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Research Article

Impact of Methamphetamine (Crystal) use on central neurotransmitter homeostasis

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ABSTRACT

This study was designed to determine the impact of Crystal use on the levels of central neurotransmitter homeostasis, Dopamine, Serotonin, and Acetylcholine, as well as oxidative stress markers. This study involved 60 male crystal users from the Rehabilitation Center in Baghdad and 20 non-crystal users; their ages ranged from 19 to 54 years. They were divided into four experimental groups based on the length of hospitalization: the first group consisted of individuals who were non-users of Crystal as control group, the second group consisted of individuals recently discharged (recovery period 1–2 months), the third group included those with a moderate recovery period (2–4 months), and the fourth group comprised long-term recoverees (more than 6 months in the rehabilitation centers. Results: The study revealed highly significant decreases ($p \leq 0.001$) in the serum levels of dopamine, serotonin, and acetylcholine, with the most pronounced reductions observed in the moderate-recovery group, followed by the recently recovered group, compared to the control group, Current study's finding regrading oxidative stress indicators showed a highly significant increase in the oxidants Malondialdehyde (MDA), and Lactate Dehydrogenase LDH, with regarding level the antioxidant variables Glutathione (GSH) and Superoxide dismutase SOD), revealed a highly significant decrease. The long-term recovery group showed no significant difference in the antioxidant variables (GSH and SOD) compared with the control group.
Conclusion: Based on the results of the current study, it was evident that the use of crystal methamphetamine has a significantly high effect on neurotransmitter levels. This was clearly demonstrated in the groups of recently admitted and mid-term rehabilitation participants. In addition, there was a significant increase in the levels of oxidative stress variables. The length of the rehabilitation period inside the treatment facility also had a substantial impact on the return of toluene concentrations (or "toluene," as stated in the study) in neurotransmitter levels, and on the oxidative stress/antioxidant balance variables in the study samples, bringing them back to normal levels.

1. INTRODUCTION

The World Health Organization (WHO) defines drugs as natural or synthetic chemical substances that affect the central nervous system, resulting in changes in consciousness, behavior, and cognitive function. Drugs include a variety of substances such as opium, cocaine, marijuana, and methamphetamine, which differ in their effects and harms to human health [1]. Drugs can be classified into legal and illegal categories, where some types are allowed for medical use under medical supervision, while the use of many other controlled substances is prohibited in most countries due to their negative effects on public health. Drug abuse is an addictive behavior that contributes to the deterioration of mental and physical health and can lead to changes in individual behavior and profound impacts on society as a whole [2]. Drugs are a global health problem and pose a threat to many communities due to the negative effects associated with their use, including neuronal destruction, effects on brain neurotransmitters, and increased psychological and physical risks; drugs cause a large number of social, health, and economic problems worldwide [3]. According to researchers, abuse does not require the appearance of withdrawal symptoms or an inability to stop using the substance, but it can lead to this over time. Abuse is considered the beginning of progression toward addiction if it persists. [4]. Addiction is a chronic and complex condition characterized by physical and psychological dependence on a particular substance or addictive behavior. Addiction is a chronic disorder that leads the individual to need the substance continually and makes it difficult to recover without appropriate treatment. Addiction dependence on the substance, causing clinical or physiological withdrawal symptoms when the substance is stopped, which does not usually occur with abuse. [5].

