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Assessment of serum trace elements and its association with severity and outcome of Iraqi patients with traumatic brain injury

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ABSTRACT

Trace elements, such as copper and iron, play vital roles in brain metabolism and oxidative stress regulation. This study investigated the role of trace element imbalance, specifically copper and iron, in traumatic brain injury (TBI) severity, type, treatment, and patient outcomes over 3 months. A case-control study was conducted on 60 patients with TBI who were admitted to hospitals in Baghdad, Iraq, within 8 h of injury, along with 40 healthy controls. Serum samples were analyzed using colorimetric methods. Patient outcomes were assessed 3 months after injury in 31 patients. The results revealed significant differences in serum copper and iron levels between patients with TBI and healthy controls. Copper levels also differed significantly between the acute phase and 3 months after injury ($p = 0.001$), whereas iron levels increased but not significantly ($p = 0.788$). Copper levels were notably lower in patients with severe injuries than in those with moderate and mild injuries ($p = 0.001$). Mean copper levels were significantly lower in patients with poor outcomes than in those with good outcomes ($p = 0.002$). The ability of copper levels to discriminate patients with TBI was evaluated using receiver operating characteristic analysis, with an AUC of 0.242 (95% confidence interval [CI], 0.124–0.361). The optimal cutoff value was 91.65 µg/dL, with 11.4% specificity and 91.1% sensitivity. The AUC for iron was 0.342 (95% CI, 0.222–0.462). The optimal cutoff value was 92.3 µg/dL, with 79.4% specificity and 18.2% sensitivity. These findings suggest that altered copper and iron homeostasis may be associated with TBI and may reflect injury severity and patient outcomes.

Keywords: *Cu, Fe, GCS, GOS, TBI, Trace elements*

1 INTRODUCTION

Traumatic brain injury (TBI) is a major global health problem, with an estimated 69 million cases occurring annually worldwide [1]. In developed countries, TBI is one of the most common causes of death and disability among young adults and children [2]. In TBI cases, both primary and secondary brain injuries occur. Primary injury occurs at the time of trauma, whereas secondary injury develops later as a result of subsequent complications [3]. Copper is an essential trace element involved in several biological functions, including cellular respiration, antioxidant defense, and neurotransmitter biosynthesis [4]. It also plays an important role in iron metabolism. Copper is required for the proper function of ceruloplasmin, a copper-containing enzyme that oxidizes ferrous iron (Fe^{2+}) to ferric iron (Fe^{3+}). This oxidation is necessary for iron to bind to transferrin, the protein responsible for transporting iron through the bloodstream [5]. Copper homeostasis is tightly regulated because imbalance can lead to toxicity and cell death [6]. Copper dysregulation has been implicated in several neurodegenerative disorders, such as Alzheimer's disease, Parkinson's disease, and amyotrophic lateral sclerosis,

mainly through oxidative stress mechanisms [7].

Iron is an essential mineral required for several biological processes, including oxygen transport, DNA synthesis, and cellular energy production [8, 9]. Iron homeostasis is tightly controlled because both deficiency and excess can cause health problems. Excess iron can disrupt redox homeostasis, leading to oxidative stress and the generation of reactive oxygen species (ROS), which may contribute to ferroptosis [8]. Ferroptosis is a recently identified form of regulated cell death (RCD) characterized by iron overload and excessive lipid peroxidation [10].

Copper (Cu) and iron (Fe) are necessary for normal brain function; however, when dysregulated, they may contribute to secondary injury in TBI. Copper homeostasis is necessary to maintain neuronal function and prevent neurodegeneration. Altered copper levels may promote neuronal apoptosis, inflammation, oxidative stress, and toxicity [11]. Iron is also implicated in TBI because of its role in neurodegenerative processes, where iron accumulation can exacerbate neurotoxicity and contribute to cognitive and motor impairment [12].

2 MATERIALS AND METHODS

2.1 Sample collection

Samples were collected from patients with traumatic brain injury (TBI) at the Neurosurgery Teaching Hospital, Dr. Saad Al-Witry Neuroscience Hospital, and Al-Kindi Teaching Hospital, Baghdad, Iraq. A total of 100 individuals, including 40 healthy controls and 60 patients with TBI, were recruited and provided consent to participate in this study. The TBI patients were 18–63 years old and had mild, moderate, or severe injury according to the Glasgow Coma Scale (GCS) [13].

