SMGroup

SM Journal of Nephrology and Therapeutics

Research Article

Impact of Long-Standing Poor Glycemic Control on the Occurrence of Contrast-Induced Acute Kidney Injury in Patients with Type-II Diabetes Mellitus Undergoing Percutaneous Coronary Intervention

Emad Abdallah[1]*, Osama Mosbah[1], Nevine Sherif[1], Noha El-Shiegh[1], Ahmed Ali[2] and Ahmed Abdullah[3]

[1]*Department of Nephrology, Theodor Bilharz Research Institute, Egypt*
[2]*Intensive Care Unit, Theodor Bilharz Research Institute, Egypt*
[3]*Cardiology Care Unit, National Heart Institute, Egypt*

Article Information

Received date: Nov 23, 2016
Accepted date: Jan 02, 2017
Published date: Jan 05, 2017

***Corresponding author**

Emad Abdallah, Associate professor of nephrology, Theodor Bilharz Research Institute, El-Nile St.Waraq El-Hadar, P.O.Box: 30 Imbaba Giza, 12411, Egypt, Phone: 00201005767492; Fax: 20235408125; Email: drabdallah96@gmail.com

Keywords Acute kidney injury; acute myocardial infarction; diabetes mellitus; HbA1c

Article DOI 10.36876/smjnt.1005

Abstract

Background: Many studies revealed that hyperglycemia on hospital admission increase the risk of Acute Kidney Injury (AKI) in patients undergoing Percutaneous Coronary Intervention (PCI), however, there is a little data regarding the effect of long-standing hyperglycemia on AKI occurrence in patients with myocardial infarction undergoing primary PCI.

Objectives: The aim of this study was to evaluate the effect of long-standing poor glycemic control on AKI occurrence in patients with Type-II Diabetes Mellitus (T2DM) and acute ST-Elevation Myocardial Infarction (STEMI) undergoing primary PCI.

Patients and Methods: We prospectively studied 120 patients with T2DM and acute STEMI undergoing primary PCI. According to glycosylated hemoglobin (HbA1c), patients were divided into 2 groups, patients with HbA1c <7% (Group 1, n=47) and patients with HbA1c ≥ 7% (Group-II, n=73). The estimated Glomerular Filtration Rate (eGFR) was estimated using the abbreviated Modification of Diet in Renal Disease equation (MDRD) and patients with eGFR <60ml/min/1.73m^2 were excluded from the study. Medical records of both groups of patients were reviewed for the occurrence of AKI. AKI was determined using Kidney Disease/Improving Global Outcomes (KDIGO) guidelines and defined as increase in serum creatinine by 0.3 mg/dl (26.5 umol/l) within 48 hours of admission.

Results: AKI was found in 3 of 47 patients (6.38%) in Group-I and in 16 of 73 patients (21.9%) in Group-II (p = 0.0436). Baseline serum creatinine and estimated Glomerular filtration rate were comparable between the two groups. There was positive significant correlation between the HbA1c levels and the incidence of AKI in the studied patients (p=0.0001). Using multivariate regression analysis, HbA1c was found to be one of the independent risk factors of Contrast-Induced Acute Kidney Injury (CI-AKI).

Conclusion: An elevated HbA1c levels were associated with a higher incidence of CI-AKI compared with an optimal HbA1c levels in T2DM patients with an eGFR of ≥60 ml/min/ 1.73 m^2 and STEMI treated with pimary PCI.

Introduction

Coronary heart disease is one of the leading causes of death worldwide and remains a substantial contributor to morbidity, mortality and healthcare expenditure. The treatment of choice for many patients with stable Coronary Artery Disease (CAD) is revascularization using Percutaneous Coronary Intervention (PCI). Advances in PCI technology have resulted in increasing numbers of patients undergoing coronary revascularization via this approach. In Europe, 15 million people had PCI in 2010, and it is estimated that 15 million patients undergo PCI in the United States every year [1].

