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Original Article

Clinical Study of Renal Profile of Acute Coronary Syndrome

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Abstract

Background: Markers of renal function have been incorporated in risk stratification of acute coronary syndrome (ACS). Global registry for acute coronary events risk score, Michigan percutaneous coronary intervention (PCI) risk score, Mayo PCI risk scores, etc. incorporate serum creatinine levels or creatinine clearance as one of the biomarkers in risk stratification with serum uric acid, limited data is available regarding its prognostic value in ACS.

Objective: To study the renal function parameters in patients of ACS.

Methods: A total of 100 patients who were admitted and fulfilled the inclusion/exclusion criteria were evaluated by history. Physical examination, electrocardiogram, echocardiography and renal function tests were performed.

Result: A total of 100 patients of diagnosed ACS were included in the study of which 75% were males and 25% were females. In 75 males, maximum 28 (37.3%) were in age group of 51-60 years followed by 16 (21.3%) in age group of 41-50 years and in 25 females maximum 11 (44.0%) were in age group 61-70 suggesting that ACS commonly occur in middle age group adults. Out of 100 diagnosed ACS patients, maximum (72%) were ST-segment elevation myocardial infarctions (STEMI) followed by 23% UA and 5% Non STEMI. Mean (+ standard deviation) serum creatinine value in STEMI patients was 2.20 + 1.61, was recorded indication mild to moderate renal insufficiency in ACS patients.

Conclusion: The long term morbidity and mortality is higher in those ACS patients who have deranged renal function, incorporating renal function parameters for regular assessment of patients at risk of developing ACS and measuring the renal function parameters in patient who have developed ACS at their first clinical visit in emergency department becomes important for risk stratification. Thus, it is concluded that renal function monitoring must be done in patients at risk or who have developed ACS for reducing the long term morbidity and mortality in such patients.

Key words: Acute coronary syndrome, Chronic kidney disease, Clinical profile, Creatinine, Creatinine clearance

INTRODUCTION

Cardiovascular diseases (CVDs) are currently are among the leading cause of death in industrialized as well as in emerging countries. Among CVDs coronary artery disease (CAD) is the most prevalent manifestation and is associated with high mortality and morbidity.[1] World Health Organization (WHO) estimates that CVDs are the number one cause of death globally with 9.4 million deaths each year of which 45% of deaths being due to coronary heart disease (CHD).[2] WHO estimates that more than 60% of the global burden of CHD occurs in developing countries. Globally, burden of CHD is projected to rise from around 47 million (in 1990) Disability-adjusted life years (DALYs) to 82 million DALYs in 2020. With India, DALYs lost per 1000 population were reported to be 20.[3]

Acute coronary syndrome (ACS) involves a range of thrombotic CADs. These are unstable angina (UA), ST-segment elevation myocardial infarctions (STEMI), and non-STEMI (NSTEMI).[4] The clinical presentations of CAD include silent ischemia, stable angina pectoris, UA, myocardial infarction (MI), heart failure and sudden death.[1]

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Despite modern treatment, the rates of death, MI, and readmission of patients with ACS remain high.[1]

A assessing the patient for cardiovascular risk is cornerstone in diagnosis and management of ACS. Risk stratification helps in appropriate referral of patients to and emergency department and assessment of the risk of future at the time of the initial assessment in the emergency department.[4,5] Different score based on different markers have been developed for risk stratification. Different risk scores that are validated include Thrombolysis in MI (TIMI) score, global registry for acute coronary events (GRACE) risk score, FRISC score, PURSUIT score.[6] Among these, GRACE and TIMI risk scores are being used commonly used in clinical practice.[7] Different markers that are used in risk stratification include that of myocardial necrosis, inflammation, hemodynamic stress and neurohormonal activation, renal impairment, vascular injury, and accelerated atherosclerosis.[6]

In addition to these, some novel biomarker and risk scores have been assessed for their prognostic value in risk stratification of ACS. These include high-sensitivity troponin (hs-cTn), CD40 ligand and interleukin-6, glycogen phosphorylase isoenzyme-BB (GP-BB), percutaneous coronary intervention (PCI) risk scores, angiographic risk scores.[7,8]

