Prediction and severity of hospital-acquired acute kidney injury in patients with and without hypertension and type 2 diabetes

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Abstract Context: The impact and prognosis of acute kidney injury (AKI) change considerably depending on the severity, clinical setting, comorbid factors, and also Geographic location.

Aims: To assess the severity and the risk factors of hospital-acquired AKI (HA-AKI) in diabetic and hypertensive patients.

Settings and Design: A prospective cohort study was conducted in 2019 with 88 hypertensive and diabetic in hospitalized patients of Distrito Federal.

Materials and Methods: A structured questionnaire and Charlson's comorbidity index (CCI) were the data collection instruments.

Statistical Analysis: The data were analyzed using descriptive and inferential methods.

Results: Hypertensive and diabetic patients were older (70 [62–76] years old, P = 0.001), with a body mass index indicating overweight (26.9 [24.0–31.1] kg/m², P = 0.01). AKI predominated among the hypertensive and diabetic patients (30 [52.6%]), and with higher severity stages (Kidney Disease Improving Global Outcomes 2 and 3) (22 [38.6%]). Hypertensive and diabetic patients presented more severity (Charlson >3, P = 0.03), suffered from kidney injury more frequently (30 [52.6%]), and with more severe stages (kidney injury or failure) (22 [38.6%]). Heart disease ([odds ratio (OR) 17.94, confidence interval (Cl) 2.23–144.44], P = 0.007) and older age ([OR 1.05, Cl 1.01–1.09], P = 0.009) were independent risk factors for predisposition to kidney injury in patients with hypertension and diabetes.

Conclusions: The hypertensive and diabetic patients were older, with a CCl >3, and evolved to more severe AKI. Heart disease and older age contributed to HA-AKI. Delays in identifying risk factors may predispose to more severe impairments. Risk assessments support early identification and can encourage professionals in directing, decision-making, and care management.

Keywords: Acute kidney injury, Diabetes mellitus, Hypertension, Multimorbidity

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INTRODUCTION

Kidney complications in the contemporary scenario are frequently secondary to chronic noncommunicable diseases (CNCDs), and therefore represent a concern for public health since, although described as controllable, they are generally not curable.^[1]

The incidence of acute kidney injury (AKI) remains with an increasing trend affecting up to one out of five hospitalized adults worldwide. This syndrome is characterized by a continuum of injury, which requires a careful clinical history, careful interpretation of laboratory and imaging tests, review of medical records, and complete physical examination so that potential changes in renal functioning^[2-4] can be identified and prevented in the clinical practice before there is loss of excretory renal function, identified by laboratory tests, such as serum creatinine.

AKI is a multifactorial syndrome associated with clinical complications. Diabetes mellitus (DM) and arterial hypertension are CNCDs with potential risk for renal impairment and increased mortality, due to microvascular lesions, even when treated, reflecting the severity of these diseases, which are generally common in multimorbid conditions.^[3,5]

AKI is characterized by a sudden decrease in the renal function and by an increase in serum creatinine of at least 0.3 mg/dL in 48 h and a reduction in urinary output^[6] and, when acquired in the hospital environment, it manifests after 24 h of admission as hospital-acquired AKI (HA-AKI).^[7]

HA-AKI is predominant in large city centers. Especially Latin America, followed by Southeast Asia, presents the highest percentage of AKI in the world.^[8] In this scenario, hemodynamic instability has shown a significant association with AKI development and progression, generally due to the severity of the disease and to the patient's comorbidities.^[9-11]

The impact and prognosis of AKI vary considerably depending on severity, clinical setting, comorbid factors, and also geographic location.^[12] Therefore, early detection can attenuate or neutralize the worst outcomes^[13] and reduce the severity, duration, and frequency of HA-AKI, which show to be unfavorable to the patients' recovery.

