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Clinical Characteristics of Constrictive Pericarditis Diagnosed by Echo-Doppler Technique in Korea

A retrospective analysis of clinical data of 71 patients with constrictive pericarditis (CP) diagnosed by echo-Doppler technique (mean age, 49±17) was done. In 27 patients (38%), the etiology was unknown, and the three most frequent identifiable causes were tuberculosis (23/71, 32%), cardiac surgery (8/71, 11%), and mediastinal irradiation (6/71, 9%). Pericardiectomy was performed in 35 patients (49%) with a surgical mortality of 6% (2/35), and 11 patients (15%, 11/71) showed complete resolution of constrictive physiology with medical treatment. Patients with transient CP were characterized by absence of pericardial calcification, shorter symptom duration, and higher incidence of fever, weight loss, and tuberculosis. The 5-yr survival rates of patients with transient CP and those undergoing pericardiectomy were 100% and 85±6%, respectively, which were significantly higher than that of patients without undergoing pericardiectomy (33±17%, p=0.0083). Mediastinal irradiation, higher functional class, low voltage in ECG, low serum albumin, and old age were the independent variables associated with a higher mortality. Tuberculosis is still the most important etiology of CP in Korea, and not infrequently, it may cause transient CP. Early diagnosis and decision-making using follow-up echocardiography are crucial to improve the prognosis of patients with CP.

Key Words : *Pericarditis, Constrictive; Echocardiography, Doppler*

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INTRODUCTION

Constrictive pericarditis (CP) is a rare disease entity characterized by ambiguous clinical presentation of dyspnea, abdominal distension, and generalized edema, which is not infrequently misdiagnosed as chronic liver or renal disease. However, as pericardiectomy can dramatically improve the patient's condition, correct diagnosis of CP is of great importance for physicians. Traditionally, invasive cardiac catheterization has been used to measure elevated intracardiac pressure due to constriction, but sometimes, endomyocardial biopsy is needed for differential diagnosis of various forms of restrictive cardiomyopathy.

Recently, echo-Doppler technique has been reported as an accurate non-invasive diagnostic method for CP; 2-dimensional echocardiography can measure the thickness of pericardium and Doppler technique can easily estimate the characteristic hemodynamics of CP (1-3). Due to a very high feasibility in clinical practice, wide application of this echo-Doppler technique in various clinical situations can contribute to early detection of CP. Moreover, as this technique is totally non-invasive, repeated examination can provide excellent opportunity to get information about temporal changes and natural history of CP. Although the changing clinical features of CP in western countries have been reported using this technique, information about clinical characteristics of CP in Korea, such as etiology, hospital course, treatment results, and prognosis, are not available yet (4, 5). The purpose of this study was to characterize the clinical features of patients with CP in Korea diagnosed by echo-Doppler technique.

MATERIALS AND METHODS

A total of 71 patients was diagnosed to have CP by echo-Doppler technique at Asan Medical Center from March 1991 to June 2000. All patients underwent meticulous transthoracic or transesophageal echocardiographic examination using a commercially available echocardiographic machine (Hewlett Packard Sonos 2500 or 5500). Typical diagnosis of CP was based on demonstration of thickened pericardium (Fig. 1) and exaggerated bouncing motion of interventricular septum with characteristic increase of right ventricular dimension during inspiration (Fig. 2A). Markedly dilated inferior vena cava without normal inspiratory collapse (Fig.

