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Incidence and Predictors of Contrast-Induced Nephropathy Following Intravenous Iodinated Contrast Media Administration.

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Abstract

Background: Contrast-induced nephropathy (CIN) is a recognised complication of intravascular iodinated contrast media (ICM) administration and contributes to increased morbidity. Reported incidence varies widely with baseline renal function and associated risk factors. This study aimed to determine the incidence of CIN and general adverse reactions following intravenous iodinated contrast administration, and to identify the clinical and demographic factors associated with their occurrence. ***Materials and Methods***: This hospital-based prospective observational study was conducted, in the Department of Radiodiagnosis of a tertiary care teaching hospital over a period of 12 months. Four hundred patients referred for intravenous contrast-enhanced computed tomography (CECT) or intravenous urography, with normal to near-normal baseline renal function (eGFR ≥45 mL/min, up to ≥30 mL/min in select circumstances) and no known contrast allergy, were enrolled by purposive sampling. Serum creatinine was measured at baseline, 24 hours and 48 hours after contrast injection. CIN was defined, per the American College of Radiology (ACR) and Kidney Disease Improving Global Outcomes (KDIGO) criteria, as an absolute rise in serum creatinine ≥0.3 mg/dL or a relative rise ≥50% from baseline. Adverse reactions were graded as mild, moderate or severe. Associations were tested using the chi-square test, with $p<0.05$ considered statistically significant. ***Results***: The overall incidence of CIN was 5.25% (21/400). A statistically significant female preponderance was observed (8.28% in females vs. 3.03% in males; $\chi^2=4.41$, $p=0.036$), as was a strong association with pre-existing hypothyroidism (29.6% vs. 3.68% in euthyroid patients; $\chi^2=45.87$, $p<0.001$). No significant association was found between CIN and age ($p=0.210$), diabetes mellitus ($p=0.142$), hypertension ($p=1.000$), cardiac disease ($p=1.000$), renal disease ($p=1.000$), concurrent drug use ($p=0.320$) or type of contrast study ($p=0.387$). General adverse reactions to contrast occurred in 3% (12/400) of patients — mild in 2.25% and moderate in 0.75%, with no severe reactions recorded — and were significantly associated with a prior history of allergy ($\chi^2=13.25$, $p<0.001$) and with hypothyroidism ($\chi^2=9.09$, $p=0.003$). ***Conclusion***: In patients with preserved baseline renal function, the incidence of CIN following intravenous iodinated contrast administration is low. Female sex and hypothyroidism emerged as significant independent associations warranting closer biochemical monitoring, whereas hypertension and diabetes alone did not increase risk when baseline renal function was normal. These findings support individualised, risk-based screening rather than blanket restriction of contrast use.

Keywords: Contrast-induced nephropathy; iodinated contrast media; acute kidney injury; hypothyroidism; adverse drug reaction; computed tomography.

INTRODUCTION

Contrast-induced nephropathy (CIN), also termed contrast-induced acute kidney injury, refers to an abrupt decline in renal function occurring after intravascular administration of iodinated contrast medium (ICM), and is considered a causative subset of the broader entity of contrast-associated acute kidney injury.[1] Its reported incidence ranges from 1–3% in patients with normal renal function to 9–38% in those with moderate renal dysfunction compounded by diabetes mellitus, heart

failure or volume depletion,2 while the overall incidence of contrast-induced acute kidney injury in the general population is estimated at 0.6–2.0%.3 CIN is associated with increased short- and long-term morbidity and mortality.4

