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Review paper

The use of intravascular contrast media in patients with impaired kidney function – joint clinical practice position statement of the Polish Society of Nephrology and the Polish Medical Society of Radiology

Michał Chmielewski[1,2,A,E,F], Zbigniew Serafin[3,A,E,F], Dorota Kamińska[4,A,E,F], Katarzyna Skrobisz[5,A,E,F], Oliwia Kozak[5,A,E,F], Piotr Olczyk[4,E,F], Przemysław Rutkowski[6,A,E,F], Marcin Adamczak[7,A,E], Edyta Szurowska[5,A,E], Magdalena Krajewska[4,A,E]

[1]Department of Nephrology, Transplantology, and Internal Medicine, Medical University of Gdansk, Poland
[2]Department of Rheumatology, Clinical Immunology, Geriatrics, and Internal Medicine, Medical University of Gdansk, Poland
[3]Department of Radiology and Diagnostic Imaging, Nicolaus Copernicus University, Collegium Medicum, Bydgoszcz, Poland
[4]Department of Nephrology and Transplantation Medicine, Wroclaw Medical University, Poland
[5]Department of Radiology, Medical University of Gdansk, Poland
[6]Department of Internal and Paediatric Nursing, Faculty of Health Sciences, Medical University of Gdansk, Poland
[7]Department of Nephrology, Transplantation, and Internal Medicine, Medical University of Silesia, Katowice, Poland

Abstract

Radiological procedures utilising intravascular contrast media (ICM) are fundamental to modern medicine, enhancing diagnostics and treatment in diverse medical fields. However, the application of ICM has been constrained in patients with compromised kidney function due to perceived nephrotoxic risks, called contrast-induced nephropathy or contrast-induced acute kidney injury. Historical evidence marked ICM as a possible contributor to kidney damage. This led to restrictive guidelines advocating limited ICM use in patients with impaired renal function, preventing crucial radiographic interventions in patients with acute kidney injury (AKI) and chronic kidney disease. Recent advances challenge these traditional views. In particular, no direct causal relationship has been confirmed between contrast administration and elevated serum creatinine concentrations in humans. Furthermore, contemporary research models and meta-analyses do not associate AKI with contrast usage. This paper, prepared by a cross-disciplinary team of nephrologists and radiologists, presents updated guidelines for ICM application amid renal function impairments, emphasising the reduced nephrotoxic risks currently understood and loosening the previous restrictive approach in patients with renal dysfunction.

Key words: intravascular contrast media, chronic kidney disease, acute kidney injury, position statement.

Introduction

Radiological procedures that include intravascular contrast media (ICM) administration are fundamental for contemporary medicine because they allow for improved diagnostics and, as a consequence, better treatment in practically every branch of medical practice. They are indispensable for oncology investigation, interventional radiology, and cardiology procedures, just to name a few. Therefore, it is not surprising that their use is becoming increasingly common. The number of contrast-enhanced computed tomography (CT) examinations is estimated to increase by 4% per year worldwide, for a current yearly total of approximately 150 million procedures [1].

Despite the evident benefits of contrast media in both diagnostics and treatment, in patients with impaired kidney function their use has often been limited or delayed due to perceived risks of so-called contrast-induced

Correspondence address:
Prof. Michal Chmielewski, Department of Nephrology, Transplantology, and Internal Medicine, Medical University of Gdansk, Poland, e-mail: chmiel@gumed.edu.pl

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nephropathy (CIN) or contrast-induced acute kidney injury (CI-AKI). Following initial observations some 70 years ago [2], for decades, ICM has been regarded as potentially nephrotoxic, restricting its use in patients with both AKI and chronic kidney disease (CKD). Results of these observational clinical studies have been strengthened by experimental studies in animals with kidney injury that demonstrated the nephrotoxic potential of ICM [3].

Potential mechanisms for this nephrotoxicity included the generation of reactive oxygen species leading to increased oxidative stress and, hence, tubular damage, haemodynamic disturbances in the kidney medulla, and/or direct toxicity to tubular epithelial cells [4]. Although the decline in kidney function associated with contrast media administration has been considered typically modest and transient, it has also been thought to be associated with serious adverse outcomes, including progressive kidney dysfunction and death [4].

