

Assessment of Renal Function in Patients of Acute Stroke- A Clinical Study

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Abstract

Background: Aim: To assess renal function in patients with acute stroke. **Methods:** Eighty- five adult patients of stroke (males- 50, females- 35) were selected and routine blood and biochemical examination such as serum creatinine, sodium, potassium, calcium, phosphorous, uric acid, fasting blood glucose, cardiac markers such as HDL, LDL, cholesterol, triglycerides were estimated. **Results:** Out of 85 cases, ischaemic stroke contributed 45 (males- 30, females-15) and haemorrhagic 40 (males- 15, females-20). Out of 85 cases of stroke, 30 had no AKI while 55 had different stages of AKI (stage 1- 35, stage 2- 15 and stage 3- 5). A significant difference was observed ($P < 0.05$). Calcium in non- AKI was 8.91 mg/dl and in AKI was 9.34 mg/dl, Phosphorous in non- AKI was 3.8 mg/dl and in AKI was 4.1 mg/dl, Uric acid in non- AKI was 6.3 mg/dl and in non- AKI was 6.7 mg/dl, FBG in non- AKI was 106.5 mg/dl and in AKI was 110.6 mg/dl, TG in non- AKI was 143.5 mg/dl and in AKI was 152.8 mg/dl, Cholesterol in non- AKI was 185.4 mg/dl and in AKI was 191.0, HDL in non- AKI was 46.5 mg/dl and in AKI was 48.7 mg/dl, LDL in non- AKI was 96.0 mg/dl and in AKI was 98.2 mg/dl. A non- significant difference was observed ($P > 0.05$). **Conclusions:** The level of cardiac markers was higher in AKI patients than non- AKI patients. There was high prevalence of AKI in patients with stroke.

Keywords:- Cardiac Markers, Acute Kidney Injury, Renal Function, Cholesterol.

INTRODUCTION

Stroke represents a continuously evolving medical and social problem, being the third leading cause of death after heart disease and cancer in developed countries.^[1] The increasing economic burden that patients with stroke impose on the already 'overloaded' social security systems of western societies, as well as the significant loss of manpower, renders the study of prognostic factors that can affect short- and long-term mortality after stroke indispensable.^[2,3] In previous years, several factors, such as C-reactive protein, glucose

levels on admission, fibrinogen concentration, erythrocyte sedimentation rate, leukocyte count, uric acid and a low tri-iodothyronine level, have been associated with a low survival rate after acute stroke.^[4]

Acute kidney injury (AKI) is a common comorbid condition in the community with different medical events which include cardiovascular disease, diabetes mellitus, hypertension and cerebrovascular stroke and hospitalisation in intensive care unit. In the immediate period following a stroke, acute kidney injury may develop as a possible

complication.^[5] AKI and its presence can be explained by the particular characteristics of the stroke-prone population: Elderly individuals (typically over 60 years), associated multiple cardiovascular comorbidities frequently treated with multiple drug associations, and usually underlying impaired renal function.^[6]

Renal dysfunction is commonly seen in hospitalized stroke patients. Ischemic stroke is frequently associated with renal dysfunction and nearly a third of patients hospitalized with intracerebral haemorrhage (ICH) have chronic kidney disease (CKD) (estimated glomerular filtration rate [e-GFR] < 60ml/minute per 1.73m²).^[7] The severity of the impairment and the requirement of renal replacement therapy for these patients in the course of their treatment are important management issues not currently well addressed by the literature. It has been established that good overall medical care can greatly influence the outcome of patients with stroke.^[8] Considering this, we selected this study to assess renal function in patients with acute stroke.

MATERIAL AND METHODS

A total of eighty- five adult patients of stroke (males- 50, females- 35) were selected for this prospective, observational study. Patients within age range 18-70 of either gender was the inclusion criteria whereas pre-existing chronic kidney disease, heart failure and glomerulitis cases were excluded. The approval was sorted from ethical review clearance committee. All selected patients' written consent was obtained after explaining the importance of the study.