The kidneys eliminate ICM almost entirely through renal tubular excretion; tubular reabsorption of these low-molecular-weight, poorly protein-bound molecules can concentrate them up to 100-fold within the renal parenchyma, promoting osmotic diuresis and a reduction in glomerular filtration rate that may culminate in acute tubular injury.3 CIN is reported to account for up to 11% of hospital-acquired renal insufficiency, making it the third most common cause of in-hospital acute kidney injury after impaired renal perfusion and nephrotoxic medication use.5 In addition to renal injury, iodinated contrast can provoke general adverse reactions, most commonly dermatological (pruritus and urticaria in roughly 70% of reactions), which are classified as immediate (within one hour) or delayed (up to seven days) and graded by severity.6 Preventive strategies for CIN include pre-procedural risk stratification, avoidance of unnecessary contrast in high-risk patients, adequate peri-procedural hydration, and, in select cases, N-acetylcysteine.7 With the steadily increasing use of iodinated contrast for both diagnostic and interventional purposes, it is important to characterise its incidence and the clinical determinants of both renal and non-renal adverse effects in routine diagnostic practice, so that at-risk patients can be identified and monitored proactively rather than being denied clinically indicated imaging.

This study was therefore undertaken to assess the incidence of general and organ-specific (renal) adverse reactions to intravenous iodinated contrast agents in patients undergoing routine contrast-enhanced radiological studies, and to evaluate the relationship of these reactions with age, sex, comorbidities, prior allergic history and procedure-related factors.

MATERIALS AND METHODS

Study design and setting: This was a hospital-based prospective observational study conducted in the Department of Radiodiagnosis, in the Department of Radiodiagnosis of a tertiary care teaching hospital over a period of 12 months, after approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants, and confidentiality of records was maintained throughout.

Study population and sampling: Patients referred to the department for intravenous urography and various intravenous contrast-enhanced CT studies were enrolled by purposive sampling. A total of 400 patients constituted the study sample.

Inclusion criteria: Patients referred for intravenous urography or intravenous contrast-enhanced CT studies, with normal serum creatinine/eGFR up to 45 mL/min (up to 30 mL/min in select circumstances), and no known allergy to iodinated contrast. Procedure-related complications such as contrast extravasation were included within the scope of the study.

Exclusion criteria: Significantly elevated serum creatinine (known chronic kidney disease with eGFR <30 mL/min), known allergy to iodinated contrast, pregnancy, and conditions confounding the interpretation of a rise in serum creatinine (e.g., severe hypovolaemia or surgery within 48 hours of contrast administration), so that isolated contrast-induced nephropathy could be assessed without overlap from contrast-associated causes.

Data collection and variables: A structured proforma, adapted from variables used in comparable published studies, was used to record age, sex, weight, comorbidities (cardiac disease, renal disease, diabetes mellitus, hypertension, hypothyroidism), prior allergic history, fasting (nil per os, NPO) duration, type of contrast study and drug history. All patients were observed for 30 minutes after contrast injection for immediate reactions, and serum creatinine was measured at baseline and at 24 and 48 hours after contrast administration to assess renal function.

Outcome definitions: CIN was defined as per the ACR Manual on Contrast Media (2024) and KDIGO criteria — an absolute rise in serum creatinine ≥0.3 mg/dL, or a relative rise ≥50% (1.5-fold) from baseline, within 48 hours of contrast administration.[1] The urine-output criterion of the KDIGO definition was not applied, as continuous urine-output monitoring was not feasible in this heterogeneous, largely ambulatory cohort. General adverse (allergic and physiological) reactions were classified as mild, moderate or severe according to standard ACR criteria, and managed per institutional protocol for acute contrast reactions.

Statistical analysis: Data were entered in Microsoft Excel 2024 and analysed using Microsoft Excel and Python. Categorical variables were expressed as frequencies and percentages, and associations between CIN/adverse reactions and clinical or demographic variables were tested using the chi-square test. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 400 patients referred for contrast-enhanced radiological studies were enrolled and followed for the development of post-contrast reactions and changes in renal function.

Demographic and baseline characteristics

The majority of patients were middle-aged: 160 (40%) were aged 31–50 years and 144 (36%) were aged 51–70 years, while the extremes of age (10–30 and 71–90 years) together accounted for 24% of the cohort. There was a mild male preponderance, with 231 patients (57.75%) male and 169 (42.25%) female. Most patients (59.5%) weighed between 51–75 kg. Ninety-five percent of patients had a fasting (NPO) duration of 6–12 hours prior to contrast injection, consistent with institutional practice (Table 1).

