Cardiogenic Shock Evolving into Acute Kidney Injury: the Importance of Early Treatment

Ana Paula Romagnoli Mattos, Carlos Eduardo Cardoso*, Aparecida Carmem de Oliveira, Alexandre Mitsuo Mituiassu, Marco Orsini, Marcos RG de Freitas, Eduardo Tavares Lima Trajano and Marco Aurélio dos Santos Silva

Professional Master’s Program Applied in Health Sciences and Medical Graduation Course of Severino Sombra University for Vassouras University, Brazil

Received: April 01, 2018; Published: May 07, 2018

*Corresponding author: Carlos Eduardo Cardoso, Professional Master’s Program Applied in Health Sciences of Severino Sombra University, Vassouras, Rio de Janeiro, Brazil

Abstract
Cardiorenal syndrome (CRS) can be defined as a clinical-pathological disorder in which a primary insult in the kidney or heart initiates a series of functional and morphological secondary dysfunctions with responses in the other organs. A 63 year-old male patient with heart failure secondary to ischemic heart disease presented decompensation associated with hyperkalemia, evolving cardiogenic shock and acute kidney injury (AKI). In this case, an early use of inotropic medications to correct the low-output syndrome, associated with the identification of the AKI, was the differential that contributed to the positive outcome of the case.

Introduction
Cardiorenal syndrome (CRS) can be defined as a clinical-pathological disorder in which a primary insult in the kidney or heart initiates a series of functional and morphological secondary dysfunctions with responses in the other organs [1]. Subtype 1 (acute CRS) is characterized by rapid worsening of cardiac function leading to acute kidney injury (AKI) [2]. The definition of acute kidney injury (KDIGO-2012) is based on three criteria: increased serum creatinine higher or equal to 0.3 mg/dL within 48 h or increased serum creatinine higher or equal to 1.5 times the baseline value, occurring in the last 7 days or deficit less than 0.5 ml/kg/h for more than 6h. Although a relatively common syndrome, treatment of a cardiorenal syndrome requires particularities, such as optimization of cardiac output with inotropic drugs, continuous intravenous use of diuretics, combination of diuretics, and sometimes ultrafiltration. Therefore, the presence of the cardiorenal syndrome is the translation of a more serious clinical situation, requiring an individualized therapy regarding the risk that each patient presents for the development of renal dysfunction and careful to avoid worsening of the clinical condition in the short term. Whenever possible you should choose drugs that help preserve kidney function [3]. The objective of the study was to report a case of CRS type 1, originating from a cardiogenic shock that resulted in AKI and to show the importance of the early treatment for the positive outcome of the case.

Case Report
A 63-year-old male patient with heart failure secondary to ischemic heart disease presented decompensation associated with hyperkalemia, evolving cardiogenic shock and AKI. He entered the emergency room of the University Hospital of Vassouras with reports of chills, paresthesias of lower limbs and hands, vertigo, drowsiness, cold extremities and oliguria about 3 days ago. Patient with previous history of alcoholism and smoking presented Diabetes Mellitus, Gout, acute myocardial infarction for 2 months with angioplasty with placement of a stent in the anterior descending coronary. The test was lucid, oriented, hypertensive (AP: 80x60 mmHg), bradycardic (HR: 44bpm), with slow peripheral capillary perfusion and pathological jugular turgor.

Regular heart rhythm, bibasal lung crepitations, and lower limb edema. He came in regular use of Furosemide, Spironolactone, Carvedilol, Digoxin, AAS, and Clopidogrel. Hospital admission tests showed potassium of 7.9 mmol/L, urea 140 mg/dL, creatinine 2.9 mg/dL, glomerular filtration estimation (MDRD): 23.5 mL/m²/1.73m², creatinine Clearance 24.5 mL/min. Gasometry: pH: 7.43; pCO₂ 29.7 mmHg; pO₂ 80 mmHg; HCO₃ 19.8 mmol/L; sO₂c 96.3% Initial measures were performed to treat hyperkalemia with calcium gluconate, glycosulphone therapy and nebulization with fenoterol. Inotropic and vasopressor support was performed with Dobutamine and Noradrenaline. Patient evolved with clinical exercise.
improvement, with resolution of symptoms of low cardiac output, increased urine output and progressive improvement of renal function. Diagnoses: CRS type 1, uncompensated heart failure cold / wet pattern.

