

# Contrast induced nephropathy in patients with primary percutaneous coronary intervention with or without metabolic syndrome

*Nefropatía inducida por contraste en pacientes con intervención coronaria percutánea primaria con o sin síndrome metabólico*

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## Key words:

Acute coronary syndrome, metabolic syndrome, percutaneous coronary intervention, contrast-induced nephropathy.

## Palabras clave:

Síndrome coronario agudo, síndrome metabólico, intervencionismo coronario percutáneo, nefropatía inducida por contraste.

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## ABSTRACT

Contrast induced nephropathy (CIN) is defined as the absolute increment of serum creatinine  $\geq 0.5$  mg/dL or an increment more than 25% of basal creatinine, without any other identified cause, within 48 hours after contrast media administration. **Objective:** Determine the CIN risk in patients with Acute Coronary Syndrome (ACS) with or without metabolic syndrome (MetS) treated with primary percutaneous coronary intervention (PCI). **Material and methods:** A prospective, observational, longitudinal and comparative study, in patients with ACS admitted to the Coronary Care Unit or Intensive Care Unit. PCI was performed with a serum creatinine (sCr) of  $\leq 1.2$  mg/dL prior intervention. Serum creatinine determinations were conducted 24-48 hours post PCI. The statistical test for analysis of free distribution quantitative variables was performed with Mann Whitney U test, and for qualitative variables Chi square test ( $\chi^2$ ). Likelihood-ratio and confidence interval of 95% with  $p = 0.05$ . **Results:** 420 patients with infarction code were studied, 323 men (76.9%), 97 women (23.1%), with ages between 56-70 years. They were divided into 2 groups: group A 176 (41.9%) with MetS and group B 244 (58%) without MetS. CIN was present in 43 patients (10.2%) group A and in 29 (6.9%) group B. RR: 2.05, CI 95% 1.33-3.15,  $p = 0.0012$ . **Conclusions:** MetS is a risk factor (RF) for the development of CIN in patients with ACS who undergo PCI. Therefore, this syndrome should be kept in mind for an early detection and prevention of the development of CIN.

## RESUMEN

La nefropatía inducida por contraste (NIC) se define como el incremento absoluto de creatinina sérica  $\geq 0.5$  mg/dL o un incremento del 25% de la creatinina basal, sin otra causa identificada, en un periodo de 48 horas posterior a la exposición al medio de contraste. **Objetivo:** Determinar el riesgo de NIC en pacientes con síndrome coronario agudo (SCA) con y sin síndrome metabólico (SM) tratados con intervencionismo coronario percutáneo (ICP). **Material y métodos:** Estudio prospectivo, observacional, longitudinal, comparativo, en pacientes con SCA admitidos a la Unidad de Cuidados Coronarios o a la Unidad de Cuidados Intensivos. La ICP fue realizada con creatinina sérica (CrS) previa  $\leq 1.2$  mg/dL. Las determinaciones de creatinina sérica se efectuaron 24-48 horas post ICP. Para el análisis de las variables cuantitativas se utilizó la prueba de U de Mann-Whitney y para variables cualitativas, prueba de Chi cuadrada ( $\chi^2$ ) con nivel de significancia e intervalos de confianza del 95% con  $p = 0.05$ . **Resultados:** 420 pacientes de código infarto fueron estudiados, 323 hombres (76.9%), 97 mujeres (23.1%) con edades de 56 a 70 años. Se dividieron en dos grupos: grupo A 176 (41.9%) con SM, grupo B, 244 (58%) sin SM. Se presentó NIC en 43 pacientes (10.2%) del grupo A y en 29 (6.9%) del grupo B. RR: 2.05, IC 95% 1.33-3.15,  $p = 0.0012$ . **Conclusiones:** El SM es un factor de riesgo (FR) para desarrollar NIC en pacientes con SCA sometidos a ICP. Por lo tanto, debe tenerse en cuenta para la detección temprana y prevención de NIC.

