



The Influence of Renal Function on In-Hospital Complications in Patients with ST-Elevation Myocardial Infarction

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Abstract

Objective: The goal of this study was to examine in-hospital complications in patients with acute ST-elevation myocardial infarction with a different renal function.

Methods: 351 patients were included in the study. Percutaneous coronary intervention was performed on all patients. 116 had a glomerular filtration rate < 60 ml/min/1.73 m² (Group 1), $120 \geq 60$ ml/min/1.73 m² and < 90 ml/min/1.73 m² (Group 2) and $115 \geq 90$ ml/min/1.73 m² (Group 3). Other parameters in the groups were comparable. The composite rate of acute pulmonary oedema and cardiogenic shock, in-hospital pneumonia, pulseless ventricular tachycardia or ventricular fibrillation, new-onset atrial fibrillation or atrial flutter, and in-hospital death were all compared among study groups.

Results: Mean glomerular filtration rate in Group 1 was 48.2 ± 10.4 ; in Group 2, 74.7 ± 8.7 ; and in Group 3, 104.1 ± 14.6 ($p < 0.001$). The incidence of atrial fibrillation or atrial flutter was higher in Group 1 than in Groups 2 and 3: 12.1%, 5.8%, and 3.5%, respectively ($p < 0.05$). Group 3 had significantly lower rates of acute pulmonary oedema and cardiogenic shock than Groups 1 and 2: 10.3%, 5.8%, and 0.9%, respectively ($p < 0.05$). There was also a significant difference between groups when comparing the rate of in-hospital pneumonia: Group 1 had reasonably higher rates of in-hospital pneumonia than Group 2 and Group 3: 13.8%, 6.7%, and 4.3% ($p < 0.05$), respectively. The authors discovered no significant differences in additional complications: pulseless ventricular tachycardia or ventricular fibrillation occurred in 2.6%, 3.3%, and 0.9%, respectively ($p > 0.05$). in-hospital death was 3.4% in Group 1; 0.8% in Group 2; and 0 in Group 3, ($p > 0.05$).

Conclusion: Patients with lower glomerular filtration rate were more likely to develop in-hospital acute pulmonary oedema and cardiogenic shock, pneumonia, and new-onset atrial fibrillation or atrial flutter in ST-elevation myocardial infarction.

Keywords

acute coronary syndrome, ST-elevation myocardial infarction, percutaneous coronary intervention, renal dysfunction, glomerular filtration rate

Introduction

Clinical trials have significantly increased efforts to establish the importance of gender and, more recently, the function of diabetes and obesity as important comorbidities for coronary artery disease (CAD) [1,2]. Renal disease has not attracted the same attention—in fact, cardiovascular events account for more than half of all deaths in individuals with end-stage renal illness [3,4]. Several studies have found a high prevalence of chronic kidney disease in adults [5]. There is also an increased frequency of cardiovascular illness with diminishing renal function (RF) [6–8]. Renal impairment at the time of admission should signal an increased risk of in-hospital complications in patients with acute myocardial infarction. According to several studies, even slight changes in glomerular filtration rate (GFR) enhance the chance of developing de novo CAD or death after the first myocardial infarction [9–12]. After acute coronary syndrome (ACS), including acute myocardial infarction (AMI), percutaneous coronary intervention (PCI), and coronary artery bypass grafting,

patients with impaired RF have a worse prognosis than those with normal RF [13]. In ST-elevation myocardial infarction (STEMI) with primary PCI on a left main culprit lesion, renal failure was associated with more complex coronary lesions and less complete revascularisation and turned out to be an independent predictor of mortality at 1-year follow-up [14]. Except for caution about prescribed doses and more liberal use of revascularisation, current guidelines recommend the same treatment for patients with renal impairment as for other patients with ACS [15,16]. Understanding the causes driving poor outcomes in people with renal impairment is critical. Much of the literature focuses on the underutilisation of evidence-based pharmacological and interventional therapy in this population [17]. Clinicians may be unaware of renal impairment, hence a specific recommendation has been prepared to assist in the diagnosis of patients with cardiovascular disease and decreased kidney function [18]. Although guidelines do not recommend treatment modification based on renal function [9,19], the risk-to-benefit trade-off may be different in the 11% of adults with renal dysfunction [20]. Given this high risk, patients with renal dysfunction could derive greater absolute benefits from an invasive strategy than patients without renal dysfunction; however, the risk for adverse outcomes is also

