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The Role of Serotonin in Regulating Depression in PTSD Patients (Clinical Study)

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Abstract:

This study aimed to uncover the role of serotonin in regulating depression in patients with post-traumatic stress disorder (PTSD): What is the role of serotonin in regulating depression in patients with post-traumatic stress disorder?

We discussed the hypotheses and concluded that serotonin plays a key role in modulating mood and reducing depressive symptoms in patients with PTSD. The study relied on a clinical approach supported by the analysis of 3 clinical cases at Aboubaker Razi Psychiatric and Mental Disorders Hospital in Annaba and psychological scales such as the Beck Depression Inventory and the Davidson Trauma Inventory.

The results found a negative correlation between low serotonin levels and the onset of depressive symptoms, as well as the effectiveness of pharmacological treatments that target this neurotransmitter, especially SSRIs (selective serotonin reuptake inhibitors). The study reinforced the importance of combining psychotherapy and pharmacotherapy to achieve better results.

Keywords: Serotonin, depression, post-traumatic stress disorder, serotonin reuptake inhibitors, biological psychotherapy.

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1. Problematic introduction:

The impact of post-traumatic stress disorder (PTSD) is not only psychological but also physical, with many patients experiencing chronic physical symptoms such as headaches, muscle pain, and gastrointestinal disorders. PTSD is also associated with an increased risk of cardiovascular disease, diabetes, and other chronic diseases. (Debray, 2020, p. 259)

Given these devastating effects of PTSD on the lives of individuals and society, there is an urgent need for a deeper understanding of this disorder, identifying the factors that contribute to its development and persistence, and searching for effective ways to prevent and treat it. In this context, understanding the neurobiological mechanisms behind PTSD is of paramount importance, as it can contribute to the development of more targeted and effective treatment strategies. (Salmona, 2023, p 53)

Among these neurobiological mechanisms is the role of neurotransmitters, the chemicals that transmit signals between neurons in the brain. Neurotransmitters are essential for regulating a wide range of brain functions, including mood, emotions, sleep, appetite, memory, and learning. Several studies have indicated a close relationship between neurotransmitter disorders and PTSD, as patients often have imbalances in the levels of certain neurotransmitters in the brain.

Among the neurotransmitters that have received significant attention in PTSD research, serotonin stands out as a key neurotransmitter that plays a critical role in regulating mood, emotions, and behaviors. Serotonin is also known as the "happiness hormone" due to its positive effect on mood and feelings of happiness and well-being. However, serotonin's role in the brain is more complex than just being the "happiness hormone." It is involved in regulating a wide range of other brain functions, including sleep, appetite, memory, learning, aggression, and impulsivity. (Lejoyeux, 2021, pp 34-37)

Numerous studies have shown that disturbing the level of serotonin in the brain is closely linked to the onset of depressive symptoms, making selective serotonin reuptake inhibitors (ISRS: Les inhibiteurs sélectifs de la recapture de la sérotonine) the first therapeutic option in the treatment of depression and anxiety. However, the relationship between serotonin and PTSD is still a matter of scientific debate. Some research suggests that low serotonin levels are the primary factor in the onset of depressive symptoms in PTSD patients, while neuroimaging studies have shown that PTSD patients have abnormalities in certain serotonin-related brain regions, such as the

amygdale and the prefrontal cortex, which may explain some of their emotional symptoms. (Boulenger. Arpon, 2016, p 324)

However, the variability in research findings makes it difficult to determine whether serotonin imbalance is a major cause of depression in TSPT patients or, given the complexity surrounding the relationship between serotonin levels and the onset of depression in PTSD patients, this study is important. aims to gain a deeper understanding of the relationship between serotonin and depression in the context of PTSD, and to identify the mechanisms by which serotonin affects mood and emotions in these patients. Several studies have shown a relationship between serotonin levels and depression in PTSD patients. For example, a study by Smith and colleagues (2018) found that plasma serotonin levels were significantly lower in chronic PTSD patients compared to healthy people, and that these levels were negatively correlated with the severity of depressive symptoms. What if PTSD patients took medications containing serotonin? What role does serotonin play in regulating depression in PTSD patients?

