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Evaluation of Renal Function in Patients of Acute Stroke and its Relationship with in-hospital Mortality

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Abstract

Introduction: *Stroke is one of the leading causes of mortality worldwide and it causes serious disability which has a profound impact on patients' long-term survival.The adverse outcomes following cerebrovascular stroke (CVS) are determined not only by the neurological deficits but also by medical co-morbidities. Associated kidney dysfunction has been found to be a major predictor in various studies. However, there is scarcity of such studies in India despite the high burden of stroke patients in India. Thus, the present study was done to evaluate 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.*

Methods: *This was a 1 year prospective, observational study conducted on 100 adult patients of acute stroke who reported to hospital .The patients underwent routine laboratory investigations including baseline biochemical investigations on the day of admission, and then subsequently on day 3, 7 and 14. Estimated glomerular filtration rate (e-GFR) was calculated and the trend of renal function in the two stroke subgroups was assessed. Admission creatinine was considered to be the baseline. AKI was defined as a creatinine increase during hospitalisation of 0.3 mg/dl or a percentage increase of at least 50% from baseline.*

Results: *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 m^2) in comparison to ischaemic stroke patients (86.60 ± 26.73 ml/min/1.73 m^2) .*

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%. Statistically no differences were observed between mortality rate of ischaemic stroke (11.9%) and haemorrhagic stroke (12.2%). Mortality rate among patients who developed AKI was 29.2%. Haemorrhagic stroke, older age, diabetes mellitus and high baseline creatinine level were found to be predictors of AKI. GCS score < 10, AKI, haemorrhagic stroke and AKI requiring renal replacement therapy were found to be associated with longer hospital stay. Mortality was significantly more common in patients with aspiration pneumonia, GCS score < 10, AKI, older age and AKI requiring Renal Replacement Therapy.

Conclusion: *Haemorrhagic stroke, older age, high baseline creatinine and diabetes mellitus were found in our study to be predictors of AKI. AKI was also found to be an independent predictor of prolonged hospital stay and increased mortality among patients of stroke.*

Key words: *Cerebrovascular stroke (CVS), AKI.*

Introduction

Stroke is defined as "the rapidly developing clinical symptoms and/or signs of local or at times global disturbance of cerebral functions, with symptoms lasting for more than 24 hours or leading to death with no apparent cause other than that of vascular origin[1]." There is growing evidence in medical literature of the role of cardiovascular and cerebrovascular diseases in renal dysfunction in view of the similarities between vascular beds of the kidney, heart and brain. Renal dysfunction may indicate a higher comorbidity burden, especially of atherosclerotic risk factors and diseases[2].

AKI is a common co-morbid 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 (AKI) may develop as a possible complication. AKI and it's presence can be explained by the particular characteristics of the stroke-prone population: Elderly individuals (typically over 60 years), associated multiple cardiovascular co-

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morbidities frequently treated with multiple drug associations, and usually underlying impaired renal function. Various common risk factors between stroke and kidney dysfunction lead to a higher morbidity and mortality in patients of stroke. Almost all types of vascular disease including stroke have been found to be associated with renal function impairment and severity of stroke could reflect the degree of injury in small renal vessels[3,4]. The estimated overall incidence of AKI in stroke in various studies is 4.63%. Incidence of AKI is more in haemorrhagic stroke (19.17%) as compared to ischaemic stroke (9.62%). Serum creatinine has been found to be an independent predictor of mortality and increased duration of hospital stay even after adjustment among stroke survivors[5-9]. Although, AKI is common and imposes a heavy burden of illness (morbidity and mortality), it is amenable to prevention, early detection and treatment.

Data on the association between renal dysfunction and outcome in patients with stroke are controversial and scarce. Various studies have been done previously to assess renal dysfunction in patients of acute stroke and have conclusively stated AKI as an independent predictor of morbidity and mortality in these patients. However, there is scarcity of such studies in India despite the high burden of the stroke patients in India. Thus, the present study was planned to evaluate 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 in patients of acute stroke.

