Pericardial Disease

Risk of Constrictive Pericarditis After Acute Pericarditis

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Background—Constrictive pericarditis (CP) is considered a rare, dreaded possible complication of acute pericarditis. Nevertheless, there is a lack of prospective studies that have evaluated the specific risk according to different etiologies. The aim of this study is to evaluate the risk of CP after acute pericarditis in a prospective cohort study with long-term follow-up.

Methods and Results—From January 2000 to December 2008, 500 consecutive cases with a first episode of acute pericarditis (age, 51±16 years; 270 men) were prospectively studied to evaluate the evolution toward CP. Etiologies were viral/idiopathic in 416 cases (83.2%), connective tissue disease/pericardial injury syndromes in 36 cases (7.2%), neoplastic pericarditis in 25 cases (5.0%), tuberculosis in 20 cases (4.0%), and purulent in 3 cases (0.6%). During a median follow-up of 72 months (range, 24 to 120 months), CP developed in 9 of 500 patients (1.8%): 2 of 416 patients with idiopathic/viral pericarditis (0.48%) versus 7 of 84 patients with a nonviral/nonidiopathic etiology (8.3%). The incidence rate of CP was 0.76 cases per 1000 person-years for idiopathic/viral pericarditis, 4.40 cases per 1000 person-years for connective tissue disease/pericardial injury syndrome, 6.33 cases per 1000 person-years for neoplastic pericarditis, 31.65 cases for 1000 person-years for tuberculous pericarditis, and 52.74 cases per 1000 person-years for purulent pericarditis.

Conclusions—CP is a relatively rare complication of viral or idiopathic acute pericarditis (<0.5%) but, in contrast, is relatively frequent for specific etiologies, especially bacterial. (***Circulation.*** 2011;124:1270-1275.)

Key Words: constrictive pericarditis ■ pericarditis ■ prognosis

Constrictive pericarditis is a rare but dreaded possible complication of acute pericarditis.[1–4] This risk is evident in particular after tuberculous pericarditis,[4] whereas it is not well established after idiopathic and viral acute pericarditis, the most common causes of acute pericarditis in developed countries.[5–7] Unfortunately, there is a lack of prospective cohort studies, and most data come from retrospective surgery series of patients submitted to pericardiectomy for permanent chronic constriction.[8–10]

The aim of the present study is to prospectively evaluate the incidence of constrictive pericarditis after acute pericarditis in a cohort study with a long-term follow-up. To the best of our knowledge, this is the first prospective study in contemporary patients with acute pericarditis.

Methods

Patients

From January 2000 to December 2008, all consecutive cases with a first episode of acute pericarditis were recorded and prospectively studied for outcomes. Baseline features were recorded including age, gender, presentation of specific features (fever >38°C, subacute course), physical (pericardial rubs) and instrumental findings (ECG changes, pericardial effusion, cardiac tamponade), response to empirical anti-inflammatory drugs at 1 week, and use of corticosteroids. Response to anti-inflammatory therapy was considered incomplete in the case of persistence of symptoms with evidence of activity disease (fever without alternative causes, pericardial rubs, new or worsening ECG changes, elevated markers of inflammation, and appearance or worsening of pericardial effusion).

Such features were sought specifically because they have been reported as possible poor prognostic predictors in patients with acute pericarditis.[11]

Reported diagnostic criteria for acute pericarditis include pericarditic typical chest pain, pericardial friction rubs, widespread ST-segment elevation or PR depressions not previously reported, and new or worsening pericardial effusion.[2,5,6,11–18] A clinical diagnosis of acute pericarditis was made when at least 2 of these criteria were present.[11–18] The presence of elevated markers of inflammation (C-reactive protein and/or erythrocyte sedimentation rate) was considered confirmatory.[18]

A minimal ST-segment elevation of 1 mm was considered significant, although no specific recommendations are available on this issue. The presence of an atrial current of injury, reflected by

elevation of the PR segment in lead aVR and depression of the PR segment in other limb leads and in the left chest leads, primarily V_5 and V_6, was considered a diagnostic ECG change.

A final diagnosis of idiopathic or viral acute pericarditis was reached at the end of the diagnostic assessment, which included chest-x ray, echocardiography, viral serology, and other specific testing according to the initial clinical presentation. Pericardiocentesis was done when a bacterial or neoplastic etiology was suspected or in case of cardiac tamponade or severe pericardial effusion without response to medical therapy after 1 week.

