

Original Article

Hepatic and Renal Function Tests and Routine Hematological Markers in Patients with Cerebrovascular Accident and Transient Ischemic Attack

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Abstract


Background and Aim: Cerebrovascular accident (CVA), also known as stroke, is a vascular syndrome that is the second leading cause of death in the world after ischemic heart disease. Transient ischemic attacks (TIAs) is a warning sign for CVA so that 20% of patients with TIA experience a subsequent stroke within 90 days. Hence, identification of laboratory markers is crucial for the prognosis and diagnosis of patients with CVA and TIA. Changes in some laboratory markers occur in patients with CVA and TIA. Thus, the present study aimed to evaluate hepatic and renal function tests and routine hematological markers in patients with CVA and TIA.

Methods: The present study was a cross - sectional analytical study. The study population was patients with CVA and TIA who were hospitalized in Abadan and Khorramshahr educational hospitals from March 21, 2019, to March 19, 2020. One hundred patients with CVA and one hundred patients with TIA were randomly selected and the necessary information (age, sex, liver enzymes, renal function tests, FBS, and routing hematologic markers including CBC, ESR, PT and PTT) of the patients was collected from HIS (Hospital Information System) of Abadan and Khorramshahr educational hospitals. Liver, kidney and hematologic diagnostic markers were evaluated by age and gender. Data analysis was performed using a t-test (to compare gender difference of laboratory markers in CVA and TIA groups) and one-way ANOVA (to compare laboratory markers among age groups of patients with CVA and TIA). Simple linear regression was used to examine the relationships between changes in FBS (mg / dl) and changes in laboratory diagnostic factors.

Results: The results of this study showed that the highest frequency of patients with CVA (n= 130, 27.3%) and TIA (n= 49, 23.8%) was observed in the age group of 55-64 years. The results indicate that the mean level of some laboratory markers such as FBS (CVA: 174.32 ± 105.83; TIA: 150.32 ± 83.32), creatinine (CVA: 1.37 ± 1.32; 1.42 ± 1.09), LDH (CVA: 696.29 ± 344.90; TIA: 538.17 ± 230.76), and ESR (CVA: 52.41 ± 37.61; TIA: 14.00 ± 8.40) was higher than the normal range in both CVA and TIA. The mean of SGOT (34.10 ± 26.40 IU / L) and ALK (331.44 ± 370.78 IU / L) enzymes were higher than normal only in CVA patients and the mean of SGPT (33.08 ± 38.55 IU / L) was higher than normal only in TIA patients. It was also observed that in patients with CVA, with each unit increase in FBS, a significant increase occurs in K⁺ level (P < 0.001), WBC (P= 0.003), and RBC (P= 0.031) count, as well as a significant decrease in Na⁺ level (P= 0.008).

Conclusion: The results of the present study showed an increase in the level of FBS, ESR and LDH both in CVA and TIA. While SGOT and ALK increased only in CVA patients, SGPT showed an increase only in TIA patients.

Keywords: Stroke; Transient Ischemic Attack; Laboratory Markers; Fasting Blood Sugar; Creatinine

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Introduction

The cerebrovascular accident (CVA), also known as stroke, is defined as a vascular syndrome with rapidly developing signs of global or focal disturbance of cerebral functions lasting more than twenty-four hours or leading to death (1). Stroke is currently the second leading cause of death in the world after ischemic heart disease. According to the reports, stroke and ischemic heart disease together were responsible for 15.2 million deaths in 2015 (2). Transient ischemic attack (TIA) (3) is defined as an episode of focal cerebral dysfunction, probably with the ischemic origin, lasting less than twenty - four hours, followed by disappearance of symptoms (4). Stroke includes two major categories: ischemic and hemorrhagic. Hemorrhagic stroke is caused by the fragility of blood vessels that leads to draining in neighboring brain tissues. The major causes of hemorrhagic stroke are trauma, hypertension, aneurysm, abnormal blood vessel, and use of cocaine. Ischemic stroke, another type of stroke, is caused by the blockage of blood supply in the brain due to the presence of blood clots (5).

The incidence rate of CVA in Iran is 23 to 103 per 100000 persons in different age groups (6). The mortality rate in central regions of Iran and in-hospital death is 24.6% and 20% respectively (7). Both of them are higher than those rates in Western countries. This high incidence can be explained by the under - diagnosis of stroke in hospitals which leads to late referral (7).

