Thyago Anzolin Coser

INCIDÊNCIA DE LESÃO RENAL AGUDA E O USO DE CONTRASTE ENDOVENOSO – ESTUDO RETROSPECTIVO

Dissertação apresentada à Universidade de Caxias do Sul, para obtenção do Título de Mestre em Ciências da Saúde.

Caxias do Sul
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NEFROPATIA INDUZIDA POR CONTRASTE APÓS TOMOGRAFIA COMPUTADORIZADA NA SERRA GAÚCHA

Thyago Anzolin Coser

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Dedicatória

À minha família, a qual é meu alicerce, apoiando-me em todos os momentos, mesmo que para isso, muitas vezes, fique privada da minha presença.

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Sumário

Esta dissertação de Mestrado Acadêmico Stricto Sensu é apresentada no formato exigido pelo Programa de Pós-Graduação em Ciências da Saúde da Universidade de Caxias do Sul. A mesma é constituída da secção de “Introdução com referências bibliográficas”, a inclusão do artigo original submetido em periódico Qualis A na classificação da Coordenação de Aperfeiçoamento de Pessoal em Nível Superior (CAPES), e as “Considerações Finais e Perspectivas”.

1 INTRODUÇÃO

Os meios de contraste iodados endovenosos (EV) são comumente aplicados na realização de exames de Tomografia Computadorizada (TC) para aprimorar a precisão diagnóstica[1]. No entanto, esse uso não é isento de riscos, pois contrastes iodados possuem toxicidade que pode causar disfunção renal[2]. As sociedades médicas definiram Nefropatia induzida por contraste (NIC), inicialmente, nos seguintes critérios: aumento absoluto da creatinina sérica (CrS) ≥0,5 mg/dL ou relativo ≥25% entre 48 a 72h após a exposição[3-7]. Atualmente, o estudo KDIGO sugere o conceito de NIC como a elevação absoluta da CrS ≥0,3 mg/dL ou relativa ≥50% entre 2 a 7 dias após a realização do exame[8].

A patogênese da NIC em modelos animais consiste em um quadro clássico de necrose tubular aguda (NTA), secundária à vasoconstrição, associado à hipoxemia medular e, consequentemente, à citotoxicidade das células tubulares. O dano vascular ocorre pela redução dos níveis de óxido nítrico e pelo aumento da endotelina e da adenosina na corrente sanguínea[9,10].

Segundo a literatura concernente à epidemiologia, a NIC é reconhecida como a terceira causa de lesão renal aguda (LRA) em pacientes hospitalizados, com incidência média de 11%[11]. Essa frequência pode variar entre 1% a 50%, de acordo com as características populacionais e o uso de contraste arterial ou venoso[12-19]. No caso de pacientes com administração exclusiva de contraste venoso, a incidência de NIC pode variar de 2,5% a 12%[15-19].

Os fatores de risco clássicos à NIC são: sexo feminino, idosos, diabete mellitus (DM), doença cardiovascular (DCV), processos infecciosos, obesidade, doença renal crônica (DRC), uso de fármacos nefrotóxicos, entre outros[8,20-24].

Em praticamente todos os casos, a diminuição da taxa de filtração glomerular (TFG) é ligeira e transitória, com a recuperação dentro de três a cinco dias [25,26]. No entanto, em alguns pacientes portadores de DRC, a CrS pode não retornar aos valores basais. Mesmo que a CrS retorne ao nível citado (basal), o desenvolvimento de NIC tem sido associado a resultados adversos a curto e longo prazo[27]. Os pacientes usualmente permanecem não-oligúricos e, em função disso, a diurese não é considerada um dos critérios para NIC[28].

Podem ainda estar presentes outras manifestações de LRA na NIC, incluindo hipercalemia, acidose, fração de excreção de sódio (FENa) menor que 1% e hiperfosfatemia. Deve-se atentar para o diagnóstico diferencial de LRA por NIC, o qual inclui ateroêmbolos renais, NTA isquêmica, nefrite intersticial aguda e alterações de hipovolemia[29].

Não há um tratamento que seja específico para NIC. Quando a LRA se desenvolve, a conduta deve ser como para qualquer outra causa de NTA, com o foco na manutenção do equilíbrio hidroeletrolítico. O melhor tratamento da NIC continua sendo a prevenção[26,27,29].

