

Uremic Pericarditis Accompanying Cardiac Tamponade after Emergency Hemodialysis

Ha Yeun Park, M.D., Seong Sik Kang, M.D., Yae Rim Kim, M.D., O Hyun Kwon, M.D.,
Kyu Bok Jin, M.D., Seung Yeup Han, M.D., Sung Bae Park, M.D., Woo Yeong Park, M.D.

*Department of Internal Medicine, Keimyung University School of Medicine,
Keimyung University Kidney Institute, Daegu, Korea*

Received: March 31, 2016

Revised: May 19, 2016

Accepted: June 02, 2016

Corresponding Author: Woo Yeong Park, M.D.,
Department of Internal Medicine,
Keimyung University School of Medicine,
Keimyung University Kidney Institute,
56 Dalseong-ro, Jung-gu, Daegu 41931, Korea
Tel: +82-53-250-7399
E-mail: wy-my@hanmail.net

• The authors report no conflict of interest in this work.

Although the incidence of uremic pericarditis was high in the past, it has decreased in recent decades with early and appropriate dialysis. However, cardiac tamponade caused by uremic pericarditis is still a life-threatening emergency and it requires urgent management. Herein we report a case of 38-year-old man with chronic renal disease who represented critical uremic pericarditis followed by cardiac tamponade despite of appropriate hemodialysis. Careful consideration of risk factors and aggressive treatment are very important for effective and safe treatment of uremic pericarditis and cardiac tamponade.

Key Words: Cardiac tamponade, Pericardial effusion, Pericardiocentesis, Renal replacement therapy, Uremia

Introduction

Uremic pericarditis is a serious complication of end-stage renal disease requiring maintenance dialysis, and it can occur before renal replacement therapy (RRT) or in the 8 weeks after RRT initiation [1]. Although the incidence of uremic pericarditis was high in the past, its occurrence has been decreased in recent decades according to development of dialysis technique and increase of kidney transplantation [2]. However, mortality of uremic pericarditis was still very high, especially accompanying with cardiac tamponade, a life-threatening emergency state [3]. Therefore, it requires an aggressive diagnosis of uremic pericarditis before occurrence of cardiac tamponade and an emergent treatment when the sign of cardiac tamponade such as a hypotension, decreased cardiac sound or low voltage on electrocardiography happened.

Here, we report a case of uremic pericarditis accompanying cardiac tamponade after intensive hemodialysis treated successfully with pericardiocentesis and continuous renal replacement therapy.

Case Report

A 38-year-old man with a history of hypertension was brought to the emergency room with severe general weakness and dyspnea for two weeks. He was diagnosed with hypertension and chronic kidney disease four years ago. His cause of chronic kidney disease was estimated chronic glomerulonephritis due to elevated serum creatinine level, 2.0 mg/dL, contracted kidneys and proteinuria. However, he did not receive any treatments in the meantime. His blood pressure was 190/100 mmHg, pulse rate 102/min, respiration rate 28/min and 37.0°C. Oxygen saturation was 98% on room air. Coarse crackles were audible in both lung fields and pitting edema was observed in both legs, but cardiac sound was normal. Laboratory findings on admission revealed a leukocyte count of 13,710/ μ L, hemoglobin 7.2 g/dL, platelets 260,000/ μ L, blood urea nitrogen and creatinine level were 260

mg/dL and 39.32 mg/dL. Arterial blood gas analysis showed pH 7.294, HCO_3^- 7.3 mEq/L and high anion gap metabolic acidosis. Chest radiography showed cardiomegaly and pulmonary edema (Fig. 1A). Electrocardiogram and echocardiogram showed left ventricular hypertrophy, normal ejection fraction and small amount of pericardial effusion (Fig. 2A&D). After emergency hemodialysis through the tunneled cuffed catheter, his symptoms and general condition has improved gradually. At the 6th hemodialysis session using heparin 5000 IU, for priming and maintenance, hypotension and mental change occurred. An acute drop in hemoglobin level (8.5 mg/dL to 6.2 mg/dL) was seen, but we could not find the cause of anemia. Chest radiography showed severe cardiomegaly and pulmonary edema (Fig. 1B), and follow-up echocardiogram showed a large amount of pericardial effusion (anterior 3.0 cm, posterior 2.32 cm, close to left ventricle 2.98 cm, right ventricle 3.02 cm) with diastolic collapse of right ventricular wall (Fig. 2B). Pericardiocentesis was performed and the amount of drainage was 1,050 mL of bloody fluid, with 2,720/ μ L white blood cells, 75% neutrophils, 2,220,000/ μ L red blood cells, 40 mg/dL glucose, 5.0 g/dL protein, LDH 5055.4 IU/L and ADA