Laboratory markers such as white blood cell (WBC) count, C - reactive protein (CRP), and erythrocyte sedimentation rate (ESR) increase in patients with stroke (8). There is also evidence that platelets contribute to the pathogenesis of stroke, and platelet consumption in the acute phase leads to a decrease in platelet count (9). Some studies have shown a post - stroke increase in liver function markers such as SGOT or AST (Aspartate transaminase) and SGPT or ALT (Alanine aminotransferase) (10). Some other studies have shown a change in renal function and its markers such as blood urea nitrogen (BUN) and creatinine, and specially BUN / Cr ratio (11).

Evaluation of serum levels of the mentioned laboratory markers can be an economical, simple, and easily available method for assessing prognosis, outcome, and even quality of response to treatment

(11-15). The results of a study have shown that in patients with intracerebral hemorrhage, the average creatinine clearance measured during the study period is higher compared to the average calculated creatinine clearance. It was also shown that fifty percent of participants with intracerebral hemorrhage experienced at least one day of increased renal clearance (11). The results of studies showed that mean platelet volume (MPV) and platelet count (PC) were observed as independent risk factors for ischemic stroke. There was a negative and significant correlation between PC and hemorrhagic stroke. As a result, MPV may be an early and important predictor for the prognosis of ischemic stroke, while for hemorrhagic stroke; PC also plays an important role in the outcome (13). It has been shown that decrease in the level of alanine aminotransferase (ALT) increases the risk of death in the elderly after ischemic stroke, so that the significant decrease in the level of this liver marker at the time of diagnosis is an independent risk factor that increases the death rate in elderly people after a stroke (14). Considering the importance of laboratory diagnostic markers in the prognosis, and diagnosis of patients with cerebrovascular accidents and transient ischemic attacks and considering the lack of a comprehensive study that examines blood, liver, and kidney markers at the same time, in this study we decided to examine hepatic, renal and hematological laboratory markers in patients with cerebrovascular accident and transient ischemic attack.

Methods

Study Protocol

The present study is a cross - sectional analytical study. After obtaining ethical permission from the ethics committee of Abadan University of Medical Sciences (Ethics code: IR.ABADANUMS.REC.1399.033) and the permission of the head and hospital officials to access the patients' files, the patients' information was collected, and the confidentiality of the information was observed. The study population was patients with CVA and TIA who were hospitalized in Abadan and Khorramshahr educational hospitals from March 21, 2019, to March 19, 2020. The inclusion criteria in this study included patients with CVA and TIA who were admitted to Abadan and Khorramshahr educational hospitals from March 21, 2019, to March 19, 2020, with the diagnosis of a specialist doctor and the information about their

laboratory diagnostic markers such as liver, kidney, heart, and coagulation markers was available in HIS. The information was recorded in the checklist and then entered into Excel software. These patients were excluded from the study if the information was incomplete or unavailable. From this community of patients (476 patients with CVA and 206 patients with TIA), 100 patients with CVA and 100 patients with TIA were randomly selected. The necessary information was collected from HIS (Hospital Information System) by referring to the medical records department of Abadan and Khorramshahr educational hospitals. The information included hematologic factors (Lymphocytes [lymph], White Blood Cells [WBCs], Neutrophils [Neut], Red Blood Cells [RBCs], Mean Corpuscular Volume [MCV], Mean Corpuscular Hemoglobin [MCH], Mean Corpuscular Hemoglobin Concentration [MCHC] Hemoglobin, Hematocrit [HCT], Platelets), liver function markers (alkaline phosphatase [ALK], lactate dehydrogenase [LDH], SGOT or AST and SGPT or ALT), and renal function markers (Blood Urea Nitrogen [BUN] and Creatinine [Cr], serum sodium [Na] and serum potassium [K]). Information was sorted by sex, age, date of admission, and laboratory markers and recorded in Excel software. Then, duplicate data were eliminated.