The first blood sample was collected within 8 h of head injury, and the second blood sample was collected 3 months after injury from 20 patients. A 10 mL blood sample was collected from each enrolled participant and centrifuged to isolate serum. Serum aliquots were stored at -43 °C until analysis. Patients were excluded if they were younger than 18 years, had a previous head injury, had known dementia, Alzheimer's disease, multiple sclerosis, or other central nervous system (CNS) pathology, were pregnant, or had polytrauma.

2.2 Patient assessments and classification

Patients enrolled in this study, aged 18–63 years, were classified according to five categories: type of injury based on initial computed tomography (CT) findings, including focal, diffuse, or combined focal and diffuse injury; mechanism of injury, including fall, violence, traffic accident, or other causes; clinical severity according to the GCS, including mild injury (GCS 13–15), moderate injury (GCS 9–12), and severe injury (GCS 3–8) [13]; treatment type, including surgical or conservative treatment; and patient outcome within 3 months after injury, assessed using the Glasgow Outcome Scale (GOS) [14]. Good outcome was defined as GOS 4–5, whereas poor outcome was defined as GOS 1–3. In the poor-outcome group, the patients included in this study had GOS = 1, representing non-survivors. Three months after injury, blood was redrawn from 20 patients with good outcomes, and serum iron and copper levels were remeasured.

2.3 Serum copper and iron assessment

Cupric ions react with the chromogen 4-(3,5-dibromo-2-pyridylazo)-N-ethyl-N-(3-sulfopropyl) aniline (Di-Br-PAESA) to form a blue compound, the intensity of which is proportional to the copper concentration in the sample [15]. Copper was assessed using the LTA copper kit, Italy. The working reagent was prepared by mixing equal quantities of reagent A and reagent B. The blank, standard, and sample tubes were prepared by adding 1 mL of working reagent and 66 µL of distilled water, standard, or sample, respectively. The tubes were mixed well and incubated for 10 min, and the absorbance was then read against the blank at 580 nm using a Humalyzer Primus.

Serum iron was determined using a photometric colorimetric method. Iron (III) reacts with chromazurol B (CAB) and cetyltrimethylammonium bromide (CTMA) to form a colored ternary complex with maximum absorbance at 623 nm. The intensity of the produced color is directly proportional to the iron concentration in the sample [16]. Iron was assessed using a Human iron kit, Germany. Briefly, 1000 µL of reagent was added to 50 µL of distilled water to prepare the reagent blank, and 50 µL of sample or standard was added for measurement. The mixture was incubated for 15 min at room temperature, and absorbance was read using a Humalyzer Primus, Germany.

2.4 Statistical analysis

The results are presented as mean ± SD. An independent t-test was used to compare age and other continuous clinical parameters between groups. One-way ANOVA followed by Tukey's post hoc test was used to compare copper and iron levels among patients and controls, injury severity groups based on GCS, and injury-

type groups. The ability of copper and iron levels to discriminate patients with TBI was assessed using the area under the receiver operating characteristic curve (AUC). According to current statistical interpretation, AUC values of 0.9 – 1.0 were considered excellent, 0.8-0.9 good, 0.7-0.8 fair, 0.6-0.7 poor, and 0.5-0.6 failed discrimination. All analyses were performed using SPSS version 23 .

3 RESULTS

A total of 100 individuals were enrolled in the study, and serum samples were obtained for analysis. The study included 60 patients with TBI and 40 healthy controls. Of the 60 patients, 31 were followed up 3 months after injury. Among them, blood was redrawn from 20 patients for repeated measurement of trace elements and GOS assessment, whereas 11 patients died within 3 months of injury. Patient characteristics and injury severity are shown in Table 1.

Table 1 Demographics of patients with traumatic brain injury and healthy controls.

