

Research Progress of Cognitive Impairment in Post-Traumatic Stress Disorder

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Abstract

Post-traumatic stress disorder (PTSD) is a kind of serious mental disorder that occurs after severe traumatic events. It is characterized by severe emotional and memory damage. This paper reviews the relevant research literature on PTSD at home and abroad in recent years, and reviews the mechanism of cognitive impairment in mental trauma to reveal the functional mechanism of cognitive impairment in post-traumatic stress disorder, in order to provide reference for future research.

Keywords

PTSD, Cognitive Function, Mechanism, Review

1. Introduction

In recent years, with the frequent occurrence of natural and man-made disasters, a series of malignant events have not only caused great loss of life and property, but also posed a major threat to people's physical and mental health. Among them, post-traumatic stress disorder is one of the serious consequences of these injuries. Post-Traumatic Stress Disorder (PTSD) is a disorder in which a person who has directly experienced a traumatic event develops a characteristic set of symptoms (intrusion/re-experiencing the trauma; avoidance; negative cognitions and mood; hyper-arousal) [1]. A survey on the incidence of dementia in a large number of veterans in the United States showed that the risk of dementia in veterans with PTSD was about twice than in veterans without PTSD [2] [3] [4]. It is not clear whether there are common risk factors for PTSD and dementia, or whether PTSD itself is an independent risk factor for dementia. However, the current research on PTSD is mainly based on clinical observation, while the research on the mechanism of PTSD is still not in-depth enough. The author

summarizes the mechanism of brain function of PTSD by combining and summarizing the research literature on the mechanism of PTSD in recent years, in order to provide reference for future related research.

2. Abnormal Changes in Mood

Brain is not only the highest organ regulating stress, but also an important target organ of stress. At present, it is generally believed that the amygdala plays an important role in emotion and emotion regulation, and the enhancement of its activity may cause excessive fear response or impaired fear memory in patients [5]. Brunetti *et al.* [6] used fMRI technology to find that the activation process of the amygdala in adult PTSD patients was abnormally increased under neutral or negative stimuli. However, Liu Mengqi *et al.* [7] firstly studied the neurobiochemical changes of bilateral amygdala in children and adolescents with PTSD by using MRS technique, and preliminarily discussed its possible role in the pathogenesis of PTSD. The study found that the left amygdala creatine (Cr) concentration was significantly higher than that of the right amygdala in the healthy control group, suggesting that the distribution of Cr concentration in the normal children and adolescents was asymmetrical. There was no significant difference in the concentration of Cr in the left amygdala of the PTSD group compared with the healthy control group. Cr is a buffer of high-energy phosphate in energy metabolism, and previous studies have shown that the concentration of Cr will decrease in the high metabolic state, and, on the contrary, it will increase in the low metabolic state [8]. This study suggested that the asymmetry of Cr concentration distribution in the amygdala of PTSD patients was destroyed, and the left amygdala was hypermetabolic. Therefore, it is speculated that the activation of the amygdala (especially the left amygdala) is abnormally increased under the stimulation of traumatic events, resulting in high metabolism, thus breaking the asymmetry of the distribution of Cr concentration in the amygdala, which may be one of the mechanisms of the recurrence of traumatic experience and the increase of persistent alertness in PTSD patients. Karl *et al.* [9] found in their Meta-analysis that the bilateral amygdala in PTSD patients was significantly reduced, and the left amygdala volume was significantly reduced in PTSD patients compared with normal healthy people, which proved on the other hand that the abnormal changes in the structure and function of the amygdala (especially the left amygdala) were closely related to the occurrence of PTSD. Rauch *et al.* [10] found that the amygdala cerebral blood flow in patients with PTSD increased in the fearful imagination environment, and decreased in the amygdala cerebral blood flow after a period of CBT. It is suggested that the increased amygdala activity is an important pathological basis of PTSD. At the same time, Rauch *et al.* [10] also found that the blood flow in the bilateral insula of PTSD patients increased, and the insula has extensive fibrous connections with many brain regions, such as the frontal lobe, temporal lobe, amygdala, and hippocampus, which is an important node in the emotional processing and au-

tonomic regulation network. Therefore, it is speculated that the activation of the insula may also be closely related to the occurrence of PTSD. Many domestic and foreign studies [11] [12] [13] [14] [15] believe that the consistency of brain precuneus in patients with PTSD is abnormally activated, suggesting that the local neuronal activity of precuneus in patients with resting state is enhanced in synchronization, indicating hyperfunction. Studies have shown that the precuneus is an important part for processing visual information [16] and is involved in extracting emotional memories [17]. It is speculated that the enhancement of precuneus function may be one of the important mechanisms of recurrent traumatic experience in PTSD patients.