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## INTRODUCTION

Chronic degenerative diseases such as type 2 diabetes mellitus (DM2), systemic arterial hypertension and dyslipidemia, have been on the rise in the present century and are considered as cardiovascular risk factors (CVRF) as well as tobacco consumption.<sup>1-3</sup> The metabolic syndrome (MetS) is defined as the association of different metabolic risk factors that predispose to the development of cardiovascular disease and type 2 diabetes mellitus. The National Education and Cholesterol Program (NCEP ATP III) defines it as the presence of abdominal obesity, glucose intolerance, dyslipidemia, and hypertension.<sup>4,5</sup>

The acute coronary syndrome (ACS) accounts for approximately 80% of cases of sudden death in adults and in most of them represents the first and last clinical manifestation.<sup>6,7</sup> Contrast induced nephropathy (CIN) is defined as the absolute increment of serum creatinine (sCr)  $\geq 0.5$  mg/dL or an increment more than 25% of basal creatinine, without any other identified cause over a period of 48 hours after exposure to contrast material.<sup>8-10</sup>

CIN is the third cause of hospital renal lesion, only next to hypoperfusion and drug toxicity. A prior renal damage is the most important risk factor according to the risk scale of Mehran, placing the patients in a high-risk group.<sup>11-13</sup> The reported incidence of CIN ranges from 1-2% in patients without renal failure with preexisting renal disease and in the presence of non-modifiable risk factors (RF), it increased up to 25%. Risk factors are divided into 2 categories: I. Non-modifiable factors: age  $> 75$  years, type 2 DM, systemic arterial hypertension, renal insufficiency (sCr  $> 1.5$  mg/dL or creatinine clearance  $< 60$  mL/min), heart failure, hepatic cirrhosis, renal transplant, multiple myeloma, acute myocardial infarction and nephrotic syndrome.

II. Modifiable factors: dehydration, hypotension, non-steroid anti-inflammatories, use of diuretics, use of  $> 140$  mL of contrast medium, repeated application of contrast material in periods shorter than 24 hours, hypoalbuminemia, use of nephrotoxic antibiotics and anemia.<sup>14-18</sup>

There are few studies referable to the impact of MetS on the development of CIN, post elective percutaneous coronary intervention (PCI) in non-diabetic patients, where CIN occurred with higher frequency in patients with MetS compared with those without it.<sup>19-23</sup>

Few studies have been performed in patients with infarction code with and without MetS, associated with CIN. MetS is alarmingly increasing in our population, so the aim of our study was to identify whether patients with ACS plus MetS have a higher risk of developing CIN than those without MetS.<sup>21,24,25</sup>

## MATERIAL AND METHODS

A prospective, observational, longitudinal, and comparative study was conducted during the period May 1, 2017 to January 30, 2018 at the Hospital de Especialidades «Antonio Fraga Mouret» del Centro Medico Nacional «La Raza» (HECMN), Instituto Mexicano del Seguro Social. The project was authorized and registered by the local health research committee with number 3501, R-2017-3501-49.

The study was performed in patients with ACS admitted to the Coronary Care Unit (CCU) or the Intensive Care Unit (ICU), and after PCI for their continuous monitoring, given the high risk of electrical or mechanical complications that may arise from the ACS itself or from PCI. The contrast medium (CM) employed in the Cath Lab was Omnipaque, a hydro soluble, non-ionic CM (350 mg of iodine/mL). The employed dose was calculated based on the formula: 5 mL of contrast per kilogram body weight/sCr (mg/dL) (maximum dose of 300 mL).<sup>26</sup> MetS was diagnosed based on the NCEP ATP III criteria (abdominal obesity, glucose intolerance, dyslipidemia, and hypertension). The CIN was defined as the absolute increase in serum creatinine  $\geq 0.5$  mg/dL or an increase more than 25% of basal creatinine without any other identified cause within 48 hours after exposure to the CM.<sup>7,15,27,28</sup>

Patients were divided into two groups according to their characteristics: group A, patients with MetS and group B, without MetS. Only were selected, patients with acute coronary syndrome, with sCr  $\leq 1.2$  mg/dL and

with glomerular filtration rate (GFR)  $\geq 60$  mL/min prior to PCI. These measurements were estimated by the formula of Cockcroft-Gault (140-age) (kg weight)/sCr mg/dL (72) or (0.85 in women).<sup>11</sup> Later, during their stay in ICU or CCU, sCr was measured at 24 and 48 hours.

