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Review Paper

Post Traumatic Stress Disorder: Narrative Review

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ABSTRACT

PTSD is an anxiety disorder that occurs after exposure to traumatic stimuli. It is characterized by repetitive memories, mood swings heightened sensitivity and avoidance of stimuli. Exposure to a traumatic events is a necessary condition for diagnosing PTSD and such traumatic events increase the chance of developing PTSD within the first 3 months. Covid - 19 epidemics had an impact on mental health. Risk factors include early life traumatic experiences or history of depression in family. The latest version of the dsm-5 includes updated methods for diagnosing PTSD that have been developed over many years. Non-pharmacological treatment can improve the well-being of more than half of PTSD patients through cognitive-behavioural therapy and eye movement sensitization reprocessing. The primary pharmacological treatment for PTSD is through the use of selective serotonin reuptake inhibitors. Without treatment, PTSD can result in more symptoms, longer recovery periods, physical, emotional and relational issues as well as increased substance abuse. PTSD can be prevented and managed through treatment and patient care.

Keywords: *Post traumatic stress disorder, exposure to traumatic event, anxiety disorder*

Post traumatic disorder (PTSD) is an anxiety disorder that occurs after exposure to a traumatic event. PTSD is a severe condition that can arise after experiencing or being exposed to traumatic event [1] Symptoms include recurrent memories, unpleasant mood swings, increased arousal and sensitivity, and avoidance of stimuli, all of which can lead to serious mental health problems, long term disability and socioeconomic burden[2]. People with PTSD report high levels of dissatisfaction in many areas of their lives, including physical health, social and work functioning[3].

Posttraumatic stress disorder is a common and often debilitating psychiatric illness with significant functional impairment in a variety of domains.[4] PTSD produces extreme distress or impairment in psychosocial functioning and is characterized by four primary categories of symptoms that continue for at least a month, which are respectively the criteria B, C, D, and E for the disease. (i) Intrusions linked with the traumatic event, e.g., involuntary distressing event-related memories, and flashbacks, or physiological reactions to cues that resemble the traumatic event; (ii) avoidance of event-related stimuli, i.e., internal (memories, thoughts,

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and feelings) or external (people, places, situations) (iii) negative alterations in event-related cognitions and/or mood, e.g. forgetfulness (iv) Enhanced excitability and reactivity towards situations such as disturbed sleep patterns or suicidal behaviour[5].

Exposure to a traumatic event is a necessary condition for diagnosing PTSD, and such traumatic events increase the chance of developing PTSD within the first three months. Although traumatic events are a significant risk factor for PTSD, not all victims develops it. Psychiatrists have long recognized the existence of PTSD symptoms. Ancient texts documented condition PTSD as abnormal mental symptoms and post battle activities. However, it wasn't until 1980. the Diagnostic and Statistical Manual of Mental Disorders, Third Edition (DSM-III) recognized PTSD as a psychiatric condition. The techniques to identify PTSD have been developed over decades and are updated in the new version of the DSM-5. In the DSM-5, PTSD is now classified as "Trauma- and Stressor-Related Disorders." Due to the high comorbidity between PTSD and DSM diagnosed mental disorder, the world health organization's 11th edition (ICD-11) is often used. Although the symptom group differ in many ways, both diagnostic categories are extensive, describe the indications of PTSD. The psychological and emotional responses to TEs are determined by the qualities of both the event and the characteristics of the individual. It is determined that more than 75% people will experience TE at least once in their lifetime, and about 10% will develop PTSD. Worldwide, the lifetime prevalence of PTSD is 1.3% to 12.5%, with a year prevalence of 0.2% to 3.7%. The current coronavirus pandemic (COVID-19) can also be considered a TE that can affect physical and mental wellbeing. The widespread presence of PTSD symptoms is quite high, 7-53.7%. The clinical presentation of PTSD varies and there is considerable overlap in symptoms between PTSD and other psychiatric diseases such as depression, anxiety, and substance abuse. Nearly all patients with PTSD encounter the criteria for other mental illnesses. Many patients suffer from PTSD due to their fear-triggered emotional responses and long term negative experiences that have an impact on their lives and families. PTSD is also linked with a number of medical conditions including chronic pain, a group of metabolic irregularities that are adverse effects for heart disease and an elevated risk of dementia.

