Correlation of Venous Congestion to Kidney Function in Patients with Decompensated Heart Failure

Thesis

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Sara Mohamed Amr

Contents

List of Abbreviations

Abb.	Full term
AII	:Angiotensin II
ACE	:Angiotensin converting enzyme
ACS	:Acute coronary syndrome
ADH	:Antidiuretic hormone
ADHF	:Advanced decompensated heart failure
ADHERE	:Acute decompensated heart failure national registry
ADMA	:Asymmetric dimethyl arginine
ADQI	:Acute dialysis quality initiative
AHF	:Acute heart failure
AKI	:Acute kidney injury
AKIN	:Acute kidney injury network
ANP	:Atrial natriuretic peptide
ARB	Angiotensin receptor blocker
BA	:Bronchial asthma
BP	:Blood pressure
BNP	:Brain natriuretic peptide
BUN	:Blood urea nitrogen
BW	:Body weight
CAD	:Coronary artery disease
CBC	:Complete blood count
CHF	:Congestive heart failure
CHOIR	:Correction of Hemoglobin and Outcomes in Renal Insufficiency
CD	:Cluster of differentiation
CI	:Cardiac index
CO	:Cardiac output
CKD	:Chronic kidney disease
Cr	:Creatinine
CrCl	:Creatinine clearance
CRS	:Cardiorenal syndrome

Abb.	Full term
CVP	:Central venous pressure
CVS	:Cerebrovascular stroke
CysC	:Cystatin C
DE	:Diuretic efficacy
DCs	:Dendritic cells
DM	:Diabetes mellitus
DOSE trial	:Diuretic Optimization Strategies Evaluation trial
DVT	:Deep vein thrombosis
EAFV	:Effective arterial filling volume
ESRD	:End stage renal disease
ESCAPE trial	:Evaluation Study of Congestive heart failure and Pulmonary Artery Catheterization Effectiveness trial
E GFR	:Estimated Glomerular Filtration Rate
EF	:Ejection fraction
g/dL	:Gram per deciliter
g/L	:Gram per liter
Hb	:Hemoglobin concentration
HF	:Heart failure
HTN	:Hypertension
IAP	:Intra-abdominal pressure
ICU	:Intensive care unit
IHD	:Ischemic heart disease
IPF	:Interstitial pulmonary fibrosis
IU	:International unit
IV	:Intravenous
JVP	:Jugular venous pressure
K	:Potassium
KDIGO	:Kidney disease improving global outcome
Kg	:Kilogram
KIM	:Kidney injury molecule
L	:Liter
LVEF	:Left ventricular ejection fraction
MAP	:Mean arterial pressure
MDRD	Modification of Diet in Renal Disease:

Abb.	Full term
Mg	:Milligram
mg/kg	:Milligram per kilogram
MHz	:Mega hertz
MI	:Myocardial infarction
Ml	:Milliliter
mm Hg	:Millimeter mercury
mmol/L	:Millimol per liter
MSNA	:Muscle Sympathetic Nerve Activity
MUSIC	:Multi-Sensor Monitoring in Congestive Heart Failure
NADH	:Nicotinamide adenine dinucleotide
NADPH	:Nicotinamide adenine dinucleotide phosphate (reduced form)
Na	:Sodium
ng/ml	:Nanogram per milliliter
NAG	:N-acetyl-beta-D-glucosaminidase
NGAL	:Neutrophil gelatinase associated lipocalin
NO	:Nitric oxide
NKF	:National kidney foundation
NSAID	:Non steroidal anti inflammatory drugs
NYHA	:New York Heart Association Functional Classification
OR	:Odd ratio
Pg/ml	:Picogram per milliliter
PCWP	:Pulmonary capillary wedge pressure
PARKIS	:Parkinsonism
PVD	:Peripheral vascular disease
PEEP	:Positive end-expiratory pressure
RAP	:Right atrial pressure
RAAS	:Renin - angiotensin - aldosterone system
RBF	:Renal blood flow
RNS	:Reactive nitrogen species
ROS	:Reactive oxygen species
RVSP	:Right ventricular systolic pressure
RIFLE	:Risk, Injury, Failure, Loss of Kidney Function, and End-stage Kidney Disease
RV	:Right ventricle

