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How to Design Economic Predictive Laboratory Panel Evaluating Acute Ischemic Stroke Outcome

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Abstract

Background: acute ischemic stroke (AIS) remains the third cause of death and disability, and acute phase responses, both increasing international normalized ratio (INR) and activated partial thromboplastin time (APTT) are associated with worse outcome. Erythrocyte sedimentation rate (ESR) serves as severity marker, and non-fasting triglycerides (TG) indicates remnants of chylomicrons and very low density lipoproteins potentially pro-inflammatory. **Aims:** to design predictive economic panel evaluating AIS. **Patients and methods:** 100(AIS) patients were included, clinically evaluated by Scandinavian Stroke Scale (SSS) and Modified Rankin Score (MRS), subjected to complete blood count (CBC) on Cell-Dyne3700, manual ESR, INR and APTT on SYSMEX-CA1500, serum uric acid (SUA), serum albumin and non-fasting (TG) on Beckman Coulter AU480. **Statistical analysis:** STATA intercooled version 9.2. **Results:** odd ratio (OR), confidence interval (CI) of (MRS) in correlation to WBCs count in quartile (Q)3, 4 (OR 8.14, CI 2.29 - 8.90, significant P = 0.01; and OD13.5, CI 3.39 - 53.68, high significant P = 0.001 respectively), to APTT in Q3 (OD 4.15, CI 1.09 - 15.82, P = 0.04), SUA in Q3 (OD 0.19, CI 0.05 - 0.68, P = 0.01), TG in Q3,4 (OD 0.24 CI 0.06 - 0.88, P = 0.03; and OD 0.09, CI 0.02 - 0.34 P = 0.001 respectively) and serum albumin in Q3(OD 0.13, CI 0.04 - 0.51, P = 0.003), insignificant correlations to ESR, INR and platelets. **Conclusion:** according to (MRS), the economic predictive panel should be included WBCs, APTT, SUA, and non-fasting TG with serum albumin as prognostic tool evaluating functional disability in AIS.

Keywords

Acute Ischemic Stroke, Predictive Laboratory Panel

1. Introduction

Stroke is a leading cause of preventable death and major disability. Biomarkers are tests that indicate the physiology of the body [1]. Many biomarkers are related to the etiology and pathophysiology of the ischemic stroke as tissue ischemia markers, thrombosis and vascular occlusion markers, inflammatory response markers, arterial recanalization markers, biomarkers of early neurological deterioration, biomarkers of infarction size and outcome, and lastly biomarkers of preventive therapy [2]. The rapid and effective management of critically ill patients depends on rapid evaluation of the stroke etiology and clinical condition. This is necessary to take prompt decision in therapy especially when time of onset is not known [3]. SSS used in several clinical trials to either select patients or rate outcome severity [4]. Other tests are non-disease specific to brain ischemia, but can be applicable in assessing the stroke severity and can predict the outcome [5]. In practice guidelines of biomarkers panels used in AIS management are limited and meta-analysis studying of various stroke biomarkers has improve the evaluation of individual pathophysiology, promoting the establishment of screening panels for high risk patients, and rational therapy tailored to each patient findings [6]. Inflammatory parameters ESR and fibrinogen have been proposed as risk markers [7]. Leukocytes may reflect the inflammatory status after AIS, so it is considered as strategy to improve the outcome of the patient [8]. Some studies showed that, the pro-inflammatory biomarkers in association with high serum lipids and prothrombotic factors predispose to internal carotid artery occlusion [9] [10]. Increased level of biomarkers of inflammation and coagulation is associated with recurrent ischemic lesions after AIS [11]. The significant value of INR and APTT is a predictive risk factor for AIS outcome [12]. In patients diagnosed as AF with long term warfarin therapy, INR is not considered as a predictive test for AIS outcome, but D-dimer is used instead [13] [14]. Ischemic modified albumin is considered as a useful diagnostic marker of both AIS, and its sensitivity and specificity is 87% [15] [16]. Another chemical investigation within the predictive panels of AIS is SUA. Its high level is associated with excellent outcomes so it used as adjuvant therapy to thrombolysis [17] [18]. Serum TG is an independent predictor of AIS outcome, but its association with cardio-embolic stroke is not a role [19]. Increase level of non-fasting triglycerides is associated with increasing risk of ischemic stroke [20].

