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Renal angina index as a predictor of acute kidney injury in patients admitted to the emergency department

Abstract

Acute renal failure (ARF) is a syndrome that occurs due to multiple causes of injury and is characterized by an abrupt decrease in glomerular filtration rate. The concept of renal angina is made to highlight the characteristics of renal injury, as an analogy to the concept of angina pectoris, which is used to increase the suspicion of acute coronary syndrome in cardiology.

Material and methods: Prospective, observational and descriptive study. The present study will be carried out on patients admitted and attended in the Emergency Department of the Hospital de Alta Especialidad (UMAE) No. 14. Adolfo Ruiz Cortínez from September 1 to November 30, 2022. The data described in the data collection instrument will be collected in order to later concentrate them in a database for analysis.

Results: Of the 73 patients who entered the study, 42 patients were male, the mean age was 55 years, the minimum 18 and the maximum 75, the mode was 70 years. Of the patients included 49 were diabetic and of these 38.36% were male, only 11 had sepsis without much difference between the two sexes, representing 15.07% of the total number of patients. Of the total number of patients only 12 were admitted to the intensive care unit, 16.44%; the male sex was the most prevalent group, only 23.9% of the total number of patients required mechanical ventilation and 10.96% required vasopressor (Of the total number of patients only 15 (20.5%) had acute renal injury and of these the most affected age group was 61 to 80 years old).

Conclusion: The percentage of patients with a positive renal angina index (>8) who had AKI was 100% of the 73 patients only 15 developed AKI. Sepsis, vasopressor use and mechanical ventilation did not have a significant value for the development of AKI within this study in comparison to the study "Renal angina: "The beginning of the end" where out of 95 patients included in their study, sepsis, vasopressor use and mechanical ventilation did have a significant value for the development of AKI.

Volume 15 Issue 1 - 2023

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Received: February 20, 2023 | **Published:** February 27, 2023

Introduction

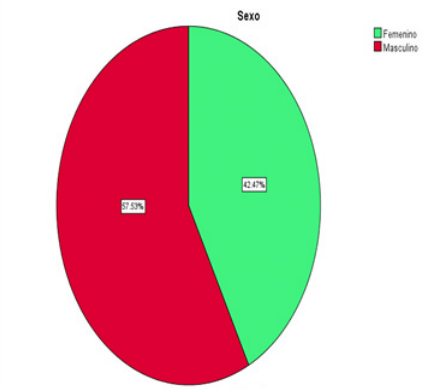
Acute kidney injury (AKI) is one of the most common diseases in critically ill adult patients admitted to the intensive care unit (ICU) and is associated with severe short- and long-term complications, including increased mortality. AKI is characterized by a rapid decrease in glomerular filtration rate (GFR).[1-5] AKI is defined as an increase in serum creatinine or a decrease in urine output over hours or days. A thorough anamnesis and physical examination can help categorize the underlying cause as prerenal, intrinsic renal, or postrenal.[8,9] More than 30 definitions of AKI have been recognized in the medical literature. In 2002 an "Acute Dialysis Quality Expert Panel Initiative" proposed a universal definition of AKI known as the RIFLE criteria (Risk Injury, Failure, Loss, End Stage), an acronym for the diagnosis, treatment and prevention of AKI.[9,10] Later the Acute Kidney Injury Network (AKIN) would meet and define AKI as an abrupt decline in renal function within 48 hours and an increase of equal to or greater than 0.3 mg/dl of creatinine or an increase of more than 50% of baseline creatinine, useful in patients already suffering from chronic renal failure, or a decrease in urine output of less than 0.5 cc/kg/hr for more than 6 hours.[10-12]

The incidence of AKI has been steadily increasing, particularly among long-lived hospitalized patients. Despite our knowledge and management of AKI have advanced, the prognosis remains poor. Both the incidence of AKI and its associated mortality rates show variation between countries, with differences attributed to factors such as the background of patients with AKI and the variety of definitions of AKI.[12-14] Goldstein introduced the term "RENAL ANGINA" in 2010. The impetus for this research is due to the realization that even a small elevation in serum creatinine can reflect significant renal damage and be associated with poor patient outcome, making creatinine a late marker of AKI. AKI researchers refer to the search for AKI biomarkers as the "search for renal troponin I," implying that AKI may allow earlier intervention, thus mitigating or even preventing progression of AKI to severe acute renal failure and the resulting morbidity and mortality.[16-18]