A intervenção primária para prevenção de NIC consiste na administração adequada de fluidos isotônicos EV previamente à administração de contraste radiológico[30-32]. Um consenso da sociedade americana de radiologia recomenda a infusão de solução salina 0,9% para pacientes com risco de NIC[33]. Um estudo clínico randomizado recente não demonstrou redução na incidência de NIC com hidratação EV para pacientes com TFG entre 30 e 59 mL/min/1,73m^2, sugerindo que a reidratação EV deve ser reservada para pacientes com TFG <30 mL/min/1,73m^2[34]. O regime ideal para a administração de fluidos é incerto; entretanto, o uso de 1 mL/kg de solução salina isotônica por 4 a 12 horas pré e pós-procedimento é preconizado[35]. Pode ser utilizado bicarbonato ou solução salina isotônica, embora exista uma ligeira preferência pela solução salina isotônica, uma vez que é menos dispendiosa e não há risco de erros de composição[8]. Embora as informações na literatura sejam conflituosas, em pacientes propensos à NIC, a acetilcisteína (NAC) oral possui indicação principalmente pela sua associação com um pequeno benefício na nefroproteção no uso de contraste radiológico intra-arterial [36,37].

Nesse panorama, ao revisar a literatura científica disponível, percebe-se que existem vários artigos que mostram não haver risco aumentado de NIC, independentemente dos fatores de risco[1,24,38-43]. Em contrapartida, alguns relatam NIC em pacientes com disfunção renal pré-existente[18,44]. A maioria dos estudos trata sobre NIC após a aplicação de contraste arterial e existem poucos artigos relacionando NIC ao contraste EV. Dessa forma, nosso estudo teve como objetivo principal correlacionar a incidência de LRA com uso de contraste EV em TC.

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3 ARTIGO

Incidence of acute kidney injury and the use of intravenous contrast - retrospective study

Abstract

Objectives: To determine the incidence of nephropathy induced by intravenous contrast on hospitalized patients submitted to computed tomography.

Methods: Retrospective cohort study that allocated 1,238 patients who were admitted to General Hospital of Caxias do Sul between August 1st, 2010 and April 30th, 2017 and who were submitted to computed tomography with or without contrast – iopromide (Bayer). The primary outcome was contrast-induced nephropathy, with both the criteria. The old (absolute increase on SCr ≥0.5 mg/dL or relative ≥25% during 48-72h after administration) and the new, based on KDIGO (absolute increase on SCr ≥0.3 mg/dL or relative ≥50% during 2–7 days after administration). The association factors consisted of the association between contrast-induced nephropathy and the following variables: female, elderly, diabetes mellitus, cardiovascular disease, infection, chronic kidney disease, association between contrast volume and serum creatinine variation, association between contrast-induced nephropathy and preventive measures.

Results: The incidence of general acute kidney injury was 11.52%. Univariate logistic regression demonstrated significance related to the absolute increase in SCr ≥0.5 mg/dL (OR 5.4 [CI: 95%: 1.45-20.15], P=0.02) in the wake of a computed tomography exam. Multivariate logistic regression initially found an association with an absolute increase in serum creatinine ≥0.3 mg/dL with elderly people (OR 1.77 [CI: 95%: 1.01-3.11], P=0.05), but the association did not keep when corrected the P. We found no association between acute kidney injury and the risk factors.

Conclusions: No criteria were found for contrast-induced nephropathy after computed tomography or association of acute kidney injury with classic risk factors.

Keywords

Contrast-induced nephropathy (CIN); acute kidney injury (AKI); serum creatinine (SCr); computed tomography (CT); intravenous (IV).

Introduction

Intravenous (IV) iodinated contrasts are ordinarily applied during computed tomography (CT) exams due to their capacity of increasing diagnostic accuracy[1]. However, this use is not risk-free, because iodinated contrasts present toxicity that can cause kidney dysfunction[2]. Medical societies defined Contrast-Induced Nephropathy (CIN), initially, in the following criteria: the absolute increase on serum creatinine (SCr) ≥0.5 mg/dL or relative ≥25% between 48 and 72 hours after administration[3-5]. Later, after the KDIGO study, the concept for CIN was changed to the absolute elevation of SCr ≥0.3 mg/dL or relative ≥50% between 2 and 7 days after the exam[6].

CIN pathogenesis on animal models consists of a classic case of acute tubular necrosis (ATN), secondary to vasoconstriction, which is associated with medullar hypoxemia and, thereafter, to tubular cells cytotoxicity. Vascular damage occurs due to the reduction of nitric oxide levels and increase in endotheline and adenosine in the bloodstream[7,8].