Scientists have made significant success in understanding the brain circuits, chemical mechanisms, and genetic pathways underlying the PTSD. Even as awareness of PTSD in past 10 years, the resulting information has not yet improved treatment outcomes for PTSD patients. The biggest cause for this is lack of foresight.

PTSD is not only related to major natural disasters or wars. Traumatic life events can also contribute to stress disorders to varying degrees. In past 20 years, major disasters have increased the number of people with PTSD worldwide. Mental illness has seriously affected people's health and lifestyle. Governments and scientists are increasingly focusing on the management of PTSD.[10]

ETIOLOGY

Although the cause of PTSD is unknown, most researchers believe that a personal proness is necessary for symptoms of a traumatic experience to appear. A small proportion of people develop clinically significant symptoms due to stressful events. Those who are more prone to PTSD have a history of depression or anxiety disorders, and a family history of anxiety and neurotic behaviour. From a biological perspective, what differentiates PTSD from a mere fear response is the body's inability to return to its pre-traumatic state. In a normal startle response, the first sympathetic current triggers the "fight-or-flight" reaction. Increases

in catecholamines and cortisol are proportional to the degree of the stressor. Corticotropin-releasing factor stimulates cortisol release via the hypothalamic-pituitary-adrenal (HPA) axis, which functions in a negative feedback loop to decrease sympathetic activity and generate more cortisol release.

Ambient cortisol levels in PTSD patients are lower than usual; this situation has been responsible to severe "adrenal exhaustion" caused by prolonged severe anxiety suppresses the HPA axis. However, current findings emphasize that cortisol levels soon after a motor vehicles accident were much lower in those who developed PTSD. The HPA axis and the sympathetic nervous system may be disconnected in patients with PTSD leading to uncontrolled release of catecholamines, which can impact memory formation during trauma and potentially worsen symptoms when exposed to traumatic triggers.[1]

EPIDEMIOLOGY

According to epidemiological research, the lifelong existence of PTSD is 13.0-20.5% for women and 6.3-8.3% for men. According to the World Mental Health Surveys, high-income countries (Northern Ireland: 3.5%, US: 2.7%, New Zealand: 2 %) had higher one year prevalence rates than low- and middle-income countries (Colombia: 0.5%, Mexico: 0.5%). Research suggests that some aspects of a traumatic incident may elevate the chances of developing PTSD. Natural disasters are associated with a significantly lower rate of PTSD (6-11%) compared to sexual assault (>42%). Overall, interpersonal violence is associated with higher rates of PTSD. According to the World Mental Health Surveys, experiencing organized, physical, or sexual violence increases the chance of developing PTSD. After analysing all the methodological considerations, self- reported torture remains the largest predictor of PTSD after cumulative exposure to a potentially traumatic experience. Studies on individual countries show that certain ethnic groups, such as Hispanics and African Americans in the US, have a higher prevalence of PTSD. This approach is more methodologically sound because it takes into account the cofounding factors of the context. It has been established through military experiments that Hispanics are more prone to developing PTSD. These differences are due to unequal access to health care resources, ethnic bias, or socioeconomic conditions, making the interpretation questionable. Epidemiological studies shows that many patients who suffers PTSD also have other comorbidities, such as depression, anxiety, and substance use disorders. These elevated rates of comorbidity could be explained by psychiatric diseases predisposing individuals to traumatic events. Exposure to traumatic situations increases the likelihood of comorbidity.[6]

PATHOPHYSIOLOGY

The amygdala is the centre of the central nervous system and the fear response. This brain structure controls our ability to feel fear and learn to avoid pain by intervening between emotion and attention in two ways. Amygdala signals enhance processing of fear-inducing information by the higher structure of the cerebral cortex. i.e. increases the emotional valence associated with information and memory in the brain, which facilitates their use in the future. In addition , the amygdala can quickly recognize danger signals through basic visual pathways that bypass the central cortex and neocortex . The amygdala examines objects and organisms in its environment before interacting with them. This brain structure has the ability to rapidly activate almost every, body system, causing it to fight fiercely or retreat quickly. In addition to alerting the body, the amygdala stimulates the neighbouring hippocampus, allowing the brain to learn and create new memories associated to danger. The clinical pathway of PTSD involves important factors such as proximity and fear, followed by re-experience, avoid/sensitization, and hypersensitivity/over-vigilance. [7]

