

Effects of Pericardiocentesis on Renal Function and Cardiac Hemodynamics

Maulin Shah¹, Hezzy Shmueli², Yatindra Patel³, Long-Co Nguyen³, Hannah Hardy¹, Florian Rader¹, and Robert J. Siegel¹

¹Cedars-Sinai Medical Center Smidt Heart Institute

²Soroka Medical Center

³Cedars-Sinai Medical Center Medicine Department

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Abstract

Background: Previous case studies have reported reversal of acute renal failure after pericardiocentesis in pericardial effusion. This study examines the effects of pericardiocentesis on pre-procedural low cardiac output and acute renal dysfunction in patients with pericardial effusion. **Methods:** This is a retrospective study of 95 patients undergoing pericardiocentesis between 2015 and 2020. Pre- and post-procedure transthoracic echocardiograms (TTE) were reviewed for evidence of cardiac tamponade, resolution of pericardial effusion, and for estimation of right atrial (RA) pressure and cardiac output. Laboratory values were compared at presentation and post-procedure. Patients on active renal replacement therapy were excluded. **Results:** Ninety-five patients were included for analysis (mean age 62.2 ± 17.8 years, 58% male). There was a significant increase in glomerular filtration rate pre- and post-procedure. Fifty-six patients (58.9%) had an improvement in glomerular filtration rate after pericardiocentesis (termed “responders”), and these patients had a lower pre-procedure glomerular filtration rate than “non-responders”. Forty-four patients (46.3%) had a greater than 10% improvement in glomerular filtration rate. There was a significant improvement in estimated cardiac output and right atrial pressure for patients in both groups. Patients who had an improvement in renal function had significantly lower pre-procedural diastolic blood pressure and mean arterial pressure. **Conclusions:** Pericardial drainage may improve effusion-mediated acute renal dysfunction by reducing right atrial pressure and thus systemic venous congestion, and by increasing forward stroke volume and perfusion pressure.

Introduction

The heart is encased by the pericardium, a sac with an inner, epicardial layer and an outer layer called the parietal pericardium. In addition to maintaining the heart in a relatively fixed position and serving as a barrier to infection, the mechanical properties of the pericardial tissue create a restraining effect on cardiac volume. At low stress, the pericardial tissue is very elastic. However, with increased stretch, it becomes stiff and resistant to further distention. This can result in increased cardiac filling pressures and impaired cardiac filling, leading to a reduction in cardiac output. Fluid accumulation between the visceral and parietal pericardium is a common mechanism of increased pericardial stretch and decreased compliance. Pericardial effusion can occur as a result of a variety of clinical conditions, including inflammation, infection, autoimmune disease, trauma, metabolic derangement (e.g., uremia), neoplasm and idiopathic processes (1).

The effect of intrapericardial fluid on cardiac hemodynamics depends on the time-course of accumulation. If the effusion accumulates rapidly, even 150 mL (2) can cause hemodynamic compromise and cardiac tamponade. Pericardial inflammation and effusions are often treated with colchicine and/or non-steroidal anti-inflammatory medications. If the effusion is not responsive to medical therapy, pericardial drainage may be required. Indications for pericardiocentesis include hemodynamic compromise due to tamponade, large

refractory effusion (>20 mm diameter measured with echocardiography), or for diagnostic necessity (3). In cases of cardiac tamponade, certain echocardiographic features often precede hemodynamic compromise and, if detected, may be an indication for pericardiocentesis before clinical instability occurs. These signs include diastolic right ventricular (RV) collapse (high specificity) (4), late diastolic or systolic right atrial (RA) collapse (earliest sign) (5), plethoric inferior vena cava with minimal respiratory variation (high sensitivity) (6) and exaggerated respiratory cycle changes ($>25\%$ respirophasic change of mitral inflow velocity and $>40\%$ respirophasic change of tricuspid valve inflow velocity) (7).

The accumulation of fluid in the pericardial space can increase intrapericardial pressure. This increased pressure is transmitted to the RV and results in exaggerated interventricular dependence, resulting in impaired diastolic left ventricular filling, decreased cardiac output, and multi-organ failure (8). Previous case studies have reported reversal of renal failure caused by pericardial effusion after treatment with pericardiocentesis (9-14).