Chronic kidney disease (CKD) is quite common with a prevalence of about 12% of adults.[9] When CKD is considered, an independent association between renal dysfunction and mortality after ACS is reported.[10] it has been reported that baseline renal dysfunction is a potent and easily identifiable determinant of outcome after an ACS.[11] Therapeutic complexities are more in the management of patients with CKD presenting with ACS as compared to the general population because of the lack of well-designed randomized trials assessing therapeutic strategies in such patients.[12]

Markers of renal function have been incorporated in risk stratification of ACS. GRACE risk score, Michigan PCI risk score, Mayo PCI risk scores, etc., incorporate serum creatinine levels or creatinine clearance (CrCl) as one of the biomarkers in risk stratification.[7] with serum uric acid, limited data is available regarding its prognostic value in ACS.[13]

Since limited data is available with renal profile of ACS patients in Indian scenario, we planned this research to study the renal parameters in ACS patients.[14-23]

Aims and Objectives

To study the renal function parameters in patients of ACS in cardiac intensive care unit (ICU) of a tertiary care hospital.

MATERIALS AND METHODS

This open label, prospective, single center, observational, cross–sectional study was conducted in patients admitted at cardiac ICU of a tertiary care hospital. Institutional ethics committee (IEC) approved the study protocol. Study patients were screened at cardiac ICU for the recruitment criteria and those who fulfilled the inclusion and exclusion criteria were enrolled in study. Patients were given patient information sheet to understand the study details. Any questions concerning the study were answered. Then the informed consent was sought form each patient in an IEC approved informed consent form (ICF). The patients were recruited with following inclusion and exclusion criteria.

Inclusion Criteria

- Age >18 years
- Either gender
- Investigated and diagnosed cases of ACS in cardiac ICU.

Exclusion Criteria

- Not willing to participate in study or give informed consent.

Patients fulfilling these criteria were enrolled and ICF was signed. Study related parameters were than noted in case record forms (CRF). ICU case record papers were observed to record the data. The data collected includes demographic parameters such as registration number, age, sex, height, weight, and body mass index (BMI). The vital signs were recorded. The clinical history of the patient was also recorded from the case files and by direct questioning to the patient and it includes smoking history, any major illness such as diabetes, hypertension, or CKD. The findings of electrocardiogram (ECG) were noted.

As per the protocol in the management of ACS at this cardiac (ICU, all the blood investigations including biomarkers and renal function tests are routinely performed. From these investigation reports, blood sugar levels, tropoin T result, serum creatinine, blood urea nitrogen (BUN), serum uric acid levels, serum albumin levels, urine routine and microscopy examination results, etc., were recorded in CRF. No active investigations on any patient were performed during the study. The CrCl was calculated for each patient with the help of following formula.

$$\text{Creatinine clearance} = \frac{\left[\,140 - \text{Age}\right] \times \text{Body weight (kg)}}{72 \times \left(\text{Serum creatinine}\right)}$$

(To be multiplied by 0.85 if the patient is female).

Statistical Analysis

The collected data was analyzed using the SPSS software version 15. The data are presented as frequency and percentages. The mean and standard deviation (SD) were calculated. For quantitative data, student's *t*-test were used and for qualitative data analysis, Chi-square test with or without Yate's correction were used. $P < 0.05$ was considered significant.

Chart 1 shows that in total, 75% patients were males and 25% patients were females.

RESULTS

Table 1 shows the age group wise distribution of males and females enrolled in the study. Out of total 75 males, 11 (14.7%) were 40 years or less, 16 (21.3%) were in between 41 and 50 years, 28 (37.3%) were in between 51 and 60 years, 12 (16.0%) were in 61-70 years age group, whereas 8 (10.7%) were 71 years or above. Out of total 25 females, the frequencies for females in given age group were 1 (4.0%), 1 (4.0%), 2 (8.0%) 11 (44.0%), and 10 (40.0%), respectively.

Chart 2 describes the smoking history of patients. No females had smoked anytime in their life. Out of 75 males, 40 (53.3%) had active smoking in their life.

