

LONG-TERM FOLLOW-UP OF IDIOPATHIC CHRONIC PERICARDIAL EFFUSION

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ABSTRACT

Background A large idiopathic chronic pericardial effusion is a collection of pericardial fluid that persists for more than three months and has no apparent cause. We conducted a prospective study of the natural history and treatment of this disorder.

Methods Between 1977 and 1992, we prospectively evaluated and enrolled patients with large idiopathic chronic pericardial effusion. We performed pericardiocentesis in most of the patients. We performed pericardiectomy when large pericardial effusion reappeared after pericardiocentesis. Follow-up ranged from 18 months to 20 years (median, 7 years).

Results During the study period, we evaluated a total of 1108 patients with pericarditis, 461 of whom had large pericardial effusion. Twenty-eight of these patients (age range, 7 to 85 years; median, 61) had large idiopathic chronic effusion and were included in the study. The duration of effusion ranged from 6 months to 15 years (median, 3 years). At the initial evaluation, 13 patients were asymptomatic. Overt tamponade was found in eight patients (29 percent). Therapeutic pericardiocentesis, performed in 24 patients, was followed by the disappearance of or marked reduction in the effusion in 8. Five of the 24 patients underwent early pericardiectomy, and in 11 large pericardial effusion reappeared. Cardiac catheterization, performed in 16 patients, showed elevated intrapericardial pressure (mean [\pm SD], 4.75 ± 3.79 mm Hg) and reduced transmural pressure (1.0 ± 2.50 mm Hg) before pericardiocentesis. Both of these abnormalities in pressure improved significantly after pericardiocentesis. Pericardiectomy, performed in 20 patients, yielded excellent long-term results. At the end of the follow-up period, 10 patients had died, but none had died from pericardial disease.

Conclusions Large idiopathic chronic pericardial effusion is well tolerated for long periods in most patients, but severe tamponade can develop unexpectedly at any time. Pericardiocentesis alone frequently results in the resolution of large effusions, but recurrence is common and pericardiectomy should be considered whenever a large effusion recurs after pericardiocentesis. (N Engl J Med 1999;341:2054-9.)

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SINCE echocardiography became a routine diagnostic tool, pericardial effusion has been a common clinical finding. In most instances, the cause of pericardial effusion can be determined; however, in some cases the cause is not apparent even after a thorough diagnostic evaluation, despite the fact that the effusion may have been present for years or even decades.¹⁻³ Patients with such find-

ings are said to have idiopathic chronic pericardial effusion.

The natural history, clinical course, and optimal management of large idiopathic chronic pericardial effusion have not been clearly defined.^{1,4-16} Therefore, we performed a prospective study of 28 consecutive patients, some of whom were followed for 20 years.

METHODS**Definitions**

We considered patients to have large idiopathic chronic pericardial effusion if they met the following criteria: the cause of effusion was not apparent after a thorough evaluation that included examination of pericardial fluid or tissue, the sum of anterior and posterior echo-free spaces exceeded 20 mm at end diastole,¹⁷ there was no progression of disease during the observation period, and the effusion persisted for more than three months. Cardiac tamponade was diagnosed when the following classic clinical criteria were met: hypotension (systolic blood pressure less than 90 mm Hg), pulsus paradoxus (a decline of more than 10 mm Hg in inspiratory systolic blood pressure), and increased jugular venous pressure.

Study Patients

We prospectively enrolled patients with suspected idiopathic chronic pericardial effusion between January 1977 and December 1992, at which time we discontinued recruitment in order to obtain long-term follow-up data on all patients. All patients were recruited from our hospital ward or outpatient clinic.

Each patient underwent a clinical evaluation,^{18,19} which included a complete medical history taking, physical examination, electrocardiography, chest radiography, echocardiography, and the following laboratory tests: complete blood count; tests of liver function; measurements of blood urea nitrogen, serum creatinine, serum glucose, serum electrolytes, serum triiodothyronine, serum thyroxine, and serum thyroid-stimulating hormone; and tests for rheumatoid factor, antibodies against toxoplasma, antibodies against DNA, and antinuclear antibodies. We also performed tuberculin skin tests and tested for tubercle bacilli in sputum or gastric aspirates. We further investigated positive or abnormal findings in specific studies. We also searched for previous chest films and echocardiograms that might be used to confirm the duration of the effusion.