The sudden onset and evolutionary severity of AKI in the hospital setting associated with the deficit in early recognition and management of HA-AKI, especially in patients with different comorbidities, generate the need to identify risk and severity predictors as indicators that can more precisely contribute for clinical prediction and individualized guidance of preventive strategies, including better therapeutic planning.^[2]

Given the above, the objective of this study is to assess the severity and risk factors of HA-AKI in diabetic and hypertensive patients.

MATERIALS AND METHODS

Research design and setting

A prospective cohort study was conducted in the one medical clinic unit of a public and tertiary-care hospital in the west region of Brasilia-Distrito Federal, Brasil.

Data collection was performed through a structured questionnaire prepared by the researchers consisting of the variables of interest, namely gender, age, race, weight, body mass index (BMI), hospitalization time, comorbidities, creatinine, urea, hemoglobin, serum sodium and potassium, blood pressure, glomerular filtration rate (GFR), and patient's severity as measured by Charlson's comorbidity index (CCI).

CCI establishes the severity of the patient's condition by adding up the weights from 1 to 6 attributed to 19 clinical conditions, where 6 indicates more severity and 1 less severity. This stratification allowed categorizing the patients according to the score obtained, where those with a score of one (1) were categorized as ill, of two (2) as moderately ill, from three (3) to five (5) as severely ill, and of six (6) as dying.^[14]

Patients aged over 18 years old, with a medical diagnosis of arterial hypertension and type 2 DM recorded in an electronic medical chart were included; as well as those with a hospitalization period of more than 48 h in one medical clinic with medical wards, and sustained change in serum creatinine ≥ 0.3 mg/dL for at least 48 h when compared to baseline. The exclusion criteria were GFR <30 mL/min/1.73, need for renal replacement therapy, and performance of surgical procedures.

Baseline creatinine was that obtained during the first hospitalization week in the medical clinic sector.^[15] Serum creatinine was monitored and its parameters were evaluated according to the recommendations of the Kidney Disease Improving Global Outcomes (KDIGO) creatinine criteria for evaluation of the evolutive renal profile and staging of renal impairment^[6] [Table 1]. The urinary output criteria

Table 1: Kidney Disease Improving Global Outcomes classification (creatinine criterion)

Stage	Serum creatinine
1	1.5–1.9 times baseline creatinine or >0.3 mg/dL
2	2.0–2.9 times baseline serum creatinine
3	3.0 times baseline or elevation of baseline creatinine
	to >4 mg/dl or initiation of RRT or in patients <18 years
	old, drop in GFR estimated to <35 ml/min/1.73 m ²

RRT: Renal replacement therapy, GFR: Glomerular filtration rate

from the KDIGO classification were not used in this study due to the scarcity of records regarding urinary volume.

HA-AKI was considered in patients with a persistent change in serum creatinine after 24 h of hospitalization in the medical clinic.^[16]

Sample size and sampling procedure

Sample calculation considered 80% power and was obtained by the formula below:^[17]

$$N = 2 \frac{\left[z_{\alpha} 2(\overline{pq})^{\frac{1}{2}} + z_{\beta}(p1q1 + p2q2)^{\frac{1}{2}}\right]^{2} (1 + (n-1)\rho)}{n(p1 - p2)^{2}}$$

Where p1 = 0.4 and p2 = 0.65 are the proportion of patients that had CCI > 3 at two distinct moments of the follow-up; q1 = 1 - p1; q2 = 1 - p2; $\overline{p} = (p1 + p2)/2$; $\overline{q} = 1 - \overline{p}$; $\rho = 0.5$ is the intraclass correlation; n is the number of measurements on the same individual; $z\alpha = 5\%$ is the percentile of the normal distribution corresponding to the significance level; $z\beta$ is the percentile of the normal distribution corresponding to the power of the test.

The population consisted in hospitalized patients with systemic arterial hypertension (SAH) and DM. Five hundred and twelve patients were evaluated from January to December 2019 and 88 patients were selected. Monitoring was carried out for 6 months.