Material and methods

Study design

This was a one year prospective, observational study conducted on 100 adult patients of acute stroke who reported to hospital. Patients in the age range of 18 - 75 years who presented within 7 days of stroke were included in the study. Patients with pre-existing chronic kidney disease, emergent cardiac disease or undergoing cardiothoracic surgery, heart failure, glomerulonephritis from any cause and urinary tract obstruction were excluded from the study.

Stroke was defined as neurological deficit of cerebrovascular origin documented by computerised tomography (CT) or magnetic resonance imaging (MRI) that persists beyond 24 hours or is interrupted by death within 24 hours. Stroke was classified as ischaemic or haemorrhagic, based on CT scan on admission. Acute kidney injury was defined by an increase in serum creatinine by ≥ 0.3 mg/dl (≥ 26.5 µmol/l) within 48 hours; or increase in serum creatinine to ≥ 1.5 times baseline, which is known or presumed to have occurred within the prior 7 days; or decrease in urine volume to < 0.5 ml/kg/h for 6 hours[10].

After obtaining clearance from the Institutional Ethics Committee and due consideration of inclusion and exclusion criteria, written consent was taken from patient or caretaker for the study. All patients were evaluated during hospitalisation. A detailed history and clinical examination was done in all subjects who were included into the study. The patients underwent routine laboratory investigations including baseline biochemical investigations on day of admission, and then subsequently on day 3, 7 and 14 for outcome.

Statistical analysis: The collected data were transformed into variables, coded and entered in Microsoft Excel. Data were analysed and statistically evaluated using SPSS-PC-17 version[11].

Quantitative data was expressed in mean, standard deviation and difference between two comparable groups was tested by student's t-test (unpaired) or Mann Whitney U' test. Qualitative data were expressed in percentage. Statistical differences between the proportions were tested by chi square test or Fisher's exact test. p value less than 0.05 was considered statistically significant.

Results

In the present study more than half of study subjects were males (58.0%). Mean age of study subjects was 60.91 ± 8.31 years. Most of the study subjects were in the age group of 56 - 65 years (39.0%). More than half of the patients had ischaemic stroke (59.0%) while rest had haemorrhagic stroke (41.0%).

Acute kidney injury was seen in 24% of stroke patients out of which 13 (13.0%) were in stage 1 AKI, 7 were in stage 2 AKI and 4 in stage 3 AKI. AKI was more common in haemorrhagic stroke patients (34.3%) compared to ischaemic stroke patients (16.9%). This difference was found to be statistically significant ($p < 0.05$). A total 10 patients with ischaemic stroke developed AKI, out of which 5 were in stage 1, 3 were in stage 2 and 2 were in stage 3 AKI. A total 14 patients with haemorrhagic stroke developed AKI out of which 8 were in stage 1, 4 were in stage 2 and 2 were in stage 3 AKI (Table I).

Table I: AKI according to type of stroke.

		Ischaemic stroke (n = 59)		Haemorrhagic stroke (n = 41)		P value
		No.	%	No.	%	
No AKI		49	83.1	27	65.7	<.05
AKI	Stage 1	5	8.5	8	19.5	
	Stage 2	3	5.1	4	9.8	
	Stage 3	2	3.4	2	4.9	

Baseline serum creatinine and blood urea was significantly higher in haemorrhagic 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/1.73m^2) in comparison to ischaemic stroke patients (86.60 ± 26.73 ml/min/1.73m^2) (Table II).

Table II: Comparison of baseline biochemical parameters in stroke patients.