This conviction in the harmfulness of ICM has led to the publication of recommendations and guidelines from numerous national and international societies of radiology and nephrology provenience, including the guidelines of the Polish Society of Nephrology, issued in 2016 [5]. Underlining the nephrotoxicity of contrast media, they have recommended restricting or avoiding their use in patients with impaired kidney function.

However, acknowledging the harmful potential of contrast media has led to a situation in which clinically indicated and potentially lifesaving radiographic procedures have been underutilised or delayed in patients with AKI and CKD. It was documented that patients with CKD are 50% less likely to undergo coronary angiography than those without CKD [6-8]. Among subjects with peripheral vascular disease, those with CKD have been significantly less likely to undergo revascularisation than patients with estimated glomerular filtration rate (eGFR) values higher than 60 ml/min/1.73 m^2 [9]. Fear of contrast toxicity can also lead to deficient oncological proceedings, as often seen in the hospital and outpatient setting when a patient is refused a contrast medium in a diagnostic evaluation of cancer.

Fortunately, this perception of contrast media use in patients with impaired kidney function is gradually changing. First, it must be stressed that a causal link between contrast administration and an increase in serum creatinine has never been proven in humans. Initial studies were limited by their observational character and the absence of control groups. Contemporary use of propensity score-adjusted models, minimising the risk for selection bias, as well as meta-analyses of well-conducted studies, does not show an increased risk of AKI after contrast administration in comparison to patients without it [10,11]. Similar results have been shown in a clinical study completed in subjects from the Polish population [12]. It could be that the transient deterioration of kidney function represents a marker of general vulnerability to adverse events due to increased underlying comorbidity, more severe illness, and/or diminished kidney reserve rather than a true nephrotoxic effect of ICM. Moreover, over the years the properties of ICM have changed, with previous hyperosmolar agents being replaced by more physiological low-osmolar (although still with higher osmolality than human plasma) and finally iso-osmolar ICM. Therefore, the generalisability of previous ICM guidelines to newer agents is questionable. Similarly, the use of gadolinium-based agents for magnetic resonance imaging (MRI) has previously been linked to a serious complication termed nephrogenic systemic fibrosis (NSF). However, as demonstrated in detail below, currently used agents do not increase the risk of NSF.

Our current understanding is that the administration of contrast media carries much less nephrotoxic risk than has been commonly cited in the past and that the true incidence of postcontrast AKI is significantly lower than initially thought.

The present document, completed by a multidisciplinary working group of nephrologists and radiologists, provides a consensus-based statement for the use of ICM in the context of impaired kidney function. The main body of the present recommendations focuses on the use of intravenous (IV) iodinated contrast media, for which our understanding, and hence the guidelines, has changed within the last decade. Specific recommendations will address the use of intraarterial ICM, as well as intravascular gadolinium-based contrast agents for clinical MRI.

Definitions

Statement 1.1

We recommend using the CKD definition as proposed by KDIGO (Kidney Disease Improving Global Outcomes) [13]: CKD is defined as abnormalities of kidney structure or function, present for > 3 months, with health implications, and requires one of two criteria documented or inferred for > 3 months – either GFR < 60 ml/min/1.73 m^2 or markers of kidney damage, including albuminuria (strong recommendation based on international, well-accepted guidelines).

This is the most general definition. CKD staging should also follow the KDIGO [13] criteria as given below:

- stage 1: kidney damage with normal or increased GFR (> 90 ml/min/1.73 m^2),
- stage 2: mild reduction in GFR (60-89 ml/min/1.73 m^2),
- stage 3a: mild to moderate reduction in GFR (45-59 ml/min/1.73 m^2),
- stage 3b: moderate reduction in GFR (30-44 ml/min/1.73 m^2),
- stage 4: severe reduction in GFR (15-29 ml/min/1.73 m^2),
- stage 5: kidney failure (GFR < 15 ml/min/1.73 m^2 or dialysis).

Kidney damage markers in stages 1 and/or 2, should include one or more of the following:

- albuminuria (albumin excretion > 30 mg/24 hr or albumin : creatinine ratio > 30 mg/g [> 3 mg/mmol]),
- urine sediment abnormalities,
- electrolyte and other abnormalities due to tubular disorders,
- histological abnormalities,
- structural abnormalities detected by imaging,
- history of kidney transplantation.

Commentary

The KDIGO guidelines are widely recognised and used for defining and staging CKD. They allow better communication among physicians and facilitate intervention at different stages of the disease.