Table 1. Demographic and baseline characteristics of the study population (n=400)

Characteristic	n	%
Age group (years)		
10–30	65	16.25
31–50	160	40.00
51–70	144	36.00
71–90	31	7.75
Sex		
Male	231	57.75
Female	169	42.25
Weight (kg)		
25–50	38	9.50
51–75	238	59.50
76–100	121	30.25
101–125	3	0.75
Fasting (NPO) duration (hours)		
2–5	5	1.25
6–9	178	44.50
9–12	202	50.50
>12	15	3.75

Comorbidities and type of contrast study

Diabetes mellitus (25.5%) and hypertension (33%) were the most prevalent comorbidities; cardiac disease (4.25%), hypothyroidism (6.75%), pre-existing (non-CKD) renal disease (0.75%) and a documented history of allergy (1.75%) were less common. Two hundred and seven patients (45.4%) were not on any regular medication; among those on treatment, metformin (18.6%) and telmisartan (18.2%) were most frequently used. Contrast-enhanced CT of the abdomen was the commonest study performed (63%), followed by CT chest (9%) and intravenous urography (5.5%) (Table 2).

Table 2. Comorbidities and clinical characteristics of the study population (n=400)

Characteristic	n	%
Diabetes mellitus	102	25.50
Hypertension	132	33.00
Hypothyroidism	27	6.75
Cardiac disease	17	4.25
Pre-existing renal disease (non-CKD)*	3	0.75
History of allergy	7	1.75
Contrast extravasation	3	0.75
Type of study (leading categories)		
CECT abdomen	252	63.00
CECT chest	36	9.00
Intravenous urography (IVP)	22	5.50
CECT neck	21	5.25
CT cardiac angiogram	19	4.75
Other studies (9 categories)	50	12.50

*Isolated pyelonephritis, renal artery stenosis and pyonephrosis with eGFR 30–45 mL/min; patients with chronic kidney disease were excluded.

Adverse (allergic and physiological) reactions

Twelve of 400 patients (3%) experienced a general adverse reaction to contrast: 9 (2.25%) mild and 3 (0.75%) moderate, with no severe reactions. The commonest mild symptoms were chills (31.6% of all reactions), nausea (15.8%) and itching (10.5%); moderate reactions comprised facial oedema, diffuse skin oedema with rash, and shortness of breath (one case each). Symptoms were generally short-lived, resolving within a few hours in most affected patients. Contrast extravasation occurred in 3 patients (0.75%) (Table 3).

Table 3. Distribution of adverse (allergic and physiological) reactions to contrast media (n=400)

Reaction severity	n	%
Mild (chills, nausea, vomiting, itching, hypotension, fever, skin rash)	9	2.25
Moderate (facial oedema, diffuse skin oedema with rash, dyspnoea)	3	0.75
Severe	0	0.00
Total with reaction	**12**	**3.00**
No reaction	388	97.00

Incidence of contrast-induced nephropathy

By the absolute-rise criterion (≥0.3 mg/dL), CIN was detected in 4.25% of patients at 24 hours and 4.5% at 48 hours; three patients with a transient rise at 24 hours had normalised by 48 hours, while four new cases emerged between 24 and 48 hours, giving an overall absolute-criterion incidence of 5.25% (21/400). By the percentage-rise criterion (≥50%), CIN was detected in 3.0% at 24 hours and 3.8% at 48 hours (overall 3.75%, 15/400); all patients meeting the percentage criterion also met the absolute criterion. Combining both criteria, the overall incidence of CIN in this cohort was 5.25% (21/400) (Table 4).