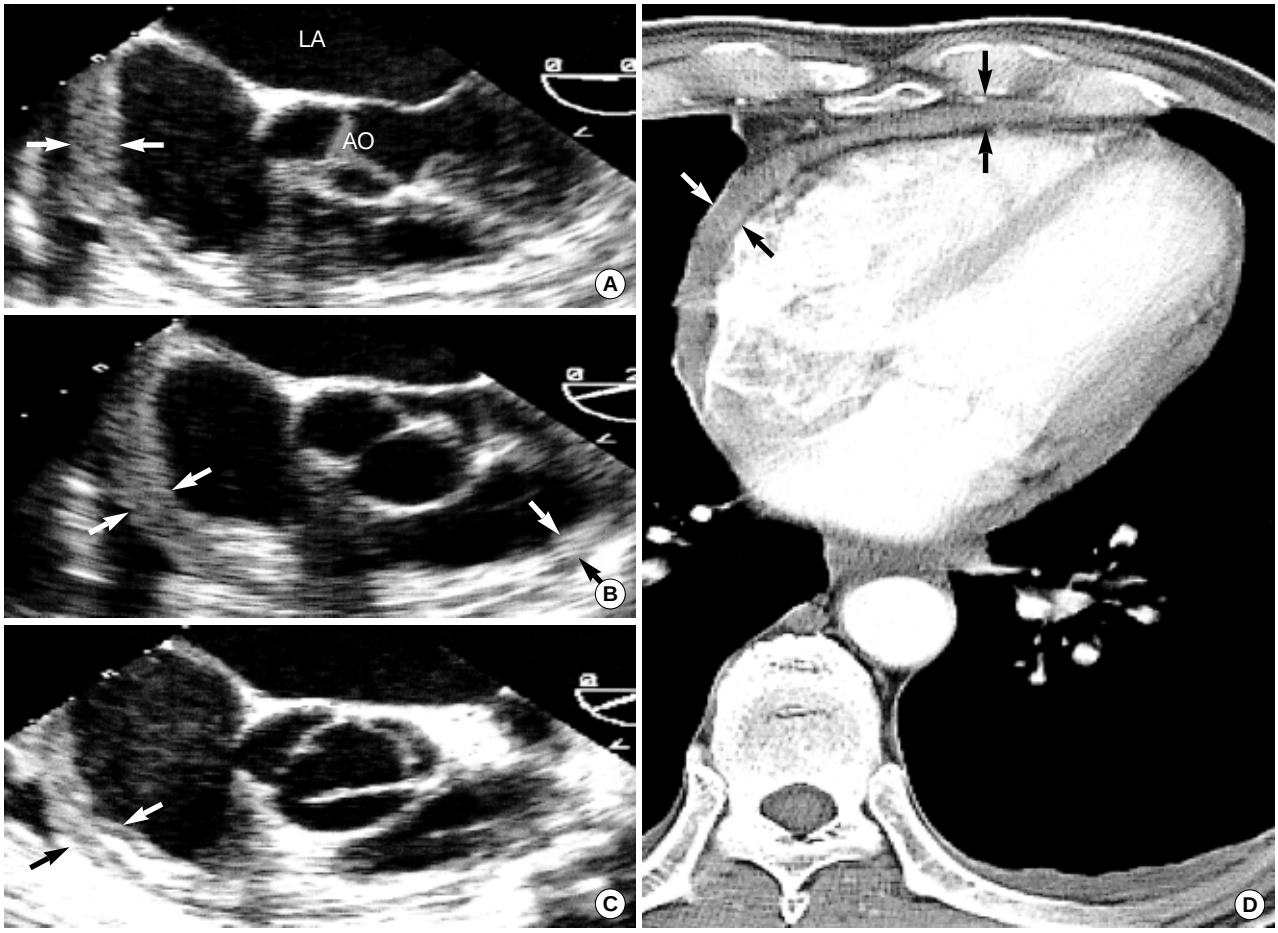

Fig. 1. Measurement of pericardial thickness with transesophageal echocardiography (A-C) and computerized tomography (D). Arrows represent total thickness of pericardium in a patient with constrictive pericarditis. Marked spontaneous echo contrast are seen in right atrium (C). LA, left atrium; AO, aorta.

2B) and inspiratory decrease (more than 25%) of transmitral early diastolic flow (Fig. 2C) with expiratory reversal of hepatic venous flow were also used as diagnostic criteria for CP. Thickened pericardium was confirmed again using other imaging modalities such as computerized tomography (55 patients) and magnetic resonance imaging (16 patients) (6, 7). Invasive cardiac catheterization was performed in 32 patients, and endomyocardial biopsy was done in 7 patients to rule out restrictive cardiomyopathy or infiltrative endomyocardial disorders.

Retrospective analysis of patients' data was done to characterize the etiology of CP, clinical presentation, hospital course with treatment, and clinical outcome. Medical records and telephone interview were used for follow-up data in all patients. Continuous variables were expressed as mean ± standard deviation, and Student t-test or Wilcoxon rank sum test was used to test the differences between groups. Chi-square or Fisher's exact test was used for categorical variables. Survival rates were calculated using Kaplan-Meier method and univariate Cox proportional hazard analysis was used to detect factors associated with survival. Multiple logistic regression model was used to define independent prognostic factors. *p*-value less than 0.05 was considered to be statistically significant.

RESULTS

Baseline Characteristics

Mean age of the patients was 49 ± 17 yr and male to female ratio was 6:4 (Table 1). Duration from symptom onset to diagnosis was 12.2 ± 0.8 months, and nine patients (13%) were treated under a clinical impression of liver cirrhosis. Past medical history of tuberculosis (Tbc) was present in 14 patients (20%), and 8 patients had medical history of previous cardiac operation. In six patients, significant amount of pericardial effusion was detected previously; among them four patients underwent percutaneous drainage and one patient underwent window operation. The other patient developed CP two months after diagnosis of pericardial effusion, for which no specific treatment was done.

Table 1. Baseline characteristics of patients (n=71)

Age, yr	49±17
Male/female	44/27
Past medical history	
Liver cirrhosis	9 (13%)
Tuberculosis	14 (20%)
Clinical presentation	
Dyspnea	55 (77%)
Peripheral edema	37 (52%)
Abdominal fullness	33 (46%)
Chest pain	12 (17%)
Cardiac tamponade	6 (8%)
Functional class	
I, II/III, IV	38 (54%)/33 (46%)
Physical findings	
Neck vein distension	59 (83%)
Hepatomegaly	36 (51%)
Ascites	34 (48%)
Electrocardiography	
Atrial fibrillation	20 (28%)
Low voltage	21 (30%)
Imaging study	
Pleural effusion	49 (69%)
Pericardial calcification	20 (28%)
Pericardial thickness	10.3±3.8 mm
Pericardial effusion	30 (42%)

Thirty-three patients (46%) were in New York Heart Association's functional class III or IV at the time of diagnosis. The presenting symptoms were dyspnea on exertion (77%, 55/71), peripheral edema (52%, 37/71), abdominal fullness with epigastric discomfort (46%, 33/71), and vague chest pain (17%, 12/71). In six patients (8%), initial clinical presentation was cardiac tamponade. Frequent findings in physical examination were neck vein distension (83%, 59/71), hepatomegaly (51%, 36/71), and ascites (48%, 34/71).

Pericardial calcification was detected in 20 patients (28%) and pleural effusion, mostly left-sided, was present in 49 patients (69%). The thickness of pericardium was 10.3± 3.8 mm. In electrocardiogram, atrial fibrillation was present in 20 patients (28%), and 21 patients (30%) showed low voltage. Initial echocardiogram revealed pericardial effusion in 30 patients (42%); 11 patients showed persistent constrictive physiology after percutaneous drainage (4 patients) or window operation (7 patients), which was compatible with effusive-constrictive pericarditis. The other 19 patients showed small and localized pericardial effusion.

