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Original Research Article

RENAL FUNCTION ASSESSMENT IN ACUTE STROKE PATIENTS AND ITS ASSOCIATION WITH IN-HOSPITAL MORTALITY

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Abstract

Introduction: Stroke is one of the world's leading causes of death, and it causes significant impairment that has a profound effect on the long-term survival of patients. Not only neurological defects, but also medical co-morbidities decide the adverse effects after cerebrovascular stroke (CVS). In different studies, related kidney dysfunction has been shown to be a significant predictor. However, considering the elevated burden of stroke patients in India, there is a shortage of such studies in India. The current research was therefore conducted to assess renal function in acute stroke patients and to investigate the possible utility of preventive and early intervention interventions to minimize morbidity and mortality due to renal dysfunction.

Methods: This was a 1-year, longitudinal, retrospective analysis of 25 adult acute stroke patients who were admitted to the hospital. On the day of admission, and then on days 3, 7 and 14, patients underwent routine laboratory investigations, including baseline biochemical investigations. The approximate glomerular filtration rate (e-GFR) was determined and the renal function pattern was evaluated in the two stroke subgroups. Creatinine was known as the baseline for entry. During hospitalization, AKI was characterized as a 0.3 mg/dl creatinine increase or a percentage increase of at least 50 percent from baseline.

Results: Of the 25 stroke patients, 15 (59%) were ischemic while the remainder were hemorrhagic (41 percent). The mean age of the subjects examined was 60.42 ± 8.45 years. In contrast to hemorrhagic stroke, the mean age of subjects with ischaemic stroke was higher (62.65 ± 7.32 years) (58.72 ± 9.86 years). Baseline Serum creatinine and blood urea were significantly higher in patients with hemorrhagic stroke relative to subjects with ischaemic stroke ($p < 0.01$), although e-GFR was significantly lower in patients with hemorrhagic stroke (56.12 ± 28.34 ml/min/1.73 m2) compared to patients with ischaemic stroke (86.34 ± 26.68 ml/min/1.73 m2).

The hospital stay period (days) was substantially higher in patients with hemorrhagic stroke (12.43 ± 5.36 days) relative to subjects with ischemic stroke (9.54 ± 2.65 days). In 24 per cent of stroke patients, acute kidney damage has been seen. In hemorrhagic stroke patients (34 percent), AKI was more common compared to ischemic stroke patients (17 percent). In contrast to non-AKII, diabetes was substantially correlated with the production of AKI (54 percent) (16 percent). In stroke patients, the mortality rate was 12% . Statistically, there were no variations between the ischemic stroke mortality rate (12%) and hemorrhagic stroke (12 percent). The mortality rate was 29% among patients who developed AKI . The predictors of AKI were found to be hemorrhagic stroke, older age, diabetes mellitus and high baseline creatinine levels. It has been found that GCS score < 10, AKI, hemorrhagic stroke and AKI requiring renal replacement therapy are associated with longer hospital stays. In patients with aspiration pneumonia, GCS score < 10, AKI, older age and AKI needing Renal Replacement Therapy, mortality was significantly more likely.

Conclusion: In our research, the predictors of AKI were found to be hemorrhagic stroke, older age, high baseline creatinine and diabetes mellitus. It was also found that AKI was an autonomous indicator of extended hospital stay and increased mortality among stroke patients.

Key words: Cerebrovascular stroke (CVS), acute kidney injury (AKI), Glasgow Coma Score (GCS).

Introduction

Stroke is characterized as' the rapidly evolving clinical symptoms and/or signs of local or often global cerebral function disturbance, with symptoms that last longer than 24 hours or lead to death with no obvious cause other than vascular origin.' In view of renal dysfunction, there is growing evidence in medical literature of the involvement of cardiovascular and cerebro-vascular diseases in renal dysfunction. In the population, AKI is a common co-morbid disorder with multiple medical events that include cardiovascular disease, diabetes mellitus, hypertension and cerebrovascular stroke, and intensive care unit hospitalization[1]. Acute kidney injury (AKI) can develop as

