



Case Report

Acute cardiac tamponade: A unusual cause of acute kidney injury

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ABSTRACT

Cardiac Tamponade is usually consequence of increased pericardial pressure with accumulation of pericardial effusion. We are reporting a case of acute kidney injury after mitral valve replacement due to acute pericardial effusion with clot. A 38-year-old female presented to CTVS department of our institute with history of palpitations and shortness of breath. On further examination and investigations she was diagnosed with severe mitral stenosis. Mitral valve replacement was done for her. In post operative period she developed cardio renal syndrome due to cardiac tamponade which presented as acute kidney injury. Re-exploration was done and clot was removed. She was shifted to ward after post operative monitoring and extubation.

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1. Introduction

Cardiac tamponade is usually consequence of increased pericardial pressure with accumulation of pericardial effusion. It may be caused by acute pericarditis, trauma, tumour, uraemia, hypothyroidism, cardiac surgery or other inflammatory/non-inflammatory conditions.

We are reporting a case of acute kidney injury after mitral valve replacement due to acute pericardial effusion with clot. The patient had normal baseline renal function but developed acute oliguric renal failure with significant increase in serum creatinine postoperatively. Pericardiotomy with clot removal led to improvement in blood pressure, immediate diuresis and quick recovery of renal function back to baseline.

2. Case Report

A 38-year-old female presented to CTVS department of our institute with history of palpitations and shortness of breath for the past 8 years which had been recently aggravated. On further history, it was found that she had history of CVA (cerebro vascular accident) with right hemiparesis 10

years back. Old records of the patient showed an area of encephalomalacia with gliosis in left fronto parieto temporal and ganglio capsulo region of left MCA (middle cerebral artery) territory with dilation of left lateral ventricular region. On examination, it was found that she had right sided residual weakness and late diastolic murmur with loud S2 on auscultation. When further investigations were done, it was found in echocardiography that she had severe MS (mitral stenosis) (MVA of 0.5 cm²), severe TR (Tricuspid regurgitation), severe PAH (pulmonary artery hypertension), dilated LA (left atrium) with clot of size of 51×29 mm and LVEF (Left ventricular ejection fraction) of 55%. ECG findings were showing atrial fibrillation. The patient had normal renal function prior to surgery.

Patient was started on tab. Digoxin 0.25mg OD, tab. Acitrom 2mg HS, tab. Ecospirin 75 mg OD, tab. Toremide 10 mg OD, tab. Metoprolol 25 mg OD. Then she was planned for an elective mitral valve replacement with LA clot removal and TV repair. Later patient was operated as planned and no intraoperative major events. Perfusion pressure of 60 mm Hg was maintained during cardio pulmonary bypass. Patient was shifted to ICU without extubation. While monitoring the patient for two hours it was found that patient developed hypotension and atrial

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fibrillation. Patient was kept on amiodarone infusion at the rate of 1mg/ min after intravenous bolus of 150 mg. Even then hypotension persisted Hence cardioversion was done with 100J of DC. Rhythm was reverted but hypotension persisted and urine output were on decreasing trend (5 to 10ml/hr). Patient was kept on noradrenaline infusion at the rate of 0.08 mcg/kg/min. Chest drains were checked frequently. Around 100 ml of blood was present and we found that there was no blockage of chest tube drain. On taking serial ABG(arterial blood gas), PH was 7.0, PCO₂= 43 mm Hg, PaO₂ of 200 mm Hg, HCO₃ of 14 mmol/dL and potassium levels were found to be persistently high (K+=6.6 mmol/dL). Post operative chest x ray was done but there was gross enlargement of cardiac shadows found and left us inconclusive. So, USG abdomen was done to see IVC (inferior venacava) diameter. IVC diameter was normal, so transthoracic echocardiogram was done in subcoastal view initially along with other views (paramedian short axis, short axis and apical) and it was found that LV was empty so IV fluids were given. Despite of giving IV fluids 1L over one hour, patient urine output remained low at 5ml/hr. On repeating and USG findings were same and urine output kept decreasing and serum potassium were the same. The patient serum creatinine increased to 2mg/dl from 0.9mg/ml prior to surgery. To evaluate postsurgical complications transoesophageal echocardiography was performed which revealed Pericardial tamponade with large clot compressing the SVC and right atrium was found. So, emergency pericardiotomy with clot removal was done. This led to improvement in blood pressure, immediate diuresis and quick recovery of renal function back to baseline. The patient was extubated on postoperative day 1 and patient was given furosemide 10 mg TDS for diuresis and warfarin started for anticoagulation. With intensive intermittent CPAP therapy and chest physiotherapy, the patient was shifted to ward on postoperative day 3.