Contrast-Induced Acute Kidney Injury (CI-AKI) continues to be one of the most common major adverse side effect of cardiac catheterization, and is associated with short- and long- term morbidity and mortality [2,3]. This is particularly true in the population presenting with acute ST-Elevation Myocardial Infarction (STEMI). Coronary Angiography (CAG) and PCI are associated with the highest rates of Acute Kidney Injury (AKI) [4,5] mainly related to the intra-arterial injection and to the high dosage of the contrast necessary, and also to the type of patients who have advanced age, one or more comorbid conditions, and more advanced vascular disease, hypertension, and Diabetes Mellitus (DM) [6].

OPEN ACCESS
ISSN: 2573-3613

The increased prevalence of Type-II Diabetes Mellitus (T2DM), a known significant risk factor of CI-AKI, also contributes to this process. A long-standing hyperglycemic milieu is considered to be responsible for the increased incidence of CI-AKI in patients with T2DM [7]. Several studies have reported that acute hyperglycemia also increases the risk of CI-AKI and therefore mortality [8-10]. This has been associated with the pathophysiological similarity of the adverse effects of both hyperglycemia and iodinated Contrast Media (CM) on kidneys (oxidative stress, endothelial dysfunction and vasoconstriction) [11-13].

Objectives

Since there are no adequate clinical studies to demonstrate whether long-standing poor glycaemic control further increases the risk of CI-AKI. Accordingly, in this study, we investigated the effect of long-standing poor glycemic control (using HbA1c, as a marker of glucose control in the last 2-3 months) on AKI occurrence in patients with T2DM and STEMI undergoing CAG and primary PCI.

Patients and Methods

Study population

This a prospective study conducted on 120 patients with T2DM admitted during the period from January 2013 to May 2016 at National Heart Institute and Theodor Bilharz Research Institute, Cairo, Egypt, with acute STEMI for CAG and treated by PCI. DM was diagnosed by history of DM diagnosed previously or history of receiving anti-diabetic medications. STEMI was diagnosed as patients had typical chest pain, serial elevation of cardiac Troponin with echo-heart changes. Primary PCI was performed on patients with symptoms from 12 to 24 h duration. CM used in procedures was Iodixanol (Visipaque, GE healthcare, Ireland) or Iohexol (Omnipaque, GE healthcare, Ireland). Following CAG and PCI procedures, normal saline (0.9%) was given intravenously at a rate of 1 ml/kg/h for 24h after contrast exposure. The hydration rate was reduced in patients with volume overload as patients with heart failure. Left Ventricular Ejection Fraction (LVEF) was assessed in all patients within the first 48h of admission. Patients with Estimated Glomerular Filtration Rate (eGFR)<60ml/min/1.73m^2 and critically ill patients on mechanical ventilation or intra-aortic balloon counter pulsation were excluded from the study.

According to glycosylated hemoglobin (HbA1c), patients were divided into 2 groups, patients with HbA1c <7% (Group-I, n=47) and patients with HbA1c ≥7% (Group-II, n=73). A cutoff point of 7% was chosen because it is the recommended target of glycaemic control for T2DM to reduce complications [14].

Random blood glucose level was measured on admission. HbA1c levels were measured from blood samples taken within 24h of hospital admission. Renal function tests (serum creatinine, blood urea, serum potassium, serum sodium, and serum uric acid) were measured on hospital admission, and at least once a daily. The eGFR was estimated using the abbreviated Modification of Diet in Renal Disease equation (MDRD) [15]. Baseline renal insufficiency was categorized as admission eGFR of <60ml/min/1.73m^2 [16]. AKI was determined using KDIGO guidelines and defined as increase in serum creatinine by 0.3 mg/dl (26.5umol/l) within 48 hours of admission [17].

Ethical issues

1) The research followed the tenets of the Declaration of Helsinki; 2) Informed consent was obtained from all patients included in the study and they were free to leave the study at any time; and 3) The research was approved by ethical committee of National Heart Institute and Theodor Bilharz Research Institute.

Statistical analysis

All data are presented as Mean±Standard Deviations (SD) or percentages. Continuous variables were compared using the unpaired two-tailed Student's t-test (using GraphPad QuickCalcs software). The p-values for the categorical variables were calculated with the chi square test. Pearson's rank correlation test was used to analyse the correlation between HbA1c and serum creatinine (using MedCalc software). Multivariate linear regression analysis was used to study the predictive factors of CI-AKI. Statistical analyses were performed using SPSS version-16 for Windows software (SPSS Inc., Chicago, IL, USA). A P-value <0.05 was considered statistically significant and P-value <0.01 was considered highly statistically significant.