In this study, we assessed the relationship between pericardiocentesis and resolution of pre-procedural low cardiac output and acute renal dysfunction. To our knowledge, this is the largest study examining the effect of pericardiocentesis on cardiac hemodynamics and renal function.

Methods

This is a retrospective study of patients requiring pericardiocentesis at Cedars-Sinai Medical Center from 2015 to 2020. We reviewed all available pre- and post-pericardiocentesis transthoracic echocardiograms (TTE) in this group of patients. Echocardiographic tamponade was defined as follows: diastolic RV or RA collapse, plethoric inferior vena cava (IVC) with minimal respiratory variation (thus estimation of high RA pressure) and exaggerated respiratory cycle changes ($>25\%$ decrease in mitral inflow velocity or $>40\%$ increase in tricuspid valve inflow velocity). RA pressure was estimated according to IVC size and presence of inspiratory collapse. Mean stroke volume was expressed via mean left ventricular (LV) outflow tract velocity-time integral (LVOT VTI) [stroke volume (cm^3) = LVOT area (cm^2) \times LVOT VTI (cm)]. Post-pericardiocentesis TTE was performed within 24 hours after the procedure. All complementary data were extracted from the electronic medical record. Renal function was measured via glomerular filtration rate (GFR), calculated according to the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI), as recommended by the National Kidney Foundation (15). Laboratory results were taken from the admission values of the index hospitalization and again from the first collection after the pericardiocentesis was performed, all within 24 hours of the procedure. Blood pressures were measured non-invasively by standard cuff sphygmomanometer.

Descriptive data is presented as frequency (n) and percentage for categorical variables and mean \pm standard deviation (SD) for continuous variables. Pre- and post-procedure variables were compared for each patient using paired sample t-test and chi square statistics as appropriate, and effect size was calculated using Cohen's d statistic. Normality of data was assumed on visualization of P-P plot. Variables between "responders" [glomerular filtration rate (post-procedure) – glomerular filtration rate (pre-procedure) > 0] and "non-responders" [glomerular filtration rate (post-procedure) – glomerular filtration rate (pre-procedure) ≤ 0] were compared using independent sample t-test or analysis of variance (ANOVA). The level of significance was set at $p < 0.05$. Data analysis was performed using SPSS v.24.0 (SPSS Inc, Chicago, IL). This study was approved by our institutional review board. Informed consent was not required due to the nature of this study. This study received no internal or external funding. Given the retrospective nature of this study, patients were not directly involved in the design, conduct, recruitment, dissemination or reporting of this study.

Results

There were 160 patients who underwent pericardiocentesis in our tertiary medical center between January 2015 – June 2020, and 95 were included for analysis. Forty-four cases were excluded due to missing complete TTE or lab results. An additional 21 cases were excluded due to active renal replacement therapy (hemodialysis, peritoneal dialysis or continuous renal replacement therapy).

Table I shows the demographic and blood pressure values pre- and post-pericardiocentesis of the study population. Mean age was 62.2 \pm 17.8 years, and 58% of patients were male. The main etiologies of pericardial effusion were post-cardiac surgery (n=32, 33.7%), malignancy (n=30, 31.6%), infectious or inflammatory pericarditis (n=14, 14.7%) and idiopathic (n=19, 20%). Seven patients (7.4%) died during the index hospitalization, and 3 more (3.2%) died within 30 days of admission, all of whom had progressive oncologic disease and malignant pericardial effusion. Fourteen patients (14.7%) presented with hypotension/shock (Systolic Blood Pressure [SBP] < 80mmHg) and 3 patients presented with infective endocarditis (3.2%). The most common chronic medications taken were beta blockers (n=37, 38.9%) and any anticoagulation (n=34, 35.8%). Sixty-eight patients (71.6%) were taking at least one anti-hypertensive medication. Colchicine treatment was given to 88 patients (92.6%) during the index admission and 37 patients (38.9%) were prescribed NSAIDs. On average, patients had a lower GFR prior to pericardiocentesis (72.0 \pm 31.7 mL/min/1.73 m²) than after pericardiocentesis (77.8 \pm 32.0 mL/min/1.73 m²). This difference, 5.8 (95% CI [2.93, 8.59]), was statistically significant (p < 0.001), and represented an effect size of 0.18 (moderate effect). Forty-four patients (46.3%) had an increase in GFR > 10% from baseline (**Table II**).

