Uraemic Pericarditis With Cardiac Tamponade: A Report of Four Cases

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Pericarditis is a common complication of renal failure. It is usually an aseptic inflammatory process with fibrin formation but little fluid. Many authorities—for instance, de Wardener (1961) and Wood (1961)—make no mention of pericardial effusion resulting from uraemic pericarditis. However, Fishberg (1954) remarked that "exceptionally there is a considerable serous or haemorrhagic effusion," and Lowry and Boyd (1960), out of a total of 227 cases of uraemic pericarditis, recorded 58 with hydropericardium and 3 with massive haemopericardium.

Cardiac tamponade in uraemic pericarditis was first reported in the literature in 1956, when Goodner and Brown recorded the deaths of two young males with chronic renal disease. Acute right heart failure with pulsus paradoxus preceded death in both cases, and at necropsy 800 and 850 ml. of blood-stained fluid were found in the pericardial cavities. Since then six further cases have been reported (Guild, Bray, and Merrill, 1957; Hutt and Holmes, 1961; Merikas, Samartzis, and Marketos, 1962; Rappaport, 1962). In four of these cases some prolongation of life was obtained by pericardial tap; one case improved spontaneously.

The following four cases of cardiac tamponade from uraemic pericarditis were seen in the renal unit at Hammersmith Hospital during 1961-2.

Case 1. Chronic Glomerulonephritis

A youth aged 18 had acute nephritis at the age of 7. This insidiously entered a chronic phase with slightly elevated blood-pressure, persistent proteinuria, anaemia, osteodystrophy, and a gradually rising blood urea. On admission to hospital on 26 June 1962 he was moderately hypertensive (blood-pressure 180/100 mm. Hg) with clinical and electrocardiographic evidence of left ventricular hypertrophy, but there was no evidence of pericarditis. Blood urea was 505 mg./100 ml., and serum sodium 134, serum potassium 4.8, serum chloride 88, serum bicarbonate 10, serum calcium 3.7, and serum phosphate 7.2 mEq/litre.

Haemodialysis was first performed on 4 July 1962 (blood urea 620 mg./100 ml.); an arteriovenous teflon fistula was inserted in the left wrist as subsequent intermittent haemodialyses were planned. He was readmitted on 26 July with pericarditis and quickly developed a large pericardial effusion with signs of cardiac tamponade—paradoxical pulse (10 mm. Hg paradoxus), small pulse pressure (blood-pressure 150–160/120), jugular venous pressure elevated 8 cm., increased cardiac dullness to percussion, a pericardial friction rub, and an enlarged tender liver. Chest *x*-ray examination and electrocardiogram confirmed the clinical diagnosis. Pericardial tap was performed on two occasions (3 and 7 August): 420 and 300 ml. of heavily blood-stained fluid were obtained. Haemodialysis was performed again on 8 August with satisfactory lowering of the blood urea to 88 mg./100 ml. His convalescence was disturbed only by a large blood-stained left pleural effusion; pericardial friction disappeared and further pericardial aspiration was not required.

Repeated attacks of left ventricular failure preceded his final admission on 16 September. Pericardial friction was absent. Haemodialysis on two occasions was only of transient benefit, and he was discharged home, at his father's request, on 23 October. Death occurred on 6 November. Necropsy was not performed.

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Case 2. Renal Amyloidosis

A man aged 40 was first seen in January 1960 because of chronic osteomyelitis of the sacrum and renal amyloidosis. He was moderately uraemic (blood urea 120 mg./100 ml. By March 1961 he had developed malignant hypertension (blood-pressure 200/120 mm. Hg). This responded fairly well to guanethidine and hydrallazine. Two months later he was readmitted because of increasing uraemia (blood urea 330 mg./100 ml.), severe anaemia (haemoglobin 37%), and congestive cardiac failure. Transient pericardial friction was heard for the first time early in June. Haemodialysis was carried out in 7 June as the blood urea had risen to 430 mg./100 ml.