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Crystal (Methamphetamine) is a powerful stimulant that affects the nervous system, the endocrine system, and metabolism, and it stimulates the release of large amounts of norepinephrine and dopamine, leading to a state of heightened neural activity and extreme alertness [6] .Methamphetamine causes severe damage to neural structures, especially in areas responsible for memory, emotions, and decision-making, and it also leads to depletion of neurotransmitters, particularly dopamine and serotonin, resulting in serious psychiatric symptoms such as hallucinations, aggression, and severe depression. The danger of crystal meth lies not only in its destructive biological impact but also in its ease of manufacture and spread among youth, making it one of the major health, psychological, and social challenges facing communities in the modern era. [7]. The metabolic process of Crystal breakdown generates a cascade of free radicals and oxidants that cause direct Oxidative stress and many diseases [8]. Regarding oxidative stress and antioxidants, crystal disrupts the balance between reactive oxygen species (ROS) and antioxidant defenses in the body. The high rate of metabolism induced by the stimulant increases free radical production, molecules that can attack proteins, lipids, and even DNA. With repeated use, the body's natural ability to produce antioxidants such as glutathione (GSH) and catalase declines, leading to a state of oxidative stress. This stress contributes to accelerated cellular damage and favors the onset of chronic inflammation that may affect the liver, heart, and brain [9]. Neurotransmitters are chemical substances that transmit signals between neurons, and the site of release of these neurotransmitters varies according to the type of neurotransmitter and its mechanism of action. Dopamine is released mainly in brain regions such as the Ventral Tegmental Area, the Nucleus Accumbens, and the Substantia Nigra. Dopamine is responsible for the feeling of reward and general mood; under normal conditions, dopamine is released in response to rewards, but it can be markedly affected by drug use, such as cocaine and methamphetamine, where its release is stimulated in large amounts, leading to a temporary sense of euphoria [10].
The metabolism of neurotransmitters refers to the chemical processes that occur after the release of these transmitters in the brain, where they are broken down or modified for reuse or disposal. The mechanism of neurotransmitter metabolism varies depending on the type of transmitter and the neuronal environment. In the case of dopamine, it is broken down by enzymes such as dopamine oxidase, which converts dopamine to homovanillic acid, and is eliminated via urine. The half-life of dopamine is very short, lasting only a few minutes in the synapses before being degraded by enzymes [11]. Serotonin is a monoamine neurotransmitter derived from the amino acid tryptophan. It is stored in vesicles before synaptic transmission, is released from the brainstem, and disseminates to the cortex and tissues outside the brain. It has more than seven receptor types (Receptor Subtypes - 5-HT1 to Hydroxytryptamine Receptor - 5-HT7); most of them act through a G protein (Guanine Nucleotide-binding Protein). A G protein is a protein that acts as an intracellular signal mediator and becomes activated when a specific receptor (for example, a Protein-Coupled Receptor GPCR) binds an external molecule such as a hormone or neurotransmitter. The G protein, in turn, activates enzymes like adenylate cyclase to produce cyclic adenosine monophosphate (cAMP) or ion channels to modify cellular activity, leading to changes in cAMP, which is a very important second messenger in cells. It transmits signals from outside the cell (such as the effect of hormones or neurotransmitters) and influences the regulation of mood, sleep, appetite, anxiety, and pain. The decrease in serotonin activity is associated with depression and sleep disorders [12]

1.1 Study Aims

Our study came behind the fact of the increasing use of Crystal in the Iraqi community, and we investigated the health effects of this chemical substance on the brain, and the oxidative stress markers with continuous use**.**

2. MATERIALS AND METHODS

2.1 Collect study samples

Samples for the study were collected in collaboration with the Ministry of Interior, General Directorate of Drugs and Psychotropic Substances Affairs. A total of 60 blood samples were collected from individuals who are drug users and are located in the rehabilitation center affiliated with the directorate above, Baghdad, Rusafa. Their ages ranged from 19 to 54 years, and they were divided into three equal groups based on the duration of their rehabilitation for the users. Additionally, 20 blood samples were taken from non-users Crystal male aged 19 to 54 years to serve as the control group. Five milliliters (5 mL) of venous blood were drawn for each subject and placed into Gel Tube test tubes for the purpose of performing biochemical tests. After centrifugation at a speed of 6000 rpm for 5 minutes, the serum was stored frozen at −20°C until the necessary tests were performed.

2.2 Estimation of Neurotransmitters Levels (DOP-SOR-ACL)

Levels of all neurotransmitters, including Dopamine (DOP), Serotonin (SERO), and Acetylcholine (ACL), being studied were estimated using the ELISA technique with a microplate reader, based on the enzyme-linked immunosorbent assay method. A specific antibody for each neurotransmitter, manufactured by the American company Cusabio, was used.

2.3 Determination levels of oxidants & Antioxidant's parameters

The levels of oxidative stress variables, represented by LDH and MDA, were assessed in the experimental groups, along with the antioxidant markers GSH and SOD, following the ELISA protocol described in section 2.4.3. The assays employed antigen-specific kits for the above variables, manufactured by the American company Cusabio, except for the superoxide dismutase (SOD) assay, the kit for which is produced by [Carlo Erba- France].