Statistical analysis: a descriptive analysis of each of the variables of the two groups expressed in frequencies, percentages and relative risk (RR) was done. Quantitative free distribution variables were summarized in median and measurement of dispersion was conducted with interquartile ranges, (IR) 25 and 75. The statistical test for analysis of free distribution quantitative variables was performed with Mann Whitney U test. For categoric variables, a bivariate analysis was performed with Chi-square ( $\chi^2$ ) test. Variables showing to be significant were further analyzed using logistic backward stepwise regression with likelihood-ratio and a confidence interval of 95%  $p = 0.05$ . All statistical analyses were performed using IBM's software SPSS v 20.  $p$  values  $\leq 0.05$  were significant.

## RESULTS

Four hundred twenty patients with infarction code were studied: 323 men (76.9%) and 97 women (23.1%), with a mean age of 62 years for men and 64 for women, interquartile range

(IR) 56-70 years. Successful PCIs were 357 (85%) and failed PCIs 63 (15%). The population was divided into two groups: group A with MetS 176 (41.9%) and group B without MetS 244 (58.1%). CIN was more frequent in men, 53 (12.6%) compared with women, 19 (4.5%). However, the stratified analysis did not reveal statistically significant differences between the two genders for the occurrence of CIN: RR:0.853, CI:95% 0.532-1.36, ( $p = 0.513$ ) (Table 1).

The presence of CIN was found in 72 patients (17.1%), of whom 43 (10.2%) were from group A and 29 (6.9%) from group B, RR: 2.05, CI: 95% 1.33-3.15, ( $p = 0.0012$ ). The average elevation in sCr at 48 hours in the group A was by 1.88 mg/dL. CI: 95% 1.82-1.91, ( $p = 0.001$ ). We observed that patients with type 2 DM without MetS 94 (22.4%) showed a RR: 1.96, CI:95% 1.14-3.36, ( $p = 0.013$ ). The comorbidities found in group A were in order of frequency as follows: systemic arterial hypertension 157 (37.3%), visceral obesity 142 (33.8%), dyslipidemia 102 (24.2%) and glucose intolerance 34 (8%) which are all components of MetS. Eighty percent of patients presented three components of MetS and 20% had the four components. The prevalence of type 2 DM with MetS was 136 patients (32.4%) (Table 2).

The electrocardiographic localization of ACS was: inferior 158 (37.6%), anteroseptal 109 (26%), and extensive anterior 66 (15.7%). ACS of inferior and anteroseptal localization showed a RR for CIN of 1.11, CI: 95% 0.72-1.71 ( $p = 0.608$ ) and RR: 1.11, CI: 95% 0.69-1.80, ( $p = 0.650$ ) respectively. Of these patients, 253 (60.2%) received thrombolysis, and 167 (39%) did not, with an average door-to-needle time of 2 hours with CI: 95% 1.92-2.01, ( $p = 0.011$ ). Angiographically, the results were as follows: multivessel disease 134 (31.9%), bivascular 130 (31%), anterior descendent 79 (18.8%). RR: 1.52, IC:95% 1.0-2.3, ( $p = 0.05$ ) (Table 3 and Figure 1).

The average time since arrival from a referral hospital to the hemodynamics room was 10 hours IR: 8-12 hours. Patients admitted with ACS in Killip Kimball class I (KK I): were 393 (43.6%), KK II: 17 (4%), KK III: 10 (2.4%). Of these, CIN occurred en KK I: 66 (16%), KK II: 4 (5.5%), KK III: 2 (2.7%),  $p = 0.824$ . The duration of the procedure was 45 average-

Table 1: Analysis of logistic regression of independent risk factors for contrast induced nephropathy with primary percutaneous coronary intervention.