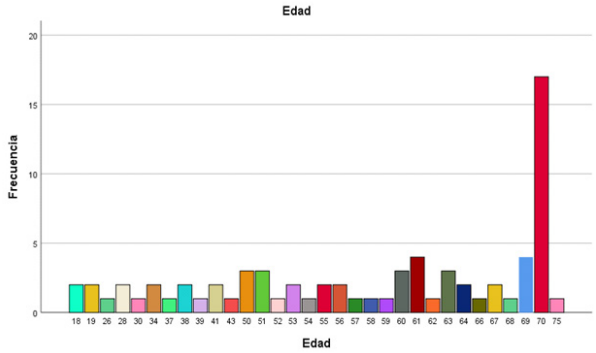
Material and methods

This is a prospective, observational and descriptive study in which all patients older than 18 years who were admitted to the emergency department UMAE HE No. 14. Veracruz, Veracruz, Ver. without previous diagnosis of nephropathy and with acute kidney injury on admission were included. Of the 73 patients who entered the study, 42 patients were male (Graph 1), the mean age was 55 years, the minimum 18 and the maximum 75, the mode was 70 years (Graph 2). Of the patients included 49 were diabetic and of these 38.36% were male, only 11 had sepsis without much difference between the two sexes, representing 15.07% of the total patients (Graph 3 and 4). Of the total number of patients only 12 were admitted to the intensive care unit, 16.44%; the male sex was the most prevalent group (Graph 5), only 23.9% of the total number of patients required mechanical ventilation (Graph 6) and 10.96% required the use of vasopressor

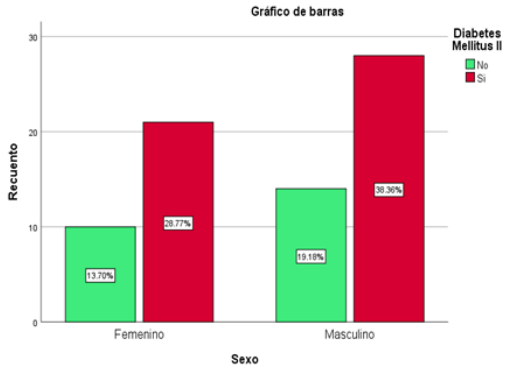
(Graph 7). Of the total number of patients only 15 (20.5%) had acute kidney injury and of these the most affected age group was 61 to 80 years old (Graph 8, 9).