2.4 Statistical Analysis

Statistical analysis was performed using SPSS software (version 27). A Randomized Complete Block Design (RCBD) was used to organize the distribution of samples into four experimental groups: newly diagnosed patients, patients with a medium length of stay, long-term patients, and a control group, with a uniform sample size of 20 individuals per group. One-way ANOVA was employed to determine differences among the four groups for each variable, followed by Least Significant Difference (LSD) tests to identify the locations of differences at a significance level of $P \leq 0.001$. To classify the groups according to significant differences, Duncan's Multiple Range Test was used, with significant differences indicated by superscripts (a, b, c). Results were presented as mean ± standard deviation (Mean ± SD), and significance was indicated as High Significance at $P \leq 0.01$ and Significance at $P \leq 0.05$ [13].

3. RESULTS

3.1 Results of neurotransmitter parameters

The results of the statistical analysis of neurotransmitter variables in this study, as shown in Table I, indicate statistically significant differences ($P \leq 0.001$) for these variables when compared with the control group.

TABLE I: NEUROTRANSMITTER CONCENTRATION IN STUDY GROUPS.

Parameters	Mean ± SD				P value
	G1	G2	G3	G4	
Dopamine (pg/mL)	900.73 ± 87.89 a	681.92 ± 109.09 b	530.68 ± 64.43 c	856.93 ± 35.32 a	<0.001 HS
Serotonin (ng/mL)	467.92 ± 21.71 a	264.69 ± 29.86 c	334.88 ± 76.27 b	449.09 ± 18.73 a	<0.001 HS
Acetylcholine (pg/mL)	429.83 ± 19.86 a	243.22 ± 38.67 b	229.49 ± 31.56 b	445.65 ± 29.42 a	<0.001 HS

S: significant correlation between parameters ($0.01 < p$ value ≤ 0.05)
HS: Highly significant correlation between parameters (p value ≤ 0.01)
Groups with different letters are significantly different.
G1: Control , G2: Newly hospitalized, G3: Intermediate hospitalized, G4: Old hospitalized
N: sample size =20 for each group.

3.2 Dopamine neurotransmitter(pg/mL).

The deviations point to a highly significant decrease ($P \leq 0.001$) in the Dopamine (DOP) values in the experimental groups of recent addicts G1 and those in mid-recovery G2, respectively (G1: 681.92 – G2: 530.68) compared to the control group (G4: 900.73). In contrast, the DOP values in the long-term recovering addicts group indicate a return to the physiological level, showing no significant difference compared with the control group, with a value of (G3: 856.93). Additionally, the mid-recovery addicts group showed the greatest considerable decrease compared with the control group (see Figure 1).

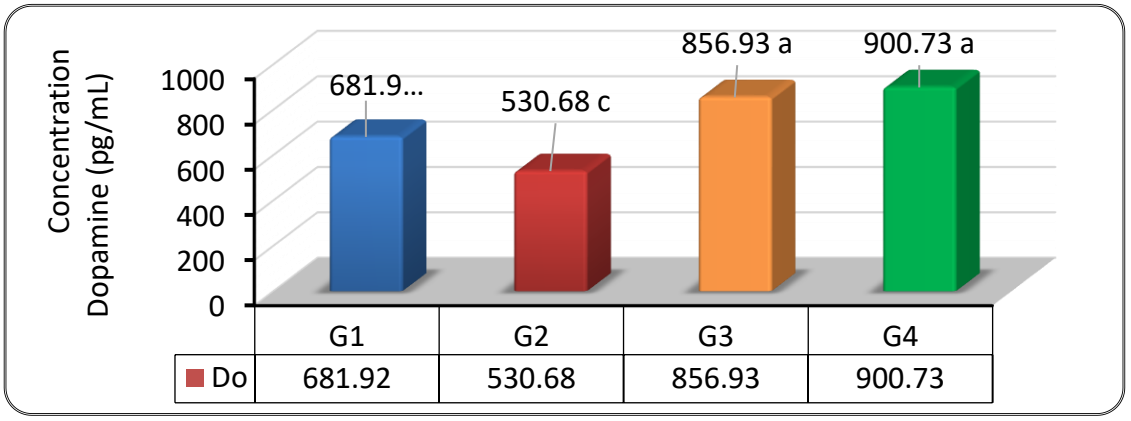

Fig. 1. Dopamine levels in study groups.