Statistical Analysis

Patients' information and laboratory findings were obtained from HIS and entered into Excel. The normality of quantitative variables was assessed using the Kolmogorov - Smirnov test. Continuous variables were reported as mean \pm standard deviation and categorical variables were reported as numbers and percentages. Data analysis was performed using a t - test (to compare gender difference of laboratory markers in CVA and TIA groups) and one-way ANOVA (to compare difference of laboratory markers among age groups of patients with CVA and TIA). Simple linear regression was used to examine the relationships between changes in FBS (mg / dl) and changes in other laboratory diagnostic factors. All statistical analyses were performed with SPSS (version 16). $P < 0.05$ was considered significant.

Results

Laboratory Findings in Patients with CVA

The total number of patients with CVA in this study was 476, of which 240 (50.4%) were women and 236 (49.6%) were men. The highest frequency of CVA cases was observed in the age group of 55-64 years (n= 130, 27.3%). However, those aged 15-24 years old and 5-14 years old had the lowest numbers of patients among CVA patients (n= 1, 2%) (Table 1).

Table 1. Frequency and percentage of CVA and TIA patients by age groups

Age (In years old)	CVA	TIA
	Frequency (n), percentage (%)	Frequency (n), percentage (%)
5-14	1 (2)	3 (1.5)
15-24	1 (2)	1 (0.5)
25-34	12 (2.5)	8 (3.9)
35-44	36 (7.6)	28 (13.6)
45-54	68 (14.3)	36 (17.5)
55-64	130 (27.3)	49 (23.8)
65-74	119 (25)	48 (23.3)
>75	109 (22.9)	33 (16)
Total	476 (100)	206 (100)

The mean FBS (174.32 ± 105.83 mg / dl) was higher than the normal range in patients with CVA (Table 2). FBS was higher in women than men but, this difference was not significant ($P= 0.46$) (Table 3).

In patients with CVA, the highest amount of FBS was observed in the age group of 55-64 years, which was not significant compared to other age groups ($P= 0.11$) (Table 4).

Table 2. Mean \pm SD and normal range of the studied factors in patients with CVA and TIA

Variable	Normal Ranges	Mean \pm SD in CVA patients	Mean \pm SD in TIA patients
FBS (mg / dl)	70-100	174.32 \pm 105.83	150.32 \pm 83.32
BUN (mg / dl)	7-20	20.65 \pm 15.47	18.86 \pm 13.32
Cr (mg / dl)	0.6-1.3	1.37 \pm 1.32	1.42 \pm 1.09
Na (mmol / L)	135-145	139.88 \pm 4.31	139.96 \pm 3.35
K (mmol / L)	3.5-5.5	5.52 \pm 13.80	4.13 \pm 0.56
ALK (U / L)	60-306	331.44 \pm 370.78	270.77 \pm 172.28
LDH (IU / L)	200-500	696.29 \pm 344.90	538.17 \pm 230.76
WBC ($\times 10^3/\text{mm}^3$)	4.5-11	8.58 \pm 2.65	8.47 \pm 3.13
RBC($\times 10^6/\text{mm}^3$)	Male: 4.3-5.9 Female: 3.5-5.5	4.69 \pm 0.99	4.61 \pm 0.83
HB (g / dL)	Male: 13.5-17.5 Female: 12.0-16.0	12.95 \pm 3.68	12.74 \pm 2.06
HCT (%)	Male: 41 - 53 Female: 36 - 46	38.47 \pm 6.95	38.87 \pm 5.93
MCV (μm^3)	80-100	83.77 \pm 8.66	85.09 \pm 9.24
MCH (pg / cell)	25.4-34.6	28.64 \pm 4.31	27.66 \pm 3.76
MCHC (% Hb / cell)	31 - 36	30.68 \pm 3.19	31.51 \pm 2.23
PLT ($\times 10^3/\text{mm}^3$)	150-400	230.23 \pm 76.23	236.01 \pm 66.22
ESR (mm / h)	0-10	52.41 \pm 37.61	14.00 \pm 8.40
SGOT (IU / L)	0-31	34.10 \pm 26.40	27.62 \pm 27.08
SGPT (IU / L)	0-31	28.62 \pm 28.31	33.08 \pm 38.55
PT (s)	11-13	12.73 \pm 2.87	12.12 \pm 1.28
PTT (s)	25-45	34.71 \pm 12.88	31.27 \pm 8.85
INR	< 1.1	1.09 \pm 0.143	1.07 \pm 0.20

Abbreviations: FBS fasting blood sugar; BUN blood urea nitrogen; Cr creatinine; ALK alkaline phosphatase; LDH lactate dehydrogenase; WBC white blood cell; RBC red blood cell; HB hemoglobin; HCT hematocrit; MCV mean corpuscular volume; MCH mean corpuscular hemoglobin; PLT platelet; ESR erythrocyte sedimentation rate; SGOT serum glutamate oxalate transaminase; PT prothrombin time; PTT partial thromboplastin time; INR international normalized ratio.

