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Case Report

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Renal Vein Congestion in Cardiorenal Syndrome Type 1: An Analysis of Two Case Reports From a Hemodynamic Perspective

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Abstract

Cardiorenal syndrome (CRS) is a complex disease that results from the close relationship between cardiac and renal physiology. We present two cases which illustrate how renal vein congestion (case 1) and reduced cardiac output (case 2) can both lead to CRS type 1. We focused on the hemodynamic differences between these two cases, as we believe that a deep understanding of the pathophysiology and the renal blood flow (RBF) and renal perfusion pressure (RPP) in these two cases is key in the correct management strategy. This report highlights that CRS type 1 must be recognized as an acute process comprised of two distinct mechanisms, due to renal vein congestion or due to low cardiac output, which can present independently or in varying degrees in the same patient. In consequence, the role of renal vein congestion needs to be recognized early in order to adequately tailor patient treatment.

Keywords: Cardiorenal syndrome; Renal vein congestion; Hemodynamics

Introduction

The close relationship between cardiac and renal physiology results in an interdependence that may lead to cardiac or renal injury when one of the organs undergoes a major insult. This interdependence causing renal or cardiac dysfunction has been termed as cardiorenal syndrome (CRS) [1]. The current classification of CRS divides it into five clinical subtypes, based on the organ believed to be the culprit and the acuity of the clinical presentation [2]. In this article, we will focus on CRS type 1, defined as the development of acute kidney injury (AKI) in patients with cardiac disease most commonly in the setting of acute decompensated heart failure [3]. We believe that the hemodynamic changes underlying CRS type 1 are complex and not necessarily reflected in the current definitions.

We present two cases with different pathophysiological pathways, but both resulting in CRS type 1. First, we will present a patient with elevated central venous pressure (CVP) leading to elevated renal vein congestion. He had venous fluid overload and poor arterial circulation. Second, we present a case of low cardiac output (CO) leading to pre-renal AKI mainly due to poor forward flow. We will analyze these cases from a hemodynamic perspective to help us differentiate between the two distinct subtypes of CRS type 1. Interestingly, these scenarios are dynamic and commonly coexist in the same patient overtime. Therefore, clinicians should have increased awareness for an accurate recognition and prompt implementation of the appropriate treatment based on hemodynamic data.

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Case Reports

Case 1

A 68-year-old woman with medical history significant for hypertension, persistent atrial fibrillation, moderate aortic stenosis, severe mitral regurgitation status post mitral valve repair, pulmonary hypertension, chronic obstructive pulmonary disease and obstructive sleep apnea was admitted for acute decompensated heart failure. Despite up-titration of loop diuretic and addition of milrinone, we observed a decrease in both mean arterial pressure (MAP) and systemic vascular resistance (SVR) alongside with worsening renal function. Pulmonary artery catheterization was performed to guide therapy; initial hemodynamics showed a markedly elevated CVP of 29 mm Hg, and an elevated pulmonary capillary wedge pressure

(PCWP) of 40 mm Hg. Cardiac index (CI) calculated via Fick equation was adequate at 2.4 L/min/m^2 with SVR was low normal at 10 mm Hg × min/mL. Milrinone infusion was then discontinued. In order to maintain adequate SVR, intravenous norepinephrine infusion was started which led to increased diuresis, reduced CVP and improved serum creatinine (Table 1).

Case 2

A 40-year-old man with medical history significant for non-ischemic cardiomyopathy and medication nonadherence was admitted with acute on chronic decompensated heart failure. Clinical exam was significant for marked pulmonary congestion and cool lower extremities, but no significant jugular venous distention or peripheral edema were present. The patient was initially managed with increasing dose of loop diuretic and addition of milrinone infusion. On the second day of admission, urine output decreased, and creatinine increased from 2.2 to 2.8 mg/dL. CVP was low at 3 mm Hg as measured via left internal jugular central venous catheter. CI was estimated to be 1.86 L/min/m^2 based on Fick equation with venous oxygen saturation obtained from the superior vena cava. Decision was made to administer intravenous fluids in addition to inotropic support via dobutamine infusion. CI then increased with subsequent improvement of urine output and serum creatinine (Table 2).

