Cardiorenal syndrome type I recovery post heart rate correction - Cardiac Output is not only stroke volume.

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Abstract

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Keywords:

Heart rate, Cardiac output, Cardio-renal syndrome, pacemaker

Cardiorenal syndrome is an umbrella of disorders of heart and kidney in either an acute or chronic fashion where the determinant factors in pathophysiology are the hemodynamic cross-talk between these organs and the neurohormonal mechanisms. Cardiac output is the product of the heart rate and the stroke volume which implies that the reduction of any these components can cause low cardiac output state. Cardiac output can be calculated non-invasively using Transthoracic Echocardiography which has high levels of reliability and reproducibility in experienced hand. We describe a case of a patient where bradarrhythmia was responsible for low cardiac output state, despite normal ejection fraction, that manifested clinically in cardiorenal syndrome type I and resolved completely after pacemaker insertion.

1 |CASE REPORT

An 78-year-old female patient with history of hypertension and dyslipidemia presented to emergency department with acute dizziness and severe general fatigue of few hours duration. She denied chest pain or shortness of breath and she did not experience similar symptoms before. She had no history of kidney disease previously and her kidney function reported normal one month back on routine check-up. Her chronic medication included amlodipine 5 mg once daily and atorvastatin 40 mg once daily. Physical examination was remarkable for overweight (body mass index 29 kg/m2), severe bradycardia of 30 beat per minute of regular rate, blood pressure of 150/80 mmHg, normal respiratory rate and oxygen saturation. Neurological, chest and abdominal examination were unremarkable. Cardiovascular examination was remarkable for severe bradycardia with no additional heart sounds or murmurs. Her electrocardiogram revealed high grade Atrioventricular (AV) block in form of Second degree heart block with P: QRS ratio of 3:1 or higher, causing extremely slow ventricular rate (Figure 1).Patient admitted to critical care unit for continuous monitoring of vital signs. Her Laboratory results showed Haemoglobin of 12 g/dl, normal coagulation profile and normal liver function test. Serum electrolytes were also normal apart from mildly elevated serum potassium and elevated serum lactate of 3 mmol/L. Her kidney function revealed creatinine of 203 µmol/L, blood urea nitrogen (BUN) of 28.5 mmol/L and albumin of 3.5 g/dL which are new findings for her as compared with her baseline level one month back. Urine analysis showed no evidence of urinary tract infection and no other abnormality .Computed tomography of brain revealed no neurological insult. Her Echocardiography study revealed normal left ventricular size and systolic function with estimated ejection fraction of 65% and grade I diastolic dysfunction. No significant valvular pathology and normal function of right ventricle. Stroke volume (SV) was calculated using echocardiographic measurement of velocity time integral (VTI) at the left ventricular outflow tract (LVOT) (Figure 2) and cross-sectional area (CSA) of LVOT at aortic annulus and was of 65 ml per beat which is within the normal range, however due to low ventricular rate, the cardiac output was only of 1.9 Liter per minute and cardiac index of 1.1 liter per minute per meter2 (L/M/M2) and these value can explain the low cardiac output state.

To improve ventricular rate the patient was give atropine of 1 mg twice with no significant response and then dopamine infusion started. During stay in critical care patient remained dizzy with normal blood pressure recording with no improvement of heart rate despite dopamine infusion of 15 mic/kg/min. Follow up of renal function after 12 hours showed more raising of creatinine and BUN and she was anuric and remained bradycardic

Patient was diagnosed as an acute kidney injury and cardiorenal syndrome type I due to low cardiac output state related to low ventricular rate that most probably due to progressive atherosclerotic changes in the atrioventricular node. The decision was to insert temporary Transvenous single lead pacemaker with target heart rate of 60 beat per minute (B/min) (Figure 3) and higher to improve cardiac output. With heart rate of 60 B/min her cardiac output increased from 1.9 to 3.9 liter per minute. 2 hours post insertion of pacemaker the patient symptoms disappeared and urine output improved with gradual recovery of the kidney function. Permanent pacemaker inserted later on and patient discharged from hospital with normal kidney function.

In our case it was a key to recognize early the state of low cardia output state secondary to bradyarrhythmia with subsequent manifestation of acute kidney injury and cardiorenal syndrome type I. This early recognition lead to successful treatment with Transvenous pacemaker.