All patients were evaluated clinically and subjected to routine blood and biochemical examination such as serum creatinine, sodium,

potassium, calcium, phosphorous, uric acid, fasting blood glucose, cardiac markers such as HDL, LDL, cholesterol, triglycerides.

Acute kidney injury was defined by an increase in serum creatinine by ≥ 0.3 mg/dl (≥ 26.5 μ mol/l) within 48 hours; or increase in serum creatinine to ≥ 1.5 times baseline or decrease in urine volume to < 0.5 ml/kg/h for 6 hours. All were subjected to CT scan and accordingly, stroke was classified as ischaemic or haemorrhagic. Results of the study was subjected to statistical analysis. Data was represented as mean. The test for statistical analysis applied was Mann Whitney U test where p value less than 0.05 was set as significant.

RESULTS

Out of 85 cases, ischaemic stroke contributed 45 (males- 30, females-15) and haemorrhagic 40 (males- 15, females-20) [Table 1].

Out of 85 cases of stroke, 30 had no AKI while 55 had different stages of AKI (stage 1- 35, stage 2- 15 and stage 3- 5). A significant difference was observed ($P < 0.05$) [Table 2, Figure 1].

The mean eGFR level in non- AKI was 78.2ml/min/1.73 m² and in AKI was 51.4 ml/min/1.73 m², blood urea in non- AKI was 53.2 mg/dl and in AKI was 60.7 mg/dl, s. creatinine in non- AKI was 1.76 mg/dl and in AKI was 1.93 mg/dl, MAP in non- AKI was 118.4 mm Hg and in AKI was 117.4, SBP in non- AKI was 156.2 mm Hg and in AKI was 160.3 mm Hg, DBP in non- AKI was 98.2 mm Hg and in AKI was 95.4 mm Hg, Sodium in non- AKI was 132.5 mg/dl and in AKI was 138.9 mg/dl, Potassium in non- AKI was 3.90 mg/dl and in AKI was 4.65 mg/dl, Calcium in non-

AKI was 8.91 mg/dl and in AKI was 9.34 mg/dl, Phosphorous in non- AKI was 3.8 mg/dl and in AKI was 4.1 mg/dl, Uric acid in non- AKI was 6.3 mg/dl and in non- AKI was 6.7 mg/dl, FBG in non- AKI was 106.5 mg/dl and in AKI was 110.6 mg/dl, TG in non- AKI was 143.5 mg/dl and in AKI was 152.8 mg/dl,

Cholesterol in non- AKI was 185.4 mg/dl and in AKI was 191.0, HDL in non- AKI was 46.5 mg/dl and in AKI was 48.7 mg/dl, LDL in non- AKI was 96.0 mg/dl and in AKI was 98.2 mg/dl. A non- significant difference was observed ($P > 0.05$) [Table 3, Figure 2].

Table 1: Type of stroke

Type	Male	Female
Ischaemic (45)	30	15
Haemorrhagic (40)	20	20
Total	50	35

Table 2: Cases of AKI in stroke patients

Parameters	Variables	Number	P value
No AKI		30	Significant
AKI (55)	Stage 1	35	
	Stage 2	15	
	Stage 3	5	

