Pericardial effusions in patients with end-stage renal disease¹

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SUMMARY Echocardiography has greatly increased the accurate recognition of pericardial effusion. Echocardiograms were performed prospectively on the total group of 35 stable asymptomatic patients on chronic haemodialysis to determine the incidence of pericardial effusion. Effusions were shown in 11 per cent (4/35); only 6 per cent (2/35) were estimated as greater than 100 ml. For comparison, records were reviewed retrospectively from 41 haemodialysis patients referred during a 27-month period for echocardiographic assessment of suspected pericardial effusion. These 41 patients came from a total group of 108 patients treated with chronic dialysis over this interval. Of 41 examined, 21 (51%) or 21 of 108 (19%) of the population at risk had an effusion. Of 21 with echocardiographic effusions, 15 (71%), or 15 of 41 (37%) of those with clinically suspected effusion, had more than 100 ml fluid. Gross (> 100 ml) pericardial effusions are infrequent in stable, asymptomatic patients with end-stage renal disease. When clinical findings suggest pericardial disease, the echocardiographic demonstration of over 100 ml pericardial fluid is indicative of new effusion, rather than coincidental pre-existing effusion.

Pericarditis has been recognised as a clinical feature of uraemia for over 100 years, and frequently was a harbinger of death in the era before dialysis (Beaudry *et al.*, 1966). More recently, recognition and treatment of uraemic pericarditis and its complications have allowed more patients to survive acute episodes and continue to be maintained on chronic haemodialysis.

Echocardiography has gained widespread use for the non-invasive detection of pericardial effusion (Feigenbaum, 1972). Careful echocardiographic evaluation may reliably define effusions as small as 20 ml (Horowitz *et al.*, 1974). We were initially impressed by the apparently high prevalence of pericardial effusion among patients on maintenance haemodialysis referred for echocardiography because clinical symptoms suggested pericardial disease. We considered the possibility that effusions might be present chronically among many patients with end-stage renal disease, perhaps as a result of

sustained volume overload. As long as such effusions were small and did not cause circulatory embarrassment, they would remain clinically silent and undetected. If this were the case, then the echocardiographic demonstration of effusion in patients on chronic dialysis would be of different clinical import than if subclinical effusions were uncommon. The current study was undertaken to define more clearly the significance of the echocardiographic demonstration of pericardial effusion in these patients and to clarify the role of echocardiography in their management.

Methods

Thirty-five asymptomatic, stable patients comprising the total group who had been dialysed chronically in our unit for at least 2 months (range 23 to 72 months) underwent echocardiographic examination during a 4-month study period (group A). None had any evidence of pericarditis during the study period. Of these 35 patients, 6 had had previous echocardiographic studies to rule out pericardial effusion at least 6 months before their inclusion in this asymptomatic group. For comparison, the medical records from a second group of all 108 patients treated with chronic haemodialysis

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during the 27-month period between June 1972 and September 1975 were reviewed retrospectively. Of these 108 patients, 41 had been studied echocardiographically because clinical symptoms of fever, chest pain, friction rub, or persistent hypotension during dialysis caused suspicion of pericarditis (group B).

Echocardiograms were performed with a Smith-Kline Instruments Ekoline Model 20A Ultrasono-

scope, which emits 1000 pulses/s. The transducer was a 2.25 MHz, 13 mm active diameter model with an acoustic lens providing beam collimation to 5 cm. Returning signals were recorded on a strip chart using either the Irex 101 or Honeywell 1856 Visicorder. M-mode sector scanning was used in all cases. Lead II of the electrocardiogram was recorded simultaneously with the echocardiogram. The

Fig. 1 Echocardiogram from an asymptomatic stable patient. A posterior echo-free space is observed which extends into but not through diastole. The pericardium is flat relative to the epicardium. ECG, electrocardiogram; IVS, interventricular septum; En, endocardium; C, chord; Ep, epicardium; P, pericardium; EFS, echo-free space.

Fig. 2 Classic echocardiographic pattern of pericardial effusion, also from an asymptomatic stable patient. A posterior echo-free space extends throughout systole and diastole, and the pericardium remains flat relative to the epicardium. See Fig. 1 for key to abbreviations.

Fig. 3 Pericardial effusion with possible early pericardial thickening. An additional linear echo (*) is present in the echo-free space between the flat pericardium and the epicardium. See Fig. 1 for key to abbreviations.

patient's chest was raised approximately 30° from the horizontal, in the supine or shallow (30°) left lateral decubitus position. The transducer was placed in the third, fourth, or fifth intercostal space at the left sternal border. The chosen transducer position permitted recording of the echoes from the mitral valve leaflets while the transducer was perpendicular to the chest wall in the sagittal plane. Inferolateral angulation of the transducer from this position allowed recording of the echoes from the ventricular structures.

A technically satisfactory study was defined as the simultaneous recording of echoes from the posterior left ventricular pericardium, epicardium, endocardium, mitral chordae tendineae, interventricular septum, and anterior heart wall. In order to locate the strong pericardial echo, the damping control was abruptly increased after the optimal gain settings for the ventricular structures had been achieved (Feigenbaum, 1972). Continuous recording of this area over several cardiac cycles at maximal damping permitted the clear observation of isolated pericardial movement. Slightly less damping often brought out the epicardium and occasionally a small separation between the posterior epicardium and pericardium. As the damping was further reduced, this small separation often was obscured. Minimal damping allowed identification of all ventricular structures and verified the patterns of pericardial, epicardial, and endocardial echoes of a satisfactory study. The volume of pericardial fluid was estimated as described by Horowitz *et al.* (1974).