3. Abnormal Changes in Memory Function

PTSD is often associated with memory impairment. Studies have shown that both the frontal and temporal lobes of the brain are directly involved in the regulation of memory. The frontal lobe is responsible for the integration of all senses and perception, the frontal cortex is involved in memory formation, and the temporal cortex is involved in memory formation and retrieval [18]. Gong Li *et al.* [19] used MRI technology to investigate the correlation between PTSD memory impairment and brain structure, and found that the scores of understanding of memory, delayed understanding memory, visual regeneration and delayed visual regeneration in PTSD patients were significantly lower than those in normal healthy people, suggesting that PTSD patients had different degrees of memory impairment. MRI results showed that the volume of gray matter in the frontal and temporal lobes of the patients with PTSD was significantly smaller than that of normal healthy people. It is preliminarily suggested that the memory impairment of PTSD is closely related to the structural changes of the frontal and temporal lobes. When Jin Kuixing *et al.* [20] studied the correlation between executive function of early PTSD and frontal lobe damage, they found that PTSD patients had obvious executive dysfunction and the gray matter volume of part of the frontal lobe decreased, which was similar to the conclusion that frontal lobe damage might be the pathological basis of cognitive function damage. The reason may be that the frontal lobe damage weakens the inhibitory effect on the amygdala, thus enhancing its own activity, affecting the normal memory processing and ultimately affecting the encoding, recognition and recognition of declarative memory. Director of learning and memory function in temporal lobe area as the hippocampus, also have scholars launched a related study, Min Guo, *et al.* [21]. The MRI hippocampal volume of 50 patients with acute PTSD was compared with that of 50 healthy volunteers, Normal hippocampal volume was correlated with age and sex. This study used the difference method to measure the hippocampal volume of correction, and matched controls in terms of age, gender, as far as possible to avoid the error caused by objective factors. Then they found that people with PTSD hippocampal volume compared with healthy controls were changed, but to different degree, patients with mild-to-moderate

hippocampal volume shrink are not obvious. The difference was not statistically significant, but the volume reduction in severe patients was significantly different [22]. It is suggested that the occurrence of PTSD is related to the damage of hippocampus, and it is speculated that the degree of hippocampus volume reduction may be related to the severity of PTSD patients. Some scholars also believe that the hippocampus damage in PTSD patients may be one of the reasons for the persistence of PTSD. Traumatic memories, once formed, are difficult to eliminate, so that people with PTSD often a persistent fear, and fear of eliminating mainly through the prefrontal cortex (mPFC) fear of amygdala response inhibition, assist the mPFC adjust the function of the amygdala, hippocampus, once the hippocampus is damaged, mPFC of amygdala inhibitory effect will be weakened [23].

4. Influence of Stress Trauma on Dementia

Clement *et al.* [24] showed that negative life events may be one of the risk factors for the onset of Alzheimer's disease (AD), and there is a certain correlation between early mental trauma and the onset of AD. Qureshi *et al.* [25] found that the incidence of dementia in veterans with PTSD was significantly higher than that in non-PTSD veterans. Tsolaki *et al.* [26] confirmed that over 70% of AD patients had experienced at least one severe traumatic event prior to the diagnosis of AD, versus only 55% of the control subjects. A case-control study by Shen Yuyou *et al.* [27] showed that AD was associated with the occurrence of major life events (spouse death, divorce, and child death). In a study of 93 patients with PTSD who survived the Holocaust in Germany, 13 patients developed dementia, of which the incidence of vascular dementia was 66%, the incidence of AD was 23%, and other dementia subtypes were 11% [28]. PTSD has not been found to be associated with a specific subtype of dementia, being associated with almost all types of dementia, especially vascular dementia and AD. Although the damage of cognitive function in people with PTSD that has been a large number of studies confirm that but at the moment about PTSD and the forward of the correlation between cognitive decline is lack of system research. PTSD patients early cognitive function change whether can be as early signs of dementia or as an independent risk factor for dementia remains to be further research.

5. The Effect of Stress Trauma on Cognition

Traumatic events often lead to significant physiological and psychological responses of individuals, increase the risk of mental and psychosomatic diseases [29], and may also cause cognitive decline [30]. Peavy *et al.* [31] found that the more traumatic events experienced, the higher the risk of cognitive impairment. Traumatic events can have long-lasting effects on the human brain that can be delayed decades or even a lifetime after the stress is experienced. Previous research data have shown that both acute trauma and long-term chronic stress may affect individual learning and memory functions, and most of the results

are from animal experiments and observational studies [32]. Aggarwal *et al.* [33] conducted a 7-year follow-up study involving 6200 adults and found that the more stressful events experienced, the greater the impact on cognitive function and the faster the rate of cognitive decline. People who suffer from PTSD after trauma in adulthood are also at increased risk of cognitive impairment [34]. It is concluded that severe traumatic events in adulthood are one of the risk factors for long-term cognitive decline.

6. Summarize

1) Major mental trauma is the main cause of memory impairment in PTSD patients, and the more severe the trauma symptoms are, the more severe the memory impairment is.

2) The frontal abstract thinking and memory ability of PTSD patients are severely impaired, and the patients generally have executive dysfunction, which has no correlation with the clinical symptoms of PTSD, suggesting that the executive dysfunction of PTSD patients is not affected by other factors and is one of the independent neurological symptoms.

3) Cognitive decline in PTSD patients may be one of the important reasons for the impairment of memory function, and cognitive decline is also the result of cognitive impairment caused by primary symptoms of PTSD.

4) PTSD patients have significant memory impairment, including visual regeneration, comprehension memory, delayed memory and visual regeneration disorders, and their memory impairment is more serious than that of patients with depression and anxiety.

7. Conclusion

Despite the rapid development of science and technology and economy, people are still suffering from a variety of natural and man-made disasters and continuous wars. Focusing on the severe international situation, the research on PTSD has attracted much attention both at home and abroad. Researchers have explored through the neuroendocrine, molecular biology, behavioral and other aspects. The continuous development of imaging technologies, functional imaging technologies, including MRI/fMRI, PET/ PET-CT and EGG, have provided strong support for us to explore the brain functional mechanism of PTSD. The relationship between PTSD and the impairment of long-term cognitive function still needs to be further systematically studied. Addressing mental factors and giving psychological intervention in advance, cognitive dysfunction can be controlled in the earliest stage of disease development, in order to reduce various mental disorders in the long term.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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