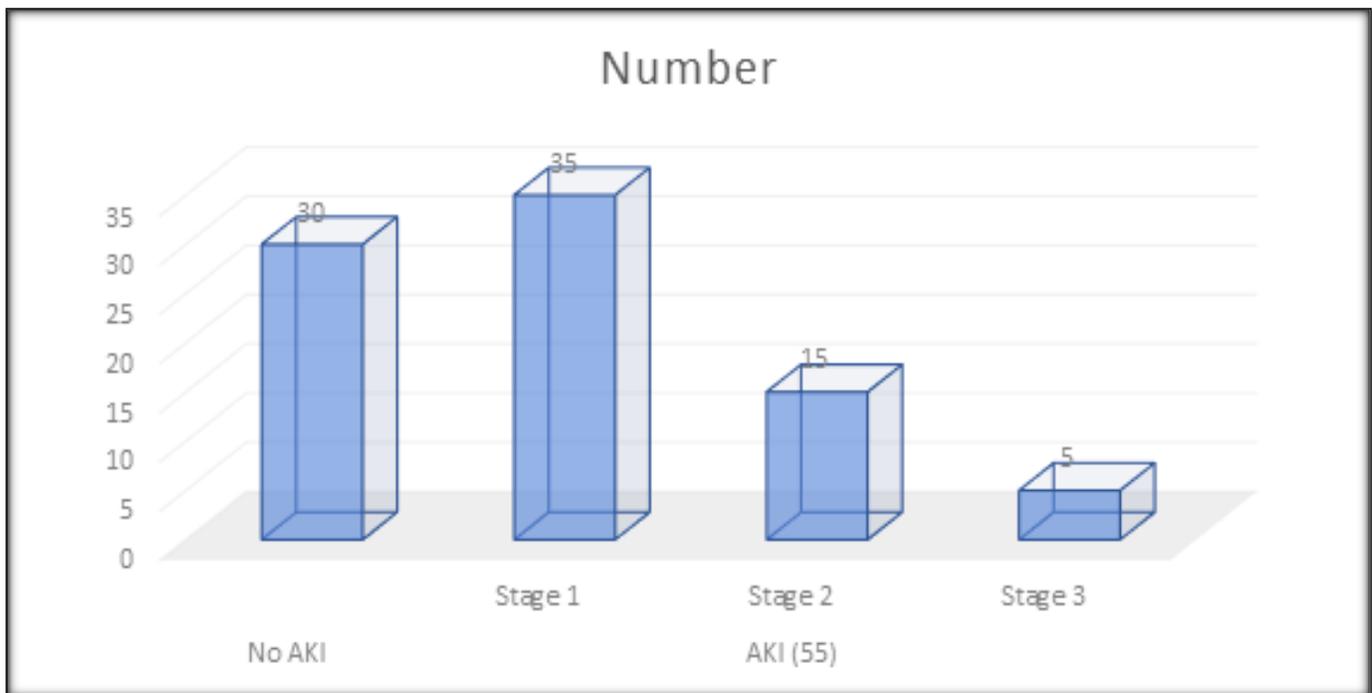


Figure 1:

Table 3: Baseline biochemical investigations of stroke patients

Parameters	Non- AKI	AKI	P value
eGFR	78.2	51.4	Significant
Blood urea	53.2	60.7	Non- significant
S. creatinine	1.76	1.93	Non- significant
MAP	118.4	117.4	Non- significant
SBP	156.2	160.3	Non- significant
DBP	98.2	95.4	Non- significant
Sodium	132.5	138.9	Non- significant
Potassium	3.90	4.65	Non- significant
Calcium	8.91	9.34	Non- significant
Phosphorous	3.8	4.1	Non- significant
Uric acid	6.3	6.7	Non- significant
FBG	106.5	110.6	Non- significant
TG	143.5	152.8	Non- significant
Cholesterol	185.4	191.0	Non- significant
HDL	46.5	48.7	Non- significant
LDL	96.0	98.2	Non- significant

