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Reversible Hyponatremia and Acute Renal Failure in Cardiac Tamponade

Ales Rozman, Vladimir Dimitric, Katja Adamic, Mateja Marc Malovrh and Julij Selb*

Department of Endoscopy/Pulmonology, University Clinic of Respiratory and Allergic Diseases Golnik, Slovenia

Abstract

Background: Cardiac tamponade can be associated with hyponatremia and/or acute renal failure. Case reports and a few case series describe conditions separately. The objective of this study was to determine the frequencies of the reversible hyponatremia and acute renal failure in patients with cardiac tamponade after pericardial drainage and to determine whether there is any association between them.

Methods: In this retrospective study medical records of patients treated by pericardial drainage between years 2010 and 2016 were reviewed. Cardiac tamponade was confirmed by cardiac chamber compression proved by echocardiography. Sample means/medians of paired data were compared with paired t-test (sodium concentration before and after drainage) and Wilcoxon signed-rank test (creatinine concentration before and after drainage).

Results: Median amount of evacuated pericardial fluid was 1100 ml (rang 250 ml to 2000 ml) in 23 included patients. The mean pre-drainage sodium level was 133.1 mEq/l ± 8.5 mEq/l and the mean post-drainage sodium level was 139.0 mEq/l ± 4.2 mEq/l ($p<0.002$). Among 23 patients 10 (43.5%) had hyponatremia before drainage and 3 (13.0%) after drainage. The median pre-drainage serum creatinine concentration was 87.5 µmol/l (range =47 - 283 µmol/l) and median post-drainage serum creatinine concentration was 70 µmol/l (range =42 - 186 µmol/l) ($p=0.014$). Sodium and creatinine concentrations were correlated in a way, that sodium concentration increases, and creatinine concentration decreases after cardiac decompression ($p<0.001$).

Conclusion: Hyponatremia and acute renal failure are associated with cardiac tamponade and recover spontaneously after pericardial drainage. Sodium concentration increase and creatinine concentration decrease after cardiac decompression are in a correlation.

Keywords: Acute renal failure; Cardiac tamponade; Hyponatremia; Pericardial drainage; Pericardial effusion

Introduction

Cardiac tamponade is compression of the heart due to the accumulation of the fluid in the pericardial sac and represents clinical emergency. The relationship between the amount of pericardial fluid and pressure is not linear, thus symptoms vary and are mostly nonspecific [1].

Cardiac tamponade can be accompanied by hyponatremia as reported in several case reports and two retrospective analyses [2-7]. Hyponatremia is frequently associated with malignant pericardial effusion and with cardiac chambers compression [2,3]. After pericardial drainage hyponatremia often improves spontaneously, without additional treatment [2-7]. The suggested underlying mechanism was the increased secretion of Antidiuretic Hormone (ADH) due to decreased cardiac output and consequential reabsorption of solute-free water in kidneys [8].

Moreover, the cardiac tamponade associated decrease in cardiac output may also be the cause of acute renal failure. The condition was so far mentioned in a few case reports, again, with rapid spontaneous resolution after pericardial drainage [9-14].

Reversible hyponatremia and acute renal failure may be the expressions of the same pathophysiological mechanisms associated with reversal of decreased cardiac output in decompressed heart, but the association has not been described yet in the medical literature. The purpose of our retrospective study was to determine the frequencies of the reversible hyponatremia and acute renal failure in patients with cardiac tamponade after pericardial drainage and to determine whether there is any association between them.

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Correspondence:
Ales Rozman, Department of Endoscopy/Pulmonology, University Clinic of Respiratory and Allergic Diseases Golnik, Golnik 36, SI-4204 Golnik, Slovenia, Tel: +386 41 313 811; E-mail: ales.rozman@klinika-golnik.si

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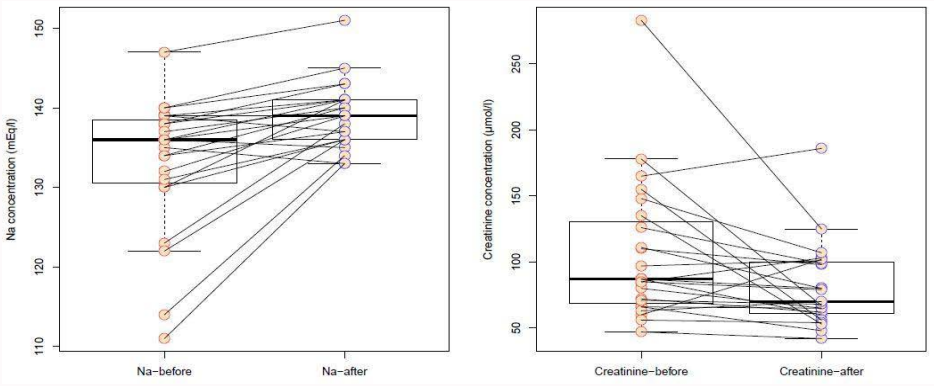

Figure 1: The serum concentrations of sodium (a) and creatinine (b) before and after pericardial drainage.