Statement 1.2

We also recommend following the KDIGO guidelines for AKI, both for the definition and staging purposes [14] (strong recommendation based on international and well-accepted guidelines) (Table 1).

Definition: Increase in creatinine ≥ 0.3 mg/dl (≥ 26 µmol/l) in 48 hours, or increase by > 50%, which is known or presumed to have occurred within the 7 previous days, or urine output < 0.5 ml/kg/h for 6 to 12 hours.

Commentary

The KDIGO guidelines for AKI definition and staging constitute a combination and a compromise between different previous definitions such as the AKIN and RIFLE [15,16]. This allows better communication among physicians and facilitates intervention at different stages of the disease.

Statement 1.3

We recommend using the following definitions for kidney damage associated with ICM administration:

- CI-AKI (contrast-induced acute kidney injury) and CIN (contrast-induced nephropathy) – AKI developing after an ICM procedure, which is causally attributed to ICM administration,
- CA-AKI (contrast-associated acute kidney injury) and PC-AKI (post-contrast acute kidney injury) – AKI developing after an ICM procedure, regardless of whether the causative impact of ICM on kidney damage has been ascertained or not.

Commentary

The terms CI-AKI and CIN assume that ICM is the cause of AKI, which currently seems to occur rarely, if ever. The adjective 'associated' in CA-AKI makes the distinction that AKI cannot be directly attributed to ICM. In the text below, we will use the term PC-AKI, which, in our opinion, best reflects chronology and not causation.

Screening

Statement 2.1. Evaluation of kidney function

We recommend estimating glomerular filtration in the evaluation of kidney function. We recommend the use of the CKD-EPI formula. When CKD-EPI is not available, the CKD-MDRD formula can be used (strong recommendation based on large well-conducted studies and current international guidelines).

Commentary

To adequately assess the risk of PC-AKI, it is necessary to adequately assess baseline kidney function. We recommend the assessment of the estimated glomerular filtration rate (eGFR) rather than serum creatinine or urea because it is more sensitive in addressing actual kidney function. The tools most commonly used for this purpose are the MDRD (modification of diet in renal diseases) and CKD-EPI (chronic kidney disease epidemiology) formulas. The MDRD formula is a widely used method that calculates GFR based on serum creatinine concentrations, age, gender, and ethnic origin [17]. It has been widely used, making it a familiar tool for healthcare professionals. Its main drawback is the risk of underestimating the GFR, especially in individuals with high muscle mass. Additionally, it is less accurate within the normal and near-normal GFR range [18]. The CKD-EPI formula takes into account

Table 1. Staging of acute kidney injury (AKI) [14]

Stage	Serum creatinine	Urine output
1	Increase ≥ 0.3 mg/dl (≥ 26 µmol/l) within 48 hrs or increase 1.5 to 1.9 times baseline creatinine serum concentration	< 0.5 ml/kg/hr for > 6 consecutive hrs
2	Increase 2 to 2.9 times the baseline creatinine serum concentration	< 0.5 ml/kg/hr for > 12 hrs
3	Increase ≥ 3 times the baseline creatinine serum concentration or increase ≥ 4.0 mg/dl (≥ 354 µmol/L) serum concentration or initiation of renal replacement therapy	< 0.3 ml/kg/hr for > 24 hrs or anuria for < 12 hrs

the same variables as MDRD [19]. It has been shown to demonstrate a steeper relationship curve between eGFR and creatinine concentration for higher creatinine values but gentler for lower creatinine values. This characteristic makes the formula more accurate, especially for higher eGFR values.

Statement 2.2. Timing of the evaluation of kidney function

In patients without a history of CKD or patients with stable renal function, we recommend that the results of the eGFR evaluation be valid for 3 months. In inpatient subjects, patients with AKI, and patients with a known rapid progression of kidney dysfunction, eGFR ought to be evaluated no more than 7 days before ICM administration (expert opinion).

Commentary

This recommendation follows other international guidelines, especially those issued by the European Society of Urogenital Radiology (ESUR) [20], as well as the previous recommendations of the Polish Society of Nephrology [5]. Given the lack of scientific data on the validity period of GFR assessment, they appear reasonable and should aid in correctly identifying patients at risk.

Statement 2.3. Risk stratification

To identify patients at risk of developing PC-AKI after an IV ICM, we propose a cut-off eGFR of < 30 ml/min/1.73m² (expert opinion).