Table III shows the echocardiography-Doppler related measurements of the entire study group. In 77 cases (81.1%), tamponade signs or physiology were present on admission. The fluid was circumferential to the heart in 84 cases (88.4%) and was moderate-large (n = 41, 43.1%) or large (n = 42, 44.2%) in the majority of cases. In 61 cases (64.2%) the IVC was > 20 mm in diameter. Two patient examples are shown in **Figure 1** and **Figure 2**.

Pre- and post-procedure RA pressures as estimated by TTE were available in 81 patients (85%). The remaining patients either had poor quality view of the IVC or were mechanically ventilated, precluding IVC estimation of RA pressure. Pre-procedural RA pressure was estimated to be ~3 mmHg in 9 cases (11.1%), ~8 mmHg in 30 cases (37.0%) and [?]15 mmHg in 42 cases (51.9%). Post-procedure TTE revealed 49 cases (60.5%) with RA pressure ~3 mmHg. The proportion of patients with improved estimated RA pressure post-procedurally was statistically significant (p < 0.001).

Pre- and post-procedure LVOT-VTI data was available for 59 patients (62.1%). Mean pre-procedure LVOT-VTI was 17.3 \pm 6.9 cm and increased to 20.7 \pm 6.5 cm post-procedurally. This difference, 3.4 cm (95% CI [1.82, 4.83]) was statistically significant (p<0.001) and represented an effect size of 0.52 (medium effect).

Table IV (supplementary data) shows pre- and post-pericardiocentesis data for the study group divided into patients that had an increase in GFR post-pericardiocentesis (“Responders”, GFR (post-procedure) – GFR (pre-procedure) > 0, n=56, 58.9%) versus patients that did not have an increase in GFR post-pericardiocentesis (“Non-responders”, GFR (post-procedure) – GFR (pre-procedure) [?] 0, n=39, 41.1%). The groups were largely similar in demographic, echocardiographic and laboratory values, differing only in pre- and post-procedure mean creatinine (Responders: 1.29 \pm 0.72 mg/dL vs. Non-responders: 1.0 \pm 0.37 mg/dL, p=0.022). However, pre-procedure hemodynamic measurements were different between the two groups. GFR responders had a significantly lower pre-procedure diastolic blood pressure (Responders: 70.5 \pm 14.4 mmHg vs. Non-responders: 77.4 \pm 15.0 mmHg, p=0.026). Additionally, responders had decreased levels of overall pre-procedure organ perfusion as assessed by pre-procedure mean arterial pressure (MAP), diastolic perfusion pressure (DPP; DPP = DBP – RA pressure), and mean perfusion pressure (MPP; MAP – RA pressure). These were all found to be significantly lower in the “responder” group, although DPP and MPP were based only on RA pressure estimates. There was no significant difference in amount of fluid drained during the procedure for responders (579 \pm 324 mL) versus non-responders (588 \pm 274 mL) (p=0.423). For the 81 patients in whom both pre- and post-procedure RA pressures were available, improvement in RA pressure was seen in both responders (n=47) and non-responders (n=34). For non-responders (n=34), pre-procedural RA pressure was estimated to be ~3 mmHg in 5 cases (14.7%), ~8 mmHg in 14 cases (41.2%) and [?]15 mmHg in 15 cases (44.1%), while post-procedural RA pressure was estimated at ~3 mmHg in 23 cases (67.6%), ~8 mmHg in 5 cases (14.7%), and [?]15 mmHg in 6 cases (17.6%). The pre- and post-procedural differences in estimated RA pressure in patients with no improvement in GFR was statistically significant (p=0.049). For responders (n=47), pre-procedural RA pressure was estimated to be

~3 mmHg in 4 cases (8.5%), ~8 mmHg in 16 cases (34.0%) and ≥ 15 mmHg in 27 cases (57.4%), while post-procedural RA pressure was estimated at ~3 mmHg in 26 cases (55.3%), ~8 mmHg in 13 cases (27.7%), and ≥ 15 mmHg in 8 cases (17.0%). The pre- and post-procedural differences in estimated RA pressure in patients with improvement in GFR was statistically significant ($p < 0.001$). For the 59 patients in whom both pre- and post-procedure LVOT-VTI measurements were available, the mean difference in LVOT-VTI (LVOT-VTI post-procedure – LVOT-VTI pre-procedure) was 3.33 ± 5.48 in GFR responders ($n=36$) versus 3.32 ± 6.32 in GFR non-responders. This difference was not statistically significant ($p=0.840$).