a potential complication in the immediate period following a stroke[2]. The basic features of the stroke-prone population can be explained by AKI and its presence: elderly people (typically over 60 years), various cardiovascular comorbidities often associated with multiple drug associations, and generally underlying impaired renal function. A number of important risk factors for stroke and kidney failure result in higher morbidity and mortality in stroke patients. Nearly all forms of vascular disease, including stroke, have been shown to be associated with impairment of renal function, and the severity of stroke in small renal vessels may represent the degree of injury[3,4]. In different trials, the median general incidence of AKI in stroke is 5 percent . Compared to ischemic stroke, AKI incidence is higher in hemorrhagic stroke (19 percent) (10 percent). Even after adjustment among stroke survivors, serum creatinine was found to be an independent indicator of mortality and increased hospital stay time. While AKI is prevalent and has a high disease burden (morbidity and mortality), it is ideal for prevention, early diagnosis and treatment[5,6]. There is controversial and scarce evidence on the relationship between renal dysfunction and outcomes in stroke patients. Different studies have previously been performed to examine renal impairment in acute stroke patients and have conclusively established AKI as an independent indicator of morbidity and mortality in these patients. However, considering the elevated burden of stroke patients in India, there is a shortage of such studies in India[7,8]. The present research was therefore designed to assess renal function in patients with acute stroke and to investigate the possible utility of preventive and early intervention interventions to minimize morbidity and mortality in patients with acute stroke due to renal dysfunction[9].

Material and Methods

Study design

This was a one-year, prospective, observational study of 25 adult acute stroke patients who were admitted to the hospital. Patients aged 18 to 75 years who had a stroke within 7 days were included in the report. The research omitted patients with pre-existing chronic kidney disease, developing cardiac disease or undergoing cardiothoracic surgery, heart failure, all-cause glomerulonephritis, and urinary tract obstruction. Stroke has been identified as a cerebrovascular neurological deficit reported by computed tomography (CT) or magnetic resonance imaging (MRI) that persists beyond 24 hours or is interrupted by death within 24 hours. Based on the CT admission scan, stroke was graded as ischemic or hemorrhagic. Acute kidney injury was characterized as an increase in serum creatinine by >0.3 mg/dl (>26.5 micro mol/l) within 48 hours; or an increase in serum creatinine by >1.5 times the baseline stated or suspected to have occurred within the previous 7 days; or a decrease in the volume of urine by <0.5 ml/kg/h within 6 hours. Written consent was obtained from the patient or caretaker for the study after receiving approval from the Institutional Ethics Committee and due consideration of the inclusion and exclusion criteria. During hospitalisation, all patients were assessed. For all topics that were included in the research, a detailed history and clinical review was carried out. On the day of admission, patients underwent routine laboratory investigations, including baseline biochemical investigations, accompanied by results on days 3, 7 and 14.

Statistical analysis: The data obtained has been transformed, coded and entered in Microsoft Excel into variables. Using SPSS-PC-17 version11, the data was analyzed and statistically evaluated. Quantitative information was expressed in mean, standard deviation, and the difference between two equivalent groups was calculated by the student t-test (unpaired) or Mann Whitney U 'test. Percentages of qualitative data were expressed. The chi square test or Fisher's exact test measured statistical differences between the proportions. The p value was found statistically important to be less than 0.05.

Results

More than half of the research subjects in the current study were males (58 percent). The mean age of the subjects examined was 60.42 ± 8.45 years. In the age group of 56 to 65 years, most of the research subjects were (39 percent). More than half of the patients had ischemic stroke (59%) and the remainder had hemorrhagic stroke (41 percent). In 24 percent of stroke patients, acute kidney damage was found, of which 3 (13 percent) were in stage 1 AKI, 2 in stage 2 AKI and 1 in stage 3 AKI. In hemorrhagic stroke patients (34 percent), AKI was more common compared to ischemic stroke patients (17 percent). It was found that this disparity was statistically important ($p < 0.05$). A total of 3 ischaemic stroke patients developed AKI, of which 1 was stage 1, 1 was stage 2, and 1 was stage 3 of AKI. A total of 4 hemorrhagic stroke patients developed AKI, of which 2 were in stage 1, 1 was in stage 2 and 1 was in stage 3 (Table I).