Pericardial injury syndromes included pericarditis resulting from recent or earlier injury of the pericardium (late post–myocardial infarction pericarditis, postpericardiotomy syndrome, and posttraumatic pericarditis). The initial injury is thought to release cardiac antigens and stimulate an immune response, eliciting an inflammatory response, which may involve the pericardium, pleura, or both. On the basis of the presumptive common autoimmune pathogenesis, pericardial injury syndromes were grouped with connective tissue diseases.[5,6,11]

Follow-Up

A structured follow-up was implemented including serial clinical visits, ECG, blood chemistry (including C-reactive protein and blood count), and echocardiograms at least at 1 month, 3 months, and then every 6 months from the initial episode of acute pericarditis. Additional investigations were organized according to the patient's symptoms and clinical evolution. Case report forms were completed for each patient (see the online-only Data Supplement). All patients underwent a basal echocardiographic examination at the time of the initial clinical diagnosis and regularly at each follow-up visit. The clinical course was recorded (stable remission, incessant course, recurrences, cardiac tamponade, persistence of pericardial effusion).

An incessant course was defined as a course with persistence of symptoms and clinical and instrumental signs of disease activity without a free interval, whereas recurrence was defined as a situation in which a new attack occurs after a period of complete disappearance of symptoms and normalization of markers of inflammation if previously elevated.[19]

The following clinical events were considered "adverse events" during follow-up: recurrent pericardial pain without objective evidence of disease, recurrent pericarditis, cardiac tamponade, and constrictive pericarditis. Blinded assessment and validation of adverse events were done by an independent committee with 2 experts on pericardial diseases (see Appendix in the online-only Data Supplement). Recurrent pericardial pain without objective evidence of disease[20] was recorded when recurrent chest pain recognized by the patient as similar to the previous pericarditic attack was reported in the absence of other objective evidence of disease activity (fever, pericardial friction rub, ECG changes, presence/worsening of pericardial effusion, and elevated markers of inflammation). Criteria for the diagnosis of recurrence included recurrent pain and 1 or more of the following signs: fever, pericardial friction rub, ECG changes, echocardiographic evidence of pericardial effusion, and elevation in the white blood cell count, erythrocyte sedimentation rate, or C-reactive protein.[14–17] The diagnosis of cardiac tamponade and constrictive pericarditis was made with consideration of the combination of clinical and echocardiographic instrumental data according to current available guidelines.[21,22] The presence of effusive-constrictive pericarditis was suspected in case of pericardial effusion with constrictive features on echocardiography and/or cardiac magnetic resonance imaging. The diagnosis of effusive-constrictive pericarditis was confirmed when pericardiocentesis failed to decrease the right atrial pressure by 50% or to a level <10 mm Hg after exclusion of other causes of persistently elevated right atrial pressure after pericardiocentesis (ie, right heart failure or tricuspid regurgitation).[23,24] For constrictive pericarditis, clinical suspicion based on initial physical and echocardiographic evaluation was confirmed by additional imaging studies (computed tomography, cardiac magnetic resonance), cardiac catheterization, and, finally, macroscopic and histology findings from surgical specimens.[25] All patients had a surgically confirmed diagnosis of constrictive pericarditis.

Table 1.   Comparison of Baseline Characteristics of the Studied Population Between Viral/Idiopathic and Nonidiopathic Causes

Feature	Idiopathic or Viral* (n=416)	Specific Etiology† (n=84)	P
Mean age, y (51±16)	50±17	53±18	0.145
Male gender (n=270)	233 (56.0)	37 (44.0)	0.058
Fever >38°C (n=77)	40 (9.6)	37 (44.0)	<0.001
Subacute course (n=22)	8 (1.9)	14 (16.7)	<0.001
Pericarditic chest pain (n=490)	410 (98.6)	80 (95.2)	0.106
Pericardial rubs (n=175)	146 (35.1)	29 (34.5)	0.984
ST-segment elevation (n=449)	375 (90.1)	74 (88.1)	0.723
Pericardial effusion (n=327)	250 (60.1)	77 (91.7)	<0.001
Large pericardial effusion (n=48)	21 (5.1)	27 (32.1)	<0.001
Cardiac tamponade (n=22)	5 (1.2)	17 (20.2)	<0.001
Aspirin or NSAID failure at 1 wk (n=98)	46 (11.1)	52 (61.9)	<0.001
Corticosteroids as initial therapy (n=72)	42 (10.0)	30 (35.7)	<0.001

Data are expressed as mean±SD for continuous variables and numbers and percentages (in parentheses) for categorical variables. Comparisons between patient groups (idiopathic/viral etiologies vs specific etiologies) were performed with the Mann-Whitney test for continuous variables and a χ^2 analysis for categorical variables. NSAID indicates nonsteroidal anti-inflammatory drugs. Overall, C-reactive protein elevation was recorded in 451 of 500 patients (90.2%) on serial determinations without significant differences between patients with or without a specific etiology.

