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Evaluation of Some Biochemical Parameters in Iraqi Methamphetamine Addicts

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Abstract

Background: Methamphetamine (METH) is a potent stimulant of the central nervous system and a highly addictive substance. With a severe neurotoxic effect on the central nervous system (CNS), it is a powerful, highly addictive, and frequently abused pharmaceutical medication. The investigation of liver enzymes, like aspartate aminotransferase (AST) and alanine aminotransferase (ALT), is linked to METH addiction, as well as certain elements, such as sodium and calcium, because of their link to elevated neurotransmission brought on by METH addiction. **Objective:** A study of the impact of METH abuse on some biochemical variables in addicted individuals. **Study design:** One hundred and twenty participants were split into three groups for the study: forty men without addiction (R1), who were not smokers or people with long-term illnesses; forty methamphetamine addicts (R2); and forty methamphetamine and other narcotic substance (mixture) addicts (R3). The participants' ages ranged from 18 to 50 years, and their duration of methamphetamine abuse was between 1 and 5 years. **Results:** In comparison to the non-addicted group (R1), the results indicate a significant drop in glucose levels in the addicted group (R2, R3) ($P < 0.05$). The levels of ALT and AST were significantly higher in the addicted group (R2, R3) than in the non-addicted group (R1) ($P < 0.05$). When compared to the non-addicted group (G1), calcium levels were noticeably greater in the addicted group (R2, R3) ($P < 0.05$). The addicted group (R2, R33) had significantly higher sodium levels than the non-addicted group (R1) ($P < 0.05$).

Keywords: Biochemical variables, methamphetamine, methamphetamine addicts

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Introduction

Methamphetamine (METH) is a highly addictive, potent, and widely used pharmacological drug (psychostimulant) with a strong neurotoxic effect on the central nervous system (CNS). METH is neurotoxic to human midbrain dopaminergic neurons [1]. It has also been shown to damage serotonin neurons in the CNS [2].

This damage encompasses adverse changes in brain structure and function, including reductions in grey matter volume in several brain regions and alterations in markers of metabolic integrity [3].

Hyperactivity, dilated pupils, flushing, excessive sweating, increased movement, dry mouth and teeth grinding, headache, irregular heartbeat, rapid breathing, high blood pressure, blurred vision, dizziness, tingling, numbness, tremors, dry skin, and acne are some of the signs and symptoms associated with methamphetamine addiction [4].

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Additionally, there is a strong correlation between METH usage and violent behaviours, amphetamine psychosis, anxiety, depression, and suicide [5].

Meth-induced psychosis shares symptoms with paranoid schizophrenia. These include seeing, hearing, or feeling things that aren't there, acting aggressively, and having delusions. or excessive drug use may exacerbate a preexisting susceptibility to schizophrenia [6]. Glucose is the mammalian brain's main energy source [7], and brain physiology depends on rigorous regulation of glucose metabolism. In line with its critical role in physiological brain function, disruption of normal glucose metabolism and its link to cell death pathways form the pathophysiological basis for many brain disorders [8,9].

Neurons in the brain of an adult have the greatest energy demands [7], necessitating constant blood glucose delivery.

Glucose metabolism provides the energy for physiological brain function through the synthesis of neurotransmitters and ATP, which is the foundation for maintaining both neuronal and non-neuronal cells. Therefore, rigorous regulation of glucose metabolism is necessary for brain physiology, and aberrant glucose metabolism in the brain is the underlying cause of several diseases that affect both the brain and the body [8,10].

Additionally, glucose metabolism provides the energy and precursors for the biosynthesis of neurotransmitters [11]. Importantly, astrocytic glycogen seems to be directly relevant for learning [12].

Many liver enzymes, such as gamma-glutamyl transpeptidase (GGT), alkaline phosphatase (ALP), aspartate transaminase (AST), and alanine transaminase (ALT). A blood test reveals elevated liver enzymes, which signify damaged or inflamed liver cells [13].

Since METH causes cellular damage, addiction, and altered neuronal function, many studies of the substance concentrate on its effects on the central nervous system. However, exposure to METH has also been linked to liver and other organ damage [14]. The recent discovery of a link between METH-induced hepatic injury, elevated peripheral and cerebral ammonia levels, and protracted dopamine and serotonin depletions highlights the importance of peripheral organ damage in mediating the neurotoxicity of METH [15].

The hepatic injury induced by METH has not been comprehensively elucidated in vivo, notwithstanding these observations.