Baseline investigations	Ischaemic stroke (n = 59)	Haemorrhagic stroke (n = 41)	P value
	Mean ± SD	Mean ± SD	
eGFR (ml/min/1.73 m^2)	86.60 ± 26.73	56.09 ± 28.41	< 0.01
S. creatinine (mg/dl)	1.05 ± 0.60	1.69 ± 1.17	< 0.01
Blood urea (mg/dl)	45.03 ±18.39	68.22 ± 47.81	< 0.01
MAP (mmHg)	111.27 ± 4.28	128.17 ± 17.74	< 0.01
Systolic BP (mmHg)	149.90 ± 7.78	174.05 ± 18.65	< 0.01
Diastolic BP (mmHg)	91.80 ± 3.53	105.41 ± 17.32	< 0.01
Sodium (meq/l)	134.25 ± 7.13	138.32 ± 7.68	> .05
Potassium (meq/l)	3.99 ± 0.44	4.32 ± 0.65	> .05
Serum calcium (mg/dl)	9.01 ± 0.51	9.11 ± 0.63	> .05
Serum phosphorus (mg/dl)	4.2 ± 0.7	4.0 ± 0.9	> .05
Serum uric acid (mg/dl)	6.4 ± 1.1	6.6 ± 1.4	> .05
Fasting blood sugar (mg/dl)	107.32 ± 22.34	109.34 ± 19.37	> .05
Triglycerides (mg/dl)	151.13 ± 34.54	149.38 ± 31.78	> .05
Total cholesterol(mg/dl)	188.45 ± 44.86	186.47 ± 40.15	> .05
HDL (mg/dl)	46.75 ± 11.54	48.43 ± 13.89	>.05
LDL (mg/dl)	96.31 ± 10.54	98.65 ± 10.99	>.05

Diabetes was significantly associated with the development of AKI (54.2%) as compared to non-AKI (15.8%) (Table III). Among baseline biochemical investigations, a lower e-GFR was found in AKI patients, and this difference was statistically significant. Other parameters like blood urea, serum creatinine, fasting blood sugar, systolic BP, diastolic BP and MAP were higher in AKI patients as compared to non-AKI patients, but the difference was not statistically significant (Table IV).

The mean blood urea, mean serum creatinine and mean eGFR constantly changed during the hospital stay, but these values could not reach statistical significance. Rest of the 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) did not show any significant change during the hospital stay (Table V). Similar findings were observed with subgroup analysis of haemorrhagic and ischaemic stroke patients.

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

Medical history	Non-AKI (n = 76)		AKI (n = 24)		P value
	No.	%	No.	%	
Gender					
Male	46	60.5	12	50.0	> .05
Female	30	39.5	12	50.0	
Medical history					
H/o Smoking	63	82.9	20	83.3	> .05
H/o Alcohol	57	75.0	19	79.2	> .05
H/o Hypertension	65	85.5	24	100.0	> .05
H/o Diabetes	12	15.8	13	54.2	< 0.01
H/o Coronary artery disease	8	10.7	3	12.5	> .05
H/o Heart failure	3	3.9	0	0.0	> .05
H/o Atrial fibrillation	15	19.7	6	25.0	> .05
H/o Stroke	11	14.8	5	20.8	> .05
H/o TIA	8	10.5	3	12.5	> .05
Hypercholesterolaemia	26	34.2	9	37.5	> .05
Carotid artery stenosis	18	23.7	1	4.2	< .05

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

Baseline investigation	Non-AKI (n = 76) Mean ± SD	AKI (n = 24) Mean ± SD	P value
eGFR (ml/min/1.73 m^2)	77.73 ± 31.41	53.83 ± 28.67	< .05
S. creatinine (mg/dl)	1.78 ± 1.86	1.95 ± 2.11	> .05
Blood urea (mg/dl)	52.86 ± 34.45	59.88 ± 38.52	> .05
MAP (mm Hg)	118.51 ± 14.53	117.22 ± 14.27	> .05
Systolic BP (mm Hg)	158.87 ± 18.10	160.65 ± 17.41	> .05
Diastolic BP (mm Hg)	97.79 ± 13.17	96.08 ± 13.42	> .05
Sodium (meq/l)	134.78 ± 6.89	137.65 ± 8.31	> .05
Potassium (meq/l)	3.95 ± 0.45	4.69 ± 0.63	> .05
Serum calcium (mg/dl)	8.98 ± 0.65	9.32 ± 0.43	> .05
Serum phosphorus (mg/dl)	3.9 ± 0.9	4.0 ± 0.7	> .05
Serum uric acid (mg/dl)	6.4 ± 1.3	6.6 ± 1.4	> .05
Fasting blood sugar(mg/dl)	107.81 ± 20.89	110.76 ± 22.15	> .05
Triglycerides(mg/dl)	149.54 ± 31.76	153.78 ± 33.57	> .05
Total cholesterol(mg/dl)	186.98 ± 41.78	191.65 ± 43.65	> .05
HDL(mg/dl)	46.53 ± 11.98	48.96 ± 13.24	> .05
LDL(mg/dl)	96.57 ± 9.34	98.78 ± 11.54	> .05

