Original Article

HEALTH SCIENCES
MEDICINE

DOI: 10.32322/jhsm.1654419
J Health Sci Med. 2025;8(3):476-482

Metabolic syndrome as a risk factor in contrast-induced acute kidney injury*

Belma Özlem Tural Balsak[1], Şehmus Özmen[2], Davut Akın[3], Mehmet Emin Yılmaz[4]

[1]Department of Endocrinology, Ankara Bilkent City Hospital, Ankara, Turkiye
[2]Division of Nephrology, Department of Internal Medicine, İstanbul Medipol University, Çamlıca Hospital, İstanbul, Turkiye
[3]Division of Nephrology, Department of Internal Medicine, Faculty of Medicine, Pamukkale University, Denizli, Turkiye
[4]Division of Nephrology, Department of Internal Medicine, Faculty of Medicine, Dicle University, Diyarbakır, Turkiye

Cite this article as: Tural Balsak BÖ, Özmen Ş, Akın D, Yılmaz ME. Metabolic syndrome as a risk factor in contrast-induced acute kidney injury. *J Health Sci Med.* 2025;8(3):476-482.

Received: 23.03.2025 ◆ **Accepted**: 11.05.2025 ◆ **Published**: 30.05.2025

ABSTRACT

Aims: The expanding use of contrast media has made contrast-induced acute kidney injury (CI-AKI) a cause of acute renal failure. This study investigated the relationship between metabolic syndrome (MS), insulin resistance, and contrast-induced acute kidney injury.

Methods: This study encompassed 94 hospitalized patients (73 male) with creatinine levels of 1.0 and above who underwent contrast-enhanced computed tomography for various reasons in the internal medicine clinics of Dicle University Faculty of Medicine. Patients whose creatinine levels were routinely measured before and 48 hours after tomography and whose anamnesis information was sufficient for the International Diabetes Federation metabolic syndrome criteria were retrospectively included in the study. HOMA-IR values were calculated.

Results: CI-AKI developed in 10 (10.6%) patients out of 94. MS was identified in 60% of the group that developed CI-AKI and 38.1% that did not. Insulin resistance was observed in 30% of the group that developed CI-AKI and 17.9% that did not. Despite the observed difference, it did not achieve statistical significance. Considerable differences were found between the two groups regarding albumin, urea, creatinine, uric acid, C-reactive protein, and hemoglobin levels. CI-AKI was significantly more common in individuals with low creatinine clearance and in those with creatinine ≥1.3 mg/dl.

Conclusion: Our study showed that a higher rate of patients with MS was detected in the group with CI-AKI. MS has been accused of being a risk factor for CI-AKI. Therefore, caution should be exercised when administering contrast to patients with MS.

Keywords: Contrast-induced acute kidney injury, insulin resistance, metabolic syndrome

*Our research's data was presented as an Oral Presentation abstract in Clinical Kidney Journal, Oxford Academy, 2009;2(Supp 2):ii178.

INTRODUCTION

Contrast-induced acute kidney injury (CI-AKI) was considered the third most common cause of acute renal failure (ARF), following hypotension and postoperative complications, according to hospital settings.[1] Nonetheless, modern findings suggest that the reported prevalence rates were biased and inflated.[2]

In 2020, the American College of Radiology and the National Kidney Foundation Consensus revealed that the prevalence of CI-AKI is significantly lower than previously reported. The guideline concluded that most cases of CI-AKI following intravenous contrast administration are not directly caused by the contrast medium itself, but rather by coincident nephrotoxic exposures.[2,3] In light of this, the term CA-AKI was recommended over CI-AKI to describe instances of AKI occurring after contrast exposure when the exact cause remains uncertain.[3]

Acute kidney injury (AKI) continued to be defined based on the established kidney disease; improving global outcomes (KDIGO) criteria, which include: an increase in serum creatinine by ≥0.3 mg/dl within 48 hours, a ≥50% rise in serum creatinine within 48 hours, a ≥1.5-fold increase within 7 days, or urine output less than 0.5 ml/kg/hour for at least 6 hours.[4]

The 2018 guidelines published by the ESUR Contrast Medium Safety Committee recommended 'post-contrast acute kidney injury' (PC-AKI) as the preferred term for renal function deterioration following contrast medium administration. It was emphasized that CI-AKI can have multiple potential causes. Key patient-related risk factors for PC-AKI include chronic kidney disease (CKD) and dehydration.[5]

Across two meta-analyses of 19,000 patients who received IV contrast medium, the incidence of PC-AKI was reported as