Commentary

Data on the relationship between IV ICM administration and the occurrence of AKI can still be regarded as inconclusive. Some studies suggest that the risk of PC-AKI increases with creatinine levels above 1.5 mg/dl [21] and GFR lower than 30 ml/min/1.73 m² [22]. The vast majority of recent controlled retrospective studies of PC-AKI, with the use of modern ICM, reported no excess risk of AKI among patients who underwent contrast-enhanced procedures, as compared to controls, even in patients with baseline eGFR < 30 ml/min/1.73 m² [23-29]. The ESUR guidelines assert that the risk of PC-AKI in patients with eGFR ≥ 30 ml/min/1.73 m² after IV ICM is very low [20]. Due to the lack of properly controlled prospective studies on PC-AKI from IV ICM, and the inconclusive results of retrospective studies in patients with eGFR < 30 ml/min, the thesis that the administration of ICM is associated with kidney damage in these subjects cannot be definitively rejected. In our opinion, patients with GFR ≥ 30 ml/min/1.73m² should be considered safe from PC-AKI.

Nephroprotective measures in high-risk patients

Statement 3.1. Hydration

We do not recommend routine hydration before administering contrast agents in any group of patients. Decisions for patients with GFR < 30 ml/min/1.73 m² or other risk factors (diabetes, patients undergoing AKI, older age, use of large volume of contrast agents) should be made individually after a thorough assessment of the patient's hydration status (recommendation based on the results of observational and interventional studies).

Commentary

The ESUR guidelines suggest IV rehydration for patients in the at-risk group [20]. Administering 1.4% sodium bicarbonate is recommended at a dose of 3 ml/kg one hour before the administration of ICM or 0.9% saline at a dose of 1 ml/kg/h 3-4 hours before and 4-6 hours after ICM administration. However, this recommendation is based on studies with patients receiving intraarterial contrast for coronary procedures [30,31]. These will be addressed below. Regarding IV ICM, IV hydration has not been shown to be more effective than oral rehydration in patients with GFR > 30 ml per min/1.73 m² [32]. There was no significant benefit from IV hydration both in patients with eGFR of 30-59 ml/min/1.73 m² [33,34] or in patients with GFR < 30 ml/min/1.73 m² [35]. Intravenous hydration did not reduce the risk of PC-AKI, dialysis, or in-hospital mortality. Similarly, studies that compared isotonic saline with sodium bicarbonate did not produce decisive results. In theory, sodium bicarbonate might prevent PC-AKI by alkalising tubular fluid and reducing the production of free oxygen radicals. However, clinical studies have not confirmed its advantage over saline [36]. A similar position is presented by the Canadian Association of Radiologists in its guidelines issued in 2022 [37]. In general, hydration should be considered depending on the clinical situation of the patient. It will be recommended in cases of clinically diagnosed dehydration, but contraindicated in overhydrated anuric AKI patients.

Statement 3.2. Drug prophylaxis

We do not recommend any drug prophylaxis before ICM administration (recommendation based on the results of observational and interventional studies).

Commentary

Many substances, such as N-acetylcysteine, have been studied for the prevention of PC-AKI. In the case of N-acetylcysteine, a large systematic review with meta-analysis did not show its effectiveness [38].

Chronic use of statins could be effective in reducing the risk of PC-AKI in patients with renal failure and ischaemic heart disease before PCI procedures [39,40]. The mechanism of this phenomenon is not yet known but is probably related to their pleiotropic antithrombotic, anti-inflammatory, and antioxidant effects [41]. There is no evidence either for the effectiveness of high-dose statin administration shortly before contrast agent administration.

Results of recent clinical studies summarised in a meta-analysis of 13 trials including over 90,000 patients documented that chronic sodium-glucose cotransporter 2 inhibitor (SGLT2i) treatment reduced the risk of AKI both in patients with and without diabetes [42]. Results of recent observational studies suggest that chronic use of SGLT2i reduces the risk of PC-AKI [43,44]. Results of an experimental study suggest that the mechanism of this phenomenon is related to the improvement of tubular cell metabolism related to decreased oxygen consumption in renal proximal tubular cells [44]. As in the case of statins, there is no evidence for the effectiveness of SGLT2i administration shortly before contrast agent administration. Such clinical studies are undoubtedly needed.