Data collection tools and procedure

Identification of the patients with elevated serum creatinine, through weekly consultations of the electronic medical records of patients, admitted to the medical clinic and verification of the renal biochemical profile (serum creatinine and estimated creatinine clearance by the chronic kidney disease (CKD) Epidemiology Collaboration - CKD Epi, as recommended by the KDIGO, in addition to the collection of the variables of interest through consultation of the electronic medical record.

The results of the laboratory tests were monitored for 6 months, from the identification of changes in serum creatinine, to trace the clinical and renal evolutive profile and the outcome.

At hospital discharge, requests for laboratory tests to maintain the biochemical dosage of serum creatinine, urea, sodium, potassium, and hemoglobin in the laboratory of a basic health unit and control the evolutive profile of the renal function were handed into the patients or legal guardians.

After hospital discharge, the researcher forwarded alerts via telephone contacts to remind the patient about the collection date corresponding to the laboratory tests. Follow-up and monitoring of the results were carried out by consulting the patient's medical record, and the identification of changes in the laboratory parameters led to referral to the nursing or medical consultation for follow-up and guidance.

The hemodynamics and laboratory reference parameters followed the protocol of the health secretariat of Distrito federal, namely mean arterial pressure (MAP) altered when ≤ 60 or ≥ 100 mmHg, diastolic blood pressure = 80–89 mmHg, serum creatinine from 0.8 to 1.4 mg/dL, and hemoglobin from 13.0 to 17.0 g/dL.^[18]

Data analyses

The descriptive analysis was performed by calculating the summary (mean and median) and dispersion (standard deviation and 25-75 percentiles) measures. For qualitative variables (categorical), frequency distribution was calculated. The normality of the study was tested by the Kolmogorov-Smirnov test and then, nonparametric tests were applied, such as the Mann-Whitney test for continuous variables and the Chi-square and Fisher's tests for categorical variables. Sensitivity analyses were performed to exclude missing values but, as no statistically significant changes were identified, it was decided not to exclude any observation. For the multivariate analysis, the backward method was adopted through logistic regression for the selection of the variables with the calculation of the odds ratio (OR) and the respective 95% confidence interval (95% CI). For the statistical analyses, a two-tailed P < 0.05was considered as evidence of statistical significance.

Ethical consideration

This study was approved by the Research Ethics Committee of the Health Sciences Teaching and Research Foundation/State Health Secretariat, CAAE: 51576215.8.0000.5553, according to Resolution 466/2012.

RESULTS

A total of 88 patients were followed, predominantly male 45 (51.1%), older adults (64 ± 14 years old), brown-skinned 20 (22.7%), single 12 (13.6%) with a BMI indicating overweight ($26.8 \pm 7.5 \text{ kg/m}^2$), high severity in at least

18 (20.5%) justified by CCI = 6, and hospitalized in a nonintensive care clinic. The majority was on spontaneous ventilation (ambient air), 49 (55.7%), and 23 (26.1%) on oxygen therapy by tracheostomy.

The median hospitalization time was predominantly prolonged, around 35 days. Type 2 DM and SAH affected more than half of the patients (n = 57; 64.8%), 32 (36.4%) were affected by respiratory diseases, and 26 (29.5%), by heart diseases. It is noteworthy that pathologies such as SAH and DM affected the oldest individuals (70 [62–76] years old, P = 0.001) and those who were overweight (26.9 [24.0–31.1] kg/m², P = 0.01).

Hospital discharge was the main clinical outcome (58; 65.9%). Mortality during hospitalization affected 15 (17.0%) patients and, after discharge, it affected 20 (22.7%) patients. In most of the patients, 72 (81.8%), antibiotic therapy was administered at a ratio of 2.5 ± 1.2 per patient. Diuretics were commonly administered medications, with the loop being proportionally superior, 66 (75.0%), to potassium sparing, 25 (28.4%). The use of antibiotics (46 [80.7%, P = 0.7]) and diuretics (45 [78.9%, P = 0.6]) was more frequent in patients with SAH and DM, although with no statistical significance [Table 2].

