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Review Article

Dissociative Identity Disorder- A Forgotten Tale in Neuropsychiatry

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Abstract

Dissociative Identity Disorder is highly prevalent in clinical settings, generally undetected, with prevalence in the community as high as 1%. However, throughout the past few decades, progress in treating this condition has lagged behind than that in other psychiatric conditions. Individuals with dissociative disorders are one who had experienced childhood trauma and/or maltreatment more frequently than ones in any other psychiatric condition, and their dissociation is the pinnacle of human response to persistent developmental stress. An interruption in one or more mental functions is the essential component of dissociation. It also acts as a determinant in general psychiatry, including neurobiological and psycho-pharmacological studies. Despite the fact that dissociation may accompany nearly all mental disorders in addition to forming disorders in and of themselves, research on the topic has not been significant. Hence, this review aims to pour some light on the neglected area of DID, its prevalence, etiology, neurobiology, clinical appearance, diagnosis and clinical aspects. .

***Keywords**: Dissociative Identity Disorder; Neurobiology; Post Traumatic Stress Disorder; Psychotherapy; Hypnotherapy; Pharmacological Intervention*

Introduction

Dissociative Identity Disorder (DID), formerly called multiple personality disorder (MPD), is a complex group of psychological conditions resulting from chronic post-traumatic stress that has originated from childhood trauma, including abuse (usually repetitive physical, sexual, or psychological abuse of extreme category), emotional neglect, or other familial and social causes. The disorder is an empirically robust form of chronic psychiatric nature in view of its involvement with neurobiological, interpersonal and cognitive non-integration as an escape mechanism to unfathomable stress. There occurs an extreme form of dissociation, a decrease in connectivity of thought processing, including memories, emotions, actions, and most importantly a shift in identity or in perception of environment. This aspect is believed to be a form of coping mechanism in which the individual shuts down one identity to overcome an extremely violent, painful or traumatic stimuli that would otherwise have not been possible with their conscious self [1].

Dissociation can be defined as changes in consciousness marked by a sense of detachment from the self and/or the environment, as well as the failure to integrate information and self-attributions that should typically be consolidated [2]. DID is one of the most chronic and complex form of dissociative disorders and as documented in a clinical case series by one study, highest frequencies of the disease are associated with childhood psychological trauma ranging from sexual (57.1% - 90.2%), physical (62.9% - 82.4%) and

emotional (57.1%) abuse as well as neglect (62.9%). Among the high-risk population, substance abusers make the top strata. Conversely, these chemical substances and even chronic alcohol abuse can lead to dissociative experiences in patients while dissociation may also serve as a cause for an individual becoming addict [3]. Dissociative disorders and DID are not uncommon medical illnesses. According to clinical investigations, between 1 and 5 percent of individuals in psychiatric programmes may fit DID's diagnostic criteria. Many of the participants in these investigations had never previously received a dissociative disorder clinical diagnosis. Clinicians' ignorance of dissociation, dissociative disorders, and the impact of psychological trauma, as well as their own bias, are the main causes of the problems in diagnosing DID [2].

The term "dissociation" was first used to describe a mental condition in which different aspects of the personality are segregated into inaccessible compartments by French psychiatrist Janet in 1924. Prince, an American psychologist, popularised the idea in 1906 by reporting a clinical case with several personalities. A comparable case was documented fifty years later by two American psychiatrists, Thigpen and Cleckley (1954) [4].

Famous people with dissociative identity disorder include comedienne Roseanne Barr, Adam Duritz and retired NFL star Herschel Walker. Walker wrote a book about his struggles with DID, along with his suicide attempts, explaining he had a feeling of disconnect from childhood to the professional leagues. To cope, he developed a tough personality that didn't feel loneliness, one that was fearless and wanted to act out the anger he always suppressed. These "alters" could withstand the abuse he felt; other alters came to help him rise to national fame. Treatment helped Walker realize that these alternate personalities are part of dissociative identity disorder, which he was diagnosed with in adulthood [5].