2. Hypotheses of the study:

2.1. General hypothesis:

High or low serotonin levels significantly contribute to the development and exacerbation of depression in PTSD patients.

2.2. Partial hypotheses:

- Low serotonin level contributes to the worsening of mood symptoms and the onset of depression in PTSD patients.

- Higher serotonin level contributes to the improvement of mood symptoms and depression in PTSD patients.

I. Theoretical aspect:

1. Definition of serotonin:

Serotonin is a chemical neurotransmitter known as the "happy hormone" (5-hydroxytryptamine or HT-5 for short) that plays an important role in regulating mood, sleep, appetite, and cognitive function. Serotonin is mainly synthesized in the brain and gastrointestinal tract from the amino acid tryptophan. It also has a role in affecting the central nervous system, gastrointestinal tract and blood vessels. According to a study published in the annual reviw of medcine, serotonin may be associated with depression and mood disorders, while increasing it may improve mood. (Naudet, 2020, p 219)

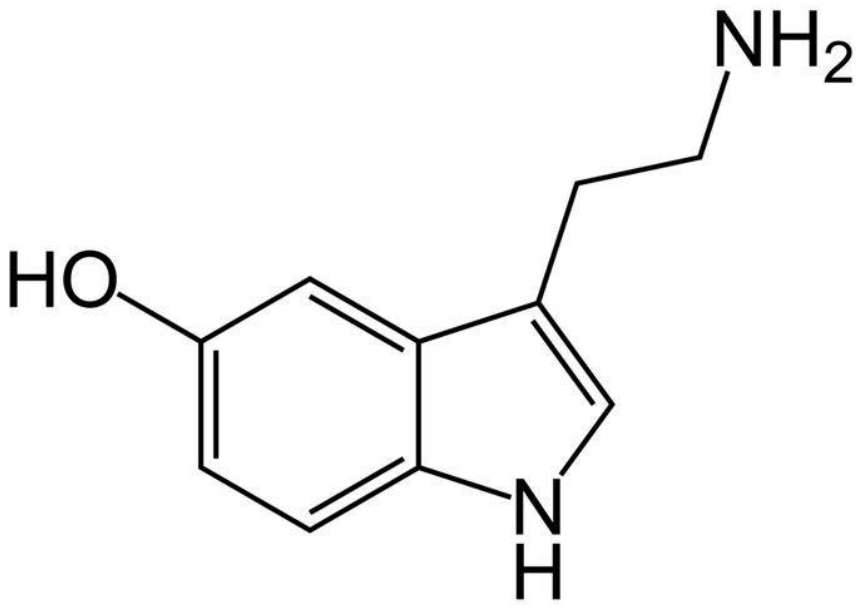
2. The mechanism by which serotonin is synthesized and produced:

Serotonin is mainly produced in enterochromaffincells, 90%, and in neurons in the central nervous system (CNS), 10%.

Serotonin (5-hydroxytryptamine, HT-5) is synthesized from the amino acid tryptophan, which is transported via plasma to the brain where it is converted to the precursor HT-5. (Hamon, 2020, p 194)

Tryptophan is an essential amino acid that is provided through the diet. L-tryptophan is hydroxylated at position 5 by the enzyme 5-tryptophan hydroxylase to produce 5-hydroxytryptophan (5-HTP), the latter is then decarboxylated (COOH-) by a decarboxylase (AADC) in the presence of vitamin B6 to produce serotonin.(Jacquot, 1989, pp 618-626)

Figure (1) The chemical structure of serotonin



3. Functions of serotonin:

Serotonin is a neurotransmitter that plays a key role in regulating many vital functions in the body and brain:

3.1. Mood and emotion regulation: Serotonin plays a key role in regulating mood and reducing anxiety and depression. Its deficiency may be associated with depression and anxiety disorders. (Bockaert et al, 2019, p 204)

3.2. Control of appetite and digestion: Serotonin affects appetite, reducing the feeling of hunger. It also plays a role in bowel movements, as more than 90% of it is found in the gastrointestinal tract. (Enders, 2015, p 58)

3.3. Production of melatonin: Serotonin helps in the production of melatonin, the hormone responsible for regulating sleep, as high levels during the day stimulate wakefulness, while low levels at night promote sleep. (Brillant, 2017, p 161)

