CARDIAC TAMPONADE SECONDARY TO PERICARDITIS IN A DIALYSIS PATIENT – A CASE REPORT

Tamponada serca na podłożu zapalenia osierdzia u pacjenta dializowanego – opis przypadku

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Abstract

Introduction: Pericarditis is a known cause of chest pain, particularly in young men. It may be complicated by cardiac tamponade, which requires urgent intervention. The aetiology of acute pericarditis may pose a diagnostic challenge. Possible causes include viral and bacterial infections, rheumatic diseases, aortic dissection, and others. Uremic pericarditis is also a potential cause, primarily occurring in dialysis patients, and less common in those with chronic kidney disease prior to initiating dialysis. **Case report:** A 65-year-old male patient was admitted to the hospital due to deterioration of general condition and dyspnoea. Based on the clinical presentation, cardiac tamponade secondary to pericarditis was suspected, which was confirmed by imaging studies. Emergency pericardiocentesis was performed. The differential diagnosis included uremic and viral aetiologies. Daily haemodialysis and corticosteroid therapy were initiated, resulting in clinical improvement. **Discussion:** Uremic pericarditis predominantly occurs in patients with chronic kidney disease and may develop both prior to and during dialysis, irrespective of serum urea levels. Intensified dialysis, as in the case described, frequently leads to clinical improvement. Pharmacological treatment for viral acute pericarditis has limited efficacy in patients with chronic kidney disease, making corticosteroid therapy the primary therapeutic option. **Conclusion:** In dialysis patients presenting with acute pericarditis, uremic aetiology, along with other potential causes, should always be considered. Early diagnosis and prompt pericardiocentesis are crucial in cases of cardiac tamponade.

Streszczenie

Wstęp: Zapalenie osierdzia jest jedną z przyczyn bólu w klatce piersiowej, szczególnie u młodych mężczyzn. Jego powikłaniem może być tamponada serca, wymagająca pilnej interwencji. Etiologia ostrego zapalenia osierdzia bywa trudna do ustalenia. Możliwe przyczyny obejmują infekcje wirusowe i bakteryjne, choroby reumatologiczne, rozwarstwienie aorty i inne. Jedną z nich jest również mocznicowe zapalenie osierdzia, występujące głównie u pacjentów dializowanych, rzadziej w przewlekłej chorobie nerek przed rozpoczęciem dializ. **Opis przypadku:** 65-letni pacjent został przyjęty do szpitala z powodu pogorszenia stanu ogólnego i duszności. Na podstawie obrazu klinicznego podejrzewano tamponadę serca w przebiegu zapalenia osierdzia, co potwierdzono w badaniach obrazowych. W trybie ostrego dyżuru przeprowadzono perikardiocentezę. Diagnostyka różnicowa obejmowała etiologię mocznicową i wirusową. Włączono codzienne zabiegi hemodializ oraz terapię glikokortykosteroidami uzyskując poprawę stanu pacjenta. **Omówienie:** Mocznicowe zapalenie osierdzia występuje głównie u pacjentów z przewlekłą chorobą nerek i może rozwijać się zarówno przed dializoterapią, jak i podczas niej, niezależnie od stężenia mocznika. Intensyfikacja dializ, jak w opisanym przypadku, często przynosi poprawę. Leki stosowane w ostrym zapaleniu osierdzia o etiologii wirusowej mają ograniczone zastosowanie u pacjentów z przewlekłą chorobą nerek, co sprawia, że steroidoterapia często pozostaje jedyną opcją terapeutyczną. **Wnioski:** U dializowanych pacjentów z ostrym zapaleniem osierdzia należy zawsze uwzględniać zarówno etiologię mocznicową, jak i inne potencjalne przyczyny. W przypadku pojawienia się tamponady serca kluczowe jest wczesne rozpoznanie i przeprowadzenie perikardiocentezy.

Keywords: pericarditis; cardiac tamponade; uremic pericarditis

Słowa kluczowe: zapalenie osierdzia; tamponada serca; mocznicowe zapalenie osierdzia

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Introduction

Pericarditis is diagnosed in about 5% of patients presenting to emergency departments with chest pain that is not related to acute coronary syndrome [1], most commonly affecting men aged 20–50 years [2]. It may give rise to a serious complication known as cardiac tamponade, one of the causes of sudden cardiac arrest in the 4H/4T classification, which requires immediate pericardial decompression. Hypotension, muffled heart sounds, and distension of the jugular veins, collectively referred to as Beck's triad, are typical clinical manifestations. Rapid diagnosis of cardiac tamponade is vital for effective treatment.

Over 95% of patients with acute pericarditis present with pleuritic retrosternal pain of varying severity [3], which may radiate to the neck, jaw, or arm, resembling myocardial infarction. It typically worsens when lying down or coughing and improves when sitting or leaning forward, when compression on the pericardium is smaller [1].