Etiology

Multiple exposure, coping, and developmental aspects must be considered in order to fully comprehend the aetiology of DID. Traumas, family situation, child development, attachment, and culture all play a part in the creation of "alternate" selves, which are embodied representations of the metaphor of "a different person" or spiritual being. These selves have unique characteristics and distinct memories of trauma. When a kid experiences instability, coercion, and-most frequently-overt serious physical or sexual abuse, DID often develops, along with a disorganised relationship to caretakers. Additionally, the infant must possess the inherent ability to disassociate to an extraordinary degree, resulting in a variety of moods that do not gradually merge. These assertions about oneself help the youngster organise overwhelming and contradictory emotions like betrayal, horror, love, and shame. The child is unable to combine distinct behavioural and emotional states into a coherent or comparatively integrated self in accordance with the appropriate socio-cultural construction(s) of self because they are overpowered by strong, conflicting needs and emotions. This process is consistent with the fragmentation of internal identities in some cultures (such as mainstream Western culture), whereas in other cultures (such as non-Western cultures), it may be consistent with the control of the individual's consciousness and identity by external spirit-dual entities. Overall, the evidence suggests that a complex interaction of traumatic events, dissociative processes, psychosocial mediators, and socially constructed self-perceptions led to the formation of DID [6].

Based on Janet's theories, "structural dissociation of the personality," another etiological model, aims to develop a comprehensive theory of dissociation that encompasses DID. According to this hypothesis, dissociation emerges from a fundamental inability to combine systems of beliefs and personality functions. The personality as a total system can become split into a "seemingly normal part of the personality" dedicated to daily functioning and an "emotional part of the personality" dedicated to coping after exposure to potentially traumatic situations. Défense in this case has nothing to do with the psychodynamic idea of defines; rather, it has to do with psychobiological processes of survival in response to life threat, such as fight/flight. It is believed that that Chronic traumatisation and/or neglect may cause subsequent structural dissociation and the emergence of extra emotional facets of the personality [2].

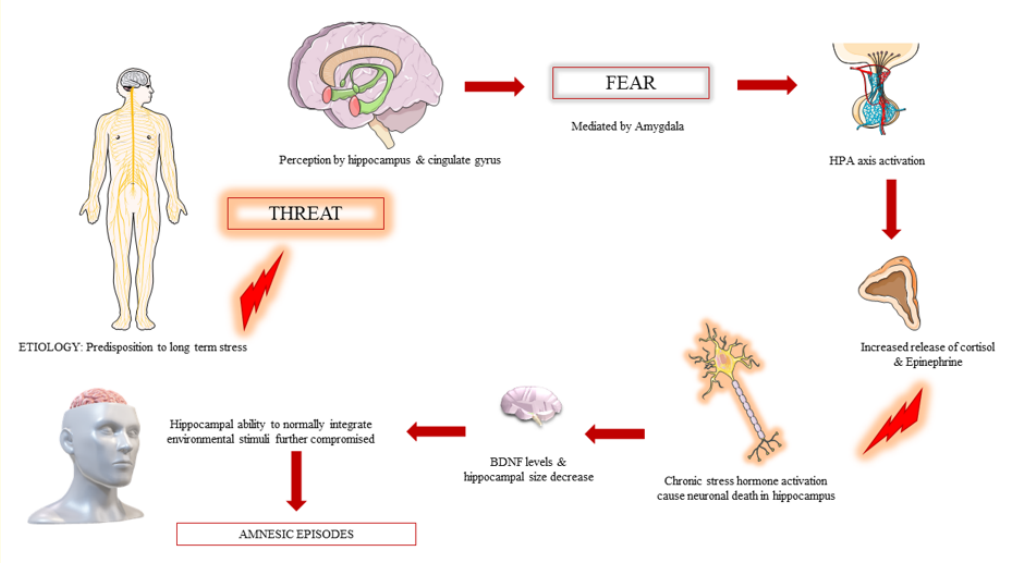

Figure 1: *Relationship between chronic stress and neurobiological symptoms of DID.*

Neurobiology of DID

Imaging and neurophysiological studies have shown discrete areas of interest in understanding DID.

Orbitofrontal region hypofunctioning

The hypofunction of the orbitofrontal region of the brain is one of the most specific hypotheses about the neurobiology of DID [7]. It has been suggested that early developmental trauma may have an impact on the orbitofrontal lobe [8]. In line with this theory, two single photon emission computer tomography (SPECT) tests carried out on DID patients while they were in their "host" identities showed bilateral orbitofrontal hypoperfusion as compared to healthy controls. Multiple scanning performed on a subgroup of these people while they were under the supervision of a different personality condition did not find any differences [9,10]. So it appears that orbitofrontal hypofunction is a trait indicator [10].