Table 3. Mean \pm SD of factors studied in CVA and TIA patients by gender

Factor	CVA			TIA		
	Mean \pm SD		P-value	Mean \pm SD		P-value
	Male	Female		Male	Female	
FBS (mg / dl)	165.13 \pm 83.01	181.68 \pm 121.39	0.464	146.97 \pm 93.49	153.17 \pm 74.63	0.74
BUN (mg / dl)	18.82 \pm 10.54	22.18 \pm 18.58	0.285	18.26 \pm 10.29	19.39 \pm 15.62	0.71
Cr (mg / dl)	1.40 \pm 1.10	1.34 \pm 1.48	0.833	1.45 \pm 0.72	1.39 \pm 1.34	0.79
Na (mmol / L)	139.48 \pm 4.67	140.22 \pm 4.00	0.393	140.05 \pm 3.57	139.88 \pm 3.195	0.82
K (mmol / L)	4.00 \pm 0.50	6.80 \pm 18.75	0.314	4.15 \pm 0.60	4.11 \pm 0.53	0.76
WBC ($\times 10^3/\text{mm}^3$)	8.28 \pm 2.81	8.82 \pm 2.52	0.31	9.08 \pm 3.64	7.93 \pm 2.53	0.1
MCV (μm^3)	84.54 \pm 9.02	83.14 \pm 8.39	0.42	85.74 \pm 10.83	84.52 \pm 7.67	0.5
MCH (pg / cell)	29.10 \pm 4.21	28.27 \pm 4.40	0.34	28.31 \pm 4.19	27.10 \pm 3.28	0.1
MCHC (Hb / cell)	30.48 \pm 3.54	30.84 \pm 2.90	0.58	32.12 \pm 0.07	30.97 \pm 2.25	0.02
RBC ($\times 10^6/\text{mm}^3$)	4.95 \pm 1.00	4.47 \pm 0.93	0.01	4.82 \pm 0.90	4.43 \pm 0.73	0.03
PTT(s)	36.44 \pm 15.39	33.28 \pm 10.32	0.260	32.69 \pm 8.79	30.00 \pm 8.87	0.264
INR	1.09 \pm 0.16	1.08 \pm 0.12	0.734	1.08 \pm 0.15	1.07 \pm 0.23	0.781
PT(s)	12.44 \pm 1.56	12.97 \pm 3.62	0.401	12.15 \pm 1.23	12.10 \pm 1.35	0.879
PLT ($\times 10^3/\text{mm}^3$)	217.77 \pm 74.76	240.15 \pm 76.62	0.152	216.59 \pm 62.05	253.12 \pm 65.75	0.013
HB (g / dL)	14.20 \pm 4.75	11.94 \pm 2.04	0.002	13.66 \pm 1.73	11.94 \pm 2.01	0.000
ALK (IU / L)	205.86 \pm 57.65	411.36 \pm 462.23	0.264	185.00 \pm 25.45	286.36 \pm 183.89	0.308
SGOT (IU / L)	29.44 \pm 18.85	37.91 \pm 31.69	0.491	15.25 \pm 6.23	33.11 \pm 31.27	0.710
SGPT (IU / L)	22.44 \pm 12.35	33.25 \pm 35.95	0.401	11.00 \pm 6.63	42.89 \pm 43.14	0.414
ESR (mm / h)	65.75 \pm 43.83	40.56 \pm 28.50	0.176	11.83 \pm 6.85	16.60 \pm 10.11	0.329
LDH (IU / L)	762.38 \pm 411.69	608.17 \pm 235.99	0.430	639.50 \pm 256.68	487.50 \pm 237.74	0.533