Table I: AKI according to type of stroke.

		Ischaemic stroke (n = 15)		Haemorrhagic stroke (n = 10)	
		No.	**%**	**No.**	**%**
No AKI		12	83**%**	7	66**%**
AKI	Stage 1	1	9**%**	1	20**%**
	Stage 2	1	5**%**	1	10**%**
	Stage 3	1	3**%**	1	5**%**

Baseline serum creatinine and blood urea were significantly higher in patients with hemorrhagic stroke compared to patients with ischaemic stroke ($p<0.01$), whereas e-GFR was significantly lower in patients with hemorrhagic stroke (56.12 ± 28.34 ml/min/1.73 m2) compared to patients with ischaemic stroke (86.34 ± 26.68 ml/min/1.73 m2) (Table II).

Table II: Comparison of baseline biochemical parameters in stroke patients

Baseline investigations	Ischaemic stroke (n = 59)	Haemorrhagic stroke (n = 41)
	Mean ± SD	Mean ± SD
eGFR (ml/min/1.73 m2)	86.34 ± 26.68	56.12 ± 28.34
S. creatinine (mg/dl)	1.12 ± 0.68	1.72 ± 1.24
Blood urea (mg/dl)	45.24 ±18.68	68.42 ± 47.86
MAP (mmHg)	111.16 ± 4.46	128.26 ± 17.64
Systolic BP (mmHg)	149.84 ± 7.34	174.26 ± 18.74
Diastolic BP (mmHg)	91.64 ± 3.56	105.34 ± 17.28
Sodium (meq/l)	134.42 ± 7.26	138.64 ± 7.34
Potassium (meq/l)	3.45 ± 0.22	4.16 ± 0.32
Serum calcium (mg/dl)	9.12 ± 0.26	9.22 ± 0.32
Serum phosphorus (mg/dl)	4.4 ± 0.14	4.2 ± 0.4
Serum uric acid (mg/dl)	6.2 ± 1.2	6.12 ± 1.8
Fasting blood sugar (mg/dl)	107.16 ± 22.17	109.17 ± 19.24
Triglycerides (mg/dl)	151.26 ± 34.27	149.19 ± 31.36
Total cholesterol(mg/dl)	188.90 ± 44.43	186.23 ± 40.30
HDL (mg/dl)	46.38 ± 11.27	48.21 ± 13.45
LDL (mg/dl)	96.15 ± 10.27	98.32 ± 10.52

Compared with non-AKI (16%), diabetes was significantly correlated with the production of AKI (54%) (Table III). A lower e-GFR was observed among the baseline biochemical investigations in AKI patients and this discrepancy was statistically significant. Other parameters were higher in AKI patients compared to non-AKI patients, such as blood urea, serum creatinine, fasting blood sugar, systolic BP, diastolic BP, and MAP, but the difference was not statistically important (Table IV). During the hospital stay, mean blood urea, mean serum creatinine and mean eGFR were continuously changing, but these values did not achieve statistical significance. There was no major improvement in the remaining parameters, including sodium (meq/l), potassium (meq/l), serum calcium (mg/dl), serum phosphorus (mg/dl), serum uric acid (mg/dl), lipid profile and serum albumin (g/dl) during the hospital stay (Table V). Related results were found in the subgroup study of patients with hemorrhagic and ischemic stroke.

Table III: Comparison of baseline medical history of stroke patients (AKI versus non-AKI).

Medical history	Non-AKI (n = 19)		AKI (n = 6)	
	No.	%	No.	%
Gender				
Male	12	63%	3	50%
Female	7	37%	3	50%
Medical history				
H/o Smoking	16	84%	5	83%
H/o Alcohol	14	74 %	5	83%
H/o Hypertension	16	84 %	6	100%
H/o Diabetes	3	16 %	3	50%
H/o Coronary artery disease	2	11%	1	17%
H/o Heart failure	1	5 %	0	0%
H/o Atrial fibrillation	4	21 %	1	17%
H/o Stroke	3	16 %	1	17%
H/o TIA	2	11 %	1	17%
Hypercholesterolaemia	6	32%	2	33%
Carotid artery stenosis	4	21%	1	17%

Table IV: Comparison of baseline biochemical investigations of stroke patients (AKI versus non-AKI).