Duration of stay in hospital (days) was significantly higher in stroke patients who developed AKI. The findings were similar with subgroup analysis of ischaemic and haemorrhagic stroke patients. However, duration of hospital

stay was higher in haemorrhagic stroke (12.71 ± 5.12 days) compared to ischaemic stroke subjects (9.41 ± 2.78) days and the difference was statistically significant ($p < 0.01$). GCS score < 10, AKI, type of stroke and requirement of renal replacement therapy for AKI were found to be associated with longer hospital stay (Table VI).

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

Investigation	Day of admission	Day 3	Day 7	Day 14	P value
Haemoglobin (g/dl)	12.42 ± 2.68	13.68 ± 1.97	12.78 ± 2.78	12.01 ± 2.16	>.05
Total leukocyte count (cells/cumm)	12651 ± 3760.7	11499 ± 2879.45	10764 ± 2334.5	10654 ± 2035.4	> .05
Blood urea (mg/dl)	45.03 ± 18.39	50.63 ± 21.33	46.76± 13.16	45.14 ± 10.34	>.05
Serum creatinine (mg/dl)	1.05 ± 0.60	1.21 ± 0.87	1.16 ± 0.77	1.04 ± 0.95	> .05
Sodium (meq/l)	134.25 ± 7.13	135.44 ± 6.35	135.71 ± 6.72	135.45 ± 8.34	> .05
Potassium (meq/l)	3.99 ± 0.44	4.01 ± 0.46	4.06 ± 0.43	3.99 ± 0.36	> .05
Serum calcium (mg/dl)	9.01 ± 0.51	9.11 ± 0.36	9.46 ± 0.75	9.31 ± 0.68	> .05
Serum phosphorus (mg/dl)	4.2 ± 0.7	3.9 ± 0.7	3.8 ± 1.1	4.1 ± 0.9	> .05
Serum uric acid (mg/dl)	6.4 ± 1.1	6.2 ± 1.1	5.9 ± 1.6	5.8 ± 1.4	> .05
eGFR (ml/min/1.73 m^2)	86.60 ± 26.73	69.65 ± 28.45	65.74 ± 26.75	63.65 ± 24.86	> .05
Fasting blood sugar (mg/dl)	108.65 ± 21.45	107.48 ± 26.12	104.23 ± 19.45	108.78 ± 23.24	> .05
Triglycerides (mg/dl)	150.41 ± 32.15	148.24 ± 30.15	139.21 ± 33.57	135.46 ± 34.45	> .05
Total cholesterol (mg/dl)	187.43 ± 42.45	191.65 ± 39.46	188.65 ± 34.45	192.34 ± 35.98	> .05
HDL (mg/dl)	47.56 ± 12.56	44.09 ± 11.87	46.73 ± 13.48	45.84 ± 12.97	> .05
LDL (mg/dl)	97.45 ± 10.86	94.83 ± 9.54	97.92 ± 9.11	94.18 ± 8.79	> .05
Serum albumin (g/dl)	3.9 ± 1.2	3.8 ± 1.6	4.1 ± 0.9	3.9 ± 1.1	> .05

Haemorrhagic stroke, older age, diabetes mellitus and high baseline creatinine level were found to be predictors of AKI (Table VII). Out of 100 patients of stroke, 12 patients died so mortality rate in stroke patients was 12%. Statistically, no differences were observed between mortality rate of patients of ischaemic stroke (11.9%) and haemorrhagic stroke (12.2%). Out of 12 patients who died, 7 had AKI, thus mortality was significantly associated with AKI in stroke patients. Mortality was significantly more common in patients with aspiration pneumonia, GCS score < 10, AKI, older age and requirement of RRT (Table VIII).