Discussion

This study examines the effects of pericardiocentesis on renal function and cardiac hemodynamics in the setting of significant pericardial effusion and echocardiographic tamponade. The main finding of this study is that pericardiocentesis in patients with cardiac tamponade can improve renal function, RA pressure and forward stroke volume (as measured by LVOT-VTI). The improvement in RA pressure and LVOT-VTI was independent of improvement in renal function in our study, as patients who had an improvement in GFR (“responders”) had similar changes in pre- and post-procedural RA pressure and LVOT-VTI as patients who did not have an improvement in GFR (“non-responders”). We hypothesize that this finding is explained by the pre-procedure difference in GFR, as responders had a significantly lower pre-procedural GFR than non-responders. The difference in pre-procedure GFR may be related to several hemodynamic measurements indicating that GFR responders had worse pre-procedural organ perfusion. First, GFR responders had decreased MAP when compared to non-responders. Moreover, DPP and MPP were also significantly lower in GFR responders, although these measurements were based on RA pressure estimates. Several studies have shown that DPP and MPP are associated with acute kidney injury (AKI) in critically ill patients (16-19), highlighting the important role of central venous pressure in organ perfusion. Pericardiocentesis likely reduces elevated renal vein pressure (as measured by RA pressure) and increases forward stroke volume (as measured by LVOT-VTI), which then improves renal perfusion and function in patients who had prior hemodynamic susceptibility to AKI. This improvement is notably similar to decompensated, low contractility states with respect to the Frank-Starling law. As RA pressure *decreases*, there is a concomitant increase in stroke volume. In normal cardiac physiology, the *increase* in RA pressure induces an increase in stroke volume.

Acute renal failure (ARF) resulting from pericardial effusions with and without tamponade has been described in case reports and retrospective analyses (9-14, 20). In each of these studies, ARF was completely reversed after pericardial drainage. Several mechanisms have been suggested to explain the pathophysiology of AKI in this setting. These include a significant decrease of transmural pressure and preload leading to decreased cardiac output resulting in renal ischemia, reduction of GFR and prerenal azotemia (12). Hormonal changes have also been implicated and demonstrated in animal models. Previous reports have shown that increasing the pericardial pressure by 5 mmHg decreases urinary sodium excretion and increases the renin secretion rate without changing the mean arterial pressure, renal blood flow, or GFR. However, a further increase in pericardial pressure to 10 mmHg decreases urinary sodium excretion, mean arterial pressure and GFR (21). These results indicate that small increases in pericardial pressure caused by effusion may alter renal hemodynamics leading to reduced GFR and oliguric ARF. In a series of 16 patients with constrictive pericarditis, patients had a decrease in cardiac output, increase in RA pressure, elevated pulmonary capillary wedge pressure and presence of high systemic and pulmonary vascular resistance (22). The plasma concentrations of norepinephrine, renin and aldosterone were also elevated in these patients. After pericardiectomy all measurements returned toward baseline levels, with reversal of the vasoconstriction state, normalization of mean arterial pressure and eventually normalization of GFR (22).

The precise mechanism of renal function recovery after pericardiocentesis is unknown. Renal blood flow is dependent on the precipitous drop in resistance through an extensive network of parallel branching vessels, which is primarily controlled at the afferent and efferent arterioles. As blood flows through the renal system, it reaches a pressure minimum at the renal vein and ultimately flows back into systemic circulation (23). However, as we found in our study, there is a significant increase in RA pressure in the setting of pericardial

effusion or echocardiographic tamponade. This increased pressure leads to effusion mediated elevation of renal vein pressure, which likely adversely alters the pressure gradient from renal arterioles to renal vein. Animal models have shown that sustained increase in renal venous pressure leads not only to decreased renal artery blood flow and oliguric renal failure, but also increased plasma renin activity, serum aldosterone, and urinary protein leak (24). Moreover, these studies showed improvement in renal function as renal venous pressure was normalized which is consistent with our findings.