Results

The patients included in this study were 120 patients with T2DM with mean age 62.7±9.2 (75% males), 47 of whom (39.2%) had HbA1c <7% and 73 of whom (60.8%) had HbA1C level ≥7%. The baseline demographic, clinical and laboratory characteristics of patients according to the HbA1c levels are presented in (Table 1). The two groups were comparable regarding age, gender, hypertension, dyslipidemia, hyperuricemia and extent of coronary artery disease.

Table 1: Demographic, clinical and laboratory characteristics of studied patients.

Variables	Group-I (HbA1c <7%) No.47	Group-II (HbA1c ≥7%) No.73	*p* value
Age (years)	59.4±9.3	60.3±8.4	0.5839
Gender (M/F)	34/13(72.3%/27.7%)	56/17(76.7%/23.3)	0.7434
Systolic BLP (mm/Hg) Diastolic BLP (mm/Hg)	132.8±23.5 89.2±16.6	138.3±28.2 92.6±21.5	0.2688 0.3588
Insulin therapy	8(17.02%)	43(58.9%)	0.0001
Glucose level at admission (mg/dl)	117.6±36.4	238.3±48.2	0.0001
HbA1c (%)	5.8±1.2	8.6±1.6	0.0001
Total cholesterol (mg/dl) Triglycerides (mg/dl)	170.4±36.7 86.3±18.4	171.8±38.2 88.4±19.2	0.8426 0.5534
Serum uric acid (mg/dl)	6.43±3.21	6.64±2.52	0.6901
Personal history of MI	8(17.02%)	19(26.03%)	0.3525
Family history of CAD	14(29.8%)	29(39.7%)	0.3633
No of stenosed coronary arteries 1 2 3	 11(23.4%) 16(34.04%) 20(42.6%)	 15(20.5%) 21(28.8%) 37(50.7%)	 0.8812 0.6895 0.4965
Time to reperfusion (h)	7.3±5.2	8.2±4.9	0.3396
C-RP (mg/l)	15.2±6.3	16.7±6.8	0.2274
C-PK (units/l)	1368.4±432.6	1412.8±471.4	0.6041
LVEF (%)	51.3±7.4	48.6±8.2	0.0701

BLP: Blood Pressure; **HbA1c:** Glycosylated Hemoglobin; **MI:** Myocardial Infarction; **CAD:** Coronary Artery Disease; **C-RP:** C - reactive protein; **C-PK:** Creatine Phosphokinase; **LVEF:** Left Ventricular Ejection Fraction;

Citation: Abdallah E, Mosbah O, Sherif N, El-Shiegh N, Ali A and Abdullah A. Impact of Long-Standing Poor Glycemic Control on the Occurrence of Contrast-Induced Acute Kidney Injury in Patients with Type-II Diabetes Mellitus Undergoing Percutaneous Coronary Intervention. SM J Nephrol Therap. 2017; 2(1): 1005. https://dx.doi.org/10.36876/smjnt.1005

Table 2: Serum creatinine changes, intravenous contrast volume applied and the occurrence of AKI according to HbA1c levels.

Variables	Group-I (HbA1c <7%) No.47	Group-II (HbA1c ≥7%) No.73	*p* value
Serrum creatinine at admission (mg/dl)	1.24±0.62	1.38±0.64	0.2388
eGFR (ml/min/1.73 m^2)	77.12±16.7	74.23±14.2	0.3121
Contast volume (ml)	140.8±12.4	138.7±14.2	0.4081
Serrum creatinine 48h after PCI	1.31±.83	1.68±.62	0.0062
Serum creatinine at discharge	1.23±0.65	1.43±0.41	0.0408
AKI	3(6.38%)	16 (21.9%)	0.0436

eGFR, estimated glomerular filtation rate; PCI, percutaneous coronary intervention; AKI, acute kidney injurry

Table 3: Multivariate regression analysis of the risk factors for CI-AKI.