Statement 3.3. Withdrawal of potentially nephrotoxic drugs

Metformin should not be used in patients with eGFR < 30 ml/min/1.73 m² (recommendation based on the summary of the product characteristics). We do not recommend discontinuing the renin-angiotensin-aldosterone system (RAAS) inhibitors (recommendation based on randomized trials and meta-analyses). We do not recommend routine discontinuation of nonsteroidal anti-inflammatory drugs (NSAIDs), diuretics, SGLT2i, or any other drugs before administration of ICM (opinion).

Commentary

It should be noted that metformin has no proven effect on the development of PC-AKI, but it increases the risk of lactic acidosis. According to the latest KIDGO and American Diabetes Association (ADA) consensus, metformin is not recommended for use in patients with GFR < 30 ml/min/1.73 m², and in patients with eGFR 30-44 ml/min/1.73 m² the dose should be reduced to 1000 mg per day [45]. There is no need to stop metformin before ICM administration in this group of patients. Patients with shock and AKI have a significantly higher risk of lactic acidosis associated with metformin [46].

Although some small studies and meta-analyses have shown an association between the use of RAAS inhibitors and the risk of PC-AKI, these were based on small groups [47-49]. A meta-analysis of 12 studies indicated that discontinuation of RAAS inhibitors in patients with CKD was associated with a lower risk of PC-AKI, but a direct analysis of use vs. absence of use of RAAS inhibitors showed no significant differences [50]. Another meta-analysis based only on randomised trials did not confirm the association between RAAS inhibitors and PC-AKI [51]. It should be remembered that high doses of NSAIDs, as well as diuretics, could, in some specific circumstances (elderly patients, dehydration), accelerate the progression of CKD [52] and increase the risk of AKI [53]. However, as yet there is insufficient evidence to recommend withholding potentially nephrotoxic drugs such as NSAIDs, diuretics, antimicrobial agents, or chemotherapeutic agents before ICM administration [54].

Statement 3.4. Follow-up

We recommend a follow-up eGFR of 48-72 hours after ICM in all in-hospital patients with eGFR ≤ 30 ml/min/1.73m². In outpatient subjects, eGFR ought to be checked in case of worsening of the clinical status of the patient (expert opinion).

Commentary

If, following ICM administration, any deterioration of kidney function appears, it usually occurs during the first 2-3 days following the procedure involving ICM [4]. Therefore, in at-risk in-hospital patients, it seems optimal to screen for a decrease in eGFR at this time interval. In outpatient patients, acute clinical events, such as a decrease in urine output, dyspnoea, etc., should warrant eGFR control. Patients should be informed that in such cases, they should contact the family physician or emergency department. It is recommended that prepared written information for the patients is given after a radiological procedure with ICM use.

Types and doses of ICM, intervals between consecutive doses

Statement 4.1. Types and dose of ICM

The choice of ICM type and dose should be made by the radiologist performing the procedure, based on considerations other than kidney function (indication, cost, availability) (recommendation based on prospective studies and meta-analyses).

Commentary

Intravascular contrast media can be broadly classified into 3 groups according to their osmolality, defined as the number of particles dissolved in one kilogram of water. High-osmolar-contrast media (HOCM) are 5 to 8 times the osmolality of blood (1500 to over 2000 mOsm/kg H_2O) and are no longer used in clinical practice. Newer agents, although called low-osmolar-contrast media (LOCM), have

an osmolality of around 900 mOsm/kg H_2O, which is more than 3 times the osmolality of blood. Finally, iso-osmolar contrast media (IOCM), according to their name, are characterised by an osmolality of around 290 mOsm/kg H_2O. Comparisons between LOCM and IOCM in terms of their potential nephrotoxicity have produced mixed results. Some studies show that particular LOCM (as iohexol and ioxaglate) could be associated with a higher risk of PC-AKI, in comparison to IOCM (iodixanol) and some other LOCMs (ioversol, iomeprol, iopromide, iopomidol) [55-57]. However, in a large meta-analysis completed in 2017 in patients with impaired kidney function undergoing coronary angiography, the use of IOCM did not show a general difference in the incidence of PC-AKI compared to LOCM [58].

The contrast agent dose from the IOCM or LOCM group varies between 1 and 1.5 ml/kg body weight. For CT scans performed in children, the dose of contrast agent increases to 2-3 ml/kg body weight up to a maximum of 50 ml. The type, amount, and dose of contrast agent should be decided by the radiologist to achieve a reliable examination result.