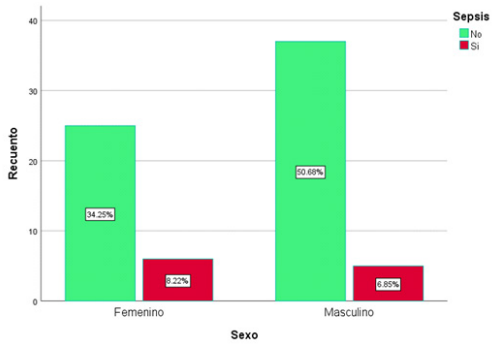
Graph 1



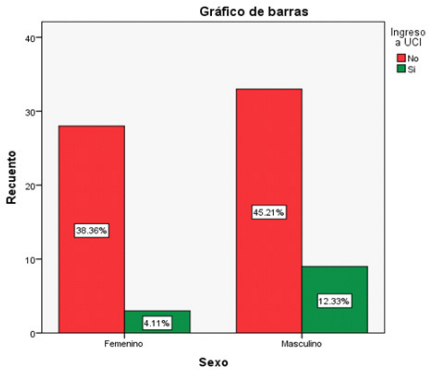
Graph 2



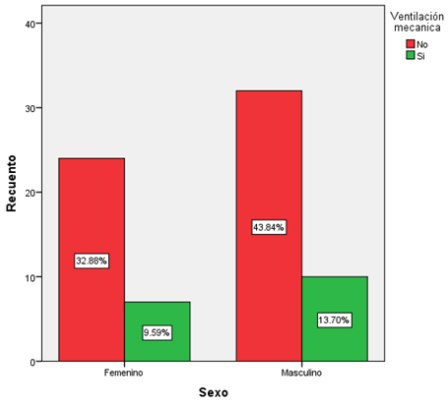
Graph 3



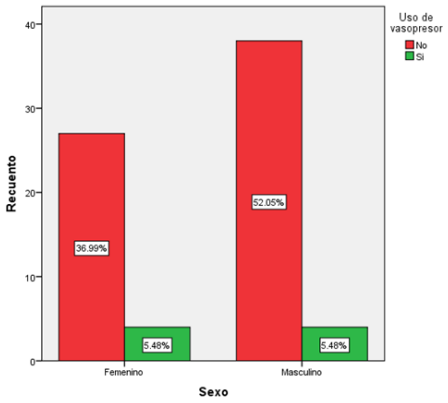
Graph 4



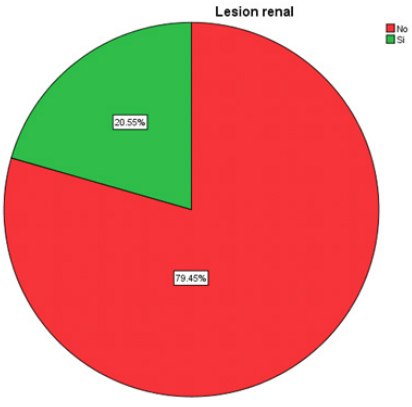
Graph 5



Graph 6

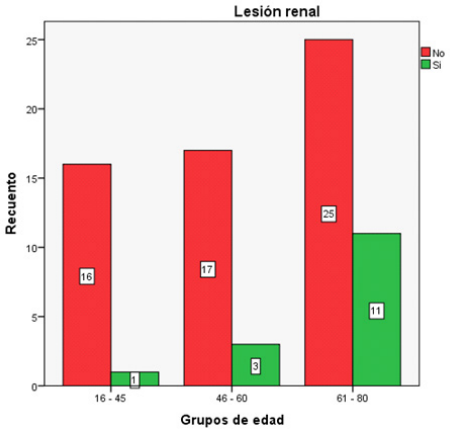


Graph 7



Graph 8

Citation: Aguilar MEM, Valdez VAC, Espinoza U. Renal angina index as a predictor of acute kidney injury in patients admitted to the emergency department. *J Anesth Crit Care Open Access*. 2023;15(1):51–55. DOI: 10.15406/jaccoa.2023.15.00551



Graph 9

Normality tests were performed using Kolmogorov-Smirnov, determining non-parametric tests for the following analyses, since the statistical significance is less than alpha 0.05, rejecting the alternate hypothesis, i.e., the data do not have a normal distribution (Table 1).

Table 1

Normality tests			
	Kolmogorov-Smirnov		
Basal creatinine	.135	73	.002
Creatinine control	.142	73	.001
Renal angina index	.259	73	.000
Subsequent creatinine	.326	73	.000

Regarding baseline and control creatinine, the measurements did not have much difference, the median was 0.60 and 0.70 respectively, the minimum value was 0.30 and 0.40, the maximum value was 1.10 for both cases and the interquartile range was 0.30 and 0.25. The values for creatinine after the interventions, median of 0.60, minimum value of 0.30, maximum value of 3.10 and interquartile range of 0.35. For the renal angina index values the values are as follows; median 3, minimum 1, maximum 16, interquartile range 3 (Table 2).