Variables	*β*	*p* value
HbA1c	0.62	0.021
LVEF	-0.37	0.041
Age	0.53	0.024
Contrast volume	0.59	0.022
Blood glucose	0.42	0.032

HbA1c, glycosylated hemoglobin; LVEF, left ventricular ejection fraction.

Patients with HbA1c level ≥7% were more likely to be treated with insulin (58.9% *vs* 17.02%; P<0.001) with significantly higher admission glucose levels (259±95 *vs* 163±71 mg/dl; p =0.0001), and higher HbA1c (8.6±1.6 *vs*. 5.8±1.2; p=0.0001).

(Table 2) compares the serum creatinine changes, intravenous contrast volume applied and the occurrence of AKI according to HbA1c levels. Baseline serum creatinine and eGFR were comparable between the two groups. The total volume of CM was not statistically different between the two groups. Serum creatinine change at 48h after PCI and on discharge was highly significant in Group-II with HbA1c ≥7% than Group-I with HbA1c <7%. AKI was found in 16 of 73 (21.9%) in Group-II of patients with HbA1c ≥7% and in 3 of 47 (6.38%) in Group-I of patients with HbA1c <7% (p = 0.0436).

There was positive significant correlation between the HbA1c levels and the incidence of AKI in the studied patients (Figure 1).

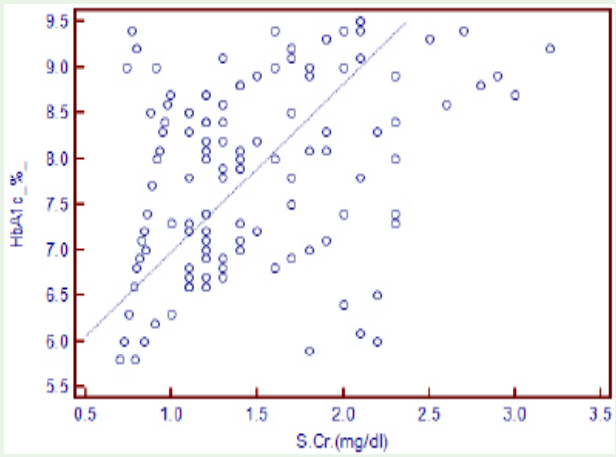

Figure1: Correlation between glycosylated hemoglobin (HbA1c) and serum creatinine (S.Cr) {r = 0.353, 95% confidence interval for r 0.186 to 0.501, p = 0.0001}.

Multivariate linear regression analysis was used to define independent risk factors of AKI. According to regression analysis, HbA1c, age, LVEF and volume of CM were found to be independent risk factors of AKI (Table 3).

After adjusting for age, sex, LVEF, multi-vessel disease, volume of CM, and blood glucose, HbA1c remained an independent risk factor for CI-AKI.

Discussion

Contrast-Induced Acute Kidney Injury (CI-AKI) is a prevalent but under diagnosed complication of PCI that is associated with increased in-hospital morbidity and mortality [3,18-20]. The importance of this complication is being increasingly recognized. Several recent North American and European epidemiological studies have shown that the incidence of AKI is increasing at an alarming rate [21]. Patients with DM, pre-existing renal insufficiency, congestive cardiac failure or advanced age are particularly susceptible to developing CI-AKI post-PCI.

The present study revealed that, elevated HbA1c levels were associated with increased incidence of CI-AKI in patients with T2DM (patients with an eGFR of ≥60 ml/min/1.73m^2) and STEMI undergoing PCI.

There are several previous reports demonstrated that acute hyperglycemia was associated with a significant increase of AKI following primary PCI [22,23]. Several studies have demonstrated that admission hyperglycemia in patients with STEMI increased the incidence of AKI, cardiac failure and mortality even in the absence of a history of T2DM [24-27].

The results of two recent studies regarding the relationship of admission hyperglycemia and CI-AKI are particularly interesting. These studies have demonstrated similar rates of CI-AKI development in T2DM patients with and without admission hyperglycemia [8,9]. However, the development of CI-AKI was more common in non-diabetic patients with admission hyperglycemia compared to those without admission hyperglycemia. The difference might possibly be explained by the administration of a more aggressive insulin therapy in patients with T2DM during hospitalization, a better hydration of these patients since T2DM is known to be a risk factor for CI-AKI, and the need for a greater stress factor in association with its secondary adverse effects in non-diabetics which generate a comparably high level of glucose.