Corresponding Author: Belma Özlem Tural Balsak, belmabalsak@gmail.com

6.4% (95% CI, 5.0-8.1) and 5.0% (95% CI, 3.8-6.5).[5,6] Although 1% of patients experienced sustained renal dysfunction over two months, the need for renal replacement therapy (RRT) was rare, with a weighted incidence of just 0.06%.[6]

Preexisting renal failure, diabetes mellitus, advanced age, use of nephrotoxic drugs, use of high amounts of contrast media, or use of ionic, hyperosmolar contrast media, congestive heart failure, anemia, and dehydration are the most important risk factors for the development of CI-AKI. Metabolic syndrome (MS) is one of the suggested risk factors for CI-AKI.[7]

Metabolic syndrome is a metabolic disease that is formed by the accumulation of a group of diseases that lead to cardiovascular diseases, the main culprit being insulin resistance.[8] Abdominal obesity, impaired glucose tolerance associated with insulin resistance and hyperinsulinemia, dyslipidemia, and hypertension are the main components of this syndrome.[9]

The exact mechanism of CI-AKI is not known in detail. Many mechanisms have been proposed for etiological factors. Iodine contrast can induce cytotoxicity in nephrons, tubular epithelial cells, and endothelial cells, leading to mitochondrial dysfunction, apoptosis, pyroptosis, necrosis, and interstitial inflammation. It may also alter renal hemodynamics, resulting in intramedullary ischemia and hypoxia.[10] In addition to renal medullary hypoxia and direct tubular toxicity caused by contrast, reactive oxygen radicals, oxidative stress, renin-angiotensin-aldosterone system activation, and increased endothelin I, which are among the etiological factors of MS, are also among these proposed mechanisms.[11-14] This study aims to examine the relationship between MS, which is considered a possible new risk factor for CI-AKI, and insulin resistance and CI-AKI.

METHODS

Ethics

Since this study was conducted before 2020, there was no ethics committee requirement at that time, and the study was retrospective, so ethics committee approval was not obtained. Prior the study, institutional approval was obtained. All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Design and Participants

This is a single-center retrospective study. Patients hospitalized in Internal Medicine Clinics at Dicle University who were going to have contrast tomography for any reason and whose creatinine values were 1.0 mg/dl and above were enrolled in this study.

Patients with serum creatinine values <1.0 mg/dl, diabetic patients receiving insulin treatment, patients undergoing hemodialysis, those unable to calculate fasting insulin, and critically ill patients requiring intensive care were excluded from the study.

Procedure

Before the tomography, serum creatinine, urea, lipid profile (cholesterol, HDL, LDL, triglyceride), albumin, uric acid, CRP, fasting insulin, fasting glucose, and hemoglobin were obtained. Serum creatinine levels were checked 48 hours after tomography. The patients' age, gender, body weight, height, waist circumference, and body-mass index (BMI) were noted.

Risk factors were evaluated by investigation of diabetes, hypertension, coronary artery disease, hyperlipidemia, stroke, heart failure, and smoking history.

The drugs used by the patients, especially in the last 48 hours before tomography [Angiotensin-converting-enzyme inhibitors (ACEI), Angiotensin receptor blockers (ARB's), acetylsalicylic acid (ASA), N-acetylcysteine, statin, diuretic, non-steroidal anti-inflammatory drugs (NSAID)], were recorded from the patient files.

Using the 2005 International Diabetes Federation (IDF) criteria, patients were categorized into those with and without MS. HOMA-IR values were calculated to measure insulin resistance (HOMA-IR=glucose (mg/dl)xinsulin (μU/dl)/405). HOMA-IR values above 2.5 suggest insulin resistance.

The patients' creatinine clearance was determined with the modification of diet in renal disease (MDRD) formula [GFR=186x(Scr) 1.154x(age) 0.203x(0.742 if female)].[7]

CKD is defined as abnormalities in kidney structure or function that persist for at least three months and have implications for health. These abnormalities may include a reduced glomerular filtration rate (eGFR & lt; 60 ml/min/1.73 m^2) or evidence of kidney damage, such as albuminuria, structural changes seen on imaging, or abnormalities in urine or blood tests. To make a diagnosis of CKD, one or more of the following criteria must be present for ≥3 months:[4]

- eGFR less than 60 ml/min/1.73 m^2
- Albumin-to-creatinine ratio (ACR) ≥30 mg/g
- Urinary abnormalities (e.g., hematuria, casts)
- Structural abnormalities of the kidney (e.g., seen on ultrasound)
- History of kidney transplantation

CKD is classified into five stages based on eGFR, as shown below:

- **Stage G1:** eGFR ≥90 ml/min/1.73 m^2 (normal or high kidney function, but with evidence of kidney damage)
- **Stage G2:** eGFR 60-89 ml/min/1.73 m^2 (mild decrease in function, with evidence of kidney damage)
- **Stage G3a:** eGFR 45-59 ml/min/1.73 m^2 (mild to moderate decrease in kidney function)
- **Stage G3b:** eGFR 30-44 ml/min/1.73 m^2 (moderate to severe decrease in kidney function)
- **Stage G4:** eGFR 15-29 ml/min/1.73 m^2 (severe decrease in kidney function)
- **Stage G5:** eGFR & lt;15 ml/min/1.73 m^2 (kidney failure, also known as end-stage renal disease-ESRD)

Echocardiography recorded the left ventricular ejection fraction (EF). The patients' blood pressure values for systole and diastole during the tomography scan were measured and recorded, and their hydration status was determined.

All patients were routinely given 100 ml of low-osmolar nonionic monomer iohexol (Omnipaque vial, 300 mg-100 ml vial, Opakim Medical Products Limited Company, İstanbul) intravenously during the tomography scan.

Acute kidney injury (AKI) is defined based on the established kidney disease; improving global outcomes (KDIGO) criteria.[4]

Statistical Analysis

Data analysis was performed using the SPSS 16.0 software after the study. Continuous variables were presented as mean±standard deviation, while frequencies were reported as percentages (%). The student T test was employed to compare group means, and the Chi-square test was used to assess differences in frequency distributions. For comparing means with non-homogeneous distributions, the Mann-Whitney U test was applied. The analysis of receiver operating characteristic (ROC) curves determined the cut-off value, sensitivity, and specificity. A p-value of <0.05 was considered statistically significant.

RESULTS

73 male (77.7%) and 21 female (22.3%) patients were enrolled in the study. MS was detected in 39 patients (41.4%). 17 of these patients were female (43.6%) and 22 were male (56.4%). CI-AKI developed in 10 patients out of 94 patients. Of the 10 patients who developed nephropathy, 9 were male and 1 was female (M/F 90/10%). This difference observed between genders did not reach the level of statistical significance (p=0.322). Patients were divided into two groups; those who developed CI-AKI and those who did not.

MS was 60% in the group that developed CI-AKI and 38.1% in the group that did not (p=0.182). Insulin resistance was detected in 30% of the group that developed nephropathy and 17.9% in the group that did not (p=0.356). The mean baseline creatinine values of patients with MS were 1.29, and the mean baseline creatinine values of patients without MS were 1.26. No significant difference was found in baseline creatinine levels between those with and without MS.

The mean age was 69.7±8.9 in the CI-AKI (+) group and 60±17.4 in the CI-AKI (-) group (p>0.05). Advanced age is considered a risk factor for CI-AKI, but in our study, no statistically significant difference was found between the groups that developed nephropathy and those that did not.

A significant difference was found between the groups that developed nephropathy and those that did not (p=0.022) in terms of baseline creatinine clearance levels (As shown in **Figure 1**). The creatinine clearances calculated with MDRD were lower in the group that developed nephropathy. Those with creatinine ≥1.3 were 60% in the group that developed nephropathy and 22.6% in the group that did not (p=0.011). The renal dysfunction is related to the nephropathy that develops after contrast application (**Figure 1, 2**).

A considerable difference was found between the two groups in the evaluated albumin, urea, creatinine, uric acid, CRP, and hemoglobin values of the patients (**Table**).

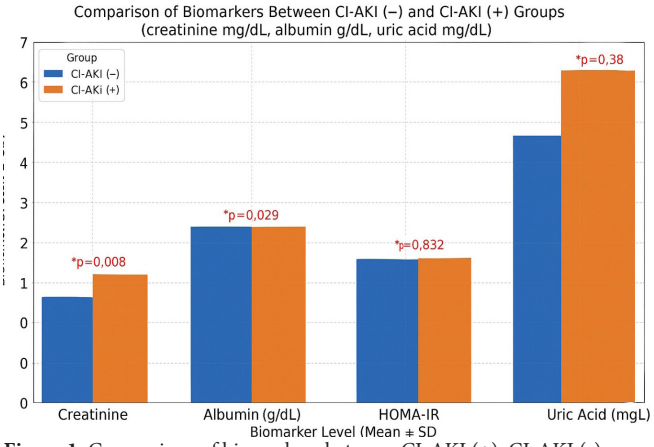

Figure 1. Comparison of biomarkers between CI-AKI (+), CI-AKI (-) groups (creatinine mg/dl, albumin g/dl, uric acid mg/dl)

