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Role of the Renal Arterial Resistive Index in Early Prediction of Contrast Induced Acute Kidney Injury after Coronary Angiography

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ABSTRACT

Background: contrast-induced acute kidney injury (CI-AKI) is defined as acute impairment of renal function after administration of intra vascular iodinated contrast media, and it is considered as the third leading cause of acute kidney injury in hospitalized patients.

Objectives: This study aimed to evaluate the clinical significance of pre-procedural Doppler based renal arterial resistive index for the prediction of CI-AKI in patients with coronary artery disease (CAD).

Patients and Methods: one hundred patients undergoing cardiac catheterization at risk of CI-AKI were studied. All patients presented with at least two CI-AKI risk factors and were free of other identifiable causes of acute kidney injury or arrhythmia. Doppler RRI was measured before and the one day after catheterization. CI-AKI was assessed and was defined by increase in serum creatinine by 25% above the pre-procedural baseline or rise in serum creatinine >0.5 mg/dl from baseline value or >25% decrease in e-GFR within 5 days after cardiac catheterization.

Results: fifteen subjects were developed CI- AKI within five days post-procedure. Post procedural RRI value was higher in CI-AKI subjects [RRI: 0.76±0.0l with CI-AKI patients vs. 0.6l±0.04 without CI-AKI P<0.00l]. In addition, the RRI was increased significantly in the first day after the procedure in patients with CI-AKI (p<0.00l). Post procedural RRI >0.744 predicted CI-AKI with a sensitivity of 94% and specificity of 92%.

Conclusions: measurement of the Doppler-based RRI before and early post-coronary catheterization in high-risk patients enabled early prediction of contrast induced acute kidney injury.

Keywords: Acute kidney injury, Contrast media, resistive index, Cardiac catheterization.

INTRODUCTION

Contrast-induced acute kidney injury (CI-AKI) is defined as acute renal impairment after administration of intra vascular iodinated contrast media. It occurs most frequently after coronary angiography. CK-AKI is associated with acute renal failure and may require urgent dialysis [(1)].

CI-AKI becomes the third most common cause of new AKI in hospitalized patients and was responsible for 11% of cases. The risk of CI-AKI has been related to the radiological procedures performed in the emergency room as primary percutaneous coronary intervention [(2)].

The development of renal injury is triggered by high osmolality and viscosity of contrast media, leading to increased renal vascular resistance [(3)], renal tubular hypoxia and eventually causing tubular cell apoptosis[(4, 5)].

Renal resistive index (RRI) has become the clinical method of choice to evaluate the renovascular hypertension and kidney allograft function with assessment of risk of AKI [(6, 7)].

Aim of the study:

To evaluate the clinical significance of pre-procedural Doppler based renal arterial resistive index, along with numerous pre- and intra- procedural risk factors, for the prediction of CI-AKI in patients with coronary artery disease (CAD) and preserved renal function, referred for coronary angiography and/or percutaneous coronary intervention.

PATIENTS AND METHODS

Study Design: This is a prospective study.

Study Setting: This study was conducted at the cardiovascular department, Tanta University Hospital and/or Damietta Cardiology Center for coronary angiography and percutaneous coronary intervention between August 20l7 and June 20l8.

Patients: We enrolled l00 consecutive patients with either stable angina (SA) or non-ST-elevation acute coronary syndrome (NSTE-ACS).

Inclusion criteria:

1. Age >l8 years.
2. Patients presented with stable angina with high pre-test probability of CAD or with positive treadmill electrocardiographic stress test or positive echocardiographic dobutamine stress test or non-ST-elevation acute coronary syndrome that met diagnostic criteria as established in ESC NSTE-ACS 2011 guidelines.
3. Presence of at least two of the following known risk factors for AKI: Glomerular filtration rate (e-GFR) < 90 ml/min/l.73m^2, age >60 yrs., peripheral vascular disease, diabetes and/or heart failure. [(8)]

Exclusion criteria:

1. Significant hemodynamic instability.
2. Acute or chronic respiratory failure (blood oxygen saturation <90%).
3. Severe heart failure.
4. Chronic kidney disease or other chronic diseases.