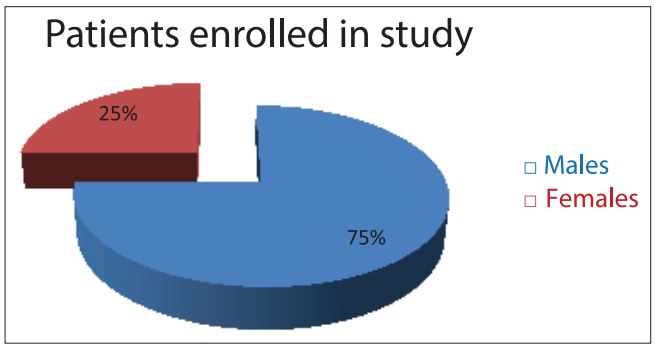

Chart 1: Gender wise distribution of patients in study

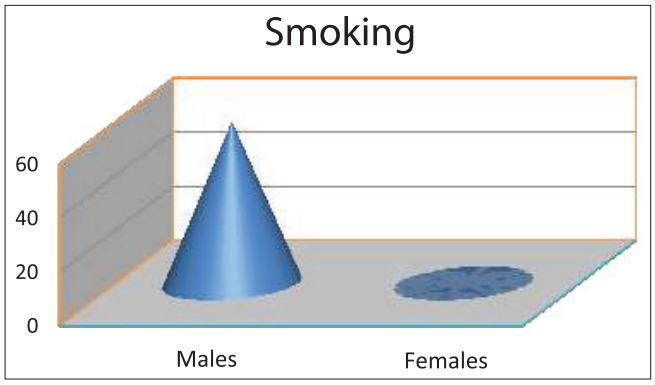

Chart 2: Smoking history of patients in the study

Table 2 gives the gender wise distribution of mean age and BMI. The mean age of males was 54.5 + 12.7 and of females was 67.2 + 10.4. BMI in males was 25.1 + 2.1 and in females, it was 25.6 + 3.2.

Table 3 gives the diagnosis of the patients enrolled in to the study. Among ACS diagnosed in total 100 patients, maximum (72%) were STEMI followed by UA with 23% patients and NSTEMI with 5% of patients.

Table 4 shows that 67 (93%) patients with STEMI and 5 (100%) patients with NSTEMI had TnT positive. Only 5 (7.0%) patients with STEMI had negative result of TnT test.

Chart 3 describes percentage of ACS patients having serum creatinine levels within and above normal limits. Of 100

Table 1: Age group wise distribution of males and females in the study

Age group	Gender (%)		Total
	Male	Female	
<40	11 (14.7)	1 (4.0)	12
41-50	16 (21.3)	1 (4.0)	17
51-60	28 (37.3)	2 (8.0)	30
61-70	12 (16.0)	11 (44.0)	23
≥70	8 (10.7)	10 (40.0)	18
Total	75	25	100

Table 2: Mean age and BMI of patients in the study

Parameter	Gender		*P*
	Male	Female	
Age (years)	54.5+12.7	67.2+10.4	0.226
BMI (kg/m^2)	25.1+2.1	25.6+3.2	0.016**

Data presented as mean+SD, ***P*<0.05 considered significant, SD: Standard deviation, BMI: Body mass index, SD: Standard deviation

Table 3: Diagnosis of patients enrolled in the study

Diagnosis	Number of patients (%)
NSTEMI	5 (5.0)
STEMI	72 (72.0)
UA	23 (23.0)
Total	100 (100)

STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non ST-segment elevation myocardial infarction, UA: Unstable Angina

Table 4: TnT positivity among the patients diagnosed with STEMI or Non-STEMI in the study

Diagnosis	Troponin-T, *n* (%)	
	Positive	Negative
STEMI	67 (93.0)	5 (7.0)
NSTEMI	5 (100)	0

STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non ST-segment elevation myocardial infarction, TnT: Troponin-T

ACS patients in the study, 67% had serum creatinine within normal limits of 1.5 mg/dl where as 33% patients had value above 1.5 mg/dl suggesting deranged renal function in these patients.

Chart 4 describes the serum urea levels in ACS patients. Out of 100 patients, 66% had serum urea level above 40 mg/dl. About 34% patients had level in normal limits.

Table 5 describes the presence of hypertension, ischemic heart disease, and CKD in patients enrolled in study. About 61.1% STEMI, 40.0% NSTEMI, and 56.5% UA patients had hypertension; 19.4% STEMI, 40.0% NSTEMI, and 34.7% UA patients had IHD and 23.6% STEMI and 21.7% UA patients had CKD. No patients with NSTEMI had associated CKD illness.

Chart 5 describes the overall serum uric acid levels in ACS patients of which 90% had levels <7 mg/dl and 10% had values above normal that is more than 7 mg/dl.

