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Dissociative Identity Disorder: A Pathophysiological Phenomenon

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Abstract

Various hollywood movies have shown a character who switches his personality, one personality does not remember the other at all. In the psychiatric world such a condition is called dissociative identity disorder (DID). The current study shows a collection of some studies showing the pathologic nature of the phenomenon. In this review, we have shown some studies including MRI studies, that demonstrated that the limbic system of DID patients decreases in size (especially hippocampus and amygdala). Moreover, a reduction in the functioning of orbitofrontal cortex is also observed with a reduced cerebral blood in DID patients. In the end, we recommend more studies in the future, to gain more insight about the pathophysiology of this mysterious condition, that will be helpful to treat and manage the patients with DID successfully in the near future.

Keywords: DID; Dissociation; Dissociative identity; Multiple personality; Split personality; DID brain; DID mechanism; DID pathophysiology

Introduction

The famous psychiatric condition, dissociative identity disorder (DID) also known previously as multiple personality disorder, has often been confused and misunderstood. Sometimes it is perceived as a spiritual phenomenon, especially in the developing world, while the scientific community believes it to be a pathophysiological disorder. The topic has been discussed in various Hollywood movies and many other TV shows and several movies across the world. In this short review, we will highlight some facts about the pathophysiological nature of this disorder, and put forward some studies supporting some alterations in the brain of such patients.

According to modern science, DID also known as multiple personality disorder or split personality disorder is a chronic psychopathological condition which commonly manifests after trauma or childhood abuse. It includes disruption in memory and identity of a person. The patient with DID presents with different identities and does not remember the event after coming back to his original identity [1]. Spiegel, et al. [2], proposed a change in the definition of DID to emphasize the disruptive nature of dissociation and amnesia for day to day as well as for traumatic events. They also proposed that the patient experience should include possession in the definition of identity disruption.

In 2013, DSM 5 was released and came up with many new definitions of Psychiatric disorders including DID. According to DSM-5, DID is defined as follows:

1. Two or more distinct identities or personality states are present, each with its own relatively enduring pattern of perceiving, relating to and thinking about the environment and self. According to the DSM-5, personality states may be seen as an “experience of possession.” These states “involve(s) marked discontinuity in sense of self and sense of agency, accompanied by related alterations in affect, behavior, consciousness, memory, perception, cognition, and/or sensory-motor functioning. These signs and symptoms may be observed by others or reported by the individual.” (Read about dissociative identity disorder alters) One important change from the fourth to the fifth edition of the DSM is that individuals may now report their perception of personality shifts rather than limiting diagnosis to shifts that others must report.

The second dissociative identity disorder criterion in the DSM-5 is:

2. Amnesia must occur, defined as gaps in the recall of everyday events, important personal information and/or traumatic events. (Dissociative amnesia: Deeply buried memories) This criterion for DID newly recognizes that amnesia doesn't just occur for traumatic events but, rather, everyday events, too.

3. The person must be distressed by the disorder or have trouble functioning in one or more major life areas because of the disorder. This criterion is common among all serious mental illness diagnoses as diagnosis is not appropriate where the symptoms do not create distress and/or trouble functioning.

4. The disturbance is not part of normal cultural or religious practices. This DID criterion is to eliminate diagnosis in cultures or situations where multiplicity is appropriate. An example of this is in children where an imaginary friend is not necessarily indicative of a mental illness.

5. The symptoms are not due to the direct physiological effects of a substance (such as blackouts or chaotic behavior during alcohol intoxication) or a general medical condition (such as complex partial seizures).

This characteristic of dissociative identity disorder is important as substance abuse or another medical condition is more appropriate to diagnose, when present, than DID” [3].