Characteristics	**TBI patients** (n = 60)	**Controls** (n = 40)
Age, year, mean ± SD	35.07 ± 14.736	37.44 ± 14.590
Gender, n (%)		
Male	52 (86.7)	29 (71.4%)
Female	8 (13.3)	11 (28.6%)
Clinically by GCS		
Mild	34 (57.1)	-
Moderate	14 (23.8)	-
Severe	12 (19.0)	-
Mechanism of injury		
Fall	19 (31.1)	-
Traffic	33 (55.6)	-
Violence	7 (11.1)	-
Others	1 (2.2)	-
Type of injury		
Focal	39 (64.3)	-
Diffuse	17 (28.6)	-
Combination [I]	4 (7.1)	-
Treatment		
Surgery	11 (17.9)	-
Conservative [II]	49 (82.1)	-
Outcome		
Good outcome [III]	20 (33.3)	-
Poor outcome [IV]	11 (18.3)	-

Note. Values are in mean ±SD and n(%). GCS, Glasgow Coma Scale (mild GCS 13–15, moderate GCS 9–12, severe GCS 3–8); [I] Combination; both focal and diffuse injuries; [II] Conservatives; no surgery treatment; [III] Good outcome; the GOS (Glasgow Coma Scale) ≥ 4; [IV] poor outcome GOS ≤ 3.

There was no significant difference in age between patients and controls (p = 0.486). Table 1 shows that males represented a higher proportion of TBI cases than females, with 86.7% males and 13.3% females. Table 2 presents the clinical parameters of patients with TBI and healthy controls. WBC count was significantly elevated in patients with TBI compared with healthy controls (15.35 ± 6.03 vs. $7.84 \pm 1.64 \times 10^9$/L).

Table 2 Laboratory parameters of patients with traumatic brain injury and healthy individuals.

Clinical parameters	**TBI Patients**	**Controls**	P**-value**
WBC ($\times 10^9/\mu$L)	15.35 ± 6.03	7.84 ± 1.64	0.001
RBC ($10^6/\mu$L)	4.84 ± 0.55	5.30 ± 0.52	0.106
HB (g/dL)	14.86 ± 2.02	13.63 ± 1.33	0.158
HCT (%)	44.82 ± 10.12	38.00 ± 3.46	0.135
Plt ($10^3/\mu$L)	219.48 ± 88.81	240.25 ± 103.500	0.811

Note. Values are in mean ±SD. $P > 0.05$ non significant, $p \leq 0.05$ significant, $p \leq 0.001$ highly significant. WBC; white blood cell, RBC; red blood cell, HB; hemoglobin, HCT; Hematocrit, plt; platelets.

As shown in Table 3, patients with TBI had significantly lower serum copper levels during the acute phase ($113.715 \pm 21.061\mu$ g/dL) compared with healthy controls ($131.825 \pm 21.979\mu$ g/dL; p = 0.001) and patients assessed 3 months after injury ($133.975 \pm 19.956\mu$ g/dL; p = 0.040). However, no significant difference in copper levels was observed between patients assessed 3 months after injury and healthy controls (p = 0.964).

Serum iron levels in patients with TBI during the acute phase (67.250 ± 24.453 μg/dL) were significantly lower than those in healthy controls (82.912 ± 26.595 μg/dL; p = 0.028). At 3 months after injury, serum iron levels in patients with TBI ($73.888 \pm 33.352\mu$ g/dL) showed partial recovery but remained statistically comparable to both acute-phase TBI patients (p = 0.787) and healthy controls (p = 0.656).

Figure 1 illustrates the mean levels of serum copper and iron (µg/dL) in three groups: patients with TBI within 8 hours of injury, healthy controls, and TBI patients after 3 months follow-up. Serum copper and iron levels varied across TBI patient subgroups, as shown in Table 4. Copper levels decreased with increasing injury severity, from 116.186 ± 7.274 μg/dL in mild cases to $93.452 \pm 5.620\mu$ g/dL in severe cases. In contrast, iron levels showed an increasing trend, from $64.039 \pm 22.764\mu$ g/dL in mild cases to $77.225 \pm 26.327\mu$ g/dL in severe cases.

Table 3 Multiple comparisons of serum copper and iron levels in traumatic brain injury patients, 3-months follow-up, and healthy controls.

