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CASE REPORT

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Benign Rise in First Follow-up Creatinine in Decompensated Heart Failure: Renal Success

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Abstract

The Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial was a randomized control trial conducted to determine the safety of using a Pulmonary Artery Catheter (PAC) to guide therapy in patients hospitalized with severe heart failure. A study from the ESCAPE trial suggests that Rise in Serum Creatinine (RSC) in acute Decompensated Heart Failure (ADHF) is not a negative sign, especially if fluid overload is optimally treated. More aggressive diuresis is usually employed on 1st day of ADHF hospitalization followed by down titration of diuretics as the degree of congestion decreases, and rise of 1st follow-up creatinine is expected. We studied the characteristics and outcomes of subjects with *versus* without rise of 1st follow-up creatinine. Rise in 1st follow-up creatinine was not associated with any of 12 ESCAPE trial outcomes variables that we examined. This argues against discontinuing diuretic therapy in an overloaded ADHF patient merely because of rise in 1st follow-up creatinine. Since the kidney in this instance is acting appropriately to diurese a congested patient, we find that this benign rise in 1st follow up creatinine is a reflection of renal success through protecting the patient from the graver danger of cardiorespiratory failure.

Introduction

Rise in Serum Creatinine (RSC) is expected during hospitalization for acute Decompensated Heart Failure (ADHF) due to effect of intravenous diuresis and renin-angiotensin-aldosterone system inhibition by angiotens in converting enzyme inhibitors or angiotensin receptor blockers. There is sufficient evidence suggesting that the RSC in ADHF is not associated with worse outcomes [1,2], especially if fluid overload is optimally treated [3]. Rises in serum creatinine in ADHF don't always indicate true kidney injury [4]. Hence, RSC and permissive acute kidney injury (AKI) [5] may be more appropriate than Worsening Renal Function(WRF) and AKI [6]. More aggressive diuresis is usually employed on arrival to emergency room and1st day of ADHF hospitalization by emergency physicians and admitting hospitalists, followed by down titration of as degree of congestion decreases, and therefore rise of 1st follow-up creatinine is expected. In this analysis from ESCAPE trial, we studied the characteristics and outcomes of subjects with -versus without- rise of 1st follow-up creatinine.

Results

Our findings confirm that rise in 1st follow-up creatinine in the setting of aggressive diuresis during ADHF is not associated with worse outcomes, and therefore aggressiveness of diuresis should mainly be guided by volume status rather than renal functions.

Paradoxically, patients with rise in 1st follow-up creatinine had lower degree of decongestion from admission-to-discharge evident in lower negative fluid balance and lower decrease in RAP. This is likely because the group without rise in 1st follow-up creatinine probably had more renal congestion due to Worse Left Ventricle (higher LVESD and LVEDD) and worse pulmonary artery pressures, and is reflected in their higher baseline creatinine, and therefore diuresis rather benefited the kidney and improved kidney functions through renal decongestion, unlike patients with rise in 1st follow-up creatinine who had less renal congestion (due to lower PASP, PADP and better LVESD and LVESD) and therefore aggressive diuresis led to creatinine rise.

To the same extent, we have previously demonstrated that creatinine rise on admission with ADHF is due to renal congestion rather than low cardiac output and therefore an improvement in renal function would be expected with diuresis in cases with renal congestion [7]. These findings also suggest that more aggressive diuresis may be initiated in more severe right-side ADHF with expected renal congestion. In this analysis, we have confirmed the benign nature of rise in 1st follow-up creatinine during ADHF hospitalization up to 6-months following discharge -even in patients with IDDM. This argues against discontinuing or minimizing diuretic therapy in an overloaded ADHF patient merely because of fear of rise in 1st follow-up creatinine. Since the kidney in this instance is acting

appropriately to diurese a congested patient, we find that this benign rise in 1st follow up creatinine is rather a reflection of renal success -not failure- through protecting the patient from the graver danger of cardiorespiratory failure.

Discussion

Among 433 ESCAPE trial patients (mean age 56 years, 74% men) with Left Ventricular Ejection Fraction (LVEF) 19%, serum creatinine was 1.51 ± 0.62 mg/dL at baseline and1.49 ± 0.63 mg/dLon 1st check (day 3). A total of 35.7% (133/373) of cases had a rise in 1st follow-up creatinine, with a magnitude of creatinine increase of 0.30 ± 0.28 mg/dL, which represents 22.9% increase relative to baseline. Among patient with rise in 1st follow-up creatinine, 41.6% (52/125) had creatinine improvement on discharge. Patients with rise in 1st follow-up creatinine had a lower baseline creatinine (1.41 ± 0.57 mg/dL vs. 1.56 ± 0.65 mg/dL, P=0.024) and higher maximum in-hospital creatinine (1.88 ± 0.91 mg/dL vs. 1.59 ± 0.72 mg/dL, P=0.002). Patient with rise in 1st follow-up creatinine were more likely to have insulin-dependent diabetes Mellitus (IDDM, 25.8% vs. 13.4%, P=0.003). Table 1 compares patients with versus without rise in 1st follow-up creatinine.