NEUROENDOCRINOLOGY

The brain's response to trauma can trigger an overactive adrenaline response, leading to the development of PTSD symptoms. This can cause significant changes in neurological activity. These patterns may endure for many years, making the individual susceptible to unpleasant occurrences in the future. .In traumatic situations, significant amounts of stress hormones are released, which weaken hypothalamus function and can result in PTSD . It causes physiological changes in the entire body that are different from other psychiatric illnesses, such as depression. Individuals with PTSD react more to the dexamethasone inhibition test than people with major depression. Most patients who suffers with PTSD have low cortisol secretion and high urinary catecholamine production, resulting in a higher norepinephrine/cortisol ratio than similar undiagnosed individuals. This is in contrary to the typical fight-or-flight response, where both catecholamine and cortisol increase after being exposed to a stressor. Levels of catecholamines in the brain are high, as are levels of corticotropin-releasing factor. These findings reflect an imbalance of the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis, the locus coeruleus-noradrenergic systems, and the limbic-frontal brain connections are all involved in fear maintenance. The HPA axis, which coordinates the hormonal response to stress and activates the LC-noradrenergic system, is linked to the excessive consolidation of memories following trauma. The amygdala is responsible for detecting and reacting to threats with conditioned and unconditioned fear responses. The HPA axis regulates the hormonal response to stress. Because dexamethasone significantly reduces cortisol in PTSD, HPA axis abnormalities are most likely due to strong negative feedback inhibition of cortisol, likely due to increased glucocorticoid receptor sensitivity. PTSD is thought to be a maladaptive learning pathway for fear response via a hypersensitive, hyperreactive, and hyperresponsive HPA axis. Low cortisol levels can contribute to PTSD. Swedish soldiers who fought in Bosnia and Herzegovina who had low pre-service salivary cortisol levels were more likely to experience PTSD symptoms after war stress than soldiers with normal levels. Because cortisol is generally vital in restoring homeostasis after a stress response, trauma survivors with low cortisol levels would be expected to have poorly regulated, i.e. longer and more disruptive response that would underlie PTSD. The locus coeruleus-noradrenergic system is hypothesized to mediate excessive consolidation of fear memory. High cortisol levels suppress norepinephrine activity, and PTSD patients have lower cortisol levels, it has been said that they are unable to control the increased noradrenergic response to severe stress. Intrusive memories and fear responses are thought to result from a response to associated stimuli. Neuropeptide Y (NPY) has been proven to inhibit norepinephrine release and have anxiolytic benefits in animal models. Research shows that PTSD patients have lower levels of NPY, which indicate increased anxiety. Other research suggests that persons with PTSD have severely low serotonin levels which often contributes to behavioural symptoms such anxiety, ruminations, irritability, Outrage, violence and suicidal thoughts. Serotonin also helps to stabilize the synthesis of glucocorticoids. Dopamine levels in PTSD patient can reduce and cause anhedonia, apathy, attention deficits , and motor deficits, while excessive levels can cause psychosis, agitation, and restlessness. Several studies have found higher levels of the thyroid hormone (triiodothyronine) in patients with PTSD. This type II form of allostatic adaptation can aid in enhancing susceptibility to adrenaline and other hormones related to stress. Chronic stress can also contribute to hypersensitivity of the norepinephrine system. Overloading of norepinephrine receptors in the frontal cortex has been linked to the negative memories and flashbacks that many people with PTSD suffer from. Impairment of other norepinephrine functions hinder the brain and memory systems from processing the situation through proper channels and the emotions an individual experiences during a episode of bad memories are unrelated to the present environment. The neuroscience of

PTSD is a debated topic in medicine. A 2012 analysis found no clear association between hydrocortisone levels and PTSD. According to the majority of research, patients with PTSD have increased corticotropin-releasing hormone levels, decreased hydrocortisone levels, and dexamethasone enhances negative risk feedback of the HPA axis.