Since METH's hepatotoxicity seems to be a contributing factor to its well-established neurotoxicity, it is important to further characterize and comprehend this hepatotoxicity [16].

Additionally, calcium has a role in the production and release of neurotransmitters, among other neural processes. and the control of neuronal membrane excitability. It regulates neuronal plasticity and metabolic activity, such as phosphorylation and proteolytic activity, and it contributes to the anticonvulsant effect of some drugs [17]. Recent studies have underlined the involvement of this ion in long-term processes, like memory[18], and changes in protein synthesis through the induction of specific genes[19]. When an action potential enters a nerve terminal, Ca+2 channels open, allowing a highly localized, brief rise in intracellular Ca+2 at the active zone, which starts synaptic transmission (Fig. 1-3). Ca+2 initiates synaptic transmission by causing synaptic vesicle exocytosis, which releases the neurotransmitters inside the vesicles [20].

The flow of sodium and potassium within nerve cells creates the electrical potential that permits the conduction of nerve impulses. Potassium alters the membrane potential as it exits the cell, enabling the nerve impulse to spread. For excitable cells, like nerve cells, which rely on this pump to respond to stimuli and transfer impulses, this physiological function is especially important [21].

Numerous medical conditions can arise from abnormal levels of these electrolytes. For instance,

hypernatremia, a disorder characterized by an excessively high sodium content, causes edema (tissue swelling brought on by excessive fluid retention), thirst, and decreased urine production [22].

Materials and Methods

Subjects

This study was conducted at the Psychiatry Teaching Hospital.

One hundred and twenty participants were split into three groups for the study: forty men without addiction (R1), who were Non-smokers and without chronic diseases; forty methamphetamine addicts (R2); and forty methamphetamine and other narcotic substance addicts (R3). The ages of the participants varied from 18 to 50 years, and their history of methamphetamine use spanned from 1 to 5 years. This information was gathered following a clinical assessment and diagnosis conducted at the Teaching Hospital for Psychiatry's laboratories, and the addicted individuals were selected for the study. Screening for methamphetamine use within the last 24 to 48 hours produced good results.

Exclusion Criteria

Subjects who suffered from any chronic diseases, were taking any medication, and subjects who were smoking were excluded from this study. Information was obtained through a reconnaissance paper after obtaining consent from them.

Collection of Blood and Serum Preparation

Ten milliliters of blood were drawn at a random time into a syringe, the blood was allowed to clot for 15-30 minutes, the clot shrank, and serum was obtained by centrifuging for approximately at a relative centrifugal force (RCF) for 15 min at1000×g. the serum specimens collected from these samples were frozen at –20 °C.

Determination of Glucose Concentration (mg/dL).

Determination of glucose in human serum by the Colorimetric method using the Linear Kit according to the manufacturer's instructions.

Determination of ALT&AST (U/l)

ALT and AST measurements in human serum by the Colorimetric method by using the AFCO Kit according to the manufacturer's instructions.

Determination of Calcium Concentration (mg/dL).

Calcium in human serum is measured using the colorimetric method using the AGAPPE Kit in accordance with the manufacturer's instructions.

Determination of Sodium Concentration (mmol/L).

Utilizing the Spin React Kit and the manufacturer's instructions, determine the amount of sodium in human serum using the colorimetric method.

Results and Discussion

As indicated in Table 1, the control group consisted of forty non-addicted men, ages 30.9±7.0 years, and the test group consisted of 80 addicted males, ages 28.2 $\pm$ 6.5 years, who had abused METH for one to five years.

Additionally, the group of addicts was split into two groups: forty were classified as R2 because they were addicted to methamphetamine, and forty were classified as R3 due to their drug addiction, which included metham phetamine.

Glucose Concentration

In comparison to the non-addict group (R1), the results in Table 2 demonstrate a significant reduction in the glucose levels in the group of addic ts (R2, R3) ($P < 0.05$).

Table 1: Addicts' Group Age (Years) and Non-Addicts' Age (Controls)

Age	N	Mean± SD	SE
addicts	80	28.2 ± 6.5	1.07
non addicts	40	30.9±7.0	1.30

Table 2: The Addict Groups' (R2, R3) Glucose Levels concerning the Group Without Addiction (R1)

groups	N	Mean ± SD	SE	Compared groups		P value
R1	**40**	**97.2 + 17.6**	**3.2**	**1**	**2**	**0.0001**
					3	**0.0001**
R2	**40**	**78.8 + 10.7**	**1.9**	**2**	**1**	**0.0001**
					3	**0.84**
R3	**40**	**78.0 + 12.0**	**2.8**	**3**	**1**	**0.0001**
					2	**0.84**

Glucose, a kind of sugar, is the primary energy source for all bodily cells.

