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www.bsmi.uz
https://newdaymedicine.com E:
ndmuz@mail.ru
Тел: +99890 8061882

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CURRENT RISK OF CONTRAST-INDUCED ACUTE KIDNEY INJURY AFTER CORONARY ANGIOGRAPHY AND INTERVENTION: A REAPPRAISAL OF THE LITERATURE

Mustafaeva Malika Rustamovna https://orcid.org/0000-0001-9280-276

Bukhara State Medical Institute named after Abu Ali ibn Sino, Uzbekistan, Bukhara, st. A. Navoi.1
Tel: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Resume***

Contrast-induced acute kidney injury (CI-AKI) is the acute impairment of renal function further to the intravascular administration of iodinated contrast media, and occurs most frequently after coronary angiography, percutaneous coronary intervention, and contrast-enhanced computed tomography. CI-AKI has been associated with the development of acute renal failure, worsening of chronic kidney disease, requirement for dialysis, prolonged hospital stay, and higher mortality rates and health care costs. Recently, a number of studies suggested that contrast media exposure might not be the causative agent in the occurrence of acute kidney injury, particularly in stable patients who receive small to moderate amounts of contrast media. However, those who undergo coronary angiography and intervention are indeed subject to an increased hazard of CI-AKI, in view of a more significant contrast media exposure as well as the presence of concomitant risk factors. Solid randomized clinical trials are therefore required to identify preventative strategies to reduce the risk of CI-AKI and its complications in these patients.

Key words: acute kidney disease, contrast-induced nephropathy, percutaneous coronary intervention.

СОВРЕМЕННЫЙ РИСК КОНТРАСТ-ИНДУЦИРОВАННОГО ОСТРОГО ПОВРЕЖДЕНИЯ ПОЧЕК ПОСЛЕ КОРОНАРНОЙ АНГИОГРАФИИ И ВМЕШАТЕЛЬСТВА: ПЕРЕОЦЕНКА ЛИТЕРАТУРЫ

Мустафаева Малика Рустамовна https://orcid.org/0000-0001-9280-276

Бухарский государственный медицинский институт имени Абу Али ибн Сины, Узбекистан,
г. Бухара, ул. А. Навои. 1 Тел: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Резюме***

Контраст-индуцированное острое повреждение почек (КИ-ОПП) представляет собой острое нарушение функции почек в результате внутрисосудистого введения йодсодержащих контрастных веществ и чаще всего возникает после коронарной ангиографии, чрескожного коронарного вмешательства и компьютерной томографии с контрастированием. КИ-ОПП было связано с развитием острой почечной недостаточности, ухудшением хронической болезни почек, необходимостью диализа, длительным пребыванием в больнице, а также более высокими показателями смертности и затратами на здравоохранение. Недавно ряд исследований показал, что воздействие контрастных веществ не может быть причиной возникновения острого повреждения почек, особенно у стабильных пациентов, которые получают небольшое или умеренное количество контрастных веществ. Однако те, кто подвергается коронарной ангиографии и вмешательству, действительно подвергаются повышенному риску КИ-ОПП ввиду более значительного воздействия контрастных веществ, а также наличия сопутствующих факторов риска. Поэтому необходимы основательные рандомизированные клинические исследования для определения профилактических стратегий, позволяющих снизить риск КИ-ОПП и его осложнений у этих пациентов.

Ключевые слова: острая болезнь почек, контраст-индуцированная нефропатия, чрескожное коронарное вмешательство.

KORONOAR ANGIGRAFIYA VA INTERVENSIYA KEYIN KONTRAST BO'LGAN O'TKIR BUYRAK SHIKASTLANISHINING HOZIRGI XAVFI: ADABIYOTLARNI QAYTA BAHOLASH

Mustafaeva Malika Rustamovna https://orcid.org/0000-0001-9280-276

Abu Ali ibn Sino nomidagi Buxoro davlat tibbiyot instituti, O‘zbekiston, Buxoro sh. A. Navoiy kochasi 1 Tel: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Rezyume***