Discussion

Cardiorenal Syndrome, despite being a relatively common condition, has a high mortality, so a rapid recognition of the syndrome as well as rapid intervention and correct treatment are very important. The patient has impaired cardiac function, therefore the decompensation of this cardiac insufficiency of the patient associated with worsening renal function and hyperkalemia characterizes Cardiorenal Syndrome type 1. The treatment for Cardiorenal Syndrome was instituted soon at the hospital admission. Inotropic drugs and diuretics, which despite being a treatment choice, have a significant effect on the type 1 CRS, the reduced response to the congestive state, the decreased response to diuretics may result from physiological phenomena of diuretic breaking (decreased diuretic efficacy secondary to post-diuretic sodium retention) [4] and post-diuretic sodium retention [5]. If diuretic-resistant fluid overload exists despite an optimized cardiac output, withdrawal of isotonic fluid can be achieved by the use of extracorporeal ultrafiltration [6,7].

Patient has evolved with hypotension, characterizing cardiogenic shock, treated with positive vasoactive and inotropic drugs. Digital Suspension because Calcium Gluconate enhances the digitalis effect and may cause digitalis intoxication [8]. Attention should be paid to the preservation of renal function, perhaps with the same vigor as we try to save and protect the heart muscle. The worsening of renal function during ST-segment elevation myocardial infarction admission is a powerful and independent predictor of in-hospital mortality at 1 year [9,10]. In this context, an increase in creatinine and cardiovascular risk in patients with systolic dysfunction after myocardial infarction. Am Soc Nephrol 17(10): 2886-2891.

Conclusion

Given that the presence of type 1 CRS defines a population with high mortality, a rapid, systematic, multidisciplinary approach involving cardiologists, nephrologists, intensive care physicians and cardiac surgeons is both logical and desirable. Therefore, in this case, an early use of inotropic medications to correct the low-output syndrome, associated with the identification of the AKI, was the differential that contributed to the positive outcome of the case.

References

1. Berbari AE, Mancia G (2010) Cardiorenal syndrome: Mechanisms, Risk and Treatment. Springer.
2. Ronco C (2008) Cardiorenal syndrome. Journal of the american college of cardiology 52 (19): 1527-1539.
3. Tang W, Mullens W (2009) Cardio-Renal Syndrome in Decompensated Heart Failure. Heart BMJ 96 (4): 255-260.
4. Ellison DH (1999) Diuretic resistance: physiology and therapeutics. SeminNephrol 19(6): 581-597.
5. Almeshari K, Ahkstrom NG, Capraro FE, Wilcox CS (1993) A volume independent component to postdiuretic sodium retention in humans. J Am Soc Nephrol 3(12): 1878-1883.
6. Ronco C, Ricci Z, Brendolan A, Bellomo R, Redogni F (2004) Ultrafiltration in patients with hypervolemia and congestive heart failure. Blood Purif 22: 150-163.
7. Costanzo MR, Guglin ME, Saltzberg MT (2007) Ultrafiltration versus intravenous diuretics for patients hospitalized for acute decompensated heart failure. J Am Coll Cardiol 113(49): 675-683.
8. Vieira Neto OM, Moyzes Neto M (2003) Distúrbios do equilíbrio hidroeletrolítico. Medicina, Ribeirão Preto 36: 325-337.
9. Jose P, Skali H, Anavekar N (2006) Increase in creatinine and cardiovascular risk in patients with systolic dysfunction after myocardial infarction. Am Soc Nephrol 17(10): 2886-2891.
10. Goldberg A, Hammerman H, Petcherski S (2005) Inhospital and 1-year mortality of patients who develop worsening renal function following acute ST-elevation myocardial infarction. Am Heart J 150(2): 330-337.
11. Berl T, Henrich W (2006) Kidney-heart interactions: epidemiology, pathogenesis, and treatment. Clin J Am Soc Nephrol 1: 8-18.
12. Tokuyama H, Kelly DJ, Zhang Y, Gow RM, Gilbert RE (2007) Macrophage infiltration and cellular proliferation in the non-ischemic kidney and heart following prolonged unilateral renal ischemia. Nephron Physiol 106(3): 54-62.