2. Patients and Methods

2.1. Study Protocol

The study was carried out on 100 patients admitted to Neurology Department, Sohag University Hospital, over a period of one year from May 2013 to May 2014, they were diagnosed as AIS. Their age ranged from 27 to 88 years old with median age 65 years; they were 52 males and 48 females. **The inclusion criteria** were diagnosis of AIS, according to the definition established by the World Health Organization, stroke is rapidly developed clinical signs of focal or global

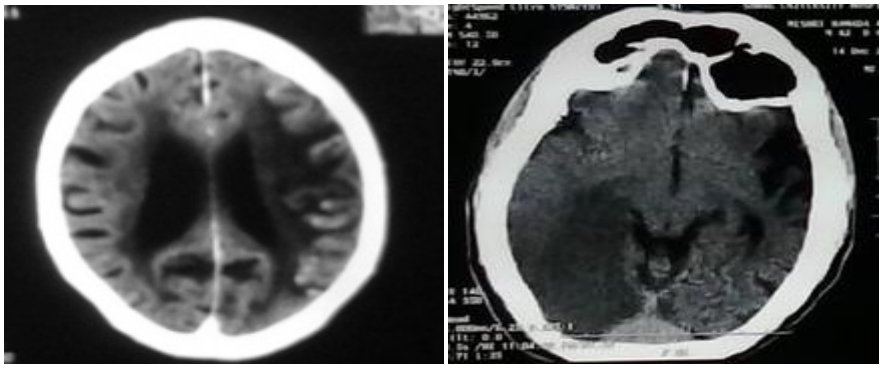
deficits of cerebral function, lasting more than 24 hours, or until death, with no apparent non-vascular cause [1]. **The exclusion criteria** were other ischemic conditions like acute myocardial infarction, limb ischemia, and renal failure, known inflammatory or malignant disease, also cerebral hemorrhage or other coagulation disorders.

2.2. Medical Ethics

Institutional Research Committee Board had been approved the research. Informed consent from the enrolled patients or their relatives was obtained.

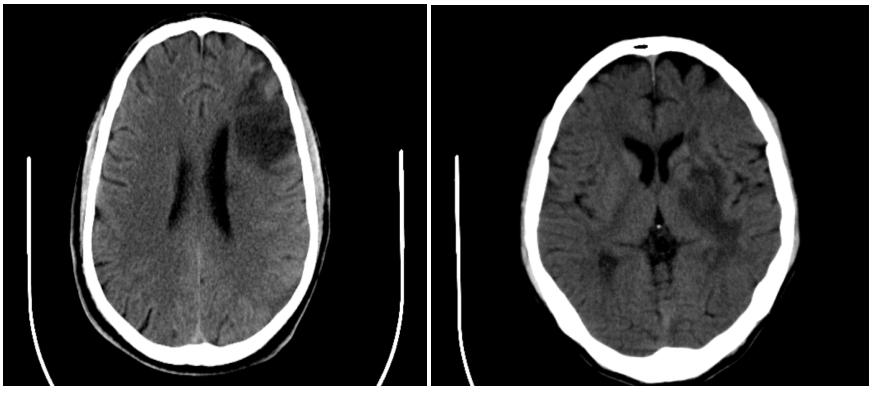
2.3. Clinical Evaluation

For each patient full history, clinical and neurological examination and brain computed tomography (CT scan) or magnetic resonance imaging (MRI), were performed within 24 h of stroke. The volume of hypo dense areas of brain CT scan, was calculated according to the formula based on length × depth × height × 1/2 (in mm) as in **Figures 1-3**. Stroke severity on admission was assessed using SSS, milder stroke was considered when SSS > 25 whereas it's considered



(a) (b)

Figure 1. CT findings. (a) Lt cerebral infarction 1.6 × 2.4 cm; (b) Rt occipital infarction 5 × 4.5 cm.



(a) (b)

Figure 2. CT findings. (a) LT Frontal infarction 3 × 3.6 cm; (b) LT Basal ganglia infarction 3 × 2.4 cm.