Patients with rise in 1st follow-up creatinine had significantly lower left Ventricular End-Diastolic Diameter (LVEDD, 6.36 cm vs. 6.75 cm, P=0.009), lower Left Ventricular End-Systolic Diameter (LVESD, 5.57 cm vs. 6.04 cm, P=0.003), lower pulmonary artery diastolic pressure (PADP, 25.9 mmHg vs. 29.0 mmHg, P=0.038) and a trend

Table 1: Comparison of ESCAPE trial patients with *versus* without rise in 1st creatinine.

	Rise in 1st creatinine (n=133)	No rise in 1st creatinine (n=240)	P-value (n=240
Demographics and comorbidities			
Age (years, m ± SD)	57.08 ± 13.22	56.09 ± 14.02	0.503
BMI (Kg/m2, m ± SD)	30.18 ± 7.51	28.64 ± 6.12	0.048
Admission weight (Kg, m ± SD)	86.58 ± 21.53	85.69 ± 19.71	0.69
Discharge weight (Kg, m ± SD)	82.98 ± 21.08	81.11 ± 19.41	0.413
Male % (n)	73.7% (98/133)	75.8% (182/240)	0.646
Black race % (n)	29.3% (39/133)	25% (60/240)	0.365
CABG % (n)	29.5% (39/132)	28.9% (69/240)	0.891
COPD % (n)	18.2% (24/132)	15.5% (37/239)	0.502
IDDM % (n)	25.8% (34/132)	13.4% (32/239)	0.003
DM on OHG % (n)	22% (29/132)	17.6% (42/239)	0.303
Hepatic disease % (n)	7.6% (10/132)	8.4% (20/239)	0.789
Stroke % (n)	9.1% (12/132)	10% (24/239)	0.767
AFib % (n)	28% (37/132)	29.7% (71/239)	0.734
ICD % (n)	22.7% (30/132)	30.1% (72/239)	0.127
Pacemaker % (n)	21.2% (28/132)	24.7% (59/239)	0.45
Baseline physical examination			
JVD % (n)	89.7% (113/126)	94.5% (222/235)	0.094
JVP>12 cmH2O % (n)	50.8% (64/126)	58.3% (137/235)	0.171
Rales % (n)	58% (76/131)	51.5% (123/239)	0.227
Hepatomegaly % (n)	54.6% (71/130)	59.7% (142/238)	0.348
HJR % (n)	77.3% (99/128)	80.9% (186/230)	0.427
Ascites % (n)	40% (52/130)	37.7% (89/236)	0.667
2+ peripheral edema % (n)	40.5% (53/131)	37.2% (89/239)	0.543
Baseline laboratory values			
Na	136.98 ± 4.16	136.39 ± 4.62	0.22
BUN	34.34 ± 25.24	35.92 ± 21.33	0.524
creatinine	1.41 ± 0.57	1.56 ± 0.65	0.024
BNP	941 ± 1242	1101 ± 1425	0.327
Hct	37.09 ± 4.69	38.09 ± 5.66	0.088
Total bilirubin	0.94 ± 0.51	0.83 ± 0.55	0.094