Table 2

Statistics					
		Basal creatinine	Creatinine control	Subsequent creatinine	IAR
N	Valid	73	73	73	73
	Lost	0	0	0	0
Media		.6600	.6904	.9274	5.32
Median		.6000	.7000	.6000	3.00
Fashion		.60	.60	.50	3
Standard deviation.		.18415	.18270	.69567	3.628
Variance		.034	.033	.484	13.163
Range		.80	.70	2.80	15
Interquartile range		0.30	0.25	0.35	3
Minimum		.30	.40	.30	1
Maximum		1.10	1.10	3.10	16
Percentiles	25	.5000	.5500	.5000	3.00
	50	.6000	.7000	.6000	3.00
	75	.8000	.8000	.8500	6.00

The percentage of patients with a positive renal angina index and had acute kidney injury was 100%, not having an expected result greater than 5 we opted for the Fisher test on Chi-square, obtaining a bilateral significance of 0.00, being less than alpha we accept the dependence relationship between the samples, i.e. they are related, also the Phi value is 0.960 so it has a very high association, i.e. the relationship between the two variables did not occur by chance (Table 3-4).

Table 3

Contingency table Renal lesion * Renal angina index					
			Renal angina index		Total
			No	Yes	
Renal injury	No	Count	57	1	58
		Expected frequency	45.3	12.7	58.0
		% within Kidney Injury	98.3%	1.7%	100.0%
		% within renal angina index	100.0%	6.3%	79.5%
	Yes	Count	0	15	15
		Expected frequency	11.7	3.3	15.0
		% within Kidney Injury	0.0%	100.0%	100.0%
		% within renal angina index	0.0%	93.8%	20.5%
Total		Count	57	16	73
		Expected frequency	57.0	16.0	73.0
		% within Kidney Injury	78.1%	21.9%	100.0%
		% within renal angina index	100.0%	100.0%	100.0%

Table 4

Chi-square tests						
	Value	gl	Asymptotic sign (bilateral)	Exact sig. (bilateral)	Exact sig. (unilateral)	Probability at point
Pearson's Chi-square	67.258[a]	1	.000	.000	.000	
Fisher's exact statistic				.000	.000	
N of valid cases	73					
a. 1 cells (25.0%) have an expected frequency of less than 5. The minimum expected frequency is 3.29.						

Gender did not show significant differences in organ failure, where the scores of women (Mdn=4; range=7) and men (Mdn= 4, Range= 13), U= 640, p= .899, g Hegdes= 0.22, For the percentage decrease in kidney injury if there was difference between both groups and if there was significant difference; the scores of women (Mdn=0; range=35) were higher than in men (Mdn= 0, Range= 13), U= 558, p= .319, g Hegdes= 0.30 . For both results there is a small effect of the independent variable on the dependent variables (Table 5).

Table 5

Symmetrical measurements				
		Value	Approximate Sig.	Accurate sig.
Nominal by nominal	Phi	.960	.000	.000
	Cramer's V	.960	.000	.000
N of valid cases		73		

Cross tests were performed to determine the ODDS RATIO, observing that only 27.3% of the patients who had sepsis manifested renal lesion, while 72.7% did not. The ODDS RATIO was 1.563, a result that is considered null, the 95% confidence interval was .360 as lower limit and 6.788 as upper limit, determining that this result is not statistically significant. It is concluded that sepsis is not a determinant factor for developing renal injury. The results for DM2 showed that only 24.5% of diabetic patients developed acute kidney injury, the ODDS RATIO value is 2.270 giving a value as a risk factor, the 95% confidence interval ranges from 0.575 to 8.968, concluding that it is not significant (Table 6-8).

Table 6

Mann Withney U independent sample comparisons					
	Female (n= 31)	Male (n= 42)	U	p	g of Hedges
	Mdn(Range)	Mdn(Range)			
Percentage of organ failure	4 (7)	4 (13)	640	.899	0.22
Percentage decrease	0 (35)	0 (13)	558	.319	0.30

Table 7

Contingency table Sepsis * renal injury					
			Renal injury		Total
			Yes	No	
Sepsis	Yes	Count	3	8	11
		% within Sepsis	27.3%	72.7%	100.0%
		% within renal lesion	20.0%	13.8%	15.1%
	No	Count	12	50	62
		% within Sepsis	19.4%	80.6%	100.0%
		% within renal lesion	80.0%	86.2%	84.9%
Total		Count	15	58	73
		% within Sepsis	20.5%	79.5%	100.0%
		% within renal lesion	100.0%	100.0%	100.0%
		Value	95% confidence interval		
				Inferior	Superior
ODDS RATIO		1.563		.360	6.788