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high in patients with renal dysfunction [21,22]. Several laboratory procedures were conducted to determine the RF. In clinical laboratories, RF is often evaluated by assessing the renal clearance of any drug. Creatinine is the most often used biochemical marker for RF [23]. It should be emphasised, however, that using creatinine to determine renal function has considerable drawbacks. Creatinine production, for example, is reduced in patients with liver illness [24]. One of the disadvantages of creatinine as a laboratory marker for RF assessment is that its plasma level is affected not only by GFR but also by muscle mass, protein consumption, and composition [25,26]. It should be noted that normal plasma creatinine levels do not always indicate normal RF. Evidence for this is that plasma creatinine levels reach the upper limit of normal only when GFR relative loss exceeds 50% [23]. The GFR is the most trustworthy metric for RF assessment [23]. There are several formulas for calculating GFR. MDRD (modification of diet in renal diseases), Cockcroft and Gault, and CKD-EPI (chronic kidney disease epidemiology collaboration) formulae are a few examples. Some data indicate that the following formulas were compared in terms of the RF predetermination value and the accuracy of the GFR decision. Poggio and co-authors, for example, established the superiority of the MDRD calculation formula over the Cockcroft and Gault method in terms of RF predetermination in their work [27]. As previously stated, plasma creatinine level enhancement requires at least 50% relative loss of GFR, so cases of renal impairment in patients with acute STEMI may not always be detected solely through creatinine control, particularly in patients undergoing primary coronary angioplasty. New trials are required to investigate the effect of renal function on patients with ACS. This study aimed to compare in-hospital complications between patients with acute STEMI and patients with varying RF.

Methods

1. Study design and population

This is a prospective, observational, single-centre study. All of the patients were ≥ 40 years old. A total of 351 STEMI patients were selected for this study and divided into three groups according to GFR. Group 1 comprised 116 patients with < 60 ml/min/1.73 m² GFR. Group 2 consisted of 120 patients with ≥ 60 ml/min/1.73 m² and < 90 ml/min/1.73 m² and Group 3 comprised 115 patients with ≥ 90 ml/min/1.73 m². GFR was estimated with the MDRD formula, which is calculated by using creatinine, age, gender, and race [28]. All of the patients were white. During the first hours after admission, all patients' demographic, clinical, and angiographic data, including age, gender, cardiovascular risk factors such as hypertension and diabetes, STEMI localisation, and angiographic characteristics, were collected. Iohexol was used as a contrast agent. All patients were provided an evaluation of renal function after PCI. None of the included patients was diagnosed with chronic kidney disease (CKD) before admission. All variables, as well as the criteria for the diagnosis of STEMI, acute heart failure (AHF), ventricular tachycardia (VT), ventricular fibrillation (VF), atrial fibrillation (AF), and atrial flutter (AFL), were defined by the European Society of Cardiology

Table 1 - The groups were comparable in terms of drugs received

Drugs received	Group 1 n = 116	Group 2 n = 120	Group 3 n = 115	p-value
Aspirin	100%	100%	100%	–
Clopidogrel	100%	100%	100%	–
Heparin and LMWHs	100%	100%	100%	–
Statins	100%	100%	100%	–
B-blockers	83.6%	84.2%	88.7%	0.49
ACE inhibitors or ARBs	83.6%	88.3%	91.3%	0.2
Aldosterone antagonists	33.6%	34.2%	29.6%	0.72
Calcium antagonist	22.4%	20.8%	13.9%	0.22
Loop diuretics	38.8%	35.8%	32.2%	0.58

LMWHs, low molecular weight heparins; ACE, angiotensin-converting enzyme; ARBs, angiotensin II receptor blockers

(ESC) guidelines [29–31]. All arrhythmias were fixed from the time of admission to before discharge. The clinical practice guidelines of the Infectious Diseases Society of America and the American Thoracic Society were used to diagnose in-hospital pneumonia (IP) [32].