HA-AKI, as well as more severe AKI (KDIGO 2 or 3-kidney injury and failure), predominated in hypertensive and diabetic patients, with 18 (58.1%) and 22 (38.6%), respectively. Patients with DM and SAH had lower baseline creatinine clearance when compared to the group without these pathologies, 62 (47–85) mL/min versus 87 (57–103) mL/min; P = 0.052, as well as more altered MAP, 46 (82.1%) versus 17 (60.7%); P = 0.04. The group with SAH and type 2 DM showed a significant predisposition to heart diseases (P < 0.001) and liver disease (P = 0.001), as well greater severity by CCI >3 (P = 0.03) [Table 3].

Heart disease was an independent risk factor in patients with SAH and type 2 DM and was shown to increase approximately 18 times the chance of HA-AKI (OR 17.94, CI 2.23–144.44), P = 0.007. Older age also showed to be a predictor, although with less intensity (OR 1.05, IC 1.01–1.09), P = 0.009 [Table 4].

DISCUSSION

The findings showed that the hypertensive and diabetic patients who evolved with HA-AKI after hospitalization in a nonintensive setting presented greater severity as measured by CCI >3, P = 0.03, in addition to multimorbidity such as heart diseases (P < 0.001), liver diseases (P = 0.001), and overweight (P = 0.01). More severe renal impairment (KDIGO 2 or 3) was also found in the group with SAH and DM. AKI is a pathology generally associated with a high morbidity burden and consequent increase in costs and mortality;^[19] it can even cause permanent changes in the renal function and development of CKD.^[4]

The hypertensive and diabetic patients with HA-AKI remained hospitalized for a long period, with a median of 38 days, which is consistent with the findings among the 1,285,045 participants from eight cohort studies of a meta-analysis.^[20] Even the GFR of the hypertensive and diabetic group was lower, 62 (47–85) mL/min when compared to the group without hypertension and diabetes, 87 (57–103) mL/min, P = 0.052.

In this context, regardless of the origin of the disease or the patient's geographical location, given the absence

Variables	Patients without SAH and DM (n=31)		Patients with SAH and DM (<i>n</i> =57)		Р
	n (%)	Median (25-75)	n (%)	Median (25-75)	
Demographic					
Age (years old)		55 (43-68)		70 (62–76)	0.001
Male gender	19 (61.3)		26 (45.6)		0.2
BMI (kg/m ²)	, , , , , , , , , , , , , , , , , , ,	23.8 (20.7-26.7)		26.9 (24.0-31.1)	0.01
White race	3 (25.0)		7 (25.9)		0.9
Antibiotic					
Use of ATB	26 (83.9)		46 (80.7)		0.7
Number of ATB	, , , , , , , , , , , , , , , , , , ,	2 (2-3)	, , , , , , , , , , , , , , , , , , ,	3 (1-4)	0.2
Diuretics					
Use of diuretic	23 (74.2)		45 (78.9)		0.6
Loop diuretic	22 (71.0)		44 (77.2)		0.5
Potassium sparing	8 (25.8)		17 (29.8)		0.7
Thiazides	3 (9.7)		6 (10.5)		0.9

Table 2: Relationship of patients with and without a history of arterial hypertension and diabetes mellitus with the clinical variables. Brasília, Brazil, 2019

Chi-square test, Mann-Whitney test, Fisher's exact test. SAH: Systemic arterial hypertension, DM: Diabetes mellitus, BMI: Body mass index, ATB: Antibiotic