The brain uses half of the body's sugar energy, making it the most energy-demanding organ due to its abundance of nerve cells, or neurons.

Glucose metabolism in the brain exhibits marked variations in resting state and is strongly influenced by intrinsic brain activation, indicating a strong link between energy metabolism and brain function [10].

Cognitive functions, including learning, memory, and thought, are intimately correlated with the brain's capacity to use glucose as fuel and with its glucose levels.

For example, the chemical messengers in the brain, known as neurotransmitters, are not produced.

For example, the chemical messengers in the brain, known as neurotransmitters, are not produced, and the neuronal connection is disrupted if there is insufficient glucose in the brain. The primary required substrate for energy metabolism is glucose, even though the brain can use other fuels [23].

In this study, the effect of MATH on glucose levels was investigated. Dr. Lev et al. (2016) found that fasting blood glucose levels in MATH-dependent patients were lower than those in the control group. Additionally, this study demonstrated MA-stimulated insulin secretion through its direct effect on the pancreas [24].

The brain's high energy consumption makes it susceptible to a variety of illnesses if energy sources are stopped, as neurons are typically intolerant of inadequate energy supplies. Numerous conditions affecting the central nervous system can result in, or

occasionally be the cause of, disruptions in the central or peripheral glucose energy metabolism [8].

Activities of Aspartate Aminotransferase (AST) and Alanine Aminotransferase (ALT)

Along with elevated ammonia and changed hepatic function, hepatocellular enzymes AST and ALT are released into the bloodstream when cells are damaged. This is a sign of meth-induced liver damage [15].

Table 3 demonstrates that, in comparison to the non-addict group (G1), the ALT and AST levels in the group of addicts were noticeably higher (R2, R3) ($P < 0.05$).

Because of its euphoric effects, widespread availability, and affordable price, methamphetamine (METH) poisoning is rather common throughout the world [25]. Since METH causes cellular damage, addiction, and altered neuronal function, many studies of the substance concentrate on its effects on the central nervous system. However, exposure to METH has also been linked to liver and other organ damage. Hepatocellular injury may arise from METH's direct impact on the liver and related peripheral effects on cardiac function [26].

The urea cycle, which converts ammonia to urea, is one of the liver's primary roles. Ammonia builds up and can be harmful when liver function is impaired [27].

Similar to METH, ammonia damages neurons by oxidative stress, inflammation, and excitotoxicity. Ammonia increases extracellular glutamate, depolarizes glutamate receptors, and produces excitotoxicity by reducing the expression of the astrocytic glutamate transporter, EAAT-1. [28]. Ammonia also increases glutamate release from astrocytes [29].

Increases in peripheral and brain ammonia have been shown to occur simultaneously with liver damage caused by METH and to have a role in the neuronal damage that is a hallmark of METH [15].

The toxicity of METH combined with its relative accessibility of availability has led to a rise in related medical issues and deaths [30]. According to Y. Kamijo et al. (2002), acute liver failure can result from methamphetamine consumption [31].

The degree of hepatic dysfunction brought on by METH is indicated by elevated levels of the clinical and diagnostic values linked to variations in blood enzyme concentrations like AST and ALT [32].

Finally, the results in this study showed a significant increase in liver enzymes (ALT, AST), which is considered a significant indicator of liver damage.

Calcium Concentration

The healthy operation of the nervous system depends on calcium ions and the preservation of cellular calcium ion homeostasis.

Calcium ions, for instance, are essential for neurotransmission, growth, and development, and they also play a role in the diverse patterns of neuronal gene expression.

Additionally, calcium seems to be necessary for the vast majority of synaptic plasticity that depends on activity, which is typically thought to be the cellular correlate of memory and learning [33].

Table 4 indicates that calcium levels were significantly higher in the addict group (R2, R3) ($P < 0.05$) than in the non-addict group (R1).