We performed pericardiocentesis in the catheterization laboratory. We used a 5-French pigtail angiographic catheter for pericardiocentesis and to measure intrapericardial pressure and pressure from the right and left ventricular chambers simultaneously. We calculated right atrial transmural pressure (right atrial pressure minus intrapericardial pressure) from the simultaneous phasic strip-chart tracings. The average of all measurements of instantaneous pressure (taken every 0.2 second) during the second half of expiration was calculated in three consecutive cycles of respiration. The catheters were removed from the pericardium after the procedure. Pericardial fluid was aspirated for the measurement of glucose, total protein, cholesterol, lactate dehydrogenase, and aden-

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osine deaminase; for cytologic studies; for aerobic and anaerobic bacterial cultures; and for examination for the presence of acid-fast bacilli. Pericardiocentesis was followed by serial echocardiography. The last 16 patients who were enrolled in the study underwent a second pericardiocentesis if pericardial effusion reappeared.

Wide anterior pericardiectomy was considered to be indicated if a large effusion reappeared and persisted for more than six months. All specimens obtained during pericardiectomy were submitted for histologic and bacteriologic studies.

Follow-up

The patients were monitored in the outpatient clinic. After pericardiocentesis, they were seen every three months during the first year and then yearly, or whenever symptoms developed. After pericardiectomy, they were seen every three to five years after the first year.

Statistical Analysis

Data on catheterization are presented as means \pm SD. We compared values before and after pericardiocentesis using Student's *t*-test for paired data.

RESULTS

Clinical Findings

Among 1108 patients with pericarditis of any type, 461 had large pericardial effusion; of these patients, 41 fulfilled our criteria for large chronic effusion. We identified specific causes or associated diseases in 11 of the 41 patients: 3 patients had hypothyroidism, 2 had radiation-induced pericardial effusion, 2 had toxoplasmosis,²⁰ 2 had severe pulmonary hypertension and right ventricular failure, 1 had an atrial septal defect, and 1 had chylopericardium. Two additional patients were not examined completely, because they declined to undergo pericardiocentesis and pericardiectomy. Therefore, 28 patients fulfilled our criteria for inclusion in the study.

There were 19 female patients and 9 male patients (age range, 7 to 85 years; median, 61). The most common reason for hospitalization was to investigate an incidental finding of chronic pericardial effusion (in 13 asymptomatic patients). Another seven patients reported atypical chest pain (pain that was not characteristic of pericarditis or angina) or mild dyspnea on exertion. Seven other patients were admitted because of overt tamponade. The remaining patient had features of acute pericarditis. No patient had constitutional symptoms.

In all patients, the chronicity of the effusion was suggested by a huge cardiac silhouette. Patients for whom no previous chest films or echocardiograms were available that might be used to confirm the duration of the effusion were not included in the study until they had had stable pericardial effusion for three months. In the case of the 15 symptomatic patients, particularly those with tamponade, this approach permitted us to be confident of the chronicity of the effusion. The duration of pericardial effusion ranged from 6 months to 15 years (median, 3 years).

In patients without overt cardiac tamponade, the

physical examination was unremarkable. Two patients had congenital facial lymphangioma. Only one patient had a pericardial friction rub.

Twenty-three patients were in sinus rhythm, and five had atrial fibrillation. Six met the criteria for low QRS voltage. Flattened or slightly negative T waves were observed in seven. On chest films, the cardiothoracic index ranged from 0.63 to 1. The lung fields were clear in all patients. One patient had moderate left-sided pleural effusion.

On echocardiography, the sum of anterior and posterior echo-free spaces at end diastole ranged from 23 to 54 mm (anterior, 3 to 35; posterior, 10 to 45). Right atrial collapse was found in five patients. The dimensions of left and right atrial and ventricular chambers were normal in all patients.

We evaluated pericardial fluid obtained from all 28 patients. In 20 patients the fluid was serofibrinous, and in 8 it was serosanguineous. No malignant cells were found. Protein concentrations ranged from 3.5 to 5.8 g per liter (normal range, 1.7 to 3.5). The activity of adenosine deaminase (measured in 17 patients) ranged from 7 to 12 U per liter (normal values, less than 20). Cultures for bacteria, fungi, and acid-fast bacilli were negative in all patients.