As to epidemiology, CIN is recognized as the third biggest cause of acute kidney injury (AKI) on hospitalized patients, with average incidence of 11%[9]. This frequency, however, can vary between 1% and 50%, according to the population characteristics and the use of arterial or venous contrast[10-16]. In the case of patients that are administered venous contrast only, the incidence of CIN can vary from 2,5% to 12%[12-16].

The classic risk factors to CIN are highlighted as: female, elderly, diabetes mellitus (DM), cardiovascular disease (CVD), infectious processes, chronic kidney disease (CKD), use of nephrotoxic drugs, among others[6,17-21].

Reviewing the available literature, it can be observed that most studies deal with CIN after the application of arterial contrast, and few articles relating CIN to venous contrast are available. In view of the small number of intravenous contrast studies[1,21-28], we have carried out a study to evaluate the relationship of venous contrast after CT with CIN in hospitalized patients and verify the relation with classic risk factors.

Methods

This was an retrospective cohort, single center study, performed between August 1st, 2010 and April 30th, 2017, conducted at General Hospital of Caxias do Sul, encompassing a population of 1.5 million people. It was approved by the Ethic and Research Committee of University of Caxias do Sul, protocol no. 1880766, and the authors signed the Confidentiality and Secrecy Term for data obtainment from the institution's electronic medical records. There is no funding source for the study.

In the period from August 1st, 2010, and April 30th, 2017, 129,205 patients were hospitalized in the institution, and 5,159 medical records of patients submitted to CT were analyzed. The amount of patients who did not fulfill the inclusion criteria was 3,921 patients, and 1,238 were validated, which amounts to 24% of the analyzed overall (Figure 1). Among the included, 51.85% were submitted to contrast CT and 48.15% to non-contrast CT according to institutional decision.

The SCr was tracked by isotope dilution mass spectrometry (IDMS). All patients submitted to contrast followed the institutional protocol, receiving an IV infusion of iopromide (Bayer) with concentration 623 mg/mL (equivalent to 300 mg/mL iodine), with osmolality 0.59 Osm/kg H_2O, non-ionic.

Inclusion criteria for this study were (Figure 1): patients admitted to the hospital and submitted to CT with or without contrast; age ≥18 years on the date of CT; basal and at 48 hours after CT SCr collection; kidney function stable during the three months previous to the exam and basal SCr between 0.4 and 4.0 mg/dL. Stable kidney function was defined as the non-occurrence of: absolute increase in SCr ≥0.3 mg/dL over a 48-hour period, SCr increase ≥1.5-fold baseline within 7 days or diuresis <0.5 mL/kg/h for a period of 6 hours[(2)].

Exclusion criteria were (Figure 1): patients on dialytic treatment; radiological contrast use on the 48 hours previous to the exam.

To check the proposed relations, the explanatory variables analyzed were: age, gender, elder (≥65 years old), DM (Guidelines of the Brazilian Diabetes Society), CVD (NYHA Criteria), infection (Sepsis criteria), CKD (KDIGO 2012), obesity (IMC ≥30 kg/m^2), previous use of nephrotoxic drugs (inhibitors of the angiotensin conversion enzyme, angiotensin receptor blockers, non-steroidal anti-inflammatory,

aminoglycosides, among others) and preventive measures (crystalloid solution, sodium bicarbonate or n-acetylcysteine).

The primary outcome was the incidence of CIN. The criteria for AKI were: SCr variation 48h after realization of CT: 0.3 mg/dL or 50%; 0.5 mg/dL or 25%.

The association factors were: association between CIN and the already listed risk factors; association between the contrast volume and the SCr variation; reduction of CIN when preventive measures are adopted.

Statistical modeling

Continuous variables were expressed as average and standard deviation (SD). Categorical variables were presented as absolute values and percentage. Differences in proportions were assessed by Chi-square test, when indicated. Normality of the distribution of continuous variables was calculated by the Shapiro-Wilk test. Student's t tests were used for the comparison of continuous variables. The variables that differed between patients with and without CIN were analyzed by logistic regression using the Stepwise Backward method with statistical use of Wald as a possible association with the occurrence of CIN. Odds Ratio (OR) and their respective confidence intervals (95% CI) were presented to quantify the effects.