Table 4. Mean \pm SD of factors studied in CVA patients by age groups

Factor	Age groups					Total	P-value
	44 \geq	45-54	55-64	65-74	\geq 75		
FBS	107.67 \pm 21.92	123.89 \pm 44.30	195.56 \pm 124.08	190.28 \pm 100.60	172.67 \pm 111.30	174.32 \pm 105.83	0.110
BUN	20.45 \pm 24.45	18.40 \pm 9.45	18.43 \pm 13.74	22.67 \pm 15.89	23.35 \pm 14.63	20.65 \pm 15.47	0.764
Cr	1.99 \pm 3.17	1.18 \pm 0.65	1.14 \pm 0.55	1.43 \pm 1.26	1.45 \pm 0.68	1.37 \pm 1.32	0.442
Na	142.27 \pm 5.35	137.40 \pm 5.56	139.74 \pm 3.20	141.52 \pm 3.62	137.61 \pm 4.28	139.88 \pm 4.31	0.003
K	3.87 \pm 0.37	4.04 \pm 0.47	4.06 \pm 0.45	4.41 \pm 0.63	11.76 \pm 35.51	5.52 \pm 13.80	0.346
PTT	31.20 \pm 4.59	28.78 \pm 4.17	33.21 \pm 6.42	35.79 \pm 12.37	41.67 \pm 23.75	34.71 \pm 12.88	0.106
INR	1.02 \pm 0.04	1.07 \pm 0.10	1.04 \pm 0.08	1.11 \pm 0.17	1.19 \pm 0.18	1.09 \pm 0.14	0.008
PT	11.70 \pm 0.50	12.51 \pm 1.05	12.13 \pm 0.93	13.63 \pm 5.08	13.21 \pm 1.50	12.73 \pm 2.87	0.260
WBC	7.83 \pm 2.14	8.74 \pm 3.05	8.62 \pm 3.07	9.04 \pm 2.30	8.19 \pm 2.48	8.58 \pm 2.65	0.730
RBC	5.32 \pm 1.34	4.46 \pm 0.61	4.88 \pm 0.82	4.58 \pm 1.12	4.26 \pm 0.83	4.69 \pm 0.99	0.047
MCV	78.30 \pm 8.0	81.10 \pm 8.6	81.58 \pm 9.4	86.71 \pm 6.51	88.33 \pm 7.16	83.77 \pm 8.66	0.003
MCH	27.00 \pm 4.38	29.68 \pm 6.18	27.80 \pm 4.61	28.81 \pm 3.49	30.47 \pm 3.22	28.64 \pm 4.31	0.154
MCHC	30.48 \pm 4.24	30.99 \pm 2.12	31.14 \pm 3.46	30.83 \pm 2.60	29.58 \pm 3.24	30.68 \pm 3.19	0.562
PLT	228.73 \pm 56.92	255.11 \pm 88.07	254.24 \pm 92.10	219.31 \pm 66.60	190.44 \pm 38.25	76.23 \pm 7.74	0.043
HB	15.55 \pm 9.20	12.41 \pm 1.96	12.98 \pm 2.06	12.73 \pm 2.39	11.91 \pm 1.85	3.68 \pm 0.37	0.125
HCT	39.03 \pm 9.46	37.32 \pm 5.35	38.81 \pm 6.61	39.51 \pm 7.26	36.59 \pm 6.52	38.47 \pm 6.95	0.691

A significant inverse relationship was observed between FBS and Na ($P = 0.008$). FBS showed a significant relationship with K^+ ($P < 0.001$), WBC

($P = 0.003$), and RBC ($P = 0.031$). In this study, FBS had no significant relationship with other diagnostic markers (Table 6).