The idea behind writing this mini review was to highlight the psychopathology behind DID. Because not much has been published about the brain changes in the people exhibiting the symptoms of DID, this article will serve as a collection of some important studies discussing

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the pathophysiological mechanism associated with DID. Moreover, in this article we will address the changes in the amygdala; hippocampus and an involvement of orbitofrontal cortex (OFC) in DID patients, with reduction in functioning and the blood flow in OFC. Furthermore, in the end we will point towards a need of more research in the future to gain more insight about a clear pathophysiology involving any possible brain changes in the DID.

The Brain and the Dissociative Identity Disorder

Pathophysiology

Because not much has been written about the pathophysiology of DID. Mostly we have been relying on speculations. In this review, we have attempted to compile some relevant studies discussing a possible pathophysiology of DID. Some interesting studies have been found and included in this review. We found some studies about bipolar disorder, schizophrenia and their possible association with DID. These kinds of studies only point toward a possibility rather than claiming something with confidence.

Co-occurrence with schizophrenia, bipolar disorder and personality disorders

As we can see that in a recent study, Ross, et al. [4] concluded that there is a strong co-existence of DID and bipolar disorder, these kinds of studies just provide a hint of a possible association between the two conditions and any changes in the brain structure. The attempts to study a possible pathology involved in DID is not new. In 1995, Ellason et al. [5] stated “A substantial number of patients with dissociative identity disorder have had previous diagnoses of schizophrenia, due to the presence of positive symptoms of schizophrenia.” They searched the pattern of positive and negative symptoms in with dissociative identity disorder patients, and compared it with others with no such symptoms. The results indicated that the positive symptom and general psychopathology scores were worse in the dissociative identity group than the norms for schizophrenia, while the negative symptoms were remarkably more severe in schizophrenia. They further stated, “Since patients with dissociative identity disorder report more positive symptoms of schizophrenia than do schizophrenics, while schizophrenics report more negative symptoms, a primary emphasis on positive symptoms may result in false-positive diagnoses of schizophrenia and false-negative diagnoses of dissociative identity disorder.”

Enough evidences exist that schizophrenia patients have brain changes in almost all lobes including the occipital lobe [6]. Therefore, the co-occurrence of DID with either schizophrenia or Bipolar is an indication that in the future we will read more about similar studies, and possibly we will find some possible alterations in the brain in such patients. However, as of now it is premature to comment on any such brain changes.

Similarly, various other personality disorders and types have been studied in relation to DID. DID has been found associated with some personality disorders with avoidant disorder (76%) being the most common; followed by self-defeating (68%); borderline (53%), and passive aggressive (45%) personality disorders. This study inclined our attention towards the persona logic crux of DID [7]. Moreover, a preliminary study by Ross [8] was carried out in 2011 where he proposed the inclusion of possession experience in DSM V. This points toward a notion that a group of scientists has always been actively involved in trying to study, understand and treat DID as a pathophysiological phenomenon.

Neuroimaging studies and the changes in the brain: Limbic system and the cortex

In 2003, Brunson et al. [9] suggested that stress was one of the major contributing factors affecting the volume of hippocampus. This further emphasizes upon our review demonstrating DID and its association with early stress and changes in volumes of stress regulating brain areas. Many studies proposed childhood trauma as the most common precipitating cause of DID. But does childhood trauma tend to change volumes of hippocampus or amygdala? remained unanswered. Much neurological research in collaboration with radiology has to be done in order to get an answer.

The emergence of neuroimaging and especially structural as well as the functional MRI revolutionized the world of neurosciences [10]. The discovery made it possible to study many neurological disorders including DID. An MRI study by Vermetten, et al. compared the female brain structure of DID patients with healthy subjects. The study demonstrated alterations in the limbic system-the hippocampus and amygdala of the DID patients were found to be remarkably smaller (19.2% and 31.6%, respectively). Amygdala controls emotions while hippocampus controls long-term memory, thus Vermetten's study does provide a good logic in explaining the pathophysiology of DID [11]. However, another MRI study demonstrated reduction in hippocampal and amygdalar volume in PTSD patients, but no considerable difference in hippocampus and amygdala of DA/DID patients and normal subjects [12]. Vermetten counter argued the study by Weniger et al. by claiming that “patients with true dissociative identity disorder without PTSD essentially do not exist” [11].