For analysis, the software R for Windows (version 3.3.2, R-Cran project, http://cran.r-project.org/) was used. We aimed to correct probable bias of information by robust selection and statistical analysis.

Results

The sample consisted of 1,238 patients, with the demographic, clinical and laboratory characteristics are presented in Table 1. There were 723 (58.41%) men and 515 (41.59%) women, with average age of 60.8±16.1 years, being higher on patients who were submitted to non-contrast CT (63.5 vs 59.3; P <0.01). There were fewer elderly patients (40.03% versus 54.53%, P <0.01), with DM (16.66% versus 23.82%, P = 0.013) and with CKD (25.54% versus 46.30%, P <0.01) in the group of patients who were submitted to contrast CT compared to those who were submitted to non-contrast CT. More preventive measures were performed (19.62% versus 11.74%; P <0.01) in the group of patients who were submitted to contrast CT

compared to those who were submitted to non-contrast CT. The other comparisons of variables related to the characteristics of patients exposed to contrast medium did not differ and are presented in Table 1.

Furthermore, regarding the possible association between contrast volume and SCr variation, 943 patients (76.17%) had no height measuring recorded, rendering impossible the analysis of the body surface area, and, thus, the verification of any association. At baseline SCr (0.8 mg/dL versus 1.1 mg/dL, P <0.01) and at 48 hours (0.8 mg/dL versus 1.05 mg/dL, P <0.01) was lower in the group of patients who were submitted to contrast CT compared to those who were submitted to non-contrast CT (Table 1).

After CT was performed, using a simple logistics regression, we found statistical significance related to the absolute increase on SCr ≥0.5 mg/dL (OR 5.4 [IC:95%:1.45-20.15], P=0.02). However, we found no statistical significance with the absolute increase of SCr ≥0.3 mg/dL and relative of SCr ≥25% and 50% (Table 2). On patients that were submitted to contrast CT, applying the KDIGO criteria for CIN, we verified an incidence of 11.52% on absolute variation and 7.47% on relative variation of SCr (Table 3).

We also performed the logistic multivariable regression with the explanatory factors related to the absolute increase of SCr ≥0.3 mg/dL and 0.5 mg/dL, as well as the relative increase of SCr ≥25% and 50% (Table 4). Another point worth noting is the multivariate analysis. It revealed only a positive association between SCr ≥0.3 mg/dL with elderly people (OR 1.77 [IC:95%:1.01-3.11], P=0.05) (Table 4), but the association did not keep when corrected the P. Furthermore, no other positive associations were presented of AKI in multivariate analysis.

Discussion

Initially, publications on CIN inferred a strong association between arterial contrast use for angiographic procedures and AKI. Due to this relevant incidence, investigations aimed to demonstrate the association of AKI with CT exams performed with IV[1,21-28].

In this study, we hoped to find some incidence of CIN on the 48 hours after the contrast injection. However, we did not perceive relevant increase of SCr on patients submitted to procedures with the use of IV. This result is corroborated by various

evidences on recent medical scientific literature, with unexpressive incidence of CIN after IV[1,24-27]. Besides, these researches concluded that IV is not a risk factor for AKI, even on patients with compromised kidney function or with comorbidities that may predispose nephrotoxicity[1,25-27]. Even so, some investigation concluded that IV is a risk factor for patients with CKD and DM[13,15,16,28].

In our study the incidence of AKI found, which had no association with IV use, was 11.52%, using KDIGO criteria. In two meta-analysis that selected 42 and 40 articles the incidence found was 4.96% e 6.4% respectively[15-16]. In another retrospective study with 126 patients with glomerular filtration rate (GFR) 60 mL/min/1.73m^2, there was 5.1% incidence of CIN[14]. Already in an heterogeneous and prospective cohort of patients submitted to CT with contrast, 11% of them developed AKI consistent with CIN[2].

Initially, by using simple logistic regression, statistical significance of the absolute increase of SCr ≥0.5 mg/dL after CT was found. However, this association disappeared by using a multivariable logistic regression, when the use of contrast and risk factors were evaluated. The positive association between SCr ≥0.3 mg/dL with elderly on the multivariate analysis had no relevance, because it was not maintained when corrected by P. Other associations with classic risk factors were not found, corroborating, thus, various evidences presented on the current scientific literature[1,21,24-27].