Table 6. Association between FBS and laboratory diagnostic factors in the linear regression model

Variable	CVA (N = 100)		TIA (N = 80)	
	B (95% CI)	P-value	B (95% CI)	P-value
Demographic				
Age	1.33 (- 0.23, 2.89)	0.094	0.726 (- 0.602, 2.053)	0.279
Sex (female)	16.56 (- 28.18, 61.29)	0.464	6.20 (- 32.24, 44.64)	0.749
Renal				
BUN	0.008 (- 0.20, 0.037)	0.563	0.002 (- 0.026, 0.029)	0.907
Cr	0.001 (- 0.002, 0.003)	0.710	0.001 (- 0.002, 0.004)	0.636
Electrolyte				
Na	- 0.011 (- 0.20, - 0.003)	0.008	- 0.10 (- 0.020, - 0.001)	0.028
K	0.002 (0.001, 0.004)	< 0.001	0.001 (- 0.001, 0.002)	0.729
Hematologic and Coagulation				
WBC	0.008 (0.003, 0.013)	0.003	0.003 (- 0.012, 0.005)	0.455
RBC	0.002 (0.000, 0.004)	0.031	0.001 (- 0.001, 0.003)	0.570
HB	0.001 (- 0.007, 0.009)	0.771	0.004 (- 0.001, 0.009)	0.146
PLT	0.148 (- 0.008, 0.304)	0.062	0.006 (- 0.191, 0.180)	0.951
MCV	0.012 (- 0.028, 0.015)	0.158	0.004 (- 0.017, 0.026)	0.687
MCH	0.003 (- 0.005, 0.012)	0.436	0.005 (- 0.004, 0.014)	0.249
MCHC	0.004 (- 0.002, 0.011)	0.167	0.004 (- 0.002, 0.010)	0.235
INR	0.000 (- 0.000, 0.000)	0.672	0.000 (- 0.001, 0.000)	0.224
PT	0.000 (- 0.006, 0.006)	0.949	0.003 (- 0.001, 0.007)	0.085
PTT	0.018 (- 0.008, 0.044)	0.180	0.030 (0.003, 0.056)	0.029

CI: confidence interval; B: regression coefficient.

Among the liver function tests, the mean and standard deviation of LDH (696.29 ± 344.90 IU / L), SGOT (34.10 ± 26.40 IU / L), and ALK (331.44 ± 370.78 IU / L) were also higher than normal ranges in patients with CVA (Table 2). Moreover, mean SGOT and SGPT in women with CVA were higher than normal and higher than in men, although this gender difference was not significant ($P = 0.49$ and $P = 0.40$, respectively) (Table 3). The mean LDH was higher than normal in men and women with CVA and higher in men than women, but this difference was not significant ($P = 0.43$) (Table 3).

Regarding renal function tests, in patients with CVA, the mean of creatinine (1.37 ± 1.32 mg / dl), BUN (20.65 ± 15.47 mg / dl), and K^+ (5.52 ± 13.80 mmol / L) were slightly higher than normal (Table 1). Although the mean BUN in women with CVA was higher than normal and also higher than the mean BUN in men, the difference was not significant ($P = 0.28$). The Mean creatinine was higher than normal in both women and men with CVA and the mean creatinine in men is higher than in women but the difference was not significant ($P = 0.83$) (Table 3). The mean creatinine and BUN in patients with CVA in the age group over 75 years had the highest values among the age groups while these changes in BUN ($P = 0.76$) and creatinine ($P = 0.44$) were not significant (Table 3).

Among hematological markers, the mean ESR (52.41 ± 37.61 mm / h) was higher than the normal ranges (Table 2) in both CVA and TIA. The mean ESR was higher than normal in men and women with CVA and higher in men than women, but these differences were not significant ($P = 0.17$) (Table 3). Associations between gender groups and serum levels of hematological markers were evaluated (Table 3). The difference among age groups was estimated in CVA patients (Table 4). Among CVA patients, PLT ($P = 0.043$), RBC ($P = 0.047$), MCV ($P = 0.003$), and INR ($P = 0.008$) (Table 4) were markers that differ by age group, respectively. In contrast, no significant difference was recorded in other markers among both groups of patients.

Laboratory findings in Patients with TIA

The number of patients with TIA was 206, of which 112 (54.4%) were female, and 94 (45.6%) were male. The age range of 55-64 years had the highest frequency of TIA patients ($n = 49$, 23.8%). The age range of 15-24 years had the lowest number of patients

with TIA (Table 1). The mean FBS in patients with TIA (150.32 ± 83.32 mg / dl) was higher than the normal range (Table 2). FBS in patients with TIA was higher in women than men but, this difference was not significant ($P = 0.74$) (Table 3). An increase in FBS leads to a decrease in Na^+ , which was significant ($P = 0.028$). An increase in FBS also leads to an increase in PTT which was significant ($P = 0.029$) in this study, FBS had no significant relationship with other diagnostic markers in patients with TIA (Table 6). The mean SGPT (33.08 ± 38.55 IU / L) and LDH (538.17 ± 230.76 IU / L) were higher than normal ranges in patients with TIA (Table 2). Mean SGOT and SGPT were higher in women than normal and higher than in men although the difference was not significant ($P = 0.71$ and $P = 0.41$, respectively) (Table 3). The mean LDH of men was higher than normal and higher than women but the difference was not significant ($P = 0.53$) (Table 3).