Table 4. Incidence of contrast-induced nephropathy (CIN) by diagnostic criterion and time interval

Criterion / time point	n/N	%
Absolute rise ≥0.3 mg/dL – 24 hours	17/400	4.25
Absolute rise ≥0.3 mg/dL – 48 hours	18/400	4.50
Absolute rise ≥0.3 mg/dL – overall	**21/400**	**5.25**
Relative rise ≥50% – 24 hours	12/400	3.00
Relative rise ≥50% – 48 hours	12/400	3.00
Relative rise ≥50% – overall	**15/400**	**3.75**
Overall CIN (either criterion)	**21/400**	**5.25**

Factors associated with contrast-induced nephropathy

On chi-square testing, female sex (8.28% vs. 3.03% in males; χ^2=4.41, p=0.036) and hypothyroidism (29.6% vs. 3.68% in euthyroid patients; χ^2=45.87, p<0.001) were significantly associated with CIN. A borderline association was noted with a prior history of allergy (28.6% vs. 4.8%; χ^2=3.75, p=0.053). Age group, diabetes mellitus, hypertension, cardiac disease, pre-existing (non-CKD) renal disease, concurrent drug use and type of contrast study showed no statistically significant association with CIN (Table 5).

Table 5. Association between contrast-induced nephropathy and clinical/demographic variables

Variable	CIN n (%)	Non-CIN n (%)	χ^2	p-value
Sex – Female (n=169)	14 (8.28)	155 (91.72)	4.41	0.036*
Sex – Male (n=231)	7 (3.03)	224 (96.97)		
Age 10–30y (n=65)	2 (3.08)	63 (96.92)	11.75	0.210
Age 31–50y (n=160)	10 (6.25)	150 (93.75)		
Age 51–70y (n=144)	8 (5.56)	136 (94.44)		
Age 71–90y (n=31)	1 (3.23)	30 (96.77)		
Diabetes present (n=102)	2 (1.96)	100 (98.04)	2.16	0.142
Diabetes absent (n=298)	19 (6.38)	279 (93.62)		
Hypertension present (n=132)	7 (5.30)	125 (94.70)	0.00	1.000
Hypertension absent (n=268)	14 (5.22)	254 (94.78)		
Hypothyroidism present (n=27)	8 (29.6)	19 (70.4)	45.87	<0.001*
Hypothyroidism absent (n=373)	13 (3.48)	360 (96.52)		
Cardiac disease present (n=17)	1 (5.88)	16 (94.12)	0.00	1.000
Cardiac disease absent (n=383)	20 (5.22)	363 (94.78)		
Allergy history present (n=7)	2 (28.6)	5 (71.4)	3.75	0.053
Allergy history absent (n=393)	19 (4.83)	374 (95.17)		
Renal disease present (n=3)	0 (0.0)	3 (100.0)	0.00	1.000
Renal disease absent (n=397)	21 (5.29)	376 (94.71)		
Drug use (any, vs. none)	–	–	17.75	0.320
Type of contrast study	–	–	14.87	0.387

*Statistically significant, p<0.05.

Adverse (allergic and physiological) reactions to contrast were significantly associated with a prior history of allergy (3/7, 42.9% vs. 9/393, 2.3%; χ^2=13.25, p<0.001) and with hypothyroidism (0/27, 0.0% vs. 12/373, 3.2%; χ^2=9.09, p=0.003), but not with age (p=0.235), weight (p=0.264), sex (p=0.396), hypertension (p=0.125) or cardiac disease (p=0.150).