For the merits of this study, being the same retrospective, we verified the absence of relevant information in the data collection in the medical records. As a result, we were unable to analyze the association between contrast volume and SCr variation, since there was no patient height registered on most of the medical records. This situation points to a need to institute the measurement and register of this data on medical records in the form of a routine. Even so, without this care being taken, and by administering a contrast dose not adjusted to the patient's specific needs, there was no association between contrast use and AKI, reinforcing, thus, the hypothesis that the use of contrast during CT really does not induce CIN.

It is also worth mentioning the fact that a guideline of the Royal College of Radiology recommends the infusion of a 0.9% saline solution for patients with risk of CIN[29]. In this perspective, a recent clinical random study did not demonstrate reduction on the incidence of CIN with IV hydration for patients with GFR between 30

and 59 mL/min/1.73m², suggesting that IV rehydration should be reserved for patients with TFG < 30 mL/min/1,73m$^{2(30)}$. Another study suggests that prevention measures of CIN were not effective on the evaluated population$^{(13)}$. In our investigation, there was no finding of CIN. So, the association between preventive measures as a protective factor for AKI after IV use could not be evaluated.

Otherwise, there was an increase on SCr measurements at 48 hours after IV application on both populations (CT with and without contrast). It was verified that this data was not associated neither with the contrast nor to the classic risk factors studied. So, it will necessary to perform more investigations in an attempt to identify why inpatients of General Hospital of Caxias do Sul submitted to CT had an increase on SCr 48 hours after the exam in our, as well as in other institutions. The results found will be valid for our hospital, but they cannot be generalized to other institutions.

In our study, the incidence of AKI was 11.52%. Besides that, no criteria for CIN were found in CT or the association between AKI with classic risk factors. This corroborates the current literature in which the incidence of CIN in EV contrast is low.

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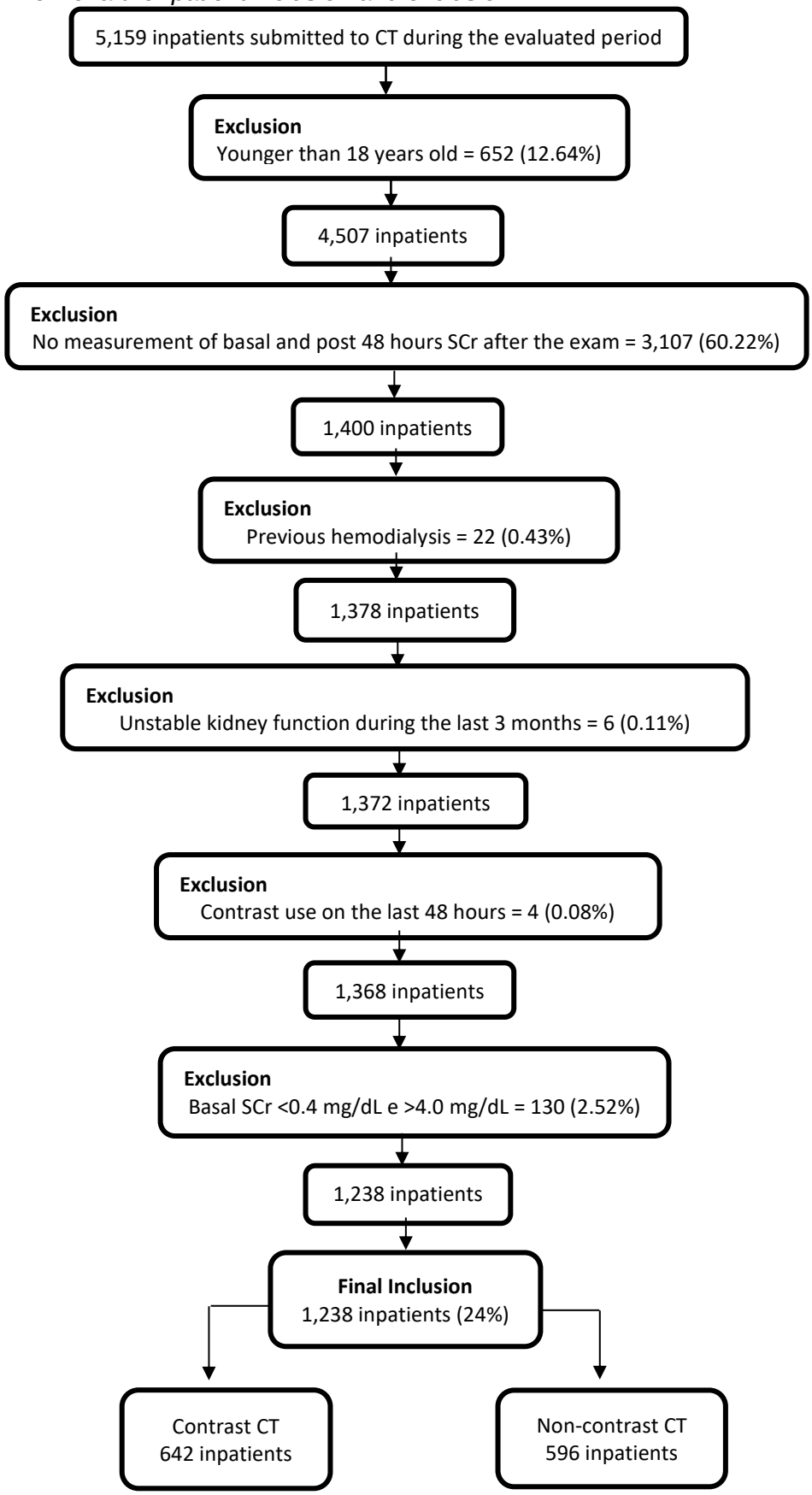
Figure 1: Flow chart for patient inclusion and exclusion



