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Risk of Acute Kidney Injury after Off-Pump Coronary Artery Bypass Grafting in Patients with Poor Glycemic Control

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ABSTRACT

Introduction: Coronary artery bypass grafting (CABG) is a commonly performed procedure in ischemic heart disease (IHD) patients. Acute kidney injury (AKI) is a frequent complication of CABG. Many patients undergoing surgery have Diabetes Mellitus, which increases risk of AKI after CABG. Limited work has been done to see the impact of poor glycemic control on incidence of AKI after off-pump CABG. However, local data on the subject was missing especially using OFF PUMP technique.
Aims & Objectives: Aim of this study was to find effects of poor glycemic control on acute kidney injury after Off-pump CABG.
Place and Duration of Study: The study was carried out at the Department of Cardiothoracic Surgery, Shaikh Zayed Hospital, Lahore from 1st October 2021 to 30th September 2022.
Material & Methods: 130 diabetic patients were divided into two equal groups of 65, exposed to poor glycemic control (HbA1c ≥ 6.5%) and unexposed with good glycemic control (HbA1c < 6.5%). Postoperative variables like urine output, serum creatinine levels and e-GFR were recorded for 72 hours. Mean ± SD were computed for quantitative variables and frequency (%) was computed for qualitative variables using SPSS 24.0.0: Risk ratio >1 was considered as significant.
Results: In the unexposed group, 58 individuals (89.2%) did not experience AKI after 72 hours, while 7 individuals (10.8%) did. Conversely, exposed, 48 individuals (73.8%) did not experience AKI in the exposed group, while 17 individuals (26.2%) did. Relative risk calculated was 2.35(CI: 1.12- 7.70), p=0.01.
Conclusion: Poor glycemic control has increased risk for developing AKI, increased serum creatinine levels and eGFR after off-pump-CABG compared to patients with good glycemic control.

Keywords: AKI, CABG, Diabetes Mellitus, Glycemic control, Acute Kidney Injury

INTRODUCTION

CABG surgery is performed to develop a bypass of diseased coronary arteries to improve blood flow to the heart. Type II diabetes mellitus (DM) is responsible for 20-50% cases of CABG, and it has increased progressively during the last two decades[1]. One of the most worrisome dangers of CABG is acute renal failure. The rate of acute renal failure after CABG has been reported at 3.7%, with mortality rate of 44.4% in these patients[2]. Another study reported 30-day mortality rate of 63.7% in patients that developed ARF after CABG, compared to 4.3% in patients that did not develop ARF[3]. In post-CABG patients, acute renal injury has various pathophysiological pathways. Acute kidney injury after bypass operation is due to quick worsening in the functionality of renal system expressed as the momentous reduction in the glomerular filtration rate[4]. It is characterized by an abrupt decline in renal function and in addition to raising the risk of cardiovascular events, chronic renal disease progression and long-term mortality; it can also raise hospital stays, healthcare expenses, and death[5]. The chance of developing CKD, which lasts for more than 90 days, is also increased in AKI survivors. AKI is significantly predicted by factors such as age, comorbidities, and exposure to sepsis, coronary artery bypass grafting (CABG), and shock[5]. Previous epidemiological studies observe the prevalence of acute kidney injury after bypass surgery was around 30%, of which 2% needed hemodialysis. It is a significant indicator for increased morbidity and mortality after surgery[6]. Diabetes mellitus, which has an average prevalence of 20–30%, is a serious problem in patients undergoing CABG. These individuals are at a higher risk of worse perioperative outcomes and shorter overall survival[7]. Type II diabetes is an independent

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predictor of higher post- CABG morbidity and mortality[8]. In patients with acute myocardial infarction, acute kidney injury (AKI) occurs due to an acute rise in glycemic level. This highlights a significant independent role of acute glycemic increase beside the high glycemic level at presentation. Using both acute and chronic glycemic levels, a study looked at how hyperglycemia may predict acute kidney damage (AKI) in diabetic individuals who had just experienced an acute myocardial infarction[9].

Gao S, et al examined baseline characteristics and outcomes in diabetic patients with AMI and demonstrated a substantial correlation between uncontrolled hyperglycemia and a higher risk of AKI[10]. Kocogulları CU, et al found that the acute kidney injury occurred in 3.6% patients with good glycemic control while in 16.7% patients with poor glycemic control after CABG[11]. According to Hertzberg, et al AKI occurred in 32% of type 1 diabetes mellitus (T1DM) patients and 20% of T2DM patients after the CABG procedure[12].

Off-pump CABG, which excludes the cardiopulmonary bypass, is found to be associated with lower incidence of acute kidney injury. However, given the fact that this complication has serious consequences local evidence was missing. This study was aimed to highlight the effects of poor glycemic control on acute kidney injury after Off-pump CABG.