Kontrastli o'tkir buyrak shikastlanishi (CI-AKI) yodlangan kontrastli vositalarni tomir ichiga yuborishgacha bo'lgan buyrak funktsiyasining o'tkir buzilishi bo'lib, koronar angiografiya, teri orqali koronar aralashuv va kontrastli kompyuter tomografiyasidan keyin tez-tez uchraydi. CI-AKI o'tkir buyrak etishmovchiligining rivojlanishi, surunkali buyrak kasalligining yomonlashishi, dializga bo'lgan ehtiyoj, uzoq vaqt kasalxonada qolish, o'lim darajasi va sog'liqni saqlash xarajatlarining oshishi bilan bog'liq. Yaqinda bir qator tadqiqotlar shuni ko'rsatdiki, kontrast moddaning ta'siri o'tkir buyrak shikastlanishining qo'zg'atuvchisi bo'lmasligi mumkin, ayniqsa kontrast moddani kichik va o'rtacha miqdorda qabul qiladigan barqaror bemorlarda. Biroq, koronar angiografiya va aralashuvdan o'tganlar, kontrastli vositalarning sezilarli darajada ta'sirini hisobga olgan holda, shuningdek, birga keladigan xavf omillari mavjudligi sababli, CI-AKI xavfini oshiradilar. Shu sababli, ushbu bemorlarda CI-AKI va uning asoratlari xavfini kamaytirish uchun profilaktika strategiyalarini aniqlash uchun qattiq randomizatsiyalangan klinik tadqiqotlar talab qilinadi.

Kalit so'zlar: o'tkir buyrak kasalligi, kontrastli nefropatiya, teri orqali koronar aralashuv.

Relevance

In this article, we provide a critical review of the recently published literature challenging the association between contrast media administration and contrast-induced acute kidney injury (CI-AKI) development. Additionally, we review the pathogenesis of acute kidney injury (AKI) in the context of coronary angiography and intervention. Finally, we stress the importance of intravascular volume expansion to minimize the risk and effects of CI-AKI.

CI-AKI: Definition of the Problem

CI-AKI is defined as the occurrence of acute renal impairment after administration of iodinated contrast media, which exert nephrotoxic effects by means of vasoconstriction, oxidative stress, osmotic tubular nephrosis, and ischemia of the outer medulla.1 Most CI-AKI cases stem from intravascular contrast media exposure during coronary angiography, percutaneous coronary intervention (PCI), and contrast-enhanced computed tomography imaging.2 Traditionally, CI-AKI has been re- ported as one of the leading causes of acute renal failure during hospitalization,2 and is associated with progression of renal failure, requirement for dialysis, prolonged hospital stay, mortality, and increased costs.1,3-5 However, recent publications have challenged these associations, and some researchers have called for a reappraisal of the actual role of contrast media in the development of AKI and its effect on clinical outcomes.

Does CI-AKI Really Exist?

Recent studies reported a low incidence of CI-AKI (2.4%- 6.4%) in patients who underwent intravenous contrast media administration for computed tomography imaging.6,7 In several studies, McDonald et al.7-9 analyzed the risk of CI-AKI and its effect on outcomes in patients who underwent computed tomography imaging with or without contrast media administration. After propensity score adjustment, the authors reported that AKI risk was similar between contrast- and non-contrast-enhanced scans, across all risk subgroups.9 These data suggest that critically ill patients who undergo computed tomography imaging might have other causes of AKI and that contrast media might not be the causative agent. Additionally, intravenous contrast media administration was not associated with higher 30-day mortality or need for dialysis. Although patients who developed AKI suffered higher rates of dialysis and mortality, contrast media exposure was not an independent predictor of either outcome, even among subjects with chronic kidney disease (CKD) or other predisposing comorbidities.8

Similarly, a recent report on 6 million hospitalizations showed no difference in the incidence of CI-AKI between patients exposed to and those not exposed to contrast media (5.5% vs 5.6%).10 This cohort included subjects who under went contrast media exposure in different settings and for a wide variety of conditions. The risk of AKI increased with a higher comorbidity burden in both groups. After adjustment for comorbidity and clinical presentation, contrast media administration was paradoxically associated with a 7.4% reduction in AKI odds. The authors concluded that the relationship between contrast media administration and AKI is highly confounded, unpredictable, and might express a risk-treatment paradox ("renalism"11). In particular, among patients with acute coronary syndrome (ACS), subjects with CKD undergo coronary angiography and intervention significantly less frequently than non-CKD patients, because of an aversion to the risk of CI-AKI, despite the fact that the would benefit the most from revascularization.12 Crucially, these patients who are not exposed to contrast media still suffer higher rates of AKI (because of the direct consequences of an unrevascularized ACS on kidney function), compared with subjects who undergo an invasive management and thereby are exposed to contrast media.8

This body of evidence might suggest that contrast media administration might not be the causative agent of increased creatinine values, and might not be associated with a higher risk of AKI, dialysis, or death, even among subjects with comorbidities predisposing to nephrotoxicity.8,9 To further support this hypothesis, the authors10 point out that, during hospitalization and in the absence of contrast media exposure,

patients' serum creatinine levels might fluctuate significantly, often exceeding thresholds considered diagnostic of CI-AKI.13 Therefore, in a relevant proportion of cases, development of AKI might be pathophysiologically unrelated to earlier (incidental) contrast media exposure. Interestingly, the same conditions considered to be CI-AKI risk factors (eg, age, diabetes, advanced CKD, heart failure, hemodynamic instability, etc) also represent risk factors for AKI in general (contrast media-unrelated), which might therefore be mediated through alternative pathways.