Table 8

Contingency table Diabetes mellitus 2 * renal injury					
			Renal injury		Total
			Yes	No	
DM 2	Yes	Count	12	37	49
		% within DM 2	24.5%	75.5%	100.0%
		% within renal lesion	80%	63.8%	67.1%
	No	Count	3	21	24
		% within DM 2	12.5%	87.5%	100.0%
		% within renal lesion	20%	36.2%	32.9%
Total		Count	15	58	73
		% within DM 2	20.5%	79.5%	100.0%
		% within renal lesion	100.0%	100.0%	100.0%
		Value	95% confidence interval		
				Inferior	Superior
ODDS RATIO		2.270		.575	8.968

We estimated a multiple linear regression model with input method to predict the effect of sepsis, diabetes mellitus 2, ICU admission, vasopressor use, and mechanical ventilation use on the renal angina index. The regression equation was statistically significant F ($_5$) = 8.248, p= .000, β - 1 = 0.99. The R value^2 = .381, indicating that 38% of the change in inference score can be explained by the model with the previously mentioned variables. The regression equation was .518 + 481 (sepsis) + .204 (DM2) + .042 (ICU admission) + .114 (mechanical ventilation) + .144 (vasopressor use), where the renal angina index score increases .518 points if presenting with sepsis, .204 if diabetic, .042 if requiring ICU admission, .114 if requiring mechanical ventilation, and .144 points if requiring vasopressor use.

Discussion

AKI is defined as an increase in serum creatinine or a decrease in urine output over hours or days. A thorough anamnesis and physical examination can help categorize the underlying cause as prerenal, intrinsic renal, or postrenal.[8,9] Of the total number of patients in this study only 15 (20.5%) had AKI and of these the most affected age group was 61 to 80 years. In agreement with a systematic review that

analyzed 312 large cohort studies showed that the pooled incidences of AKI during hospitalizations using the KDIGO definition (used in 154 of the studies) was 21.6%.[2,3]

Taking into account the main objective, which was the application of the renal angina index as a predictor of acute kidney injury. In our study the percentage of patients with a positive renal angina index (greater than 8 points) who had acute kidney injury was 100%. Gender did not show significant differences in organ failure, where the scores of women (Mdn=4; range=7) and that of men (Mdn= 4, Range= 13), U= 640, p= .899, g Hegdes= 0.22, for the percentage decrease in kidney injury if there was difference between both groups and if there was significant difference; the scores of women (Mdn=0; range=35) were higher than in men (Mdn= 0, Range= 13), U= 558, p= .319, g Hegdes= 0.30. For both results there is a small effect of the independent variable on the dependent variables.

In comparison to a study conducted in Japan and Thailand of 851 patients (523 in Japan and 328 in Thailand), only 263 patients met the inclusion criteria for the initial analysis and 24 reached the primary outcome at 48 hours with development of acute kidney injury. In comparison to our study where out of 73 patients only 15 developed acute kidney injury at 72 hours.[12]

It is important to note that in our study 27.3% of the patients who had sepsis manifested renal injury while 72.7% did not, the ODDS RATIO was 1.563, a result that is considered null, the 95% confidence interval was .360 as the lower limit and 6.788 as the upper limit, determining that sepsis in our study group was not significant for the development of AKI. This is in contrast to what is mentioned in the study published in Japan and Thailand where sepsis was the most frequent cause of admission to the ICU with the development of AKI, in our study sepsis was not significant for the development of AKI in the following 72 hours of hospital admission.[10],12

The only marker of kidney injury used in this study was creatinine and control serum levels as well as uresis at 72hrs after hospital admission, thus improving the prediction of AKI development.[2,3] This study could aim to classify a low-risk group that could identify patients at low risk for AKI in whom it would not be necessary to use AKI biomarker testing as they would have low yields and in the high-risk group for the development of AKI, it could potentially allow physicians to optimize the use of biomarkers with high yields for timely diagnosis to decrease short-term mortality.[13]

Conclusion

The percentage of patients with a positive renal angina index (>8) who had AKI was 100% of the 73 patients only 15 developed AKI. Sepsis, vasopressor use and mechanical ventilation did not have a significant value for the development of AKI within this study in comparison to the study "Renal angina: "The beginning of the end" where out of 95 patients included in their study, sepsis, vasopressor use and mechanical ventilation did have a significant value for the development of AKI.