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5. Hematological or autoimmune diseases.
6. Neoplastic diseases.
7. Presence of other identifiable causes of acute kidney injury such as sepsis, hypovolemia, hypotension, cardiogenic shock and nephrotoxic drugs.

Methods:

This study was approved by Tanta University Ethics Committee and all patients gave written informed consents before participation. Full medical history and clinical data were taken from all the patients emphasizing on cardiovascular and acute kidney injury risk factors including age, gender, renal impairment, hypertension, diabetes mellitus, smoking and their duration, symptoms and signs suggestive of heart failure, peripheral vascular disease and current medications. Electrocardiogram and trans-thoracic echocardiography were done with assessing the patient rhythm whether it is sinus or any other rhythm, Left ventricular systolic function and Left atrium volume index were assessed. Data regarding coronary angiography information were collected such as Access site (Femoral or Radial), type of the procedure (diagnostic or interventional), type of contrast media, volume of contrast and severity of coronary artery disease. Both blood urea nitrogen (BUN) and creatinine were assessed together with assessment of creatinine clearance before and after coronary angiography procedure using Cockcroft-Gault Equation.

$$eC_{Cr} = \frac{(140 - \text{Age}) \times \text{Mass (in kilograms)} \times [0.85 \; if \; Female]}{72 \times \text{Serum Creatinine (in mg/dL)}}$$

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Renal Duplex evaluation

The renal resistive index (RRI) measurement was performed by trained sonographer before and within 24 hours after coronary angiography using commercially available machines (Philips Envisor HD, Toshiba Aplio 500) with 3.5 MHz pulsed wave Doppler probe. The patients were examined in a supine position using a lateral or postero-lateral approach. First correct B-mode acoustic window with a precise regulation of focus and gain was acquired; subsequently, the color box was opened. With the activation of the pulsed wave Doppler module, the sample volume was placed in the lumen of proximal inter-lobar arteries and the speed–time curve was recorded. The Doppler gain was set to obtain a clear outline of flow waves with minimal background noise. The Doppler spectrum was considered optimal when at least three similar consecutive waveforms visualized. The RRI will be calculated as follows [10]:

Peak systolic velocity - End diastolic velocity) / Peak systolic velocity

Post procedural data

Mehran's *et al.* [8] risk score for prediction of contrast-induced nephropathy was calculated for all patients. Occurrence and severity of contrast induced nephropathy was assessed. Contrast induced nephropathy defined as relative increase in serum creatinine by 25% between baseline level and peak level or rise in serum creatinine of >0.5 mg/dl from baseline value or a >25% decrease in e-GFR within 5 days after cardiac catheterization. Outcome variables, such as the need for IV hydration and dialysis were assessed. Then studied populations were divided into two groups: contrast induced nephropathy (CIN) group and non-contrast induced nephropathy (non-CIN) group.

Statistical method

We used MedCalc ver.15.8 as a statistical tool. Mann-Whitney's, Wilcoxon's, Chi square, factorial ANOVA, logistic and multiple regression analysis were used and also Spearman's correlation, and ROC Curve analysis were involved to test the data significance. Then data were presented in tables and figures. We considered P-values less than 0.05 (5%) was considered to be statistically significant.

RESULTS

The mean age of all patients was (59.42 ± 3.69) years. Comparison between the studied groups revealed significant increase in pre-procedural troponin, FBS, BUN and creatinine in CIN group compared to non-CIN group ($p < 0.0l$ respectively), and revealed significant decrease in pre-procedural creatinine clearance in CIN group compared to non-CIN group ($p < 0.0l$ respectively). According to the echocardiographic measures there were significant decrease in pre-procedural LVEF in CIN group compared to non-CIN group ($p = 0.0007$) and there were non-significant difference as regards pre-procedural LA-VI and E/e' ($p > 0.05$)**.** Comparison between the studied groups as regards renal Duplex data revealed that there was significant increase in pre-procedural RRI and RPI in CIN group compared to non-CIN group with significant statistical difference ($p < 0.0l$ respectively). The pre-procedural and post-procedural measurements revealed significant increase in post-procedural BUN, creatinine, RRI and RPI measurements in CIN group with significant difference ($p <0.0l$ respectively) and revealed significant decrease in post-procedural creatinine clearance measurements in CIN group with significant difference ($p = 0.000l$), **Table (1)**.