3.3 Serotonin neurotransmitter (pg/mL).

Results showed a highly significant decrease ($P \leq 0.001$) in the level of Serotonin neurotransmitter specifically in the groups of recently and mid addicts recovery, (G1: 264.69 - G2: 334.88) respectively, when compared with the control group(G4:467.92). In contrast, the Sero values in the long-term recovering addicts group indicate a return to the physiological level, showing no significant difference compared with the control group, with a value of (G3:449.09). Additionally, the recent recovery addicts group showed the greatest considerable decrease compared with the control group (see Figure 2).

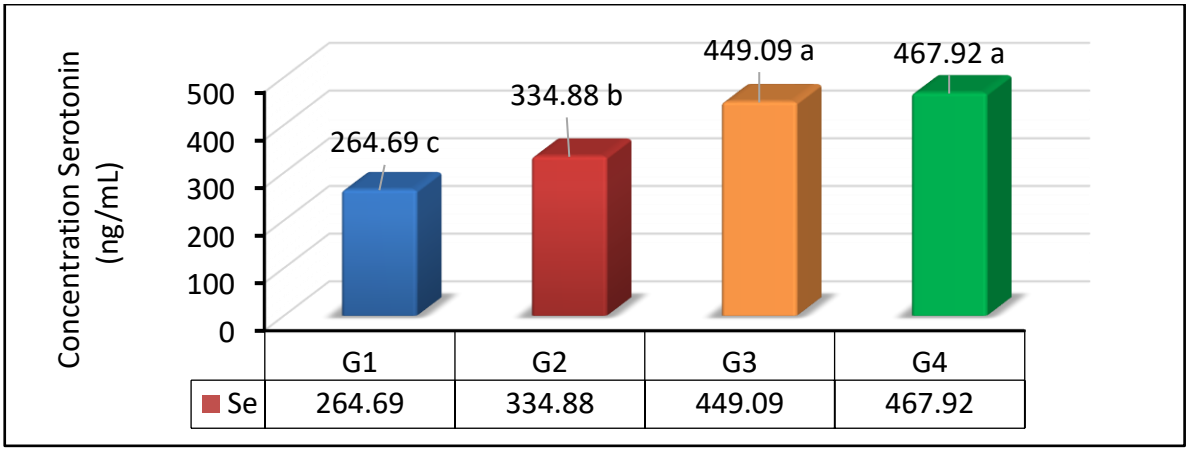

Fig. 2. Serotonin levels in the study group

3.4 The result of Acetylcholine (pg/mL).

Results showed a highly significant decrease ($P \leq 0.001$) in the level of Acetylcholine neurotransmitter specifically in the groups of recently and mid addicts recovery, (G1: 243.22 - G2: 229.49) respectively, when compared with the control group (G4:429.83). In contrast, the Ach values in the long-term recovering addicts group indicate a return to the physiological level, showing no significant difference compared with the control group, with a value of (G3:445.65). Additionally, the recent and mid recovery addicts group showed no significant changes between them (see Figure 3).