Biomarkers	Patients with TBI (n = 60)	Healthy individuals (n = 40)	Patients after 3 months (n = 20)
Copper (μg/dL)	113.71521.061	131.82521.979	133.97519.956
Iron (μg/dL)	67.25024.453	82.91226.595	73.88833.352
			***P*-value**
Copper (μg/dL)	Patients	Controls	**0.001**
		Patients after 3 months	**0.040**
	Controls	Patients	**0.001**
		Patients after 3 months	0.964
Iron (μg/dL)	Patients	Controls	**0.028**
		Patients after 3 months	0.787
	Controls	Patients	**0.028**
		Patients after 3 months	0.656

Note. Values are in mean $\pm SD$, $P > 0.05$ non significant, $p \leq 0.05$ significant, $p \leq 0.001$ highly significant.

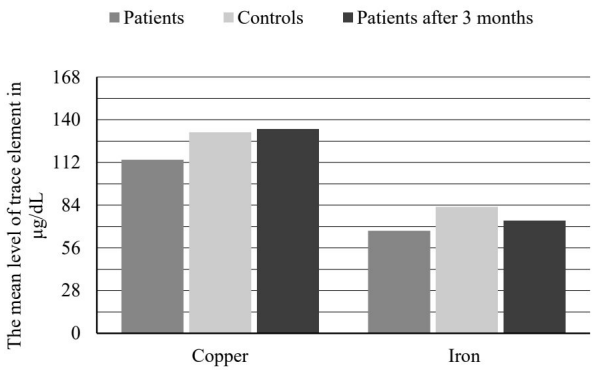

Fig. 1 Mean levels of serum copper and iron.

Differences were also observed according to the mechanism of injury. Patients with violence-related injuries had the lowest iron levels ($62.680 \pm 26.135\mu$ g/dL), whereas patients with fall-related injuries showed lower copper levels (106.618 ± 10.872 μg/dL). Regarding injury type, patients with combined focal and diffuse injuries showed higher copper ($112.133 \pm 0.339\mu$ g/dL) and iron levels (93.150 ± 7.420 μg/dL) compared with those with focal or diffuse injuries.

Patients with poor outcomes had substantially lower copper levels than those with good outcomes (102.993 ± 12.682vs.$146.475 \pm 43.765\mu$ g/dL). However, iron levels were slightly higher in the good-outcome group than in the poor-outcome group (73.887 ± 33.352vs.$68.028 \pm 27.041\mu$ g/dL).

Table 4 Serum levels of copper and iron by clinical characteristics in patients with traumatic brain injury.

Characteristics	Copper (µg/dL) MeanSD	Iron (µg/dL) MeanSD
Clinically by GCS		
Mild	116.1867.274	64.03922.764
Moderate	110.3327.438	66.65030.194
Severe	93.4525.62	77.22526.327
Mechanism of injury		
Fall	106.61810.872	64.23626.879
Traffic	112.69012.908	71.88724.819
Violence	136.72051.248	62.68026.135
Others	123.700	43.400
Type of injury		
Focal	110.06912.655	55.55322.223
Diffuse	110.25513.356	73.83728.099
Combination[I]	112.7400.339	93.1507.424
Treatment		
Surgery	108.41811.915	60.60026.321
Conservatives [II]	112.13310.158	67.21024.548
Outcome		
Good outcome [III]	146.47543.765	73.88733.352
Poor outcome [IV]	102.99312.682	68.02827.041

Note. GCS, Glasgow Coma Scale (mild GCS 13-15, moderate GCS 9-12, severe GCS 3-8); ^{I}. Combination; both focal and diffuse injuries ^{II}. Conservatives; no surgery treatment ^{III}. Good outcome; the GOS (Glasgow Coma Scale) ≥ 4 ^{IV}. poor outcome GOS ≤ 3.

Table 5 shows a significant decrease in serum copper levels in patients with severe injuries compared with those with mild and moderate injuries (p = 0.001). However, no significant differences in serum iron levels were observed across GCS severity groups. The results also showed no significant differences in serum copper or iron levels according to injury type or treatment subgroup. Serum copper levels were significantly lower in patients with poor outcomes than in those with good outcomes (p = 0.002). To assess the diagnostic performance of copper and iron, the area under the receiver operating characteristic curve (AUC) was calculated for each marker to discriminate patients with TBI from healthy controls, as shown in Figures 2 and 3 and Table 6.