CI-AKI: Contrast-induced acute kidney injury

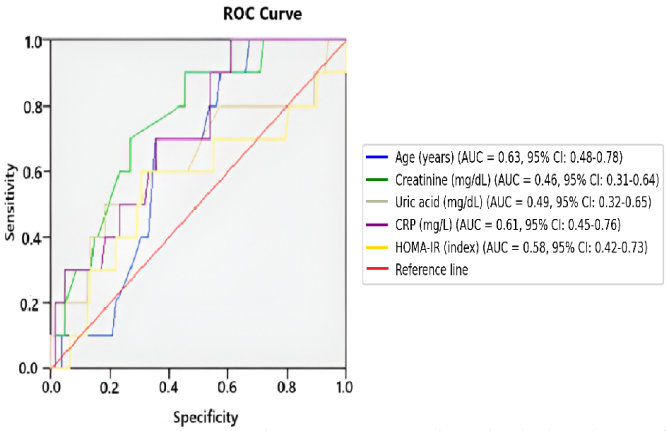

Figure 2. Receiver operating characteristic curves for individual predictors of contrast-induced acute kidney injury, including age (years), creatinine (mg/dl), uric acid (mg/dl), C-reactive protein (mg/L), and HOMA-IR (index)

HOMA-IR: Homeostatic model assessment of insulin resistance

There was no significant difference between the two groups in the evaluated parameters of fasting blood sugar, insulin, cholesterol, triglyceride, LDL, HDL, HOMA-IR, systolic blood pressure, diastolic blood pressure, waist circumference, weight, and BMI.

In our study, the number of diabetic patients was 5 (6%). None of the diabetic patients developed contrast nephropathy.

Congestive heart failure (CHF) is one of the previously determined risk factors for CI-AKI. CHF was detected in 30% of the group that developed CI-AKI and 8.3% of the group that did not. This difference was statistically significant (p=0.036).

No significant difference was observed between the two groups in the evaluation of hypertension, smoking, coronary artery disease, and stroke history of the patients (As shown in **Table**).

Among the evaluated patients, ASA use was 30% in the group that developed nephropathy and 13.1% in the group that did not develop nephropathy; Statin use was 10%, 6%, ACEI use was 40%, 25%, and Diuretic use was 40%, 21%, correspondingly. No significant difference was detected between all these groups.

Table. Demographic and laboratory characteristics of patients with and without contrast-induced acute kidney injury

Parameter	CI-AKI (+) (n=10)	CI-AKI (–) (n=84)	p-value
Age (years)	69.7±8.9	60.0±17.4	0.116
Systolic blood pressure (mmHg)	119±18.5	118±15.0	0.935
Diastolic blood pressure (mmHg)	76±12.6	75±12.0	0.839
Waist circumference (cm)	96±13	94±13	0.634
Weight (kg)	62±11.7	71±15.0	0.063
Body-mass index (kg/m^2)	22.4±3.4	25.0±5.5	0.064
Urea (mg/dl)	84±34	49±25	<0.001*
Creatinine (mg/dl)	1.66±1.08	1.20±0.30	0.008*
Albumin (g/dl)	2.4±0.6	3.0±0.7	0.029*
Fasting blood glucose (mg/dl)	88.6±22.0	89.0±23.0	0.921
Insulin (µIU/ml)	10.3±11.9	7.16±8.6	0.291
Hemoglobin (g/dl)	10.4±1.7	12.1±2.3	0.021*
Total cholesterol (mg/dl)	139±37	156±40	0.230
Triglyceride (mg/dl)	138±48	137±75	0.994
HDL-C (mg/dl)	31±15	32±13	0.788
LDL-C (mg/dl)	95±55	95±31	0.991
Uric acid (mg/dl)	7.13±3.3	5.58±2.0	0.038*
CRP (mg/L)	102±96	49±65	0.025*
eGFR (MDRD, ml/min/1.73 m^2)	52±16.8	65±17.2	0.022*
HOMA-IR (index)	1.8±1.5	1.6±2.2	0.832
Congestive heart failure (%)	30%	8.3%	0.036*
Metabolic syndrome (%)	60%	38.1%	0.182
Sex (male/female, %)	90%/10%	76%/23%	0.322
Diabetes mellitus (%)	0%	6%	0.428
Stroke (%)	0%	1.2%	0.729
Hypertension (%)	40%	28.6%	0.455
Smoking (%)	60%	47%	0.437
Coronary artery disease (%)	30%	11.9%	0.117
Aspirin use (%)	30%	13.1%	0.156
Statin use (%)	10%	6%	0.621
ACEI use (%)	40%	25%	0.310
Diuretic use (%)	40%	21%	0.190
Hydration status (%)	40%	66.7%	0.097
Insulin resistance (%)	30%	17.9%	0.356
Chronic kidney disease (%)	60%	34.5%	0.115
Creatinine ≥1.3 mg/dl (%)	60%	22.6%	0.011
Hypoalbuminemia (%)	90%	66.7%	0.131