*A positive viral serology was recorded in 166 cases (39.9%).

†Specific etiologies included autoimmune causes (pericardial injury syndromes and connective tissue diseases) in 36 patients (7.2%), neoplastic cause in 25 patients (5.0%), tuberculosis in 20 patients (4.0%), and purulent bacterial infection in 3 patients (0.6%).

Statistical Analysis

Data were expressed as mean±SD for continuous variables and counts with percentages for categorical variables. Comparisons between patient groups (idiopathic/viral etiologies versus specific etiologies) were performed with the Mann-Whitney test for continuous variables and a χ^2 analysis or Fisher exact test (when the number of observations obtained for analysis was relatively small) for categorical variables. Time-to-event distributions were estimated by the Kaplan-Meier method and compared with the log-rank test. A P value of <0.05 was considered to show statistical significance. Analyses were performed with the software package SPSS 13.0 (Chicago, IL).
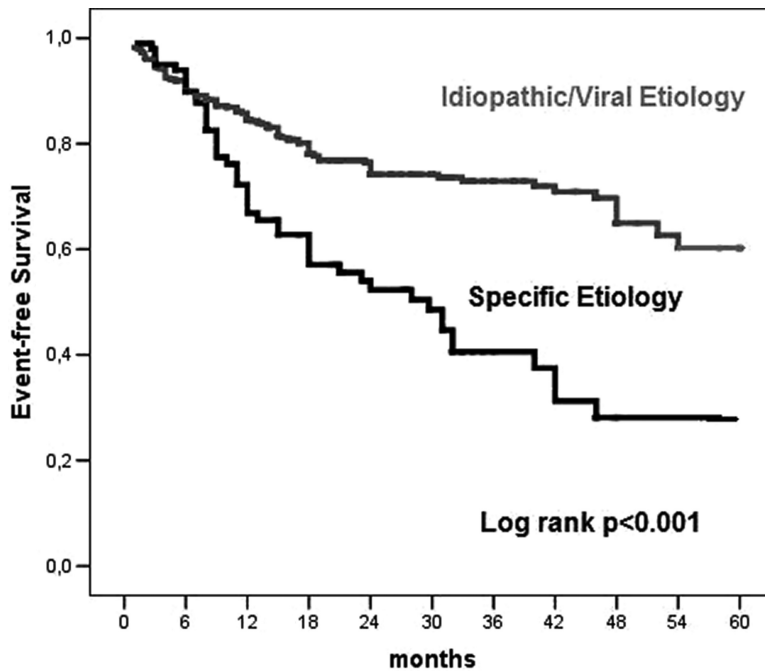
Incidence rates were computed as the number of new cases of disease during a period of time divided by the person time at risk. Person time was the estimate of the actual time at risk in years that all persons contributed to the study. Confidence intervals for incidence statistics were calculated with the use of MedCalc software, version 11.5.1 (Mariakerke, Belgium).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Acute Pericarditis Study Population

In the study period, 500 cases of acute pericarditis (mean age, 51±16 years; 270 men) were recorded. Among them, 416 patients (83.2%; mean age, 50±17 years; 233 men) had a diagnosis of viral or idiopathic acute pericarditis, and 84 patients (16.8%; mean age, 53±18 years; 37 men) had a diagnosis of nonviral, nonidiopathic acute pericarditis.

Figure. Event-free survival in patients with (black line) or without (gray line) a specific (nonidiopathic/ nonviral) etiology of acute pericarditis. Differences of event-free survivals are evident after 6 months and increase in the first 48 months. Medians for survival time in patients with viral/idiopathic etiologies vs specific etiologies were 88.8 months (95% confidence interval, 80.0 to 97.6) vs 39.3 months (95% confidence interval, 28.7 to 50.0), respectively. Recurrent pericardial pain without objective evidence of disease, recurrent pericarditis, cardiac tamponade, and constrictive pericarditis were considered adverse events during follow-up.

Patients at risk:
Idiopathic/viral:
416 394 387 380 372 366 354 338 320 298 275
Specific etiology:
84 79 70 54 47 40 32 20 8 8 8

Serology data supported the diagnosis of viral infection in 166 patients (Coxsackie in 66 patients, Ebstein-Barr virus in 25 patients, cytomegalovirus in 24 patients, parvovirus in 24 patients, influenza/parainfluenza viruses in 15 patients, and adenovirus in 12 patients). The specific etiologies (nonviral, nonidiopathic) of acute pericarditis were connective tissue disease or pericardial injury syndrome in 36 cases (7.2%), neoplastic pericarditis in 25 cases (5.0%), tuberculosis in 20 cases (4.0%), and purulent pericarditis in 3 cases (0.6%). Baseline data of the studied population are reported in Table 1.