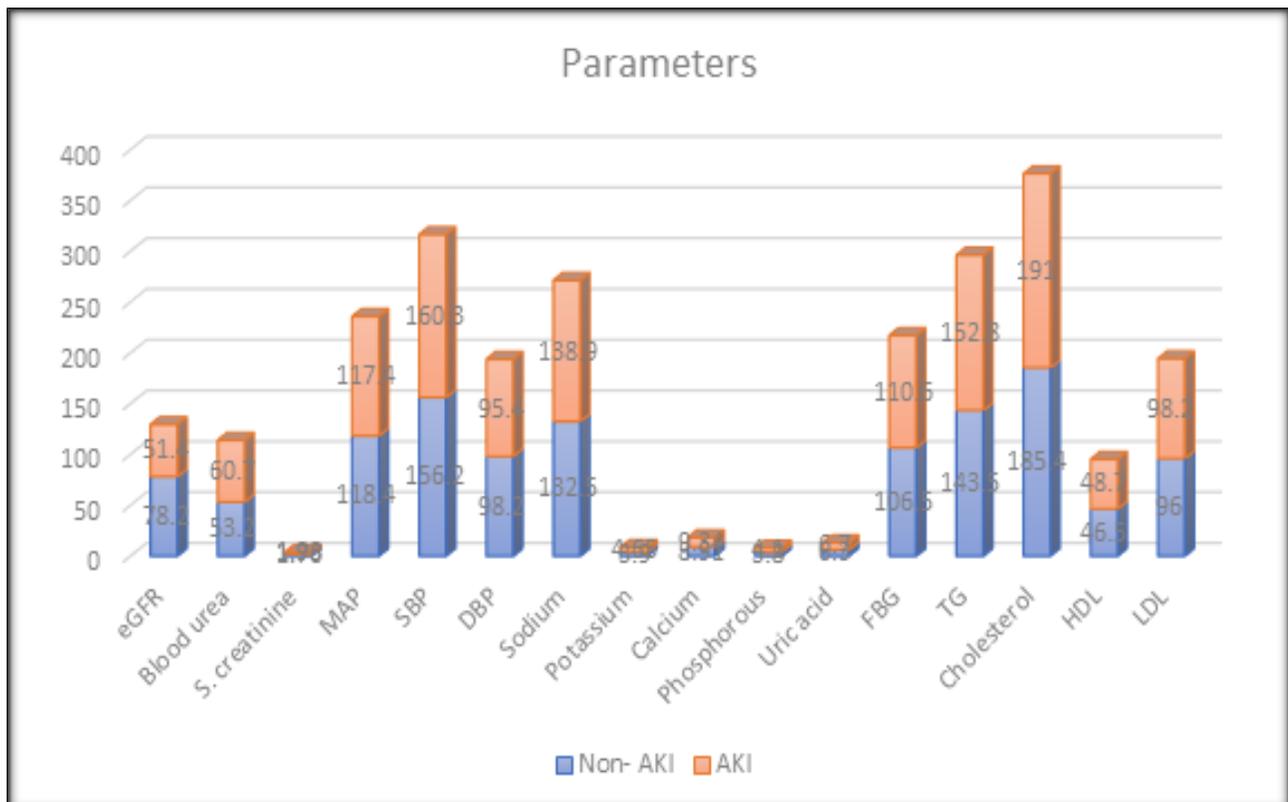


Figure 2:

DISCUSSION

This prospective, observational study was conducted to assess renal function in patients with acute stroke. Out of 85 cases, ischaemic stroke contributed 45 (males- 30, females-15) and haemorrhagic 40 (males- 15, females-20). Cerebrovascular accident itself has a high burden of morbidity and mortality, and additionally, CKD is an independent predictor of poor clinical outcome and mortality after an initial stroke.^[9] Co-existence of adverse conditions, such as anemia, oxidative stress, platelet dysfunction, electrolyte imbalance and hyperhomocysteinemia, in patients with CKD have been implicated as the reason why these patients have poorer outcomes compared to the normal population.^[10] Moreover, even mild stages of CKD increases the risk of future ischemic and haemorrhagic strokes. Renal function impairment has been associated with a high prevalence of cardiovascular disease (CVD). Patients with reduced renal function are at high risk for the subsequent development of CVD disease including stroke.^[11] Several studies in the last decade revealed that not only the risk of mortality but also the new cardiovascular events after myocardial infarction or heart failure are higher among patients with renal dysfunction.^[12,13,14] Although acute stroke is an emergency disease and shares the same atherosclerotic risk factors with ischaemic heart disease, the association of renal function and stroke is poorly investigated.^[15] Friedman investigated elderly stroke survivors and found that serum creatinine concentration independently predicted mortality during a follow-up period of 18 months.