Baseline investigations	Non-AKI (n = 19)	AKI (n = 6)
	Mean ± SD	Mean ± SD
eGFR (ml/min/1.73 m2)	77.36 ± 31.22	53.42 ± 28.32
S. creatinine (mg/dl)	1.39 ± 1.43	1.44 ± 2.22
Blood urea (mg/dl)	52.43 ± 34.22	59.44 ± 38.26
MAP (mmHg)	118.25 ± 14.26	117.11 ± 14.22
Systolic BP (mmHg)	158.43 ± 18.05	160.32 ± 17.21
Diastolic BP (mmHg)	97.34 ± 13.09	96.04 ± 13.22
Sodium (meq/l)	134.34 ± 6.45	137.32 ± 8.15
Potassium (meq/l)	3.45 ± 0.22	4.32 ± 0.34
Serum calcium (mg/dl)	8.46 ± 0.33	9.16 ± 0.22
Serum phosphorus (mg/dl)	3.4 ± 0.4	4.4 ± 0.34
Serum uric acid (mg/dl)	6.2 ± 1.15	6.3 ± 1.2
Fasting blood sugar (mg/dl)	107.42 ± 20.42	110.23 ± 22.24
Triglycerides (mg/dl)	149.32 ± 31.34	153.33 ± 33.12
Total cholesterol(mg/dl)	186.45 ± 41.36	191.32 ± 43.36
HDL (mg/dl)	46.22 ± 11.46	48.42 ± 13.21
LDL (mg/dl)	96.24 ± 9.17	98.36 ± 11.23

In stroke patients who developed AKI, the hospital stay period (days) was substantially higher. The results of the subgroup study of ischaemic and hemorrhagic stroke patients were similar. However, the hospital stay period was higher in patients with hemorrhagic stroke (12.43 ± 5.36 days) relative to subjects with ischaemic stroke (9.54 ± 2.65 days) and the difference was statistically relevant ($p < 0.01$). GCS score < 10, AKI, stroke form and renal replacement therapy criteria for AKI have been shown to be correlated with longer hospital stays (Table VI

Table V: Biochemical parameters in stroke patients during hospital stay.

Investigation	Day of admission	Day 3	Day 7	Day 14
Haemoglobin	12.21 ± 2.34	13.34 ± 1.46	12.39 ± 2.39	12.21 ± 2.08
Total leukocyte count (cells/cumm)	12648 ± 3735.6	11445 ± 2838.21	10732 ± 2364.2	10626 ± 2017.2
Blood urea (mg/dl)	45.10 ± 18.15	50.32 ± 21.16	46.34 ± 13.08	45.07 ±10.17
Serum creatinine (mg/dl)	1.02 ± 0.40	1.42 ± 0.44	1.08 ± 0.34	1.02 ± 0.46
Sodium (meq/l)	134.21 ± 7.26	135.22 ± 6.16	135.32 ± 6.46	135.22 ± 8.17
Potassium (meq/l)	3.46 ± 0.22	4.02 ± 0.23	4.03 ± 0.21	3.46 ± 0.18
Serum calcium (mg/dl)	9.02 ± 0.26	9.22 ± 0.18	9.23 ± 0.36	9.15 ± 0.32
Serum phosphorus (mg/dl)	4.1 ± 0.35	3.45 ± 0.14	3.4 ± 1.2	4.2 ±0.45
Serum uric acid (mg/dl)	6.2 ± 1.2	6.4 ± 1.2	5.45 ± 1.3	5.4 ±1.2
eGFR (ml/min/1.73 m2)	86.30 ± 26.36	69.32 ± 28.24	65.36 ± 26.34	63.34 ±24.42
Fasting blood sugar (mg/dl)	108.32 ± 21.22	107.24 ± 26.6	104.46 ± 19.22	108.39 ± 23.12
Triglycerides (mg/dl)	150.82 ± 32.30	148.21 ± 30.30	139.43 ± 33.28	135.23 ± 34.21
Total cholesterol (mg/dl)	187.21 ± 42.22	191.32 ± 39.23	188.32 ± 34.22	192.17 ± 35.44
HDL (mg/dl)	47.13 ± 12.23	44.04 ± 11.41	46.36± 13.24	45.42 ± 12.32
LDL (mg/dl)	97.11 ± 10.46	94.35 ± 9.38	97.36 ± 9.22	94.09 ± 8.58
Serum albumin (g/dl)	3.44 ± 1.4	3.4 ± 1.12	4.2 ± 0.42	3.4 ± 1.33

