Psychiatric Comorbidities and Cognitive Dysfunction in Post-Traumatic Stress Disorder.

Liana Brooks*

Division of Cognitive and Behavioral Science, Pacific University for Psychology, California, USA

Introduction

Post-Traumatic Stress Disorder (PTSD) is a debilitating psychiatric condition that arises following exposure to traumatic events. Beyond its hallJunk symptoms—intrusive memories, hyperarousal, avoidance, and negative mood—PTSD frequently co-occurs with other psychiatric disorders and is often associated with cognitive dysfunction. These comorbidities and cognitive impairments not only complicate diagnosis and treatment but also significantly affect the quality of life and daily functioning of individuals with PTSD [1, 2, 3, 4, 5].

Common psychiatric comorbidities in PTSD include depression, anxiety disorders, and substance use disorders. These conditions can mask or amplify PTSD symptoms, creating a complex clinical picture. For instance, depression can exacerbate emotional numbness, while anxiety may heighten hypervigilance. Substance use, often a coping mechanism, can lead to poor treatment adherence and worsened outcomes [6, 7, 8].

Cognitive dysfunction in PTSD typically affects attention, working memory, and executive functioning [9, 10]. Neurobiological studies suggest that changes in the hippocampus, amygdala, and prefrontal cortex contribute to these impairments. Such deficits hinder emotional regulation, learning, and problem-solving, thereby impairing social and occupational functioning.

Conclusion

The interplay between psychiatric comorbidities and cognitive dysfunction in PTSD presents significant clinical challenges. A comprehensive treatment approach that addresses both the psychological and cognitive aspects is crucial for improving patient outcomes. Early intervention, cognitive rehabilitation, and integrated therapy models are vital steps toward holistic recovery in individuals living with PTSD.

References

1. Evans M, Wilkinson M, Giannpolou A. Fast-acting insulin aspart: the rationale for a new mealtime insulin. Diabetes Therapy. 2019;10:1793-800.
2. Giorgino F, Battelino T, Bergenstal RM, et al. The Role of Ultra-Rapid-Acting Insulin Analogs in Diabetes: An Expert Consensus. Journal of Diabetes Science and Technology. 2023:19322968231204584.
3. Hoogwerf BJ, Pantalone KM, Basina M, et al. Results of a 24-week trial of technosphere insulin versus insulin aspart in type 2 diabetes. Endocr Pract. 2021;27(1):38-43.
4. Glastras SJ, Cohen N, Dover T, et al. The clinical role of insulin degludec/insulin aspart in type 2 diabetes: an empirical perspective from experience in Australia. J. Clin. Med.ine. 2020;9(4):1091.
5. Shah VN, Al-Karadsheh A, Barnes C, et al. Pharmacokinetic similarity of switching SAR341402 insulin aspart biosimilar and NovoLog insulin aspart versus continuous use of NovoLog in adults with type 1 diabetes: The GEMELLI X trial. Diabetes Obes. 2023.
6. Sivaprasad S, Sen S, Cunha-Vaz J. Perspectives of diabetic retinopathy—challenges and opportunities. Eye. 2023;37(11):2183-91.
7. Kuo JZ, Wong TY, Rotter JI. Challenges in elucidating the genetics of diabetic retinopathy. JAMA ophthalmology. 2014;132(1):96-107.
8. Stitt AW, Lois N, Medina RJ, et al. Advances in our understanding of diabetic retinopathy. Clinical science. 2013;125(1):1-7.
9. Duh EJ, Sun JK, Stitt AW. Diabetic retinopathy: current understanding, mechanisms, and treatment strategies. JCI insight. 2017;2(14).
10. Stitt AW, Curtis TM, Chen M, et al. The progress in understanding and treatment of diabetic retinopathy. Prog Retin Eye Res. 2016;51:156-86.

***Correspondence to:** Liana Brooks *, Division of Cognitive and Behavioral Science, Pacific University for Psychology, California, USA. Email: l.brooks@mindscience.org

***Received:** 27-May-2025, Manuscript No. AACPCP-25-167449; **Editor assigned:** 01-Jun-2025, PreQC No. AACPCP-25-167449 (PQ); **Reviewed:** 15- Jun-2025, QC No. AACPCP-25-167449; **Revised:** 22- Jun-2025, Manuscript No. AACPCP-25-167449 (R); **Published:** 29- Jun-2025, DOI:10.35841/AACPCP-9.2.219*

***Citation:** Brooks L. Psychiatric Comorbidities and Cognitive Dysfunction in Post-Traumatic Stress Disorder. Cogn Psychol. 2025; 9(2):219*