Switching" and inter-identity changes

There were changes between identity states on beta activity in the frontal and temporal regions according to a QEEG study [11]. Increased frontal QEEG delta activity in a hypnotically induced personality state has been seen in a patient with DID. In four of the eleven personality states examined in a QEEG study [12] on a patient with DID, there were left temporal and posterior-temporal-occipital alterations in the theta and beta-2 frequencies. In five DID patients, one study [13] found that in the temporal, frontal, parietal, and central regions, the average alpha coherence on QEEG was lower for the host personality state than for the alter personality state.

Differences in the corticolimbic system's biochemical response to trauma have been noted, in addition to the structural alteration in the hippocampus and amygdala areas of the brain's volume. the link between brain morphology, psychological disorders, and developmental

trauma. According to animal studies, stress causes the cortico-limbic system to produce glutamate, which has been linked to neurotoxicity, behavioural abnormalities, and brief dissociative episodes. It's also possible that glutamate release is what causes alterations in brain plasticity. Similar to this, N-Methyl-D-Aspartate (NMDA) antagonists that cause glutamate release have been used in similar animal models. Studies utilising NMDA antagonists, such as ketamine, do enhance glutamate release in humans, possibly producing the associated dissociative trance-like condition. Glutamate-inhibiting medications need to be investigated further to prove its potential as an early therapy for patients with traumatic events [14].

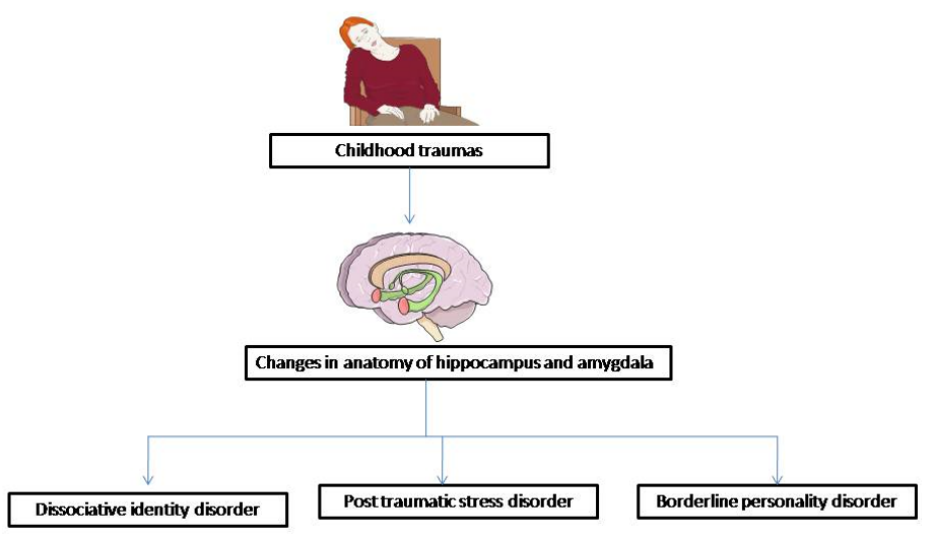

Figure 2: *A connection between childhood traumas, brain changes and different psychiatric disorders.*

Signs and symptoms

When two or more identities/personality accompany in an individual, each one having its own relatively unique behaviour of perceiving, thoughts and relation to environment as well as self, a basic characteristic of DID is formulated as per DSM-IV-TR. Also, two of these identities should necessarily have taken control of an individual's behaviour on recurrent basis. Dissociative amnesia, which is defined by an inability to recall significant personal information that is too extensive to be explained by regular forgetfulness, is usually present alongside DID. Depersonalization, derealization, spontaneous autohypnotic symptoms, pseudo-psychotic symptoms like being passively influenced by and/or hearing the hallucinated voices of alter identities, and multiple somatoform symptoms are all frequently experienced by patients with dissociative identity disorder. Most dissociative identity disorder patients have been found in clinical investigations to also meet the DSM-IV-TR criteria for posttraumatic stress disorder (PTSD). A complex range of concurrent symptoms linked with psychosis, mood, anxiety, affect management, and personality functioning make up the distinctive traits. Childhood attachment-based trauma appears to be a universal feature in the production of DID, while societal idioms of self-produce elements of cultural specificity. DID is the result of a combination of covert and overt developmental, interpersonal, and cultural forces. In practically every culture that researchers have thoroughly examined for the breadth of dissociative symptoms, DID is present [6].