Predictors of AKI were found to be hemorrhagic stroke, older age, diabetes mellitus and elevated baseline creatinine levels (Table VII). 3 people died out of 25 stroke patients, so the mortality rate for stroke patients was 12%. Statistically, there was little difference between the mortality rate of ischaemic stroke patients (12 percent) and hemorrhagic stroke patients (12 percent). 2 of the 3 patients who died had AKI, so mortality in stroke patients was substantially correlated with AKI. In patients with aspiration pneumonia, GCS score < 10, AKI, elderly age and RRTT requirement, mortality was significantly more likely (Table VIII).

Table VI: Predictors of hospital stay among stroke patients.

	Mean ± SD
GCS score	
< 10	12.38 ± 5.26
□□10	9.42 ± 3.17
AKI status	
AKI	14.23 ± 4.22
No AKI	9.26 ± 3.20
Hypertension	
Yes	10.45 ± 4.14
No	9.22 ± 3.26
Type of stroke	
Ischaemic	9.21 ± 2.34
Haemorrhagic	12.33 ± 5.06
Need for renal replacement therapy	
Yes	22.11 ± 1.21
No	10.46 ± 3.78

Table VII: Predictors of AKI among stroke patients.

	Non-AKI	AKI
Haemorrhagic stroke (n = 10)	7 (70%)	3 (30%)
Ischaemic stroke (n = 15)	12 (80%)	3 (20%)
Hypertension (mmHg)	16 (86%)	6 (100%)
Age (in years)	59.11 ± 7.22	64.14 ± 9.32
Baseline creatinine (mg/dl)	1.28 ± 0.42	1.40 ± 1.03
Diabetes mellitus	3 (16%)	3 (54%)
Smoking	13 (83%)	5 (83%)
Alcohol	14 (75%)	5 (79%)

Table VIII: Predictors of death among stroke patients.

	Alive (n = 22)	Died (n = 3)
Aspiration pneumonia	7 (32%)	2 (67%)
GCS score < 10	6 (27%)	3 (100%)
AKI	5 (23%)	2 (67%)
Age	60.23 ± 7.56	66.25 ± 9.84
Hypertension	19 (86%)	3 (100%)
Renal replacement therapy	0 (0.0%)	1 (33%)
Type of stroke		
Ischaemic	13 (59%)	2 (67%)
Haemorrhagic	9 (41%)	1 (33%)

Discussion

AKI may be correlated with the short-term evolution following stroke as a potential complication, which is often ignored and underestimated in clinical trials. We demonstrated in our research that the presence of AKI is not an uncommon finding in patients with stroke. In the population, AKI is a common co-morbid disorder with multiple medical events that include cardiovascular disease, diabetes mellitus, hypertension and cerebrovascular stroke, and intensive care unit hospitalization[10]. There is growing evidence in medical literature of the function of the kidney, like stroke, in the development of cardiovascular disease. While the majority of stroke patients had mild AKIs that were "clinically" reversible in most cases, it should be emphasized that this does not always hold true at the tissue level. During kidney repair, endothelial damage, tubular inflammation, and activation of the intrarenal fibrotic pathways can gradually damage the structure of the kidney, resulting in proteinuria, hypertension, and a progressive decrease in renal function. Renal dysfunction, especially in atherosclerotic risk factors and diseases, may suggest a higher comorbidity burden[11]. The degree of renal impairment present in patients with stroke tends to simply be a marker of end-organ damage due to long-standing arterial stiffness of small and large arteries due to atherosclerosis and its related vascular risk factors (e.g., aging, smoking, high blood pressure, diabetes mellitus, and cardiovascular disease) or to be independent of other