3.4. Influence on cognition and memory: Serotonin is involved in cognitive processes such as learning, memory, and decision-making, and has a role in optimizing cognitive functions. (Stahl, 2021, p 73)

3.5. Regulation of body temperature: Serotonin contributes to the regulation of body temperature, which helps keep physiological functions stable. (Kim et al, 2023, p 275)

3.6. Impact on the heart and blood vessels: Serotonin affects blood pressure and vasodilation, making it an important factor in heart health. (Blangy et al 2021, p 336)

3.7. Sexuality: A relationship has been found between serotonin levels and sexual desire, where high levels of serotonin reduce anxiety and impulsivity but also sexual desire, a fact that explains why many antidepressant medications can reduce sexual desire.

3.8. Reducing feelings of nausea: When we eat something toxic, unpleasant or not properly tolerated by our body, the gut increases serotonin production to increase intestinal transit. (Delvaux. Mayer, 2017, p 282)

4. The role of serotonin in depression:

Serotonin is synthesized through enzymatic reactions, and any mutation that affects these processes may cause deficiencies at several levels such as decreased production, impaired uptake, or dysfunctional receptors leading to depression.

There is also a hypothesis that suggests that serotonin hyperactivity may contribute to depression, as increased serotonin at synapses leads to sensitization and increased receptor excitability. Some studies have shown that depressed and suicidal patients have increased 5-HT2 and B-adrenergic receptors in the frontal cortex of the brain. (Ferreri, 2018, pp. 52-54)

4.1. Serotonin receptors and their role in depression:

Every behavior is regulated by several serotonin receptors located in different areas of the brain, affecting anxiety, appetite, energy balance, and movement.

Receptors important in depression include 5-HT1A-B, 5-HT2A, and 5-HT3.

5-HT1 receptors are densely located in the dorsal raphe nuclei and cerebral cortex and play a role in eating disorders, depression, and obsessive-compulsive disorder.

5-HT2A and 5-HT2C receptors, which bind to G proteins, are found in brain regions responsible for emotions, such as the amygdala, hippocampus, and prefrontal cortex, and are among the most important receptors targeted in the treatment of depression.

Antidepressants reduce the density of these receptors, which helps alleviate symptoms.

Thus, serotonin is a key element in understanding depression, whether due to its deficiency or hyperactivity, opening the way for the development of treatments that target the balance of its neural activity in the brain. (Hamon, 2021, pp. 67-71)

5. Serotonin and PTSD:

Post-traumatic stress disorder (PTSD) is a mental disorder that appears following exposure to a traumatic event, affecting emotional regulation, memory, and stress response.

Serotonin (HT-5) plays a central role in regulating mood, anxiety, and sleep. Dysfunction in the serotonin hormone system is associated with PTSD symptoms, including hypervigilance and trauma reliving. (Miller et al, 2018, pp680-690)

Studies suggest that polymorphisms in the serotonin transporter gene (SERT, 5-HTTLPR) influence vulnerability to PTSD, especially in individuals exposed to early trauma. (Peretti et al, 2021, pp68-69)

II. Field aspect:

1. Methodology of the study: Due to the nature of the current study, the case study method was used, which is the most commonly used method for such studies.

We used the clinical method, as it is a clinical study that relies on interviews and uses tests and observations to reach the goals set by the clinical method, as Khalid Abdul Razzaq Al-Jar defines it as a method that relies on similarity: We all differ in quantity and agree in quality, but the feelings remain the same and the difference lies only in the amount.

The clinical method focuses on clinical observation in order to collect data and information that helps the specialist to understand the patient, his suffering, and the type of therapeutic sponsorship that applies to the case.

2. Study instruments: After observing and interviewing the subjects, we applied the Beck Depression Scale and the Davidson Trauma Scale.

3. Results of the two tests:

Based on the results of applying the two tests to the studied sample of 03 cases, the results showed a clear distribution of depression and trauma levels according to the Beck and Davidson scales. In the Beck Depression Scale, the cases recorded levels of depression that fell into the "very severe" category (37+ scores), while severe depression (24-36 scores), moderate depression (16-23 scores), mild depression (10-15 scores), and no depression (less than 9 scores). In general, the arithmetic mean of the depression scores in the sample was around points, with the dominance of specific symptoms such as sleep disturbances and chronic guilt.