Dissociative symptoms can accompany practically all mental diseases, such as borderline personality disorder, conversion disorder, and obsessive-compulsive disorder, in addition to being a diagnostic category unto themselves. Regardless of the primary condition,

dissociation is frequently associated with a history of childhood trauma, suicidality, self-mutilating behaviour and elevated general mental comorbidity. Psychiatric conditions including PTSD and schizophrenia have dissociative subcategories proposed. However, depending on the complexity of the prevalent dissociative symptom pattern, concomitant dissociation may also be considered as a concurrent diagnosis for certain of these psychiatric diseases [3].

According to the study by Hart., *et al*. memories of traumatic events in patients with DID initially manifest as sensorimotor fragments, subsequently take on more sensory dimensions, and eventually include a narrative component that is not necessarily "personalised." The growth of a narrative memory appears to occur more slowly in DID participants than in PTSD patients. Patients with DID may also lose memory of emotionally significant but non-traumatic situations, in contrast to sedations. The sensory component (re-experiences, dreams/nightmares, and intrusive thoughts) is less prominent than in traumatic memories, yet memories of these events also frequently come back in fragments [15]. The hippocampi and amygdalae of DID patients are smaller than those of healthy controls, according to a structural MRI research. Additionally, decreased hippocampus and amygdala sizes have been observed in patients with borderline personality disorder with a history of early maltreatment when compared to healthy controls [16].

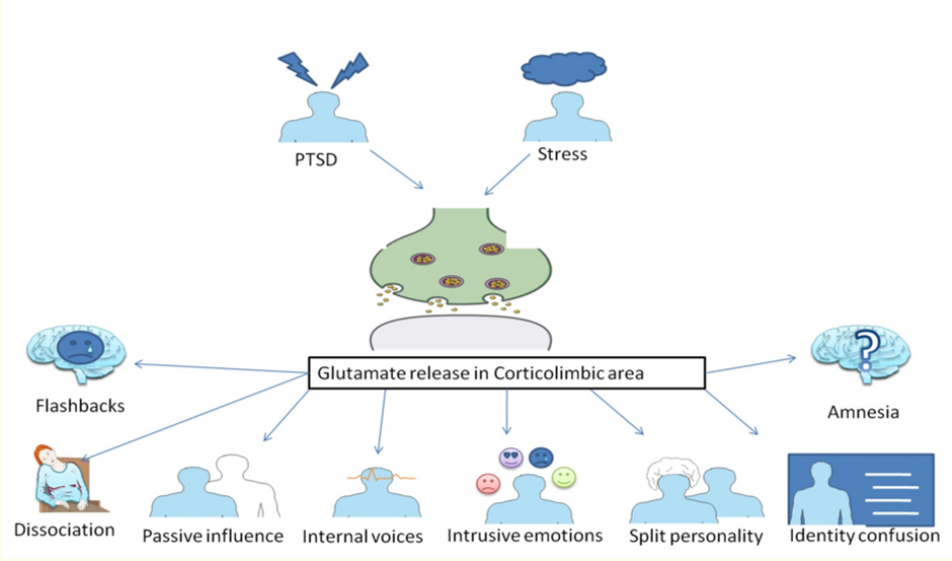

Figure 3: *Pathophysiological changes in DID followed by glutamate release in corticolimbic regions.*

Diagnosis

Insufficient training in recognising trauma-related dissociation, limited access to accurate scientific information about DID, symptom overlap with other disorders (such as schizophrenia, bipolar disorder, and borderline personality disorder), and the ongoing aetiology debate have all contributed to a resistance to considering a diagnosis of DID, which has hampered effective treatment [17]. Dissociative disorders have been excluded from large-scale epidemiological studies for many years due to the absence of dissociative disorder sections in frequently used general psychiatric screening questionnaires. The inclusion of dissociative disorders in general psychiatric screening studies is critical to prevent false negative diagnoses in future research, could very well facilitate better differential diagnosis between dissociative and other psychiatric disorders, and will also help to gather detailed information about true comorbidities. Despite studies using specific instruments having started to correct this perception. Wechsler Adult Intelligence Scale-Revised, Rorschach Inkblot Test,

Minnesota Multiphasic Personality Inventory-2, Millon Clinical Multiaxial Inventory-III, among others) can help understand the patient's personality structure and may provide information that can help differentiate DID from disorders that it is frequently confused with [2].