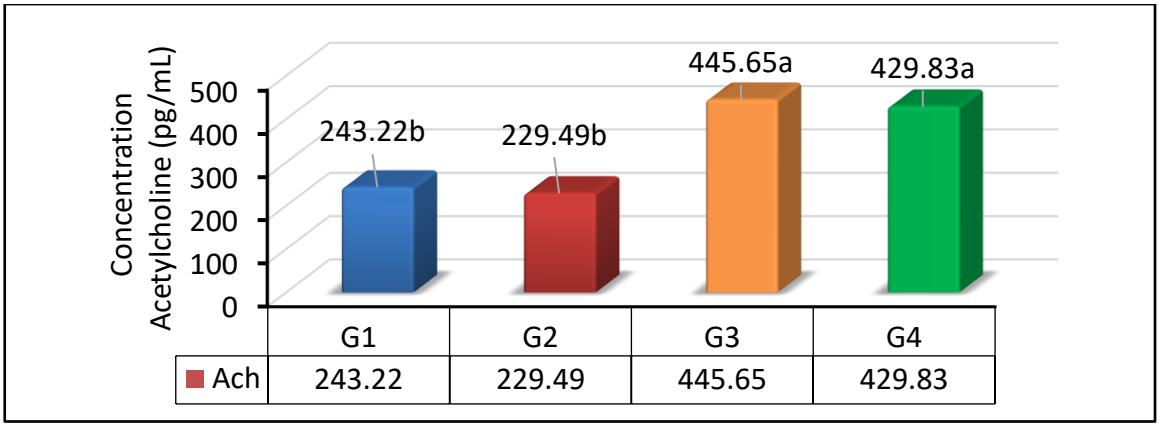

Fig. 3. Acetylcholine levels in the study groups.

4. RESULT OF OXIDATIVE STRESS AND ANTIOXIDANT PARAMETERS

4.1 Oxidant's parameter (MDA).

Results in Table II showed a highly significant increase ($P \leq 0.001$) in the levels of oxidant parameters (MDA and LDH) in all the groups(G1, G2, and G3) when compared with the control group G4.

TABLE II. SHOWED THE LDH (µMOL/ML) AND MDA (NG/ML) LEVELS.

Parameters	Mean ± SD				P value
	G1	G2	G3	G4	
MDA (ng/mL)	417.36± 5.69 c	948.9± 58.16 a	810.56± 8.96 b	508.38± 1.09 c	<0.001 HS
LDH (µmoL/mL)	0.18 ± 0.02 c	0.30 ± 0.05 a	0.22 ± 0.02 b	0.19 ± 0.02 c	<0.001 HS
GSH (µg/mL)	70.36 ± 1.19 a	62.62 ± 2.44 c	67.81 ± 1.86 b	71.08 ± 1.12 a	<0.001 HS
SOD (pg/mL)	1713.23± .37 a	948.77± 19.6 b	976.44± 6.92 b	1729.01± 7.1 a	<0.001 HS

S: significant correlation between parameters ($0.01 < p$ value ≤ 0.05)
HS: Highly significant correlation between parameters (p value ≤ 0.01)
Groups with different letters are significantly different.
G1: Newly hospitalized, G2: Intermediate hospitalized, G3: Old hospitalized, G4: Control
N: sample size =20 for each group.

4.2 MDA Results

The results are shown in Figure 4, high elevation, significant increase in the levels of MDA in recently recovered, mid-addicts. Recovery. In contrast, the (MDA) values in the long-term recovering addicts group indicate a return to the physiological level, showing no significant difference compared with the control group, with a value of (G3).