Diabetes doubles the risk of developing AKI compared with non-diabetic patients. The incidence of AKI in diabetic patients varies from

5.7 to 29.4 %. The administration of iodinated radiocontrast media to diabetics acutely reduces renal parenchymal oxygenation, a reduction that is most prominent in the renal medulla, since it already functions at low oxygen tension [28]. The biologically active endothelins are produced by proteolysis of the precursor prepro endothelins under the action of endothelin-converting enzyme that plays a key role in increasing circulating and renal endothelin levels found both in diabetes and after exposure to contrast agents. This may explain the particular susceptibility of diabetic patients to contrast media [28].

The increased incidence of AKI in diabetic patients has also been attributed to hypersensitivity of renal vessels of diabetics to adenosine, a vasoconstrictive agent, since experimental studies have shown increased adenosine-induced vasoconstriction in the kidneys of diabetic animals and the administration of adenosine receptor antagonists reduces the risk of development of contrast-induced AKI in both diabetic and non-diabetic patients [29,30]. Moreover, hyperglycemia might cause hypovolemia by increasing the Osmotic Diuresis.

To date, there have been few studies on the relationship between long-standing poor glycaemic control and CI-AKI. In their retrospective study, Ding et al [31] have reported higher glycated albumin and HbA1c levels in patients with CI-AKI compared to those without CI-AKI (8.3±1.6% *vs* 7.5±1.2% for HbA1c, respectively, $p<0.001$). However, in this study, it might be incorrect to suggest uncontrolled glucose levels as the primary cause of CI-AKI. Since the rate of pre-existing Chronic Kidney Disease (CKD) was statistically significantly higher, the LVEF was lower, the number of elderly patients was higher and a greater amount of CM was used in patients with CI-AKI compared to those without CI-AKI ($p<0.001$, for all). However, in our study, eGFR, age, LVEF, and CM volume were comparable between the two groups. In the other study, Yoshikawa et al [32] have reported a 5% increase in serum creatinine and a decrease of 4ml/min/1.73m^2 in eGFR in patients with an HbA1c of ≥6.5% compared to those with an HbA1c of <6.5% following coronary computed tomography angiography ($p<0.001$). However, such a small change in the values neither fits the definition of CI-AKI nor has any known clinical implications. A recent repo by Akyuz et al [33] demonstrated no difference in the rate of AKI following elective PCI in Type-II diabetic patients undergoing elective PCI. Marenzi et al [23] demonstrated similar rates of AKI development following primary PCI in T2DM patients with and without admission hyperglycemia.

The results of the present study might be explained with the long and more marked effect of chronic intra-renal mechanisms on kidneys due to long-standing poor glycemic control (i.e., changes in the intra-glomerular haemodynamics modulated in part by local activation of the renin-angiotensin system, biochemical derangements, proteinuria, and hypoxia) in addition to the direct effect of hyperglycemia in terms of the development of CI-AKI in T2DM [34].

Our study has several limitations: 1) Limited number of patients; 2) Urine albumin was not measured and as CKD is defined by an eGFR of ≥60 ml/min/1.73m^2 only in the presence of albuminuria for patients with T2DM, it can be presumed that some of the patients have CKD and others do not; 3) As patients with acute STEMI cannot receive pre-procedural hydration, extrapolation of the results to these patients may not be appropriate; 4) These results might also not be valid for radiological procedures that use the intravenous rather than the intra-arterial route; 5) These results might not be valid for patients with Type-I DM; and finally, 'hospital-induced nephropathy', a newly recognized aspect, described as a substantial day-to-day variation in serum creatinine in hospitalized patients regardless of CM injections, might have been a confounding factor [35].

Conclusion

In conclusion, an elevated HbA1c level is associated with a higher incidence of CI-AKI compared with an optimal HbA1c level in patients with T2DM (patients with an eGFR of ≥60ml/min/1.73m^2) and STEMI undergoing CAG and/or PCI.