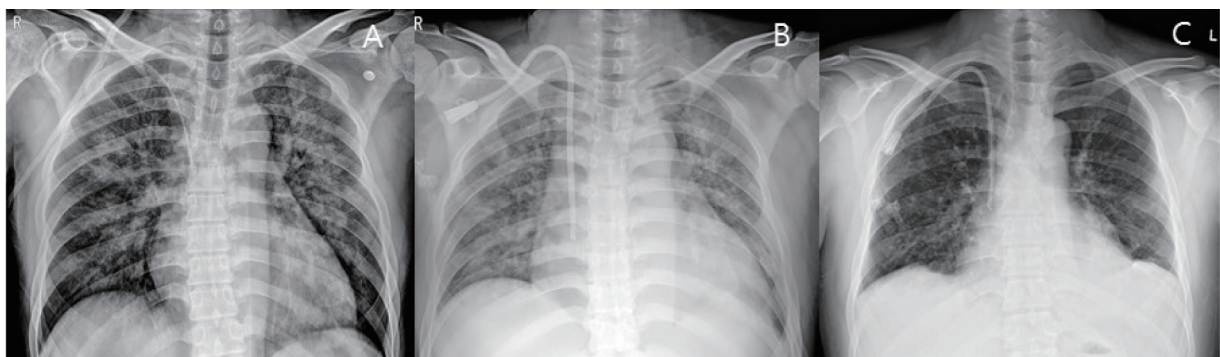


Fig. 1. (A) Initial chest X-ray shows cardiomegaly and pulmonary edema. (B) Chest X-ray shows severe cardiomegaly and pulmonary congestion after the 6th hemodialysis session. (C) After 5 days of pericardiocentesis and continuous renal replacement therapy, chest X-ray shows much improved cardiomegaly and pulmonary edema.

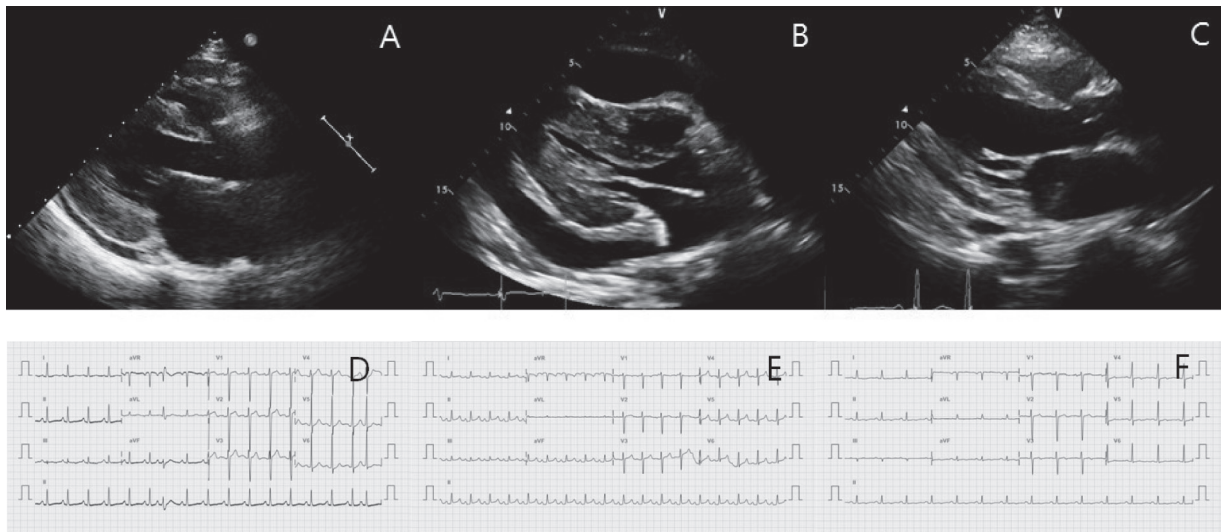


Fig. 2. (A) Initial echocardiogram shows normal left ventricular ejection fraction and small amount of pericardial effusion. (B) Echocardiogram shows a large amount of pericardial effusion (anterior 3.0 cm, posterior 2.32 cm, close to left ventricle 2.98 cm, right ventricle 3.02 cm) with diastolic collapse of right ventricular wall. (C) After 5 days of pericardiocentesis and continuous renal replacement therapy, echocardiogram shows much improved cardiomegaly and pulmonary edema. (D) Initial electrocardiogram shows left ventricular hypertrophy. (E) Electrocardiogram shows low voltages in all leads. (F) After treatment, electrocardiogram shows restored voltages in all leads.