Our study showed that out of 85 cases of stroke, 30 had no AKI while 55 had different stages of AKI (stage 1- 35, stage 2- 15 and stage 3- 5). Aggarwal et al,^[16] evaluated renal function in patients of acute stroke and explore the potential usefulness of preventive measures and early interventions to reduce morbidity and mortality due to renal dysfunction. Amongst 100 patients of stroke, 59 (59.0%) were ischaemic while rest were haemorrhagic stroke (41.0%). Mean age of study subjects was 60.91 ± 8.31 years. Mean age of ischaemic stroke subjects was higher (62.27 ± 7.20 years) as compared to haemorrhagic stroke (58.95 ± 9.45 years). Baseline Serum creatinine and blood urea was significantly higher in hemorrhagic stroke patients compared to ischaemic stroke subjects ($p < 0.01$) while e-GFR was significantly lower in haemorrhagic stroke patients (56.09 ± 28.41 ml/min/1.73 m²) in comparison to ischaemic stroke patients (86.60 ± 26.73 ml/min/1.73 m²). Duration of stay in hospital (days) was significantly higher in haemorrhagic stroke (12.71 ± 5.12 days) compared to ischaemic stroke subjects (9.41 ± 2.78) days. Acute kidney injury was seen in 24% of stroke patients. AKI was more common in haemorrhagic stroke patients (34.3%) as compared to ischaemic stroke patients (16.9%). Diabetes was significantly associated with development of AKI (54.2%) as compared to non-AKI (15.8%). Mortality rate in stroke patients was 12%.

We observed that The mean eGFR level in non-AKI was 78.2 ml/min/1.73 m² and in AKI was 51.4 ml/min/1.73 m², blood urea in non- AKI was 53.2 mg/dl and in AKI was 60.7 mg/dl, s. creatinine in non- AKI was 1.76 mg/dl and in AKI was 1.93 mg/dl, MAP in non- AKI was 118.4 mm Hg and in AKI was 117.4, SBP in non-AKI was 156.2 mm Hg and in AKI was 160.3

mm Hg, DBP in non- AKI was 98.2 mm Hg and in AKI was 95.4 mm Hg, Sodium in non- AKI was 132.5 mg/dl and in AKI was 138.9 mg/dl, Potassium in non- AKI was 3.90 mg/dl and in AKI was 4.65 mg/dl. Shrestha et al,^[17] estimated glomerular filtration rate (e-GFR) and the trend of renal function in the two stroke subgroups (haemorrhagic and ischemic) with renal impairment defined as e-GFR < 60mL/minute per 1.73m². Among 52 patients, 25 had haemorrhagic stroke (mean age 59.81 ±14.67) and 27 had ischemic stroke (mean age 56.12 ± 13.08). The mean e-GFR (mL/minute per 1.732m²) at admission in the haemorrhagic stroke subgroup was 64.79 ± 25.85 compared to 86.04 ± 26.09 in the ischemic stroke subgroup. Sixteen out of 25 (64%) patients in the haemorrhagic stroke subgroup and 9 out of 27 (33.3%) patients in the ischemic subgroup developed renal impairment. The location of the bleed (p=0.8), volume of hematoma and surgical intervention did not predispose the patients to renal impairment. One out of 16 patients with haemorrhagic stroke (who

eventually died), and 2 out of 9 patients with ischemic stroke required renal replacement.

We observed a non- significant difference in serum calcium, phosphorous, uric acid, FBG, TG, Cholesterol, HDL, LDL in non- AKI was 96.0 mg/dl and in AKI was 98.2 mg/dl. Tsagalis et al,^[18] found that almost 1/3 (28.08%) of our acute stroke patients presented with moderate (group B) or severe (group C) renal dysfunction as estimated by eGFR. After adjusting for basic demographic, stroke risk factors and stroke severity on admission, eGFR was an independent predictor of stroke mortality at 10 years. Patients in groups B and C had an increased probability of death during follow-up: Hazard ratio = 1.21 with 95% CI 1.01–1.46, p < 0.05 and Hazard ratio = 1.76 with 95% CI 1.14–2.73, p < 0.05 respectively, compared to patients belonging to group A.

CONCLUSIONS

There was high prevalence of AKI in patients with stroke. The level of cardiac markers were higher in AKI patients than non- AKI patients.

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