On the Davidson Trauma Scale, one case met the criteria for a diagnosis of full-blown PTSD based on the presence of at least one reliving symptom, 3 avoidance symptoms, and a minimum of 2 arousal symptoms. The overall mean score was points out of 68, with symptom severity varying between sub-dimensions, with the "arousal" dimension (e.g., hypervigilance and irritability) scoring the highest. A clear clinical correlation was also observed between the severity of depression and trauma, as other cases diagnosed with major depression (≥24 on the Beck scale) constituted the majority of all PTSD cases, while trauma with moderate or severe depression was present in two cases. It should be noted that these results reflect acceptable reliability of the two scales (Beck reliability: 0.77, Davidson reliability: 0.78-0.99) in the local environment.

4. Analyze the results in light of the hypotheses:

4.1. Hypothesis 1: Recall the first hypothesis: "Low serotonin level contributes to the worsening of mood symptoms and the onset of depression in PTSD patients."

Our research hypothesis centers on the role of serotonin as a critical neural component in the development of post-traumatic stress disorder (PTSD) and depression in individuals who have experienced severe psychological trauma. Serotonin (5-HT) is a central neurotransmitter with a prominent role in regulating mood, sleep, appetite, and cognitive functions. Recent research suggests that imbalances in this neural system may contribute substantially to the development of complex psychiatric disorders such as PTSD and depression. Our study aims to discuss how serotonin alterations contribute to explaining the clinical symptoms of these disorders, drawing on recent scientific studies and real-life clinical cases.

Several studies suggest that changes in the serotonergic system may play a pivotal role in the development of PTSD and depression symptoms.

A recent neuroimaging study (Smith et al., 2022) showed that patients with PTSD show significantly reduced serotonin levels in the amygdala (AMYGDALA) and hippocampus (HIPPOCAMPUS), two areas responsible for fear processing and emotional memory. These findings support our hypothesis that serotonin disruption promotes the fixation of traumatic memories and the maintenance of alertness. The case of Samir: Samir, who was severely traumatized after the death of his mother and the suicide of his brother, represents a typical case of serotonin disorder after a devastating emotional trauma. He exhibited symptoms of tantrums, hypervigilance, and sleep disturbances, along with pronounced depressive manifestations such as loss of meaning and persistent sadness. This overlap between the symptoms of PTSD and depression strongly supports the hypothesis that serotonergic dysregulation could be common to both conditions.

On the other hand, a systematic study (Brown & Taylor, 2023) showed that low availability of serotonin receptors in the prefrontal cortex is directly related to the severity of depressive symptoms. This supports research findings that serotonin regulates one's mood, and that its imbalance leads to feelings of sadness and loss of interest.

The case of Kawthar: Kawthar, who survived a car accident in which she lost her best friend, developed symptoms of social withdrawal and severe depression with compulsive flashbacks to the accident. The sudden change in her psychological life, from an active teacher to a person suffering from

chronic sadness and sleep disturbances, points to a dysregulation of serotonin, especially with symptoms of rumination and a tendency to withdraw.

The case of Muhammad: Mohammed, who survived a horrific bus accident, suffers from recurrent nightmares, panic attacks, and deep depression. His accurate description of the accident demonstrates distorted and painful storage of the traumatic memory, reinforcing the hypothesis that impaired serotonin regulation contributed to the fixation of this traumatic memory and led to the onset of PTSD symptoms with depressive features.

Table (1) Analytical comparison of clinical conditions and their relationship to serotonin

Case	Shocking event	Psychological symptoms	The supposed serotonin imbalance
Muhammad	Fatal bus accident	Flashback, anxiety, sleep disturbance, deep sadness, depressive symptoms	Post-traumatic stress disorder, emotional dysregulation and traumatic memory
Samir	Mother's death and brother's suicide, witness to the event	Anger Attacks Depression Avoidance, Altered Personality, Substance Abuse	Chronic serotonin deficiency, neurotransmitter dysfunction due to repeated trauma
Kawthar	Car accident in which her best friend died	Mood disorder, isolation, nightmare flashbacks	Disruption of serotonin-related sensory and emotional responses

4.2. Hypothesis 2: Reminder of Hypothesis 2: "Elevated serotonin level contributes to improved mood symptoms and remission of depression in TSPT patients."