Risk factor	RR	CI	p
Male gender	1.93	0.69-2.21	0.4660
Smoking	1.12	0.66-1.92	0.6520
Type 2 Diabetes mellitus	1.96	1.14-3.36	*0.0130
Metabolic Syndrome	2.05	1.33-3.15	*0.0012
ACS inferior localization	1.11	0.72-1.71	0.6000
ACS anteroseptal	1.11	0.69-1.80	0.6500
Bivascular disease	1.33	0.87-2.05	0.1860
Multivessel disease	1.52	1.0-2.3	*0.0500

ACS = Acute coronary syndrome, RR = Relative risk, CI = Confidence interval 95%.

\*  $p < 0.05$ .

minutes, IR: 40-60 min. The amount of CM (Omnipaque, a hydro soluble, non-ionic, contrast material) employed had a median

of 120 mL, IR: 100-153 mL ( $p = 0.868$ ). No association was observed between the amount of CM used and the development of CIN.

**Table 2: Characteristics of patients with acute coronary syndrome who developed or not contrast induced nephropathy.**

n = 420	Group A n = 176 n (%)	Group B n = 244 n (%)	p
Sex	M = 124 (29.5) W = 52 (12.4)	M = 199 (47.4) W = 45 (10.7)	0.0800
Mean age, IR.	62 (60-64)	64 (62-66)	*0.0470
Smoking	137 (32.6)	193 (46)	0.7570
Sedentary lifestyle	165 (39.3)	231 (55)	0.6880
Systemic arterial hypertension	157 (37.3)	59 (14.0)	*0.0010
Visceral obesity	142 (33.8)	34 (8.0)	*0.0010
Dyslipidemia	102 (24.2)	74 (17.6)	*0.0010
Glucose intolerance	34 (8.0)	142 (34.4)	*0.0440
Previous infarction	34 (8.0)	25 (6.0)	*0.0080
Type 2 DM	136 (32.4)	94 (22.4)	*0.0001
CIN	43 (10.2)	29 (6.9)	*0.0012
sCr: 48 hours mg/dL (CI)	1.88 (1.82-1.91)	1.02 (0.87-1.2)	0.0010*
Contrast media mL. IR	120 (100-153)	120 (100-153)	0.8680

DM2 = Type 2 diabetes mellitus, CIN = Contrast induced nephropathy, IR = Interquartile range, mL = Milliliters, sCr = Serum creatinine, CI = Confidence interval 95%.  
\* $p < 0.05$ .

**Table 3: Clinical, electrocardiographic and angiographic data, patients with and without contrast induced nephropathy.**

	CIN n = 72 n (%)	Without CIN n = 348 n (%)	RR	CI	p
Sex	M: 53 (73.6) W: 19 (26.4)	M: 270 (77.6) W: 78 (22.4)	1.000 1.930	0.63-1.61 0.69-2.21	0.9680 0.4660
Type 2 DM	49 (68.0)	181 (52.0)	1.966	1.14-3.36	*0.0130
Metabolic syndrome	43 (59.7)	133 (38.2)	2.050	1.33-3.15	*0.0012
Without metabolic syndrome	29 (40.2)	215 (61.7)	0.486	0.31-0.74	0.0800
ACS inferior	29 (40.3)	129 (37.1)	1.110	0.72-1.71	0.6080
ACS A/S	20 (27.8)	89 (25.6)	1.110	0.69-1.80	0.6500
# Affected vessels					
1	15 (20.8)	141 (40.5)	0.440	0.26-0.75	*0.0010
2	27 (37.5)	103 (29.6)	1.330	0.87-2.05	0.1860
3	30 (41.7)	104 (29.9)	1.520	1.0-2.3	*0.0500

CIN = Contrast induced nephropathy, DM2 = Type 2 diabetes mellitus, ACS = Acute coronary syndrome, A/S = Anteroseptal, RR = Relative risk, CI = Confidence interval 95%.