	Patients without SAH and DM (n=31)		Patients with SAH and DM (n=57)		Р
	n (%)	Median (25-75)	n (%)	Median (25-75)	
Scores					
CCI>3	10 (32.3)		32 (56.1)		0.03
Mechanical ventilation					
Use of TCT	7 (22.6)		16 (28.1)		0.6
Oxygen therapy					
O ₂ mask	4 (12.9)		5 (8.8)		0.7
Laboratory variables					
Hemoglobin (g/dL)		10.4 (8.7-11.9)		10.8 (9.6-12.7)	0.2
Hemodynamics					
Altered MAP	17 (60.7)		46 (82.1)		0.04
Comorbidities					
Respiratory	9 (29.0)		23 (40.4)		0.3
Heart disease	1 (3.2)		25 (43.9)		< 0.001
Hepatopathy	11 (35.5)		4 (7.0)		0.001
Others					
KDIGO 2 or 3 (kidney injury or failure)	11 (35.5)		22 (38.6)		0.8
HA-AKI	18 (58.1)		30 (52.6)		0.6
Baseline CrCl (mL/min)		87 (57–103)		62 (47-85)	0.052
Death	3 (9.7)		12 (21.8)		0.1
Death after medical clinic	13 (43.3)		23 (41.8)		0.9
Blood transfusion	3 (10.0)		8 (14.0)		0.7
Days of hospitalization (days)		33 (18–75)		38 (22–60)	0.9

Table 3: Relationship of patients with and without arterial hypertension and diabetes mellitus with variables related to severity. Brasília, Brazil, 2019

Chi-square test, Mann–Whitney test, Fisher's exact test. CCI: Charlson's comorbidity index, TCT: Tracheostomy, 0₂: Oxygen, MAP: Mean arterial pressure, CrCI: Creatinine clearance, Ha-AKI: Hospital-acquired acute kidney injury, KDIGO: Kidney Disease Improving Global Outcomes

Table 4: Multivariate analysis of risk factors for diabetic and hypertensive patients with hospital-acquired acute kidney injury. Brasília, Brazil, 2019

Variables	Coefficients	Р	OR	95% CI (OR)	
				Lower	Upper
Age	0.050	0.009	1.05	1.01	1.09
Heart disease	2.887	0.007	17.94	2.23	144.44
Constant	-2.982	0.013	0.05		

Chi-square=27.44, degrees of freedom of the model=2, P<0.001, n=88. OR: Odds ratio, CI: Confidence interval

of a successful pharmacological therapy to treat AKI, early identification of at-risk patients is fundamental to preventing the progression of AKI to CKD, characterized by the irreversible loss of nephrons.^[4]

It is noteworthy that the acute kidney disease in the majority group of hypertensive and diabetic patients persisted predominantly during the 6-month follow-up period, which configures progression to CKD. According to the KDIGO classification, CKD is defined as an abnormality of the renal structure or function, represented by the change in serum creatinine and GFR, present for a period longer than 3 months.^[21]

Advanced age and heart diseases showed to be independent risk factors for HA-AKI in hypertensive and diabetic patients and therefore increased the chance of this syndrome occurring in the hospital setting (OR 1.05, CI 1.01–1.09), P = 0.009 and (OR 17.94, CI 2.23–144.44), P = 0.007, respectively. In addition to revealing at-risk patients, these findings reinforce the need to monitor serum creatinine and/or urinary output as predictive measures for AKI. A careful assessment of volume and hemodynamics in these patients can represent a differential to achieving renal recovery.^[4]

The incidence of AKI is increasing in individuals of different ages; however, in older adults, the greater predisposition can be related to frequent multimorbidity and renal senility.^[22,23] In the current study, the age of the patients with SAH and DM, especially, was 70 years old, of which 52.6% developed HA-AKI, with a median creatinine clearance of 62 mL/min. Consequently, mortality presented more incidence in this group when compared to the group without SAH and DM (21.8% vs. 9.7%). Deterioration of the renal function during hospitalization can be corroborated by different factors, such as advanced age, use of medications, cardiac function, and infection, in addition to underlying diseases such as SAH and DM.^[24]

In addition to aging, overweight (P = 0.01) has also been identified among hypertensive and diabetic patients with HA-AKI. They are both recognized as indicators for HA-AKI, as they exert an influence on diabetes control and on the precise assessment of renal function. Heart diseases stood out for increasing approximately 18 times the chances of AKI. Heart failure, for example, is characteristically a disease of advanced age and its decompensation, as well as of other heart diseases, can trigger hemodynamic changes and reduce blood supply to the renal tissue, causing AKI.^[22]