The second hypothesis in our paper concerns the role of serotonin in improving mood symptoms and remission of depression in TSPT patients. Serotonin is a neurotransmitter that plays a pivotal role in regulating mood,

anxiety, and stress response. Its altered levels have been linked to a number of psychiatric disorders, including depression and PTSD. In this hypothesis, we propose that elevated serotonin levels may contribute to the improvement of a patient's clinical symptoms and limit the worsening of trauma-related depression.

Recent scientific studies are helping to clarify the role of serotonin in the treatment of psychological symptoms associated with PTSD and depression. One study that supports this hypothesis is Felipe Corchs' (2009) study on the effect of tryptophan depletion on patients recovering from PTSD using SSRIs. The results of the study show that patients who were exposed to tryptophan depletion (the primary substance in the synthesis of serotonin) showed an increase in their responses to trauma-related stimuli. These findings suggest that the level of serotonin may play a pivotal role in reducing the response to traumatic stimuli, improving mood symptoms and reducing depression.

Another study that supported this hypothesis was James W. Murrougha's (2011) study, which showed reduced serotonin transporter binding in the amygdala in PTSD patients. It is thought that this poor association with serotonin may be responsible for the increased symptoms of anxiety and depression in these patients. This finding supports the idea that improving serotonin levels may help restore normal neurological function, thereby reducing the negative symptoms associated with trauma.

Annmarie MacNamara's (2016) study supports this hypothesis by revealing the effect of SSRIs in regulating emotions in PTSD patients. The results of the study showed that treatment with drugs such as paroxetine enhances the brain's ability to better process emotions, leading to improved mood and reduced depressive symptoms.

Samir is an excellent example of how elevated serotonin levels can help improve PTSD symptoms. From the beginning of his treatment, Samir used SSRIs, which helped to significantly improve his mood. Initially, his symptoms included bouts of anger and constant anxiety, but over time, these symptoms gradually began to subside. This improvement can be attributed to improved serotonin levels in the brain,

In addition, Drevets et al. (2020) study shows that antidepressant treatment can raise plasma serotonin levels and lead to an improvement in depressive symptoms, suggesting that serotonin plays an important role in improving a patient's psychological state.

Table (2) Comparison of cases, medications prescribed, and symptoms before and after treatment

Case	Prescribed medication	Symptoms before treatment	Changes after treatment
Muhammad	Sertraline 50mg	Violent flashbacks, excessive anxiety, sleep disturbance, depression, feelings of guilt	Reduced flashback, improved sleep quality, lower anxiety level, more stable mood
Samir	Paroxetine 20mg	Severe anger outbursts, depression, substance abuse, difficulty with impulse control, social avoidance	Improved mood, reduced anger outbursts, gradual onset of psychological stability, reduced addiction
Kawthar	Sertraline 50mg	Social isolation, nightmares, trauma flashbacks, persistent sadness	Decrease in nightmares, improved sleep, gradually increasing social interaction

5. General analysis of the results:

In light of the hypotheses, personal and family data, and the response of the cases to the Davidson PTSD Scale and the Beck Depression Scale, it was observed that all cases showed varying levels of depression and anxiety, reflecting a complex picture of the interaction between psychological trauma and mood disorders, and the outcomes of these cases are closely related to the role of the hormone serotonin, which plays a key role in regulating mood and emotional response.

Trauma negatively affects neurotransmitter systems, primarily serotonin, resulting in low levels of serotonin in the brain. This decrease explains many of the symptoms seen in the studied cases, such as persistent sadness, loss of interest, sleep disturbances, and a tendency to social isolation.

Numerous scientific studies show that PTSD patients are often deficient in serotonin secretion, which increases the likelihood of developing chronic depression, and that serotonin maintenance therapy combined with cognitive-behavioral psychotherapy (CBT) is essential. The goal is to restore chemical balance, reduce the worsening of mood and behavioral symptoms, and improve patients' quality of life in the long term.