Table 1: Comparison between the two groups regarding the quantitative data

Variable	CIN group (15)	Non-CIN group (85)	Mann-Whitney's U test
	Median (IQR)	Median (IQR)	P value
Age	59 (59 – 63.5)	60 (57 – 61.2)	= 0.907
BMI	28 (26 – 29.3)	28.7 (27.5 – 31.5)	= 0.108
Systolic BP (mmHg)	140 (120 – 150)	130 (120 – 140)	= 0.199
Diastolic BP (mmHg)	90 (72.5 – 90)	70 (70 – 90)	= 0.009**
FBS (mg/dL)	106 (105 – 116)	103 (100 – 107)	= 0.0009**
BUN (mg/dL)	45 (40 – 51)	22 (17.5 – 28)	< 0.0001**
Creatinine(mg/dL)	1.1 (1 – 1.1)	1 (0.8 – 1)	< 0.0001**
Creatinine Clearance (CrCL) (mg/dL)	74 (65.7 – 85)	102 (88 – 124.2)	< 0.0001**
LA-VI	35 (34.6 – 40)	35 (32 – 36)	= 0.133
LVEF (%)	50 (43.5 – 52)	57 (48.5 – 63.5)	= 0.0007**
E/e'	9 (8.3 – 10)	8.5 (7.6 – 9.3)	= 0.058
RRI pre-procedural	0.71 (0.71 – 0.72)	0.61 (0.59 – 0.67)	< 0.0001**
RPI pre-procedural	1.55 (1.54 – 1.56)	1.28 (1.25 – 1.33)	< 0.0001**
Volume of contrast media (ml)	300 (300 – 300)	250 (200 – 300)	= 0.012*

BMI: body mass index, BP: blood pressure. FBS: fasting blood sugar, BUN: blood urea nitrogen. RRI: renal resistive index, RPI: renal pulsatility index.

Regarding gender of the patients, the majority (75%) of patients were males while (25%) were females Comparative studies between CIN and non-CIN groups revealed highly significant increase in diastolic BP and smoking in CIN group compared to non-CIN group with highly significant statistical difference ($p < 0.05$ respectively). Prevalence of PVD, MI and RAS blockade is more in CIN group compared to non-CIN group with highly significant statistical difference ($p < 0.01$ respectively) and non-significant difference as regards obesity, dyslipidemia, HTN, DM, HF and history of AKI ($p > 0.05$). There is significant increase in NSTE-ACS and NSIAD usage in CIN group compared to non-CIN group ($p < 0.0l$), **Table (2)**.

Table 2: Comparison between the two groups regarding the qualitative data.

Variable		CIN group (15)	Non-CIN group (85)	Chi square test
				P value
Gender	Male	11 (73.3%)	65 (76.5%)	= 0.947
Smoking	Yes	11 (73.3%)	33 (38.8%)	= 0.027*
Obesity	Yes	3 (20%)	43 (50.6%)	= 0.056
Dyslipidemia	Yes	10 (66.7%)	65 (76.5%)	= 0.627
HTN	Yes	15 (100%)	70 (82.4%)	= 0.169
DM	Yes	10 (66.7%)	62 (72.9%)	= 0.851
PVD	Yes	11 (73.3%)	11 (12.9%)	< 0.0001**
MI	Yes	10 (66.7%)	23 (27.1%)	= 0.006**
RAS blockade	Yes	15 (100%)	42 (49.4%)	= 0.0008**
HF	Yes	7 (46.7%)	26 (30.6%)	= 0.355
History of AKI	Yes	0 (0%)	5 (5.9%)	= 0.748
NSAID use	Yes	7 (46.7%)	0 (0%)	< 0.0001**
Statin use	Yes	10 (66.7%)	75 (88.2%)	= 0.0776
Indication of CA	NSTE-ACS	9 (60%)	21 (24.7%)	< 0.0001**
Troponin	+ve	6 (40%)	10 (11.8%)	= 0.017*
Severity of CAD	LM more than 50%	11 (73.3%)	12 (14.1%)	< 0.0001**
Referral to CABG	Yes	3 (20%)	12 (14.1%)	= 0.844
Hypotension during procedure	Yes	5 (33.3%)	5 (5.9%)	= 0.0051**

HTN: hypertension, DM: diabetes mellitus, PVD: peripheral vascular disease, MI: myocardial infarction, RAS: renin-angiotensin-aldosterone system, AKI: acute kidney injury, HF: heart failure, NSAID: non-steroidal anti-inflammatory drugs.