Pearson's correlation test was used to assess the correlations among the parameters shown in Table 7. No significant correlation was observed between serum iron and copper levels or between serum iron levels and WBC count. However, a significant correlation was observed between serum copper levels and WBC count (p = 0.012).

Table 5 Statistical analysis of serum levels of copper and iron in patients with traumatic brain injury.

Biomarkers	The characteristics		P-value
Clinically, GCS			
Copper (µg/dL)	Mild	Moderate	0.082
		Severe	**0.001**
	Moderate	Mild	0.082
		Severe	**0.001**
	Severe	Mild	**0.001**
		Moderate	**0.001**
Iron (µg/dL)	Mild	Moderate	0.950
		Severe	0.423
	Moderate	Mild	0.950
		Severe	0.650
	Severe	Mild	0.423
		Moderate	0.657
Type of injury			
Copper (µg/dL)	Focal	Diffuse	0.999
		Combination	0.957
	Diffuse	Focal	0.999
		Combination	0.966
	Combination	Focal	0.957
		Diffuse	0.966
Iron (µg/dL)	Focal	Diffuse	0.191
		Combination	0.104
	Diffuse	Focal	0.191
		Combination	0.565
	Combination	Focal	0.107
		Diffuse	0.565
Treatment			
Copper (µg/dL)	Surgery		0.417
	Conservatives		
Iron (µg/dL)	Surgery		0.982
	Conservatives		
Outcome			
Copper (µg/dL)	Poor outcome		**0.002**
	Good outcome		
Iron (µg/dL)	Poor outcome		0.715
	Good outcome		

Note. GCS, Glascow Coma Scale (mild GCS 13-15, moderate GCS 9-12, severe GCS 3-8) ; Combination; both focal and diffuse injuries, Conservatives; no surgery treatment, Good outcome; the GOS (Glasgow Coma Scale) ≥ 4, poor outcome; mortality or GOS ≤ 3 $P > 0.05$ non significant, $p \leq 0.05$ significant, $p \leq 0.001$ highly significant