His final admission was on 28 June because of increasing vomiting and fatigue. Examination showed a very dyspnoeic man with classical signs of cardiac tamponade—paradoxical pulse (40 mm. Hg paradoxus), jugular venous pressure elevated to the angle of the jaw, impalpable cardiac impulse, triple rhythm, pericardial rub no longer audible, and 4 cm. of tender, enlarged liver. Chest *x*-ray examination revealed a large globular heart shadow and a left-sided pleural effusion, while an electrocardiogram was also suggestive of a pericardial effusion with decreased voltage of the QRS complexes and flattening and inversion of the T waves. His blood urea was 335 mg./100 ml.; serum sodium 130, serum potassium 7.3, serum chloride 90, and serum bicarbonate 14 mEq/l:; haemoglobin 39%; with burr cells seen on film. Pericardial tap was not performed. Death occurred two days later on 30 June.

Necropsy showed chronic osteomyelitis of the sacrum, with amyloid deposits in the small contracted kidneys, thyroid, adrenals, spleen, oesophagus, and ileum. There was a haemorrhagic pericarditis with 300 ml. of blood in the pericardial sac. The haematocrit of this fluid was 24%. The visceral and parietal layers of the pericardium were covered with shaggy fibrinous exudate.

Case 3. Acute Tubular Necrosis

A married woman age 51 had a radical mastectomy for scirrhous carcinoma of the left breast in June 1959. Bilateral oophorectomy was also performed, followed by a course of deep *x*-ray therapy. Metastatic spread to the mid-dorsal spine had occurred by December 1960. Bilateral adrenalectomy was performed in April 1961; unfortunately a right nephrectomy was also necessary because of trauma to the renal vein. Transient hypotension during the operation was followed by post-operative oliguria (urine volume 20-160 ml. in the first six days) and a rising blood urea.

On admission to hospital on 20 April 1961, she had acidotic respirations and moderate oedema of the legs and sacrum. Her blood-pressure was 160/80 mm. Hg; the jugular venous pressure was not raised, and there was no pericardial friction rub. A chest *x*-ray examination showed nothing abnormal, and an electrocardiogram merely revealed T-wave inversion in leads I and VI. Her blood urea was 375 mg./100 ml.; serum sodium 122, serum potassium 5.8, serum chloride 79, and serum bicarbonate 13 mEq/l.; haemoglobin 49%; white-cell count 13,000/c.mm. The blood film showed a leucoerythroblastic picture.

Haemodialysis was carried out on 22 April. Considerable oozing occurred from incisions in her groin and arm. Chloramphenicol 1 g. daily was started for a coliform urinary infection. Pericardial friction was first heard on 25 April. By 29 April signs of early tamponade were noted—pulsus paradoxus, raised jugular venous pressure—while the electrocardiogram showed slight S-T segment elevation over the chest leads and generally low voltage. Meanwhile her blood urea had risen to 440 mg./100 ml. and further

haemodialysis was performed on 30 April. Signs of cardiac tamponade became more pronounced during the three days following dialysis—pulsus paradoxus of 45 mm. Hg, blood-pressure 115/70 mm. Hg, jugular venous pressure elevated to the angle of jaw, and heart sounds becoming fainter with disappearance of the pericardial friction rub. The clotting-time was 4½ minutes. On 3 May she complained of a sudden episode of shortness of breath and soon became unconscious and pulseless. On aspiration of 650 ml. of heavily blood-stained fluid from the pericardial sac there was an immediate return of pulse and consciousness. However, she relapsed and died shortly afterwards. Previous to this her oliguric renal failure had been improving and just before death her daily urine volume had risen to 350 ml.

Necropsy revealed a uraemic pericarditis with 500 ml. of what appeared to be pure blood in the pericardial cavity. Histological examination failed to show tumour deposits in the pericardium. The changes of acute tubular necrosis were present in the solitary left kidney.

Case 4. Prostatic Hypertrophy—Chronic Pyelonephritis

A 59-year-old man was admitted to the surgical department on 14 October 1961. He had a 12-months history of frequency of micturition (nocturia X 4), and for six months had experienced anorexia, vomiting, headache, and occasional hiccups. A few days before admission he developed a severe choking retrosternal pain. Examination revealed an ill-looking, dehydrated man. His blood-pressure was 160/70 mm. Hg. A pericardial friction rub was present. There were crepitations at the left lung base. Rectal examination revealed an enlarged prostate. A chest *x*-ray film showed atrial fibrillation only. Blood urea was 355 mg./100 ml.; serum sodium 130, serum potassium 8.2, serum bicarbonate 7, serum calcium 3.6, and serum phosphate 7.8 mEq/l.; haemoglobin 44%; and white-cell count 8,000/c.mm.