Table 6 gives some of the major renal function parameters in patients of ACS. Mean values of serum creatinine in STEMI, NSTEMI, and UA were 2.20 + 1.91, 1.16 + 0.31, and 1.93 + 1.57, respectively. The mean value for CrCl in STEMI was 99.88 + 35.80, in NSTEMI was 117.20 + 5.19, and in UA was 100.89 + 32.36. The value of serum urea in STEMI, NSTEMI and UA were 72.67 + 59.19, 39.80 + 11.05, and 65.76 + 56.97. Mean serum uric acid concentration was 4.92 + 1.34, 4.20 + 1.32 and 4.48 + 1.37 foe STEMI, NSTEMI, and UA patients, respectively.

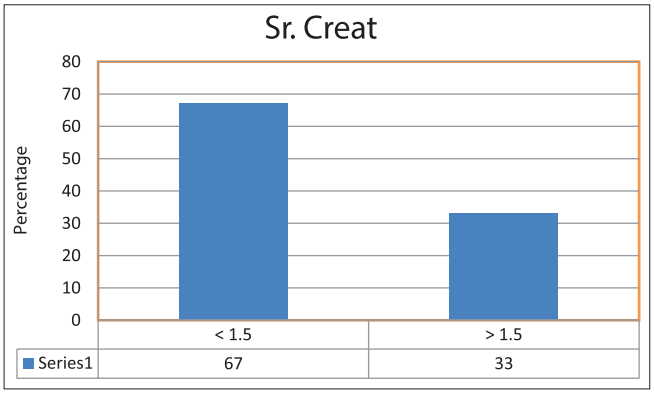

Chart 3: Serum creatinine values in acute coronary syndrome patients

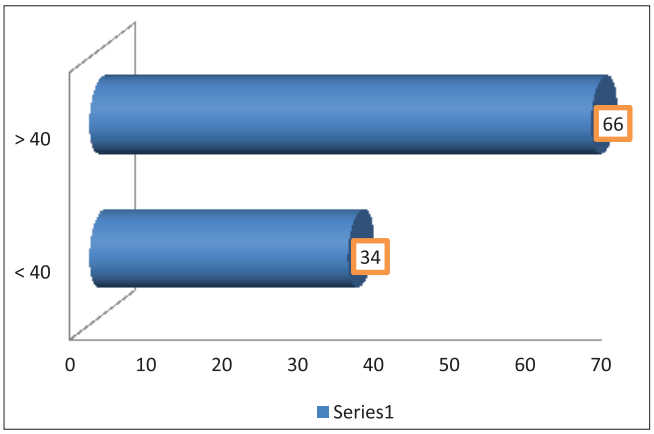

Chart 4: Serum urea in acute coronary syndrome patients

Table 7 describes urinary protein levels in ACS patients in the study. Of the 7 STEMI patients, 22.2% had nil proteins in urine, 38.9% had 1+ trace proteins, 18.1% had 1+, 13.9% had 2+, and 6.9% had 3+ grade protein excretions. No NSTEMI had grade 2+ or 3+ protein excretion, out of 23 UA patients, 47.8% had nil excretion, 17.4% had trace, and 1+, 8.7% had 2+ and 3+ proteins excretion in urine.

Table 8 shows the urinary albumin: Creatinine ratio (ACR) in ACS patients in the study. None of patients with NSTEMI had ACR >30. Of total 72.76.3% STEMI

Table 5: Associated hypertension, IHD and CKD in diagnosed acute coronary syndrome patients in the study

Diagnosis	Associated illness, *n* (%)		
	Hypertension	IHD	CKD
STEMI	44 (61.1)	14 (19.4)	17 (23.6)
NSTEMI	2 (40.0)	2 (40.0)	0
UA	13 (56.5)	8 (34.7)	5 (21.7)
Total	59	24	22

IHD: Ischemic heart disease, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non ST-segment elevation myocardial infarction, UA: Unstable Angina, CKD: Chronic kidney disease

Table 6: Major renal function parameters in patients with ACS

Diagnosis	Renal parameter*			
	Serum creatinine	Creatinine clearance	Serum urea	Serum uric acid
STEMI	2.20+1.91	99.88+35.80	72.67+59.19	4.92+1.34
NSTEMI	1.16+0.31	117.20+5.19	39.80+11.05	4.20+1.32
UA	1.93+1.57	100.89+32.36	65.76+56.97	4.48+1.37