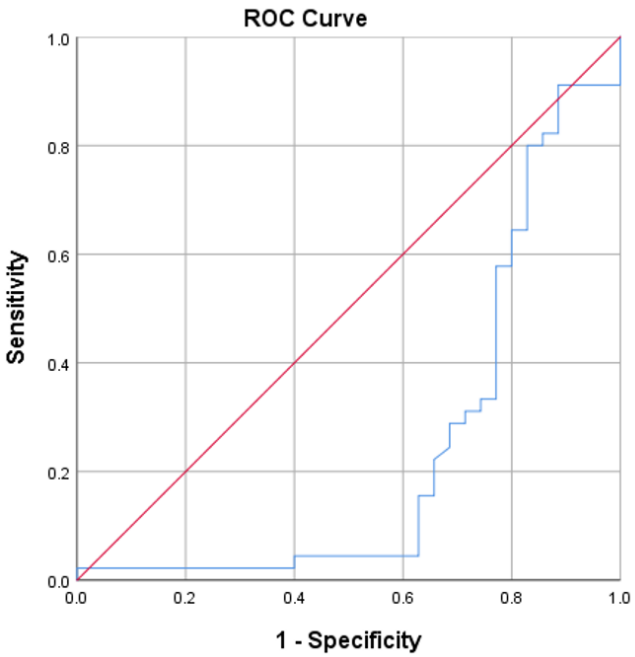

Fig. 2 ROC curve of Copper

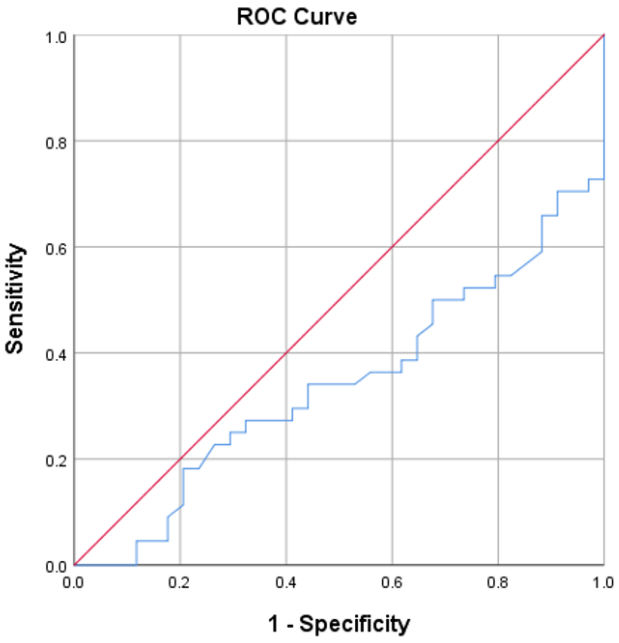

Fig. 3 ROC curve of Iron

Table 6 Receiver Operating Characteristics of copper and iron in the Traumatic Brain Injury.

Parameters	AUC	SE	Sensitivity (%)	Specificity (%)	95% -CI	Cut-off Value	*P*-value
Fe	0.342	0.061	18.2	79.4	0.222-0.462	92.300	0.017
Cu	0.242	0.060	91.1	11.4	0.124-0.361	91.650	<0.001

Table 7 Correlations between biochemical parameters in traumatic brain injury.

Correlation between	TBI patients r-value	*P*-value	Sig.
Copper & Iron	-0.141	0.360	N.S
WBC & Iron	0.139	0.375	N.S
Copper & Iron	-0.374*	0.012	S

Note. *. Correlation is significant at the 0.05

4 DISCUSSION

According to our clinical data, most patients with TBI were males, and the mortality rate within 3 months after injury was approximately 18%. In addition, mortality was higher among males than females and increased with age. Similar findings have been reported in several studies, in which mortality increased with age and was higher among males with TBI [17, 18].

Copper is a cofactor for superoxide dismutase (SOD), an enzyme essential for neutralizing reactive oxygen species (ROS) generated after TBI [19]. Serum copper may be depleted because it is mobilized into tissues after injury. Animal models have shown increased brain copper uptake after TBI, which is correlated with SOD activity and neuronal repair processes [20]. In addition, blood–brain barrier (BBB) disruption in TBI may allow copper to redistribute from serum into the brain parenchyma. Studies in rodent TBI models have demonstrated a transient decrease in serum copper levels alongside increased cerebral copper levels [21]. These findings are consistent with the study by Johary et al., who measured copper levels in 89 cases and 102 healthy controls and reported significantly lower serum copper levels in patients than in healthy controls (p = 0.001) [22]. Essa et al. also reported similar findings after measuring serum copper in 60 patients with severe head injury [23].

Our results showed that serum iron levels were significantly lower in patients with TBI than in controls (p = 0.028). The reduction in serum iron levels after TBI may result from several interrelated mechanisms. Pro-inflammatory cytokines, particularly interleukin-6 (IL-6), stimulate hepatic hepcidin upregulation. Hepcidin is a protein that maintains iron balance in the body [24] by promoting ferroportin degradation, which sequesters iron within macrophages and hepatocytes and limits systemic iron availability [25]. Concurrently, iron may be redistributed to the injured brain to support reparative processes, such as myelination, neurotransmitter synthesis, and energy metabolism. This redistribution may be facilitated by BBB disruption and transferrin receptor-mediated uptake; therefore, increased iron uptake by the brain may reduce serum iron levels [26]. Finally, systemic inflammation activates the acute-phase response, diverting iron into storage proteins, such as ferritin, as part of a nutritional immunity strategy that restricts pathogen access. Moreover, elevated oxidative stress may increase iron demand for antioxidant enzymes, such as catalase, and reduce circulating reserves [27].

Interestingly, Spearman's correlation test showed a significant negative correlation between WBC count and serum copper levels (r = -0.374, p = 0.012), as shown in Table 7. TBI triggers a systemic inflammatory response, leading to the release of pro-inflammatory cytokines, such as IL-6 and tumor necrosis factor-alpha (TNF-α). These cytokines act as signaling molecules that stimulate leukocyte migration to the site of injury to combat infection and facilitate tissue repair [28]. During inflammation, copper may be redistributed from serum to tissues, including the brain, to support antioxidant defenses [29].