The diagnostic and statistical manual of mental disorders IV lists the following diagnostic criteria for DID:

A. Dual or multiple unique identities or personality states (each with its own relatively different pattern of perception, relation, and thoughts about the surrounding and self).

B. At least two of these characters or identities frequently seize control over the person's actions.

C. A failure to remember significant details about oneself that are too numerous to be accounted for by ordinary forgetfulness.

D. The disturbance is not brought on by a general medical condition or the immediate physiological consequences of a substance (such as blackouts or erratic behaviour while intoxicated by alcohol) (e.g. complex partial seizures). Children's symptoms cannot be attributed to making up playmates or engaging in other imaginative play [2].

Structured clinical interviews for DSM-IV [SCID] and the Composite International Diagnostic Interview (CIDI), which measure general mental psychopathology, lack portions that are appropriate for screening dissociative disorders. As a result, most general psychiatric epidemiological studies of a large size that used general psychiatric instruments failed to accurately detect dissociative disorders and produced biased reports in this setting. To close this gap, studies utilising methods for screening dissociation and dissociative disorders have been carried out. The most frequently utilised of these are self-rating tools like the dissociative experiences scale (DES), structured or semi-structured diagnostic interviews like the dissociative disorders interview schedule (DDIS), and the structured clinical interview for dissociative disorders (SCID-D) [3].

Treatment

The initial DID treatment recommendations were created in 1994, and then updated in 1997, 2005, and 2011. The International Society for the Study of Trauma and Dissociation's Treatment Guidelines for Dissociative Identity Disorder in Adults provide the current standard of care for DID treatment [18]. The treatment of DID is stage-oriented, starting with supportive and strengthening work, similar to the treatment of other traumatised populations [19].

Each identity appears to have its "own" first-person perspective, a sense of its "own" self, and an understanding of other parts as being "not self" in the majority of DID patients. Changes in emotional state or environmental demands cause people to switch between their identities, which causes a new identity to arise and take over executive function. The therapist must continuously balance the conflicting points of view of several identities because they each have unique roles, experiences, emotions, memories, and beliefs. Usually speaking in the first person, the ego in charge may repudiate other aspects or simply be oblivious of them. The goal of identity negotiation and conflict resolution is to assist the identities in understanding one another as valid components of the self [2].