CT = Computed tomography; SCr = Serum creatinine.

Table 1: Demographic, clinical and laboratorial data of patients exposed or not to contrast

	Total CT (N=1,238)	**Contrast CT (N=642)**	**Non contrast CT (N=596)**	***P***
Age (years±SD)	60.8 ± 16.1	59.3 ± 15.8	63.5 ± 16.08	<0.01
Female	515 (41.59)	248 (38.62)	267 (44.79)	NS
Advanced age	582 (47.01)	257 (40.03)	325 (54.53)	<0.01
Diabetes	249 (20.11)	107 (16.66)	142 (23.82)	0.013
Cardiovascular disease	109 (8.80)	36 (5.60)	73 (12.24)	NS
Infection	201 (16.23)	107 (16.66)	94 (15.77)	NS
Chronic kidney disease	440 (35.54)	164 (25.54)	276 (46.30)	<0.01
Obesity	95 (7.67)	52 (8.09)	43 (7.21)	NS
Previous nephrotoxic drugs use	712 (57.51)	366 (57.01)	348 (58.38)	NS
Preventive measures	196 (15.83)	126 (19.62)	70 (11.74)	<0.01
Basal SCr	median 1.27	median 0.8	median 1.1	<0.01
(mg/dL)	(IQR: 0.6; 1.4)	(IQR: 0.62; 1.2)	(IQR: 0.8; 1.6)	
48h SCr	median 1.30	median 0.8	median 1.05	<0.01
(mg/dL)	(IQR: 0.6; 1.4)	(IQR: 0.6; 1.1)	(IQR: 0.7; 1.6)	

CT = Computed tomography; SD = Standard deviation; SCr = Serum creatinine; IQR = Interquartile range; NS = Non-significant. Values are expressed as n (%) unless stated otherwise.

Table 2: Increase of serum creatinine after computed tomography

	OR	95% CI	*P*
SCr increase ≥0.3 mg/dL	1.15	0.62 - 2.15	0.65
SCr increase ≥0.5 mg/dL	5.4	1.45 - 20.15	0.02
SCr increase ≥25%	2.6	0.90 - 7.4	0.07
SCr increase ≥50%	3.5	0.92 - 13.1	0.06

SCr = Serum creatinine; OR = Odds ratio; CI = Confidence interval.

Table 3: SCr incidence of acute kidney injury after computed tomography

	Total (N=1,238)	Contrast CT (N=642)	Non-contrast CT (N=596)
SCr increase ≥0.3 mg/dL	197 (15.91)	74 (11.52)	123 (20.63)
SCr increase ≥0.5 mg/dL	128 (10.33)	41 (6.38)	87 (14.59)
SCr increase ≥25%	229 (18.49)	102 (15.88)	127 (21.30)
SCr increase ≥50%	113 (9.12)	48 (7.47)	65 (10.90)

CT = Computed tomography; SCr = Serum creatinine.