CI-AKI: Contrast-induced acute kidney injury, HDL-C: High-density lipoprotein cholesterol, LDL-C: Low-density lipoprotein cholesterol, CRP: C-reactive protein, MDRD: Modification of diet in renal disease formula, HOMA-IR: Homeostatic model assessment of insulin resistance, ACEI: Angiotensin-converting enzyme inhibitor, eGFR: Estimated glomerular filtration rate

DISCUSSION

With the advancement of imaging techniques, CI-AKI has emerged as a prevalent issue in clinical practice in recent years.[1] CI-AKI prolongs hospital stays, contributing to higher morbidity and mortality rates and escalating treatment costs. As a result, it is crucial to identify the risk factors for CI-AKI early and implement the currently recommended preventive measures.

The risk of developing CI-AKI can be predicted by preexisting renal failure, diabetes, hypovolemia, anemia, advanced age, CHF, excessive use of contrast medium, use of non-ionic hyperosmolar contrast medium, and concurrent use of other nephrotoxic agents. MS has been evaluated as a risk factor for the development of CI-AKI. MS is a group of diseases that are based on insulin resistance, abdominal obesity, diabetes or impaired glucose tolerance, hypertension, high triglyceride levels, and decreased HDL, leading to increased cardiovascular risk.[8,9]

In this study, patients with plasma creatinine values >1.0 mg/dl and those who underwent tomography with contrast media for any reason were evaluated. Patients were grouped using IDF criteria in terms of MS, and HOMA-IR was calculated to determine insulin resistance. Patients ' creatinine levels were assessed before contrast administration and 48 hours following imaging, and creatinine clearance was calculated based on the MDRD formula.

Various mechanisms contribute to the pathophysiology of CI-AKI. These are renal medullary hypoxia and direct tubular toxicity. These mechanisms are also further increased by increased RAS activity, reactive oxygen species, oxidative stress, and increased endothelin 1 activity, which are etiological factors of MS.[15]

Renin and angiotensin 2 are potential mediators leading to intrarenal vasoconstriction in experimental CI-AKI studies. Abdominal obesity additionally increases the amount of RAS components.[16] Increased endothelin 1 and decreased nitric oxide (NO) result in vasoconstriction by decreased renal medullary blood flow.[17,18] Decreased NO activity and increased endothelin I are distinct features of MS. However, HT, dyslipidemia, glucose intolerance, and hyperuricemia are components of MS, which increase the risk of developing CI-AKI. Toprak and colleagues identified metabolic syndrome as a risk factor for the development of CI-AKI in their study. In this research, patients who underwent coronary angiography were categorized into two groups; MS and non-MS. It was observed that CI-AKI development significantly differed between the two groups, with a higher incidence in the MS group. MS increased the risk of nephropathy by 4.26, impaired glucose tolerance by 4.72, high triglyceride by 4.06, and multiple vessel involvement by 3.14 times. These were evaluated as predictors of CI-AKI in MS patients.[7]

In our study, the prevalence of MS was 60% in the group that developed CI-AKI and 38.1% in the group that did not develop CI-AKI. Despite the higher MS rate, this difference did not achieve statistical significance (p=0.182). The lack of significance could be attributed to the relatively small sample size. Out of the 10 patients who developed CI-AKI, 6 (60%) had MS.

We calculated HOMA-IR values to evaluate insulin resistance. Insulin resistance was found to be 30% in the group that developed CI-AKI and 17.9% in the group that did not develop CI-AKI (p=0.356). Although a higher rate of insulin resistance was observed in the group that developed CI-AKI, statistical significance was not observed. This may be due to the small number of patients.