2. Treatment protocol

Patients with severe valvular heart disease, cancer, acute cerebrovascular disease, previously diagnosed CKD, or other serious non-cardiac medical conditions with a life expectancy of less than one year were excluded from the study. Patients that declined to take part in the study were also excluded. All enrolled STEMI patients in three groups underwent primary PCI using a standard technique through the radial artery route following ESC Guidelines on acute myocardial infarction in patients with ST-segment elevation [24]. All patients received as many stents as was deemed clinically appropriate for the infarct-related artery. A subset of patients underwent staged complete revascularisation. All patients in the groups received standard pharmacological treatment for STEMI following current guidelines, including aspirin, clopidogrel, statins, beta-blockers, heparin, angiotensin-converting enzyme inhibitors, or angiotensin II receptor blockers, diuretics, and other medications (Table 1).

3. Study outcomes

In-hospital complications were reported by the treating physician and included the composite rate of APE and CS, pulseless VT/ VF, new-onset AF/AFL, IP, and in-hospital death (ID). At the end of the hospital stays, the rates of in-hospital complications were compared between groups.

4. Statistical analysis

Survey data collection and digitisation was performed using SPSS 26.0 (SPSS Inc., USA) software. Typical statistical methods were used. All calculated p-values were two-tailed and considered statistically significant when < 0.05 . Continuous variables are summarised as mean and standard deviation (SD), and categorical variables are summarised according to frequency and group percentage.

The analysis of patients' data included in the study was performed using the following statistical tests:

- The ANOVA test was used to compare continuous variables between the three groups in the study.
- Student's *t* statistics were used to compare continuous variables between two subgroups.
- Pearson chi-square test was used to compare the frequencies of the indicators compared in the study.
- Categorical variables between groups were analysed using Fisher's z-test as necessary.

Results

Thus, 351 patients with STEMI were included. Mean GFR in Group 1 was 48.2 ± 10.4 ml/min/1.73 m², in Group 2 74.7 ± 8.7 ml/min/1.73 m², and in Group 3 104.1 ± 14.6 ($p < 0.001$). The average age of patients in Group 1 was 66.2 ± 10.1 , 66.2 ± 9.1 , in Group 2 64.5 ± 6.8 , and in Group 3 ($p = 0.23$). The percentage of males was 64.7% in Group 1, 65% in Group 2 and 75.7% in Group 3 ($p = 0.12$). There was a high prevalence of cardiac risk factors and/or comorbid conditions in the groups: 22.4% in Group 1, 23.3% in Group 2, and 24.3% in Group 3 had a history of diabetes ($p = 0.94$), while 68.1% in Group 1, 69.2% in Group 2, and 63.5% in Group 3 had a history of hypertension ($p = 0.62$). Prior myocardial infarction was reported by 11.2% in Group 1, 10% in Group 2, and 4.3% in Group 3 ($p = 0.14$). Anterior STEMI was present in 46.6% of Group 1, 42.5% of Group 2, and 40% of Group 3 ($p = 0.6$). Multivessel disease rates were also comparable between the three groups: 46.6% in Group 1, 52.5% in Group 2, and 41.7% in Group 3 ($p = 0.25$). Also, 7.8% in Group 1, 13.3% in Group 2 and 10.4% in Group 3 underwent staged complete revascularisation ($p = 0.38$). In Group 1, the contrast volumes used were 234.8 ± 65.1 ml, in Group 2 and Group 3, 231.7 ± 63.7 and 224.5 ± 23 , respectively ($p = 0.34$). The incidence of left ventricular ejection fraction in Group 1 was $39.1 \pm 8.5\%$, in Group 2 $44.5 \pm 3.2\%$, and in Group 3 $41.9 \pm 6.8\%$ ($p = 0.16$) (Table 2). The incidence of APE and CS was significantly lower in Group 3 compared to Group 1 and Group 2: in Group 1, Group 2, and Group 3 10.3% (12 of 116 patients), 5.8% (7 of 120 patients), and 0.9% (1 of 115 patients) ($p < 0.05$), respectively. APE and CS rate did not differ significantly between Group 1 and Group 2 ($p > 0.05$). No significant between-group differences were found for pulseless VT or VF: in Group 1, Group 2, and Group 3 pulseless VT or VF was 2.6% (3 of 116 patients), 3.3% (4 of 120 patients), and 0.9% (1 of 119 patients) ($p > 0.05$), respectively. Group 1 showed a considerably higher AF or AFL rate: in Group 1, Group 2, and Group 3 the AF or AFL rate was 12.1% (14 of 116 patients), 5.8% (7 of 120 patients), and 3.5% (4 of 115 patients) ($p < 0.05$), respectively. No significant between-group differences were found for AF or AFL in Group 2 and Group 3 ($p > 0.05$). Also, the authors found significant between-group differences for IP. Group 1 disclosed reasonably higher rates of IP rates than Group 2 and Group 3: in Group 1, Group 2, and Group 3 13.8% (16 of 116 patients), 6.7% (8 of 120 patients), and 4.3% (5 of 115 patients) ($p < 0.05$), respectively. There was no significant difference in IP between