DISCUSSION

In this prospective study of 400 patients undergoing intravenous contrast-enhanced CT and intravenous urography, the overall incidence of CIN was 5.25%, occurring predominantly in patients with preserved baseline renal function. This is comparable to, though marginally higher than, the 2–5% incidence reported by McDonald et al.8 and Bruce et al.9 in patients without baseline renal dysfunction, and close to the 4.96% pooled incidence reported by Moos et al. in a meta-analysis of 42 studies and 18,790 patients undergoing intravenous contrast-enhanced CT.10 The modest variation across studies likely reflects differences in patient selection, contrast agent and volume, and the diagnostic threshold used for CIN.10

A notable observation was the dynamic pattern of creatinine change: three patients (0.75%) with a transient rise at 24 hours normalised by 48 hours, while four new cases (1%) emerged only at 48 hours. This underscores that a substantial proportion of CIN manifests within the first 24 hours, but continued surveillance to 48 hours — and, in higher-risk patients, potentially beyond — is required to avoid missing delayed-onset cases, consistent with the recommendation of Thomsen and Morcos to extend monitoring to 72 hours in high-risk populations.

The majority of participants were middle-aged (76% aged 31–70 years), mirroring national trends of higher diagnostic-imaging utilisation in this age group,11 and no significant association was found between age and CIN, likely reflecting the disproportionate representation of middle-aged patients in this cohort rather than a true absence of age-related risk. A mild male preponderance (57.75%) was observed, consistent with previous large-scale series of contrast-related outcomes,12 and the weight distribution, concentrated in the 51–75 kg range, paralleled findings from Asian cohorts reported by Katayama et al.13

Female sex was significantly associated with CIN (8.28% vs. 3.03% in males; p=0.036), consistent with reports by Iakovou et al., who found an increased risk of CIN in women after percutaneous coronary intervention.14 This may relate to sex-based differences in baseline renal reserve, body composition, or hormonal influences on renal haemodynamics, and reinforces sex as a factor worth incorporating into pre-procedural risk assessment.

Baseline renal function is widely regarded as the strongest predictor of CIN; Mehran et al. identified pre-existing renal dysfunction (eGFR <45 mL/min/1.73 m²) as the strongest independent predictor of CIN after percutaneous coronary intervention,15 and this is supported by StatPearls, which similarly emphasises chronic kidney disease as a major risk factor even though CIN can occur with normal renal function if other risk factors coexist.16 By design, this study excluded patients with significant pre-existing renal impairment to isolate the effect of contrast exposure itself; consequently, the observed incidence of 5.29% among patients without CKD reflects the baseline risk in a population with preserved renal reserve, consistent with the pre-procedural screening strategy advocated by Barrett and Parfrey to reduce CIN rates through early identification and proactive management of at-risk patients.17

Although diabetes mellitus was prevalent in this cohort (25.5%), its presence was not significantly associated with CIN (1.96% vs. 6.38% in non-diabetics; p=0.142), a finding consistent with McCullough et al., who noted that diabetes alone is a comparatively weak predictor of CIN unless accompanied by underlying renal dysfunction,18 and with Barrett and Parfrey, who found no markedly higher CIN rates in diabetics without pre-existing renal disease.17 Ad et al. similarly emphasised that the risk conferred by diabetes is amplified chiefly through coexisting renal impairment and altered medullary oxygenation rather than hyperglycaemia per se.19 Likewise, hypertension, present in a third of the cohort, showed no significant association with CIN (p=1.000), in keeping with reports that renal function and contrast volume, rather than hypertension itself, are the principal determinants of risk.20

The strongest comorbid association observed was with hypothyroidism, present in only 6.75% of patients but associated with a markedly higher CIN incidence (29.6% vs. 3.48% in euthyroid patients; p<0.001). This corroborates the findings of Gurdogan and Ari, who reported that hypothyroidism increases CIN risk even with normal baseline creatinine,21 and of Lin et al., who found a significantly higher incidence of CIN in hypothyroid patients undergoing percutaneous coronary intervention.22 Thyroid hormone deficiency is thought to reduce renal blood flow and glomerular filtration through decreased cardiac output and altered renal vascular resistance, potentially compounding the haemodynamic and tubulotoxic effects of iodinated contrast. Given the consistency of this association with prior literature and its magnitude in the present cohort, thyroid function may merit routine consideration in pre-contrast risk stratification, although larger studies are required to confirm hypothyroidism as an independent risk factor.