Notably, nine patients in our study had normal estimated pre-procedural RA pressures. Although plethoric IVC and lack of inspiratory collapse are often considered essential to the diagnosis of cardiac tamponade, a study examining 279 patients with large pericardial effusion found that low-pressure cardiac tamponade was present in 9.6% of patients with large pericardial effusion and in 20% of patients meeting catheterization-based criteria for cardiac tamponade (25). Our analysis corresponded with this data, showing that 11.1% of patients with pericardial effusion had normal RA pressure. This phenomenon is likely in the setting of hypovolemia and therefore reduced filling pressures.

Conclusions

We found that pericardial drainage is associated with improvement in effusion-mediated acute renal dysfunction. This is likely due to a decrease in right atrial pressure and thus systemic venous congestion with a concomitant increase in forward stroke volume and perfusion pressure.

Limitations

This was a retrospective analysis, and therefore only associations between pericardiocentesis and improvement in renal function can be assessed. We excluded patients on renal replacement therapy and with missing laboratory or echocardiographic data. LVOT VTI and RA pressure were estimated via echocardiogram rather than measured by direct invasive measurements, and thus may be limited by technical factors. Some patients had normal renal function, so we may be underestimating the effect of pericardiocentesis on renal function.

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***Co-first authors. These authors contributed to the manuscript equally.**

Table I. Demographics

Age, mean (SD)
Male
Female
Length of Stay, mean (SD)
Post-operative effusion
Inflammatory effusion

Malignancy related effusion
 Idiopathic/Unknown cause of effusion
 In-admission mortality
 30-day mortality
 Pericarditis
 Past oncologic disease
 Current oncologic disease
 History of Heart Failure
 History of Diabetes Mellitus
 History of Coronary Artery Disease
 History of Hypertension
 History of Chronic Renal Failure (not dialysis dependent)
 History of Atrial Fibrillation
 History of Valvular disease
 Pacemaker
 Endocarditis on admission
 Hypotension on admission (SBP < 80)
 ACEi/ARB
 Beta-blocker
 Calcium Channel Blocker
 Diuretics
 Alpha-agonist
 Nitrates
 Amiodarone
 Aldactone/Eplerenone
 Any anticoagulation
 On any BP lowering medication
 SBP, systolic blood pressure. ACEi, Angiotensin converting enzyme inhibitor. ARB, Angiotensin receptor blocker. NSAID

Table II. Demographics Stratified by GFR improvement > 10%

Age, mean (SD)
 Sex
 Male, n (%)
 Female, n (%)
 Length of Stay, mean (SD)
 Suspected Cause, n (%)
 Post-operative
 Infectious/Inflammatory
 Malignancy
 Idiopathic/Unknown

 In-admission mortality
 30-day mortality
 Pericarditis
 Past oncologic disease
 Current oncologic disease
 Heart Failure
 Diabetes Mellitus
 Coronary Artery Disease

Hypertension
 Chronic Renal Failure (not dialysis dependent)
 Atrial Fibrillation
 Valvular disease
 Pacemaker
 Endocarditis (on admission)
 Hypotension (SBP < 80)
 Concurrent Medications, n (%)
 ACEi/ARB
 Beta-blocker
 Calcium Channel Blocker
 Diuretics
 Alpha-agonist
 Nitrates
 Amiodarone
 Aldactone/Eplerenone
 Any anticoagulation
 On any BP lowering medication
 SBP, systolic blood pressure. ACEi, Angiotensin converting enzyme inhibitor. ARB, Angiotensin receptor blocker. NSAID

Table III. Echocardiographic, Hemodynamic and Laboratory Values

GFR (mL/min/1.73 m²)
 Systolic Blood Pressure (mmHg)
 Diastolic Blood Pressure (mmHg)
 Creatinine (mg/dL)
 BUN (mg/dL)
 Right atrial pressure (mmHg) (n=81)
 ~3 mmHg – n (%)
 ~8 mmHg – n (%)
 15 mmHg – n (%)
 Mean Arterial Pressure (mmHg)
 Diastolic Perfusion Pressure (mmHg)
 Mean Perfusion Pressure (mmHg)
 LVOT VTI (cm) (n=59)
 Dilated IVC on TTE – n (%)
 Tamponade on TTE – n (%)
 GFR, Glomerular Filtration Rate. BUN, Blood urea nitrogen. LVOT VTI, Left ventricular outflow tract-velocity time integ

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