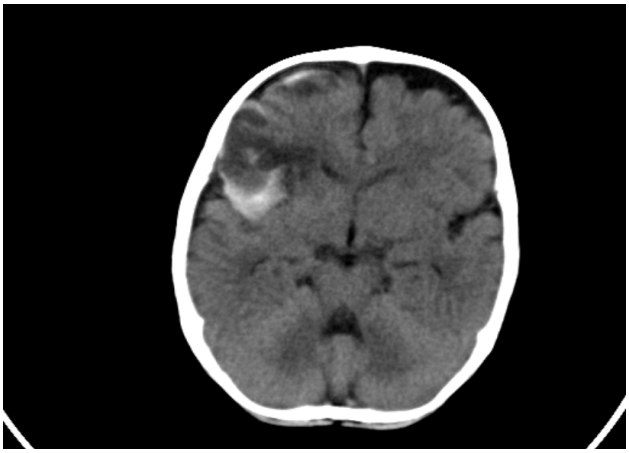


Figure 3. RT. Frontoprietal old infarction 4 × 3.7 cm.

more severe when SSS was ≤ 25 [4]. Functional outcome was measured using MRS on day seven; lesser degree of disability was defined as MRS of 1 - 3 whereas greater degree of disability was defined as MRS 4 - 6 [1]. Hypertension was defined as diastolic blood pressure (DBP) ≥ 90 mmHg and/or a systolic blood pressure (SBP) ≥ 140 mmHg. Diabetic patients were considered when fasting blood glucose is more than 126 mg/dl or random blood glucose is more than 200 mg/dl.

3. Laboratory Investigations

Sample processing: 7 ml of venous blood (non-fasting sample) was withdrawn from each patient, divided as follow:2ml was delivered into trisodium citrate vacutainer; which centrifuged at 3000/rpm for 10 min at 18˚C - 22˚C to separate platelet poor plasma (P.P.P) used for INR and aPTT, another 3ml of blood was delivered into K-EDTA vacutainer used for CBC and then diluted 4:1 with trisodium citrate solution (3.2%) used for ESR, the remaining blood was clotted, then centrifuged at 3000/rpm for 5 - 10 min, at room temperature, the separated serum was used for the remaining investigations.

4. Methods

CBC was performed on the Abbott Cell-Dyne 3700, (USA) automated hematology analyzer. ESR was performed using Westergren's method; the reading was adjusted to the 1st hour. INR and aPTT were determined using the Sysmex-CA1500 System (Germany) fully automated blood coagulation analyzer; the reagents were SIEMENS' Thromborel®S,Cat.No.OUHP29/54690523, Pathromb-tin®SLCat.No.OQGS29/53667823). Renal function tests including SUA, liver function tests with serum albumin, blood glucose and non-fasting TG were performed on Beckman Coulter AU480 (USA) fully automated system; all reagents were supplied by Coulter Beckman.

5. Statistical Analysis

Data was analyzed using STATA intercooled version 9.2. The data were not

normally distributed; Mann-Whitney test was used. Evaluation of the data was by definition of mean ± SD. Logistic regression analysis was used to calculate Odds Ratio and 95% Confidence interval (OR and 95% CI), threshold for statistical significance was established at $P < 0.05$.

6. Results

This study was performed over one year from 2013- to 2014 on 100 patients admitted to University Hospital, Neurology Department, diagnosed as AIS; their mean age was 64.83 ± 11.58 years old, males to females' ratio was 52/48. As regard the risk factors twenty patients (20%) were smokers, sixty two (62%) were hypertensive, the diabetic cases were 29 (29%), and cardiac patients were 48 (48%), one third of them were atrial fibrillation (AF) sixteen patients. Thirty three patients (33%) had positive history of previous attack. These data were presented in Table 1. The neurological data of studied population showed that the Glasgow Coma Scale (GCS) was 11.84 ± 2.75 and median was 11, GCS was ≥8 in 89 patients(89%), where it was <8 in eleven patients (11%). stroke severity using (SSS), mean ± SD was 23.69 ± 17.52 and median was 17.5. The disability scale at discharge by (MRS), was 3.86 ± 1.37 and median was 4, MRS was ≥3 in 38% of patients, where as it was <3 in 62% of patients. CT scan was normal in

Table 1. Patients' characteristics and risk factors.

