radiofrequency ablation as an adjunct to standard multimodal care in military patients with severe PTSD.
Methods: From May to July 2025, 61 male military patients (mean age 42.4±9.8 years) with clinically confirmed PTSD (Mississippi Scale score >100) were enrolled. Exclusion criteria included structural brain lesions on CT/MRI. Twenty-four patients consented to interventional treatment (SGB and/or RFA) in addition to standard therapy (psychotherapy, supportive physiotherapy, and nutrition). Thirty-seven patients served as controls, receiving only standard treatment. Procedures were performed under fluoroscopic guidance with intraoperative stimulation for accurate localization. Clinical outcomes were assessed using the Mississippi PTSD Scale and quantitative EEG (dominant alpha/beta rhythm ratio) before intervention and at day 14. Safety outcomes included intra- and postoperative complications.
Results: In the intervention group, PTSD severity decreased by 41 points on the Mississippi Scale compared to 22 points in controls ($p<0.001$). EEG analysis revealed a 41% restoration of alpha rhythm in the intervention group versus 9% in controls ($p<0.001$), with a parallel reduction in beta rhythm (−38% vs −13%). Vegetative symptoms (hyperhidrosis, blood pressure fluctuations) markedly improved in the intervention group by days 14–21. Clinical response rate to SGB/RFA reached 94.6%. The most frequent expected side effect was transient Horner's syndrome; one case of self-limited generalized seizure (4.1%) was reported.
Conclusions: Stellate ganglion block and radiofrequency ablation represent effective and relatively safe adjunctive interventions for severe PTSD in military patients. These techniques offer rapid symptom reduction (1–2 days), sustained benefit (up to 3 weeks), and objective neurophysiological improvement, making them valuable options in multidisciplinary hospital settings. Further large-scale, controlled studies are warranted to confirm long-term efficacy and broaden applications to psychosomatic disorders.

Disclosure of Interest: None Declared

EPV1189

The Effects of Post-Traumatic Stress Disorder on Memory Function

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Introduction: Post-traumatic stress disorder (PTSD) is a mental health condition that develops after exposure to a highly traumatic event. It is characterized by re-experiencing, avoidance, hyperarousal, and negative alterations in mood. Emerging evidence indicates that these symptoms frequently co-occur with memory dysfunction. A core feature of PTSD is the duality of memory disturbances: traumatic amnesia, the inability to recall all or part of a traumatic event, and emotional hypermnesia, the vivid, intrusive recall of trauma.
Objectives: This study aims to explore the relationship between PTSD and memory dysfunction.
Methods: A literature review was conducted to examine the relationship between PTSD and memory dysfunction. PubMed was searched using the terms "posttraumatic stress disorder" and "memory." Articles were selected based on relevance to PTSD and memory outcomes. Key findings regarding the interaction between trauma exposure, memory disturbances, and clinical implications were summarized.
Results: Memory disturbances, including intrusive memories and dissociative amnesia, are central to the clinical presentation of PTSD. While PTSD primarily affects trauma-related memories, individuals may also struggle to recall everyday events, segment ongoing activities, or learn and remember neutral information. Memory alterations can manifest as reduced recall (traumatic amnesia) or exaggerated recall (hypermnesia), including vivid flashbacks and nightmares. Individuals with PTSD often have difficulty retrieving specific autobiographical memories, particularly positive ones, which may contribute to symptom severity. Deficits in episodic and contextual memory, along with failure of extinction learning, underscore the hippocampus's key role in PTSD. Individuals with PTSD often exhibit bilateral hippocampal atrophy, reflected in reduced hippocampal volume on neuroimaging. Trauma-related hippocampal reduction may result from the hippocampus's high sensitivity to stress hormones, particularly glucocorticoids and adrenal steroids. Beyond the hippocampus, the amygdala is another subcortical structure critically involved in PTSD, especially in mediating intrusive and hyperarousal symptoms. The amygdala plays a central role in fear conditioning and the encoding of fear memories. Although structural changes are uncommon, neuroimaging studies consistently demonstrate amygdala hyperactivation in response to trauma-related or negative emotional stimuli. This heightened activation may underlie the hallmark symptoms of re-experiencing and hypervigilance observed in PTSD. Finally, functional imaging studies reveal disrupted connectivity among the hippocampus, amygdala, and prefrontal cortex, contributing to impaired contextual processing, emotional dysregulation, and the persistence of maladaptive fear memories.
Conclusions: Understanding the mechanisms that cause memory dysfunction in PTSD may guide targeted therapeutic interventions.

Disclosure of Interest: None Declared

EPV1190

Bridging Mind and Heart: Inflammatory Signatures in Post-AMI PTSD Patients

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Introduction: Post-traumatic stress disorder (PTSD) develops in approximately 10–20% of patients following acute myocardial infarction (AMI). Emerging evidence suggests that PTSD may drive persistent inflammatory activation —marked by elevated C-reactive protein (CRP), interleukin-6 (IL-6), interleukin-1β (IL-1β), and matrix metalloproteinase-9 (MMP-9) potentially increasing the risk of recurrent ischemic events and worsen prognosis.
Objectives: To synthesize recent evidence on associations between PTSD and inflammatory biomarkers among AMI survivors, and to examine whether inflammatory dysregulation may contribute to recurrent cardiac events or adverse outcomes.

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