Table 2 - Baseline comparability of the study groups

Variables	Group 1 <i>n</i> = 116	Group 2 <i>n</i> = 120	Group 3 <i>n</i> = 115	<i>p</i> -value
GFR (ml/min/1.73 m ²)	48.2 ± 10.4	74.7 ± 8.7	104.1 ± 14.6	< 0.001
Age, years	66.2 ± 10.1	66.2 ± 9.1	64.5 ± 6.8	0.23
Male	64.7%	65%	75.7%	0.12
Hypertension	68.1%	69.2%	63.5%	0.62
Diabetes	22.4%	23.3%	24.3%	0.94
PMI	11.2%	10%	4.3%	0.14
Anterior STEMI	46.6%	42.5%	40%	0.6
Patients with multivessel disease	46.6%	52.5%	41.7%	0.25
Patients who underwent complete revascularisation	7.8%	13.3%	10.4%	0.38
Killip > II class at the time of admission	3.4%	2.5%	0.9%	0.41
LV EF	$39.1 \pm 8.5\%$	$44.5 \pm 3.2\%$	$41.9 \pm 6.8\%$	0.16
Used contrast volume (ml)	234.8 ± 65.1	231.7 ± 63.7	224.5 ± 23	0.34

GFR, glomerular filtration rate; STEMI, ST-elevation myocardial infarction; PMI, prior myocardial infarction; LV EF left ventricular ejection fraction

Group 2 and Group 3 ($p > 0.05$). ID rate did not significantly differ between the three groups: In-hospital death was 3.4% in Group 1 (4 of 116 patients), 0.8% in Group 2 (1 of 120 patients), and 0 in Group 3 (0 of 115 patients), ($p > 0.05$). Also, we compared contrast-induced nephropathy requiring dialysis: in Group 1 it was 1.7%, in Group 2 and in Group 3 it was 0 ($p = 0.1$).

Discussion

The main finding of this study was the discovery of a direct relationship between RF at the time of hospitalisation and certain in-hospital complications, such as AHF, AF or AFL, IP, and ID. There are several methods for assessing RF, including testing for creatinine, creatinine clearance, urea, the mean of urea and creatinine clearance, and cystatin C, to name a few. CKD is defined as kidney damage or $\text{GFR} < 60$ ml/min/1.73 m² for 3 months or more, irrespective of cause [33]. In the current study, none of the patients were diagnosed with CKD before admission. In the absence of kidney damage, a GFR of 60–89 must be considered a normal RF. Kidney function decreases with age, which is well expressed by determining GFR [34]. It is worth paying attention to the GFR value in the present study: the mean GFR in Group 1 was 48.2 ± 10.4 ; in Group 2, 74.7 ± 8.7 ; and in Group 3, 104.1 ± 14.6 ($p < 0.001$). We reasoned that if all other data were comparable, there had to be statistically significant differences in intra-hospital complications between patients with different GFRs. The current study found that in-hospital complications differed between STEMI patients with different GFRs. Thus, it can be stated that a decrease in the GFR, regardless of the presence of chronic renal dysfunction, complicates the course of the disease and leads

Table 3 - Effect of glomerular filtration rate on in-hospital complications

Outcome variable	Group 1 n = 116	Group 2 n = 120	Group 3 n = 115	p Value
ALE and CS	10.3%	5.8%	0.9%	< 0.05
Pulseless VT or VF	2.6%	3.3%	0.9%	> 0.05
AF or AFL	12.1%	5.8%	3.5%	< 0.05
In-hospital pneumonia	13.8%	6.7%	4.3%	< 0.05
In-hospital death	3.4%	0.8%	0	>0.05
CIN needing dialysis	1.7%	0	0	> 0.05