The best IAR cutoff point was 8.0 to predict LRAL. The regression equation was statistically significant F ($_5$) = 8.248, p= .000, β- 1 = 0.99. Indicating a correlation between the determinants and the Renal Angina Index acceptable.

The alternate hypothesis is accepted: the renal angina index is a primary identifier of acute kidney injury in patients admitted to the emergency department, so we suggest that this index can be implemented as a predictor in our department.

Acknowledgments

None.

Conflicts of interest

We have no conflict of interest.

References

1. Díaz de León Ponce MA, Briones Garduño JC, Carrillo Esper R, et al. Acute renal failure (ARF) classification, pathophysiology, histopathology, clinical picture, diagnosis and treatment, a logical version. *Rev Mex Anest.* 2017;40(4):280–287.
2. Ding X, Rosner MH, Ronco C. Acute Kidney Injury – Basic Research and Clinical Practice. *Contrib Nephrol. Basel, Karger.* 2018;193:1–12.
3. Jacob J, Dannenhoffer J, Rutter A. Acute Kidney Injury. *Prim Care.* 2020;47(4):571–584.
4. Negi S, Koreeda D, Kobayashi S, et al. Acute kidney injury: epidemiology, outcomes, complications, and therapeutic strategies. *Semin Dial.* 2018;31(5):519–527.
5. Pickkers P, Darmon M, Hoste E, et al. Acute kidney injury in the critically ill: an updated review on pathophysiology and management. *Intensive Care Med.* 2021;47(8):835–850.
6. Ronco C, Bellomo R, Kellum JA. Acute Kidney Injury. *Lancet.* 2019;394:1949–1964.
7. Cruz López J, Monares Zepeda E, Galindo Martín CA, et al. Renal angina: <<The beginning of the end >>. *Med Crit.* 2019;33(3):121–124.
8. Teo S, Endre Z. Biomarkers in acute kidney injury (AKI). *Best Pract Res Clin Anaesthesiol.* 2017;31(3):331–344.
9. Ostermann M, Liu K, Kashani K. Fluid Management in Acute Kidney Injury. *Chest.* 2019;156(3):594–603.
10. Uchino S, Bellomo R, Goldsmith D, et al. An assessment of the RIFLE criteria for acute renal failure in hospitalized patients. *Crit Care.* 2006;34(7):1913–1917.
11. Palevsky PM, Liu KD, Brophy PD, et al. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury. *Am J Kidney Dis.* 2013;61:649–72.
12. Hoste E, Kellum J, Selby NM, et al. Global epidemiology and outcomes of acute kidney injury. *Nat Rev Nephrol.* 2018;14:607–625.
13. Poston J, Koyner J. Sepsis associated acute kidney injury. *BMJ.* 2019;k4891.
14. Matsuura R, Srisawat N, Claure–Del Granado R, et al. Use of the Renal Angina Index in Determining Acute Kidney Injury. *Kidney International Reports.* 2018;3(3):677–683.
15. Menon S, Goldstein S, Mottes T, et al. Urinary biomarker incorporation into the renal angina index early in intensive care unit admission optimizes acute kidney injury prediction in critically ill children: a prospective cohort study. *Nephrol Dial Transplant.* 2016;31(4):586–594.
16. Teixeira J, Ambruso S, Griffin B, Faubel S. Pulmonary Consequences of Acute Kidney Injury. *Semin Nephrol.* 2019;39(1):3–16.
17. Roy J, Devarajan P. Acute Kidney Injury: Diagnosis and Management. *Indian J Pediatr.* 2019;87(8):600–607.
18. Montomoli J, Donati A, Ince C. Acute Kidney Injury and Fluid Resuscitation in Septic Patients: Are We Protecting the Kidney?. *Nephron.* 2019;143(3):170–173.