References

1. Lloyd-Jones D, Adams RJ, Brown TM, Carnethon M, Dai S, de SG, et al. Heart disease and stroke statistics-2010 update: a report from the American Heart Association. Circulation. 2010; 121: 46-215.
2. Marenzi G, Lauri G, Assanelli E, Campodonico J, De Metrio M, Marana I, et al. Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. J Am Coll Cardiol. 2004; 44: 1780-1785.
3. Rihal CS, Textor SC, Grill DE, Berger PB, Ting HH, Best PJ, et al. Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation. 2002; 105: 2259-2264.
4. Mehran R, Nikolsky E. "Contrast-induced nephropathy: definition, epidemiology, and patients at risk", Kidney International. 2006; 100: 11-15.
5. Solomon R. Contrast-induced acute kidney injury: Is there a risk after intravenous contrast? Clinical Journal of the American Society of Nephrology. 2008; 3: 1242-1243.
6. Fuiano G, Mancuso D, Indolfi C, Mongiardo A, Sabbatini M, Conte G, et al. "Early detection of progressive renal dysfunction in patients with coronary artery disease," Kidney International. 2005; 68: 2773-2780.
7. Mc Cullough PA, Adam A, Becker CR, Davidson C, Lameire N, Stacul F, et al. CIN Consensus Working Panel. Risk prediction of contrast-induced nephropathy. Am J Cardiol. 2006; 98: 27-36.
8. Marenzi G, Metrio MD, Rubino M, Lauri G, Cavallero A, Assanelli E, et al. Acute hyperglycemia and contrast-induced nephropathy in primary percutaneous coronary intervention. Am Heart J. 2010; 160: 1170-1177.
9. Stolker JM, MuCullough PA, Rao S, Inzucchi SE, Spertus JA, Maddox TM, et al. Pre-procedural glucose levels and the risk for contrast-induced acute kidney injury in patients undergoing coronary angiography. J Am Coll Cardiol. 2010; 55: 1433-1440.
10. Naruse H, Ishii J, Hashimoto T, Kawai T, Hattori K, Okumura M, et al. Pre-procedural glucose levels and the risk for contrast-induced acute kidney injury in patients undergoing emergency coronary intervention. Circ J. 2012; 76: 1848-1855.
11. Ceriello A. Cardiovascular effects of acute hyperglycemia: pathophysiological underpinnings. Diabetes Vasc Dis Res. 2008; 5: 260-268.
12. Persson PB, Tepel M. Contrast medium-induced nephropathy: the pathophysiology. Kidney Int.2006; 69: 8-10.
13. Goldenberg I, Matetzky S. Nephropathy induced by contrast media: pathogenesis, risk factors and preventive strategies. CMAJ. 2005; 172: 1461-1471.
14. American Diabetes Association. Standards of medical care in diabetes-2010. Diabetes Care. 2010; 33: 11-61.
15. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine:

A new prediction equation. Modification of diet in renal disease study group. Ann Intern Med. 1999; 130: 461-470.

16. National Kidney Foundation (NKF) Kidney Disease Outcome Quality Initiative (K/DOQI) advisory board. K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification, and stratification. Am J Kidney Dis. 2002; 39: 1-266.

17. Kidney Disease; Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl. 2012; 2: 1-138.

18. Blackman DJ, Pinto R, Ross JR, Seidelin PH, Ing D, Jackevicius C, et al. Impact of renal insufficiency on outcome after contemporary percutaneous coronary intervention. Am Heart J. 2006; 151: 146-152.

19. Lindsay J, Apple S, Pinnow EE, Gevorkian N, Gruberg L, Satler LF, et al. Percutaneous coronary intervention-associated nephropathy foreshadows increased risk of late adverse events in patients with normal baseline serum creatinine. Catheter Cardiovasc Interv. 2003; 59: 338-343.

20. McCullough PA, Adam A, Becker CR, Davidson C, Lameire N, Stacul F, et al. Epidemiology and prognostic implications of contrast induced nephropathy. Am J Cardiol. 2006; 98: 5-13.

21. Ali T, Khan I, Simpson W, Prescott G, Townend J, Smith W, et al. Incidence and outcomes in acute kidney injury: a comprehensive population-based study. J Am Soc Nephrol. 2007; 18: 1292-1298.