Items	Statistics
Age in years Median (range)	65 (27 - 88)
Sex Females/males	48/52
Risk Factors	**(No. and %)**
Smoking	
No	80
Yes	20
Previous stroke	
No	67
Yes	33
Hypertension	
No	38
Yes	62
Diabetes	
No	71
Yeas	29
Cardiac	
No	52
Ischemic	32
AF	16

forty one (41%) of studied population, while it revealed ischemic changes in nine patients (9%), old infarction in seven patients (7%), recent infarction in 39 cases (39%) and multiple lacunae's in 4% of them (**Figures 1-3**). Comparison between patients with mild or moderate to those with severe SSS at admission according to the tested panel, we find that; in patients with less severe stroke (SSS > 25); ESR was 26.51 ± 21.67 mm/h, total WBCs was (8.89 ± 3.69) × 10^9/l, neutrophils was (6.23 ± 3.58) × 10^9/l, platelets count was (276.64 ± 91.76) × 10^9/l, INR was 1.13 ± 0.15, aPTT was 31.30 ± 5.19 Sec., serum uric acid was 6.67 ± 2.26 mg/dl, non-fasting triglycerides was 168.72 ± 120.27 mg/dl and serum albumin was 3.53 ± 0.53 g/dl. while in patients with more severe stroke (SSS ≤ 25); ESR was 27.28 ± 22.51 mm/h, WBCs was (11.94 ± 4.42) × 10^9/l, neutrophils was (9.78 ± 6.46) × 10^9/l, platelets was (270.07 ± 92.13) × 10^9/l, INR was 1.13 ± 0.09, aPTT was 33.16 ± 6.23 Sec., serum uric acid was 6.96 ± 3.43 mg/dl, non-fasting TG was 124.11 ± 73.81 mg/dl, serum albumin was 3.49 ± 0.71 g/dl. The difference between these two groups was statistically highly significant for WBCs count (P = 0.001), neutrophils count (P = 0.004), and significant for triglycerides (P = 0.04). While it was insignificant for ESR (P = 0.77), platelets count (P = 0.42), INR (P = 0.64), aPTT (P = 0.11), uric acid (P = 0.89) and serum albumin (P = 0.31). Given the random variance among AIS patients to the investigated panel; the OR of test's quartiles (Qs) demonstrated that, (Q1) was used as reference to other quartiles (Q2), (Q3), (Q4)), when these (Qs) were assessed according to SSS; the resulting data were demonstrated in **Table 2**. In comparing patients with good to those with poor outcome according to MRS; the functional disability showed that 38 patients had good prognosis with score less than 3 in day seven, while the other 62 patients had bad prognosis in which the MRS was more than 3 by the day seven. The difference between these two groups was presented in **Table 3**. The OR of functional disability according to the MRS in relation to the random variance quartiles of investigated panel were presented in **Table 4**.

7. Discussion

Because stroke constitutes a major health problem not only affect the patient but also his/her family, and productive availability of the community especially if the affected patients within the adult age group, or even the old age group who need much care. So the accurate diagnosis with appropriate treatment can offer beneficial outcome with less severe disability. Design an economic panel predicting functional disability which is a common AIS squeals', is considered a strategic decision in hospital management programs to safe time, money and efforts, also provides the patients with the most suitable therapy within the target period for excellent functional discharge. Some researcher has reported stroke is a leading cause of death and adult disability [21]. Cerebral ischemia with subsequent reperfusion initiates an inflammatory response with increase in leukocytes, platelets and acute-phase proteins. With the elevated plasma viscosity and pronounced reduction in cerebral circulation; infarction size is increased as mentioned by Lakshmi

Table 2. Odds ratio (95% Confidence interval) in 1st quartile of SSS to the laboratory panel.

