Posttraumatic stress disorder clinical features

Memory impairment and mood disorders are among the most troubling sequelae following traumatic brain injury (TBI). The relationships between comorbid psychiatric disorders and memory function have not been well illustrated.

More depressive symptoms rather than anxiety symptoms and less years of education are significant predictors for posttraumatic memory dysfunction [1].

PTSD may be related to impulsivity, particularly attentional impulsivity, even after controlling anxiety and depression among inpatients with alcohol use disorder (AUD). [2].

Psychiatric symptoms typically emerge in a tardive fashion post-TBI, with negative effects on recovery. Patients with PTSD, as well as rodent models of PTSD, demonstrate structural and functional changes in brain regions mediating fear learning, including prefrontal cortex (PFC), amygdala (AMYG), and hippocampus (HC). These changes may reflect loss of top-down control by which PFC normally exhibits inhibitory influence over AMYG reactivity to fearful stimuli, with HC contribution. Considering the susceptibility of these regions to injury, Schneider et al., examined fear conditioning (FC) in the delayed post-injury period, using a mouse model of mTBI. Mice with mTBI displayed enhanced acquisition and delayed extinction of FC. Using Proton magnetic resonance spectroscopic imaging ex vivo, they examined PFC, AMYG, and HC levels of gamma-aminobutyric acid (GABA) and glutamate as surrogate measures of inhibitory and excitatory neurotransmission, respectively. Eight days post-injury, GABA was increased in PFC, with no significant changes in AMYG. In animals receiving FC and mTBI, glutamate trended toward an increase and the GABA/glutamate ratio decreased in ventral HC at 25 days post-injury, whereas GABA decreased and GABA/glutamate decreased in dorsal HC. These neurochemical changes are consistent with early TBI-induced PFC hypoactivation facilitating the fear learning circuit and exacerbating behavioral fear responses. The latent emergence of overall increased excitatory tone in the HC, despite distinct plasticity in dorsal and ventral HC fields, may be associated with disordered memory function, manifested as incomplete extinction and enhanced FC recall [3].

1)
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2)
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