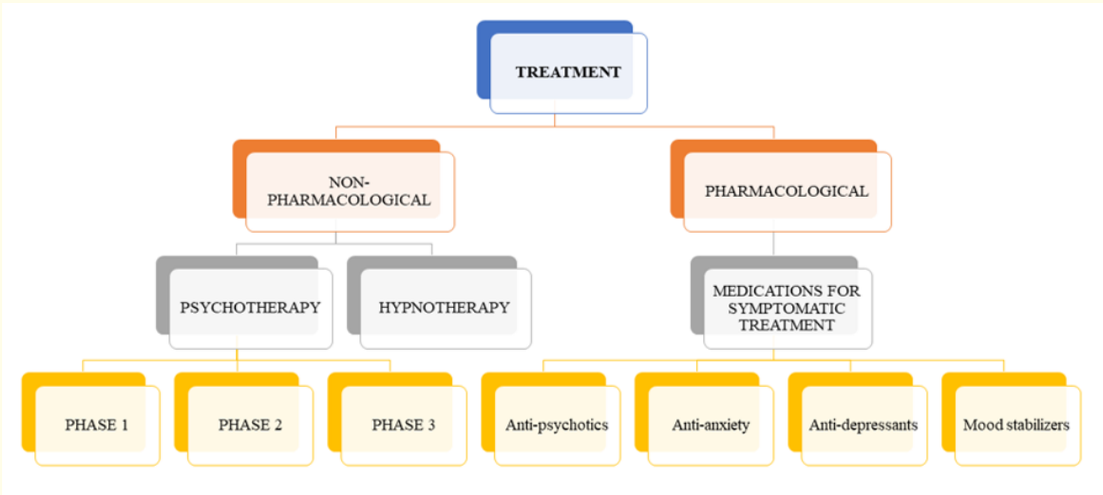

Figure 4: *Treatment strategies for DID.*

Non-pharmacological

Due to the lack of a solid pathophysiological pathway for the illness, this is thought to be the greatest treatment option for patients when compared to medication. More crucially, alter conflict or somatic encoding of any traumatic memory causes the somatoform symptoms that are frequently seen in victims of physical abuse. Therefore, it is preferable to approach these symptoms in a psychotherapy or hypnotherapeutic manner. Typically, these symptoms are long-lasting. Similar to pseudoseizures, occurrences of pseudoseizures in DID patients can be distinguished from epileptic attacks by recording epileptiform EEG discharges.

Psychotherapy

A viable type of integration or harmony among alternate identities is a desired therapy result. It can be difficult to understand when words like integration and fusion are employed. All work on fragmented mental processes throughout treatment is referred to by the broad, longitudinal process known as integration. The "Phase-oriented method" is the most often used psychotherapy approach in this regard. The stages of treatment described below reflect the main area of therapeutic concentration throughout each stage; overall, they help the DID patient gain safety, stability, and a stronger capacity for adapting to daily life [2]:

- Phase 1: Establishing safety, stabilization, and symptom reduction.
- Phase 2: Confronting, working through, and integrating traumatic memories.
- Phase 3: Integration and rehabilitation.

Hypnotherapy

Hypnosis, which continues to be the most widely used family of specialised techniques, has long been useful in the treatment of DID. Recent worries about the possibility of recovering confabulated and concretized pseudo-memories with hypnosis have been allowed to obscure the fact that hypnosis can provide anxiety relief, the chance to create sanctuary for the persecuted personalities in "safe place," and allied techniques, as well as unmatched opportunities to explore and influence the alter system, containment of affect, control of the abreactive process, facilitation of integration, and a variety of other techniques [19,20].

Pharmacological

Although medication does not treat the primary symptoms of DID, it is quite effective in treating specific target symptoms and treating co-occurring illnesses that respond to medication. Most DID patients use medication since they frequently also have other diagnoses. The design of a treatment plan is heavily influenced by whether a given diagnosis is present in any one individual or is the outcome of an illness that affects the entire population of people. This makes therapy difficult since it can be difficult to draw boundaries between these things. However, symptomatic therapies have been developed in accordance with the specific symptoms that a person is displaying.

Despite significant symptoms and dysfunction, many DID patients won't respond well to drugs, despite the best efforts to the contrary. Patients with DID require encouraging and sympathetic information regarding the existing dearth of pharmaceutical anodynes for their suffering. At the same time, efforts should be made to mitigate the pain or grief, whether or not medication is used. Clinicians should thoroughly familiarise themselves with hypnotherapeutic and psychotherapy techniques for easing DID patients' symptoms. These are typically more effective than any known pharmaceutical therapies for DID in many therapeutic settings. To go past our present dependence on anecdotal and ad hoc evidence, we must wait for more conclusive scientific studies on medication for DID.

Sadly, it may be challenging to do thorough research on psychopharmacologic therapies in DID. This is because DID individuals have a wide range of symptoms, it's possible that there are subgroups of dissociative patients that may respond to medicine differently, and

it's likely that drug effects will be minimal. The final possibility may require the execution of very sizable investigations in order to avoid missing a significant clinical effect. Studies are also required to define the state-dependent drug reactions in DID. As our understanding of dissociation, DID, PTSD, and the psychobiology of trauma expands over the coming ten years, we can only expect to find more potent psychopharmacologic treatments for MPD patients. In the end, trials to discover psychopharmacologic treatments that actually focus on the dissociation process by supporting, and helping them recover from their worst psychological trauma [21].