Discussion

The main mechanism of CRS type 1 is known to be an acute reduction in CO leading to a reduced perfusion to the kidneys. Nevertheless, in acute heart failure exacerbation, we often see systemic vein congestion leading to increased renal venous pressure and renal vein congestion. [4]. These two processes frequently coexist in the same patient leading to AKI. Increasing diuresis and promoting a negative fluid balance has been the mainstay of treatment of acute heart failure exacerbations in clinical practice. However, there is an alternative scenario where patients may present with prerenal kidney injury in setting of cardiogenic shock without increased renal venous pressure. The recognition of this situation is critical, and a misdiagnosis may lead to an opposite therapeutic approach with undesired outcomes.

The initial case is consistent with renal vein congestion evidenced by improvement in the renal function despite weaning the inotropic agent and the gradual decrease in CI. CVP in isolation was the only invasively measured hemodynamic parameter to correlate with improved serum creatinine and urinary output. Given that CRS was secondary to renal vein congestion, constant infusion of a loop diuretic improved serum creatinine level. Conversely, the second case was more consistent with a prerenal syndrome, as serum creatinine initially worsened with aggressive diuresis, but additional inotropic support then led to a gradual increase in CO, reduced creatinine level and improved diuresis.

We believe that a deep understanding of the renal blood flow (RBF) and renal perfusion pressure (RPP) is key in managing these patients. We assessed the RPP and its relationship with changes in serum creatinine and diuresis (Tables 1 and 2). Mullens et al defined RPP as the difference between MAP and CVP. In their study, RPP was not significantly different in patients with CRS type 1 versus those with preserved renal function (63 ± 15 vs. 65 ± 12 mm Hg, P = 0.2) [3], suggesting that this parameter may not be entirely associated with the effective RBF. This observation differs from the hemodynamic trend of our first case, where the RPP was initially decreased at 37 mm Hg, despite milrinone infusion and an adequate CO (6.6 L/min) and CI (3.0 L/min/m^2). Subsequent norepinephrine infusion effectively increased both SVR and RPP which resulted in improved diuresis and decreased serum creatinine. We hypothesize that norepinephrine increased MAP, thereby increasing RPP and effective RBF leading to improved diuresis and creatinine levels. The use of norepinephrine can be a bridge to increase RPP via improvement of MAP.

In contrast, our second case clearly demonstrates that calculated RPP was adequate (104 mm Hg) in the setting of a reduced CO of 3.9 L/min and CI of 1.86 L/min/m^2. Renal function only improved with inotropic support and intravenous volume administration. Given this correlation, how does one explain this discrepancy regarding the effect of RPP on AKI in CRS type 1? Is RPP a true marker of renal perfusion? Although one could argue that an increase in RPP led to improved renal function of the first patient, we believe the parameter does not consider the pathophysiology of renal vein congestion. It would be an oversimplification to define renal perfusion by CVP and MAP alone considering the complexity of the autoregulation of the renal blood flow [5].

We recognize that large prospective randomized trials have compared different therapies for this syndrome without mention of the underlying pathogenesis. The Cardiorenal Rescue Study in Acute Decompensated Heart Failure (CARRESS-HF) found that pharmacological therapy was superior to ultrafiltration, the latter being associated with higher rate of adverse events [6]. Conversely, the Ultrafiltration Versus IV Diuretics for Patients Hospitalized for Acute Decompensated Congestive Heart Failure (UNLOAD) trial showed that ultrafiltration reduced readmission rates in patients with decompensated heart failure and hypervolemia [7]. These conflicting results indicate that CRS type 1 must be analyzed from a deeper hemodynamic perspective, not only considering volume overload or decreased CO alone.