Overt cardiac tamponade was present in eight patients (29 percent). It was the reason for hospitalization in seven patients who had had pericardial effusion for one to eight years (median, four). During follow-up, overt tamponade developed in one additional patient after she had had pericardial effusion for two years. The development of tamponade was both unexpected and unexplained in all eight patients.

Findings at Catheterization

Cardiac catheterization was planned in the last 18 patients to be enrolled in the study and was performed in 16. Intrapericardial pressure fell from 4.75 ± 3.79 mm Hg (range, 0 to 13) before pericardiocentesis to -4.06 ± 2.46 mm Hg (range, -10 to 1) after pericardiocentesis ($P < 0.001$) (Fig. 1). Right atrial transmural pressure increased from 1.0 ± 2.50 mm Hg (range, 0 to 10) to 5.94 ± 3.51 mm Hg (range, 1 to 15) ($P < 0.001$) (Fig. 2). Right atrial pressure fell from 6.25 ± 3.75 mm Hg (range, 0 to 14) to 3.0 ± 3.56 mm Hg (range, -4 to 8) ($P = 0.001$). The inspiratory decrease in systolic arterial pressure declined from 13.38 ± 8.19 mm Hg (range, 6 to 36) to 6.08 ± 1.44 mm Hg (range, 4 to 8) ($P = 0.009$). The cardiac index increased from 2.58 ± 1.02 liters per minute per square meter (range, 1.29 to 4.76) to 2.89 ± 0.75 liters per minute per square meter (range, 1.61 to 4.20) ($P = 0.03$).

Therapeutic Procedures

None of the patients received antituberculosis chemotherapy or corticosteroids. Pericardiocentesis was performed in 24 patients (to treat tamponade in 7 and

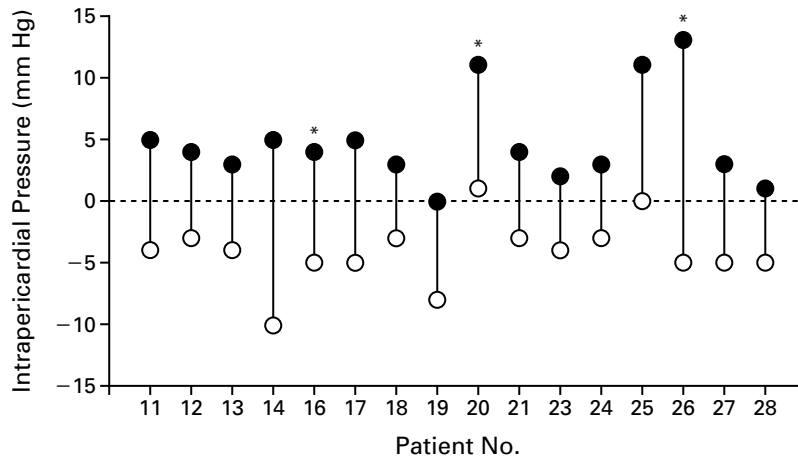


Figure 1. Intra-pericardial Pressure before Pericardiocentesis (Solid Circles) and after Pericardiocentesis (Open Circles) in 16 Patients. Asterisks indicate patients with overt tamponade.

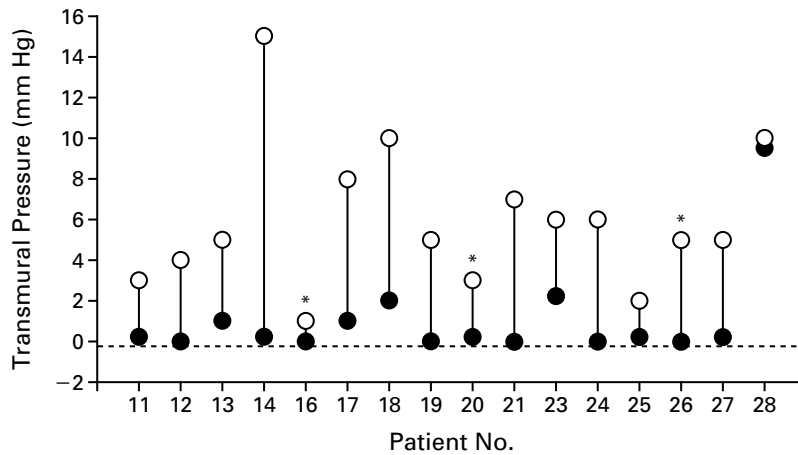


Figure 2. Right Atrial Transmural Pressure before Pericardiocentesis (Solid Circles) and after Pericardiocentesis (Open Circles) in 16 Patients. Asterisks indicate patients with overt tamponade.