Baseline echocardiography & CPET			
LVEDD (cm, m ± SD)	6.36 ± 1.03	6.75 ± 1.17	0.009
LVESD (cm, m ± SD)	5.57 ± 1.13	6.04 ± 1.20	0.003
LVEF	21 ± 10	19 ± 9	0.197
IVC (ins) (cm, m ± SD)	1.60 ± 0.68	1.66 ± 0.72	0.597
IVC (exp) (cm, m ± SD)	2.18 ± 0.53	2.23 ± 0.64	0.51
Collpasability index	28.4 ± 20.5	28.8 ± 20.9	0.9
Peak VO2 ($Mlmin^{-1}kg^{-1}$, m ± SD)	11.2 ± 2.6	11.8 ± 4.0	0.538
Baseline central hemodynamic parameters			
RAP (mmHg, m ± SD)	13.2 ± 7.6	13.7 ± 6.9	0.726
PASP (mmHg, m ± SD)	53.2 ± 15.4	57.8 ± 13.3	0.056
PADP (mmHg, m ± SD)	25.9 ± 8.3	29.0 ± 9.1	0.038
PCWP (mmHg, m ± SD)	24.4 ± 8.7	26.0 ± 9.1	0.306
CI($L/min/m^2$, m ± SD))	2.09 ± 0.78	1.92 ± 0.52	0.147
COP (L/min, m ± SD)	4.11 ± 1.70	3.79 ± 1.10	0.219
Furosemide utilization			
Baseline furosemide (mg, m ± SD)	244 ± 165	244 ± 196	0.994
In-hospital furosemide (mg, m ± SD)	281 ± 209	327 ± 481	0.33
% increase of furosemide*	50 ± 160	103 ± 491	0.276
Parameters of decongestion			
Weight loss (Kg, m ± SD)	2.88 ± 4.21	4.12 ± 5.23	0.029
RAP decrease (mmHg, m ± SD)	1.91 ± 7.15	5.26 ± 10.18	0.05
IVC (ins) decrease (cm, m ± SD)	0.15 ± 0.60	0.39 ± 0.76	0.089
IVC (exp) decrease (cm, m ± SD)	0.16 ± 0.53	0.38 ± 0.67	0.073
Outcomes			
In-hospital death % (n)	40% (10/25)	30% (15/50)	0.386
30-day death % (n)	5.3% (7/133)	4.3% (11/240)	0.769
6-month death % (n)	18.8 % (25/133)	20.8% (50/240)	0.638
6-month rehosp % (n)	55.6% (74/133)	58.3% (140/240)	0.614
6-month HF rehosp % (n)	42.9% (57/133)	46.7% (112/240)	0.479
6-month death cardiac rehospm card transplant% (n)	51.1% (68/133)	57.5% (138/240)	0.236
6-month death any rehospm card transplant% (n)	63.9% (85/133)	67.9% (163/240)	0.432
Death & CV rehosp% (n)	48.6% (17/35)	57% (45/79)	0.407
Hospital duration(d, m ± SD)	8.42 ± 6.12	9.55 ± 6.86	0.118
Total days in-hosp (1^{st} 180 days)	15.5 ± 15.6	18.1 ± 19.0	0.186
No. of hospitalization (6-month)	2.0 ± 1.1	2.1 ± 1.4	0.539
Days to 1^{st} rehosp (d, m ± SD)	64 ± 45	66 ± 46	0.834

SD: Standard Deviation; BMI: Body Mass Index; CABG: Coronary Artery Bypass Graft; COPD: Chronic Obstructive Pulmonary Disease; IDDM: Insulin-Dependent Diabetes Mellitus; DM: Diabetes Mellitus; OHG: Oral Hypoglycemics; ICD: Implantable Cardiac Defibrillator; JVD: Jugular Venous Distension; JVP: Jugular Venous Pressure; HJR: Hepatojugular Reflux; LVEDD: Left Ventricular End-Diastolic Diameter; LVESD: Left Ventricular End-Systolic Diameter; LVEF: Left Ventricular Ejection Fraction; IVC: Inferior Vena Cava; RAP: Right Atrial Pressure; PASP: Pulmonary Artery Systolic Pressure; PADP: Pulmonary Artery Diastolic Pressure; PCWP: Pulmonary Capillary Wedge Pressure; CI: Cardiac Index; COP: Cardiac Output; CV: Cardiovascular*

towards lower pulmonary artery systolic pressure (PASP, 53.2 vs. 57.8, P=0.056).There was no difference between both groups in Right Atrial Pressure (RAP), pulmonary capillary wedge pressure, IVC during inspiration and expiration, B-Type Natriuretic Peptide (BNP), Jugular Venous Distension (JVD), and JVD>12 cm, presence of rales, S3 gallop, hepatomegaly, positive hepatojugular reflux, presence of ascites, or presence of 2+ pitting edema. There was also no difference between both groups with regards to cardiac output (4.11 vs. 3.79, P=0.219), LVEF, and peak VO^2.

Patients with rise in 1st follow-up creatinine required lower, but non-significant, in-hospital furosemide dose (281 mg vs. 327 mg, P=0.330), and lower increase in furosemide dose in-hospital relative to baseline (50% vs 103%, P=0.276), compared with subjects without rise in 1st follow-up creatinine. Despite higher BMI on admission (30.2Kg/m^2 vs. 28.6 Kg/m^2, P=0.048) and discharge (29.00 Kg/m^2 vs. 27.12 Kg/m^2, P=0.017), patients with rise in 1st follow-up creatinine had less degree of decongestion evident in the significantly lower negative fluid balance (2.88 vs. 4.12 L, P=0.029) and a significantly lower decease in RAP from baseline to final check (-1.91 vs. -5.26, P=0.05) and a trend towards less decrease in inspiration and expiration IVC diameter from admission-to-discharge. Rise in 1st follow-up creatinine was not associated with any of 12 ESCAPE trial outcomes variables that we studied (Table 1). We have also repeated the analysis of these outcomes in patients with IDDM and we found no significant difference in all 12 outcomes (all P>0.1).

Conclusion

Our study confirms that rise in 1st follow-up creatinine in the setting of aggressive diuresis during ADHF is not associated with worse outcomes, and therefore aggressiveness of diuresis should mainly be guided by volume status rather than renal functions.

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