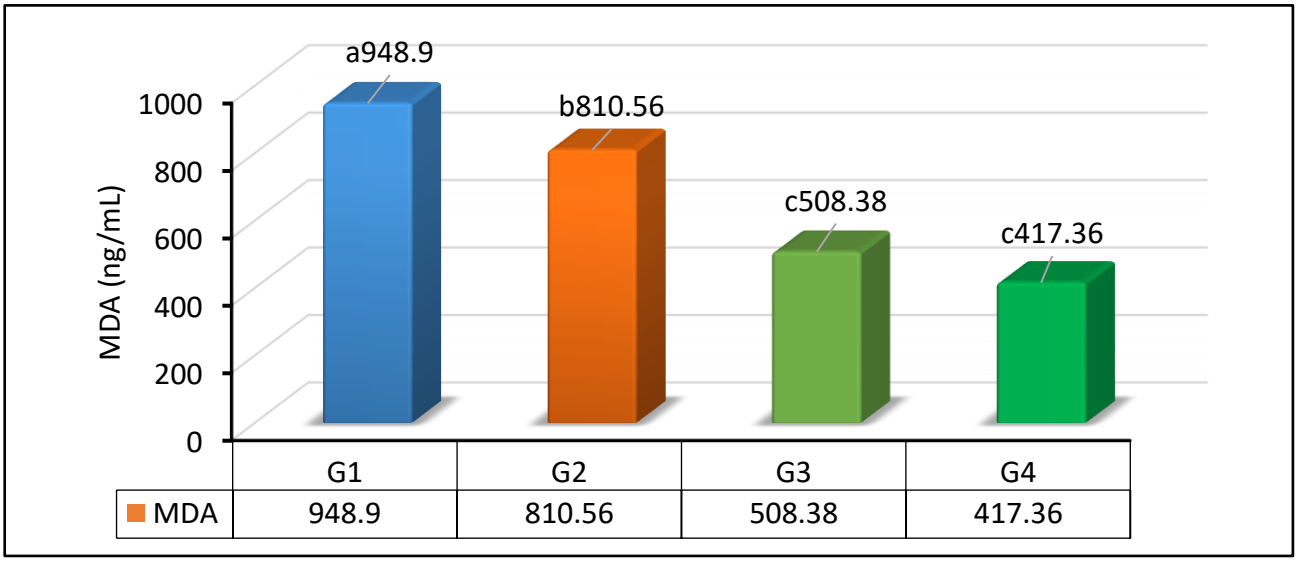

Fig. 4. MDA levels in the study groups.

4.3 LDH Results

The results are shown in Figure 5, high elevation, significant increase in the levels of LDH in recently recovered, mid-addicts. Recovery. In contrast, the (LDH values in the long-term recovering addicts group indicate a return to the physiological level, showing no significant difference compared with the control group, with a value of (G3).

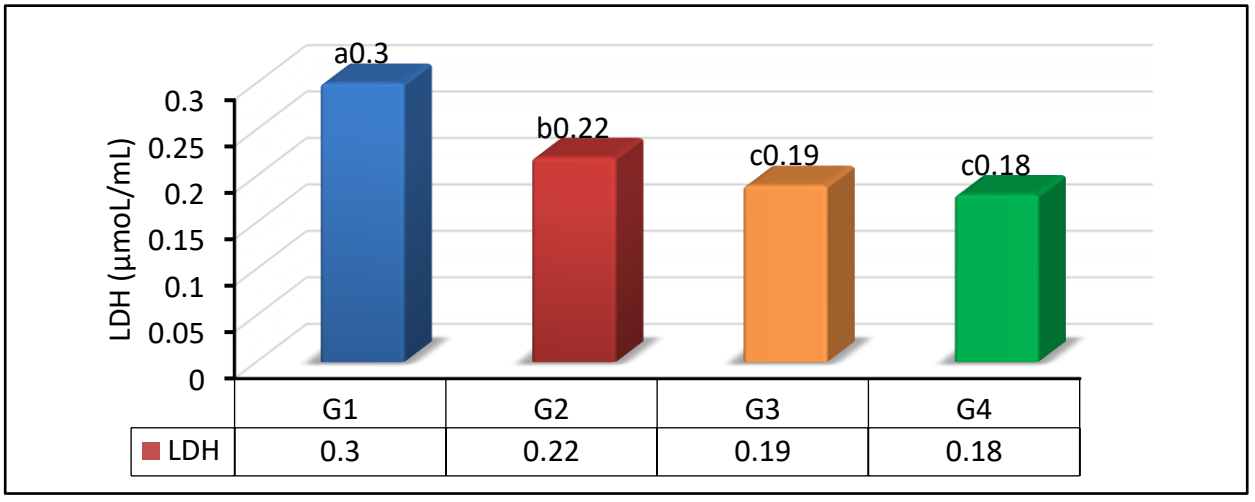

Fig. 5. LDH levels in the study groups.

4.4 SOD and GSH levels

The results are shown in Figures 6,7, a high, significant decrease in the levels of SOD and GSH in recently recovered, mid-addicts. Recovery. In contrast, the (SOD and GSH) values in the long-term recovering addicts group indicate a return to the physiological level, showing no significant difference compared with the control group, with a value of (G3).