"Resonium-A" was given by mouth because of hyperkalaemia. On 16 October peritoneal dialysis was begun because of continuing oliguria and rising blood urea (465 mg./100 ml.). By 18 October there were signs of cardiac tamponade (15 mm. of pulsus paradoxus) and a pericardial tap was performed; only 30 ml. of blood-stained fluid was obtained, however. Later the same day the patient became dyspnoeic and his blood-pressure fell to 120/60 mm. Hg. Coarse crepitations were audible all over both lungs. Despite venesection and tourniquets applied to his limbs death occurred soon after.

Necropsy confirmed the clinical diagnosis of pericardial effusion and pulmonary oedema. The pericardial sac contained 800 ml. of heavily blood-stained fluid. The prostate was hypertrophied, with a trabeculated bladder and active cystitis. There was also bilateral hydronephrosis, hydroureter, and pyelonephritis.

Discussion

Cardiac tamponade is not a common complication of uraemic pericarditis. However, it is important to recognize the development of this complication as it is readily amenable to treatment. Its appearance is not necessarily a prelude to death, for one patient (Case 1) lived for three months after pericardial aspiration. Guild *et al.* (1957) have emphasized the reversibility of uraemic cardiac tamponade in their report of two patients who survived this complication after successful aspiration.

The development of tamponade in cases of pericardial effusion depends not only on the amount of fluid within the sac but also on the rapidity of collection of this fluid (Yu, Lovejoy, Joos, Nye, and Simpson, 1953). In each of the above four cases the symptoms and signs arose acutely over a period of a few hours or days, and the speed of clinical deterioration was more rapid than is usual in cardiac tamponade from other causes. This may have been related to the fact that the pericardial fluid was pure blood in two cases, and heavily blood-stained in the other two, suggesting bleeding into the pericardial cavity. Uraemic subjects often have a bleeding tendency, and in Case 3 this was apparent from persistent oozing from the groin and arm incisions performed for haemodialysis. Indeed, in this patient cardiac tamponade appears to have been aggravated by the second haemodialysis; the critical factor might have been heparinization. Anticoagulants cannot be held responsible for the development of haemopericardium in the other three cases (see accompanying Table). Indeed, in Case 1, resolution of the pericardial effusion was hastened after his final dialysis and pericardial friction was not heard again.

Case No.	Sex and Age	Renal Disease	Blood Urea at Time of Death (mg/100 ml.)	Duration of Observed Pericarditis	Interval Between Haemodialysis and Cardiac Tamponade	Pericardial Contents at Necropsy
1	M 18	Chronic glomerulonephritis	>600	2 weeks	3 weeks	No necropsy
2	M 40	Renal amyloidosis. Chronic osteomyelitis of sacrum. Malignant hypertension	335	9 weeks	3 weeks	300 ml. blood
3	F 51	Post-operative acute tubular necrosis in solitary kidney (Metastatic breast carcinoma)	119	8 days	3 days	500 ml. blood
4	M 59	Benign prostatic hypertrophy with bilateral hydronephrosis and chronic pyelonephritis	520	4 days	No haemodialysis	800 ml. blood-stained fluid

The exact cause of uraemic pericarditis is still unknown, but in clinical practice the incidence and severity of pericarditis seem to be directly related to the severity of renal failure. The Table shows no direct relationship between the measured level of blood urea and cardiac tamponade, but such a relationship might have been obscured in Cases 2 and 3 by treatment with haemodialysis during the last few weeks of life.

Summary

Cardiac tamponade is a rare complication of uraemic pericarditis. Four new cases are described. The condition may be successfully treated by pericardial aspiration, and uraemic patients should therefore not be allowed to die of tamponade if their renal failure is potentially reversible.

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