The aetiology of acute pericarditis can pose a diagnostic challenge. Possible causes include viral and bacterial infections, rheumatic diseases (such as sarcoidosis or lupus), neoplastic processes, trauma, aortic dissection, and iatrogenic factors [2]. Uremic pericarditis, which was previously considered a complication of advanced untreated chronic kidney disease (CKD), has also been distinguished. Currently, it is observed mainly in dialysis patients. In 2017, Bentata et al. found uremic pericarditis in only 1.7% of cases, with its incidence markedly reduced by the use of highly efficient dialysis membranes [4, 5]. The introduction of medium cut-off (MCO) membranes has allowed for eliminating medium- and high-molecular-weight uremic toxins without the need for large convection volumes or significant albumin loss [6].

The pathophysiology of uremic pericarditis is complex and involves the accumulation of toxic metabolites (such as methylguanidine and guanidinoacetate), nitrogen-containing metabolic products, as well as electrolyte and acid–base imbalance [7]. Hyperparathyroidism, hyperuricemia, and hypocalcaemia may also contribute to the development of this disorder [8]. In patients with CKD, elevated urinary albumin excretion and volume overload increase endothelial permeability and promote fluid accumulation in the pericardial sac, which also contributes to pericarditis [9].

Case report

A 65-year-old patient presented to the Department of Internal Medicine, Nephrology, and Dialysis with general deterioration, weakness, and exertional dyspnoea. He had developed an upper respiratory tract infection followed by reduced exercise tolerance one week before admission.

On admission, the patient was in serious condition with significant respiratory distress, blood pressure of 95/80 mmHg, oxygen saturation of 95%, and faint heart sounds on auscultation. The patient had a history of abdominal aortic aneurysm repair, followed by renal artery occlusion complicated by acute kidney injury two months later. Creatinine was approximately 10 mg/dL, with preserved diuresis and no oedema or other signs of fluid overload. This was likely related to anatomical variation in the patient's renal vasculature, which included accessory arteries. An unsuccessful revascularization attempt was made, after which the patient was started on chronic haemodialysis. The man additionally presented with poorly controlled hypertension, renal cysts, lower limb atherosclerosis, and a history of hepatitis B.

Chest X-ray on admission showed a markedly enlarged cardiac silhouette compared with the image taken two months earlier (Fig. 1). Based on Beck's triad (hypotension and muffled heart sounds) and imaging findings, a suspicion of cardiac tamponade was raised. Electrocardiography (ECG) showed low QRS voltage, which is typical of this condition.

Point-of-care echocardiography (ECHO) found a large pericardial effusion, prompting emergency pericardiocentesis, during which 840 mL of bloody fluid was evacuated. The drain was removed three days later, having yielded an additional 50 mL. Only trace fluid was detected on follow-up ECHO.

Laboratory workup showed elevated inflammatory markers, while blood and pericardial fluid cultures were negative. Troponin levels were measured to rule out acute coronary syndrome and were stable. Computed tomography angiography (CTA) performed to rule out aortic dissection detected fluid in both pleural cavities, the rectovesical recess, the perihepatic space, and the perisplenic region. Because of signs of hypervolemia, the patient was put on daily haemodialysis, and glucocorticoids were added to the treatment regimen. His clinical condition improved, and dyspnoea resolved.

Discussion

The differential diagnosis in the discussed case primarily included uremic and infectious pericarditis, as aortic dissection was ruled out by CT angiography. A recent mild