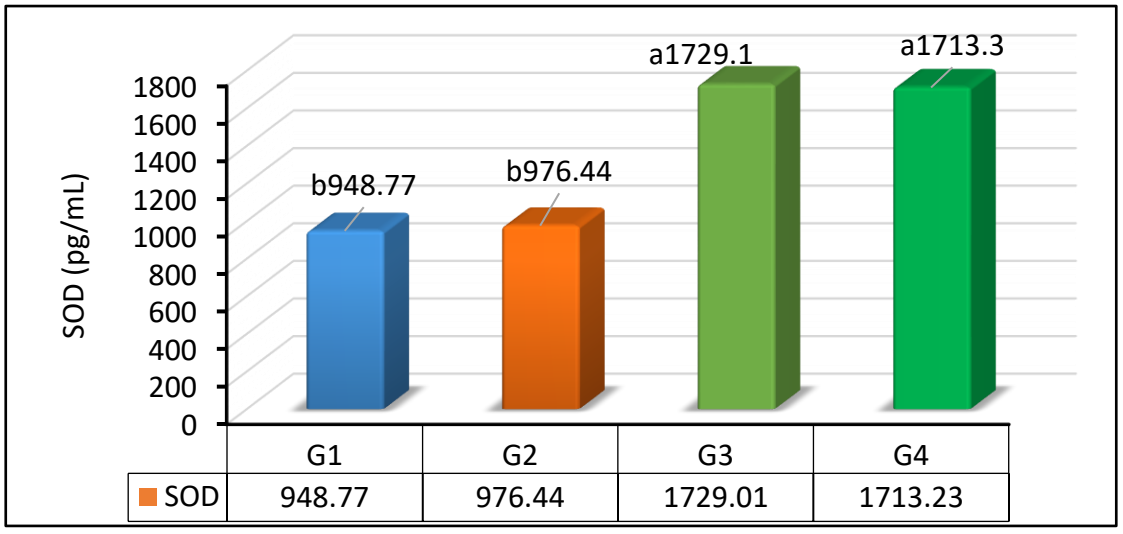

Fig. 6. SOD levels in the study groups.

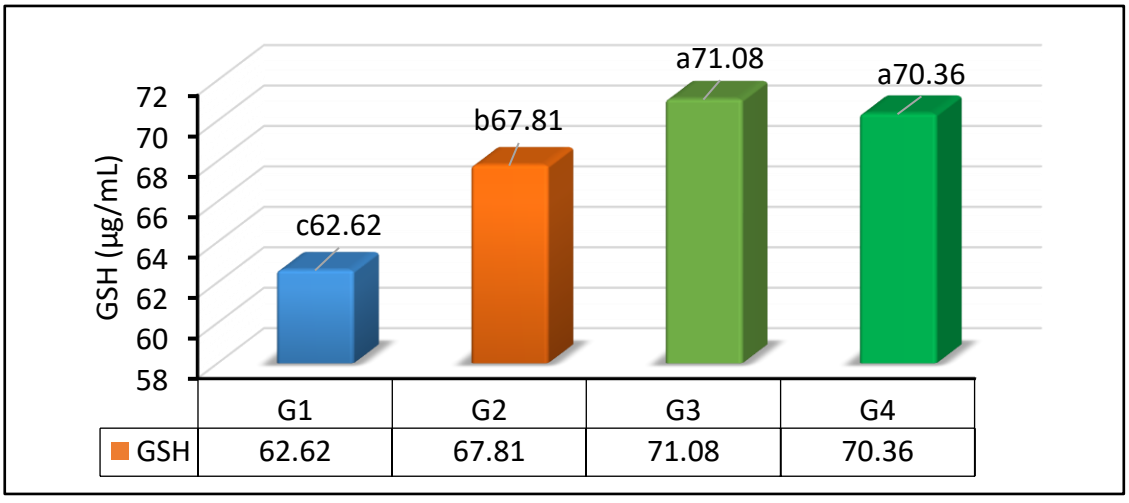

Fig. 7. GSH levels in the study groups.

5. DISCUSSION

5.1 Neurotransmitters

The study results show a clear decrease in dopamine levels compared with the control group, reflecting a sustained reduction in brain dopamine system activity due to long-term inhibition resulting from chronic substance use. This is in line with the findings of the study by [14]. Indicated a clear dysregulation of dopamine enzyme receptors (DAT) in stimulant users, leading to suppression of reward system functions. [15] also showed that the decrease in dopamine levels during the intermediate recovery stage G2 is not a transient phenomenon, but rather a natural consequence of acute depletion of the neural system due to stimulant use. Regular physical activity appears to act as a facilitator for restoring dopamine functions by increasing receptor expression and stimulating neurotrophic factors. It becomes evident that continuous physical intervention represents an effective strategy to enhance chemical neurobalance during the recovery phase. The study results indicated a notable decline in the group of new patients, and previous studies have suggested that serotonin levels are inversely related to stress states and mood disorders. The decrease in serotonin is closely linked to depression and anxiety, especially in the early stages of mental illness, because serotonin is directly affected by psychological stress and an unstable environment [16]. Therapeutically, [17] in their comprehensive review of antidepressants, indicated that restoring serotonin balance requires a relatively significant amount of time after starting treatment, which supports the clear differences between groups according to the duration of residence/community stay.Recent studies indicate that acetylcholine influences cognitive and affective functions, and that disturbances in its levels are linked to neurological and psychiatric symptoms. Picciotto pointed out that a decrease in acetylcholine can affect the regulation of anxiety, attention, and memory, which