The scientific literature has established that serotonin plays a pivotal role in regulating mood and emotions, and has a direct impact on how the brain processes traumatic experiences. Felipe Corchs' (2009) study showed that acute tryptophan depletion increases the response to trauma-related stimuli in PTSD patients who recovered with SSRIs, supporting the essential role of serotonin in promoting psychological resilience and confirming that trauma leads to an imbalance of hormones and neurotransmitters, of which serotonin is the most affected. Through my field experience, I have observed that cases that have experienced severe trauma, such as the loss of a family member or witnessing a traumatic event, do indeed show hypersensitivity to any trauma-related stimulus, even years later, which supports the findings of this study.

James W. Murrougha et al (2011) supported this, finding an abnormally low binding of the serotonin transporter 5-HTT in the amygdala in PTSD patients, which is associated with increased anxiety and depression. This biological abnormality is a logical explanation for the continued psychosocial suffering of the studied cases, even years after the trauma. This is what we observed in some of the cases, where generalized anxiety and chronic stress were inherent in them, with no clear justification in the present, which can be explained by this biological imbalance.

This opens the way for pharmacological interventions targeting serotonin receptors (such as ISRS inhibitors) to improve symptoms, as relative improvement was observed in the studied cases after ketyl incorporation, although the improvement was not complete, highlighting the need for continuity of treatment and follow-up of biological changes, in the same context, Annmarie MacNamara's study (2016) found that treatment with ISRS inhibitors, such as paroxetine, improves emotion regulation in TSPT patients, especially by improving brain function, which explains the variability in patients' responses to treatment depending on brain activity before the intervention. such as paroxetine, improves emotion regulation in TSPT patients, especially by improving brain function, which explains the variability in patients' responses to treatment depending on brain activity before the intervention, as this study found that some patients showed clear improvement with medication, while others did not show a noticeable

response, suggesting individual biological differences as indicated in the study.

By following the case and its psychological history, we observed that complex traumatic symptoms such as “re-personalization” or “remoisiement de la personnalité traumatique” - a change in one's perception of oneself and the world - may be a direct result of the prolonged negative impact of trauma on the serotonin system. This is supported by MacNamara's (2016) study, which asserts that the neurological changes that accompany TSPT lead to a “reprogramming” of emotional and behavioral cognition. This was clearly observed in one case, who expressed herself in sentences such as: “I no longer know my soul”, reflecting a ruptured identity and a radical change in self-perception.

On the other hand, Zadima Zarrouk Belaid's (2018) study confirms a strong relationship between PTSD and depression, supporting the clinical correlation between the two disorders, as supported by biochemical data. This is also confirmed by the study of Drevets, Turecki, and Sun (2020), which showed that serotonin levels in depressed patients are significantly lower than in healthy people, and that there is a relationship between kynurenine metabolites and symptom severity, which was actually reflected in the severity of symptoms in some patients, as I observed that the psychological state clearly deteriorates with the absence or interruption of treatment, which indicates the fragility of the biochemical balance.

As such, the results of this study not only show how serotonin is associated with depression and PTSD, but - similar to James et al (2011) - emphasize that low serotonin may be an important biomarker for diagnosis and therapeutic intervention. This is also supported by Ewa Alicja Ogłodek (2022), who showed how interactions between genes and environmental factors such as trauma can affect serotonin receptors and cause lasting changes in mood and emotion.

James Murrough et al (2011) and Annmarie MacNamara (2016) also discussed the relationship between serotonin and the brain circuits responsible for fear and attention, especially the amygdale and the prefrontal cortex. The dysfunction of these areas explains the patients' hyperarousal, which was also observed in the second case (Samir), especially in the form of sudden outbursts of anger and avoidance behaviors. This observation confirms that the exaggerated neurological response is not only a psychological issue, but has organic roots that require dual intervention.

From a therapeutic point of view, the results of the study supported the importance of combining CBT with ISRS-based pharmacotherapy, and I felt that this combination was the most effective, especially in cases with dual suffering from chronic traumatic and depressive symptoms.

Malaak et al (2025) emphasized that the combination of these two modalities leads to significant improvements in mood, psychological insight, and adaptability. The follow-up of the cases indicates a relative improvement after incorporating ketyl into the treatment plan, although the improvement was not complete, which indicates the need to continue treatment and follow up the biological effect.