MATERIAL AND METHODS

This prospective cohort study was carried out at Department of Cardiothoracic Surgery, Shaikh Zayed Hospital, Lahore from 1st October 2021 to 30th September 2022. After taking approval from IRB (SZMC/IRB/0072/2021) and informed consent, 130 Diabetic patients meeting selection criteria were selected. Patients were divided into two equal groups on the basis of their glycemic control according to HbA1c levels at the time of recruitment. 65 patients were included in exposed group with poor glycemic control (HbA1c $\geq$ 6.5 mg/dl) and 65 patients were included in unexposed group with good glycemic control (HbA1c < 6.5mg/dl). Non-probability, consecutive sampling was used. All diabetic patients aged 35-65 years, both genders undergoing elective CABG with normal pre op baseline serum creatinine < 1.4mg/dl were included. Patients with rheumatic /valvular heart disease, chronic kidney disease stage V, end-stage renal disease on hemodialysis and urgent/emergent CABG were excluded from the study. Power of the study was set at 80%, with a significance level of 5%. Variables like name, age, gender, and body mass index (BMI), smoking history, dyslipidemia, and ejection fraction, HbA1c levels were recorded. All patients had off-pump CABG by the same surgical team. After surgery, patients were shifted to ICU. Serum creatinine, urine output and e-GFR were recorded every 24 hours up to 72 hours. Acute Kidney Injury (AKI) was defined as an increase in serum creatinine of ≥0.3 mg/dL within 48 hours compared to baseline values, as per KDIGO criteria. Data was analyzed by using SPSS v.24. Mean ± SD were computed for quantitative variables, Frequency (%) was computed for qualitative variables

RESULTS

A total of 130 individuals were included in the analysis. The central tendency of the age distribution yielded a mean age of 49.7 years (σ= 8.2, 35-65) and mean BMI was 27.6 Kg/m^2 (σ= 3.5Kg/m^2, 21-38). The mean duration of Myocardial Infarction was 38 days(σ=16.1), mean duration of surgery was 3.5 hours and mean number of Grafts was 2.8(2-5). Distributions of all variables were insignificant in both groups except bifurcation of pre and per-operative variables which showed difference between the exposed and unexposed groups in mode of diabetes treatment and ejection fraction. (Table 1). Mean pre-operative creatinine was 0.96 mg/dL (σ= 0.21), while post-operative levels after 24 hours show a mean of 1.10 mg/dL (σ= 0.36), at 48 hours, the mean was1.14 mg/dL (σ= 0.60) and at 72 hours it was 1.31 mg/dL (σ= 0.75). The study also focused on urine output levels in relation to pre- operative and post-operative periods over 24, 48, and 72 hours. For pre-operative levels, the mean urine output was 2619 ml/24 hours (σ= 979). Post-operative urine output was recorded as mean of 2487ml/24 hours (σ= 879) at 24 hours, 2217ml/24 hours (σ= 987) for 24-48 hours post-op and 1830ml/24 hours (σ= 819) at 48-72 hours post-op. Overall, at the end of 3rd post- operative day, 24 (18.5%) patients had developed acute kidney injury. 7 of these cases had good pre-operative glycemic control according to our criteria while 17 had poor pre-operative glycemic control. This translates to a relative risk of 2.35 (10.8% vs 26.2%) which is significant with p-value of 0.01.(Table 2)

Table 3 and 4 show mean serum creatinine level and urine output per 24 hours. In summary, the results underscore that patients in the exposed group have a significantly higher risk of developing AKI after 72 hours compared to those with unexposed. The calculated RR and its confidence interval further support this conclusion, highlighting the potential effect of diabetes mellitus on the occurrence of acute kidney injury after CABG.

Table 1: Demographic & Lab. variables of Unexposed (good GC, HbA1c < 6.5%) vs. Exposed (poor GC, HbA1c ≥ 6.5%)

Variables	Categories	Unexposed	Exposed	p-value
Age				0.19
	Mean ± SD	49.4 ± 8.2	50.1 ± 8.3	
Gender				0.46
	Female	24 (36.9)	20 (30.1)	
	Male	41 (63.1)	45 (69.2)	
Body mass index (kg/m²)				0.31
	Mean ± SD	27.3 ± 3.5	27.9 ± 3.4	
Mode of diabetic treatment				0.003
	Oral	32 (49.2)	25 (38.5)	
	Insulin	30 (46.2)	23 (35.4)	
	Both	3 (4.6)	17 (26.2)	
Smoking status				0.72
	No	37 (56.9)	39 (60.0)	
	Yes	28 (43.1)	26 (40.0)	
Dyslipidemia				0.63
	Balanced	9 (13.8)	11 (16.9)	
	Imbalanced	56 (86.2)	54 (83.1)	
Ejection fraction				0.004
	<30%	4 (6.2)	18 (27.7)	
	30-50%	35 (53.8)	24 (36.9)	
	50-70%	26 (40.0)	23 (35.4)	
Myocardial infarction (days)				0.12
	Mean ± SD	36.9 ± 17.5	41.4 ± 14.7	
CABG surgery (hours)				0.91
	Mean ± SD	4.5 ± 1.2	4.6 ± 1.1	
Number of grafting				0.53
	Mean ± SD	2.7 ± 0.5	2.8 ± 0.4	