may explain the sharp decline in acetylcholine levels among new and mid-stay patients [18]. It also suggests that behavioral and cognitive effects arising from acetylcholine disturbance are often more pronounced in the early stages of exposure to psychological stress. Another study indicated that long-term neural adaptation may lead to relative stabilization of acetylcholine levels, thereby enhancing cognitive performance over time, as reflected in relatively higher levels among long-stay patients [19]. These findings support the idea that the length of hospital stay and the associated changes in the psychological and social environment may directly affect neural chemical balance, particularly concerning acetylcholine, with implications for overall neural health. The results also demonstrate statistically significant differences in neurotransmitter levels among the different groups, indicating a strong association between hospital stay duration and neurochemical changes, which in turn reflects the impact of hospital stay on neurotransmitter regulation and the potential influence on mental and neurological health of patients.

6. ANTIOXIDANT PARAMETERS SOD AND GSH TO THE OXIDATIVE STRESS MARKER MDA.

The study also showed that an increase in MDA in the early stages of the disease could indicate a deficiency in antioxidant defenses, leading to evident cellular damage, especially in unstable patients or those treated for the first time or isolated within the hospital **[20].** The data clearly suggest that both MDA and LDH can be used as genuine biomarkers to assess disease severity, the progression of oxidative stress, and the degree of cellular stability in patients. The elevated values in G1 underscore the importance of early monitoring of oxidative stress and its activation within treatment programs during the initial days of hospital admission.

7. SOD AND GSH

The results align with the study by **[9],** which found that patients with drug use disorders experience a decrease in antioxidants such as GSH, with an increase in oxidative stress markers like MDA, reflecting a clear imbalance in the redox homeostasis, especially in the acute stages.The findings also agree with a study conducted in Georgia that measured the activities of SOD, GPx, and CAT in cases of opioid and alcohol withdrawal, showing that GPx activity is significantly reduced during withdrawal, which reinforces the role of GSH in antioxidant defense during this period **[10].**These results are supported by the clinical findings of Wimberley et al. (2024), who conducted vital research on animal models and linked decreased GSH in the brain to changes in GST enzyme pathways and their effect on neurotransmitters and addictive behavior, reinforcing the view that the GSH level is a precise indicator of progression or regression in addiction states. Furthermore**, [9]** indicates that SOD is a crucial biological marker for assessing oxidative stress, with its activity dependent on the balance of metal ions such as copper and zinc, and its level decreases in conditions of inflammation, infection, prolonged exposure to drugs, or physiological and psychological stress. The results show statistically significant differences in GSH and SOD levels between the different groups. The higher values in the elderly patient group and the control group suggest a role for these enzymes in protecting against oxidative stress. In contrast, the decline in GSH and SOD levels among new patients and those with medium-length stays indicates the impact of hospital admission on antioxidant status, highlighting the need for strategies to bolster cellular protection for these patients, including both external antioxidants and pharmacological interventions, with highly significant differences ($P < 0.001$, HS) in SOD levels between groups. The elevated levels in the elderly patient group and the control group point to a restoration or maintenance of antioxidant capacity. Conversely, the sharp decline in new patients and those with medium stays demonstrates the acute deterioration associated with hospital admission, underscoring the need for nutritional and therapeutic support to stimulate the antioxidant enzymes in these patients**.**

8. CONCLUSION

Independent of the study findings, we conclude that the use of the crystal drug showed a significant effect on the concentration of central neurotransmitters, according to the long-term period of use and the recovery time, and there was a highly significant effect on liver enzymes and oxidative stress status.

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Non-patents or copyrights related to the work.

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