On the other hand, the study of Rahma Ahmed Al-Kaso (2021) indicates that a decrease in serotonin also leads to an increase in oxidative stress indicators. The study cases (Samir, Mohammed, and Kawthar) show a range of complex psychological and physiological symptoms, such as hyperarousal, anger outbursts, and chronic fatigue, which can be partially explained by the findings of this study, and the improvement of oxidative The improvement in oxidative markers after using sertraline, as shown in the study, provides a partial explanation for the improvement in some aspects of Samir's psychological stability after undergoing treatment, and confirms that medications not only have a psychological effect, but also work to restore an internal biological balance that includes both the nervous and immune systems, linking the psychological and biological aspects in a more comprehensive manner. This integration of neurological and mood indicators supports our findings in this study and highlights the importance of viewing TSPT as a complex disorder that involves multiple levels: Biological, psychological, and social.

By analyzing the clinical data and comparing the results of the studied cases with the findings of recent scientific studies, it is clear that PTSD and depression share complex biological and psychological mechanisms, in which the hormone serotonin is the cornerstone. The cases studied showed clear indications of mood and emotion dysregulation, which is directly related to imbalances in the central serotonin system. The results also confirmed the importance of this hormone in explaining chronic and overlapping symptoms, reinforcing the need to adopt a dual therapeutic approach that targets both psychological and biochemical aspects.

Therefore, any effective therapeutic intervention must take into account the biological specificity of each case, and rely on an accurate assessment of serotonin levels, as well as ongoing psychological care. These findings call for more applied research that integrates clinical observation and

neurobiochemical analysis to better understand the relationship between serotonin, depression, and PTSD, thus contributing to the development of more effective and specialized treatment strategies.

6. Conclusion:

We conclude after our presentation of the study conducted in its various stages, in which we tried to adhere to the methodological controls of scientific research and went through all its known stages from asking questions to start, doing the exploratory study put the research path on the right path, so we defined the study questions and hypotheses, and determined - based on the theoretical balance that was collected and categorized - the tools of field investigation from observing these patients diagnosed with PTSD and depression. The tools of field investigation from the observation of these patients diagnosed with PTSD and depression, interviewing psychologists and doctors who follow their treatment and their health files, and we developed two scales, the Davidson Trauma Scale and the Beck Depression Scale, as a complementary test to ensure the validity of the results obtained from our study of 3 cases, which we carefully applied during their implementation. Taking into account the specificity of the studied topic, we followed the clinical approach in this regard... We conclude that the hormone serotonin is one of the main biological factors in regulating the psychological and emotional state of individuals.

Low levels have been shown to be associated with worsening symptoms associated with depression and post-traumatic stress disorder.

Evidence has shown that an imbalance of this neurotransmitter negatively affects the ability of individuals to adapt to traumatic events and leads to difficulty in restoring psychological and cognitive balance, hindering the patient's return to a normal and stable lifestyle.

These findings support the importance of adopting an integrated approach to diagnosis and treatment that takes into account not only psychological and behavioral aspects, but also the brain and biological changes associated with these disorders.

Separating the psychological and biological dimensions is no longer feasible in light of the complexity that characterizes contemporary mental disorders. The study emphasizes the need to enhance community awareness of the long-term effects of trauma and the importance of early intervention to treat serotonin dysregulation as a preventive step to avoid worsening symptoms and deteriorating quality of life.

It also highlights the need to adopt holistic treatment strategies that take into account the complex interplay between biological, psychological and social factors, as a multidimensional approach has proven effective in improving treatment outcomes and increasing patients' ability to adapt and regain their social and occupational functions.

The results of the study also point to the importance of supporting patients through sustainable psychological rehabilitation programs, focusing on relaxation techniques, cognitive reconstruction, and the use of supportive medications in the presence of documented chemical imbalances.

Although this study has provided a clear scientific framework for understanding the role of serotonin in the emergence and development of psychiatric disorders, there is still room for future studies that aim to explore deeper and more nuanced mechanisms of this neurotransmitter's influence. Future research should include the study of genetic factors that may contribute to an individual's sensitivity to low serotonin, as well as environmental factors such as lifestyle, nutrition, and exposure to repetitive stressors.

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