Item	Odds ratio (95 Confidence interval)	P-values
ESR mm/h		
Q1 (2 - 9, n = 25)	1	
Q2 (11 - 20, n = 25)	1.00 (0.33 - 3.06)	1.00
Q3 (23 - 37, n = 25)	1.18 (0.38 - 3.63)	0.78
Q4 (37 - 97, n = 25)	1.00 (0.33 - 3.06)	1.00
WBCs × 10^9/l		
Q1 (4.16 - 7.7, n = 25)	1	
Q2 (7.8 - 9.7, n = 25)	3.43 (1.03 - 11.48)	0.045 S
Q3 (9.8 - 12.5, n = 25)	8.14 (2.29 - 28.90)	0.001 HS
Q4 (12.9- 28.3, n = 25)	12.67 (3.31 - 48.50)	<0.0001 HS
Platelets × 10^9/l		
Q1 (175 - 207, n = 25)	1	
Q2 (208 - 259, n = 25)	1.00 (0.32 - 3.17)	1.00
Q3 (260 - 320, n = 25)	0.44 (0.14 - 1.37)	0.16
Q4 (323 - 570, n = 25)	0.72 (0.23 - 2.23)	0.56
INR		
Q1 (0.96 - 1.06, n = 26)	1	
Q2 (1.07 - 1.12, n = 26)	1.17 (0.39 - 3.50)	0.78
Q3 (1.13 - 1.18, n = 23)	1.33 (0.43 - 4.16)	0.62
Q4 (1.19 - 1.91, n = 25)	1.09 (0.36 - 3.29)	0.88
aPTT (sec)		
Q1 (24.4 - 28, n = 25)	1	
Q2 (28 - 31.7, n = 28)	1.08 (0.37 - 3.19)	0.88
Q3 (32 - 35, n = 22)	2.89 (0.85 - 9.81)	0.09
Q4 (35.7 - 58.8, n = 25)	1.63 (0.53 - 4.98)	0.40
Uric acid (mg/dl)		
Q1 (1.4 - 4.6, n = 24)	1	
Q2 (4.8 - 6.1, n = 26)	0.59 (0.18 - 1.90)	0.38
Q3 (6.2 - 8.5, n = 22)	0.29 (0.08 - 0.96)	0.04 S
Q4 (8.6 - 17.7, n = 28)	1.14 (0.33 - 3.90)	0.83
Triglyceride (mg/dl)		
Q1 (61 - 84, n=27)	1	
Q2 (85 - 121, n=23)	0.79 (0.24 - 2.60)	0.70
Q3 (122 - 160, n=26)	0.42 (0.14 - 1.30)	0.13
Q4 (172 - 553, n=24)	0.30 (0.09 - 0.95)	0.04 S
Albumin (g/dl)		
Q1 (2.3 - 3, n = 25)	1	
Q2 (3.1- 3.5, n = 32)	0.53 (0.18 - 1.58)	0.26
Q3 (3.6 - 3.8, n = 20)	0.47 (0.14 - 1.58)	0.22
Q4 (3.9 - 6.4, n = 23)	0.61 (0.19 - 1.98)	0.41

S Significant P value < 0.05, HS high significant P-value 0.001.

Table 3. Modified Rankin Score between good and poor outcome in relation to the investigation panel.

Variables	Modified Rankin score		P-values
	Good outcome ≤ 3 no. = 38	Poor outcome > 3 no. = 62	
ESR mm/h Mean ± SD	24.05 ± 21.56	29.26 ± 22.29	0.18
WBCs × 10^9/l Mean ± SD	8.55 ± 3.03	11.90 ± 4.59	<0.001 S
Platelets × 10^9/l Mean ± SD	268.43 ± 77.39	275.63 ±99.76	0.73
INR Mean ± SD	1.11 ± 0.09	1.14 ± 0.13	0.10
aPTT Sec. Mean ± SD	31.34 ± 5.58	32.99 ± 5.97	0.12
Uric acid mg/dl Mean ± SD	6.78 ± 1.94	6.87 ±3.49	0.32
Triglyceride mg/dl Mean ± SD	176.4 ± 105.9	123.0 ± 88.68	0.005 S
Serum albumin g/dl Mean ± SD	3.59 ± 0.47	3.44 ± 0.72	0.06

S Significant P value < 0.05, HS high significant P-value < 0.001.

Table 4. Odds ratio in the 1st quartile of Modified Rankin score in relation to investigation panel.