Abb.	Full term
SD	:Standard deviations
S.cr	:Serum creatinine
SNS	:Sympathetic nervous system
SOAP	:The Sepsis Occurrence in Acutely ill Patients
SOD	:Superoxide dismutase
TNF	:Tumor necrosis factor
TREAT	:Trial to Reduce Cardiovascular Events with Aranesp
VAD	:Ventricular assist device
WRF	:Worsening renal function

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Introduction

The **Heart** and the **kidneys** share responsibility for maintaining hemodynamic stability and end-organ perfusion through a tight-knit relationship that controls cardiac output, volume status, and vascular tone. Connections between these organs ensure that subtle physiologic changes in one system are tempered by compensation in the other. As such, hemodynamic control remains stable through a wide range of physiologic conditions ***(Viswanathan and Gilbert, 2011)***.

The interaction between the heart and the kidneys is modulated by the cardiorenal axis. The sympathetic nervous system (SNS), renin-angiotensin-aldosterone system (RAAS), and arginine vasopressin (AVP) are the primary neurohormones that maintain the integrity of effective arterial blood volume, hence the cardiorenal axis ***(Blankstein and Bakris, 2008).***

Increasingly, heart-kidney interactions are being recognized as fundamentally important in the prognosis of each organ individually as well as the prognosis of the overall patient ***(Ronco et al., 2008a),*** as both acute and chronic heart failure may push the kidneys beyond their ability to maintain glomerular filtration, regulate fluid and electrolytes, and clear metabolic waste.

Additionally, kidney disease and heart failure have been suggested not to represent single clinical entities but rather to represent manifestations of a broader vascular injury associated with aging that affects multiple organs **(*Triposkiadis et al., 2012).***

The incidence of heart failure with preserved ejection fraction (HFPEF) remains enigmatic. Epidemiology suggests that it is common ***(Bhatia et al., 2006),*** and is reported to include about 50% of the general heart failure population **(*Fonarow et al., 2007)*** while the prevalence of HFPEF is still increasing over the last years when compared to the prevalence of heart failure with reduced ejection fraction (HFREF) **(*Bhatia et al., 2006).*** Its prevalence is higher in the elderly **(*McDonald, 2008).***

Acute kidney injury may complicate one-third of heart failure admissions resulting in a threefold increase in length of stay, a greater likelihood for hospital readmission, and a 22% higher mortality rate ***(Wencker, 2007).***

Renal dysfunction is one of the most important comorbidities in patients with chronic HF and is accentuated, or becomes more evident, during episodes of acute decompensated heart failure (ADHF) ***(Damman et al., 2014).***

The special relationship and the interdependence of the kidneys and the heart are well recognized. The manner in which dysfunction of one organ affects the other has recently led to the characterization of the cardiorenal syndrome (CRS). CRS is a complex disease in which heart and kidney are simultaneously affected and their deleterious effects are reinforced in a feedback cycle, with

accelerated progression of renal and myocardial damage **(*Ronco et al., 2010a).***

While it is true that decreased forward flow as a result of decreased cardiac output in decompensated heart failure can cause acute deterioration in kidney function, there are several reasons why this mechanism fails to completely explain the development of the CRS. From a broader point of view, taking into account the dynamic and close interplay between heart and kidney, the CRS has been recently viewed as "a pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction in one organ may induce acute or chronic dysfunction in the other organ" ***(Ronco and Maisel 2010).***

For quite some time, venous congestion has been suspected as a cause of renal dysfunction (RD). Notably, in experiments dating back to the 1860s, it was shown that partial occlusion of the renal vein led to an immediate decline in renal blood flow, glomerular filtration rate (GFR), and sodium excretion, with resolution of the abnormalities after relief of the congestion ***(Gnanaraj et al., 2013).*** Furthermore, abdominal congestion has been associated with CRS ***(Verbrugge et al., 2013).***

The sequence of events shown in studies in human subjects have also demonstrated that increased central venous and increased jugular venous pressure (JVP) on examination are associated with worsening kidney function ***(Damman et al., 2009)*** as well as increased mortality ***(Mullens et al., 2009).***