

Uremic Pericardial Effusion: A Case Report

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Abstract

A 24 year old man, recently known to have hypertension was admitted to our hospital for acute shortness of breath with central chest pain. His investigations revealed end stage renal disease with a normochromic normocytic anaemia. There was cardiomegaly on his chest radiograph and initial echocardiography did not reveal a pericardial effusion. Haemodialysis was initiated and his renal profile steadily improved. His serial chest radiographs from day 10 post hemodialysis showed increasing heart size. Echocardiography revealed a new pericardial effusion without the signs of pericardial tamponade. His pericardial effusion was completely resolved 4 weeks after admission with more intensive haemodialysis regimens, including daily short dialysis. This supports the notion that patients with uremic pericarditis resolve rapidly with intensive dialysis.

Keywords: End stage renal failure, Haemo dialysis, pericardial effusion, Uremic Pericarditis

Case report

A 24-year-old man was admitted to Melaka general hospital with acute shortness of breath with central chest pain. Over the last 2 months, he has noticed progressive fatigue and recently describes orthopnoea. He denied oliguria and puffiness of the face. He did not have fever, chill, nausea or vomiting, loin or abdominal pain and haematuria. He was diagnosed with hypertension a year ago but is not on regular medication and follow up. There was no relevant family history. He was a smoker and occasionally consumed alcohol.

On examination the patient was markedly pale, tachypnoeic and dyspnoeic. There was no puffiness of the face and pitting pedal oedema. He was hypertensive, with a blood pressure of 180/100 mmHg and a pulse rate of 105 beats per minute. The temperature was normal and respiratory rate was 21 breaths per minute. His lungs were clear. The heart sounds were normal and there were no

murmurs or pericardial rub. The abdomen was soft with no organomegaly or ascites. The remainder of the examination was normal.

The laboratory –test results revealed normocytic normochromic anaemia, markedly elevated level of blood urea and serum creatinine ,acute kidney injury(AKI) stage III with normal levels of serum electrolytes. Arterial blood gas showed metabolic acidosis. Urinalysis revealed protein 1+ and was otherwise normal. His chest radiograph revealed cardiomegaly with no sign of heart failure. Echocardiography showed ejection fraction (EF) of 56% with normal left ventricular function and there was no pericardial effusion. The cardiac enzymes showed no evidence of myocardial infarction. Other laboratory results were shown in Table 1.

His anaemia was corrected by packed cell transfusion and haemodialysis was initiated. Ten days later, he developed fever, cough and shortness of breath. His chest radiograph revealed enlarged heart with right sided pleural effusion. He was treated as a case of para pneumonic pleural effusion. It was completely resolved after treatment with broad spectrum antibiotics. After 2 weeks, he complained of retrosternal discomfort especially on lying down. He denied fever, cough and shortness of breath. On examination, the temperature was normal, the blood pressure 170/70 mm Hg, and the pulse 92 beats per minute and the respiratory rate 20 breaths per minute. The first and second heart sounds were normal: a pericardial rub was heard. The rest of the examination was normal. An electrocardiogram showed sinus tachycardia. Chest radiograph revealed globular shaped cardiomegaly suggestive of pericardial effusion(Figure 2).His echocardiography showed EF of 57%, pericardial effusion of 2.1cm inferiorly and 2.6cm laterally, no right atrium (RA) or right ventricle (RV) collapse (Figure 1).

His clinical condition was stable on regular intensive haemodialysis.After 4 weeks of regular haemodialysis, his pericardial effusion was completely resolved on echocardiography.

Discussion

Pericardial disease including uremic pericarditis and pericardial effusion is one of the common complications of end stage renal failure. Two forms have been described, uremic pericarditis and dialysis associated pericarditis. Uremic pericarditis may occur in 35-50% of patients with advanced renal failure in the predialysis era and dialysis-associated pericarditis account to 8-12% of patients after the onset of dialysis.^{1,2} However, prompt dialysis has dramatically decreased the prevalence of uremic pericarditis. But this problem is still devastating blow to the dialysis patient. About 3-5% of cases were fatal resulting from cardiac tamponade or arrhythmias.³ Large pericardial effusion may develop in up to 20% of patients. Pericardial effusion in non-dialyzed patient is usually of limited amount and cardiac tamponade does not occur. In contrast among dialysed patients, chronic pericardial effusion develops variable degree of cardiac temponade.

The recognized clinical features are fever and pleuritic chest pain but many patients are asymptomatic.⁴ Pericardial rubs may present transiently but it can persist in large effusions.⁵

According to various studies, leucocytosis is commonly seen in 40 to 76% of cases with uremic pericardial effusion. The cardiomegaly is found in 97 to 100% of cases in chest radiography secondarily to pericardial effusion or left ventricular hypertrophy.⁶ The typical ECG changes of

acute pericarditis; diffuse ST and T wave elevations may not always be observed in patients with uremic pericarditis. This may be explained by the absence of invasion of inflammatory cells into the myocardium.⁷ Typical ECG changes (e.g., electrical alternans) may be found in cases with cardiac tamponade. ECG evidence of pericardial involvement is seen in 51 to 100% of symptomatic cases with uremic pericarditis⁸. Transthoracic Echocardiography is a useful tool in the diagnosis of pericardial effusion. In moderate or large pericardial effusion, impending cardiac tamponade can be identified by the presence of right ventricular diastolic collapse in echocardiography.