\* $p < 0.05$

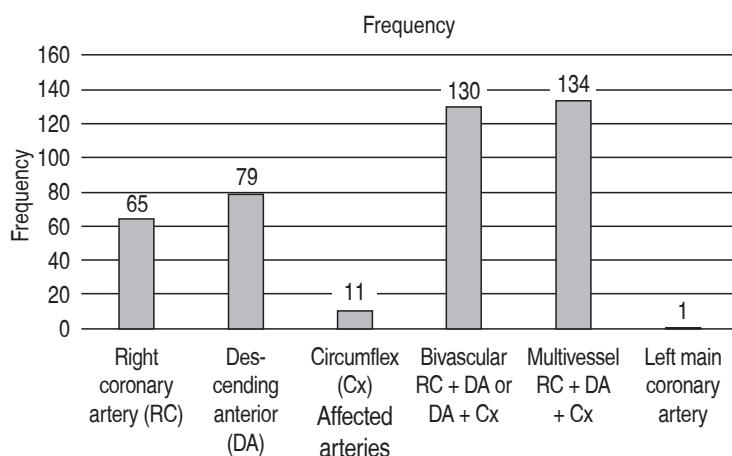


Figure 1: Percutaneous coronary intervention angiographic findings.

## DISCUSSION

In this study it was found that patients with ACS plus MetS, who underwent PCI presented twice as much the risk for CIN as those without MetS. Likewise, it was found that patients with bivascular and multivessel disease, showed a higher risk for CIN compared with the remaining angiographic findings.

CIN is a frequent complication in patients who undergo PCI. In the present study, patients with prior renal damage, which is an unmistakable RF for CIN were not included.<sup>14,25,29</sup> The already known RF inherent to the patient like type 2 DM were similar to those reported in the literature.<sup>3,10</sup> Furthermore, there are several studies in the literature of patients with previous renal damage or an elective population for the performance of a diagnostic PCI, where they found that prior renal damage was a RF for the development of CIN as established by the study of Mehran.<sup>8</sup> Another study reported that MetS is a RF for CIN in a cohort of 219 patients studied during three years, where only patients over 60 years of age and with mild to moderate impairment of renal function were included.<sup>3,8,16</sup> However, in our study we included patients with a mean age of 63 years, with ACS that did not present alteration of the renal function prior to PCI and in this way, it was decreased the bias caused by the previous alteration of renal function and thus showing the association between MetS and the development of CIN.<sup>12,16</sup>

The risk age for CIN in our population was 10 years less than that reported in the literature and no statistically significant difference between genders was found for the presence of CIN. This contrast with other reports in that there is a higher risk in a population over 70 years of age with a greater prevalence in men.<sup>9,10,16</sup>

As expected, combined hyperlipidemia, simple hypercholesterolemia, MetS, and low HDL-c have been associated with multivessel coronary artery disease independent of other CVD risk factors.<sup>25,26,30</sup> According to our results we also found that in multivessel coronary artery disease was associated to MetS and CIN.

ACS with inferior and anteroapical localization proved to be a RF, although without statistically significant difference. Of the total of patients admitted with ACS, the greatest percentage was seen in KK I prior to PCI, and only 2.4% was in stage III which improved after the performance of PCI. No association was observed between the KK class III and the development of CIN. This could be due to the fact that there were only 10 patients in KK III.

CIN is a diagnosis that connotes a great responsibility to the clinician, thus it is of utmost importance to know that patients with metabolic syndrome have a risk for CIN. This knowledge will allow us to employ the necessary therapeutic strategies in order to protect the patient and decrease the incidence of CIN.

Therefore, we suggest the following: 1. Every patient should have a sCr determination prior to exposure to a contrast medium. 2. The patient should be adequately hydrated prior to PCI. 3. Use an isoosmolar CM and 4. Do not use more than 120 mL of CM. One last suggestion is to monitor sCr and urinary output every 24 hours.