Methods

Patients

Patients were searched through Clinic's electronic database to retrieve cases by international classification of diseases (ICD-10-AM, version 6) codes I30, I31 and I32 between years 2010 and 2016. We searched also Clinic's cytology database for samples of pleural effusion during the same period. Electronic and paper charts were reviewed for the history, vital signs and other physical findings, laboratory data, cardiac echo findings, chest X-rays and treatment of each individual patient. Cardiac tamponade was diagnosed by clinical presentation, chest X-ray and cardiac echo, where besides pericardial effusion cardiac chamber compression ought to be described (end diastolic right atrial and/or early diastolic right ventricular invagination) either as clearly seen or as "suspected" [15].

Patients undergoing pericardial drainage were eligible for enrolment if serum sodium and creatinine concentrations were measured within 2 days prior drainage and within 5 days after drainage. Hyponatremia was defined as sodium concentration below 135 mEq/l.

Patients without complete cardiac echo data, electrolyte measurements and those who received hyponatremia treatment including diuretics were excluded. All pericardial drainages were performed through percutaneous approach under ultrasound guidance. Patients with benign diagnosis were followed up for at least 2 years. The study was performed in a single tertiary medical center, approved by Clinic's medical board and conducted in accordance with the Declaration of Helsinki.

Statistical analysis

All the statistical analyses were conducted using R statistical software (R Core Team (2017). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria, https://www.R-project.org). Data was checked for normality using Shapiro-Wilk test. Sample means/medians of paired data were compared with paired t-test (sodium concentration) and Wilcoxon signed-rank test (creatinine concentration). A p-value <0.05 was considered statistically significant.

Results

Thirty-one patients were identified in the study period. Eight patients were excluded due to incomplete data or use of diuretics. Twenty-three patients were included into analysis and their clinical data are summarized in Table 1.

Table 1: Patient clinical data.

	n	%
Sex		
Men	12	52
Women	11	48
Age (yrs)		
Median (range)	65 (45 – 87)	
Diagnosis		
Malignant	13	57
Lung cancer - adenocarcinoma	10	44
Lung cancer - large cell	1	4.3
Lung cancer – squamous cell	1	4.3
Malignant melanoma	1	4.3
Benign	10	44
Pneumonia with pericarditis	3	13
Tuberculosis	1	4.3
Mycobacterium kansasii	1	4.3
Coxackievirus pericarditis	1	4.3
Cardiac injury (myocardial infarction)	1	4.3
Acute pericarditis – not specified	3	13

Median amount of evacuated pericardial fluid was 1100 ml (rang 250 ml to 2000 ml). In two patients, only a partial evacuation with cardiac chamber decompression has been succeeded due to technical difficulties and clogged catheter.

The mean pre-drainage sodium level was 133.1 mEq/l ± 8.5 mEq/l and the mean post-drainage sodium level was 139.0 mEq/l ± 4.2 mEq/l. The post-drainage sodium level was significantly higher with the main increase of 6.0 mEq/l ± 6.4 mEq/l (p=0.002). Among 23 patients 10(43.5%) had hyponatremia before drainage and 3(13.0%) after drainage. Among patients with malignant pericardial effusion 5/13(38.5%) had low sodium level before and 2/13(15.4%) after drainage. Among patients with benign pericardial effusion 5/10(50.0%) had low sodium level before and 1/10(10.0%) after drainage. In all groups of patients, serum sodium concentration raised significantly after pericardial drainage (Table 2).

The median pre-drainage serum creatinine concentration 87.5 μmol/l (range =47 - 283 μmol/l) was significantly higher (p=0.014) than the median post-drainage serum creatinine concentration 70

Table 2: The comparison of serum sodium concentration before and after pericardial drainage.

	Before drainage (mEq/l)	After drainage (mEq/l)	Difference (mEq/l)	p
All patients (n = 23)	133.1 ± 8.5	139.0 ± 4.2	6.0 ± 6.4	0.002
Etiology of effusion				
Malignant (n = 13)	134.1 ± 6.9	138.4 ± 3.5	4.3 ± 6.0	0.024
Benign (n = 10)	131.8± 10.5	139.9±4.9	8.1 ±6.6	0.004
Baseline sodium level				
Normal (n = 13)	138.5 ± 3.0	140.6 ± 4.5	2.2 ± 2.6	0.011
Low (n = 10)	126.1 ± 8.3	137.0 ± 2.5	10.9 ± 6.6	0.001

mol/l (range =42 - 186 mol/l).

To test the relation between the changes in sodium and creatinine concentrations before and after pericardial drainage, we hypothesized, that changes of two variables would fall into one of the four categories (Cre_{rise} Na_{rise}, Cre_{rise} Na_{fall}, Cre_{fall} Na_{fall} and Cre_{fall} Na_{rise}) with the probability of 0.25 if they are not correlated. The distribution in our sample was the following: Cre_{rise} Na_{rise} = 5, Cre_{rise} Na_{fall}= 0, Cre_{fall} Na_{fall} = 3 and Cre_{fall} Na_{rise} =15). The p-value for getting a result of 15 positive hits in a sample of 23 draws, where we sample from a binominal distribution with the 0.25 probability of a positive draw, was <0.001. Sodium and creatinine concentrations are therefore correlated in a way, that sodium concentration increases, and creatinine concentration decreases after cardiac decompression (Figure 1).