Comparison between pre-procedural and post-procedural measurements revealed significant increase in post-procedural BUN, creatinine, RRI and RPI measurements in CIN group with significant difference (p < 0.0l respectively); **Table (3).**

Table 3: Comparison between CIN patients as regards serial laboratory and renal Duplex assessments

Variables	Pre-procedural measurement	Post-procedural measurement	Wilcoxon's test
	Median (IQR)	Median (IQR)	P value
BUN (mg/dl)	45 (40 – 5l)	50 (45 – 60)	= 0.03*
Creatinine (mg/dl)	l.l (l – l.l)	l.9 (l.9 – 2)	= 0.000l**
Creatinine Clearance (CrCl) (ml/dl)	74 (65.7 – 85)	40 (37 – 48)	= 0.000l**
RRI	0.7l (0.7l – 0.72)	0.76 (0.75 – 0.77)	= 0.000l**
RPI	l.55 (l.54 – l.56)	l.59 (l.58 – l.6)	= 0.04*

Table 4: Comparison between the studied groups of patients as regards serial laboratory and renal Duplex measurements using repeated measures ANOVA test (2-Factor study)

Variables	Repeated 2 measures ANOVA (2-F: between the studied)	
	F value	p value
BUN (mg/dl)	33.46	<0.00l**
Creatinine (mg/dl)	l07.2l	<0.00l**
Creatinine Clearance (CrCl) (ml/dl)	2l7.35	<0.00l**
RRI	89.95	<0.00l**
RPI	2.l7	= 0.l44

ANOVA: analysis of variance, 2-F: 2-factor study. #logarithmic transformation was done to non-parametric data.

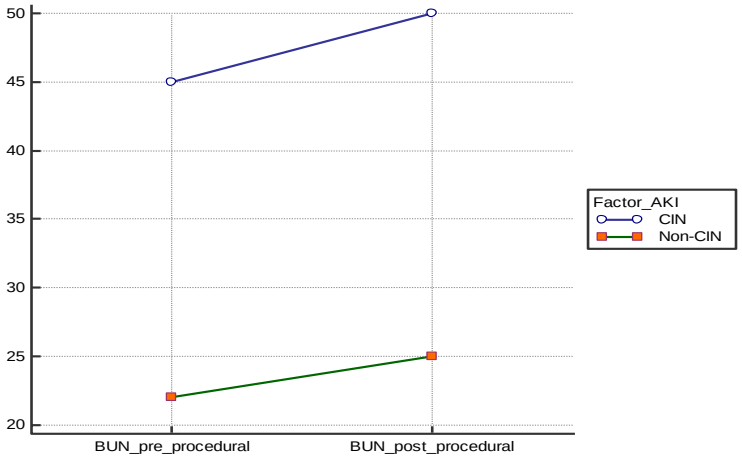

Figure 1: Comparison between the studied groups of patients regarding serial BUN assessments.

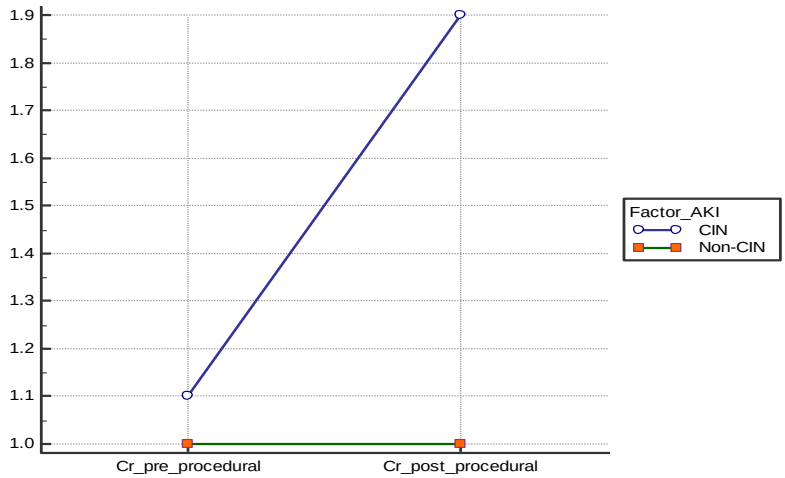

Figure 2: Comparison between the studied groups of patients regarding serial creatinine assessments.