Etiology

Pericardial specimens obtained by pericardiectomy, and pleural and pericardial effusions were used for pathologic, microbiologic, and serologic examinations to determine the etiology of CP. In 27 patients (38%), the etiology was unknown, and the three most frequent identifiable causes were tuberculosis (23/71, 32%), cardiac surgery (8/71, 11%), and mediastinal irradiation (6/71, 9%) (Table 2).

Table 2. Causes of constrictive pericarditis (n=71)

Idiopathic	27 (38%)
Tuberculosis	23 (32%)
Granuloma with caseous necrosis	9
Miliary tuberculosis	4
Sputum AFB (+)	1
Pleural ADA > 40 IU/L	2
Radiologic findings*	5
Response to therapeutic trial	2
Cardiac surgery	8 (11%)
Mediastinal irradiation	6 (9%)
Malignancy	2 (3%)
Uremia	2 (3%)
Viral infection	2 (3%)
Postmyocardial infarction	1 (1%)

AFB, acid fast bacilli.
*Calcified granuloma and lymphadenopathy in mediastinum and pulmonary artery or typical pleuropericarditis in chest computerized tomography

Tuberculosis

In 23 patients (32%), the final diagnosis was Tbc and in 9 patients thereof, it was confirmed by the surgical specimen. Chronic granulomatous lesion with typical caseation necrosis with positive acid-fast bacilli (AFB) staining or positive culture or PCR result in the pericardium was present in eight patients, and one patient showed typical caseation necrosis not in the pericardium but in the pleural specimen. Among the other 14 patients without pericardiectomy, miliary Tbc was present in 4 patients, and 1 patient revealed positive AFB staining in the sputum study. Pleural adenosine deaminase (ADA) level was higher than 40 IU/L in two patients, and characteristic radiological findings suggesting Tbc, such as typical tuberculoma, mediastinal lymphadenopathy and pleuropericarditis, were present in five patients. The other two patients showed dramatic clinical improvements after therapeutic trial of antituberculous drugs.

Among total 71 patients, 14 patients (20%) had past medical history of Tbc, but only in 6 patients Tbc was the underlying etiology of CP; thus, the past medical history of Tbc was not helpful for the determination of the underlying etiology. Four patients had chronic medical illnesses increasing the risk of tuberculous infection. Chronic administration of steroid was necessary in two patients with chronic interstitial lung disease and antiphospholipid syndrome each. Myeloproliferative disorder and hypogammaglobulinemia were the other underlying illnesses in two patients.

Cardiac surgery

In eight patients (11%), CP developed after successful cardiac surgery. The procedure were coronary bypass surgery

in two, aortic valve replacement in two, mitral valve replacement in one, mitral valve repair with maze procedure in one, and cardiac transplantation in two patients. In all patients, the clinical course was uneventful after the first cardiac surgery, and the mean time-intervals from operation to the onset of symptom and diagnosis of CP were 10.2±13.6 and 14.9±20.1 months (1-56 months), respectively.

Mediastinal irradiation

In six patients, development of CP was related with previous mediastinal irradiation, and most of them (5 patients) developed CP 142 months (44-182 months) after radiation treatment for breast cancer. In the other one patient, adenocarcinoma cell was confirmed in the pericardial fluid and cervical lymph node; palliative radiotherapy was performed and, eight months later, echocardiography done for evaluation of peripheral edema diagnosed CP.

Other etiologies

CP was caused by malignancy in two patients; both presented with cardiac tamponade, and malignant cells were confirmed after pericardiocentesis. CP developed in about two months. Viral infection was highly suspected in two patients by positive serologic test for Epstein-Barr virus and positive PCR for Enterovirus in the pleural fluid.

Treatment

Among total 71 patients, curative pericardiectomy was attempted in 35 patients (49%) average 4.4±10.9 months after diagnosis of CP. Postoperative mortality developed in two patients, whose cause of death was ventricular arrhythmia and postoperative multi-organ failure, respectively. Other two patients showed persistent constrictive physiology after incomplete pericardiectomy and died of congestive heart failure four and seven months after operation, respectively.

Pericardiectomy was not performed in the other 36 patients (51%); two patients died suddenly while waiting for pericardiectomy, and operation was contraindicated in six patients due to poor performance status at the time of diagnosis. With therapeutic trial of antituberculous medication, 11 patients showed dramatic clinical improvements with disappearance of constrictive physiology in follow-up echocardiographic examination, and the scheduled pericardiectomy was canceled. Other six patients refused pericardiectomy. Among the remaining 11 patients who showed persistent constrictive physiology in follow-up echocardiographic examination, low dose diuretics were tolerable in six patients and technical problems increasing the chance of incomplete pericardiectomy, such as relatively thin pericardium in imaging studies and localized pericardial thickening, made surgeons refuse operation in 5 patients. Table 3 compares the clinical features between patients with pericardiectomy and those with medical treatment only. Patients with pericardiectomy showed a lower prevalence of radiological findings suggestive of tuberculous lesions (9% vs 47%), and higher prevalence of pericardial calcification (40% vs 17%), hepatomegaly (66% vs 36%), and ascites (60% vs 36%), compared with those with medical treatment only.