Table (3) ALT, AST Levels (U/l), for Addict Groups (R2, R3) concerning the Group without Addiction (R1)

parameters	groups	N	Mean ± SD	SE	Compared groups		P value
ALT (U/L)	R1	40	4.30 ± 1.68	0.30	1	2	0.008
						3	0.012
	R2	40	6.33 ± 3.75	0.68	2	1	0.008
						3	0.889
	R3	40	6.45 ± 2.78	0.62	3	1	0.012
						2	0.889
AST (U/L)	R1	40	6.7 + 1.17	0.21	1	2	0.021
						3	0.001
	R2	40	8.4 + 3.03	0.55	2	1	0.021
						3	0.121
	R3	40	9.7 + 3.9	0.88	3	1	0.001
						2	0.121

Table (4) The Calcium Levels in the Addict (R2, R3) concerning the Group without Addiction (R1)

groups	N	Mean ± SD	SE	Compared groups		P value
R1	40	8.3 + 1.9	0.43	1	2	0.0001
					3	0.0001
R2	40	13.7 + 2.0	0.54	2	1	0.0001
					3	0.55
R3	40	13.5 + 1.9	0.50	3	1	0.0001
					2	0.55

In neurons, variations in intracellular calcium levels serve as signals for a number of different functions. Ca^{+2} is the main neurotransmitter release trigger, and it has been the subject of much research in recent decades [34].

Since calcium ions are a crucial intracellular second messenger that controls critical functions in practically all mammalian cells, they are the most common signal transduction molecule in the body. Calcium, for instance, is essential for normal neural activity [35].

Since it is now widely known that Ca^{+2} plays a crucial role in many physiological processes in the nervous system, it seems sensible that variations in Ca^{+2} within cells levels or Ca^{+2} signalling could be a contributing factor to several diseases.

For instance, it is commonly known that aging causes alterations in Ca^{+2} metabolism and homeostasis [36]. Additionally, METH markedly raised the amounts of calcium in the cytosol.[37] Ca+2 influx into mitochondria and nuclei is caused by an increase in the cytoplasm's Ca^{+2} content. Ca+2 speeds up and interferes with regular metabolism in mitochondria, which results in cell death [38].

Amyotrophic lateral sclerosis and neurodegenerative illnesses, including Huntington's, Parkinson's, and Alzheimer's disease, are exacerbated by changes in calcium levels [39]. Epilepsy and migraine are also influenced by disrupted signalling via Ca+2 channels [40]. Traumatic brain injury and cerebral ischemia are two of the most well-researched conditions thought to involve disrupted Ca+2 homeostasis [41].

Sodium Concentration

The concentration of sodium ions is a great biomarker that is required for cardiac activity, nerve impulse transmission, blood and bodily fluid management, and several metabolic processes.[42] Table (3-5) demonstrates that, in comparison to the non-addict group (R1), sodium levels were significantly higher in the addict group (R2, R3) ($P < 0.05$).

An electrochemical gradient of K+ ions leaving the cell and Na+ ions entering it is created by the Na, K-pumps found in the membranes of the majority of mammalian cells, which pump K+ ions enter the cell and Na+ ions out of it. The pumping process requires an electrochemical gradient of the cations, which is produced by the energy transducer Na, K-pump, which transforms chemical energy from ATP hydrolysis [43].

Active transport is defined as the transport that is directly linked to a chemical process. The transport is electrogenic, meaning that two K+ ions are brought into the cell and three Na+ ions are taken out for every ATP molecule that is hydrolysed.

Addicts in this study had greater salt levels than healthy people (the control group), which could result in health issues as previously noted.

Table (5) The Sodium Levels in the Addict (R2, R3) concerning the Group without Addiction (R1)

groups	N	Mean ± SD	SE	Compared groups		P value
R1	40	132.6 + 11.4	2.4	1	2	0.0001
					3	0.0001
R2	40	156.2 + 25.5	5.2	2	1	0.0001
					3	0.78
R3	40	155.6 + 20.1	4.6	3	1	0.0001
					2	0.78

Conclusion

The current study demonstrated how METH addiction affects clinical parameters that mimic the imbalance in the brain brought on by shifting energy balance, leading to the conclusion that: Neurodegenerative disorders are exacerbated by changes in calcium levels. However, symptoms like weakness, nausea, loss of appetite, and a strong sense of thirst might be brought on by a high blood sodium level.

METH abuse may cause liver damage depending on the duration of meth(years) and the addict's age.

Society needed to be aware of the riskiness of drug use, especially METH, as it causes rapid addiction in addition to many side effects, including an increase in criminal behavior.

Ethical Statements

Originality and Plagiarism: We confirm that this work is original and has not been published elsewhere.

Transparency and Disclosure: It was confirmed that no conflict of personal or financial interest affects the work.

Ethical approval: The research was completed with the approval of the Laboratories Department at Ibn Rushd Training Hospital for Psychiatry.

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