Targeted recognition of the predictors of HA-AKI by the multidisciplinary health team can improve the effectiveness of the treatment of underlying diseases and prevent or mitigate additional renal problems in the patients, particularly among those hospitalized or undergoing long-term treatments.^[25]

In relation to the clinical outcome in the nonintensive setting, a number of studies point to the association of comorbidities with HA-AKI as a reason for the increased mortality risk.^[26,27] The results of this study reveal that most of the patients were discharged (65.9%) and that 17.0% evolved to death. Even so, the more severe stages of HA-AKI identified especially among the diabetic and hypertensive patients, characterized by the more expressive predominance of KDIGO 2 or 3, greater number of comorbidities, use of antibiotics, and altered MAP, allows highlighting the importance of preventive measures, such as serial creatinine dosage in at-risk patients to avoid or mitigate the repercussions of this syndrome in the health system and to support early diagnosis.^[28]

The limitations of this study include its unicentric approach and its reduced sample size, which can impair the generalization of the results. The absence and inaccuracy of urinary volume records was also a condition that limited the assessment of the renal function's evolution process.

CONCLUSION

Hypertensive and diabetic patients in a nonintensive setting presented more severity in relation to the other patients, as they evolved to KDIGO 2 and 3 HA-AKI more frequently. The independent predictors for nonintensive HA-AKI in diabetic and hypertensive patients were age and heart disease, but heart disease significantly increased the chance of HA-AKI in a nonintensive environment.

Delays in identifying risk factors may predispose to more severe impairments. Risk assessments support early identification and can encourage professionals in directing, decision-making, and care management.

Conflicts of interest

There are no conflicts of interest.

Authors' contributions

Tayse Tâmara da Paixão Duarte: Contributed to the conception and design, data acquisition, data interpretation and approved the version to be published.

Amanda Viana Mota: Contributed to data acquisition, data interpretation and approved the version to be published.

Marcia Cristina da Silva Magro: Contributed to the conception and design, analysis and interpretation of data and approved the version to be published.

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REFERENCES

- Rasul FB, Kalmus O, Sarker M, Adib HI, Hossain MS, Hasan MZ, et al. Determinants of health seeking behavior for chronic non-communicable diseases and related out-of-pocket expenditure: Results from a cross-sectional survey in northern Bangladesh. J Health Popul Nutr 2019;38:48.
- Hodgson LE, Sarnowski A, Roderick PJ, Dimitrov BD, Venn RM, Forni LG. Systematic review of prognostic prediction models for acute kidney injury (AKI) in general hospital populations. BMJ Open 2017;7:e016591.
- Safadi S, Hommos MS, Enders FT, Lieske JC, Kashani KB. Risk factors for acute kidney injury in hospitalized non-critically ill patients: A population-based study. Mayo Clin Proc 2020;95:459-67.
- Gonsalez SR, Cortês AL, Silva RC, Lowe J, Prieto MC, Silva Lara LD. Acute kidney injury overview: From basic findings to new prevention and therapy strategies. Pharmacol Ther 2019;200:1-12.
- Regassa LD, Gete YK, Mekonnen FA. Time to acute kidney injury and its predictors among newly diagnosed type 2 diabetic patients at government hospitals in Harari Region, East Ethiopia. PLoS One 2019;14:e0215967.
- Kellum JA, Lameire N, Aspelin P, Barsoum RS, Burdmann EA, Goldstein SL, *et al.* Kidney disease: Improving global outcomes (KDIGO). Clinical practice guideline for acute kidney injury. Kidney Int 2012;2 Suppl 1:1-138.
- González GC, Hurtado M, Contreras K, García PK, Rodríguez P, Accini M, *et al.* Non-critical care hospital-acquired acute kidney injury. Analysis of 101 cases. Rev Med Chil 2018;146:1390-4.
- Hoste EA, Kellum JA, Selby NM, Zarbock A, Palevsky PM, Bagshaw SM, *et al.* Global epidemiology and outcomes of acute kidney injury. Nat Rev Nephrol 2018;14:607-25.
- Ostermann M, Liu K. Pathophysiology of AKI. Best Pract Res Clin Anaesthesiol 2017;31:305-14.
- Raimundo M, Crichton S, Syed Y, Martin JR, Beale R, Treacher D, et al. Low systemic oxygen delivery and BP and risk of progression of early AKI. Clin J Am Soc Nephrol 2015;10:1340-9.
- Post EH, Kellum JA, Bellomo R, Vincent JL. Renal perfusion in sepsis: From macro – To microcirculation. Kidney Int 2017;91:45-60.
- 12. Ostermann M, Joannidis M. Acute kidney injury 2016: Diagnosis and diagnostic workup. Crit Care 2016;20:299.
- Stanifer JW, Maro V, Egger J, Karia F, Thielman N, Turner EL, *et al.* The epidemiology of chronic kidney disease in Northern Tanzania: A population-based survey. PLoS One 2015;10:e0124506.
- Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: Development and validation. J Chronic Dis 1987;40:373-83.
- Macedo E, Bouchard J, Mehta RL. Renal recovery following acute kidney injury. Curr Opin Crit Care 2008;14:660-5.
- Vanmassenhove J, Kielstein J, Jörres A, Biesen WV. Management of patients at risk of acute kidney injury. Lancet 2017;389:2139-51.
- 17. Goldstein H, Diggle PJ, Liang KY, Zeger SL. Analysis of longitudinal