NEUROANATOMY

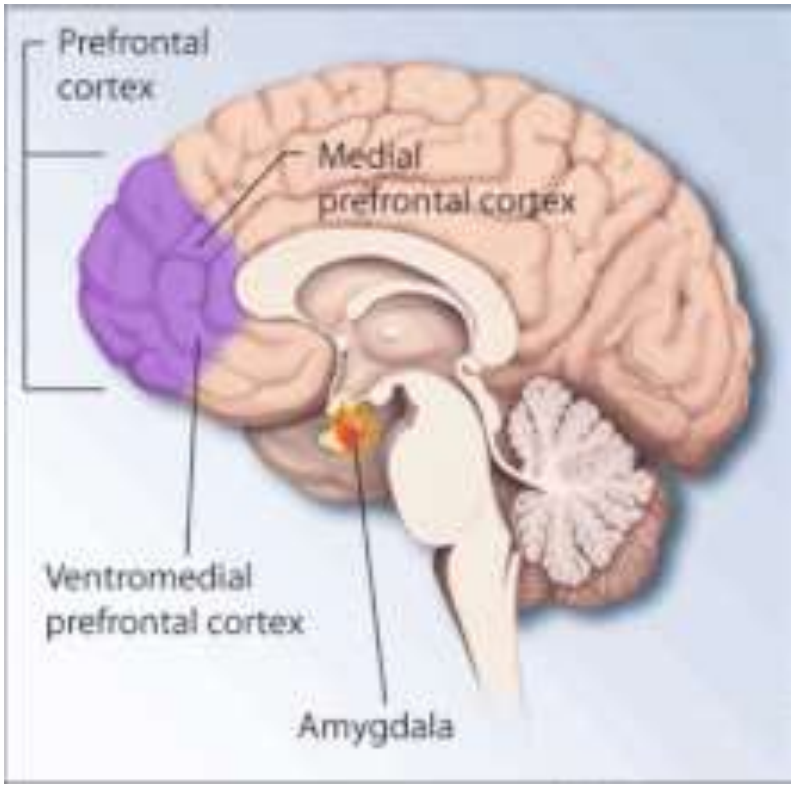
A systematic review has showed that studies found a link between brain, intracranial volume, and hippocampus, insular cortex, and anterior cingulate. Most of the previous studies focuses on PTSD in Vietnam War experts. Individuals with PTSD exhibit reduced activity in the areas responsible for emotion regulation in brain, including the dorsal and rostral anterior cingulate cortex and the ventromedial prefrontal cortex. The amygdala plays a important role in the formation of emotional memories, especially memories related to fear . In severe stress, the hippocampus, which is responsible for keeping memories in the proper framework of place and time and retrieving memories, is inhibited. one idea is that this repression may be the source of the flashbacks experienced by patients with PTSD. When a person suffering from PTSD is exposed to stimuli comparable to the TE, the body interprets the event as recurring since the patient is unable to remember.

The amygdalocentric hypothesis of PTSD posits that the medial prefrontal cortex and hippocampus, specifically stimulate and control the amygdala for extinction. This is steady with a view this is a disruptive disorder.

The amygdala's basolateral nucleus (BLA) is critical for comparing conditioned and unconditioned responses to stimuli and for developing correlations that lead to the fear conditioning seen in PTSD. The BLA triggers the amygdala's central nucleus (CeA), which develop response for fear (including the behavioural reaction to threat and the increased startle response). The prefrontal cortex afferents play essentially the role of inhibitory inputs in controlling BLA-to-CeA transmission and are thought to contribute to the inhibition of conditioned response of anxiety.

Functional neuroimaging studies in PTSD reveal significant heterogeneity, with amygdala activation being more prevalent than in anxiety disorders or phobic disorders. When comparing the dorsal (approximately the CeA) and ventral (roughly the BLA) clusters, hypersensitivity is more strong in the ventral cluster, whereas hypoactivity is visible in the dorsal cluster. These differences may explain both the dulled emotions in PTSD (due to CeA sensitivity) and the fear-related component of PTSD.

Evidence suggests that in PTSD, endogenous cannabinoid levels, notably anandamide, are lowered, and CB1 are raised to compensate. Unusual fear response and hypersensitivity in trauma survivors are believed to be correlated with an increase in CB1 receptor accessibility in the amygdala, but not restlessness in trauma survivors .[8]



Brain regions affected by stress and post-traumatic stress disorder. [8]

RISK FACTORS

Most of the risk factors are common to several psychiatric diseases, including female gender, low socio-demographic background, previous mental disorder, history of mental disorders in family, and traumatic childhoods. In terms of PTSD-specific vulnerability factors, this disorder develop following chronic trauma or personal traumatic situations. Subjective reactions to trauma, including immediate dissociation reactions and catastrophic evaluations of the event's end, are substantially related with subsequent PTSD severity. The post-trauma environment is crucial, as limited social support and continuing stressors increase the chances of PTSD.