In renal function tests, the mean creatinine (1.42 ± 1.09) was higher than the normal ranges in TIA patients (Table 2). Mean creatinine in women and men with TIA is higher than the normal and mean creatinine in men is higher than in women but, this change was not significant ($P = 0.79$) (Table 3). Mean creatinine and BUN in patients with TIA in the age group over 70 years had the highest values among the age groups and these changes were significant in BUN ($P = 0.03$) but in creatinine, this change was not significant ($P = 0.78$) (Table 5).

The mean ESR (14.00 ± 8.40 mm/h) was higher than the normal ranges (Table 2). The mean ESR was higher than normal in men and women with TIA and higher in women than men but, this difference was not significant ($P = 0.32$) (Table 3).

The difference among age groups was assessed in TIA patients (Table 5). RBC ($P = 0.001$), MCV ($P = 0.020$), and HB ($P = 0.025$) (Table 5) were markers that differ by age groups, respectively (Table 5).

Table 5. Mean \pm SD of factors studied in TIA patients by age group

Factor	Age groups				Total	P-value
	49 \geq	50-59	60-69	\geq 70		
FBS	181.60 \pm 138.46	148.47 \pm 71.22	154.00 \pm 73.50	127.23 \pm 38.79	150.32 \pm 83.32	0.281
BUN	12.58 \pm 3.32	16.00 \pm 8.41	20.10 \pm 18.94	24.48 \pm 12.93	18.86 \pm 13.32	0.030
Cr	1.19 \pm 1.05	1.49 \pm 1.54	1.39 \pm 0.92	1.54 \pm 0.83	1.42 \pm 1.09	0.787
Na	140.88 \pm 4.55	139.68 \pm 2.98	140.10 \pm 3.41	139.39 \pm 2.53	139.96 \pm 3.35	0.559
K	4.07 \pm 0.41	4.12 \pm 0.59	4.16 \pm 0.45	4.15 \pm 0.73	4.13 \pm 0.56	0.962
PTT	31.18 \pm 10.28	29.25 \pm 6.69	33.47 \pm 12.09	30.47 \pm 3.79	31.27 \pm 8.85	0.627
INR	1.03 \pm 0.04	1.19 \pm 0.37	1.05 \pm 0.12	1.04 \pm 0.11	1.07 \pm 0.20	0.139
PT	11.64 \pm 0.67	12.89 \pm 2.06	11.95 \pm 1.02	12.06 \pm 0.89	12.12 \pm 1.28	0.099
WBC	8.60 \pm 3.23	8.57 \pm 1.21	9.42 \pm 3.48	7.41 \pm 3.60	8.47 \pm 3.13	0.203
RBC	5.11 \pm 0.88	4.86 \pm 0.65	4.52 \pm 0.80	4.13 \pm 0.70	4.61 \pm 0.83	0.001
MCV	81.49 \pm 8.63	82.88 \pm 7.37	84.75 \pm 6.11	89.79 \pm 11.65	85.09 \pm 9.24	0.020
MCH	27.14 \pm 4.11	26.63 \pm 3.08	27.96 \pm 3.66	28.59 \pm 4.03	27.66 \pm 3.76	0.364
MCHC	32.07 \pm 2.11	31.25 \pm 2.28	31.28 \pm 2.64	31.51 \pm 1.92	31.51 \pm 2.23	0.679
PLT	237.06 \pm 51.92	266.39 \pm 63.89	232.38 \pm 73.24	214.78 \pm 65.72	236.01 \pm 66.22	0.099
HB	13.84 \pm 1.57	13.16 \pm 1.57	12.43 \pm 2.69	11.82 \pm 1.69	12.74 \pm 2.06	0.025

Discussion

In this study, we found that serum levels of LDH, ESR, creatinine, and FBS were higher than normal ranges among the CVA and TIA patients. We detected a higher ESR among both CVA and TIA patients. In the study of Emsley et al. ESR was elevated at 5 to 7 days after ischemic stroke compared with controls, and this elevation persisted at 3 months but not at 12 months (16).