CRS type 1 is recognized as an acute process comprised of two distinct mechanisms that can be present in the same patient in varying degrees and the role of renal vein congestion needs to be recognized early [8]. We believe future trials should tailor therapy to a specific hemodynamic pattern and patient population. Study endpoints should focus more on an improvement of hemodynamics in addition to cardiovascular morbidity and mortality.

Conclusions

The identification of the mechanisms behind AKI in acute heart failure exacerbation can be challenging. Before committing to

Table 1. Hemodynamics Recorded During Admission for Case 1

Case 1	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
BSA	2.2	2.2	2.2	2.2	2.2	Swan discontinued	Swan discontinued	Swan discontinued
Hgb	9.1	8.5	8.4	8.4	8.7			
VO_2 (used for Fick calculation)	250	250	250	250	250			
HR (day average)	101	111	114	101	114	110	110	88
BP (day average)	116/69/85	92/49/63	132/69/90	153/69/97	134/71/92	113/71/85	124/68/87	105/70/82
RAP	29	26	20	25	20			
PAP	70/40/55	74/34/45	97/32/54	80/40/53	67/30/42			
PCWP	40	34	32					
SvO_2	60	64.4	71.2	63.5	59.8			
SaO_2	97	97	96	97	94			
CO	5.3	6.6	8.8	6.5	5.9			
CI	2.4	3	4	3	2.68			
SVR	10.5	5.8	5	11	9.8			
PVR	2.8	1.6	2.5					
Norepinephrine			2					
Milrinone	0.375	0.2	0.2	0.1	Discontinued	Discontinued	Discontinued	Discontinued
Lasix (mg/h)	20	20 (+ 40 mg IV × 1)	20	20	20	10		
Chlorothiazide	250 mg IV q12h	250 mg IV q12h	250 mg IV q12h	250 mg IV q12h	250 mg IV q12h			
Midodrine	10 mg q6h	10 mg q6h	10 mg q6h	10 mg q6h	10 mg q6h			
Intake	712.5	1,408.3		1,551.5	1,747.5	456.3	740	1,080
Output	501	2,460		4,640	6,725	3,305	1,035	900
I/O net	211.5	-1,051		-3,090.5	-4,977.5	-2,848.7	-295	180
Creatinine (mg/dL)	1.9	2.3	2.6	2.6	2.6	2.3	2	1.6
BUN (mg/dL)	12	10	14	16	14	10	8	8

Case 1 shows cardiorenal syndrome type 1 primarily due to renal vein congestion. Acute kidney injury occurred in presence of elevated RAP despite adequate CI. Renal function improved with diuresis and maintenance of adequate SVR. Day number corresponds to day of admission. BSA: bovine serum albumin; Hgb: hemoglobin; VO_2: oxygen consumption; HR: heart rate; BP: blood pressure (displayed as systolic/diastolic/mean); RAP: right atrial pressure; PAP: pulmonary pressure (displayed as systolic/diastolic/mean); PCWP: pulmonary capillary wedge pressure; SvO_2: mixed venous oxygen saturation; SaO_2: arterial oxygen saturation; CO: cardiac output; CI: cardiac index; SVR: systemic vascular resistance; PVR: pulmonary vascular resistance; BUN: blood urea nitrogen; IV: intravenous.