Table 3. Comparison of clinical features between patients with and without pericardiectomy

	With pericardiectomy (n=35)	Without pericardiectomy (n=36)
Etiology		
Idiopathic/Tbc*	19 (54%)/9 (26%)	8 (22%)/14 (39%)
Age, yr	47.5±15.9	50.9±18.0
Symptoms		
Dyspnea	26 (74%)	29 (81%)
Epigastric discomfort*	21 (60%)	12 (33%)
Functional class III or IV	16 (46%)	17 (47%)
Physical findings		
Neck vein distension*	34 (97%)	25 (69%)
Hepatomegaly*	23 (66%)	13 (36%)
Ascites*	21 (60%)	13 (36%)
Radiologic findings		
Tuberculosis lesion*	3 (9%)	17 (47%)
Pericardial calcification*	14 (40%)	6 (17%)
Pericardial thickness (mm)	10.2±3.2	10.9±4.5
Symptom duration (months)	17.0±25.9	7.6±13.0

*p<0.05

Table 4. Comparisons of clinical features between patients with transient constrictive pericarditis and those with chronic persistent constrictive pericarditis

	Transient CP (n=11)	Chronic Persistent CP (n=60)
Age (yr)	41.7±19.2	50.6±16.3
Etiology		
Tuberculosis*	10 (91%)	13 (22%)
Symptom duration*	1.8±1.1 Mo	14.1±22.2 Mo
Symptoms		
Dyspnea*	5 (46%)	50 (83%)
Peripheral edema	4 (36%)	33 (55%)
Fever*	5 (46%)	2 (3%)
Weight loss*	4 (36%)	1 (2%)
Functional class III or IV*	1 (9%)	32 (53%)
Physical findings		
Neck vein distension*	5 (45%)	54 (90%)
Hepatomegaly	6 (55%)	30 (50%)
Ascites*	2 (18%)	32 (53%)
Sinus tachycardia*	5 (46%)	8 (13%)
Radiologic findings		
Tuberculosis lesion*	9 (82%)	11 (18%)
Pericardial calcification*	0 (0%)	20 (33%)
Pericardial thickness* (mm)	13.4±4.4	10.0±3.6

* p<0.05, CP, constrictive pericarditis

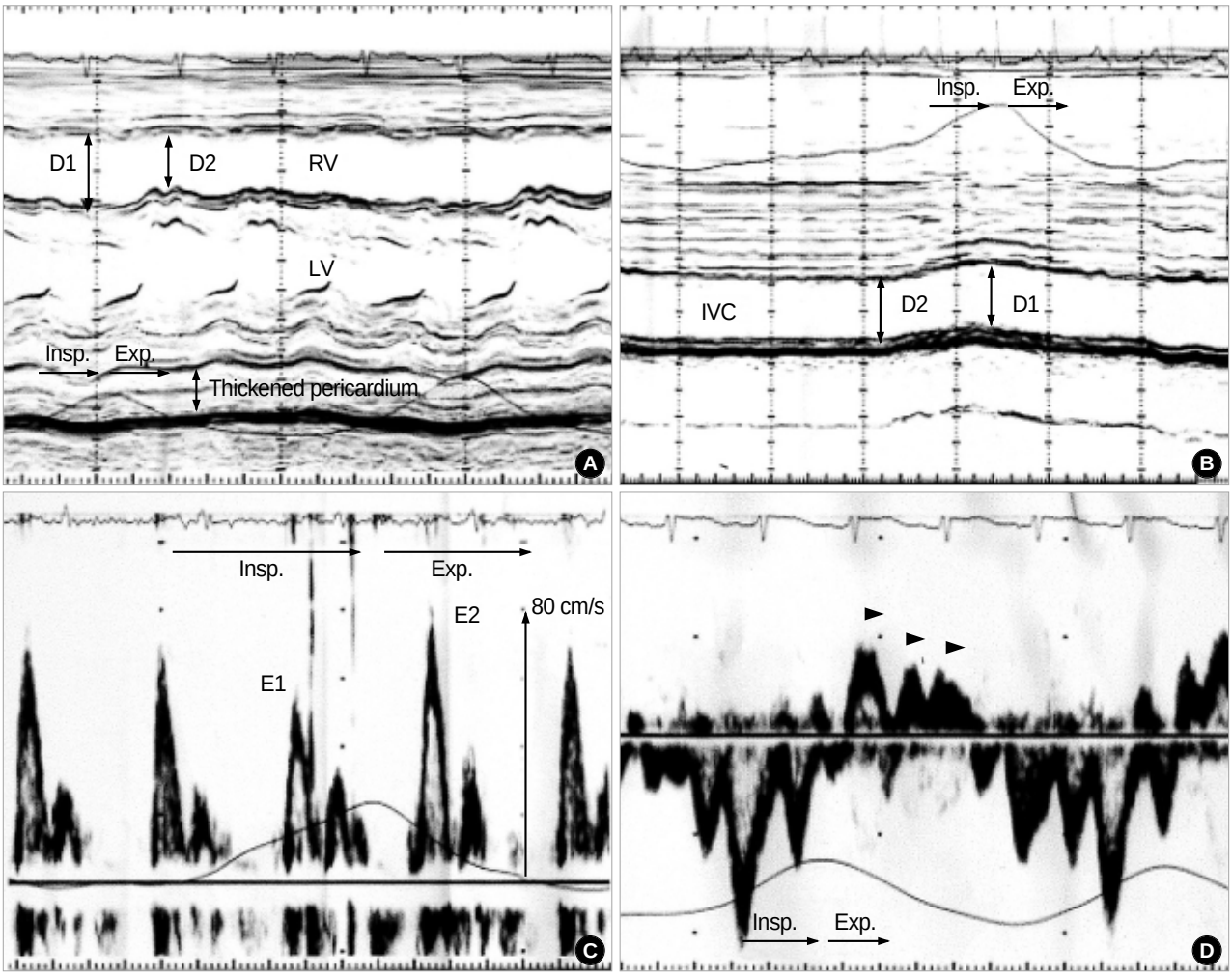

Fig. 2. Representative examples of echo-Doppler findings in constrictive pericarditis. In M-mode tracing (A), thickened pericardium and inspiratory increase of right ventricular dimension are easily confirmed. The inferior vena cava (IVC) does not show normal inspiratory collapse (B). Transmitral flow velocity recording (C) shows characteristic inspiratory decrease of early diastolic inflow velocity (E1) and expiratory reversal of hepatic venous flow is also characteristic (D). Insp., inspiration; Exp., expiration.