as an elective procedure in 17). The volume of pericardial effusion ranged from 250 to 2100 ml (mean, 980). There were no complications of the procedure. The outcome after pericardiocentesis was assessed in only 19 of the 24 patients; 5 of them underwent early pericardiectomy because of reaccumulation of fluid and recurrent tamponade. In 11 patients, pericardial effusion reappeared, and the volume was similar to that present before pericardiocentesis. Another five patients had a marked reduction in the echo-free space, from a mean of 33.8 mm (range, 23 to 54) to 11.0 mm (range, 4 to 17) (after a second pericardiocentesis in one patient). The three remaining patients

had complete resolution of pericardial effusion (after a second pericardiocentesis in one patient). In these eight patients, the duration of pericardial effusion ranged from 6 months to 9 years (median, 3 years), and the mean follow-up after pericardiocentesis was 60 months (range, 13 to 96). Overall, we found that pericardiocentesis resolved or substantially reduced the pericardial effusion in 8 of the 19 patients (42 percent).

Pericardiectomy was performed in the 20 patients in whom pericardiocentesis alone was inadequate in managing the effusion. No deaths were associated with pericardiectomy. In 2 patients it was indicated

to treat overt tamponade, in 5 it was performed soon after therapeutic pericardiocentesis, and in the remaining 13 it was performed as an elective procedure because of a long-term relapse (lasting more than six months) after pericardiocentesis. In 2 of the 20 patients, effusion recurred after pericardiectomy and the volume was the same as that present before the procedure. When the surgical protocols were reviewed, it was found that pericardial resection had been incomplete, so both patients underwent a second pericardiectomy. Postoperative pleural effusion occurred in one patient; it resolved spontaneously. All pericardial specimens appeared grossly normal. Histologic examination revealed mild, chronic nonspecific pericarditis in the 19 available specimens. After extensive pericardiectomy, pericardial effusion did not recur in any patient, nor did persistent pleural effusion develop.

At the end of the diagnostic workup, no specific cause of pericardial effusion could be identified. Accordingly, all 28 patients were considered to have chronic idiopathic pericardial effusion.

Follow-up

No patient was lost to follow-up. There were 10 deaths. In the three patients who died of cancer, neoplasia became apparent 48 months (lung cancer), 37 months (ovarian cancer), and 6 months (hepatocellular carcinoma) after pericardiectomy. None of these patients had histologic or cytologic evidence of cancer in the pericardium. One patient died of renal failure; she had had normal renal function at base line 18 months earlier. One patient died suddenly at home within 30 minutes after the onset of acute chest pain and dyspnea; his last clinical evaluation had shown moderate pericardial effusion that had remained stable during six years of follow-up. The diseases associated with death in the remaining five patients were stroke in two patients, complications of coronary-artery bypass grafting in one patient, and dementia in two patients.

Overall follow-up ranged from 18 months to 20 years (median, 7 years). Follow-up was limited to 18 months in one patient because of a change of residence. In the other 17 surviving patients, follow-up ranged from 6 to 20 years (median, 9), and their most recent follow-up visit was in late 1998. One patient had clinical and echocardiographic evidence of restrictive cardiomyopathy 13 years after pericardiectomy. After an extensive evaluation, which included computed tomography, constrictive pericarditis was ruled out. Pericardial effusion was absent in 14 of the 17 patients; it was mild in 2 and moderate in 1.

DISCUSSION

Our study population comprised patients with large idiopathic chronic pericardial effusion that was treated according to a predefined protocol. Large id-

idiopathic pericardial effusion can be challenging in terms of diagnosis and therapy. Our hospital is a referral center for pericardial disease; thus, the frequency of this disorder may have been overestimated, although only four patients came from outside our catchment area. In any case, the prevalence of this disorder was only 2.2 percent among patients with pericardial disease. Previous information about this disorder has largely been limited to isolated case reports,⁴⁻⁹ small series,^{1,10-14} and reviews^{8,13,15} published before echocardiography became available; moreover, these studies included patients with a variety of easily identifiable causes. A recent study¹⁶ was subject to bias in that it included only patients treated with surgical pericardial drainage and excluded patients with tamponade. Therefore, it is not surprising that an appropriate approach to treatment has not yet been developed.