Preexisting renal failure is considered one of the most significant risk factors for CI-AKI. A GFR <60 ml/min is a key risk factor for the development of CI-AKI.[19] In a study by Davidson et al.[20] involving 1144 patients who underwent percutaneous coronary angiography (PCAG), preexisting renal failure was identified as the primary risk factor for triggering CI-AKI. Another study found that CI-AKI developed in 2% of 378 patients undergoing PCAG, but in 30% of those with a baseline creatinine level greater than 1.5 mg/dl.[21] Additionally, in a cohort of 2034 patients, a pre-procedure serum creatinine level of 2 mg/dl or higher was found to be the strongest risk factor for CI-AKI.[22] In our study, a significant correlation was found between creatine clearance and CI-AKI. Again, CI-AKI development reached a significant level in the group with a creatinine level of 1.3 and above. In our study, the development of CI-AKI was found to be more frequent in patients with poor hydration status, with a trend toward statistical significance (p: 0.097). If contrast agent use is necessary in patients with impaired renal function, optimal hydration should be provided, and agents with the lowest dose and low osmolarity should be used.

DM plays a role in the development of CI-AKI. In a study conducted by Lautin et al.,[23] the incidence of CI-AKI was determined as 2% in patients without diabetes and azotemia. The incidence was determined as 16% in diabetics with preserved renal function and 38% in diabetics with impaired renal function. Although the risk of CI-AKI is lower in diabetic patients with preserved renal function and without proteinuria or microalbuminuria, those with impaired renal function constitute the highest risk group for CI-AKI due to the synergistic effect. The number of diabetic patients in this study was limited to 5 patients, and CI-AKI was not detected in any of them. This number was limited because most of the diabetic patients admitted to our hospital were on insulin treatment, and we excluded patients taking insulin from the study.

HT is stated as a minor risk factor for the development of CI-AKI in some articles.[17,18,24] In one study, HT was reported as an independent risk factor for CI-AKI in 8628 patients who underwent percutaneous catheterization. HT is also a component of MS Toprak et al.[7] found no correlation between HT and CI-AKI in their study. No significant difference was found in our study either.

The relationship between hypercholesterolemia and CI-AKI has been investigated in the literature.[25,26] According to these studies, hypercholesterolemia causes the development of CI-AKI by reducing NO synthesis. In our study, we could not find a relationship between hypercholesterolemia and CI-AKI. Toprak et al.[7] found a strong relationship between high triglycerides and CI-AKI in their study. In our study, no significance was found between triglyceride, HDL, LDL levels, and CI-AKI.

EF is a measure used to indicate left ventricular function. It is 67±9% in normal individuals. In patients with stage IV heart failure, especially if the EF value is below 50%, the cardiac performance of the patients is impaired as well as their renal perfusion, and when these patients are exposed to contrast media, the development of CI-AKI is higher than in normal individuals.[27-29] In our study, CHF was found to be higher in the group that developed CI-AKI (p=0.036).

Hyperuricemia is among the minor risk factors for the development of CI-AK.[30] It is also a minor component of metabolic syndrome. High uric acid level plays a role in the pathogenesis of CI-AKI by activating RAS, reactive oxygen species, and inhibiting NO production.[31,32] In a cohort study of 1,440 patients, serum uric acid levels of ≥8.0 mg/dl were found to be significantly associated with an elevated risk of contrast-induced acute kidney injury (CI-AKI).[33]

In our study, a statistically significant difference was found between high uric acid and CI-AKI, as shown in Figure 1 (p=0.038).

Although the relationship between age and CI-AKI is still debated, the 2019 ACR guidelines advise renal function assessment in individuals over 60 years prior to ICM administration. By contrast, the 2018 ESUR guidelines[10] did not recognize age itself as a risk factor for CI-AKI. The apparent association may rather reflect the increased prevalence of renal dysfunction and other age-related comorbidities.[34]

In our study, the mean age in the group that developed CI-AKI was 69.7±8.9, and the mean age in the group that did not develop CI-AKI was 60±17.4. There was no significant relationship between age and contrast nephropathy (p=0.116).

Limitations

The study had several limitations. It had all the limitations of a retrospective design and a single-center study. Additionally, a small sample size likely contributes to non-significant results, such as the relation between MS and insulin resistance, which did not reach significance. Moreover, we acknowledge the increased risk of type I errors due to multiple comparisons. Diabetic patients who were hospitalized and receiving insulin therapy were excluded. Therefore, a limited number of diabetic patients treated with oral antidiabetic agents could be included. Additionally, since hemodialysis may alter insulin levels, patients with end-stage CKD undergoing hemodialysis were also excluded from the study. Critical patients who were full and required to follow up in intensive care conditions were excluded from the study. These exclusions might lead to selection bias and impact our results. Despite these limitations, this pilot study provides preliminary evidence of a relationship between CI-AKI, MS, and insulin resistance.