Table VI: Predictors of hospital stay among stroke patients.

	Mean ± SD	P value
GCS score		
< 10	12.19 ± 5.13	< .05
≥ 10	9.95 ± 3.38	
AKI status		
AKI	14.46 ± 4.44	< 0.001
No AKI	9.59 ± 3.41	
Hypertension		
Yes	10.91 ± 4.28	> .05
No	9.55 ± 3.58	
Type of stroke		
Ischaemic	9.41 ± 2.78	< 0.01
Haemorrhagic	12.71 ± 5.12	
Need for renal replacement therapy		
Yes	22.00 ± 1.41	< 0.001
No	10.29 ± 3.59	

Table VII: Predictors of AKI among stroke patients.

	Non-AKI	AKI	P value
Haemorrhagic stroke (n = 41)	27 (65.9%)	14 (35.1%)	< .05
Ischaemic stroke (n = 59)	49 (83.1%)	10 (16.9%)	
Hypertension (mmHg)	65 (85.5%)	24 (100.0%)	> .05
Age (in years)	59.79±7.73	64.46±9.26	< .05
Baseline creatinine (mg/dl)	1.16±0.83	1.80±1.06	< .01
Diabetes mellitus	12 (15.8%)	13 (54.2%)	< .01
Smoking	63 (82.9%)	20 (83.3%)	> .05
Alcohol	57 (75.0%)	19 (79.2%)	> .05

Table VIII: Predictors of death among stroke patients.

	Alive (n = 88)	Died (n = 12)	P value
Aspiration pneumonia	27 (30.7%)	11 (91.7%)	< .01
GCS score < 10	24 (27.3%)	12 (100.0%)	< .01
AKI	19 (19.3%)	7 (58.3%)	< .01
Age	60.10±7.86	66.83±9.81	< .05
Hypertension	77 (87.5%)	12 (100.0%)	> .05
Renal replacement therapy	0 (0.0%)	4 (33.3%)	< .01
Type of stroke			
Ischaemic	52 (59.1%)	7 (58.3%)	> .05
Haemorrhagic	36 (40.9%)	5 (41.7%)	

Discussion

The short term evolution following stroke can be associated with AKI as a possible complication, which is frequently overlooked and underestimated in clinical trials. In our study, we demonstrated that the occurrence of AKI is not a rare finding in stroke patients. AKI is a common co-morbid condition in the community with different medical events which include cardiovascular disease, diabetes mellitus, hypertension and cerebrovascular stroke and hospitalisation in intensive care unit.

There is growing evidence in medical literature of the kidney's role in the development of cardiovascular disease, including stroke. Although the majority of stroke patients had mild AKI that in most cases were "clinically" reversible, it should be emphasized that in the tissue level, this does not always hold true. Endothelial damage, tubular inflammation, and activation of intrarenal fibrotic pathways during kidney repair may gradually damage kidney structure, leading to proteinuria, hypertension, and progressive decline in renal function. Renal dysfunction may indicate a higher comorbidity burden, especially in atherosclerotic risk factors and diseases. It seems that the degree of renal dysfunction present in stroke patients may simply be a marker of end-organ damage from long standing arterial stiffness of small and large arteries due to atherosclerosis and its associated vascular risk factors (e.g., ageing, smoking, hypertension, diabetes mellitus and cardiovascular diseases) or independent of other risk factors for atherosclerosis. In other words, various common risk factors between stroke and kidney dysfunction lead to a higher morbidity and mortality in patients of stroke.