Elzinga et al. [13], studied working memory in 16 patients with DID or dissociative disorder not otherwise specified (DDNOS). Dorsolateral prefrontal and parietal cortex regions were found to be activated in the patients and the control groups (same regions are involved in working memory). The difference was that the patients demonstrated an increased activation in these regions and made lesser errors with increasing task and more anxiety and less concentration, in comparison to the controls [13].

Cerebral blood flow and DID

Moreover, according to Sar et al. regional cerebral blood flow (rCBF) in DID patients was reduced in the bilateral orbitofrontal cortex regions (just like attention deficit disorder), and augmented in median and superior frontal regions and occipital areas (bilaterally) [14]. Considering “decision making” and its association with the orbitofrontal cortex, Sar stated that the reduced functioning of the OFC leads in impulsivity and the expression of the new personality in DID could be a sign of impulsive behavior. Cerebral blood flow has also been studied by other scientists. Reinders, et al. [15] calculated rCBF of DID patients in a neutral personality state (NPS) compared with a traumatic personality state (TPS). The subjects were asked to listen to a memory script. There were no differences between the NPS and TPS when listening to the neutral script, as well as no difference between the neutral and traumatic scripts for the NPS. While listening to the traumatic script, it was observed that a deactivation pattern of brain areas in the NPS exist. The study concluded that on a neurobiological level, the patients with DID possess different autobiographical selves. Furthermore, among the brain areas that were deactivated in the NPS versus the TPS is the medial prefrontal cortex. These kinds of studies point toward an idea about the involvement of orbitofrontal cortex in DID, as OFC is a part of prefrontal cortex (bilaterally) [16]. In a previous study by Sar et al. they showed that the regional cerebral

blood flow was reduced in the left and right orbitofrontal cortex of the DID patients and higher in their left (dominant) lateral temporal [16]. Some other authors have also discussed the OFC involvement [16-18]. In another study by Reinder et al. [19], regional cerebral blood flow data demonstrated different neural networks to be linked with different processing of the neutral and trauma-related memory script by NIS and TIS. They found that patients with their own access to autobiographical trauma-related memory had variations in brain activations in distinct mental states of self-awareness [19].

It is quite clear from the discussion above that there are some alterations in the brain structure of the patients suffering from this mysterious psychiatric disorder. Obviously, the data present currently in the databases and relevant journals is not enough and nor this paper contains all the information. This mini review is just an attempt to bring an attention toward a possible pathophysiological mechanism involved in DID and pointing toward a notion of its pathological nature.

Conclusion

DID is a pathophysiological disorder. The patients suffering with DID have been found to have some alterations in their brain morphology. Amygdala and hippocampus have been found to be affected in DID as well. Usually the reduction in size of the hippocampus of DID patients is observed. Moreover, studies also show an involvement of orbitofrontal cortex in these patients, with reduction in functioning and the blood flow in OFC. These kinds of findings can surely be helpful for the psychiatrists as well as the patient, because a complete understanding of the mechanism of such a unique mental disorder could be useful in finding a possible permanent solution for this medical problem. A solution more inclined towards the prevention, treatment and management. Studies like these can probably bring a new aspect in the prevention and treatment of DID. For example, as discussed above that some studies demonstrated the coexistence of schizophrenia and bipolar with DID, may be in the near future the same or similar kind of management of schizophrenia and bipolar, could be found to be useful in helping the DID patients; involving a psychotherapy and drug treatment. If not, then the treatment for the diseases involving hippocampus and amygdala like anxiety and memory disorders could be proven to be useful for these patients.