CONCLUSION

Consequently, although CI-AKI is rare in the general population, it can be serious in high-risk patients. Being aware of risk factors helps identify these patients before the procedure. Given the absence of a specific treatment for CI-AKI, prevention through early risk recognition remains essential. Metabolic syndrome has recently been blamed for the development of CI-AKI. Similar pathophysiological events are present in both metabolic syndrome and CI-AKI development, supporting the idea that an interaction between the two events may be involved. In our study, metabolic syndrome was present in 6 out of 10 patients who developed CI-AKI (60%). However, due to the small sample

size, no statistically significant result could be reached. In conclusion, our study showed that a higher rate of patients with MS was detected in the group with CI-AKI. However, the results provide preliminary evidence of a non-significant trend linking MS and insulin resistance to CI-AKI, alongside reaffirming established risk factors, including renal impairment and congestive heart failure. Therefore, caution should be exercised when administering contrast to patients with MS. Our results have to be confirmed by larger-scale, prospective longitudinal studies.

ETHICAL DECLARATIONS

Ethics Committee Approval

Since this study was conducted before 2020, ethics committee approval was not obtained.

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Detrenis S, Meschi M, Musini S, Savazzi G. Lights and shadows on the pathogenesis of contrast-induced nephropathy: state of the art. *Nephrol Dial Transplant*. 2005;20(8):1542-1550.
2. Davenport MS, Cohan RH, Khalatbari S, Ellis JH. The challenges in assessing contrast-induced nephropathy: where are we now? *AJR Am J Roentgenol*. 2014;202:784-789. doi:10.2214/AJR.13.11369
3. Davenport MS, Perazella MA, Yee J, et al. Use of intravenous iodinated contrast media in patients with kidney disease: consensus statements from the American College of Radiology and the National Kidney Foundation. *Radiology*. 2020;294:660-668. doi: 10.1148/radiol.2019192094
4. Khwaja A. KDIGO clinical practice guidelines for acute kidney injury. *Nephron Clin Pract*. 2012;120(4):c179-c184. doi:10.1159/000339789
5. van der Molen AJ, Reimer P, Dekkers IA, et al. Post-contrast acute kidney injury-part 1: Definition, clinical features, incidence, role of contrast medium and risk factors: recommendations for updated ESUR Contrast Medium Safety Committee guidelines. *Eur Radiol*. 2018;28(7):2845-2855. doi:10.1007/s00330-017-5246-5
6. Moos SI, van Vemde DN, Stoker J, Bipat S. Contrast induced nephropathy in patients undergoing intravenous (IV) contrast enhanced computed tomography (CECT) and the relationship with risk factors: a meta-analysis. *Eur J Radiol*. 2013;82(9):e387-e399. doi:10.1016/j.ejrad.2013.04.029
7. Toprak O, Cirit M, Yesil M, 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. doi:10.1159/000092481
8. Lebovitz HE. Insulin resistance: definition and consequences. *Exp Clin Endocrinol Diabetes*. 2001;109(Suppl 2):S135-S148. doi:10.1055/ s-2001-18576
9. Lebovitz HE. Oral therapies for diabetic hyperglycemia. *Endocrinol Metab Clin North Am*. 2001;30(4):909-933. doi:10.1016/s0889-8529(05)70221-8
10. Li Y, Wang J. Contrast-induced acute kidney injury: a review of definition, pathogenesis, risk factors, prevention and treatment. *BMC Nephrol*. 2024;25(1):140. doi:10.1186/s12882-024-03570-6
11. Gussenhoven MJ, Ravensbergen J, van Bockel JH, Feuth JD, Aarts JC. Renal dysfunction after angiography; a risk factor analysis in patients with peripheral vascular disease. *J Cardiovasc Surg (Torino)*. 1991;32(1):81-86.
12. Kidney Disease Outcome Quality Initiative. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. *Am J Kidney Dis*. 2002;39:17-31.
13. Maddox TG. Adverse reactions to contrast material: recognition, prevention, and treatment. *Am Fam Physician*. 2002;66(7):1229-1234.
14. Scharnweber T, Alhilali L, Fakhran S. Contrast-induced acute kidney injury: pathophysiology, manifestations, prevention, and management. *Magn Reson Imaging Clin N Am*. 2017;25(4):743-753. doi:10.1016/j.mric.2017.06.012