Here, in line with Norris et al., we found that LDH was another marker that was elevated in both CVA and TIA groups (17). Although Norris et al did not find a significant rise in LDH levels among stroke groups compared to the control group; they detected elevated LDH levels among the hemorrhagic stroke group. A possible reason can be related to cardiac involvement and this concept was further confirmed by progressive ischemic changes, the timing of elevated cardio-specific CK isoenzyme, and increased incidence of cardiac arrhythmias (17).

The present study showed increased serum levels of ALK among patients with CVA. Similarly, Muscari, et al. reported this finding and explained it as the participation of the liver during the response to acute ischemic stroke by releasing enzymes (10). Hence, it can be speculated that elevated levels of ALK can indicate an interaction between the brain and peripheral immune system (18).

We also found that FBS is elevated among both CVA and TIA groups. With each unit increase in FBS, a

significant increase in the amount of K^+ , white blood cells, and erythrocytes, but a significant decrease in Na^+ was observed in patients with CVA. The study by Sahetapi et al. reported the elevated level of fasting blood sugar in stroke groups (3). However, Singh et al. (19) observed no difference between healthy people and patients with stroke since both groups had a normal range of fasting blood. This controversy may attribute to the small sample size. It has been shown that hyperglycemia was correlated with poor functional outcomes (20) and also a higher risk of mortality (21). However, it could be a better predictor of prognosis in patients without a history of diabetes (20).

In line with other laboratory findings, we found that the mean creatinine levels were abnormal in the studied groups. In contrast, Snarska et al. reported a normal mean of creatinine among patients with both types of stroke, ischemic and hemorrhagic stroke, at admission. However, those who died had significantly raised levels of creatinine within 48 hours which fulfilled the criteria of acute kidney injury (22).

The results of our study have also detected abnormal levels of SGOT among patients with CVA while Muscari et al. (23) have demonstrated normal levels of SGOT among these patients. However, they have shown an elevation of SGOT levels after the 7th day compared to admission time. It could refer to the protective role of SGOT. SGOT can be metabolized and thus neutralize the toxic glutamate that releases from damaged brain tissue after a stroke in the brain.

Interestingly, the level of SGOT had a direct correlation with cerebral infarct size indicating higher infarct size results in a higher release of glutamate which in turn lead to higher level of SGOT. Furthermore, it has been shown that lower production of SGOT in this situation leads to a higher mortality rate (24).

A limited number of patients in the TIA group and some missed data among them were the first limitations of our study. Additional investigations with a larger sample size for both CVA and TIA groups are needed to prove our results. Another limitation of this study is that it was performed in a single center, and for a more confident result, this study should be repeated in a multicenter study. Overall, this study showed that the frequency of patients with CVA and TIA is higher in the elderly. Some hepatic and renal markers and FBS were observed above normal ranges in these patients. It was also observed that with increasing age in these patients, FBS increases, and each unit of increased FBS is associated with an increase in renal markers and white blood cell and red blood cell counts and changes in electrolytes is observed.

Conclusion

The present study found that an increase occurs in the level of some inflammatory markers in patients with CVA and TIA along with an increase in blood sugar and alterations in some liver and kidney function tests. A set of biomarkers showing pathways related to the pathophysiology of these diseases may be used for diagnosis, but further studies are needed to determine if they can be used for treatment planning.

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Note declared.

Conflict of Interest

The authors announce that they haven't any conflicting interests regarding the research or the publication of the manuscript.

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Ethics

The Ethics Committee of the Abadan University of Medical Sciences (Ethics code:

IR.ABADANUMS.REC.1399.033) approved this study.

Authors' contributions

E. R. contributed to conceptualization and project administration. A. M., A. H., K. K., S. J., M. T., and M. F. contributed to the methodology. A. M., M. F., and E. R. contributed to writing the original draft. N. K. and A. Z. contributed to the data analysis. E. R. and S. G. contributed to the review and editing

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