EDITORIAL

Associations of complex post-traumatic stress disorder in refugees and displaced populations

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DESCRIPTION

A complex form of Post-Traumatic Stress Disorder (PTSD) was first described in survivors of World War II concentration camps. It has been well studied in Western populations, including survivors of mass conflict and human rights violations, and survivors of prolonged and repeated interpersonal trauma from which there is no escape under conditions of captivity and violence, with majority much of the literature focused on sexual assault and childhood abuse. It is suggested that those with the emerging consensus is that individuals exhibiting a constellation consistent with Complex Post Traumatic Stress Disorder (CPTSD) carry a higher functional burden than those with are at risk of psychological comorbidities and associated impairment, to a greater degree than those with PTSD, and may not respond to the conventional trauma-focused clinical treatments respond to different treatment. Prior to the introduction of CPTSD in the International Classification of Diseases 11th edition (ICD-11), there were two earlier formulations of a complex form of PTSD in the literature. Disorder of Extreme Stress Not Otherwise Specified (DESNOS) was one such formulation proposed for inclusion in the Diagnostic and Statistical Manual of Mental Disorders 4th edition (DSM-IV) however, field trials did not support the validity and it was ultimately not included but was ultimately excluded due to the lack of validity for the proposed category wise section. The second earlier formulation of a complex form of PTSD is Enduring Personality Change after Catastrophic Experience (EPCACE), which was included in the ICD-10, and described a change in self-organisation following trauma. Clinicians working with survivors of complex trauma (e.g. refugees, childhood abuse survivors) at the time recognised the clinical utility of EPCACE, although, applicable to refugee populations and others, though there was limited research on empirical evidence for this construct in these populations. In addition to the tripartite structure, the DSM-5 revised broadened the definition of PTSD based on factor analytic research, with the addition of a fourth symptom group constellation including-negative alterations in cognitions and mood, symptoms that relate to impairment in regulatory capacities following exposure to traumatic events the addition of three new symptoms, and a dissociative subtype. The limitations of these changes are discussed elsewhere.

ICD-11 PTSD is diagnosed by the presence of at least one symptom from each of three categories: 1) Re-experiencing the traumatic event; 2) Avoidance of internal or external reminders of the event; and 3) Hyperarousal. In addition to meeting all criteria for PTSD, a diagnosis of CPTSD requires at least one symptom from three further categories related to disturbances in self-organisation: 1) Problems in affect regulation; 2) Altered self-concept; and 3) Impaired interpersonal relations. The exposure criteria defining the traumatic event are the same for PTSD and CPTSD in ICD-11.

CONCLUSION

The prevalence of CPTSD in refugees and displaced populations ranged from 3% to 86% in the studies included in our review. Consistent with elsewhere in the literature, the studies in our review supported an association with prolonged, repeated trauma as well as more specific to refugee and displaced populations, post-migration living difficulties. Further research on this construct in this population group, including effective treatments, is required. The review identifies several key evidence gaps. Future research needs to address 1) Development of culturally validated tools to measure CPTSD in refugee and displaced populations; 2) Use of consistent measurement tools for CPTSD, trauma and CMDs in research in this field; 3) Studies with prospective designs; and 4) Studies examining effective treatment of CPTSD in refugees and displaced populations. This study identified programmed cell death 10 (PDCD10) proteins as a target of miR-107. Moreover, in a consequent in vivo assay, tail vein injection of a recombinant lenti virus expressing miR-107 was applied to mice treated with 6-OHDA, resulting in the up-regulation of miR-107 and consequently repressing 6-OHDAeffects. The same delivery mechanism was used by other workers to introduce miR-326 lent viral vectors into mice, and after analysing the brain tissue, it was observed that overexpression of miR-326 was favourable, since it inhibited JNK signaling pathway, leading to an improvement of cognitive function; accumulation of Aβwas reduced andVAV1protein expression was restrained.

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