Concurrent drug use, including agents with potential nephrotoxic interactions such as diuretics and renin-angiotensin system blockers, was not significantly associated with CIN in this study (p=0.320), although the biological plausibility of such interactions is well established: nonsteroidal anti-inflammatory drugs impair renal prostaglandin-mediated autoregulation, diuretics can potentiate volume depletion, and angiotensin-converting enzyme inhibitors may blunt

compensatory renal haemodynamic responses in patients with pre-existing renal impairment.23 Azzalini et al. similarly reported that the combination of high-dose contrast with such agents increased CIN risk chiefly in patients with reduced renal reserve,24 and larger, drug-specific studies would be needed to clarify these interactions in an otherwise low-risk population such as this cohort.

CIN incidence varied numerically across the type of contrast study performed — highest with intravenous urography (18.2%) and CT chest (11.1%), and absent in several less-represented categories — but this difference was not statistically significant (p=0.387), and is likely explained by the disparate sample sizes across study types rather than a true modality-specific effect, since the mass of iodinated contrast delivered is broadly comparable across these examinations. This is consistent with the meta-analysis by Moos et al., which similarly found no significant difference in CIN incidence between different types of contrast-enhanced CT, while identifying renal insufficiency, allergy and hypothyroidism as the principal determinants of risk.10

General adverse reactions to contrast were infrequent (3%) and exclusively mild-to-moderate in severity, consistent with the 0.5–3% incidence reported by Chiu and Chu in a review of hypersensitivity reactions to iodinated contrast.25 These reactions were significantly linked to a prior history of allergy and to hypothyroidism, but not to age, sex, weight, hypertension or cardiac disease, suggesting that a targeted allergy and thyroid history, rather than routine screening for cardiovascular comorbidity, may be most useful in anticipating who is likely to react adversely to contrast administration.

Strengths and limitations

The principal strength of this study is its prospective design with paired serial creatinine measurements at 24 and 48 hours, allowing characterisation of the temporal pattern of CIN in a real-world diagnostic imaging population. Limitations include the single-centre design, reliance on purposive rather than random sampling, exclusion of patients with significant pre-existing renal impairment (limiting generalisability to higher-risk populations), the small absolute number of CIN events in some subgroups (e.g., allergy, cardiac disease) limiting statistical power, and the inability to monitor urine output as an additional KDIGO criterion. Larger, multi-centric studies incorporating high-risk subgroups and individual nephrotoxic drug classes are needed to refine risk-prediction models for CIN.

CONCLUSION

Among patients with preserved baseline renal function undergoing intravenous iodinated contrast studies, the overall incidence of contrast-induced nephropathy was low (5.25%), and general adverse reactions were infrequent and predominantly mild. Female sex and hypothyroidism were significant, independent correlates of CIN, whereas hypertension and diabetes mellitus alone did not increase risk when baseline renal function was normal, reaffirming baseline renal function as the pivotal determinant of CIN risk. A history of allergy and hypothyroidism similarly predicted general adverse reactions. These findings support individualised, risk-based patient evaluation — with particular attention to sex, thyroid status and allergy history — over blanket restriction of contrast-enhanced imaging, and highlight the need for larger, multi-centric studies to refine risk-prediction models and guide targeted preventive strategies.

DECLARATIONS

Ethical approval: This study was approved by the Institutional Ethics Committee of Dr. Pinnamaneni Siddhartha Institute of Medical Sciences & Research Foundation. Written informed consent was obtained from all participants.
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REFERENCES