No significant correlation was observed between serum iron levels and WBC count. However, a previous study identified an inverse correlation between iron levels and leukocyte count, supporting the established relationship between serum iron and inflammatory processes. Inflammatory cytokines can influence iron homeostasis and metabolism in neurological disorders. These findings suggest that early cytokine-induced changes in iron metabolism may contribute to decreased serum iron concentrations [30].

Table 3 shows that both serum iron and copper levels increased 3 months after TBI. The patients who were followed up after 3 months had good outcomes, defined as GOS 4–5. Copper levels differed significantly between acute-phase patients and patients assessed 3 months after head injury. This improvement may be due to resolution of the acute inflammatory response and restoration of systemic homeostasis. In addition, BBB repair may limit metal leakage into the brain, while reduced oxidative stress may decrease the demand for metal-dependent

antioxidant enzymes, such as SOD and catalase [31].

Based on Table 5, no significant differences in serum copper or iron levels were observed between treatment subgroups, including surgical and conservative treatment, or among injury types, including focal, diffuse, and combined focal and diffuse injuries. In addition, no significant differences in serum iron levels were observed among injury severity groups, including mild, moderate, and severe TBI, or between patient outcome groups within 3 months after head injury.

Serum copper levels were significantly lower in patients with severe TBI (GCS 3–8) than in those with mild TBI (GCS 13–15) and moderate TBI (GCS 9–12). These results agree with the study by Rashid et al., which included 34 patients with head injury who were assessed within 8 h of trauma and reported significantly lower serum copper levels in severe head injury than in moderate head injury ($p < 0.001$) [32].

The results showed that serum copper levels were associated with 3-month mortality in patients with TBI ($p = 0.002$). Serum copper levels were significantly lower in patients with poor outcomes, defined as death within 3 months after TBI (GOS 1), than in patients with good outcomes (GOS 4–5). This finding does not agree with the study by Ankita et al., which included 64 patients with severe TBI and 22 healthy controls to investigate heavy metals and trace elements. They found no significant difference in serum copper levels between survivors and non-survivors during the first, second, and third days of admission ($p > 0.05$) [33].

Despite statistically significant differences, the low AUC values indicate that both Fe and Cu performed poorly as diagnostic markers for TBI based on ROC analysis, as shown in Table 6. These markers may be involved in TBI pathophysiology, but they are not clinically reliable as standalone diagnostic tools.

5 CONCLUSION

This study provides evidence that serum copper and iron levels are altered in patients with traumatic brain injury and may be associated with injury severity and 3-month outcomes. Lower serum copper levels were associated with greater injury severity and poor outcome within 3 months after head injury, whereas significantly decreased serum iron levels were observed in patients with TBI compared with healthy controls. However, because copper and iron showed low diagnostic performance based on ROC analysis, they should not be considered reliable standalone diagnostic biomarkers for TBI. Further studies with larger sample sizes and serial measurements are needed to clarify the role of trace element homeostasis in TBI progression and outcome prediction.

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Author contributions

Zahraa H. Alabasi: Methodology, Investigation, Formal analysis, Data curation, Resources, Writing–review editing, Funding acquisition. Abdulnasser M. Al-Gebori: Conceptualization, Writing–review editing, Supervision, Project administration. Mohammed Ameen Ahmed Al-Rawi: Methodology, Investigation, Resources, Project administration.

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Data availability

N/A

DECLARATIONS

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could affect the work described in this article.

Consent to publish

N/A

Ethical approval

Ethical approval for this study was obtained in accordance with the local regulations in Iraq. Formal written permission were granted by Neurosurgery Teaching Hospital, Dr. Saad Al-Witry Neuroscience Hospital, and Al-Kindi Teaching Hospital, which authorized the collection and use of blood samples for research purposes.

All procedures were conducted in accordance with ethical standards for Iraqi Ministry of Health and the principles of the Deceleration of Helsinki. Patients confidentiality and privacy were strictly maintained throughout the study.

Abbreviation

CNS Central nervous system; CT Computed tomography; Cu Copper; Fe Iron; GCS Glasgow Coma Scale; GOS Glasgow Outcome Scale; IL-6 interleukin-6; RCD Regulated cell death; ROS Reactive Oxygen Spacies; SOD superoxide dismutase; TBI Traumatic brain injury; TNF-α tumor necrosis factor alpha.

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