Among 36 patients without curative pericardiectomy, 11 patients (31%) showed complete normalization of constrictive physiology with dramatic disappearance of dyspnea and peripheral edema (Fig. 3). Clinical improvement was observed average 2.1±1.0 months after medical treatment, and all received antituberculous medication. The final etiology was Tbc in 10 patients, and the other 1 patient was proved to have transient CP due to Epstein-Barr virus infection. Among 10 patients with transient CP due to Tbc, 3 patients showed disseminated Tbc in bone marrow, peritoneal cavity, and cervical lymph nodes. Table 4 compares clinical features between patients with transient CP and those with persistent CP. Patients with transient CP showed a shorter time interval from the onset of symptom to diagnosis (1.8±1.1 months vs 14.1±22.2 months) and higher prevalences of Tbc (91% vs 22%), fever (46% vs 3%), weight loss (36% vs 2%), and sinus tachycardia (46% vs 13%) compared with those with persistent CP. However, the prevalences of ascites (18% vs 53%), dyspnea of functional class III or IV (9% vs 53%), and pericardial calcification (0% vs 33%) were significantly lower in patients with transient CP than in those with persistent CP.

Follow-Up

Clinical follow-up was possible in all patients. During the mean follow-up period of 37.7±32 months, 16 patients (23%) died, and the 5-yr survival rate was 73±7%. In patients with pericardiectomy, total six patients died; in two patients, the mortality was associated with surgery and other two died of congestive heart failure due to incomplete pericardiectomy. The other two patients underwent the second cardiac surgery to correct mitral and tricuspid regurgitation, and died due to postoperative complications. In patients

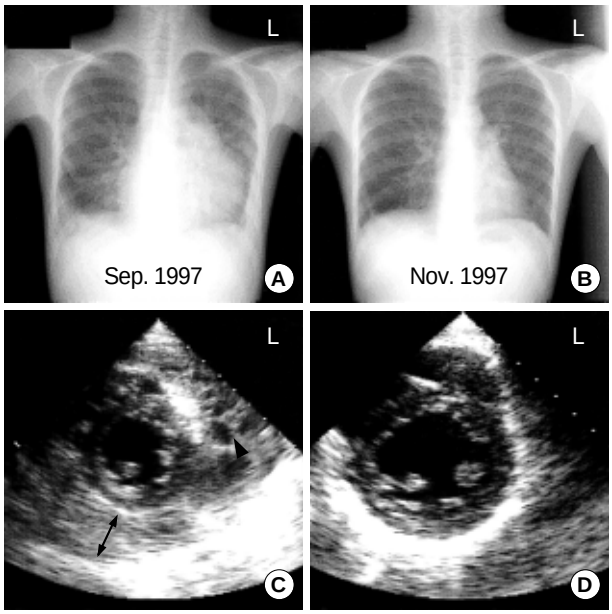

Fig. 3. One representative example of transient constrictive pericarditis. In initial chest radiography (A) and echocardiography (C) of a 17-yr-old boy with congenital hypogammaglobulinemia, thickened pericardium with increased cardiac shadow and both pleural effusions can be observed. Active pulmonary tuberculosis is also highly suspicious, and therapeutic trial of anti-tuberculosis drugs was attempted, which dramatically improved the patient's condition with normalization of cardiac shadow (B) and pericardial thickening (D) within two months.

without pericardiectomy, total 10 patients died; 2 patients died suddenly while waiting for pericardiectomy, and the other 8 died from congestive heart failure. The 5-yr survival rates of patients with transient CP (n=11) and pericardiectomy (n=35) were 100%, and 85±6%, respectively, which were significantly higher than that of patients (n=25) without pericardiectomy (33±17%, p=0.0083, Fig. 4). In patients with CP, independent factors associated with survival were functional class at the time of diagnosis, low voltage in ECG, mediastinal irradiation, serum albumin, and age (Fig. 4, Table 5). In patients with pericardiectomy, the completeness of surgical procedure was the most important factor determining the survival.

DISCUSSION

In this clinical study, we have confirmed that Tbc is an important etiology of CP in Korea comprising about 1/3 of total cases and, although pericardiectomy is an effective treatment modality, the prognosis of CP is still poor with a mortality rate higher than 20%. Besides mediastinal irradiation for malignancy, clinical factors associated with the chronicity of the disease, such as higher functional class at the time of diagnosis, low voltage in ECG, and low serum albumin,

Table 5. Multivariate clinical predictors of survival in patients with constrictive pericarditis

	Adjusted hazard ratio	95% Confidence Interval	p-value
Total patients (n=71)			
Functional class	26.26	2.80-245.98	0.004
Low voltage in ECG	5.46	1.56-19.09	0.008
Mediastinal irradiation	7.14	1.53-33.35	0.012
Serum albumin	0.26	0.08-0.89	0.031
Age	1.04	1.00-1.09	0.049
Patients with pericardiectomy (n=35)			
Successful pericardiectomy	69.00	7.30-652.26	0.0002

were independent predictors of poor prognosis, which might represent the importance of early diagnosis and intervention of CP. The finding that 15% of total patients with CP can completely recover without complications within two months of medical treatment demonstrates the clinical usefulness of echo-Doppler technique in early detection and follow-up of this disease entity.