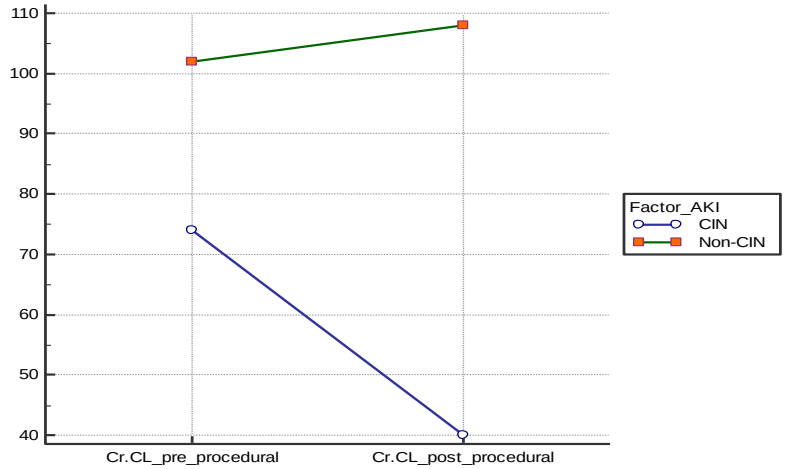

Figure 3: Comparison between the studied groups of patients regarding serial creatinine clearance assessments.

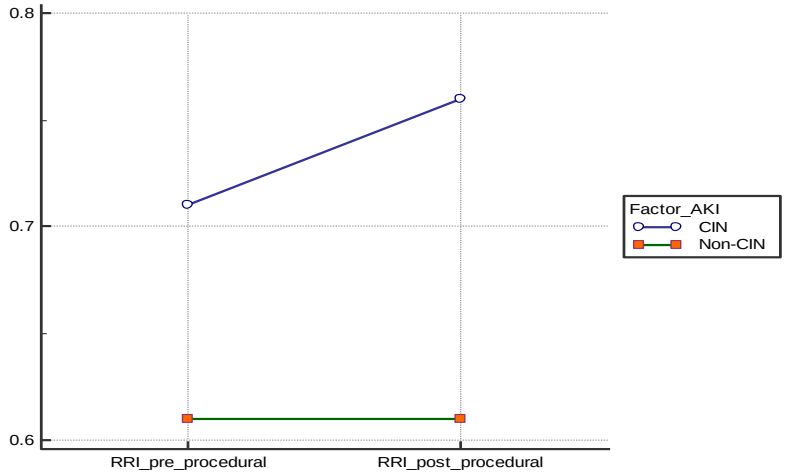

Figure 4: Comparison between the studied groups of patients regarding serial RRI assessments.

Table 5: Spearman's correlation analysis for baseline clinical/pre-procedural/CA Factors associated with post-procedural RRI:

Associated Factor	Post-procedural RRI	
	Rho	P
Age (years)	0.08l0	=0.4233
BMI	-0.0395	=0.6962
Systolic BP (mmHg)	0.0656	=0.5l66
Diastolic BP (mmHg)	0.0l04	=0.9l8l
PVD	0.0974	=0.3348
MI	0.00333	=0.9738
NSAID use	0.390	=0.000l**
Troponin (+ve)	0.l34	=0.l828
FBS (mg/dl)	0.4l9	<0.000l**
BUN (mg/dl)	0.574	<0.000l**
Creatinine (mg/dl)	0.606	<0.000l**
Creatinine Clearance (CrCl)	-0.565	<0.000l**
LA-VI	0.290	=0.0034**
LVEF	-0.l99	=0.0474*
E/e'	0.l72	=0.0868
Pre-procedural RRI	0.720	<0.000l**
Pre-procedural RPI	0.647	<0.000l**
Volume of contrast media	0.l47	=0.l437
Severity of CAD	0.l08	=0.l028
Referral to CABG	0.0663	=0.5l24
Hypotension during procedure	0.l28	=0.2058

rho: Spearman's rho (correlation coefficient).

