Diagnosis and Management of Fluid Overload in Heart Failure and Cardio-Renal Syndrome: The "5B" Approach

Claudio Ronco, MD, Manish Kaushik, MD,* Roberto Valle, MD,† Nadia Aspromonte, MD,‡ and W. Frank Peacock IV, MD§*

Summary: Cardio-Renal syndrome may occur as a result of either primarily renal or cardiac dysfunction. This complex interaction requires a tailored approach to manage the underlying pathophysiology while optimizing the patient's symptoms and thus providing the best outcomes. Patients often are admitted to the hospital for signs and symptoms of congestion and fluid overload is the most frequent cause of subsequent re-admission. Fluid management is of paramount importance in the strategy of treatment for heart failure patients. Adequate fluid status should be obtained but a target value should be set according to objective indicators and biomarkers. Once the fluid excess is identified, a careful prescription of fluid removal by diuretics or extracorporeal therapies must be made. While delivering these therapies, adequate monitoring should be performed to prevent unwanted effects such as worsening of renal function or other complications. There is a very narrow window of optimal hydration for heart failure patients. Overhydration can result in myocardial stretching and potential decompensation. Inappropriate dehydration or relative reduction of circulating blood volume may result in distant organ damage caused by inadequate perfusion. We suggest consideration of the "5B" approach. This stands for balance of fluids (reflected by body weight), blood pressure, biomarkers, bioimpedance vector analysis, and blood volume. Addressing these parameters ensures that the most important issues affecting symptoms and outcomes are addressed. Furthermore, the patient is receiving the best possible care while avoiding unwanted side effects of the treatment.
Semin Nephrol 32:129-141 © 2012 Elsevier Inc. All rights reserved.
Keywords: Cardio-Renal syndrome, fluid overload, biomarkers, NGAL, bioimpedance, blood pressure, fluid balance

Fluid overload is a common result of cardiovascular disease (especially heart failure) and kidney disease. When the heart and kidney present a combined dysfunction such as in the case of cardio-renal syndromes (CRS), overhydration is almost the rule.[1,2] The diagnosis, objective quantification, and management of this problem is integral in attempting to improve clinical outcomes, including mortality and quality of life. Many clinical conditions lead to fluid overload, including decompensated heart failure and acute kidney injury (AKI) after the use of contrast media, the administration of nephrotoxic drugs (eg, amphotericin B), drugs associated with precipitation of crystals (eg, methotrexate, acyclovir), or shock caused by cardiogenic, septic, or traumatic causes. Adequate fluid status should be obtained but a target value should be set according to objective indicators and biomarkers. Once the fluid excess is identified, a careful prescription of fluid removal by diuretics or extracorporeal therapies must be made. While delivering these therapies, adequate monitoring should be performed to prevent unwanted effects such as worsening of renal function or other complications. Thus, the clinical challenge becomes the use of all currently available methods for objective measurement to determine the patient's volume status.

The term *CRS* is used to include the vast array of interrelated derangements between the heart and kidney, and to stress the bidirectional nature of their interactions. Generally, CRS are defined as pathophysiologic disorders of either organ system, in which acute or chronic dysfunction of one may induce acute or chronic dysfunction of the other.[3] CRS can be categorized into 5 subtypes that reflect the pathophysiology, time frame, and nature of concomitant cardiac and renal dysfunction (Table 1). CRS are therefore typical conditions in which fluid overload may occur and may require specific diagnosis and management. The various types of CRS may present with different signs and symptoms but fluid overload represents one of the common pathways toward hospitalization and bad outcomes.

For CRS type 1, the hemodynamic mechanisms induced by heart failure represent the etiologic events lead-

**Department of Nephrology Dialysis and Transplantation, International Renal Research Institute, St. Bortolo Hospital, Vicenza, Italy.*
†Department of Cardiology, Ospedale Civile, Chioggia, Venezia, Italy.
‡Cardiovascular Department, San Filippo Neri Hospital, Rome, Italy.
§Department of Emergency Medicine, Cleveland Clinic, Cleveland, OH.
Financial support for this work: none.
Financial disclosure and conflict of interest statement: Dr. Claudio Ronco receives speaking honoraria from Alere, Abbot, Gambro and Pfizer. Dr. Peacock receives Research Grants (>$10,000) Abbott, Alere, Baxter, Brahms, Novartis, The Medicine's Company; Consultant (<$10,000) Abbott, Alere, Lily, The Medicine's Company; Speaker's Bureau (<$10,000) Abbott, Alere; Ownership Interest (<$10,000) Comprehensive Research Associates LLC, Vital Sensors, Emergencies in Medicine LLC.
Address reprint requests to W. Frank Peacock IV, MD, Department of Emergency Medicine, Cleveland Clinic, Desk E19, 9500 Euclid Ave, Cleveland, OH 44195-0001. E-mail: Peacocw@ccf.org
0270-9295/ - see front matter
© 2012 Elsevier Inc. All rights reserved.
doi:10.1016/j.semnephrol.2011.11.016