*Data presented as mean+SD, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non ST-segment elevation myocardial infarction, UA: Unstable Angina, ACS: Acute coronary syndrome, SD: Standard deviation

Table 7: Urinary protein levels in diagnosed ACS patients in the study

Diagnosis	Urinary proteins				
	Nil	Trace	1+	2+	3+
STEMI	16 (22.2%)	28 (38.9%)	13 (18.1%)	10 (13.9%)	5 (6.9%)
NSTEMI	2 (40.0%)	1 (20.0%)	2 (40.0%)	0	0
UA	11 (47.8%)	4 (17.4%)	4 (17.4%)	2 (8.7%)	2 (8.7%)

STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non ST-segment elevation myocardial infarction, UA: Unstable Angina, ACS: Acute coronary syndrome

patients and of total 23, 78.3% UA patients had ACR <30. Among STEMI patients, 11.1% had ACR between 500 and 1000, 5.5% had ACR in between 1001 and 1500 and 4.1% had ACR above 1500. Among UA patients, 13.1% had ACR between 500 and 1000 and only one patient had ACR in-between 1001 and 1500 and above 1500.

Table 9 shows serum homocystiene concentration in STEMI and UA groups. In only 12 patients (10 STEMI and 2 UA), serum homocystiene was determined as per the clinical need by the cardiologist. In 10 STEMI patients, mean values for serum homocystiene were 31.28 + 7.18 and that for two UA patients were 37.66 + 7.85.

DISCUSSION

In the field of cardiology, large-scale clinical trials have provided enormous data on the treatment and outcomes for hundreds of thousands of patients.[26-34] Many of these

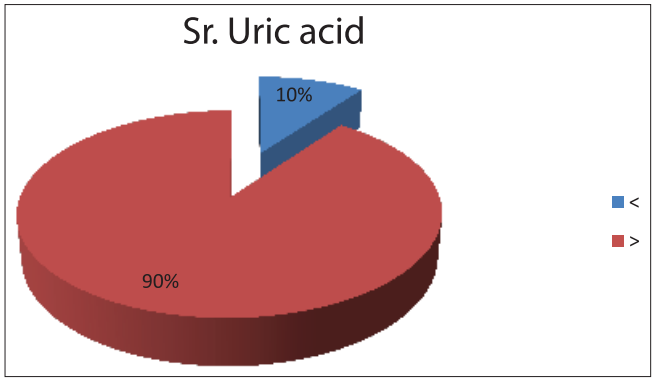

Chart 5: Serum uric acid levels in acute coronary syndrome patients

Table 8: Urinary albumin: Creatinine ratio in diagnosed ACS patients

Diagnosis	Urinary albumin: Creatinine ratio			
	<30	500-1000	1001-1500	≥1501
STEMI	55 (76.3%)	8 (11.1%)	4 (5.5%)	3 (4.1%)
NSTEMI	5 (100%)	0	0	0
UA	18 (78.3%)	3 (13.1%)	1 (4.3%)	1 (4.3%)

STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non ST-segment elevation myocardial infarction, UA: Unstable Angina, ACS: Acute coronary syndrome

Table 9: Mean serum homocystiene concentration in diagnosed ACS

Diagnosis	Serum homocystiene	
	n	Mean±SD
STEMI	10	31.28+7.18
UA	2	37.66+7.85

STEMI: ST-segment elevation myocardial infarction, UA: Unstable Angina, ACS: Acute coronary syndrome, SD: Standard deviation

efforts have focused on patients with ACS, which range STEMI to NSTEMI to UA.[35] ACS represents a life-threatening manifestation of atherosclerosis. It is usually precipitated by acute thrombosis induced by a ruptured or eroded atherosclerotic coronary plaque, with or without concomitant vasoconstriction, causing a sudden and critical reduction in blood flow.[1] WHO estimates that CVDs are the number one cause of death globally with 9.4 million deaths each year of which 45% of deaths being due to CHD.[2]