In case of detecting CIN, the following are proposed: optimize patient's hydration; prepare management for acute renal failure and optimize hemodynamic stability in order to keep satisfactory tissue organ perfusion, as well as to bear in mind that MetS is a RF for the occurrence of CIN.

Among the limitations of our study, it is important to mention that we did not have a follow up of the patients after discharge from the hospital where they spent three days.



Therefore, we consider it necessary to conduct a study following these patients during the whole hospitalization period and to know whether CIN remitted or progressed to renal function replacement therapy.

## CONCLUSIONS

MetS should be considered a risk factor for the development of CIN in patients with ACS who undergo PCI. Consequently, this fact must be kept in mind in the pre and post PCI management to optimize treatment and decrease CIN progression.

## Ethical aspects

The study was conducted based on the fundamental bioethical aspects of autonomy, beneficence and justice. In accordance with the norms of the declaration of Helsinki and the world medical assembly from 1964 to 2008, bearing in mind the CIOMS guidelines, the Belmont report and the General Health Law of Mexico with regard to article 17. Section I.

**Conflict of interests:** The authors declare that they have not conflict of interests.

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## REFERENCES

- Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012; 380 (9859): 2095-2128.
- Ferreira G. Epidemiología de la enfermedad coronaria. *Rev Esp Cardiol*. 2014; 67 (2): 139-144.
- Bouzas-Mosquera A, Vázquez-Rodríguez JM, Calviño-Santos R, Peteiro-Vázquez J, Flores-Ríos X, Marzoa-Rivas R y cols. Nefropatía inducida por contraste y fracaso renal agudo tras cateterismo cardiaco urgente: incidencia, factores de riesgo y pronóstico. *Rev Esp Cardiol*. 2007; 60 (10): 1026-1034.
- Oda E. Metabolic syndrome: its history, mechanisms, and limitations. *Acta Diabetol*. 2012; 49: 89-95.
- Toprak O, Cirit M, Yesil M, Byrne DW, Postaci N, Bayata S et al. Metabolic syndrome as a risk factor for contrast-induced nephropathy in non-diabetic elderly patients with renal impairment. *Kidney Blood Press Res*. 2006; 29 (1): 2-9.
- Robson J, Boomla K, Hart B. Estimating cardiovascular risk for primary prevention: outstanding questions for primary care. *BMJ*. 2000; 320: 702-704.
- Van de Werf F, Bax J, Betriu A, Blomstrom-Lundqvist C, Crea F, Falk V et al. Management of acute myocardial infarction in patients presenting with persistent ST segment elevation: The Task Force on the Management of ST segment elevation acute myocardial infarction of the European Society of Cardiology. *Eur Heart J*. 2008; 29 (23): 209-245.
- Antonio L, Bartorelli A, Marenzi G. Contrast induced nephropathy. *J Interv Cardiol*. 2008; 21: 74-85.
- Feldkamp T, Luedemann M, Spehlmann ME, Freitag-Wolf S, Gaensbacher J, Schulte K et al. Radial access protects from contrast media induced nephropathy after cardiac catheterization procedures. *Clin Res Cardiol*. 2017; 107 (2): 148-157.
- Mehran R, Nikolsky E. Contrast-induced nephropathy definition, epidemiology, and patients at risk. *Kidney Int Suppl*. 2006; 100: S11-15.
- Valdez DR, Wong ER, Flores YE, Basoni RAE, Esquivel MCG, González OLA y cols. Nefropatía por medio de contraste en angiografía cardiaca. *Med Int Mex*. 2010; 26 (3): 226-236.
- Arana E, Catalá F. Nefropatía inducida por contraste en pacientes de riesgo con insuficiencia renal explorados con tomografía computarizada: revisión sistemática y meta análisis de ensayos clínicos aleatorizados. *Med Clin Barc*. 2010; 135:343-350.
- Barbieri L, Verdoia M, Schaffer A, Cassetti E, Di Giovine G, Marino P et al. Pre-diabetes and the risk of contrast induced nephropathy in patients undergoing coronary angiography or percutaneous intervention. *Diabetes Res Clin Pract*. 2014; 106 (3): 458-464.
- Aspelin P, Aubry P, Fransson SC, Strasser R, Willenbrock R, Berg KJ et al. Nephrotoxic effects in high-risk patients undergoing angiography. *N Engl J Med*. 2003; 348 (6): 491-499.
- Golshahi J, Nasri H, Gharipour M. Contrast-induced nephropathy; A literature review. *J Nephropathol*. 2014; 3 (2): 51-56.
- Amiri A, Ghanavati R, Riahi Beni H, Sezavar SH, Sheykhvatan M, Arab M. Metabolic syndrome and the iodine-dose/creatinine clearance ratio as determinants of contrast-induced acute kidney injury. *Cardiorenal Med*. 2018; 8 (3): 217-227.
- Ronco C, House A, Haapio M. Cardiorenal and Reno cardiac syndromes: the need for a comprehensive classification and consensus. *Nat Clin Pract Nephrol*. 2008; 4: 310-311.
- Macías NJ, Robles PN, Gil GP, López AJ. Valoración de la función renal en el anciano. *JANO*. 2008; 1710: 43-47.
- Li Y, Zhao L, Yu D, Wang Z. Metabolic syndrome prevalence and its risk factors among adults in China: A nationally representative cross-sectional study. *PLoS One*. 2018; 19: 13-17.