Statement 4.2. The interval between consecutive ICM doses

The interval between 2 consecutive administrations of ICM in routine radiological examinations should depend on the patient's renal function (recommendation based on the results of observational studies and ICM pharmacokinetics). It should be stressed that this recommendation does not apply in emergency or life-threatening situations, when repeated doses of ICM should be administered based on the clinical demand irrespective of the kidney function.

Commentary

The pharmacokinetics of ICM should determine the waiting intervals between successive CT or MRI examinations [59]. In patients with normal or moderately impaired renal function (eGFR > 30 ml/min/1.73 m^2), 75% of ICM is excreted within 4 hours of administration. An interval of 4 hours between ICM administrations should be maintained. Patients with severe renal impairment (eGFR < 30 ml/min/1.73 m^2) should have a consecutive dose of ICM delayed by 48 hours. Iodine contrast agents and gadolinium contrast agents can be administered on the same day for routine examinations if patients have normal or moderately impaired renal function. Gadolinium contrast agents attenuate X-rays. When excreted into the urinary tract, they can cause interpretive errors in urinary CT studies. For abdominal examinations, a contrast-enhanced CT examination should be performed before a contrast-enhanced MR examination. Otherwise, for patients with advanced kidney insufficiency (eGFR < 30 ml/min/1.73m^2), the interval between the administration of contrast agents of gadolinium and iodine should not be shorter than 7 days. For chest and brain examinations, the order of CT and MR examinations does not matter.

Gadolinium-based magnetic resonance imaging (MRI)

Statement 5.1.

We do not recommend evaluating kidney function for gadolinium-based magnetic resonance imaging performed with the lowest-risk contrast media (recommendation based on well-designed observational studies).

Commentary

Contrast agents used in magnetic resonance imaging are gadolinium based. They are classified according to their biochemical structure into linear and macrocyclic and further subdivided according to their charge as ionic or non-ionic character. Macrocyclic chelates are more stable than linear ones, and ionic linear chelates are more stable than non-ionic linear chelates [60]. Stability refers to the ability of the ligand to retain the Gd^{3+} ion within the complex. The free gadolinium ligand is toxic; thus, stability is the most important factor for gadolinium toxicity. Renal function has been considered a crucial determinant of gadolinium toxicity, and so-called nephrogenic systemic fibrosis (NSF) has been associated with exposure to gadolinium-based contrast agents. Nephrogenic systemic fibrosis results from impaired gadolinium-based contrast media excretion in patients with severe renal insufficiency, allowing gadolinium chelates of lower stability to dissociate, releasing free gadolinium. The condition is characterised by progressive tissue fibrosis that features thickening and hardening of the skin. In some patients, fibrosis of deeper structures can occur, within muscle, fascia, lungs, and heart [61]. The risk of NSF was found to increase in patients with CKD 4 and 5, in patients on dialysis, and in patients with AKI [62]. However, this risk differs significantly with the use of various agents, being as high as 3-11% after linear gadodiamide exposure, and practically negligible after macrocyclic gadopentetate dimeglumine [62].

Linear agents presenting with the highest risk of NSF (Gadodiamide Omniscan®, Gadopentetate dimeglumine Magnevist®) have been suspended by the European Medicines Agency (EMA) and are no longer in use. Gadoversetamide Optimark® was also withdrawn from the European market. Since this restrictive policy on the use of gadolinium-based contrast media was introduced, the reports of NSF have significantly decreased. In a systematic review of 16 studies analysing the risk of NSF in gadolinium-based contrast media with high affinity, Gd^{3+} binding (lowest-risk group) was less than 0.07% [63].

This means that contrast-enhanced MRI with low-risk gadolinium-based agents should not be avoided in

Table 2. Gadolinium-based contrast agents grouped according to the associated risk of nephrogenic systemic fibrosis (based on ESUR CMSC guideline ver. 10.0)