31.8 IU/L. He was diagnosed with uremic pericarditis accompanying cardiac tamponade by severe uremia and the usage of heparin. Effusion cultures for bacteria, fungi, and acid-fast organisms were negative. Cytology examination showed no neoplastic cells. His general status, the result of follow-up chest radiography (Fig. 1C) and electrocardiogram and echocardiogram (Fig. 2C&F) has improved after pericardiocentesis and continuous renal replacement therapy for 5 days.

However, fever and pneumonic consolidation in both lung fields occurred with blood cytomegalovirus (CMV) polymerase chain reaction (PCR) study positive, 526 copies/mL and he received ganciclovir (1.25 mg per kg after dialysis) intravenously for seven days until the CMV PCR titer became negative and his symptoms have improved. He was discharged after about a month and now he is receiving regular hemodialysis at the other dialysis center.

Discussion

It is generally recognized that pericarditis occurs more frequently in new dialysis patients than in stabilized patients [4], but because of early renal replacement therapy recently, underdialysis in chronic hemodialysis patients is more frequent cause of pericarditis. This shows that severe uremia is one of the most important risk factors of uremic pericarditis, but there is no definite correlation with occurrence of uremic pericarditis and blood urea nitrogen and creatinine level.

Uremic pericarditis is characterized by the appearance of fibrinous exudates on both parietal and visceral pericardial surfaces [5]. The friable pericardial surface may bleed, leading to hemorrhagic pericardial effusion [6]. Hiraide *et al.* [7] reported a case of cardiac tamponade developed while heparin was being used as an anticoagulant, not recurred when

nafamostat mesilate was substituted. In our case, until 3rd session of dialysis, we used nafamostat mesilate as an anticoagulant during hemodialysis. As there was no evidence of active bleeding since 4th session of dialysis, we used unfractionated heparin. Although his uremic status was stable using heparin until 5th session of dialysis, abrupt drop of hemoglobin and hypotension occurred during 6th session of dialysis and cardiac tamponade was diagnosed by echocardiogram. Although pericardial effusion has improved after pericardiocentesis and continuous renal replacement therapy with nafamostat mesilate without an additional operation, he still had a serious bleeding tendency and even was performed an emergent bronchial artery embolization because of severe hemoptysis. This case showed that even if the status of patient was stable after intensive dialysis, the patient with severe uremic state had high bleeding tendency and could happen to hemorrhagic uremic pericarditis and cardiac tamponade.

Acute pericarditis and pericardial effusion can also happen by viral infection and viral pericarditis caused by coxsackie virus or influenza is most common [8]. In this case, CMV pneumonitis was detected in the middle of the treatment of pericarditis. Although we could not explain that CMV infection led to acute pericarditis directly because we did not perform a biopsy of pericardium, but his serum CMV PCR study showed 526 copies/mL and he had typical symptoms of pericarditis like chest pain and dyspnea. Some research has reported the cases of acute pericarditis by CMV infection [9-11]. Also, Sever *et al.* [12] analyzed the development of pericarditis on renal allograft recipients to investigate prevalence, etiology and outcomes of pericarditis. Of the 1,497 patients, pericarditis occurred in 34 patients. Pericarditis was attributed to uremia in fourteen patients, CMV infection in three, both uremia and CMV infection in four and nonspecific bacterial infection in three patients [12].

Conclusion

Although the incidence of uremic pericarditis has been decreased over time by development of hemodialysis, uremic pericarditis accompanying cardiac tamponade can lead to serious hazards if not treated properly. Pericarditis can occur by several causes such as delayed dialysis or viral infection in chronic kidney disease patients, and bleeding tendency of patients and the choice of anticoagulant can affect the occurrence of uremic hemorrhagic pericarditis. Therefore, careful consideration of these factors is important for effective and safe treatment of uremic pericarditis and cardiac tamponade.

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