20. Oda E. Historical perspectives of the metabolic syndrome. *Clinics in Dermatology*. 2017; 36 (1): 3-8.
21. Ozcan OU, Adanir Er H, Gulec S, Ustun EE, Gerede DM, Goksuluk H et al. Impact of metabolic syndrome on development of contrast-induced nephropathy after elective percutaneous coronary intervention among nondiabetic patients. *Clin Cardiol*. 2015; 38 (3): 150-156.
22. Patti G, Colonna G, Pasceri V, Pepe LL, Montinaro A, Di Sciascio G. Randomized trial of high loading dose of clopidogrel for reduction of periprocedural myocardial infarction in patients undergoing coronary intervention. results from the ARMYDA-2 (antiplatelet therapy for reduction of myocardial damage during angioplasty) Study. *Circulation*. 2005; 111 (16): 2099-2016.
23. Olshahi J, Nasri H. Gharipour M. Contrast-induced nephropathy: a literature review. *J Nephropathic*. 2014; 3 (2): 51-56.
24. Chong E, Shen L, Tan HC. Risk scoring system for prediction of contrast induced nephropathy in patients with pre-existing renal impairment undergoing percutaneous coronary intervention Singapore. *Med J*. 2012; 53 (3): 164-169.
25. Yuan Y, Qiu H, Hu XY, Luo T, Gao XJ, Zhao XY et al. Risk factors of contrast induced acute kidney injury in patients undergoing emergency percutaneous coronary intervention. *Chin Med J (Engl)*. 2017; 130 (1): 45-50.
26. Cho JY, Jeong MH, Hwan Park S, Kim IS, Park KH, Sim DS et al. Effect of contrast-induced nephropathy on cardiac outcomes after use of nonionic isosmolar contrast media during coronary procedure. *J Cardiol*. 2010; 56 (3): 300-306.
27. National Cholesterol Education Program (NCEP) Expert panel o Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III), Third Report of the National Cholesterol Education program (NECP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation* 2002; 106 (25):3143-421.
28. Grundy SM, Cleeman CJ, Daniels CS, Donato KA, Eckel RH, Franklin BA et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung and Blood Institute Scientific Statement. *Circulation*. 2005; 112 (17): 2735-2752.
29. Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol*. 2004; 44 (7): 1393-1399.
30. Abd Alamir M, Goyfman M, Chaus A, Dabbous F, Tamura L, Sandfort V et al. The correlation of dyslipidemia with the extent of coronary artery disease in the multiethnic study of atherosclerosis. *J Lipids*. 2018; 2018: 5607349.

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