Quartile of laboratory panel	Odds ratio (95% Confidence interval)	P-values
ESR mm/h		
Q1 (2 - 9, n = 25)	1	
Q2 (11 - 20, n = 25)	1.64 (0.53-5.09)	0.39
Q3 (23 - 37, n = 25)	1.64 (0.53-5.09)	0.39
Q4 (37 - 97, n = 25)	1.96 (0.62-6.19)	0.25
WBCs × 10^9/l		
Q1 (4.2 - 7.7, n = 25)	1	
Q2 (7.8 - 9.7, n = 25)	3.85 (1.18 - 12.61)	0.25
Q3 (9.7 - 12.5, n = 25)	8.14 (2.29 - 28.90)	0.001 HS
Q4 (12.9 - 28.3,n = 25)	13.5 (3.39 - 53.68)	<0.0001 HS
Platelets × 10^9/l		
Q1 (175 - 207, n = 25)	1	
Q2 (208 - 259, n = 25)	0.58 (0.18 - 1.91)	0.37
Q3 (260 - 320, n = 25)	0.42 (0.13 - 1.36)	0.15
Q4 (323 - 570, n = 25)	0.69 (0.21 - 2.85)	0.55
INR		
Q1 (0.96 - 1.06, n = 26)	1	
Q2 (1.07 - 1.12, n = 26)	1.00 (0.34 - 2.98)	1.00
Q3 (1.13 - 1.18, n = 23)	1.96 (0.60 - 6.35)	0.26
Q4 (1.19 - 1.91, n = 25)	2.20 (0.69 - 7.06)	0.18

Continued

aPTT (sec)		
Q1 (24.4 - 28, n = 25)	1	
Q2 (28 - 31.7, n = 28)	1.07 (0.36 - 3.13)	0.90
Q3 (32 - 35, n = 22)	4.15 (1.09 - 15.82)	0.04 S
Q4 (35.7- 58.8, n = 25)	1.64 (0.53 - 5.09)	0.39
Uric acid (mg/dl)		
Q1 (1.4 - 4.6, n = 24)	1	
Q2 (4.8 - 6.1, n = 26)	0.67 (0.19 - 2.34)	0.53
Q3 (6.2 - 8.5, n = 22)	0.19 (0.05 - 0.68)	0.01 S
Q4 (8.6 - 17.7, n = 28)	0.63 (0.18 - 2.21)	0.47
Triglyceride (mg/dl)		
Q1 (61- 84, n = 27)	1	
Q2 (85 - 121, n = 23)	0.40 (0.10 - 1.59)	0.19
Q3 (122 -160, n = 26)	0.24 (0.06 - 0.88)	0.03 S
Q4 (172 - 553, n = 24)	0.09 (0.02 - 0.34)	<0.0001 HS
Albumin g/dl		
Q1 (2.3 - 3, n = 25)	1	
Q2 (3.1 - 3.5, n = 32)	0.42 (0.12 - 1.40)	0.16
Q3 (3.6 - 3.8, n = 20)	0.13 (0.04 - 0.51)	0.003 HS
Q4 (3.9 - 6.4, n = 23)	0.47 (0.13 - 1.72)	0.25

S: Significant P value < 0.05, HS: high significant P-value 0.001.

et al. (2011) [22]. Within the findings of this study we noted that the age predominance of the patients was mostly around 6 and 7 decade of life, this in accordance with Scarborough *et al.*, (2009); who reported the incidence of stroke increases rapidly with age [23]. The gender frequency in this work showed the male positive rate was 52% and the female positive rate was 48%; these data are agree with Townsend (2012); who said it is approximately 25% higher in men than in women. But Zhou *et al.*, (2013) found that women had worse outcomes [24] [25]. Biomarker panels can clarify the pathophysiological mechanisms of AIS and the approach to suitable therapy [26]. In this study, the elevated WBC count was associated with stroke severity at admission as noticed in SSS and more disability at discharge by MRS, these findings are in agreement with Peng *et al.*, (2011) and Nardi *et al.*, (2011) who noticed an increased WBC count at admission was significantly correlated to in-hospital death or disability later on [27] [28]. Also the demonstrated neutrophilia was adjuvant predictor correlated to how much the stroke is severe and the prognosis is bad as observed by SSS and MRS respectively. These results are in accordance with Pregl (2016) who reported that leukocytic count is useful marker for cerebral infarction [7]. ESR is an indicator of red blood cell aggregation, our findings however revealed neither the admission SSS nor the discharge MRS scores had a significant correlations with the ESR levels in AIS patients; this may be related to the small sample size of the patients or to the effects of hematocrit, plasma albumin levels, tempera-