The authors of the aforementioned reports7-10 conclude that the incremental risk of AKI that can be attributed to contrast media is modest and probably overestimated.10 However, these studies present important limitations, namely their observational nature and their "big data approach." Because the authors relied on administrative discharge summaries to adjudicate AKI events, they could not capture variables of critical importance such as the volume of contrast media administered, laboratory exams (including serum creatinine values), and the temporal sequence between contrast media administration and AKI development, thus losing precision in the assessment of such a complex phenomenon. As a consequence, their findings and conclusions should be regarded with caution, because they do not provide any definitive evidence of the lack of association between contrast media exposure and subsequent AKI.

CI-AKI in Patients Who Undergo Coronary Angiography and Intervention

Another important limitation of the aforementioned re- ports is represented by the fact that most of their data come from populations that underwent noninvasive procedures, and high-quality data specific to a population that underwent coronary angiography and intervention are scarce.

The incidence of CI-AKI is somehow higher after coronary angiography and intervention (7.1%-10.5%14,15), compared with computed tomography imaging (2.4%-6.4%6,7), a finding that can be explained by differences in baseline characteristics and clinical presentation between the 2 patient populations, route of contrast media administration, and contrast media volumes (Fig. 1). In fact, subjects who undergo coronary angiography and intervention often have a higher burden of comorbidities and are frequently more critically ill than those who undergo computed tomography imaging: diabetes, advanced CKD, heart failure, cardiogenic shock, and ACS are commonly encountered in patients who undergo PCI and have been independently associated with a higher risk of CI-AKI.14 Additionally, intra-arterial as opposed to intravenous contrast media administration has been linked to a higher risk of CI-AKI, although the exact underlying mechanisms are poorly understood.16,17 These might involve direct toxic effects by undiluted contrast media reaching the nephrons, but are possibly compounded by concomitant alternative causes of AKI during cardiac catheterization, such as hypo- tension, microshowers of athero emboli to the renal arteries, and bleeding.1,17 Indirect support for this concept has been presented by radial artery access studies, which

have reported lower rates of CI-AKI with radial as opposed to femoral access18 (with radial access the abdominal aorta and renal