The majority of patients with uremic pericarditis resolve rapidly by intensive dialysis⁷. Frequent dialysis usually leads to resolution of the pericarditis within one to two weeks⁸. Intensive dialysis including daily short dialysis is effective in majority of patients with uremia who develop pericarditis prior to dialysis. About 25 to 33% of cases with dialysis-induced pericarditis did not respond to intensive dialysis.⁸

Pericardiocentesis, drainage of a pericardial effusion, is the treatment of choice if it fails to improve within 7 to 14 days with intensified dialysis or if it increases in size.⁹ Early and timely dialysis plays as a major role in prevention of the development of uremic pericarditis.

Conclusion

Uremic pericarditis is one of the common complications of uremia and the life threatening complications like pericardial effusion with acute cardiac tamponade can occasionally develop. The incidence, mortality and morbidity of pericarditis in patients with renal failure can be decreased by advance management. The early and timely intensive dialysis is the most effective treatment for these patients. Surgical intervention, pericardiocentesis can be considered in cases with cardiac tamponade.

Acknowledgements

The special thank goes to my helpful supervisor, Datuk Prof. Dr. Jaswan for proofreading this report. The authors of this report would like to extend our sincere acknowledgement to all those who provide the essential case documents and data of this case possible. Appreciation also goes to the contributions of our medical staff at Melaka General Hospital.

Conflict of Interest: None declared.

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Table 1: Laboratory variables

| Variable | On Admission | After 2weeks | After 4 weeks (On Discharge) | Reference Range, Adults |
|--|--------------|--------------|---------------------------------|----------------------------|
| Haemoglobin(g/dl) | 3.5 | 7 | 10.4 | 13.5-17.5 |
| White-cell count(per mm ³) | 15.1 | 10.9 | 11.6 | 4-11 |
| Differential count (%) | | | | |
| Neutrophils | 51 | 69 | 67 | 40-70 |
| Lymphocytes | 32 | 24 | 30 | 22-44 |
| Monocytes | 7 | 6 | 8 | 4-11 |
| P.C.V (%) | 9.9 | 20.6 | 29 | 40-50 |
| M.C.V.(fL) | 85 | 84 | 83 | 83-101 |
| M.C.H.(pg) | 28.5 | 28.6 | 28.1 | 27-32 |
| M.C.H.C.(g/dL) | 33.6 | 34 | 34.1 | 31.5-34.5 |
| Platelet count(per mm ³) | 262 | 198 | 310 | 150-400 |
| Sodium (mmol/liter) | 134 | 135 | 135 | 135-145 |
| Potassium (mmol/liter) | 4.7 | 4.8 | 3.7 | 3.4-4.8 |
| Chloride (mmol/liter) | 104 | 96 | 96 | 100-108 |
| PH | 7.21 | 7.3 | 7.42 | 7.35-7.45 |
| Pa CO ₂ (mmHg) | 31 | 29.8 | 33 | 35-45 |
| Pa O ₂ (mmHg) | 127 | 89 | 90 | 80-100 |

| | | | | |
|-----------------------------------|--------------|-------|------|-------------|
| Bicarbonate(mmol/liter) | 12 | 14.2 | 22 | 21-29 |
| Base Excess(moll/litre) | - 15 | -10.7 | -2 | -2 |
| Blood urea(moll/liter) | 76 | 35.8 | 13.7 | 2.5-6.6 |
| Creatinine(umol/liter) | 2277 | 1245 | 673 | 60-120 |
| Glucose(moll/liter) | 5.6 | | | |
| Iron(umol/litre) | 13.1 | | | 10.6-28.3 |
| TIBC(umol/litre) | 35.8 | | | 40.8-76.6 |
| Calcium(moll/litre) | 1.27 | 1.89 | 2.15 | 2.15-2.55 |
| Phosphate(moll/litre) | 2.84 | 2.22 | 1.57 | 0.87-1.45 |
| Magnesium(moll/Litre) | 0.88 | | | 0.65-1.05 |
| Uric acid(umol/litre) | 691 | | 472 | 202.3-416.5 |
| Troponin T | Negative | | | |
| Creatinine Kinase(U/l) | 126 | | | 55-170 |
| Urine for culture and sensitivity | No growth | | | |
| Blood culture and sensitivity | No growth | | | |
| Hepatitis B surface antigen | Non Reactive | | | |
| Anti- Hepatitis C virus | Non Reactive | | | |
| HIV 1&2 Ag-Ab | Non Reactive | | | |



Figure 1: Echocardiography after 2 weeks of hospital admission showed pericardial effusion of 2.1 cm inferiorly and 2.6 cm laterally, no RA or RV collapse.



Figure 2: Chest radiograph (after 2 weeks) revealing markedly enlarged cardiac silhouette and normal- appearing lung parenchyma suggestive of pericardial effusion