22. Marenzi G, De Metrio M, Rubino M, Lauri G, Cavallero A, Assanelli E, et al. Acute hyperglycemia and contrast-induced nephropathy in primary percutaneous coronary intervention. Am Heart J. 2010; 160: 1170-1177.

23. Moriyama N, Ishihara M, Noguchi T, Nakanishi M, Arakawa T, Asaumi Y, et al. Admission hyperglycemia is an independent predictor of acute kidney injury in patients with acute myocardial infarction. Circ J. 2014; 78: 1475-1480.

24. Ishihara M, Inoue I, Kawagoe T, Shimatani Y, Kurisu S, Nishioka K, et al. Impact of acute hyperglycemia on left ventricular function after reperfusion therapy in patients with a first anterior wall acute myocardial infarction. Am Heart J. 2003; 146: 674-678.

25. Ishihara M, Kojima S, Sakamoto T, Asada Y, Tei C, Kimura K, et al. Japanese Acute Coronary Syndrome Study Investigators. Acute hyperglycemia is associated with adverse outcome after acute myocardial infarction in the coronary intervention era. Am Heart J. 2005; 150: 814-820.

26. Timmer JR, Ottervanger JP, de Boer MJ, Dambrink JH, Hoorntje JC, Gosselink AT, et al. Hyperglycemia is an important predictor of impaired coronary flow before reperfusion therapy in ST-segment elevation myocardial infarction. J Am Coll Cardiol. 2005; 45: 999-1002.

27. Nakamura T, Ako J, Kadowaki T, Funayama H, Sugawara Y, Kubo N, et al. Impact of acute hyperglycemia during primary stent implantation in patients with ST-elevation myocardial infarction. J Cardiol. 2009; 53: 272-277.

28. Khamaisi M, Raz I, Shilo V, Shina A, Rosenberger C, Dahan R, et al. "Diabetes and radiocontrast media increase endothelin converting enzyme-1 in the kidney", Kidney International. 2008; 74: 91-100.

29. Pflueger A, Larson TS, Nath KA, King BF, Gross JM, Knox FG. "Role of adenosine in contrast media-induced acute renal failure in diabetes mellitus", Mayo Clinic Proceedings. 2002; 75: 1275-1283.

30. Pakfetrat M, Nikoo MH, Malekmakan L, Tabande M, Roozbeh J, Ganbar Ali RJ, et al. "Comparison of risk factors for contrast-induced acute kidney injury between patients with and without diabetes", Hemodial Int. 2010; 14: 387-392.

31. Ding FH, Lu L, Zhang RY, Zhu TQ, Pu LJ, Zhang Q, et al. Impact of elevated serum glycated albumin levels on contrast-induced acute kidney injury in diabetic patients with moderate to severe renal insufficiency undergoing coronary angiography. Int J Cardiol. 2013; 167: 369-373.

32. Yoshikawa D, Isobe S, Sato K, Ohashi T, Fujiwara Y, Ohyama H, et al. Importance of oral fluid intake after coronary computed tomography angiography: an observational study. Eur J Radiol. 2011; 77: 118-122.

33. Akyuz S, Kemaloglu Oz T, Altay S, Karaca M, Yaylak B, Gungor B, et al. Association between Glycosylated Haemoglobin Level and Contrast-Induced Acute Kidney Injury in Patients with Type-II Diabetes Mellitus. Cardiorenal Med. 2014; 2: 95-102.

34. Kanwar YS, Sun L, Xie P, Liu F, Chen S. A glimpse of various pathogenetic mechanisms of diabetic nephropathy. Annu Rev Pathol. 2011; 6: 395-423.

35. Newhouse JH, Kho D, Rao QA, Starren J. Frequency of serum creatinine changes in the absence of iodinated contrast material: implications for studies of contrast nephrotoxicity. Am J Roentgenol. 2008; 191: 376-382.

Citation: Abdallah E, Mosbah O, Sherif N, El-Shiegh N, Ali A and Abdullah A. Impact of Long-Standing Poor Glycemic Control on the Occurrence of Contrast-Induced Acute Kidney Injury in Patients with Type-II Diabetes Mellitus Undergoing Percutaneous Coronary Intervention. SM J Nephrol Therap. 2017; 2(1): 1005. https://dx.doi.org/10.36876/smjnt.1005