Risk factors include:

- early life traumatic experiences
- Depression history in family
- physical or sexual abuse
- substance abuse
- Depression, anxiety, or similar mental disorder.
- Stressful lifestyle
- Lack of assistance following the trauma.[8]

Comorbidities

PTSD can be linked with other anxiety disorders and depression of varying intensity, making diagnosis difficult. Substance abuse is a typical comorbidity that develops with PTSD. Temporal discordance is the strongest indicator of comorbidity. In other words, depression and/or anxiety disorders before a traumatic incident that leads to PTSD, along with substance addiction following the traumatic event, provide the most compelling evidence for multiple psychiatric diseases.

Medical disorders are frequently associated with PTSD, particularly among older individuals. Exposure to trauma raises the likelihood of poor physical health. Bos carino found a direct association between trauma and many other medical disorders in a 20-year study of males who had been subjected to extreme stress. David et al compared male soldiers treated in a PTSD rehabilitation unit or alcohol dependency and found that PTSD patients tend to develop diabetes, heart disease, obesity, and osteoarthritis than drug abusers.[7]

DIFFERENTIAL DIAGNOSIS

In 1980, the DSM's third edition classified 17 PTSD symptoms into three categories. This diagnosis has been modified and improved several times over a period of many investigations. The DSM-5's most recent version categorizes the twenty symptoms of PTSD into four clusters: Activated avoidance, intrusion into the mind, negative thoughts. and mood changes, and noticeable arousal reactivity alterations. To summarize, the diagnostic criteria can be stated as follows: the individual must have been exposed to a stressor leading to at least one intrusion symptom, one avoidance symptom, two negative changes in thinking and mood symptoms, and two dysfunctional arousal and reactive turbulence symptoms lasting for a month. Interestingly, PTSD was shifted from the area of anxiety disorders to a recent class called "trauma and stress-related disorders" in the DSM-5, which shows diversity in PTSD and awareness. In the most new version of ICD-11, the WHO and ICD adopted a new method of diagnosing PTSD by summarizing symptoms into six categories. Several weeks after a major stressor exposure, at least one symptom of each cluster must occur. In order to distinguish PTSD from other conditions that have similar symptoms, such as adjustment disorder, anxiety disorder, obsessive-compulsive disorder, and personality disorder, both diagnostic criteria emphasize the duration and exposure to trauma. Individuals diagnosed with major depressive disorder (MDD) may or may not have experienced traumatic events, but they have no signs of persistent disturbing thoughts or other symptoms linked to post-traumatic stress disorder (PTSD). Long-term confusion and disorientation are more distinct indicators of traumatic brain injury (TBI). Based on the PTSD diagnosis, the ICD-11 further recognizes a sister condition called complex PTSD, which is marked by disruptions, a low self-concept, and relationship problems. Disturbances in self-organization (DSO) accompanied by PTSD are the primary signs of CPTSD. Brewin et al. conducted a study to compare the DSM-5 and ICD-11 criteria requirements, prevalence, comorbidities, and validity for PTSD in order to assess the diagnostic's practical applicability. It appears that the presence of PTSD in adult participants using the ICD-11 is much lower than the DSM-5 because, according to their analysis, the DSM sets less strict avoidance criteria than the ICD-11, which sets stricter diagnostic requirements for recurrent symptoms. The data showed a significant difference in the patients' classifications between the DSM-5 and ICD-11, with just a small percentage of instances overlapping. This reveals that different diagnostic systems appear to identify different cases. Studies comparing comorbidity and PTSD in children show a range of outcomes and equivalent severity and quality of life. Young children (age $\leq$ 5 years) are diagnosed based on their physical and psychological development, as per DSM-5 guidelines. Despite numerous studies as well as modifications to the diagnostic criteria for PTSD, it remains unknown which kinds and degrees of stress can induce PTSD. PTSD symptoms are believed to be sufficiently caused by fear responses, particularly those linked to battle injuries. PTSD is often connected to other forms of stress, such as shame and guilt, and is often associated with moral harm during deployment. However, mental health education during and after deployment may not be enough to moderate the link between moral harm and poor mental health outcomes. Research on PTSD diagnosis and diagnostic criteria is limited, with structured clinical interviews being

uncommon and self-report instruments like the Posttraumatic Diagnostic Scale and the Impact of Events Scale more commonly used. Further examination of physical and psychological maladjustment is needed to better understand and manage PTSD.