Since the first description in 1669 by Lower et al. (8), CP has been well known among physicians. The recent global trend in the etiology of CP is that the prevalence of Tbc is decreasing significantly, and clinical importance of cardiac surgery and mediastinal irradiation is increasing dramatically due to the increase of old population and high incidences of coronary artery disease, chronic renal disease, and thoracic tumor (9, 10). In Korea, however, Tbc is still the most frequent identifiable etiology of CP. Tuberculous CP is known to be from the reactivation of previous infection, and, due to the high prevalence of Tbc in Korea, a past history of Tbc is not enough for diagnosis of tuberculous CP. However, among 23 patients with tuberculous CP in this study, past history of Tbc was suspected in 13 patients; among them, only 3 patients received complete antituberculous medication for their lesions in the past. In the past 10 yr, 8 patients developed CP after cardiac surgery, especially in the latter half, and we believe that CP due to previous cardiac surgery will increase progressively.

Pericardial calcification has been described as a very characteristic finding of CP. However, the prevalence of calcification was only 28% in this study. Pericardial thickness did not show any difference between patients with pericardial calcification and those without (9.7±2.5 mm vs 10.9 ±4.3 mm, p=0.253). Patients with pericardial calcification showed a larger left atrial diameter (52.2±7.5 mm vs 43.2 ±5.8 mm, p<0.00) and a longer time-interval from the onset of symptom to diagnosis (23.2±21.7 months vs 7.9 ±19.0 months, p=0.009), which suggests that pericardial calcification is an index of chronicity of the disease (11). Among nine patients who were treated under the impression of liver cirrhosis, six patients showed characteristic pericardial calcification in routine chest radiography; thus,

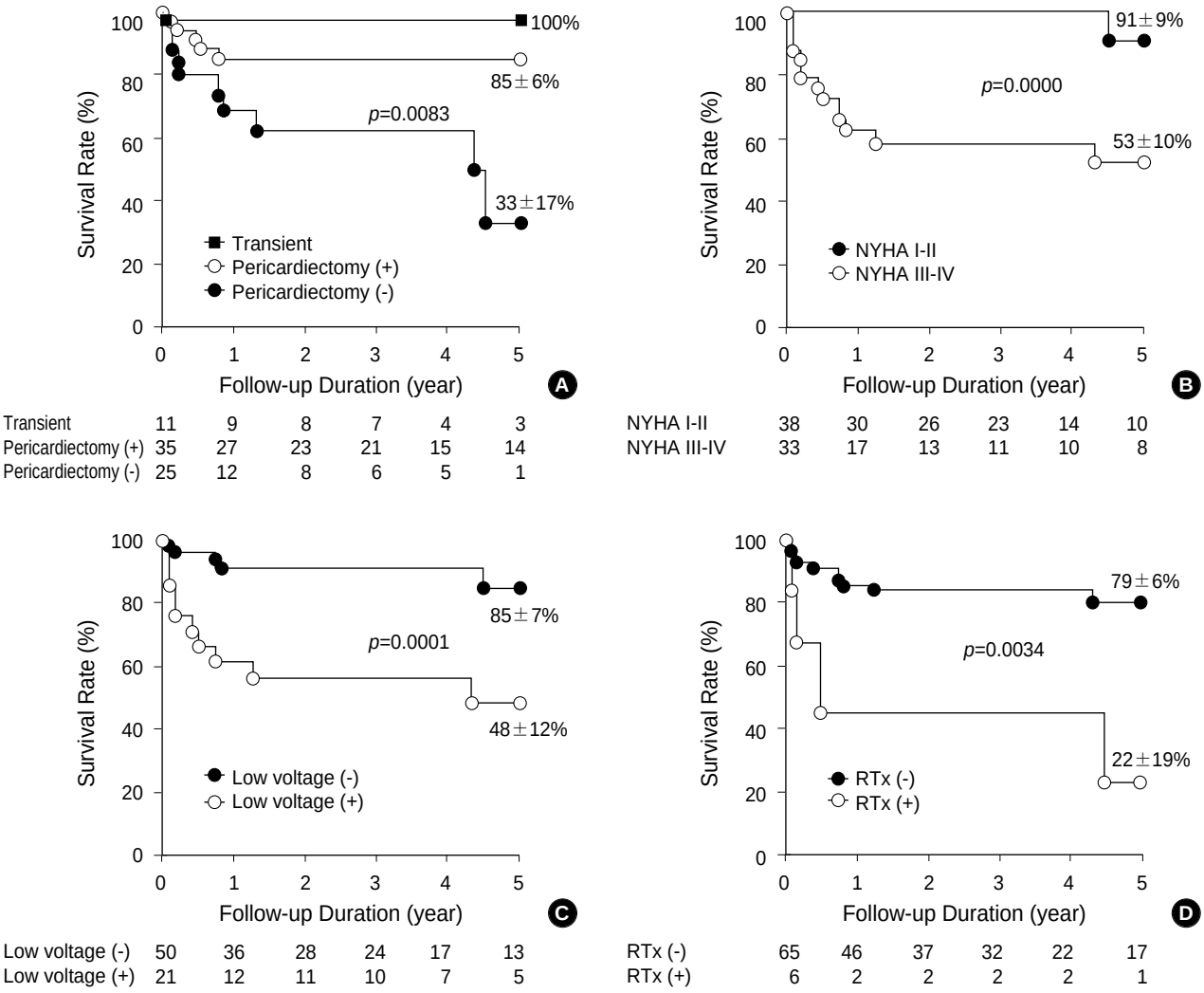

Fig. 4. Survival curves of patients with constrictive pericarditis. NYHA, New York Heart Association functional class; RTx, radiation treatment.

more careful evaluation for possibility of CP would be necessary in patients with chronic liver or renal disease (12, 13), recurrent ascites (14), or pleural effusion (15).