STEMI is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent ECG ST elevation and subsequent release of biomarkers of myocardial necrosis.[24] UA and NSTEMI are considered to be closely related conditions whose pathogenesis and clinical presentations are similar but of differing severity. Once it has been established that no biochemical marker of myocardial necrosis (troponin I [TnI], TnT, or the MB isoenzyme of creatine phosphokinase [CK-MB]) has been released, the patient with an ACS may be considered to have experienced UA, whereas the diagnosis of NSTEMI is established if a marker of myocardial injury has been released.[25] of the number of available markers and assays that detect myocardial necrosis, the cardiac TnT and TnI and the creatinine kinase-MB (CK-MB) isoform are the most commonly used, with Tn gaining acceptance as the markers of choice in ACS.[36]

CKD is defined as persistent kidney damage, as reflected by a glomerular filtration rate (GFR) of <60.0 ml per min per 1.73 m^2 of body surface area for more than three mortality in the follow-up of patients who have undergone coronary artery bypass grafting or a PCI and those who have suffered an acute MI.[11] Also mild renal impairment is associated with an increased risk of CAD and stroke. Suggesting that CVD may develop early in the course of renal dysfunction.[30]

With evolving new treatments for the management of ACS, risk stratification has become the centerprise of initial evaluation for these patients. Risk stratification has now evolved more to include the assessment of the risk of future cardiac events, which can be predicated on the basis of clinical features and biomarkers at the time of the initial assessment in the emergency department.[5] For evaluating risk of ACS, various risk scores have been developed and are used clinically. The TIMI and GRACE risk scores are recommended for risk stratification.[37] GRACE score predict cumulative 6-month risk of mortality or MI, with predictor variables including age, heart rate, systolic blood pressure (SBP), creatinine level, Killip class of heart failure, cardiac arrest at admission, ST-segment deviation, and cardiac enzymes.[7] Creatinine has been included in

many clinical scores to assess the prognosis of critically ill patients. In a study measuring CrCl on admission in patients with cardiogenic shock consecutive to ST elevation MI and for whom a PCI was performed, and independent strong association between CrCl on admission and 1-year-mortality was reported, which was significantly higher when CrCl was <67.5 ml/min.

With serum uric acid, hyperuricemia has been implicated as a marker of poor outcome, both in the general population and in patients with stroke and heart failure. There is limited data in the context of ACS with contradictory results.[13] Given the role of renal function parameters association with ACS morbidity and mortality, we studied the renal function profile in diagnosed ACS patients at tertiary care hospital.

A total of 100 patients of diagnosed ACS were included in the study of which 75% were males and 25% were females. In 75 males, maximum 28 (37.3%) were in age group of 51-60 years followed by 16 (21.3%) in age group of 41-50 years and in 25 females maximum 11 (44.0%) were in age group 61-70 suggesting that ACS commonly occur in middle age group adults. This is consistent with age group range of 51-71 years in a study by Akerblom *et al.* Mean age in males was 54.5 years and in females was 67.2 years, which was statistically non-significant ($P = 0.226$). Smoking was common in males only with 53.3% males reported active cigarette smoking. No female had smoked ever in their life. The mean BMI varied in two genders and difference was statistically significant) $P = 0.016$). BMI is also an important risk factor for the development of ACS. In a study by Wolk *et al.*; the mean values of BMI in stable and unstable CAD were 28.8 + 4.7 and 30.5 + 5.7, respectively, and reported a positive relation between BMI and the risk of acute coronary events for even mildly elevated BMI values.

Out of 100 diagnosed ACS patients, maximum (72%) were STEMI followed by 23% UA and 5% NSTEMI. Tn are among the priority biomarkers in ACS and are used clinically to differentiate the establishment of ischemia in NSTEMI and UA. Out of 72 diagnosed STEMI patients, 67 (93%) had TnT test positive and 5 (7%) had negative results with TnT test. In STEMI patients with negative TnT-test, ECG remains the main tool for the diagnosis in combination with other biomarkers. With positive TnT and no ST-elevation in ECG, diagnosis of NSTEMI is established. Among the associated illnesses in ACS patients, CKD was present in 17 (23.6%) to total 72 STEMI patients and in 5 (21.7) of 23 UA patients.

CKD and ACS are highly prevalent and highly relevant clinically for patients, physicians, and healthcare systems.[10] The short-term as well as long-term prognosis of ACS patients with poor renal function is worse than those with normal renal function. The risk of cardiovascular (CV) events and mortality is inversely proportional to the estimated GFR (eGFR).[29] The independent association between renal dysfunction and mortality after ACS has been reported.[10] Death from cardiac causes is 10-20 times more common in CKD patients than in age-and gender-matched population.[29] In this study, renal function parameters such as serum creatinine, CrCl, serum urea, serum uric acid, and urinary ACR were estimated in diagnosed ACS patients during the course of hospital stay.