ture and anticoagulants as said by Lakshmi *et al.*, (2011) [22]. Increased ESR may also have an indirect role in the formation of arterial thrombosis through its effects on the platelets [9] [22]. Kisialioul *et al.*, (2012) had reported that circulating platelets has importance in AIS recurrence, because of its role not only depending on their direct effects on endothelium but also by acting as a connection for other cells in vascular system [29]. Platelet activation and aggregation are critically involved in the pathophysiology of atherosclerosis, thrombosis and ischemic stroke [30]. We didn't find any significant relation between stroke severity or outcome disability and platelet count for unexplained etiology. However, the impact of enhanced platelet activation in AIS on antithrombotic therapy had been established [24]. Coagulation and hemostatic markers are important in management of AIS patients [3]. In this work the results of aPTT were significantly associated with MRS in Q3, this denotes bad prognosis. Prolonged aPTT was associated with worse stroke outcome, suggesting it's neuro-protective effect at short time (Kisialioul *et al.*, 2012) [29]. But the INR values in this study were not significantly associated with SSS or MRS, so INR can't be considered in the evaluation of AIS patients of this study, but it is more useful in monitoring thrombolytic therapy to avoid serious cerebral hemorrhage this opinion is in agree with Glushakova *et al.*, (2016) and Pan *et al.*, (2017) [3] [21]. Also in patient with AF on warfarin to prevent new lesions [31], Jickling *et al.*, (2015) reported that, plasma lipids, proteins, RNA and other biomarkers had a utility in diagnosis and evaluation stroke [5]. Increased serum TG is associated with endothelial dysfunction, atherosclerosis and the production of a prothrombotic state, the non-fasting TG were associated with increasing risk of ischemic stroke these data were reported by Varbo *et al.* (2011) [20]. In our study the higher non-fasting TG the lower of stroke severity and discharge disability was found, this in accordance with the results Dziedzic, (2004) who had correlated the SSS to non-fasting TG levels within 36 hours of admission [32]. Others reported that higher fasting TG predicts less severe disability [33]. Another study had demonstrated that low serum TG is an independent predictor of mortality after cardio-embolic stroke [19]. Serum albumin is the most abundant protein found in plasma, acts as carrier molecule, and preserves oncotic pressure also antioxidant defender in inflammatory process [34]. Among the laboratory panel used we found that, the high normal serum albumin values were associated with less discharge disability by MRS.A study was done by Lu *et al.*, (2012) showed a high-dose of human albumin, improving neurological status and reducing infarction size [35]. These are in agreement with our data in which low serum albumin was associated with elevated MRS at 7^{th} days, so albumin has neuro-protective mechanism. Ertekin *et al.* (2013) had reported that ischemic modified albumin represented a significant diagnostic value in AIS [15]. The role of SUA in AIS is still controversial, and a possible synergic role of SUA with thrombolytic therapies needs further investigations. Higher SUA levels in AIS patients receiving recombinant t-PA have been associated with better outcome at

day 90 and smaller infarct volume [18] [36]. In this study we noticed that, the elevated SUA was associated with less severe stroke and better outcome, so high SUA may be an independent predictor of AIS [17] [37]. All the discussed aspects of the laboratory procedures in AIS can postulate the importance of rapid and economic prognostic panel of investigations introduce as guidelines in practice during management the AIS patients. Limitations about this study were the small patients size may responsible the controversial results with other data, also lack of normal control in comparison due to illegibility to do brain CT or MRI to normal person, finally limited resources for follow up the infarction size at discharge.

8. Conclusion

The data of this work is necessitated the use of WBCs with differential neutrophils counted as independent outcome predictors as it has significant correlations to both SSS and MRS in the Q2, Q3, Q4, and APTT, which has a significant correlation to the MRS in Q3, also should be included. Both ESR and INR showed a non-significant correlation to both SSS and MRS so have no value as an adjuvant predictive tool for stroke outcome. Whereas the blood chemistry investigations mandate the use of serum albumin, non-fasting TG and SUA within the screening panel, they have a significant correlation to MRS in Q3, Q4 so considered as independent predictors for evaluating the functional disability of AIS patients. The small sample size in this work may be responsible for insignificant values of ESR and INR and platelets.

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