data. J R Stat Soc 2002;158:345.

- SESDF S de S do DF. Governo Do Distrito Federal Secretaria De Estado De Saúde Subsecretaria De Atenção Integral À Saúde; 2018.
 p. 1-31. Available from: http://www.saude.df.gov.br/wp-conteudo/ uploads/2018/04/hipertencao-e-diabetes-Manejo_da_HAS_e_DM_ na_APS.pdf. [Last accessed on 09 Jul 2021].
- Mehta RL, Cerdá J, Burdmann EA, Tonelli M, García-García G, Jha V, *et al.* International Society of Nephrology's 0by25 initiative for acute kidney injury (zero preventable deaths by 2025): A human rights case for nephrology. Lancet 2015;385:2616-43.
- James MT, Grams ME, Woodward M, Elley CR, Green JA, Wheeler DC, *et al.* A meta-analysis of the association of estimated GFR, albuminuria, diabetes mellitus, and hypertension with acute kidney injury. Am J Kidney Dis 2015;66:602-12.
- KDIGO. Clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int 2013;3:1-163.
- 22. Yokota LG, Sampaio BM, Rocha EP, Balbi AL, Sousa Prado IR, Ponce D. Acute kidney injury in elderly patients: Narrative review on incidence, risk factors, and mortality. Int J Nephrol Renovasc Dis

2018;11:217-24.

- Selmi Y, Ariba YB, Labidi J. Epidemiology, diagnosis, and etiology of acute kidney injury in the elderly: A retrospective analysis. Saudi J Kidney Dis Transpl 2019;30:678-85.
- Wang C, Pei YY, Ma YH, Ma XL, Liu ZW, Zhu JH, *et al.* Risk factors for acute kidney injury in patients with acute myocardial infarction. Chin Med J (Engl) 2019;132:1660-5.
- Pavkov ME, Harding JL, Burrows NR. Trends in hospitalizations for Acute Kidney Injury – United States, 2000-2014. MMWR Morb Mortal Wkly Rep 2018;67:289-93.
- Bamoulid J, Philippot H, Kazory A, Yannaraki M, Crepin T, Vivet B, et al. Acute kidney injury in non-critical care setting: Elaboration and validation of an in-hospital death prognosis score. BMC Nephrol 2019;20:419.
- Patschan D, Müller GA. Acute kidney injury in diabetes mellitus. Int J Nephrol 2016;2016:6232909.
- Koza Y. Acute kidney injury: Current concepts and new insights. J Inj Violence Res 2016;8:58-62.