Mean (+SD) serum creatinine value in STEMI patients was 2.20 + 1.61, was recorded indication mild to moderate renal insufficiency in ACS patients. Shlipak *et al.* studied association of renal insufficiency with treatment and outcomes after MI in 130 099 elderly patients. Patients were categorized according to the initial serum creatinine level no renal insufficiency (creatinine level <1.5 mg/dl). Mild renal insufficiency (creatinine level, 1.5-2.4 mg/dl), or moderate renal insufficiency (creatinine level, 2.5-3.9 mg/dl), mild (hazard ratio, 1.68 [95% Cl: 1.63-1.73]) and moderate (hazard ratio, 2.35 [Cl: 2.26-2.45]) renal insufficiency were associated with substantially elevated risk for death during the first month of follow-up. 1 year mortality was 24% in patients with no renal insufficiency, 46% in patients with mild renal insufficiency, and 66% in patients with moderate renal insufficiency ($P < 0.001$). Therefore renal insufficiency was found to be an independent risk factor for death in elderly patients after MI. Rozic *et al.* reported that in NSTEMI patients most significant independent early predictor of 30-day mortality was admission heart failure (OR 41.21, 95% Cl: 3.520-484.66, $P = 0.003$), followed by admission serum creatinine (OR 0.989, 95% cl 0.981-0.997, $P = 0.008$) and TnT (OR 0.263, 95% Cl: 0.080-0.861). Cakar *et al.* studied the effect of admission creatinine levels on 1-year mortality in acute MI and divided patients in two groups based on serum creatinine levels as elevated group (serum creatinine >1.3 mg/dl) and normal group (serum creatinine <1.2 mg/dl). The mean creatinine level was 1.78 + 7 mg/dl in the elevated group and 0.9 + 0.18 mg/dl in the normal group ($P < 0.0001$). The mortality rate of the elevated group ($n = 7$, 25.9%) was higher than that of the normal group ($n = 9$, 6.8%). A significant increase in 1 year mortality is also observed ($P = 002$).

CrCl was calculated for ACS patients. Mean value of CrCl in STEMI patients was 99.88 + 35.80, in NSTEMI patients was 117.20 + 5.19 and for UA patients, it was 100.89 + 32.80. Smith *et al.* studied renal impairment in long-term mortality risk prediction after acute MI in 118.753 patients. Mean creatinine was 1.3 + 0.7 mg/dl and CrCl was 55 + 21 m/min. by 10 years, 68% of patients Dhad died.

Compared with normal renal function, even mild renal impairment increased the 10 year risk for mortality by 10%. Severe renal impairment had doubled the risk for mortality at 1 year, and this increased risk persisted at both 5 and 10 year.

The mean value of serum urea in STEMI patients was 72.67 + 59.19, and in NSTEMI patients, it was 39.801 + 11.05, and in UA patients, mean serum urea was 65.78 + 56.97. Serum urea is one of most frequently determined clinical indices for estimating renal function, it is useful in differential diagnosis of acute renal failure and pre-real condition. Kirtane *et al.* studied an independent association of elevated BUN for adverse outcome in patients of ACS. They reported a stepwise increase in mortality with increase in BUN in ACS patients independent of the serum creatinine levels. Ostfeld *et al.* reported that each 1 mg/dl increase in BUN was associated with an average increased odds of having a CAD burden score greater or equal to the 75th percentile of 12% (OR 1.12 (1.05, 1.19 $P < 0.01$).