Although it is generally accepted that pericardiectomy is the only way to break the vicious cycle of progressive deterioration of cardiac relaxation and filling in CP (16), complete disappearance of constrictive physiology with medical supportive care in some patients with CP has been reported in the literature (17-20). Transient inflammation or fibrosis of the pericardium associated with viral or bacterial infection or immunologic mechanism after acute effusive pericarditis has been proposed as a mechanism of this transient CP (21). In our study, 15% of total patients (11/71) showed a typical clinical course of transient CP. One interesting finding is that, in our series, Tbc was the etiology in almost all the patients with transient CP (91%, 10/11). Nine patients showed characteristic radiological findings suggestive of tuberculous lesions in chest radiography or computerized tomography without pericardial calcification, and all responded dramatically to therapeutic trial of antituberculous medications with disappearance of constrictive physiology within two months (Fig. 3).

As echocardiography is now routinely used as a screening test for patients complaining of dyspnea and peripheral edema, it may contribute to the diagnosis of CP at relatively early stage and more frequent observation of transient CP cases is anticipated. The timing of surgical intervention has become a real clinical issue; Senni et al. recommended early pericardiectomy to prevent persistent diastolic abnormality after surgery that has been reported to have positive correlation with the symptom duration (22). The occurrence of sudden cardiac death in our series of patients with CP while waiting for pericardiectomy reinforces the importance of timely surgical intervention. Fortunately, however, patients with transient CP have characteristic clinical features related with subacute inflammatory process. Compared to those

with chronic persistent CP, they have a shorter symptom duration (1.8±1.1 months vs 14.1±22.2 months), a lower prevalence of ascites or severe dyspnea, and higher prevalences of fever and weight loss (Table 4). Thus, if a patient with constrictive physiology has these clinical features of relatively acute inflammatory process without pericardial calcification, therapeutic trial with antituberculous medications for one or two months with echocardiographic follow-up could be a rational treatment option in Korea. If chest radiography or computed tomography shows tuberculous lesions in other areas, it would be a perfect condition. Clinical decision-making for appropriate timing of surgery based on tissue characterization of pericardium by computerized tomography or magnetic resonance imaging might be one ideal way, which needs a systematic investigation.

In patients with chronic persistent CP, pericardiectomy is a gold standard treatment option. Except patients with transient CP, the mortality of patients who did not undergo pericardiectomy during follow-up was 40% (10/25), which was significantly higher than 17% of those with pericardiectomy. Among 17 patients including 12 patients who refused surgery due to mild symptoms and 5 patients in whom successful pericardiectomy could not be anticipated due to various technical reasons, 6 patients died during follow-up. Thus, if a possibility of transient CP is ruled out, prompt trial of pericardiectomy is strongly recommended to prevent progressive irreversible fibrosis into the myocardium (23-25).

Pericardiectomy is a challenging surgical procedure demanding operator's high level of skill, and besides complications of general cardiac surgery, unique complications such as acute heart failure with cardiac tamponade (26) and mitral regurgitation (27) have been described. In this study, the overall operative mortality was 6% (2/35), and two patients died four and seven months after surgery due to incomplete pericardiectomy. Four patients underwent the second cardiac surgery; among them, three patients underwent surgery to repair mitral and/or tricuspid regurgitation, and repeated pericardiectomy was attempted in one patient. In 35 patients who underwent pericardiectomy, the most important factor determining the prognosis was successful pericardiectomy. Specifically, the prognosis of CP due to mediastinal irradiation even after pericardiectomy is very poor, as pericardiectomy is often incomplete, and the second operation is frequently needed due to already damaged myocardium and cardiac valves. Thus, some investigators have recommended cardiac transplantation rather than pericardiectomy in these selected groups of patients (9, 28-30). Considering the fact that radiation-induced CP develops at least several or 10 yr after the treatment, the incidence will increase with recent improvement of long-term survivals of patients with breast cancer or malignant lymphoma. Aggressive approaches to prevent radiation injury to the heart are actually the only practical way to overcome the difficulties of this disease. The observation that clinical variables representing the chronicity of the disease, such as marked dyspnea at the time of diagnosis, low voltage in ECG, and low serum albumin, are independent adverse prognostic factors reemphasizes the clinical importance of early detection of CP.