DISCUSSION

Cardiac output measurement is an important parameter in cardiac function assessment. The two components that defined CO are HR and SV and the product of these 2 components will determine the amount of blood pumped by the heart per each minute. The amount of blood that pushed out of the ventricles with every beat called stroke volume. The 3 variables that determine the SV are preload, contractility, and afterload of each ventricle. Depending on the metabolic needs of the body the range of cardiac output can widely vary. Cardiac index is defined as the cardiac output divided by the body surface area.

Transthoracic echocardiography (TTE) is a non-invasive tool that can be used to calculate the stroke volume of the left ventricle by measuring the following variables: 1- the velocity-time integral (VTI) at LVOT and

2- the cross-sectional area of the LVOT (1'). VTI is the distance that blood travels with each beat. Using pulse wave (PW) Doppler at LVOT site from Apical 5-Chamber View, the operator can trace the LVOT signal and get the value of LVOT VTI in centimeter (Figure2) (2'). On the other hand cross-sectional area of LVOT at aortic annulus can be calculated by measuring LVOT dimeter at aortic annulus in centimeter from Parasternal Long Axis View (2'). Assuming a circular geometry of the LVOT, CSA of LVOT can be calculated through the following formula: π (D/2)2 where D is LVOT diameter and π value is approximately 3.14. Having the above mentioned parameters, the SV can be calculated from the following formula: SV at LVOT (cm3) = CSA (cm2) x VTI (cm). Once Stroke volume value calculated then cardiac output is the product of SV by heart rate. The CO calculation using TTE has shown very good correlation with thermodilution-derived cardiac output measurements (3') that considered gold standard of CO measurement.

CRS is an umbrella of pathologies of heart and kidney in which dysfunction of one organ may lead to similar dysfunction of the other organ and can be either in an acute or chronic condition (4').Based on which organ failing first, in 2008 two major categories had been identified as cardiorenal and renocardiac syndromes(5'). The sequential involvement of organs and the acuity of disease can distinguish 5 types of CRS according to the Consensus Conference of the Acute Dialysis Quality Initiative. CRS type I and II consistent of acute and chronic cardiorenal syndrome respectively, whereas CRS type III and IV imply acute and chronic renocardiac syndrome respectively. CRS type 5 is a secondary CRS where a systemic process resulting in heart failure and kidney failure. The determinant factors in pathophysiology of CRS are the hemodynamic cross-talk between organs and the activation of neurohormonal systems (Renin Angiotensin Aldosterone axis, sympathetic nervous system, and arginine vasopressin secretion), which will take place as a response to hemodynamic factors (6). The venous congestion or reduction in the cardiac output as a result of cardiac dysfunction will lead to reduction in glomeral filtration rate (GFR) in CRS Type I and II (7'). Type I CRS is a common condition in 25% to 33% of patients admitted with acute decompensated heart failure (8'). Ronco et al described in the literature 4 subtypes of type I CRS (9'). Subtype 1 of CRS type I consistent of new cardiac injury with subsequent new kidney injury whereas subtype 2 is a new cardiac injury that result in acute-on-chronic kidney injury. Subtype 3 happens when an acute-on-chronic cardiac decompensation leads to new kidney injury and whenever acute-on-chronic cardiac decompensation leads to acute-on-chronic kidney injury, subtype 4 considered (9'). Aoun et al reported a case of severe bradycardia as a reversible cause of cardio-renal –cerebral syndrome (10).

Our case demonstrated a subtype 1 of CRS type I when a bradyarrhythmia resulted in denovo cardiac injury and low cardiac output state that subsequently led to acute kidney injury. Early recognition of the role of severe bradyarrhythmia in the low cardiac output state using TTE resulted in early correction of a reversible cause with subsequent recovery of kidney function.

CONCLUSION

Cardiac output is not only stroke volume. Severe bradyarrhythmia can be a cause of low cardiac output state and lead to cardiorenal syndrome type I.

CONFLICT OF INTEREST

The author(s) declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article

AUTHOR CONTRIBUTIONS

OM: wrote the article, ZB and TZ shared in the discussion and, with MA,BH,MA and AH, in collecting the data and revision of the manuscript. Our working website is www.kockw.com (Kuwait Oil Company, Ahamdi Hospital).

CONSENT

Informed consent was obtained from the patient for the publication of this case report.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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