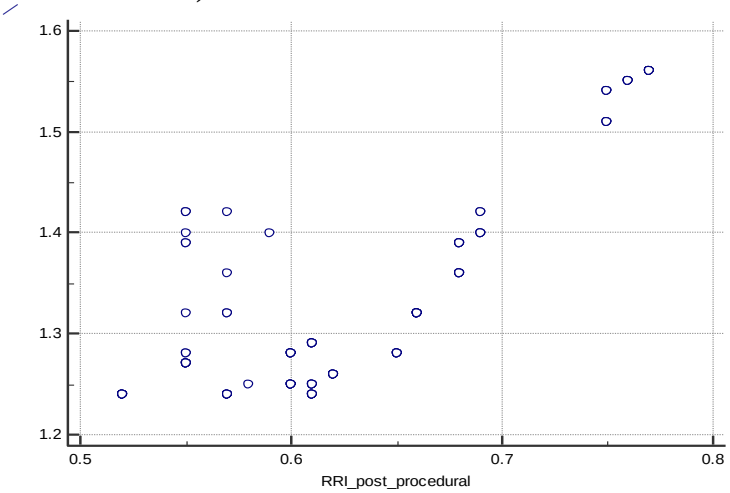

Figure 5: Correlation between pre-procedural RPI and post-procedural RRI.

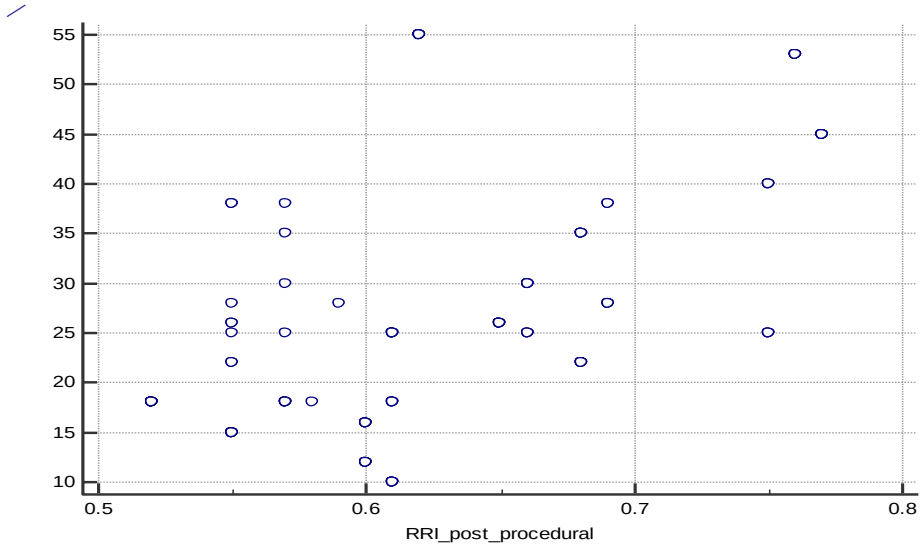

Figure 6: Correlation between pre-procedural BUN and post-procedural RRI.

ROC curve analysis:

Table 6: Roc-curve of some pre-procedural Factors to predict patients with CIN (l5) from patients without (85)