Gadolinium-based contrast agents – risk classification (based on esur cmsc guideline version 10.0)	
Highest risk	Gadodiamide (Omniscan®) Ligand: Nonionic linear chelate (DTPA-BMA) Gadopentetate dimeglumine (Magnevist®) Ligand: Ionic linear chelate (DTPA) Gadoversetamide (Optimark®) Ligand: Nonionic linear chelate (DTPA-BMEA)
Intermediate risk	Gadobenate dimeglumine (Multihance®) Ligand: Ionic linear chelate (BOPTA) Gadoxetate disodium (Primovist®, Eovist®) Ligand: Ionic linear chelate (EOB-DTPA)
Lowest risk	Gadobutrol (Gadovist®, Gadavist®) Ligand: Nonionic Cyclic Chelate (BT-DO3A) Gadoterate meglumine (Dotarem®, Magnescope® plus generic products) Ligand: Ionic Cyclic Chelate (DOTA) Gadoteridol (Prohance®) Ligand: Non-ionic cyclic chelate (HP-DO3A)

patients with kidney dysfunction, because the risk of complications, including NSG, is marginal. In patients in whom an intermediate-risk agent is planned, eGFR should be assessed, and in patients with impaired renal function, such media can be used only for hepatobiliary imaging. The lowest-risk agents can be used for all MRI examinations irrespective of kidney function (Table 2). The measurement of eGFR before examination is not mandatory. The amount of contrast agent administered should guarantee good-quality imaging, irrespective of kidney function.

Intra-arterial contrast administration

Statement 6.1.

For procedures with intra-arterial ICM with a second-pass renal exposure, we advocate proceeding as in IV ICM procedures, classifying persons with eGFR < 30 ml/min as at-risk patients, i.e. we do not recommend routine hydration before administering contrast agents (see Statement 3.1). For procedures with an intraarterial ICM with a first-pass renal exposure, preventive measures are recommended for patients already at a stage of eGFR < 45 ml/min/1.73m^2. We recommend a follow-up serum creatinine measurement 48 to 72 hours after intraarterial ICM injection in the abovementioned at-risk patients.

Commentary

To understand the potential nephrotoxicity of ICM in intraarterial administration, it is important to differentiate between first-pass and a second-pass renal exposure [20]. First-pass renal exposure indicates that ICM reaches the kidneys directly, in a relatively undiluted form. This occurs when injection is performed into the thoracic aorta or the suprarenal abdominal aorta. In clinical practice, the most common first-pass administration is associated with coronary angiography. The second pass renal exposure occurs when the contrast agent flows through the pulmonary circulation and then reaches the renal arteries. This situation most frequently occurs in interventional radiology. Obviously, first-pass renal exposure results also in second-pass renal exposure. There is solid evidence, based on randomised trials, that the risk of PC-AKI after intraarterial ICM administration with first-pass renal exposure is increased as compared to the administration of IV ICM [64,65]. The potential pathophysiological mechanisms include the multiplicity of ICM injections during an intraarterial procedure versus a single injection for a typical IV procedure, and/or the ICM volume, which is typically higher in intraarterial interventions. However, it might also be that the increased risk of AKI following intraarterial ICM is not caused by the ICM itself. Instead, other factors might be involved, such as co-morbidities and/or cholesterol microembolisation associated with catheter insertion.

In the present statement, we agree with previous guidelines from other national societies [20,37]. For procedures with an intraarterial ICM with a second-pass renal exposure, we advocate proceeding as in an IV ICM procedure. In patients, in whom an intraarterial ICM with first-pass renal exposure is administered, preventive measures are recommended for patients already with eGFR < 45 ml/min/1.73m^2. These include considering IV fluids, depending on the patient's hydration status, as described above, temporal withdrawal of metformin, and a follow-up of kidney function 48 to 72 hours after exposure. Hydration is recommended in patients undergoing coronary angiography and angioplasty, where the benefits of hydration with isotonic fluids before the procedure have been proven [66,67]. A lower risk of PC-AKI was observed in patients with serum creatinine > 1.5 mg/dl rehydrated with 0.45% saline undergoing percutaneous coronary intervention [30]. In clinical practice, many interventional cardiologists use a so-called Mehran score, which takes into account the acknowledged risk factors for deterioration of kidney function: hypotension, intra-aortic balloon pump, congestive heart failure, chronic kidney disease, diabetes, age > 75 years, anaemia, and volume of contrast [68]. It is a practical tool because it not only defines high-risk groups but also indicates with good accuracy the highest contrast media volume that may be safely administered. Its applicability has been challenged recently [69], but it remains useful for everyday practice.

Special groups of patients

Statement 7.1. Patients with AKI

For an emergent presentation, we recommend proceeding with an indicated contrast-enhanced imaging study without delay, temporally withdrawing every nephrotoxic drug before contrast exposure, and hydrating the patient according to his hydration status with IV saline or natrium bicarbonate (expert opinion).