Follow-Up Data and Risk of Constrictive Pericarditis
Follow-up data were available for all patients, and no data were lost at follow-up. During a median follow-up of 72 months (range, 24 to 120 months), patients with a nonviral, nonidiopathic etiology had a worse event-free survival compared with those with an idiopathic/viral etiology (Figure). Major adverse events included the following (Table 2): recurrent chest pain without objective evidence of disease in 85 cases (17.0%), recurrent pericarditis in 152 cases (30.4%), cardiac tamponade in 22 cases (4.4%), and permanent chronic constrictive pericarditis in 9 cases (1.8%). Transient constriction was detected by echocardiography in 75 of 500 patients (15.0%) with resolution within 3 months. Effusive constrictive pericarditis was recorded in 5 patients. All patients with chronic constriction had a surgically confirmed diagnosis of constrictive pericarditis. On histopathological examination of pericardiectomy specimens, 1 patient (11.1% of all cases of permanent chronic constrictive pericarditis) had normal pericardial thickness. A detailed list of clinical and instrumental data of patients

who developed permanent chronic constrictive pericarditis during follow-up is reported in Table 3.

Compared with patients with a specific etiology, patients with idiopathic/viral acute pericarditis had a lower risk of each adverse event: recurrent chest pain (14.9% versus 27.4%, respectively; $P=0.006$), recurrent pericarditis (25.0% versus 57.1%, respectively; $P<0.001$), cardiac tamponade (1.2% versus 20.2%, respectively; $P<0.001$), and constrictive pericarditis (Table 2). The risk of constrictive pericarditis was lower in patients with an idiopathic/viral etiology: 2 cases of 416 patients with idiopathic/viral pericarditis (0.48%) versus 7 cases of 84 patients with a nonviral/nonidiopathic etiology (8.3%). The incidence rate of constrictive pericarditis was 0.76 cases per 1000 person-years for idiopathic/viral pericarditis, 4.40 cases per 1000 person-years for connective tissue disease/pericardial injury syndrome, 6.33 cases per 1000

Table 2.   Adverse Events After a Mean Follow-Up of 60 Months

Adverse Event	Idiopathic or Viral (n=416)	Specific Etiology (n=84)	P
Recurrent chest pain (n=85)	62 (14.9)	23 (27.4)	0.006
Recurrent pericarditis (n=152)	104 (25.0)	48 (57.1)	<0.001
Cardiac tamponade (n=22)	5 (1.2)	17 (20.2)	<0.001*
Chronic constrictive pericarditis (n=9)	2 (0.48)	7 (8.3)	<0.001*

Data are expressed as numbers and percentages (in parentheses). Comparison between patient groups (idiopathic/viral etiologies vs specific etiologies) were performed with χ^2 analysis or Fisher exact test (when the number of observations obtained for analysis was relatively small).

*P values by Fisher exact test.

Table 3.   Clinical and Instrumental Findings of Patients Who Developed Constrictive Pericarditis

Feature	Patients With Constrictive Pericarditis (n=9)
Mean age, y	55±20
Male gender	5 (55.6)
Clinical features at presentation	
Fever >38°C	6 (66.7)
Subacute course	4 (44.4)
Incessant course	5 (55.6)
Large pericardial effusion	6 (66.7)
Cardiac tamponade	4 (44.4)
Nonidiopathic/nonviral etiology	7 (77.8)
Medical therapy features	
Aspirin or NSAID failure at 1 wk	6 (66.7)
Corticosteroids as initial therapy	3 (33.3)
Colchicine	2 (22.2)
Symptoms/signs*	
Symptoms of heart failure	6 (66.7)
Chest pain	2 (22.2)
Abdominal symptoms	2 (22.2)
Elevated jugular venous pressure	8 (88.9)
Pulsus paradoxus	2 (22.2)
Kussmaul's sign (lack of an inspiratory decline in jugular venous pressure)	2 (22.2)
Pericardial knock	5 (55.6)
ECG*	
Nonspecific ST- and T-wave changes	6 (66.7)
Low voltages	3 (33.3)
Atrial arrhythmias	2 (22.2)
Chest radiograph*	
Presence of pericardial calcifications	3 (33.3)
Echocardiography*	
Pronounced respiratory variations in ventricular filling, mitral inflow velocity changes >25%	9 (100.0)
Dilatation of the inferior vena cava and hepatic veins (plethora) with absent or diminished inspiratory collapse	9 (100.0)
Computed tomographic scan*	
Thickened pericardium	8 (88.9)
Cardiac magnetic resonance imaging*	
Increased pericardial thickening and inferior vena cava plethora	8 (88.9)
Enhanced ventricular interdependence	8 (88.9)
Hemodynamic findings*	
Increased right atrial pressure	8 (88.9)
"Square root" signs in RV and LV diastolic pressure tracings	8 (88.9)
Equalization of LV and RV diastolic plateau pressure tracings	8 (88.9)
Mirror-image discordance between RV and peak LV systolic pressures during inspiration	8 (88.9)