Variable	AUC	SE	Best Cut off point (Criterion)	Sensitivity (%)	Specificity (%)	P value	95% CI
Troponin	0.64l	0.0678	>0	40	88.24	= 0.037*	0.539 to 0.735
FBS	0.769	0.0559	>l03	l00	5l.76	<0.000l**	0.674 to 0.848
BUN	0.866	0.0477	>38	80	94.l2	<0.000l**	0.783 to 0.926
Creatinine	0.852	0.0542	>l.03	80	94.l2	<0.000l**	0.767 to 0.9l5
CrCl	0.878	0.04l7	≤99	l00	64.7l	<0.000l**	0.798 to 0.935
LA-VI	0.622	0.073l	>33	86.67	49.4l	= 0.096l	0.5l9 to 0.7l7
LVEF	0.774	0.0537	≤52	l00	6l.l8	<0.000l**	0.679 to 0.85l
E/e'	0.653	0.0765	>9.7	40	94.l2	= 0.045*	0.552 to 0.746
RRI	0.994	0.00406	>0.68	l00	94.l2	<0.000l**	0.953 to l.000
RPI	.000	0	>l.42	l00	l00	<0.000l**	0.964 to l.000

ROC (Receiver operating characteristic), AUC= Area under curve, SE= Standard Error.

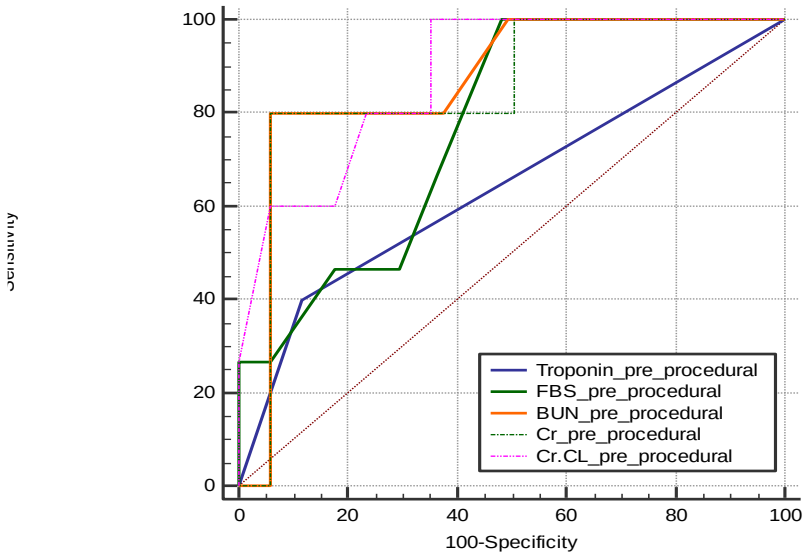

Figure 7: ROC curve of pre-procedural laboratory data.

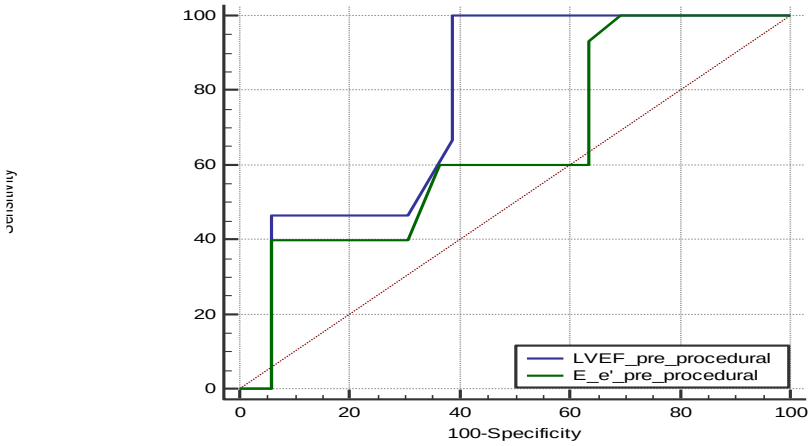

Figure 8: ROC curve of pre-procedural echocardiographic data.

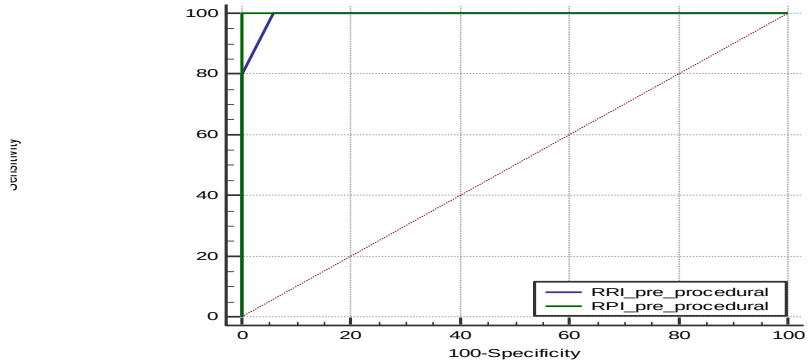

Figure 9: ROC curve of baseline pre-procedural RRI and RPI.