3. Discussion

The episode of AKI occurred due to an acute decompensated cardiac failure as a result of pericardial tamponade following cardiac surgery. This illustrates the importance of heart-kidney interactions in immediate postoperative period. This explains the cardiorenal syndrome 1(CRS) and classification of cardio-renal syndrome (Table 1) proposed by Ronco et al.¹

The common causes for pericardial effusion include chronic kidney disease, infection, malignancy, collagen vascular disease, drugs, congestive cardiac failure and hypothyroidism.^{2,3} Acute kidney injury is also one of the cause secondary to acute pericardial tamponade following cardiac surgery.⁴

The Diagnosis of cardiac tamponade in postcardiac surgery is challenging due to the predisposition of localized pericardial fluid collection and the absence of a classical

Table 1: Cardio-Renal Syndrome classification¹

Class	Description
CRS 1	Acute HF causing AKI
CRS 2	Chronic HF causing progressive CKD
CRS 3	Acute cardiac dysfunction causing acute renal dysfunction
CRS 4	CKD causing chronic cardiac dysfunction
CRS 5	Systemic disorder causing both cardiac & renal dysfunction

Beck's triad (distended neck veins, distant heart sounds and hypotension) for the diagnosis of pericardial tamponade. When cardiac tamponade developed less than 72 hours after surgery, classical echocardiographic findings were absent in 80% patients.⁴ In our case, TEE was performed which revealed pericardial effusion with large clot compressing right atrium and SVC.

In cardiac tamponade, rapidly increasing fluid accumulation in the elastic pericardial cavity inhibits the filling of the right atrium. Once the intra-pericardial pressure exceeds that in the right atrium, cardiac output drops due to reduced filling of the right atrium and ventricle, leading to hypotension. Regulation of renal blood flow is important to maintain a stable GFR despite changes in systemic blood pressure (80-180 mmhg), below this level with hypotension, the kidney does not autoregulate. Therefore urine output falls in proportion to the reductions in arterial pressure. The subsequent reductions in renal perfusion trigger mechanism in the kidney in order to maintain a constant renal blood flow and glomerular filtration.

Autoregulation of blood flow is necessary to maintain constant organ perfusion despite variations in the arterial pressure. This function is present in all tissues mainly brain and kidney. Autoregulation of RBF is mainly caused by myogenic responses (MR) and tubuloglomerular feedback (TGF). TGF is a regulating mechanism in the kidney that leads to vasoconstriction of the afferent arteriole in response to an increase in the luminal concentration of NaCl at the macula densa in the early distal tubule. During hypotension, renal vascular resistance decrease in order to maintain renal blood flow and GFR largely through a MR and TGF.

In our case, the renal effects of cardiac tamponade have been occurred within 2 hours after cardiac surgery with severe hemodynamic collapse.⁵

A further increase in pericardial collection reduces both the mean arterial pressure and GFR. In this patient, prior to re exploration patient had a decrease in cardiac output, increase in right atrial pressure and high systemic vascular resistance, elevated potassium levels and nil urine output with adequate IV fluids. All these effects are reversed by re exploration and clot removal, as evidenced by normalization of mean arterial blood pressure and GFR. Thus, a strong clinical suspicion for the diagnosis of acute pericardial

tamponade led to acute oliguric kidney injury in postcardiac surgery.

4. Conclusion

Acute kidney injury may be due to pre-oxygenation renal, renal, post-renal causes. Cardiac tamponade is the pre-renal cause of AKI. Early diagnosis and intervention reverses the kidney injury. Transthoracic echocardiography, abdomen ultrasonography, serum electrolytes, urea, creatinine level, regular monitoring of urine output helps to rule out causes of AKI in post cardiac surgery patients. Patients should be managed according to the cause and if needed renal replacement therapy should be initiated.

5. Source of Funding

None.

6. Conflict of Interest

None.

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