Table 1. CRS

General definition
Pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ induces acute or chronic dysfunction in the other
CRS type I (acute CRS)
Abrupt worsening of cardiac function leading to AKI
CRS type II (chronic CRS)
Chronic abnormalities in cardiac function causing progressive and permanent chronic kidney disease
CRS type III (acute renocardiac syndrome)
Abrupt worsening of renal function causing acute cardiac disorders
CRS type IV (chronic renocardiac syndrome)
Chronic kidney disease contributing to decreased cardiac function, cardiac hypertrophy, and/or increased risk of adverse cardiovascular events
CRS type V (secondary CRS)
Systemic condition (eg, diabetes mellitus, sepsis) causing both cardiac and renal dysfunction

ing to sodium and water retention (Fig. 1). They basically can be summarized into two main aspects: the arterial underfilling and the venous congestion. Important compensatory mechanisms occurring in response to hemodynamic alterations can be divided into two phases: vasoconstriction or vasodilation (Fig. 2). In the first, activation of the sympathetic nervous system, renin-angiotensin-aldosterone system (RAAS), vasopressin, and endothelin result in decreased water and sodium excretion and, depending on the degree of renal functional impairment, increased urine concentration. To compensate for such condition, vasodilatation occurs via natriuretic peptide (NP) release, activation of the kinin-kallikrein system, secretion of vasodilatory prostaglandins, and expression of endothelial relaxation factor, thus increasing water and sodium excretion. However, this second phase may be inadequate to counter the initial vasoconstrictor effects, and disease progression may occur. In most cases, inappropriate water retention also is caused by a nonosmotic release of arginine-vasopressin that, in heart failure, worsens vasoconstriction via the stimulation of V1 receptors and dramatically increases the back-transport of water in the distal tubule via the stimulation of V2 receptors and aquaporin activation.

When cardiac disease (or heart failure) results in renal hypoperfusion, renal medullary ischemia is the consequence. Initially functional, it ultimately results in tissue damage. Further hypoperfusion and sustained tubular-glomerular feedback often will sustain the hemodynamic effect. In such clinical situations, the important objective is the maintenance of renal blood flow. This may be accomplished by acting on cardiac output, thus maintaining intravascular volume and renal perfusion pressure. There is a very narrow window of optimal hydration in such conditions. Overhydration can result in myocardial stretching and potential decompensation. Inappropriate dehydration or relative reduction of circulating blood volume may result in distant organ damage caused by inadequate perfusion. Renal function may be affected by both situations.

Efficient management of cardiac output requires optimization of heart rate, rhythm, preload, afterload, myocardial contractility, and, if required, surgical intervention in the instance of anatomic instability. Left ventricular assist devices are considered when these approaches fail.

Ultimately, knowledge of the degree of cardiac output is vital because there is no scientific case for fluid administration when the cardiac output exceeds 2.5 L/min/m^2 in patients not receiving inotropes. Particularly when sepsis is present, if the cardiac output is high and the patient is hypotensive, vasopressors, rather than fluids, are required irrespective of central venous pressure (CVP), pulmonary artery occlusion pressure (PAOP), or right ventricular end diastolic volume levels. Thus, in sepsis, vasopressors, such as norepinephrine (and dobutamine), are required to maintain renal perfusion even if the cardiac output is normal or high. If heart failure is present either with preserved left ventricular function or decreased ejection fraction, abnormal cardiac output and altered hemodynamics may result in oliguria and inappropriate water retention and diuretics and/or extracorporeal ultrafiltration must be considered.

In other words, fluids may be either required or removed in conjunction with other therapeutic strategies. In heart failure, most patients present with overhydration and the main strategy is to remove fluid and obtain the target hydration status in the absence of hemodynamic perturbation and worsening of renal failure.

5Bs

In the clinical routine, physicians are challenged by the unstable hemodynamics of the patient even in the presence of fluid overload and congestion. Strategies to achieve optimal hydration often include diuretic therapy or ultrafiltration even though a precise target for fluid status is missing. Thus, a comprehensive approach to fluid management is required: the following are five aspects of the approach to fluid overload in the context of CRS, a mnemonic termed the *5Bs* (Table 2).

Balance of Fluids

There is a large body of evidence suggesting that fluid overload is a dangerous situation.[4] In several studies a clear association between fluid balance and clinical outcome was shown. In a prospective cohort of 113 patients with acute respiratory distress syndrome (ARDS) evaluated for up to 14 days after intubation patients who lost 3 kg or more weight had a much higher survival rate than those who gained 3 kg or more weight (67% and 0%, respectively, on day 14) and these patients showed a lower daily/cumulative fluid balance.[5] In a retrospective analysis of 89 patients with acute lung injury (ALI)/ARDS who had pulmonary artery catheters and extravas-

Download English Version:

https://daneshyari.com/en/article/3896565

Download Persian Version:

https://daneshyari.com/article/3896565

Daneshyari.com