Psychotic-like symptoms: More often than not, actual or intercurrent psychoses or paranoia in DID is rare. Generally, they are dissociative pseudopsychotic manifestations like hallucination or even pseudohallucinations. This can also be attributed to the patients having "flashbacks" of the actions of their alters or instances arising from PTSD. Studies have reported moderate beneficial effects in patients when treated with low dose neuroleptics like haloperidol transiently. However, this use is only practiced when there is an overwhelming agitation or catastrophic levels of PTSD symptoms. In others, clinicians usually tend to avoid this use due to precipitation of adverse events like tardive dyskinesia [21,22].

Anxiety: One of the most common factors accompanying DID is a feeling of anxiety. This could be due to uneasiness about future events or something that has been done by any alter. Pharmacologically, long term stress gives rise to anxiety that in turn in chronic conditions leads to abnormal activation of autonomic nervous system as depicted in figure 1. This causes a shrinkage in hippocampal and amygdala size and volume as described earlier leading to a decrease in cognitive abilities. For theoretical reasons, Braun., *et al*. investigated propranolol and clonidine alone and in combination to treat switching, impulsivity, and anxiety in dissociative disorder patients, particularly those with DID. In open trial investigations, it was discovered that propranolol, when taken in accordance with their experimental design, is a helpful addition to psychotherapy in very nervous and/or quickly switching dissociative disorder patients. It is applied in fairly high dosages while being closely monitored by a doctor. Although it is best to start using it as an inpatient, outpatient administration has been done effectively, albeit much more slowly. When utilising higher doses of propranolol, the long-acting kind appears to be beneficial. In these patients, these medications can be used in addition to benzodiazepines, and their impact has been recorded as additive. For the same purposes, clonidine is useful, particularly in people who cannot take propranolol. If necessary, it can be used with propranolol [23].

PTSD: This is one condition that is present synergistically with DID patients requiring a definitive cure. Neurobiological evidence suggests DID on basis of clinical observations that it is one severe form of PTSD [17]. The PTSD cluster comprises of phobia, anxiety and panic where primary treatment strategy is again based on non-pharmacological therapies. Pharmacologically, it is believed that there is an involvement of GABAergic, serotonergic, endorphinergic and nor-adrenergic pathways in PTSD. In most literatures, medicines have been said to play an adjunctive role in treatment of DID associated PTSD mainly because intrusive and paroxysmal symptoms like hyperarousal, insomnia, nightmares & flashbacks, for which a single psychotropic is of no use [24]. Hence, use of serotonergic anti-depressants like fluoxetine and mood stabilizing agents such as lithium. Ant-anxiety agents like benzodiazepines have shown to improve the sleep pattern significantly. However, the debate on their proper usage is still a subject of conflict between clinicians.

Other mood disorders: These are called the affective symptoms that can be very intense but are typically present in specific alters. Mood shifts in DID patients are extremely rapid, even more than that occurring in rapid-cycling bipolar patients, which may be attributed to a shift in different alters. DID depicts a case of secondary affective disorders where the patient is already presenting with a case of psychiatric illness beforehand. Thus, use of anti-depressants, lithium and carbamazepine will depend upon the actual status of patient [25].

Conclusion

We talked about the pathophysiology of DID in our review along with the structural and chemical alterations in an individual's brain with DID. Numerous studies elucidating various mechanisms, such as the relationship between the limbic system and the affected amygdala and hippocampus, an increase in activation in the dorsolateral prefrontal and parietal cortex, and the limbic system and the

dorsolateral prefrontal and parietal cortex (all these regions are associated with short-term and long-term memory), may explain the primary symptom of changing the personality and forgetting completely about the previous personality. Hippocampus of DID individuals are smaller than normal. The orbitofrontal cortex shows various alterations, including decreased blood flow and functionality. This raises the possibility of additional symptoms, such as personality and thought-process alterations. We also emphasised the neurotransmitter glutamate's potential significance in the development of dissociation symptoms. More research is necessary to clarify this connection, though. If glutamate levels are discovered to be elevated in DID as our review shows, it may make a possible target for intervention in DID using glutamate blockers depending on how this association develops. However, as was already stated, it is still too soon to advise against such a course of treatment. These kinds of discoveries can undoubtedly benefit patients, neuroscientists, and psychiatrists. Despite numerous researches, it is still unclear why the illness manifests as transitory amnesia as opposed to chronic memory loss, especially given that there is evidence of anatomical alterations in the brain. Hence much more research is still needed in order to get better insights of this disorder.

