., 2014; Takara & Kondo, 2014). A possible role of rumination has also been speculated, suggesting that ruminative thoughts arising from interpersonal difficulties, conflicts and traumas may represent a transnosographic dimension encompassing major depression and PTSD towards ASD, and triggering suicidal behaviors (Kato et al., 2013).

Some authors explored the role of neurological soft signs (NSS), such as alterations in language, motor coordination and other

*Correspondence regarding this article should be directed to: liliana.dellosso@med.unipi.it
functions within the debate about the relationship between ASD and trauma related disorders. Increased NSS have been separately found in different psychiatric conditions, including Asperger’s Disorder (currently merged into ASD) (Tani et al., 2006; Janssen et al., 2009; Valenti et al., 2012) and chronic PTSD (Gurvits et al., 2000). Interestingly, in the latter higher rates of reported neurodevelopmental problems were found in patients with chronic PTSD compared to exposed subjects who did not develop PTSD, suggesting alterations in neurodevelopmental processes as important vulnerability factors for PTSD.

In light of these most recent evidences, we argue that future research should carefully explore the relationships between ASD symptoms and post-traumatic stress disorders spectrum. This would not only lead to better understanding of the etiopathogenic mechanisms underlying such disorders within the framework of a neurodevelopmental perspective, but also will help to overcome the limitations of current nosographic system that, lacking in comprehensive etiopathological models, often leads to an artificial enhancement of comorbidity.

REFERENCES