Table 2: Bifurcation of incidence of AKI after 72 hours with respect to grouping variables Unexposed versus Exposed

Variables	Categories	Unexposed	Exposed	RR	(CI), p-value
Incidence of acute kidney injury after 72 hours				2.35	(1.12-7.70), 0.01
	AKI absent	58 (89.2)	48 (73.8)		
	AKI present	7 (10.8)	17 (26.2)		

Table 3: Bifurcation of creatinine levels in Unexposed versus Exposed

Variables	Categories	Unexposed	Exposed	p-value
Creatinine levels (pre-operative) mg/dL				0.78
	Mean ± SD	0.98 ± 0.23	0.97 ± 0.20	
Creatinine levels (post-operative after 24 hours) mg/dL				0.26
	Mean ± SD	1.03 ± 0.30	1.10 ± 0.42	
Creatinine levels (post-operative after 48 hours) mg/dL				0.005
	Mean ± SD	0.99 ± 0.27	1.30 ± 0.76	
Creatinine levels (post-operative after 72 hours) mg/dL				0.003
	Mean ± SD	1.10 ± 0.40	1.50 ± 0.95	

Table 4: Bifurcation of urine output at different times of Unexposed versus Exposed patients.

Variables	Categories	Unexposed	Exposed	p-value
Urine Output (pre-operative) ml/24 hours				0.07
	Mean ± SD	2690.5 ± 995.5	2575.1 ± 912.2	
Urine Output (post-operative after 24 hours) ml/24 hours				0.05
	Mean ± SD	2567.9 ± 911.2	2412.3 ± 827.8	
Urine Output (post-operative after 48 hours) ml/24 hours				0.001
	Mean ± SD	2301.2 ± 957.6	1985.5 ± 878.6	
Urine Output (post-operative after 72 hours) ml/24 hours				0.001
	Mean ± SD	1970.5 ± 847.8	1802.2 ± 789.3	

DISCUSSION

Typically, the patients that need to undergo CABG exhibit chronically elevated blood glucose concentrations due to stress, impaired glucose tolerance, or diabetes upon hospital admission. Research indicates a progressive relationship between glucose levels and cardiovascular disease, extending beyond the current diabetes mellitus threshold[13,14]. A 2009 research suggested diagnosing diabetes when HbA1c is ≥6.5%[15].

AKI, following cardiac surgery, leads to prolonged stays in ICU and hospital, resulting in increased healthcare expenses and higher mortality rates[16,17]. The risk factor for AKI includes advanced age, DM, poor LV, preexisting kidney dysfunction, urgent or emergent surgery and administration of nephrotoxic substances[17,18]. Both groups exhibited similar demographic data and risk factors for AKI, including creatinine levels, urine outputs and eGFR values.

Moreover, certain investigations have indicated a heightened incidence of postoperative acute kidney injury (AKI) following CABG among individuals with type-2 DM[19]. The level of HbA1c serves as a parameter for assessing the long-term management of glycemic control in patients affected by DM[20]. Typically, HbA1c levels within the range of 4-6% are considered normal. In a study by Tekumit et al., it was discovered that an HbA1c level of 6.1% represented a borderline threshold for patients undergoing CABG[21]. Through their retrospective analysis, another study conveyed that preoperative HbA1c levels exceeding 6% were linked to both 30-day postoperative mortality and the occurrence of AKI among DM patients who had undergone open heart surgery[22].

In our investigation, participants were categorized based on their HbA1c levels, with an HbA1c level of <6.5% serving as the threshold for the borderline category. Notably, patients exhibiting levels exceeding ≥6.5% displayed a notably elevated occurrence of AKI, as classified by KDIGO criteria. Our findings demonstrated that higher HbA1c levels, in contrast to findings in other studies, posed a risk factor for AKI[23-26]. While a consensus is lacking regarding the relationship between AKI and HbA1c levels among patients without known renal issues, HbA1c levels surpassing 7% are categorized as a Class 1A risk factor for individuals with chronic renal disease[26]. Within our patient cohort, the identified threshold for AKI was determined to be an HbA1c level of ≥6.5%.

CONCLUSION

In conclusion, our study provides valuable insights and highlights that patients with poor glycemic control had more chances of developing increased serum creatinine level, low urine output and decreased eGFR leading to AKI even after using OFF-PUMP technique of CABG.

Limitations and Recommendations

There is a requirement for additional investigations involving larger patient cohorts and data from multiple medical centers. As the local evidence is missing, so this study would help in attaining local evidence which in future will lead to implement the screening and prevention of patients from acute kidney injury after CABG leading to improved patient outcomes in-terms morbidity and mortality.

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