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CASE REPORT

Acute renal failure due to pericardial tamponade in a 60-year-old male patient

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Abstract

Acute renal failure (ARF) is still a considerable factor in hospital morbidity and mortality. This clinical condition occurs in up to 25% of critically ill patients. Mortality in these patients varies widely depending on the cause. ARF in the context of a large pericardial effusion and pericardial tamponade has not often been reported. This paper presents a case of life-threatening pericardial tamponade and a consecutive rapid onset of ARF. Successful treatment with pericardiocentesis was performed, which was followed by restitution of renal function.

Key Words: *Acute renal failure, pericardial effusion, pericardial tamponade*

Introduction

An increasing number of patients are developing acute renal failure (ARF) each year. In recent years, there have been considerable advances in our understanding and technical capabilities, but there is no consensus about the optimal treatment. The reasons are complex but include the fact that many patients are older, with comorbid conditions [1]. The clinical condition of ARF is said to occur in anywhere from 1% to 25% of critically ill patients [2], depending on the population and the definition criteria. Furthermore, mortality in these populations ranges from 28% to 90% [3]. Trials of prevention and therapy are not comparable because widely disparate definitions have been used. However, most definitions of ARF have common elements, including the use of serum creatinine and, often, urine output. ARF in the context of a large pericardial effusion and pericardial tamponade has not been reported very often, and it may not be thought about or considered as a cause-and-effect relationship when one or the other is present [4]. Therefore, this report presents a case of life-threatening pericardial tamponade with consecutive rapid onset of ARF.

Case report

A 60-year-old male patient was admitted to the pulmology department for evaluation. The patient's history revealed that 20 months earlier he had had a myocardial infarction which was treated by percutaneous coronary intervention and implantation of two stents. During his cardiological work-up there was a suspicious finding on his chest radiograms. Following further diagnostic evaluation it was established that the patient had pleural mesothelioma with a malignant exudative pleuritis of his right pulmonary lobe, unsuccessfully treated with chemotherapy. His current medication included a β-blocker, an angiotensin-converting enzyme (ACE) inhibitor, acetylsalicylic acid, a statin and alopurinol.

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This time he presented with prostration, loss of appetite and a sensation of pressure in his abdomen that had lasted for 6 days before admission. A chest radiogram showed a large lateral ascending shadow resembling a pleural effusion on his right pulmonary lobe, and an enlarged heart silhouette. The ECG showed sinus rhythm at 102 bpm and a chronic infarction.

The patient weighed 74 kg, had normal body temperature, his blood pressure was 120/80 mmHg and his breathing frequency was normal. His abdomen was distended with signs of ascites and hepatomegalia. His basic laboratory tests showed normal renal function, high leucocyte levels, anaemia, a C-reactive protein (CRP) level of 18.9 mg/l and elevated liver tests. The patient remained under observation.

On the fourth day of hospitalization, he became oliguric, with a drop in blood pressure to 100/60 mmHg. His laboratory parameters of renal function worsened (Table I). A saline infusion was administered, followed by intravenous furosemide. The dose of β-blockers and ACE inhibitors was halved. No urine was found in the bladder and a consulting nephrologist advised further rehydration. During the next 6 days satisfactory diuresis was achieved.

On the 11th day of hospitalization the patient again became oliguric and the next day anuric with a drop in blood pressure to 90/60 mmHg. An ultrasound showed an enlarged liver, ascites and both kidneys reduced in size.

A nephrologist concluded that the patient had ARF and ordered transfer to nephrology department the same day. There he received an extensive work-up and diuretic therapy, but there was no improvement in his diuresis during the next 2 days. His blood pressure again dropped below 100 mmHg systolic and his daily diuresis was 50–100 ml. Sodium levels fell and potassium levels increased (Table I).

On the 15th day of hospitalization ultrasound of the heart showed a large pericardial effusion around the whole heart, with a thickness of 15–26 mm, forming a life-threatening pericardial tamponade. In addition, there were some tumour masses visible on the pericardium and in the apex of the left ventricle. Pericardiocentesis was carried out on the same day and 970 ml of pericardial content evacuated. Later that day, the patient's blood pressure increased to 105/70 mmHg and his diuresis measured 2550 ml. On the next day diuresis was 2700 ml; there was no drainage from the pericardium, so it was instilled with 7.5 mg of bleomycine and the drain was evacuated on the next day.

In the following 6 days the patient's diuresis stabilized at around 1200–1500 ml/day, and his blood pressure increased and remained within the normal range (Table I). After a total of 23 days in the hospital he was discharged from the nephrology department with a blood urea level of 19.0 mmol/l and serum creatinine level of 107 μmol/l.

Discussion

The most frequent pericardial emergency is cardiac tamponade. The diagnosis of cardiac tamponade is based on clinical presentation and physical findings, confirmed by echocardiography and cardiac catheterization. Tamponade is an absolute indication for urgent drainage, by either pericardiocentesis or surgical pericardiotomy [5]. The presence of pericardial effusion is not rare in patients with renal failure. Uraemic pericardial effusions with cardiac tamponade used to occur frequently, but with improvements in dialysis techniques and modern therapy their incidence is rare today [6–8]. In this patient with a pleural tumour and pleural effusion a rapid onset of renal failure was recorded. During the evaluation all the diagnostic methods were aimed at renal failure and therapeutic interventions were undertaken to gain back diuresis by boosting renal function with diuretics and hydration, and a large pericardial effusion was

Table I. Renal function parameters, electrolytes and blood pressure changes during hospitalization.

Day of hospitalization	Urea (mmol/l)	Creatinine (μmol/l)	Sodium (mmol/l)	Potassium (mmol/l)	Systolic BP	Diastolic BP	Diuresis/24 h (ml)
2	12.9	132.0	131.0	5.8	120	70	1000
4	17.8	204.0	125.0	5.2	100	60	300
5	22.0	267.0	123.0	5.1	110	70	3000
12	30.2	268.0	127.0	4.7	90	50	100
15	45.5	472.0	125.0	6.6	105	70	2550
21	19.0	107.0	143.0	4.4	120	80	1500

BP = blood pressure.

found that threatened with pericardial tamponade. After the immediate tamponade threat was eliminated, a rapid resolution and fast return of renal function were noted. Similar findings are rare in the literature and were mostly due to unusual tumour manifestations or other causes, mostly viral or parasitic [9,10]. Although it is not possible to exclude other possible causes of rapid onset of the renal failure in this patient, such as induced by ACE inhibitors or other medication, it is likely that pericardial tamponade was responsible.

In conclusion, in patients with thoracic tumours or any other disease that could induce pericardial effusion, it is necessary to exclude it as a cause of renal failure.

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