Commentary

Patients with AKI might be more susceptible to contrast-induced kidney damage than those without AKI, although no controlled studies report on this risk. However, in this group of patients, the risk of worsening kidney function should always be weighed against the risk derived from delayed or missed diagnoses due to the avoidance of ICM [70]. Prophylactic dialysis is not needed before or after the contrast procedure.

Statement 7.2. Patients with a single kidney

We recommend treating patients with a single kidney in a similar way to the general population, based on the evaluation of eGFR (expert opinion).

Commentary

The risk of PC-AKI in people with a single kidney should be classified according to overall kidney function (eGFR) and clinical circumstances. The presence of a solitary functioning kidney should not affect decision-making about the risk of PC-AKI [71].

Statement 7.3. Patients on dialysis (haemodialysis, peritoneal dialysis)

It is not necessary to correlate the time of injection of ICM with the dialysis session. An additional haemodialysis session is not necessary to remove ICM unless symptomatic fluid overload develops. Contrast media can be administered to patients on peritoneal dialysis or haemodialysis regardless of their residual urine output, and no change in dialysis schedule is required (recommendation based on observational studies).

Commentary

Based on a systematic review of 9 studies, we have learned that there is little or no effect of ICM on residual renal function in dialysis patients [72]. Therefore, the presence or absence of residual urine output should not influence the decision to use ICM in dialysis patients.

Limitations and conclusions

The widespread belief in ICM nephrotoxicity has created the current situation, which all clinicians and patients know from their daily experience: refusal to execute contrast-enhanced radiological procedures, disqualification from contrast-associated interventions, and significant delays in diagnostics, including oncological evaluations.

Fortunately, this situation is gradually changing. With the introduction of modern ICM, and perhaps more importantly, thanks to the current, better-designed and executed studies and analyses, our understanding of the potential nephrotoxicity of ICM has changed. For a vast majority of patients with normal to moderately impaired kidney function (i.e. with eGFR > 30 ml/min/1.73 m^2), there is now consensus that ICMs are safe and do not exert any significant risk of deterioration of AKI or CKD. In cases of severe renal dysfunction (i.e. when eGFR < 30 ml/min/1.73 m^2), the risk of PC-AKI cannot be ruled out, although previous reports on its incidence and severity have been considerably overstated.

However, even in these patients, the risk of PC-AKI should not be considered a contraindication to the administration of ICM, particularly in emergent clinical situations. In each of such cases, the benefits of ICM should be balanced against the potential risks of PC-AKI. For this,

Table 3. Practical concise recommendations for the clinicians

Type of ICM intervention	At-risk group	Action
Intravenous ICM	eGFR < 30 ml/min	Re-analyse the necessity of ICM administration Check for metformin – withdraw Hydrate the patient if clinically indicated Interval of 48 hours between consecutive ICM administrations For in-patients, check eGFR 48-72 hours following ICM administration For outpatients, check eGFR in case of worsening of the clinical status following ICM administration
Intraarterial ICM with a first-pass exposure	eGFR < 45 ml/min	As above
Intraarterial ICM with a second-pass exposure	eGFR < 30 ml/min	As above
The gadolinium-based procedure with the lowest-risk media	None	None

constant cooperation between the practitioner ordering the radiological procedure and the radiologist is necessary, either as routine clinical-radiological meetings or as radiology consultations. In some patients, equally clinically valuable results could be obtained with less invasive modalities, such as an ultrasound examination. In others, MRI with IV administration of the contemporary lowest-risk macrocyclic gadolinium formulas would be a better option. However, it should be stressed that in all patients with an indication for ICM, the procedure ought to be performed, irrespective of kidney function. Decreased GFR, regardless of the stage of AKI/CKD, cannot be regarded *per se* as a contraindication to ICM administration (Table 3).

These guidelines are limited by the lack of well-designed prospective randomised trials in the area of potential ICM nephrotoxicity. We base our current opinion on the data derived from registries, retrospective studies, and meta-analyses. However, their design makes them credible, and, in the absence of prospective evaluations, we must rely on them. Our position remains in line with the guidelines and statements of other national and international societies and groups of experts [20,37,54,73,74].

We strongly believe that, for the benefit of our patients, these recommendations, prepared jointly by a multidisciplinary team of nephrologists and radiologists, ought to be disseminated among medical communities.

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Conflict of interest

The authors report no conflict of interest.

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