REFERENCES

1. Oh JK, Hatle LK, Seward JB, Danielson GK, Schaff HV, Reeder GS, Tajik AJ. *Diagnostic role of Doppler echocardiography in constrictive pericarditis. J Am Coll Cardiol 1994; 23: 154-62.*
2. Palka P, Lange A, Donnelly JE, Nihoyannopoulos P. *Differentiation between restrictive cardiomyopathy and constrictive pericarditis by early diastolic Doppler myocardial velocity gradient at the posterior wall. Circulation 2000; 102: 655-62.*
3. Hatle LK, Appleton CP, Popp RL. *Differentiation of constrictive pericarditis and restrictive cardiomyopathy by Doppler echocardiography. Circulation 1989; 79: 357-70.*
4. Kim SG, Hyun IY, Song JK, Kang YK, Lee JO, Kang TW, *et al. A case of radiation induced pericarditis and myocardial fibrosis. Korean J Int Med 1989; 37: 686-93.*
5. Cha KS, Bae Y, Ahn YK, Park JC, Seo JP, Park JH, *et al. Postoperative effusive constrictive pericarditis in ventricular septal defect repair. Korean J Echocardiogr 1997; 5: 36-41.*
6. Frank H, Globits S. *Invited magnetic resonance imaging evaluation of myocardial and pericardial disease. J Magn Reson Imaging 1999; 10: 617-26.*
7. Hayashi H, Kawamata H, Machida M, Kumazaki T. *Tuberculous pericarditis: MRI features with contrast enhancement. Br J Radiol 1998; 71: 680-2.*
8. Lower R. *Tractatus de corde. Londom: J Allestry 1669. Cited in The Pericardium. Philadelphia: W.B. Saunders 1981.*
9. Ling LH, Oh JK, Shaff JV, Danielson GK, Mahoney DW, Seward JB, Tajik AJ. *Calcific constrictive pericarditis in the modern era: evolving clinical spectrum and impact on outcome after pericardiectomy. Circulation 1999; 100: 1380-6.*
10. Schiavone WA. *The changing etiology of constrictive pericarditis in a large referral center. Am J Cardiol 1986; 58: 373-5.*
11. Ling LH, Oh JK, Breen JF, Schaff HV, Danielson GK, Mahoney DW, Seward JB, Tajik AJ. *Calcific constrictive pericarditis: is it still with us? Ann Intern Med 2000; 132: 444-50.*
12. Lowe MD, Harcombe AA, Grace AA, Petch MC. *Restrictive-constrictive heart failure masquerading as liver disease. Br Med J 1999; 318: 585-6.*
13. Shua-Haim JR, Ross JS. *Chronic constrictive pericarditis: condition of the heart presents with symptoms similar to cirrhosis. Geriatrics 1999; 54: 74.*
14. Van der Merwe S, Dens J, Daenen W, Desmet V, Fevery J. *Pericardial disease is often not recognized as a cause of chronic severe ascites. J Hepatol 2000; 32: 164-9.*
15. Sadikor RT, Fredi JL, Light RW. *A 43-yr-old man with a large recurrent right sided pleural effusion. Chest 2000; 117: 1191-4.*
16. Myers PB, Spodick DH. *Constrictive pericarditis: clinical and pathologic characteristics. Am Heart J 1999; 138: 219-32.*

17. Benaing III G, Kaplan S. *Purulent pericarditis. Am J Dis Child 1963; 106: 289-94.*
18. Sagrista-Sauleda J, Permanyer-Miralda G, Candell-Riera J, Angel J, Soler-Soler J. *Transient cardiac constriction: an unrecognized pattern of evolution in effusive acute idiopathic pericarditis. Am J Cardiol 1987; 59: 961-6.*
19. Allaria A, Michelli D, Capelli H, Berri G, Gutierrez D. *Transient cardiac constriction following purulent pericarditis. Eur J Pediatr 1992; 151: 250-1.*
20. Woods T, Vidarsson B, Mosher D, Stein JH. *Transient effusive-constrictive pericarditis due to chemotherapy. Clin Cardiol 1999; 22: 316-8.*
21. Oh JK, Hatle LK, Mulvagh SL, Tajik AJ. *Transient constrictive pericarditis: diagnosis by two-dimensional Doppler echocardiography. Mayo Clin Proc 1993; 68: 1158-64.*
22. Senni M, Redifield MM, Ling LH, Danielson GK, Tajik AJ, Oh JK. *Left ventricular systolic and diastolic function after pericardiectomy in patients with constrictive pericarditis. J Am Coll Cardiol 1999; 33: 1182-8.*
23. McCaughan BC, Schaff HV, Piehler JM, Danielson GK, Orszulak TA, Puga FJ, Pluth JR, Connolly DC, Mc Goon DC. *Early and late results of pericardiectomy for constrictive pericarditis. J Thorac Cardiovasc Surg 1985; 89: 340-50.*
24. Tirilomis T, Unverdorben S, van der Emde J. *Pericardiectomy for chronic constrictive pericarditis: risks and outcome. Eur J Cardiothorac Surg 1994; 8: 487-92.*
25. Ha HC, Park SD, Kim SH, Huh GB, Lee JS, Cho SR, Kim SM. *Pericardiectomy in constrictive pericarditis. Korean J Thorac Cardiovas Surg 1995; 28: 243-9.*
26. Sunday R, Robinson LA, Bosek V. *Low cardiac output complicating pericardiectomy for pericardial tamponade. Ann Thorac Surg 1999; 67: 228-31.*
27. Terada Y, Mitsui T, Yamada S. *Mitral regurgitation after pericardiectomy for constrictive pericarditis. Jpn J Thorac Cardiovas Surg 1999; 47: 27-30.*
28. Veinot JP, Edwards WD. *Pathology of radiation-induced heart disease: a surgical and autopsy study of 27 cases. Human Pathol 1996; 27: 766-73.*
29. Chello M, Mastroroberto P, Romano R, Zofrea S, Bevacqua I, Marchese AR. *Changes in the proportion of types I and III collagen in the left ventricular wall of patients with post-irradiative pericarditis. Cardiovasc Surg 1996; 4: 222-6.*
30. Ni Y, von Segesser LK, Turina M. *Futility of pericardiectomy for postirradiation constrictive pericarditis. Ann Thorac Surg 1990; 49: 445-8.*