Table 2. Hemodynamics Recorded During Admission for Case 2

Case 2	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 15
BSA	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1
Hgb	11.9	13	12.3	12	11.5	11	10.9	10.9	10.7
VO_2 (used for Fick calculation)[a]	262.5	262.5	262.5	262.5	262.5	262.5	262.5	262.5	262.5
HR (day average)	110	109	100	101	101	91	91	86	75
BP (day average)	140/91/107	166/90/115	127/84/98	114/72/86	134/75/95	130/71/91	135/94/108	150/95/113	147/100/119
RAP	3	3	3	3	3	3	3	3	3
PAP									25/11/16
PCWP									8
SvO_2	54.4	62.1	65.8	77.4	66.1	79.3	68.9	75.2	66
SaO_2	96	97	98	98	97	96	98	96	93
CO	3.9	4.25	4.87	7.81	5.43	10.5	6.09	8.51	6.69
CI	1.857142857	2.023809524	2.319047619	3.719047619	2.585714286	5	2.9	4.05238095	3.19
SVR	26.4	26.3	19.5	10.6	16.9	8.3	17.2	12.9	17.3
PVR									1.2
Milrinone	0.75	0.7	0.7	0.7	0.7	0.7	0.7	0.7	Discontinued
Dobutamine		1	2	2	2	2	2	2	7
Lasix	40 mg IV × 1								
Other		IV albumin 25 g × 1					IV albumin 25 g × 1		
Intake	709.8	1,941.4	1,617.6	1,602.3	796.1	1,701.1	1,954.5	1,473.5	
Output	2,075	925	720	1,800	600	725	Not measured	600	
I/O net	-1,365.2	1,016.4	897.6	-197.7	196.1	976.1		873.5	
Creatinine (mg/dL)	2.2	2.8	2.5	2.2	2	1.9	1.8	1.6	1.7
BUN (mg/dL)	56	61	62	59	56	51	54	52	49

Case 2 shows cardiorenal syndrome type 1 primarily due to low CO. Acute kidney injury occurred in the setting of low CI despite normal RAP. Renal function improved with volume resuscitation and inotropic support. Day number corresponds to day of admission. BSA: bovine serum albumin; Hgb: hemoglobin; VO_2: oxygen consumption; HR: heart rate; BP: blood pressure (displayed as systolic/diastolic/mean); RAP: right atrial pressure; PAP: pulmonary pressure (displayed as systolic/diastolic/mean); PCWP: pulmonary capillary wedge pressure; SvO_2: mixed venous oxygen saturation; SaO_2: arterial oxygen saturation; CO: cardiac output; CI: cardiac index; SVR: systemic vascular resistance; PVR: pulmonary vascular resistance; BUN: blood urea nitrogen; IV: intravenous. [a]VO_2 was based on the patient's BSA multiplied by 125 mL and mixed venous oxygen saturation obtained from superior vena cava via left internal jugular central venous catheter.

an aggressive diuresis vs. initiation of inotropes strategy, point of care ultrasound to measure the inferior vena cava (IVC) diameter could be used as a surrogate of renal vein congestion. A plethoric IVC (> 21 mm not collapsing with inspiration), as well as presence of hepatojugular reflux and jugular vein distention could help to identify this clinical scenario in patients with favorable body habitus. For ventilated patients, the evaluation of hepatic veins by transthoracic echocardiography can guide the decision of whether or not the patient needs diuresis and the presence of right ventricular failure and elevated liver function tests supports this mechanism. In more complicated cases, where the information obtained at bedside is inconclusive, a right heart catheterization would be the gold standard to evaluate volume status before initiation aggressive diuresis.

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None to declare.

Conflict of Interest

None to declare.

Informed Consent

The manuscript has been sufficiently de-identified to protect the identity of each patient. Patients have since died and unable to provide informed consent.

Author Contributions

Sajid Mirza drafted the manuscript and provided the rest of the team with hemodynamic information from the case. He was involved with patient's care and had the idea to write this article. Ahmad Alkhalil drafted the manuscript with Sajid Mirza. He was involved in writing and editing. Edward Rojas edited the manuscript, contributed with review of the literature and discussion section. Arturo Perez-Peralta edited the text in the manuscript and created tables. Giselle A. Suero-Abreu edited the manuscript, adapted format to the journal guidelines. Njambi Mathenge reviewed later versions of the manuscript, suggested changes in the writing and discussion. Anthony Lucev edited the manuscript, contributed with the discussion section. Bernard Kim revised final version of the manuscript. Added content to the discussion and contributed to final redaction.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

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