TREATMENT

The American Psychiatric Association recently published guidelines for the prevention of PTSD. These guidelines include recommendations for treatment of patients with acute stress disorder. Acute stress disorder is a typical prelude to PTSD. Worldwide Society for Traumatic Stress Studies' practice recommendations, edited by Foe et al. provide an in-depth evaluation of PTSD treatments.

GENERAL PRINCIPLES

The initial step is to relieve or eliminate PTSD symptoms and indications, as well as any trauma-related comorbidities. The physician then works to promote adaptation and restore the patient's psychologic feeling of safety and trust. Finally, general treatment aims to reduce the generalization of the initial trauma and protect the PTSD patient from future reoccurrences.

NONPHARMACOLOGIC TREATMENT

In randomized clinical trials, the most PTSD patients treated with psychotherapy improved or recovered. Bradley et al. did a comprehensive meta-analysis of psychotherapy for PTSD, comprising articles published between 1980 and 2003. Cognitive-behavioural therapy and eye movement sensitization reprocessing improve the condition of more than half (53%) of people with PTSD. The authors emphasized, however, that most patients continued to experience substantial PTSD symptoms and indications after treatment. There was also an absence of long-term follow-up data. The major duty of the primary care physician in PTSD is to provide psychoeducation and referrals as needed. These doctors should maintain a weekly follow-up schedule for a few weeks after treatment begins. Follow-up visits can be biweekly, then monthly. Referrals to support groups may be appropriate.

Nonpharmacologic therapy methods for PTSD are based on a bio psychologic knowledge of the disorder. PTSD symptoms are linked to a extensive range of psychologic processes, including attention, beliefs, cognitive-affective reactions, coping methods, memory, and social-support networks. A new review by Brewin and Holmes provides an overview of the three basic theories of PTSD: emotional processing theory, dual representation theory, and cognitive theory.[7]

COGNITIVE BEHAVIOURAL THERAPY

CBT attempts to change a person’s feelings and actions by altering the thought or behaviour patterns that are accountable for negative emotions. A previous study in 2018 found strong evidence that CBT-exposure therapy is effective for reducing PTSD and other symptoms of depression. CBT has been shown to be an efficient therapy and Individuals in CBT learn to identify and replace thoughts that cause them fear or distress. The main aim is to figure out how specific thoughts about events trigger PTSD-related stress. A study evaluating an online form of CBT for patients with mild-to-moderate PTSD discovered that the online technique was as effective and less expensive than the same treatment administered face-to-face. A 2021 Cochrane evaluation evaluating the delivery of CBT via the Internet concluded that Internet-based therapy had equal beneficial benefits to face-to-face treatment. In exposure therapy, both imagined and actual trauma experiences are incorporated into the treatment process.

Some organizations have supported the need for discovery. The US Veterans administration has aggressively trained mental health professionals in long exposure therapy and behaviour therapy in order to better treat PTSD in US veterans. Recent research history on behaviour therapies could provide effects which are equal to well validated treatments. Many of these treatments include high exposure and have been effective in treating the core issues of PTSD and repetitive indication.

Eye movement desensitization and reprocessing

Francine Shapiro developed and studied a type of psychotherapy known as eye movement desensitization and reprocessing (EMDR). She discovered that when she thought about distressing memories, her eyes would move quickly. Controlling her eye movements while thinking made the thoughts less distressing

Shapiro and Maxfield proposed an adaptive information processing hypothesis in 2002 to explain why this could work. According to this hypothesis, evaluation of eye movement can be used as a way to process memories, causing the person and memory to change to more adaptive. EMDR is similar to cognitive behaviour therapy in that it involves exposure (revisiting the traumatic event), cognitive processing, and relaxation/self-monitoring.

Several trials of EMDR lasting for 4 months have been conducted in adults, children, and adolescents. In a previous review in 2018, there is now moderately strong evidence that EMDR can rapidly reduce PTSD symptoms and depressive symptoms. EMDR lowers PTSD symptoms for a short period of time to the point where one in every two persons wont met the definition for PTSD, however only tiny number of people participated in the trial, thus conclusions should be treated with caution pending additional research. There was insufficient solid data to determine whether EMDR helped remove PTSD in adults.

In children and adults, a meta-analysis of MetaNSUE (using randomised controlled trials) found that EMDR was equally effective as CBT and better than waitlist or placebo. There was some indication that EMDR could prevent depression. There was no research which compares EMDR to other effective treatments or medications. Adverse effects were mostly unexplored. Women who are sexual assault victims will be benefited more than those who had undergone other forms of stressful incidents.