ALE, acute lung oedema; CS, cardiogenic shock; VT, ventricular tachycardia; VF, ventricular fibrillation; AF, atrial fibrillation; AFL, atrial flutter; CIN contrast-induced nephropathy

to in-hospital complications (Table 3). This study found that patients with GFRs less than 90 ml/min/1.73 m² were more likely to develop complications such as ALE and CS. Patients with GFRs less than 60 ml/min/1.73 m² were more likely to have AF or AFL paroxysms. Also, this study demonstrated a relationship between IP rate and RF at the time of admission. In particular, it was shown that patients with a GFR below 60 ml/min/1.73 m² frequently suffer from IP. It should be noted that there is not enough data in the literature about IP rates in STEMI patients. Also, one study described the correlation between IP and CKD [35]. We supposed that this correlation may be found in STEMI patients with renal dysfunction. Even in the current ESC guidelines for STEMI, no significant differences in the treatment of patients with renal dysfunction with a GFR below ≥ 30 ml/min/1.73 m² have been described [29]. This study suggests that GFR value at admission deserves more attention and that differentiation of GFR values into narrower intervals may be required for STEMI patients to make in-hospital complications more predictable. This study may catalyse other researchers to pay closer attention to patient GFR values at the time of admission to investigate GFR-related in-hospital complications as well as further short- and long-term prognosis.

1. Study limitations

This study has some limitations. The study included only patients ≥ 40 years of age. The authors suggest that if patients were included in the study without age restrictions, the patient groups would differ significantly in the basic parameters. The second limitation is that only one method was chosen to determine the GFR. It may be more efficient to calculate GFR using at least two methods. The Cockcroft and Gault method of calculating GFR, for example, could have been another option, but it was not possible to collect information on patients' exact weights during the study.

Conclusion

RF at the time of admission is an independent predictor of in-hospital complications. A GFR of less than 90 ml/min/1.73 m² increases the in-hospital APE and CS rate in STEMI patients

undergoing primary PCI. A GFR of less than 60 ml/min/1.73 m² increases IP and also AF or AFL rates in STEMI patients undergoing primary PCI. RF at the time of admission does not affect pulseless VT or VF nor ID rates in acute STEMI patients undergoing primary PCI. Evaluation of kidney function based on GFR in STEMI patients may make in-hospital complications more predictable.

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Competing Interests

The authors report no competing interests.

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Abbreviations

STEMI, ST-elevation myocardial infarction; GFR, glomerular filtration rate; PE, acute pulmonary oedema; cardiogenic shock, CS; in-hospital pneumonia, IP; pulseless ventricular tachycardia, VT; ventricular fibrillation, VF; new-onset atrial fibrillation, AF; atrial flutter, AFL; coronary artery disease, CAD; renal function, RF; acute myocardial infarction, AMI; percutaneous coronary intervention, PCI; modification of diet in renal diseases, MDRD; chronic kidney disease epidemiology collaboration CKD-EPI; chronic kidney disease, CKD; acute heart failure, AHF; ventricular tachycardia, VT; ventricular fibrillation, VF; atrial fibrillation, AF; atrial flutter, AFL; European Society of Cardiology, ESC; in-hospital death, ID.

AHF, acute myocardial infarction, AMI; atrial fibrillation, AF; atrial flutter, AFL; atrial flutter, AFL; cardiogenic shock, CS; chronic kidney disease epidemiology collaboration CKD-EPI; chronic kidney disease, CKD; coronary artery disease, CAD; European Society of Cardiology, ESC; GFR, glomerular filtration rate; in-hospital death, ID; in-hospital pneumonia, IP; modification of diet in renal diseases, MDRD; new-onset atrial fibrillation, AF; PE, acute pulmonary oedema; percutaneous coronary intervention, PCI; pulseless ventricular tachycardia, VT; renal function, RF; STEMI, ST-elevation myocardial infarction; acute heart failure, ventricular fibrillation, VF; ventricular fibrillation, VF; ventricular tachycardia, VT;

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