DISCUSSION

Contrast-induced nephropathy is a common and serious complication of contrast agents used in imaging studies. Renal resistive index has been studied as an early predictor of AKI in many clinical situations; however, its value in predicting acute kidney injury following administration of contrast agents has not been fully studied previously. This parameter depends on conventional serum creatinine, which rise was shown to be delayed and may even decrease during the first 24 hours after the procedure. In the current study, there is non-significant difference as regards age and sex of the patients (p > 0.05), these results came in agreement with **Wybraniec** and his colleagues [11].

The current study showed that hypertension was significantly related to CI-AKI. l00% of patients who developed AKI were hypertensive compared to 82.4% who didn't develop AKI (P=0.l69). Hypertension was found to be an independent predictor of CI-AKI. In 2004, **Bartholomew *et al.* [12],** found that hypertension was a risk for CIN in patients who underwent PCI (OR = 2.0, p = 0.000l). In the current study there was no significant difference in the incidence of CI-AKI among patients with and without DM. Diabetic patients constituted 66.7% of patients who developed CI-AKI compared to 72.9% of those who did not develop CI-AKI (P=0.85l). Similarly **Valente *et al.* [13],** in a study on l94 patients undergoing urgent PCI revealed that there was no significant relation between DM and CI-AKI (p=0.ll).

In the current study, there was no significant difference in the mean volume of contrast given between patients who developed CI-AKI (258.8±50.7 mL) and those who did not develop CI-AKI (248.2±6l.0) (P > 0.05). There was a significant increase in the risk of CI-AKI in patients who received contrast volume exceeding the maximum allowed contrast dose (23.5% versus 8.2%) (P=0.03l). ***Marenzi et al.*** [14] in their study on l79 patients undergoing primary PCI, showed that there was significantly increased risk of developing CI-AKI with higher contrast volumes (378±200 mL for patients who developed CI-AKI compared to 286±l25 mL for patients who didn't develop CI-AKI) (P = 0.008). Similarly, ***Evola et al.*** [15] in a study on 59l patients undergoing coronary procedures, there was significantly increased risk of developing CI-AKI with increasing the mean dose of contrast media (l36.9 ± l0l vs. ll5.l ± 83.56) (P=0.02).

Pre-procedural RRI was higher in subjects who subsequently developed CI-AKI in contrast to other

group of subjects who didn't develop CI-AKI (0.7l± 0.0l vs. 0.6l ± 0.4 p *<0.00l).* This finding differs from what has been found in similar studies which evaluated pre procedural RRI in cardiac surgery in which pre procedural RRI wasn't significantly different between subjects who developed AKI and subject who didn't (0.68 ±0.06 compared with 0.69 ±0.07, respectively)in cardiac surgery settings.**(16)**

In the present study, the post procedural RRI threshold of 0.75 predicted CI-AKI in patients undergoing cardiac catheterization which was similar to that found by ***Bossard et al.*** **(l6)** in cardiac surgery setting and approached the predictive values reported in humans (>0.70). The post procedural RRI threshold of >0.75 achieved 94% sensitivity and 92% specificity closely matching values found in similar studies which investigated potential role of RRI in predicting AKI. *Bossard et al*. (l6), who studied 65 patients after cardiac bypass graft surgery with risk factors for AKI, they reported that, in the immediate postoperative period RRI value >0.74 predicted the occurrence of delayed AKI with a sensitivity of 85 % and a specificity of 94 %.

CONCLUSION

To conclude, our study showed that the Doppler based renal resistive index that is measured early after cardiac catheterization in high risk patients for contrast induced nephropathy is beneficial in predicting contrast induced acute kidney injury in contrast to serum creatinine related markers, whose increase were shown to be delayed. Assessing RRI might allow for optimizing peri-procedural renal function sparing strategies.

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