15. Kusirisin P, Chattipakorn SC, Chattipakorn N. Contrast-induced nephropathy and oxidative stress: mechanistic insights for better interventional approaches. *J Transl Med*. 2020;18(1):400. doi:10.1186/s12967-020-02574-8
16. Hansel B, Giral P, Nobecourt E, et al. Metabolic syndrome is associated with elevated oxidative stress and dysfunctional dense high-density lipoprotein particles displaying impaired antioxidative activity. *J Clin Endocrinol Metab*. 2004;89(10):4963-4971. doi:10.1210/jc.2004-0305
17. Gleeson TG, Bulugahapitiya S. Contrast-induced nephropathy. *AJR Am J Roentgenol*. 2004;183(6):1673-1689. doi:10.2214/ajr.183.6.01831673
18. Itoh Y, Yano T, Sendo T, Oishi R. Clinical and experimental evidence for prevention of acute renal failure induced by radiographic contrast media. *J Pharmacol Sci*. 2005;97(4):473-488. doi:10.1254/jphs.crj05002x
19. Toprak O, Cirit M. Risk factors and therapy strategies for contrast-induced nephropathy. *Ren Fail*. 2006;28(5):365-381. doi:10.1080/08860220600683524
20. Davidson CJ, Laskey WK, Hermiller JB, et al. Randomized trial of contrast media utilization in high-risk PTCA: the COURT trial. *Circulation*. 2000;101(18):2172-2177. doi:10.1161/01.cir.101.18.2172
21. Gruberg L, Mintz GS, Mehran R, et al. The prognostic implications of further renal function deterioration within 48 h of interventional coronary procedures in patients with pre-existent chronic renal insufficiency. *J Am Coll Cardiol*. 2000;36(5):1542-1548. doi:10.1016/s0735-1097(00)00917-7
22. Taliercio CP, Vlietstra RE, Ilstrup DM, et al. A randomized comparison of the nephrotoxicity of iopamidol and diatrizoate in high risk patients undergoing cardiac angiography. *J Am Coll Cardiol*. 1991;17(2):384-390. doi:10.1016/s0735-1097(10)80103-2
23. Lautin EM, Freeman NJ, Schoenfeld AH, et al. Radiocontrast-associated renal dysfunction: incidence and risk factors. *AJR Am J Roentgenol*. 1991;157(1):49-58. doi:10.2214/ajr.157.1.2048539
24. McCullough PA, Soman SS. Contrast-induced nephropathy. *Crit Care Clin*. 2005;21(2):261-280. doi:10.1016/j.ccc.2004.12.003
25. Andrade L, Campos SB, Seguro AC. Hypercholesterolemia aggravates radiocontrast nephrotoxicity: protective role of L-arginine. *Kidney Int*. 1998;53(6):1736-1742. doi:10.1046/j.1523-1755.1998.00906.x
26. Yang DW, Jia RH, Yang DP, Ding GH, Huang CX. Dietary hypercholesterolemia aggravates contrast media-induced nephropathy. *Chin Med J (Engl)*. 2004;117(4):542-546.
27. Manske CL, Sprafka JM, Strony JT, Wang Y. Contrast nephropathy in azotemic diabetic patients undergoing coronary angiography. *Am J Med*. 1990;89(5):615-620. doi:10.1016/0002-9343(90)90180-l
28. Schillinger M, Haumer M, Mlekusch W, Schlerka G, Ahmadi R, Minar E. Predicting renal failure after balloon angioplasty in high-risk patients. *J Endovasc Ther*. 2001;8(6):609-614. doi:10.1177/152660280100800614
29. Gruberg L, Mintz GS, Mehran R, et al. The prognostic implications of further renal function deterioration within 48 h of interventional coronary procedures in patients with pre-existent chronic renal insufficiency. *J Am Coll Cardiol*. 2000;36(5):1542-1548. doi:10.1016/s0735-1097(00)00917-7

30. Mudge GH. Uricosuric action of cholecystographic agents. A possible factor in nephrotoxicity. *N Engl J Med*. 1971;284(17):929-933. doi:10.1056/NEJM197104292841701
31. Ishizaka N, Ishizaka Y, Toda E, Nagai R, Yamakado M. Association between serum uric acid, metabolic syndrome, and carotid atherosclerosis in Japanese individuals. *Arterioscler Thromb Vasc Biol*. 2005;25(5):1038-1044. doi:10.1161/01.ATV.0000161274.87407.26
32. Perlstein TS, Gumieniak O, Hopkins PN, et al. Uric acid and the state of the intrarenal renin-angiotensin system in humans. *Kidney Int*. 2004;66(4):1465-1470. doi:10.1111/j.1523-1755.2004.00909.x
33. Wu MJ, Tsai SF, Lee CT, Wu CY. The predictive value of hyperuricemia on renal outcome after contrast-enhanced computerized tomography. *J Clin Med*. 2019;8(7):1003. doi:10.3390/jcm8071003
34. Kodzwa R. ACR manual on contrast media: 2018 updates. *Radiol Technol*. 2019;91(1):97-100.