In our study, acute kidney injury was seen in 24% of stroke patients out of which 13.0% were in stage 1, 7.0% were in stage 2 and 4.0% in stage 3. Our results were consistent with findings of Tsagalis G *et al*, who reported that a total of 26.7% patients developed AKI after admission to the hospital. On the basis of the AKIN staging system, 21.3% patients were classified as stage 1, 2.9% as stage 2, and 2.5% as stage 3[8]. The high incidence of AKI in our population can be explained by the increased age (mean age 70.3 ± 11.9 year), low baseline GFR, and the use of a high-sensitivity definition for the detection of AKI. Pre-existing renal dysfunction may be a major contributor to the occurrence of AKI.

AKI was more common in haemorrhagic stroke patients (34.3%) compared to ischaemic stroke patients (16.9%). This difference was found to be statistically significant ($p < 0.05$). The higher incidence of renal impairment seen in the haemorrhagic stroke subgroup may be due to the inherent difference in the management of haemorrhagic and ischaemic stroke, in regards to the use of mannitol, nephrotoxic drugs, various antibiotics, etc., Khatri *et al* also reported higher incidence of AKI (18%) in their study, with significantly higher rates among intracerebral haemarrhage cases compared to ischaemic stroke (21% v 14%)[13].

Out of 100 patients of stroke, 12 patients died. Statistically, no differences were observed between mortality rate of ischaemic stroke and haemorrhagic stroke patients. AKI was also significantly associated with mortality. Out of 24 patients who developed AKI, 7 died, so mortality was 29.2% in AKI patients. This is consistent with studies using the same data set in other acute care settings. In a study by Covic *et al* on patients of stroke, the overall incidence of AKI was 14.5% with unadjusted 30-day mortality rate of 43.1% compared to 12.8% for subjects without AKI[14].

In our study, haemorrhagic stroke, older age, diabetes mellitus and high baseline creatinine level were found to be predictors of AKI. Similar studies have also reported that Patients with AKI were older, had a higher prevalence of atrial fibrillation and heart failure, and presented with a more severe neurologic deficit than patients without AKI[8].

GCS score < 10, AKI, haemorrhagic stroke, and need for renal replacement therapy were found to be associated with longer hospital stay. Spratt *et al* reported disability at discharge from the acute hospital (Rankin score > 2), age more than 65, diabetes and infection a significant risk factor for prolonged stay[15]. Shrestha *et al* also reported higher mean hospital stay in patients of haemorrhagic stroke group (14.58 ± 7.19 days) compared to ischaemic stroke group (9.86 ± 5.12 days)[12].

In our study, mortality was significantly more common in patients with aspiration pneumonia, GCS score < 10, AKI, older age and need for renal replacement therapy. So, AKI was found to be predictor of mortality. Similar studies have also reported that AKI was an independent predictor of 10-years mortality ($p < 0.01$) after stroke and for the occurrence of new composite cardiovascular events ($P < 0.05$) after adjustment for available confounding variables[8]. Another study reported that delay in recovery of consciousness and new onset acute myocardial infarction and aspiration pneumonia, age (> 60 years), severity of neurodeficit (GCS < 7, grade/N motor weakness), size of lesion (infarct > 1 lobe, haemorrhage> 60 ml) were found to be the most significant predictors of mortality[16].

The severity of impaired kidney function in patients hospitalised with acute stroke is associated with increased all-cause mortality independent of age, sex, and major co-morbidities. Our findings highlight the importance of assessing patients with stroke at risk of developing AKI based on the parameters like haemorrhagic stroke, higher age, high baseline creatinine and diabetes mellitus, that were found in our study to independently predict its

development. Also, there was a comparable risk of prolonged hospital stay and increased mortality in patients of stroke. Additional studies are required to determine if development of AKI after stroke is a causal relationship or is it simply a marker of end-organ damage from long-standing arterial stiffness of small and large arteries due to atherosclerosis and its associated vascular risk factors like ageing, smoking, hypertension, diabetes mellitus. Further studies should be planned to determine whether interventions designed to aggressively either prevent the development of AKI or treat early manifestations of AKI would result in reduced stroke mortality.

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