Likewise, it is also a possibility that these modes of management may have no effect in treating DID at all. It is far from the reality as of today, to use the same mode of treatment for DID (as some of the above mentioned disorders). It is obviously not possible to test these treatment modalities on a DID patient and no sane scientist or physician will ever recommend testing any new mode of treatment on an already affected patient. Neither do we recommend such a treatment. This is quite obvious that we will have to wait for many decades and a lot of future studies consistently need to be published for many years to come, in order know more about this rare disorder

and bring a universal effective modality of treating and managing DID effectively. Until then, we have no other option to rely solely on the previous modes of treatments that have been used to treat these patient and remain hopeful that a permanent cure for DID is discovered and solve this mystery.

References

1. Ross CA (1998) Dissociative Identity Disorder. *Handbook of Dissociation*.
2. Spiegel D, Loewenstein RJ, Lewis-Fernández R, Sar V, Simeon D, et al. (2011) Dissociative disorders in DSM-5. *Depress Anxiety* 28: E17-45.
3. American Psychiatric Association, DSM-5 Task Force (2013) *Diagnostic and statistical manual of mental disorders: DSM-5*. Arlington, VA: American Psychiatric Association, Washington D.C.
4. Ross CA, Ferrell L, Schroeder E (2014) Co-occurrence of dissociative identity disorder and borderline personality disorder. *J Trauma Dissociation* 15: 79-90.
5. Ellason JW, Ross CA (1995) Positive and negative symptoms in dissociative identity disorder and schizophrenia: a comparative analysis. *J Nerv Ment Dis* 183: 236-241.
6. Tohid H, Faizan M, Faizan U (2015) Alterations of the occipital lobe in schizophrenia. *Neurosciences (Riyadh)* 20: 213-224.
7. Dell PF (1998) Axis II pathology in outpatients with dissociative identity disorder. *J Nerv Ment Dis* 186: 352-356.
8. Ross CA (2011) Possession experiences in dissociative identity disorder: a preliminary study. *J Trauma Dissociation* 12: 393-400.
9. Brunson KL, Chen Y, Avishai-Eliner S, Baram TZ (2003) Stress and the developing hippocampus: a double-edged sword? *Mol Neurobiol* 27:121-136.
10. Tohid H (2016) Advancements in the Field of Neuroscience. *Int J Neurol Brain Disord* 3: 1-2.
11. Vermetten E, Schmahl C, Lindner S, Loewenstein RJ, Bremner JD (2006) Hippocampal and Amygdalar Volumes in Dissociative Identity Disorder. *The Am J Psychiatry* 163: 630-636.
12. Sar V, Unai SN, Ozturk E (2007) Frontal and occipital perfusion changes in dissociative identity disorder. *Psychiatry Res* 156: 217-223.
13. Elzinga BM, Ardon AM, Heijnis MK, Ruiter MB, Dyck R, et al. (2007) Neural correlates of enhanced working-memory performance in dissociative disorder: a functional MRI study. *Psychol Med* 37: 235-245.
14. Sar V, Unai SN, Ozturk E (2007) Frontal and occipital perfusion changes in dissociative identity disorder. *Psychiatry Res* 156: 217-223.
15. Reinders A (2003) One Brain, Two Selves. *Neuro Image* 20: 2119-2125.
16. Manton S (2016) Disorganized Attachment and the Orbitofrontal Cortex as the Basis for the Development of Dissociative Identity Disorder. *Art & Sciences Writing Program*.
17. Sar V, Unal SN, Kiziltan E, Kundakci T, Ozturk E (2001) HMPAO SPECT study of regional cerebral blood flow in dissociative identity disorder. *J Trauma Dissociation* 2: 5-25.
18. Schore AN (2010) Attachment and the Regulation of the Right Brain. *Attach Hum Dev* 2: 23-47.
19. Reinders AA, Nijenhuis ER, Quak J, Korf J, Haaksma J, et al. (2006) Psychobiological characteristics of dissociative identity disorder: a symptom provocation study. *Biol Psychiatry* 60: 730-740.