When there is no large, high -quality randomized trial comparing EMDR with eye movements verses the absence of eye movements, the debate about no effectiveness. The meta-analysis authors published in 2013 noted, they discovered that in laboratory research, the data suggests that thinking about distressing memories while undertaking a job that increases eye movements less alertness and anxiety related to such memories."[8]

PHARMACOLOGIC TREATMENT

After a stressful experience, no single treatment or combination of drugs prevents the onset of acute stress reaction or subsequently PTSD. However selective serotonin reuptake inhibitors are considered the first-line pharmacological treatment for PTSD and are the only ones that have obtained FDA approval. fluoxetine, sertraline, paroxetine, fluvoxamine, citalopram, and escitalopram are all selective serotonin reuptake inhibitors. Selective serotonin reuptake drugs may diminish or eradicate the clinical characteristics of PTSD's three symptom clusters (reexperiencing, avoidance/numbness, and hyperarousal). Other antidepressants, such as tricyclic antidepressants, serotonin and norepinephrine reuptake

inhibitors (venlafaxine and duloxetine), and monoamine oxidase inhibitors, may be beneficial in treating PTSD.

In patients who suffers with PTSD, benzodiazepines may control anxiety and enhance sleep. One notable drawback of this class of medicines is that PTSD sufferers who are prone to addiction may abuse or become dependent on benzodiazepines. Furthermore, these medicines may interact with the cognitive processing of trauma required for psychotherapy interventions to be effective. Psychotropic drugs other than antidepressants and anti-anxiety medications may be required to treat lingering PTSD symptoms and indicators. Second-generation (newer, atypical) antipsychotic medicines may be required to address comorbid psychiatric symptoms or anxiety that is resistant to existing medications. Second-generation agents include clozapine, risperidone, olanzapine, quetiapine, ziprasidone, and aripiprazole. Clozapine is the least appealing choice among second-generation antipsychotic medicines due to comorbidities such as leukopenia, seizures, and myocarditis/cardiomyopathy. At clinically effective doses, first-generation antipsychotic drugs are more bound to produce significant motor side effects (tardive dyskinesia and the extrapyramidal syndromes of dystonic reactions, drug-induced Parkinsonism, and akathisia) than the second-generation antipsychotics. Second-generation antipsychotics tends to cause weight gain than first-generation medicines. Clozapine and olanzapine are most likely linked to drug-induced weight gain and metabolic syndrome-related symptoms. Ziprasidone and aripiprazole are least likely to cause weight gain. Risperidone and quetiapine have a moderate ability to promote weight gain. Various symptom clusters and mood instability can be treated with so-called mood stabilizer anticonvulsants (divalproex, carbamazepine, topiramate, and lamotrigine). PTSD symptoms, including as nightmares and flashbacks, may be caused by adrenergic hyperarousal in the CNS. To address this, studies of 2-adrenergic agonists and blockers are being conducted. There is growing interest in using prazosin, a postsynaptic alpha-adrenoceptor blocker, to treat PTSD-related nightmares. Almost definitely, the PTSD patient who has been referred to a psychopharmacologist will require many psychotropic drugs from various classes.[7]

CONCLUSION

PTSD is a highly prevalent and impairing condition. The primary care physician will be the first health care professional to engage the potential subject with PTSD. Each sign and symptoms of PTSD may differ in each person. Combat related trauma is a common cause of PTSD among men and rape among women. The first reaction to traumatic stress is largely biological and controlled by the amygdala. The neocortex and hippocampus control the memories and executive decisions, respectively, which lead to short, medium, and long-term effects in subjects exposed to severe stress. The reaction to the traumatic event decides whether acute or chronic PTSD persists.

Education and supportive psychotherapy can be in the field of the primary caregiver for the patient with PTSD. Education and supportive psychotherapy may be within the domain of the PTSD patient's primary caregiver. A primary care physician may also start selective serotonin reuptake inhibitors. Turning to experts should be a ready option. The continuous collaboration of the primary care physician with the patient and specialist provides the best course of treatment for potential PTSD patients. Systematic studies on the involvement of medical professionals with PTSD. Psychiatrists, psychologists, neurophysiologists, and neuroradiologists will lead to earlier detection of PTSD, better treatment, and better outcomes for this condition. The role of internal medicine in these efforts is crucial.

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