Another marker of renal impairment is the serum uric acid. Mean value of serum uric acid in STEMI patients was 4.92 + 1.34, in NSTEMI patients was 4.20 + 1.32, and UA patients, it was 4.48 + 1.37. Nadkar and Jain reported the statically significant higher levels of serum uric acid in MI patients on duty 1 as compared to controls and these levels were more than 7.0 g/dl. In patients who died in 3 days of hospitalization. Chen *et al.* retrospectively studied uric acid levels in 502 patients of acute STEMI. Higher levels were observed in patients with three vessel disease (330.67 + 2106.47 μmol/L). Hyperuricemia patients with acute MI have a higher rate of left ventricular systolic and diastolic dysfunction including heart failure. Berzein and Kremzer reported serum uric acid as independent marker of coronary calcification in asymptomatic CAD. Timoteo *et al.* reported that serum uric acid has been a forgotten prognostic marker in ACS. In study of 683 patients, they reported best cut-off of uric acid to predict 1-year mortality of 6.25 mg/dl with sensitivity of 59% and specificity of 72%.[13] in our study, mean values of serum uric acid were higher in patients of STEMI than NSTEMI or UA. Thus, serum uric acid can be taken as an independent predictor of all-cause mortality-term after the whole spectrum of ACS and has an added value for risk stratification. Albuminuria has also been a marker for renal impairment. On urinalysis, 21.1% of STEMI patients had proteinuria of Grade 2 or more. No NSTEMI patient had proteinuria of Grade 2 and above. About 4 (17.4%) UA patients had proteinuria of Grade 2 and more. The more precise risk evaluating factor than proteinuria alone is the ACR. Majority of the patients had ACR <30. Among STEMI patients, 11.1% had ACR in between 500 and 1000, whereas 9.1% had ACR above. 1000. In UA patients, only two patients had ACR above 1000. Beton *et al.* studied the effect of albuminuria in 1-year mortality of MI patients. They reported that at multivariable analysis, the ACR was the strongest among several independent predictors of mortality (adjusted relative risks: 3.6 (95% Cl: 2.1-6.2) on the 1st day, 4.9 (95% Cl: 2.9-8.2) on the 3rd day and 4.0 (95% Cl 2.3-6.8) on the 7th day). Brantsma *et al.* in study reported that for each doubling of baseline urinary albumin excretion, the hazard ratio for a CV event was 1.36 (95% confidence interval [Cl] 1.31-1.42). Study by Roest *et al.* in postmenopausal women confirmed the predictive role of urinary albumin for the risk of future cardiovascular mortality independent of hypertension and diabetes. Study by Kweon *et al.* reported that higher normal ranges of urine ACR are independently associated with carotid intima-media thickness. Sadak *et al.* reported a direct association between micro-albuminuria and extension of atherosclerotic coronary lesions ($P = 0.009$) Thus, ACS can be regarded as a one of the important and independent risk factor for mortality in ACS patients.

Serum homocystiene was examined in some of the patients. In 10 STEMI patients, the mean value was 31.28 + 7.18 and in two UA patients, it was 37.66 + 7.85. Omland *et al.* reported that in a multivariate model of predication, serum homocystiene (. 14.1 μmol/L) was significantly associated with all-cause-mortality (RR 1.78 (95% Cl – 1.06 – 2.9)] ($P = 0.03$).

Thus, study finds that renal parameters studied have important role in prognosis of ACS patients. These factors have an independent risk of mortality and morbidity in ACS patients.

Limitations of the Study

Though the renal dysfunction is reported to be an independent risk factor for morbidity and mortality in ACS patients. Our study had some limitations. The study population was evaluated only at a single visit and no follow-up of patients after the discharge was made to ascertain health status. Data were collected at single point of time. The sample size studied was small and results would be more meaningful with the higher sample size. Thus, a study with large cohort of patients and for longer duration with long-term follow-up is warranted to focus more on risk of ACS in CKD patients in Indian scenario.

CONCLUSION

ACS are among the major causes of morbidity and mortality globally including developing countries like India. Spectrum of ACS incorporates STEMI, NSTEMI, and UA. Risk of developing ACS has been established with risk factors such as smoking, obesity, hypertension,

and diabetes. Renal impairment has also been mortality following ACS. Each renal parameter including serum creatinine, CrCl, serum urea, serum uric acid levels, and urinary ACR has been reported to predict the risk of ACS independently of the other risk factors. Development of ACS in CKD patients complicates the management strategies. In this study, we found that the renal function parameters were deranged in diagnosed patients of ACS. All the parameters described above were deranged in ACS patients who had CKD. The long term morbidity and mortality is higher in those ACS patients who have deranged renal function, incorporating renal function parameters for regular assessment of patients at risk of developing ACS and measuring the renal function parameters in patient who have developed ACS at their first clinical visit in emergency department becomes important for risk stratification. Thus, it is concluded that renal function monitoring must be done in patients at risk or who have developed ACS for reducing the long term morbidity and mortality in such patients.

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