Table 4: Risk factors for acute kidney injury after computed tomography

Risk factors	OR	95% CI	*P*
SCr increase ≥0.3 mg/dL			
Contrast use	0.72	0.42-1.24	0.24
Female	1.22	0.70-2.11	0.48
Advanced age	1.77	1.01-3.11	0.05
DM	0.63	0.31-1.28	0.20
CVD	0.78	0.25-2.41	0.67
Infection	1.40	0.72-2.72	0.32
CKD	1.25	0.69-2.24	0.46
Obesity	1.26	0.64-2.47	0.51
Previous nephrotoxic drug use	0.88	0.51-1.51	0.64
SCr increase ≥0.5 mg/dL			
Contrast use	0.82	0.41-1.64	0.58
Female	1.20	0.59-2.45	0.61
Advanced age	1.37	0.66-2.83	0.40
DM	0.48	0.18-1.26	0.14
CVD	0.61	0.13-2.81	0.53
Infection	1.61	0.72-3.61	0.25
CKD	1.57	0.74-3.34	0.24
Obesity	2.18	0.98-4.83	0.06
Previous nephrotoxic drug use	1.14	0.57-2.30	0.71
SCr increase ≥25%			
Contrast use	1.08	0.65-1.79	0.76
Female	1.28	0.76-2.14	0.35
Advanced age	1.38	0.82-2.34	0.23
DM	0.76	0.39-1.48	0.42
CVD	1.04	0.37-2.91	0.94
Infection	1.65	0.90-3.03	0.10
CKD	0.69	0.38-1.24	0.22
Obesity	1.08	0.57-2.04	0.82
Previous nephrotoxic drug use	0.87	0.53-1.43	0.59
SCr increase ≥50%			
Contrast use	0.87	0.43-1.78	0.71
Female	0.87	0.43-1.77	0.70
Advanced age	1.53	0.74-3.19	0.25
DM	0.59	0.22-1.55	0.28
CVD	0.34	0.04-2.63	0.30
Infection	1.50	0.64-3.48	0.35
CKD	0.72	0.32-1.66	0.45
Obesity	2.12	0.96-4.68	0.06
Previous nephrotoxic drug use	1.13	0.55-2.29	0.74

SCr = Serum creatinine; OR = Odds ratio; CI = Confidence Interval; DM = Diabetes mellitus; CVD = Cardiovascular disease; CKD = Chronic kidney disease.

4 CONSIDERAÇÕES FINAIS E PERSPECTIVAS

Em nosso estudo, a incidência de LRA foi de 11,52%. Entretanto, não foram encontrados critérios para NIC em TC, nem associação de LRA com os fatores de risco clássicos. Isso corrobora a literatura atual em que a incidência de NIC em contraste EV é baixa.

Também não foi possível analisar a associação entre o volume de contraste e a variação da creatinina sérica (CrS) por não haver a mensuração de altura dos pacientes na grande maioria dos prontuários. Em contrapartida, mesmo não havendo esse cuidado e administrando-se uma dose de contraste não ajustada a cada indivíduo, não houve associação entre contraste e LRA, reforçando a hipótese de que o uso de contraste em TC realmente não desenvolve NIC.

Da mesma forma, a associação de medidas preventivas como fator protetor para LRA após o uso de contraste EV não pôde ser avaliada, pois não houve a detecção de NIC.

Os achados elencados acima demonstram que não houve relação de LRA com a aplicação de contraste EV. Em contrapartida, houve um aumento da CrS na mensuração em 48 horas após a realização do contraste EV em ambas as populações (TC com e sem contraste). Foi verificado que esse dado não estava associado nem ao contraste nem aos fatores de risco clássicos estudados. Dessa forma, faz-se necessária a realização de mais investigações na tentativa de identificar o porquê de pacientes internados em nosso hospital e submetidos à TC aumentar os níveis de CrS 48 horas após a realização do exame.

5 ANEXOS

UCS UNIVERSIDADE DE CAXIAS DO SUL

Leandro Tasso <ltasso@ucs.br>

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1 mensagem

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23-Apr-2018

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Editor

INCIDÊNCIA DE LESÃO RENAL AGUDA E O USO DE CONTRASTE ENDOVENOSO – ESTUDO RETROSPECTIVO

Thyago Anzolin Coser

Dissertação de Mestrado submetida à Banca Examinadora designada pelo Colegiado do Programa de Pós-Graduação em Ciências da Saúde da Universidade de Caxias do Sul, como parte dos requisitos necessários para a obtenção do título de Mestre em Ciências da Saúde, Linha de Pesquisa: Farmacologia e Biomarcadores

Caxias do Sul, 22 de maio de 2018.

Banca Examinadora:

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