In order to attempt to determine if myocardial contractility was altered in uraemic patients at the time that effusions were present, the fractional shortening was measured in symptomatic and asymptomatic patients. The left ventricular internal diameters at end-diastole (LVID_d) and end-systole (LVID_s) were measured and used to calculate fractional shortening (FS), an index of circumferential myocardial contractility (McDonald *et al.*, 1972; McDonald, 1976), which was defined as

$$(\%)FS = \frac{LVID_d - LVID_s}{LVID_d} \times 100 \quad [1]$$

Normal values for fractional shortening in our laboratory are 28 to 41 per cent. Informed consent was obtained from all asymptomatic patients.

Results

Of the 35 asymptomatic patients in group A, 4 (11%) had pericardial effusions and only 2 (6%) had effusions estimated as more than 100 ml (Horowitz *et al.*, 1974) (Fig. 1-3). This was in distinct contrast to the findings among the symptomatic

Table Left ventricular fractional shortening in patients with end-stage renal disease

	No.	Fractional shortening (%)
Asymptomatic stable	23	36.2 ± 1.8
Symptomatic:		
With effusion	20	30.6 ± 2.3
No effusion	19	26.0 ± 2.3

*P < 0.05 [normal = 28-41%].
NS = not significant.

patients in group B. Of 108 patients at risk during the study period, 41 had echocardiograms performed because clinical symptoms suggested pericardial disease and 51 per cent (21/41) had an effusion. Six of these 41 patients had small effusions estimated to be less than 100 ml, while 15 of 41 patients (37%) had effusions greater than 100 ml. Some patients had signs we interpret as organisation of the pericardial fluid or pericardial thickening (Fig. 3).

Records of excellent technical quality satisfactory for calculation of fractional shortening were obtained in 23 patients in group A and 39 patients in group B (Table). Mean values for fractional shortening were similar among symptomatic patients with or without pericardial effusions, but the values were less than those among asymptomatic patients (Table). Eleven patients with effusions had multiple (2 to 7) echocardiographic examinations performed, either to follow the course of a single effusion or to assess several episodes of pericardial disease. Improvement or deterioration in ventricular function as measured by fractional shortening did not correlate with changes in the volume of effusion in this small series.

Discussion

Uraemic pericarditis continues to cause significant morbidity and mortality among patients in haemodialysis programmes (Bailey *et al.*, 1968; Comty *et al.*, 1971; Baldwin and Edwards, 1976). Past studies of clinically evident uraemic pericarditis have indicated a prevalence of between 16 and 41 per cent among patients in chronic dialysis programmes. Many patients have developed pericarditis 3 months or longer after starting dialysis, in spite of technically adequate and frequent dialysis (Beaudry *et al.*, 1966; Bailey *et al.*, 1968; Comty *et al.*, 1971). In the past, only frank pericarditis or tamponade which produced signs such as chest pain, friction rub, or hypotension have been detected. The clinical

assessment of rises in the jugular venous pulse has proved unreliable because obvious and sometimes rapid changes in intravascular volume are associated with dialysis. Several investigators have observed that measurements of blood urea nitrogen or creatinine do not predict the patients who will develop pericarditis (Comty *et al.*, 1971).

With the advent of echocardiography, a simple, sensitive, and uniformly safe method to diagnose pericardial effusion has become available. We used echocardiography to study prospectively a group of asymptomatic stable patients undergoing chronic haemodialysis. This method allows reliable identification of patterns associated with even small amounts of pericardial fluid, but we did not attempt to quantify the volume of effusion when the echocardiographic pattern suggested only 20 ml to 100 ml of pericardial fluid. Unsuspected effusions of 100 ml or larger were infrequent (6%). For comparison, the echocardiograms were reviewed from 41 patients who had symptoms suggestive of pericardial disease at the time of study. In 51 per cent of these patients, pericardial effusions were shown, and in 37 per cent the effusions were estimated to be greater than 100 ml. These observations imply that when clinical findings suggest pericardial disease, the echocardiographic demonstration of 100 ml or more of pericardial fluid is highly indicative of new effusion, rather than of coincidental pre-existing effusion. We believe such patients warrant careful and continuing clinical observation, as well as frequent echocardiographic follow-up.

Necropsy studies of patients who have died as a result of uraemic pericarditis have shown evidence of an associated myocarditis, suggesting possible impairment of ventricular function (Langendorf and Pirani, 1947; Gouley, 1940; Baldwin and Edwards, 1976). Our current echocardiographic assessment of left ventricular function in small numbers of patients showed that fractional shortening was not uniformly depressed when pericardial effusions were present, and that changes in left ventricular performance did not parallel changes in the volume of pericardial effusion measured echocardiographically.

The measurement of change in left ventricular dimensions is subject to error in those patients with sufficient pericardial effusion to allow the heart to swing about within the pericardium, so the sound beam strikes different parts of the walls at end-diastole and end-systole. It is interesting that the patients with symptoms but no effusion had the lowest values for fractional shortening. Additional prospective studies of larger numbers of patients with end-stage renal disease will be necessary to clarify these findings.

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