The lack of specific findings even with thorough initial investigation and in the long-term follow-up in all patients supports our characterization of their disease as idiopathic. Remarkably, during the recruitment period, we found no tuberculous or neoplastic pericarditis in the patients who fulfilled our criteria for large chronic effusion. We did not perform early pericardial biopsy to rule out tuberculosis because we have not seen chronic asymptomatic effusion as the presenting feature of tuberculous pericarditis.^{18,21} In addition, we have been able to find in the literature only two cases of well-documented chronic pericardial effusion due to tuberculosis that met our criteria.^{22,23} We do not believe that tuberculosis needs to be aggressively ruled out in cases of large chronic pericardial effusion. The presence of congenital facial lymphangioma in two patients, an association that has been reported previously,^{5,14} suggests that in some patients chronic pericardial effusion may reflect an anomaly of the lymphatic system. Thus, in agreement with previous reports, our experience shows that large chronic pericardial effusion may be due to a variety of causes, although in the majority (28 of 41 patients in our study) the cause remains unknown after a thorough evaluation.

Unlike the patients with acute pericarditis, most of the patients with large chronic pericardial effusion were women and were older than 50 years. Nearly half the patients (13 of 28) were asymptomatic, and 7 had only mild exertional dyspnea or nonspecific chest pain. An important finding was the high frequency (29 percent) of overt tamponade occurring at some time in the course of the disease. The duration of pericardial effusion in these eight patients ranged from 1 to 8 years (median, 4); tamponade appeared without an apparent precipitating cause in spite of long periods during which the disease was stable. Patients with large chronic pericardial effusion are thus at constant risk for unforeseen overt tamponade.

The incidence of tamponade in patients with chronic large pericardial effusion is difficult to establish be-

cause of the scarcity of published data — patients with tamponade were excluded from some studies.^{11,16} However, most reports^{2,3,12,15,24-26} stress that a remarkable feature of the disorder is its good long-term outcome. Our experience does not confirm this impression, even when we consider that the high frequency of tamponade in our study could be the result of selection bias. The study by Colombo et al.²⁷ is in agreement with our experience. We found that the disorder has a good prognosis only if its treatment includes the prevention of severe tamponade. Our hemodynamic findings provide a possible rationale for the development of overt tamponade. Mildly elevated intrapericardial pressure was found in all but one of the asymptomatic patients, and right atrial transmural pressure was near 0 mm Hg in most, an indication of mild cardiac tamponade.^{28,29} These pressures reverted toward normal after pericardiocentesis.

Our results suggest that pericardial drainage may be effective for the treatment of the disorder. Previous information on this treatment is scanty, although isolated cases in which chronic pericardial effusion was resolved after pericardiocentesis have been published.^{2,30,31} In our study, pericardiocentesis was helpful, since there was long-term resolution of effusion after either one or two pericardiocenteses in 8 of 19 patients (42 percent). By reducing intrapericardial pressure, thorough draining of pericardial fluid may favorably modify the disturbed balance between production and reabsorption of pericardial fluid, thus facilitating its resolution. In addition to its therapeutic value, pericardiocentesis may occasionally identify specific causes of chronic large pericardial effusion, such as toxoplasmosis²⁰ and chylopericardium.³²⁻³⁶

We recommend wide anterior pericardiectomy for patients in whom large pericardial effusion recurs or persists after pericardiocentesis. We have not used creation of a pericardial-peritoneal window,³⁷ creation of a pleuropericardial window,^{11,23,38,39} or percutaneous balloon pericardiotomy,⁴⁰ which have been reported to be useful. However, the most common use for these procedures has been as temporary expedients, mostly in patients with cancer.

In summary, idiopathic chronic pericardial effusion should be diagnosed only after a thorough evaluation of possible underlying causes. As a rule, the condition is well tolerated for long periods, but overt tamponade can supervene unexpectedly at any time. We recommend pericardiocentesis, not passive observation, because it eliminates or greatly decreases effusion over the long term in a substantial proportion of patients. Pericardiectomy should be considered whenever large effusion recurs after pericardiocentesis.

We are indebted to Henry Dargie, M.D., and Ralph Shabetai, M.D., for their editorial contributions and assistance in the preparation of the manuscript.

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