Bibliography

1. Şar Vedat., *et al*. "Revisiting the etiological aspects of dissociative identity disorder: a biopsychosocial perspective". *Psychology Research and Behavior Management* 10 (2017): 137-146.

2. International Society for the Study of Trauma and Dissociation. "Guidelines for treating dissociative identity disorder in adults, third revision". *Journal of Trauma and Dissociation: The Official Journal of the International Society for the Study of Dissociation (ISSD)* 12.2 (2011): 115-187.

3. Sar Vedat. "Epidemiology of dissociative disorders: An overview". *Epidemiology Research International* 2011 (2011).

4. Paris Joel. "The rise and fall of dissociative identity disorder". *The Journal of Nervous and Mental Disease* 200.12 (2012): 1076-1079.

5. "Dissociative Identity Disorder (Multiple Personality Disorder): Signs, Symptoms, Treatment".

6. Dorahy Martin J., *et al*. "Dissociative identity disorder: An empirical overview". *The Australian and New Zealand Journal of Psychiatry* 48.5 (2014): 402-417.

7. Forrest KA. "Toward an etiology of dissociative identity disorder: a neurodevelopmental approach". *Consciousness and Cognition* 10.3 (2001): 259-293.

8. Shore Allan N. "Affect dysregulation and disorders of the self". New York: WW (2003).

9. Sar Vedat., *et al*. "Frontal and occipital perfusion changes in dissociative identity disorder". *Psychiatry Research: Neuroimaging* 156.3 (2007): 217-223.

10. Sar Vedat., *et al*. "HMPAO SPECT study of regional cerebral blood flow in dissociative identity disorder". *Journal of Trauma and Dissociation* 2.2 (2001): 5-25.

11. Lapointe AR., *et al*. "Similar or disparate brain patterns? The intra-personal EEG variability of three women with multiple personality disorder". *Clinical EEG and Neuroscience* 37.3 (2006): 235-242.

12. Hughes John R., *et al*. "Brain mapping in a case of multiple personality". *Clinical Electroencephalography* 21.4 (1990): 200-209.

13. Hopper Annedore., *et al*. "EEG coherence and dissociative identity disorder: Comparing EEG coherence in DID hosts, alters, controls and acted alters". *Journal of Trauma and Dissociation* 3.1 (2002): 75-88.

14. Rutkofsky IH., *et al*. "The neuropsychiatry of dissociative identity disorder: why split personality patients switch personalities intermittently". *Journal of Cell Science and Therapy* 8.2 (2017): 1-8.

15. Van Der Hart., *et al*. "Memory fragmentation in dissociative identity disorder". *Journal of Trauma and Dissociation* 6.1 (2005): 55-70.

16. Vermetten Eric., *et al*. "Hippocampal and amygdalar volumes in dissociative identity disorder". *American Journal of Psychiatry* 163.4 (2006): 630-636.

17. Reinders Antje ATS and Dick J Veltman. "Dissociative identity disorder: out of the shadows at last?" *The British Journal of Psychiatry* 219.2 (2021): 413-414.

18. Brand Bethany L., *et al*. "Separating fact from fiction: An empirical examination of six myths about dissociative identity disorder". *Harvard Review of Psychiatry* (2016).

19. Kluft Richard P. "An overview of the psychotherapy of dissociative identity disorder". *American Journal of Psychotherapy* 53.3 (1999): 289-319.

20. Spiegel David. "Hypnosis in the treatment of victims of sexual abuse". *Psychiatric Clinics of North America* 12.2 (1989): 295-305.

21. Loewenstein Richard J. "Rational psychopharmacology in the treatment of multiple personality disorder". *Psychiatric Clinics of North America* (1991).

22. Braun Bennett G. "Multiple personality disorder and posttraumatic stress disorder". *International Handbook of Traumatic Stress Syndromes. Springer, Boston, MA* (1993): 35-47.

23. Braun Bennett G. "Unusual medication regimens in the treatment of dissociative disorder patients: I. Noradrenergic agents". *Dissociation: Progress in the Dissociative Disorders* (1990).

24. Putnam Frank W. "Diagnosis and treatment of multiple personality disorder". Guilford Press (1989).

25. Torem Moshe S. "Psychopharmacology". Handbook of Dissociation (1996): 545-566.

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