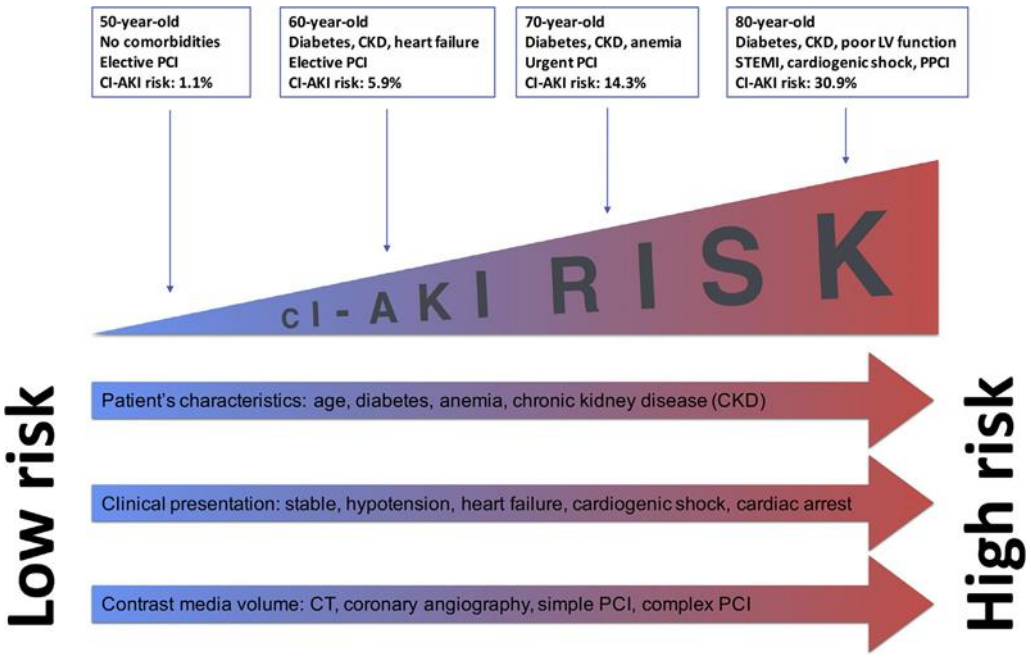

Figure 1. Contrast-induced acute kidney injury (CI-AKI) risk varies as a function of patient's characteristics, clinical presentation, and contrast media volume. Risk figures in the examples were calculated using the Society for Cardiovascular Angiography and Interventions SCAI PCI risk calculator available at: http://scaipciriskapp.org/assess/porc. CKD, chronic kidney disease; CT, computed tomography; LV, left ventricular; PCI, percutaneous coronary intervention; PPCI, primary percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction.

Artery ostia are not touched by wires and catheters). Finally, although contrast media volumes for coronary angiography and computed tomography imaging are currently quite low (50-100 mL), patients who undergo PCI receive higher contrast loads (usually > 150 mL),14 which expose them to an increased risk of CI-AKI. In fact, a large registry of ACS patients from Alberta indicates that subjects who undergo coronary angiography and intervention are exposed to an 18% increase in the risk of CI-AKI, compared with propensity score-matched patients managed conservatively.19 Because a linear relationship exists between contrast media volume and risk of CI-AKI (an increase of 12%-28% per 100 mL administered 20-22) and larger amounts of contrast media are administered during coronary intervention compared with radiological imaging, the highest incidence and clinical effects of CI-AKI will be observed in patients who undergo complex PCI.

Does Intravenous Volume Expansion Prevent CI-AKI?

The recently published A Maastricht Contrast-Induced Nephropathy Guidelines (AMACING) trial23 reported a low incidence of CI-AKI (2.7%) in patients exposed to contrast media mostly in the context of radiological imaging (interventional procedures were performed in 15% of patients). A strategy of aggressive, guidelines-recommended intravenous hydration in these patients was not associated with decreased rates of CI-AKI, and no prophylaxis was reported to be noninferior and cost-saving in this context. The reasons underlying these findings likely mirror those explaining the observed lack of association between contrast media administration and development of CI-AKI in radiological imaging: the selection of patients at low to moderate risk (reduced contrast media amounts in noncritically ill patients) implies that exposure to a risk factor (contrast media) might not be sufficient to trigger the development of the associated condition (CI-AKI) in a significant proportion of cases. Similarly, prophylactic strategies aimed at decreasing the odds of developing that condition might not show their benefit because of selection of a target population with too low a risk profile. Additional limitations of AMACING are represented by sample size recalculation while the study was ongoing (from n ¼ 1300

to n ¼ 660), and an unusually large non- inferiority margin chosen by the investigators (2.1%; the estimated incidence of CI-AKI in the hydration group being 2.4%).

Indeed, there seems to be an inverse dose-response relationship between volume of intravenously administered fluids and incidence of CI-AKI in higher-risk patients. In the Prevention of Contrast Renal Injury With Different Hydration Strategies (POSEIDON) trial,24 the incidence of CI-AKI was greatly reduced (16.3% vs 6.7%) using a left ventricular end- diastolic pressure-guided hydration scheme, because of a much higher mean fluid volume administered, compared with a standard hydration protocol (1727 vs 812 mL). Thus, the data in aggregate suggest that CI-AKI is sensitive to intra- vascular volume depletion and is potentially lessened with volume expansion given over a few hours before intravascular contrast administration.

However, only 28% of patients in POSEIDON underwent PCI and mean contrast media volume was approximately

110 mL. Additionally, emergent coronary angiography and intervention and decompensated heart failure represented exclusion criteria. These factors potentially limited the extent of benefit of hemodynamic-guided fluid administration for the prevention of CI-AKI. Solid randomized evidence in a truly high-risk population is therefore lacking.

Conclusions

CI-AKI is a real complication, although until recently its risk has been overestimated, particularly in cohorts of stable patients who undergo radiological imaging. Subjects who undergo coronary angiography and intervention have a higher prevalence of CI-AKI risk factors and are subject to a greater risk of CI-AKI, especially in the setting of urgent intervention, and therefore greatly benefit from targeted preventative measures to expand intravascular volume and augment renal perfusion. We look forward to solid randomized clinical trials that will identify optimal strategies to reduce the incidence, severity, and sequelae of CI-AKI, to optimize the outcomes of patients who undergo invasive management. In particular, an adequately-sized randomized trial on the role of prophylactic hydration in a truly high-risk population of subjects who undergo PCI is warranted.

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