Case Report

Acute Cardiac Tamponade: An Unusual Cause of Acute Renal Failure

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Key Words

Autoregulation • Acute renal failure • Tamponade

Abstract

We are reporting a case of acute renal failure after cardiac surgery due to acute pericardial effusion. The patient had normal baseline renal function but developed acute oliguric renal failure with a significant increase in serum creatinine postoperatively. Pericardiotomy led to an improvement in blood pressure, immediate diuresis and quick recovery of renal function back to baseline. Pericardial tamponade should be included in the consideration of causes of the cardiorenal syndrome.

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Case Presentation

A 65-year-old male underwent an elective coronary artery bypass graft X1 (LIMA to LAD) operation with an aortic valve replacement. The patient had a past medical history significant for coronary artery disease, hypertension, diabetes, micro-albuminuria and hyperlipidemia. The patient had normal renal function prior to surgery. The surgery and immediate postoperative care were uncomplicated and the patient was extubated on day 3. The same day, the patient was given furosemide for diuresis to facilitate extubation and warfarin was started for anticoagulation. On postoperative day 5, the patient's serum creatinine (sCr) increased to 2.35 mg/dl from 0.9 mg/dl prior to surgery. The Nephrology Department was consulted for an evaluation of acute renal failure (ARF). The patient had a net negative

Fig. 1. Renal function pre and post pericardiotomy.

Table 1. CRS classification

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

AKI = Acute kidney injury; HF = heart failure; CKD = chronic kidney disease.

fluid balance of 4.5 liters over the previous 2 days prior to nephrology consultation. His spot urine sodium was 22 mEq/l with a fractional excretion of sodium <1%, suggesting a pre-renal cause of ARF. Despite 3 liters of saline given over 12 h, the patient's urine output remained low at 5–15 ml/h (fig. 1). To evaluate postsurgical complications, a bedside echocardiogram was performed which revealed a pericardial effusion without right atrial compression or evidence of pulsus paradoxus. However, due to a strong suspicion of impending pericardial tamponade, the patient was taken to surgery for a pericardiotomy and 800 ml of hemorrhagic fluid was drained. After the pericardiotomy, the patient experienced an improvement in urine output and a substantial decline in sCr to his baseline value over the next few days.

Discussion

This case illustrates the importance of heart-kidney interactions in the acute care setting. The episode of ARF occurred due to acute decompensated heart failure as a result of a pericardial effusion. The finding that kidney function normalized following pericardiotomy highlights the complex relationship as a class 1 cardiorenal syndrome (CRS) and reinforces the utility of the cardiorenal classification scheme (table 1) in management as proposed by Ronco et al. [1].

Pericardial effusion is a known complication of chronic kidney disease with untreated advanced uremia [2, 3]. Other common causes of pericardial effusion include infection, ma-

lignancy, collagen vascular disease, drugs, trauma, congestive heart failure and hypothyroidism [4–6]. Few cases of ARF secondary to acute pericardial tamponade have been reported in the literature [7–9]. The diagnosis of cardiac tamponade in postcardiac surgery is challenging due to the predisposition for localized pericardial fluid collection and the absence of a classical Beck's triad (distended neck veins, distant heart sounds and hypotension) for the diagnosis of pericardial tamponade, as well as the reduced sensitivity of echocardiography to detect hemodynamically significant tamponade after cardiac surgery [6]. In a retrospective analysis of prospectively collected data on 2,297 patients who underwent cardiac surgery at a tertiary center, the diagnosis tamponade was confirmed in 148 patients on re-exploration. When cardiac tamponade developed <72 h after surgery, classical echocardiographic findings were absent in 80% of patients.

Normal mean right atrial filling pressures resulting from returning venous blood volume are approximately 6–8 mm Hg [6]. In cardiac tamponade, rapidly increasing fluid accumulation in the in-elastic pericardial cavity inhibits the filling of the right atrium. Once the intra-pericardial pressure exceeds that in the right atrium (and subsequently the right ventricle), cardiac output drops due to reduced filling of the right atrium and ventricle, leading to hypotension. While kidney autoregulation occurs over a wide range of arterial pressures (approx. 80–180 mm Hg), below this level – under conditions of hypotension – the kidney does not autoregulate. Thereby, urine flow falls in proportion to the reductions in arterial pressure. The subsequent reductions in renal perfusion trigger mechanisms 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 but is particularly pronounced in some organs like the brain and kidney. Autoregulation of renal blood flow is mainly caused by myogenic responses (MR) and tubuloglomerular feed back (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. In addition to MR and TGF, there are several other mechanisms that serve to regulate perfusion including vasopressin, natriuretic peptides, and activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system. During hypotension, renal vascular resistance decreases in order to maintain renal blood flow and glomerular filtration largely through a MR and TGF.

Since our patient was volume-depleted due to diuretic use, we hypothesize that a state of 'low-pressure cardiac tamponade' existed. Low-pressure cardiac tamponade is a form of cardiac tamponade in which a comparatively low pericardial pressure results in cardiac compression because of low filling pressure. The intra-pericardial pressure exceeded the right atrial pressure 'earlier' or at 'lower pressure' than seen in cases without volume depletion [9]. In a retrospective analysis, low-pressure cardiac tamponade was identified in 20% of patients with catheterization-based criteria of tamponade [10].

The renal effects of cardiac tamponade have been reported to occur even before hemodynamic collapse [7]. Increasing the pericardial pressure by 5 mm Hg decreases urinary sodium excretion and increases renin production; but the mean arterial blood flow and glomerular filtration rate (GFR) remain unchanged. A further increase in the pericardial pressure, however, reduces both the mean arterial pressure and GFR [7]. In a series of 16 patients with constrictive pericarditis, prior to pericardiectomy patients had a decrease in cardiac output, increase in right atrial pressure, elevated pulmonary capillary wedge pressure and presence of high systemic and pulmonary vascular resistance [11]. The plasma concentrations of norepinephrine, renin and aldosterone were also elevated in these patients. All these effects are reversed by drainage of pericardial fluid, as evidenced by a reversal of the vasoconstriction state, normalization of mean arterial pressure and eventually normalization of GFR.

Thus, a strong clinical suspicion for the diagnosis and a low threshold for emergent pericardial drainage are necessary to prevent ARF.

Acute pericardial tamponade as a cause of oliguric renal failure in postcardiac surgery should be considered in the long list of CRSs, especially CRS type 1. In some cases, this is associated with elevated liver enzymes, a sodium-avid state with or without volume depletion and complete recovery of renal function following treatment of the primary disorder.

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