atherosclerosis risk factors. In other words, different common risk factors for stroke and kidney failure result in higher morbidity and mortality in stroke patients[12]. Acute kidney damage was seen in our study in 24 percent of stroke patients, 13 percent of whom were in stage 1, 7 percent were in stage 2 and 4 percent were in stage 3. Our findings were consistent with the results of Tsagalis G et al[8], who stated that after admission to the hospital, a total of 27 percent of patients developed AKI. 21 percent of patients were graded as stage 1, 3 percent as stage 2, and 3 percent as stage 3 on the basis of the AKIN staging scheme. The increased mean age, low baseline GFR, and the use of a high-sensitivity description for the detection of AKI can explain the high incidence of AKI in our population. A significant contributor to the incidence of AKI could be pre-existing renal dysfunction. In hemorrhagic stroke patients, AKI was more frequent (34 percent) compared to ischemic stroke patients (17 percent). It was found that this disparity was statistically important ($p < 0.05$). The higher incidence of renal failure seen in the subgroup of hemorrhagic stroke may be due to the inherent disparity in the treatment of hemorrhagic and ischemic stroke with regard to the use of mannitol, nephrotoxic drugs, different antibiotics, etc. In their study, Khatri et al[13] also reported a higher incidence of AKI (18%) with slightly higher rates of intracerebral hemorrhage in their study (21 percent v 14 percent). 3 people died out of 25 stroke patients. Statistically, there was no difference between the mortality rate of patients with ischaemic stroke and hemorrhagic stroke. Mortality was also significantly correlated with AKI. Of the 6 patients who acquired AKI, 2 died, so the mortality in AKI patients was 33 percent. This is consistent with research in other acute care environments using the same data collection. In a study by Covic et al[14] on stroke victims, the overall incidence of AKI was 15%, with a 43% unadjusted 30-day mortality rate, compared to 13% for non-AKI topics. In our study, AKI predictors were found to be hemorrhagic stroke, older age, diabetes mellitus, and high baseline creatinine levels. Similar studies have also indicated that AKI patients were older, had a higher incidence of atrial fibrillation and heart failure, and had a neurological deficiency that was more severe than those without AKI. It has been found that GCS score < 10, AKI, hemorrhagic stroke, and the need for renal replacement therapy are correlated with longer hospital stays. Spratt et al[15] identified impairment at acute hospital discharge (Rankin score > 2), more than 65 years of age, diabetes and infection as a major risk factor for extended stay. In patients with hemorrhagic stroke, Shrestha et al[12] also reported a higher mean hospital stay relative to the ischemic stroke community. In our study, in patients with aspiration pneumonia, GCS score < 10, AKI, older age and need for renal replacement therapy, mortality was significantly more likely[13,14]. So, AKI was discovered to be a mortality indicator. Similar studies have also recorded that AKI was an independent 10-year mortality indicator ($p < 0.01$) after stroke and new composite cardiovascular events ($P < 0.05$) after adjustment for available confounding variables. Another research stated that the most important predictors of mortality were delays in consciousness recovery and new onset of acute myocardial infarction and aspiration pneumonia, age (> 60 years), severity of neuro-deficiency (GCS < 7, grade/N motor weakness), size of lesion (infarction > 1 lobe, hemorrhage > 60 ml). In patients hospitalized with acute stroke, the incidence of impaired kidney function is associated with increased all-cause mortality independent of age, sex, and significant co-morbidities[15,16].

Conclusion:

Based on the parameters such as hemorrhagic stroke, higher age, high baseline creatinine and diabetes mellitus found in our study to independently predict its growth, our findings highlight the significance of evaluating patients with stroke at risk of developing AKI. There was also a comparable chance of prolonged hospitalization and increased mortality in stroke patients. Additional studies are needed to determine whether AKI development after stroke is a causal relationship or is merely a marker of end-organ damage due to atherosclerosis and its related vascular risk factors such as aging, smoking, hypertension, diabetes mellitus due to long-standing arterial stiffness of small and large arteries. Further research should be planned to evaluate whether treatments aimed at actively preventing the occurrence of AKI or treating early AKI symptoms will lead to decreased mortality from stroke.

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