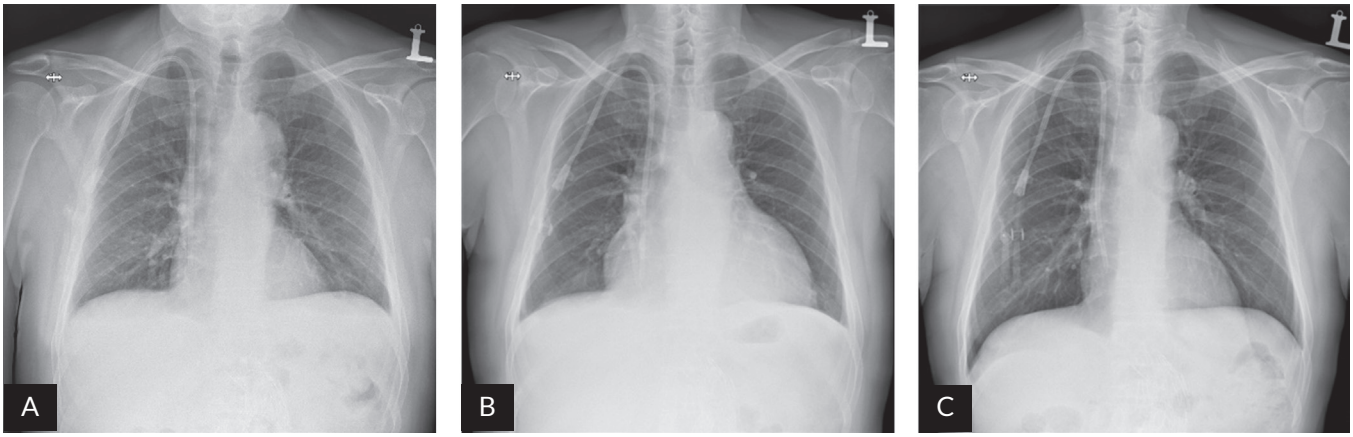

Fig. 1. Chest X-ray: **A.** Before admission (March 2024); **B.** On admission, suspected tamponade (May 2024); **C.** Post-treatment follow-up X-ray (June 2024)

respiratory infection may suggest viral aetiology, with coxsackieviruses and adenoviruses being the most common causes, though SARS or HIV can also be responsible [10]. However, no polymerase chain reaction (PCR) testing was performed to unequivocally confirm viral aetiology. Elevated C-reactive protein (CRP) and leukocytosis indicated an ongoing infection, although the patient did not present any symptoms of infection on admission and maintained an oxygen saturation of 95%. The rapid onset of symptoms after an infection may have indicated viral aetiology of pericarditis and tamponade.

A history of renal failure and chronic dialysis therapy may suggest uremic aetiology. Uremic pericarditis can occur both pre- and post-dialysis. It is known as dialysis-associated pericarditis, which occurs primarily in haemodialysis patients, less frequently in those on peritoneal dialysis. The possible causes include inadequate dialysis therapy, individual patient predispositions, and poor adherence to medical recommendations [11]. From a clinical perspective, uremic pericarditis differs from acute pericarditis. It typically has a slow onset, and aside from a pericardial rub, other symptoms may be absent. Leukocytosis and fever are uncommon, and there are often no significant ECG abnormalities [12]. Rutsky and Rostand reported pericarditis in 13% of patients on chronic dialysis during a 13.7-year follow-up period. Cardiac tamponade and impending tamponade developed in 20% and 22% of patients with pericarditis [11].

Pericarditis in advanced CKD is managed by either initiating or intensifying dialysis [4]. Patients with uremic pericarditis typically respond rapidly to renal replacement therapy [13]. The regimen for intensified dialysis is 4 hours of dialysis daily for 7–14 days. Patients should be closely monitored, with ECHO performed every 3 days to assess pericardial effusion [13, 14]. Systemic anticoagulation should be avoided due to the increased risk of bleeding and potential pre- or post-tamponade complications [15].

For viral pericarditis, patients should receive colchicine for 3 months along with nonsteroidal anti-inflammatory drugs (NSAIDs) to relieve symptoms and reduce the risk of recurrence. In recurrent cases, colchicine should be continued for at least 6 months. Corticosteroids are often used if pericarditis does not improve with NSAIDs and colchicine. Interleukin-1 (IL-1) blockers have demonstrated efficacy in some patients with repeated long-lasting (several years) recurrences, and may be preferred over corticosteroids [16]. The use of NSAIDs, colchicine, and IL-1 blockers is limited in patients with renal failure [17]. GCs are preferred in G5 CKD, as in the described case.

Given the ambiguous aetiology of the pericarditis, treatment targeting both uremic and infectious causes was initiated, leading to rapid clinical improvement. Uremic pericarditis shows a greater improvement with intensified dialysis in patients on renal replacement therapy for less than two months. Consequently, patients on long-term renal replacement therapy require intensified treatment [17].

Uremic pericarditis can also occur in well-dialyzed patients with mildly elevated serum urea due to other toxic metabolites and high levels of free radicals [8]. Even mild uraemia has been observed to create a pro-oxidant state [18]. Pupima et al. [19] showed that dialysis reduces toxic metabolite levels, but is ineffective in correcting oxidative stress or lowering inflammatory biomarkers. This may account for the development and progression of pericarditis in patients on long-term renal replacement therapy despite normal blood urea. Although uremic aetiology is usually suspected in dialysis patients with pericarditis, other causes should always be considered. Regardless of aetiology, rapid diagnosis and treatment are crucial in emergency cases, such as cardiac tamponade secondary to pericarditis.

Conclusions

Determining the aetiology of tamponade poses a diagnostic challenge in dialysis patients, as uremic pericarditis, seen only in renal failure, often comes to the fore. However, other causes should also be considered in the diagnosis and treatment planning. Early identification of tamponade and prompt pericardiocentesis are vital.

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