Discussion

Cardiac tamponade is a life-threatening condition, due to numerous malignant and benign diseases. In the present study we described the incidences of hyponatremia and acute renal failure, which spontaneously recovered after cardiac decompression and the relation between them. Although both conditions were described in case reports and case series separately, relationship between them has not been addressed yet [2-7,9-14].

We detected hyponatremia in 43,5% of patients with cardiac tamponade, what was less in comparison with previously published data, where incidences were 65% and 54.2% [2,3]. We didn't observe the positive relationship between malignant disease and hyponatremia as published before, because the incidence of hyponatremia in the group of patients with malignant disease was only 38,5% in contrast to the group of patients with benign diseases where incidence reached 50%. However, the spontaneous recovery in patients with benign disease was better than in patients with malignancy, which suggests some additional mechanisms of hyponatremia in malignant disease [16]. The increase in serum sodium concentration raised significantly in all groups of patients after cardiac decompression. Sodium raised even in the group of patients with baseline sodium concentration within the reference interval.

In parallel with hyponatremia we observed a reversible renal failure with increased creatinine concentration. Creatinine concentration decreased significantly after pericardial drainage. Although our sample of patients was small, a clear correlation between hyponatremia and worsening of the renal function seemed to exist.

Pathophysiological mechanisms running in the background are complex, but fragments of evidence from the existing literature enabled us to connect both manifestations of compressed hearth in a scheme of compensatory mechanisms of the body.

The increased pericardial pressure compresses cardiac chambers after the volume of pericardial effusion overwhelms the pericardial capacity to stretch. Cardiac chambers become progressively smaller, cardiac inflow is reduced and effective circulatory volume is consequentially decreasing [1].

The condition is detected by hypothalamus, which releases Antidiuretic Hormone (ADH) in order to restore circulatory volume by free water retention in the kidneys [8]. The elevated serum ADH was detected in dogs with experimental cardiac tamponade and declined rapidly after pericardiocentesis [17].

At the same time, mean arterial pressure and consequently Glomerular Filtration Rate (GFR) are decreased in pericardial tamponade [18]. Renal denervation prevented these changes in dogs, what suggested sympathetic nerve mediated mechanism [18].

In a small case series of patients with constrictive pericarditis increased concentrations of plasma norepinephrine, renin and aldosterone were detected [19]. Pressures in the right atrium and in the pulmonary vasculature were increased in parallel with increased pulmonary and systemic vascular resistance [19]. After pericardiectomy all measurements in all 8 patients returned toward normal levels [19].

Retention of water in the body which manifests as edema maintains the arterial pressure, and diuretic therapy at this point can dangerously compromise the patient. Pericardial drainage and heart decompression rapidly restore cardiac output and accumulated body water is detected mainly by increased transmural atrial pressure and consequential atrial distension, which stimulates Atrial Natriuretic Peptide (ANP) secretion [20-22]. Although atrial pressure is high during cardiac tamponade, transmural pressure is not elevated because of opposing pericardial fluid pressure and ANP is not released [18]. Massive diuretic response usually follows pericardial drainage with the restoration of kidney function and serum sodium concentration [5,10-12].

It is unclear, why some patients develop hyponatremia in cardiac tamponade and the other don't. It seems, that the effects of pericardial fluid volume and the time course of its accumulation on cardiac output vary in individual patients and thus also the magnitude of the adaptive response. The assumption was confirmed by our observation, where serum sodium concentration raised in all patients significantly after pericardial drainage even in those, who didn't meet the criteria for hyponatremia.

The constellation of hyponatremia and increased serum creatinine has important clinical consequences: diuretic treatment should not be started, until cardiac tamponade is excluded, to avoid harmful

effects on patient's compensatory mechanisms.

Our study had several limitations. The first is the retrospective design with incomplete set of data in some patients that had to be excluded from the analysis. This might include some selection bias. Next disadvantage is relatively low number of patients, which didn't allow us to perform subgroup analyzes. Interpretation of cardiac echo is, again, a subjective process to some extent, where certain investigators might differently assess cardiac chamber compression. Besides routine laboratory, no hormone assays were performed before and after pericardiocentesis. And finally, pericardial pressures or pressures in cardiac chambers and pulmonary vasculature were not measured before and after pericardial drainage. The study was focused on direct clinical effects of pericardial drainage and didn't address the underlying mechanisms which can be the object of future, prospective trial.

Cardiac tamponade compromises effective circulatory volume and can cause hyponatremia and acute renal failure. Both conditions spontaneously recover after pericardial drainage, without additional therapy. Diuretic therapy might harm patients if cardiac tamponade was not excluded as the cause of hyponatremia and acute renal failure, despite clinical signs of cardiac failure.

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