1. American College of Radiology. ACR Manual on Contrast Media. Reston (VA): American College of Radiology; 2024.
2. Sambyal BS, Kansal V, Nayak AK, Chauhan P, Jeldi K. Contrast-induced nephropathy: incidence and reversibility in patients undergoing high-risk percutaneous coronary intervention. J Mar Med Soc. 2024. doi:10.4103/jmms.jmms_15_24.
3. Faggioni M, Mehran R. Preventing contrast-induced renal failure: a guide. Interv Cardiol Rev. 2016;11(2):98–104.
4. Geenen RWF, Kingma HJ, van der Molen AJ. Contrast-induced nephropathy: pharmacology, pathophysiology and prevention. Insights Imaging. 2013;4(6):811–20.
5. Tublin ME, Murphy ME, Tessler FN. Current concepts in contrast media-induced nephropathy. AJR Am J Roentgenol. 1998;171(4):933–9.
6. Bottinor W, Polkampally P, Jovin I. Adverse reactions to iodinated contrast media. Int J Angiol. 2013;22(3):149–54.
7. Shams E, Mayrovitz HN. Contrast-induced nephropathy: a review of mechanisms and risks. Cureus. 2021;13(5):e14842.

8. McDonald JS, McDonald RJ, Comin J, Williamson EE, Katzberg RW, Murad MH, et al. Frequency of acute kidney injury following intravenous contrast medium administration: a systematic review and meta-analysis. Radiology. 2013;267(1):119–28.
9. Bruce RJ, Djamali A, Shinki K, Michel SJ, Fine JP, Pozniak MA. Background fluctuation of kidney function versus contrast-induced nephrotoxicity. AJR Am J Roentgenol. 2009;192(3):711–8.
10. Moos SI, van Vemde DNH, 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):e387–399.
11. Radiological Society of North America. Trends in diagnostic imaging utilization among Medicare and commercially insured adults from 2003 through 2016. Radiology. 2019.
12. Lee H, Song S, Oh YK, Kang W, Kim E. Is gender still a predisposing factor in contrast-media associated adverse drug reactions? A systematic review and meta-analysis of randomized trials and observational studies. Eur J Radiol. 2017;89:81–9.
13. Katayama H, Yamaguchi K, Kozuka T, Takashima T, Matsuura K, Nakata H, et al. Full-scale investigation into adverse reaction in Japan: risk factor analysis. Invest Radiol. 1991;26(Suppl 1):S33–36.
14. Iakovou I, Mehran R, Dangas G, et al. Impact of gender on the incidence and outcome of contrast-induced nephropathy after percutaneous coronary intervention. J Invasive Cardiol.
15. 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):1393–9.
16. Contrast-induced nephropathy. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.
17. Barrett BJ, Parfrey PS. Preventing nephropathy induced by contrast medium. N Engl J Med. 2006;354(4):379–86.
18. 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(6A):5K–13K.
19. Andreucci M, Solomon R, Tasanarong A. Side effects of radiographic contrast media: pathogenesis, risk factors, and prevention. Biomed Res Int. 2014;2014:741018.
20. Dangas G, Iakovou I, Nikolsky E, Aymong ED, Mintz GS, Kipshidze NN, et al. Contrast-induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. Am J Cardiol. 2005;95(1):13–9.
21. Gurdogan M, Ari H. The effect of thyroid functions on contrast-induced nephropathy in patients undergoing coronary angiography and percutaneous coronary intervention. Am J Cardiol.
22. Lin YS, Lin CC, Chen TH, et al. Low free triiodothyronine is associated with contrast-induced acute kidney injury and long-term outcome in elderly patients who underwent percutaneous coronary intervention. PMC.
23. Ahmed S, Antman EM, Ellinor PT, et al. Concurrent use of diuretics, angiotensin converting enzyme inhibitors, and angiotensin receptor blockers with non-steroidal anti-inflammatory drugs and risk of acute kidney injury: nested case-control study. BMJ.
24. Azzalini L, Vilca LM, Lombardo F, et al. Risk of contrast-induced nephropathy in patients undergoing complex percutaneous coronary intervention. Int J Cardiol.
25. Chiu TM, Chu SY. Hypersensitivity reactions to iodinated contrast media. Biomedicines. 2022;10(5):1036.