Data are expressed as numbers and percentages (in parentheses) unless indicated otherwise. NSAID indicates nonsteroidal anti-inflammatory drugs; RV, right ventricular; and LV, left ventricular.

*Features with the development of constrictive pericarditis.

Table 4.   Incidence of Constrictive Pericarditis According to Etiology

Etiology	Constrictive Pericarditis Evolution, No. (%)	Incidence/ 1000 Person-Years	95% CI Incidence Rate
Idiopathic/viral; n=416 (83.2%)	2 (0.48)	0.76	0.09–2.75
Pericardial injury syndrome and connective tissue diseases; n=36 (7.2%)	1 (2.8)	4.40	0.11–24.49
Neoplastic; n=25 (5.0%)	1 (4.0)	6.33	0.16–35.26
Tuberculosis; n=20 (4.0%)	4 (20.0)	31.65	8.62–81.03
Purulent; n=3 (0.6%)	1 (33.3)	52.74	1.34–293.86
All; n=500	9 (1.8)	2.85	1.30–5.41

Incidence rate is assessed as the number of new cases of disease during a period of time divided by the person time at risk. Person time is the estimate of the actual time at risk in years that all persons contributed to the study; 95% confidence intervals (CI) for incidence statistics are calculated.

person-years for neoplastic pericarditis, 31.65 cases for 1000 person-years for tuberculous pericarditis, and 52.74 cases per 1000 person-years for purulent pericarditis (Table 4).

Potential Risk Factors for Constrictive Pericarditis

The relative number of patients with constrictive pericarditis is small, and therefore it was not appropriate to perform a multivariate analysis. Nevertheless, bivariate analyses were done to explore potential risk factors for constriction. Patients who developed constrictive pericarditis during follow-up showed a higher frequency of specific features compared with those without such evolution: fever >38°C (66.7% versus 14.5%, respectively; $P<0.001$), incessant course (55.6% versus 6.9%, respectively; $P<0.001$), nonidiopathic/ nonviral etiology (77.8% versus 15.7%, respectively; $P<0.001$), large pericardial effusion (66.7% versus 8.6%, respectively; $P<0.001$), cardiac tamponade (44.4% versus 3.7%, respectively; $P=0.002$), and aspirin/nonsteroidal anti-inflammatory drug failure at 1 week (66.7% versus 18.7%, respectively; $P=0.002$).

A trend was found toward a higher rate of use of corticosteroids (33.3% versus 14.1%) and a lower rate of use of colchicine (22.2% versus 50.5%) in those who developed constrictive pericarditis.

Discussion

Permanent constrictive pericarditis is a rare, dreaded possible complication of acute pericarditis that is reported after nonidiopathic etiologies in particular. A few large series of patients with constrictive pericarditis diagnosed at pericardiectomy have been described, including 95 patients from Stanford,[8] 135 patients from the Mayo Clinic,[9] and 163 patients from the Cleveland Clinic.[10] The reported frequency of various causes in these reports, which is influenced by the selection of cases for referral and surgery, is as follows: idiopathic/viral in 42% to 49% of cases, after cardiac surgery in 11% to 37% of cases, after radiation therapy in 9% to 31% (mainly Hodgkin's disease or breast cancer), connective tissue disorder in 3% to 7% of cases, and after bacterial infection (tuberculous or purulent pericarditis) in 3% to 6% of

cases. Miscellaneous causes (malignancy, trauma, drug-induced, asbestosis, sarcoidosis, uremic pericarditis) were also reported in 1% to 10% of cases. More recently, 5 cases of surgically confirmed cases of constrictive pericarditis after orthotopic heart transplantation were described.[26]

Tuberculosis accounted for up to 50% of cases of constrictive pericarditis in historical series. Tuberculous pericarditis is now rare in developed countries (up to 5% of unselected cases) but remains common in developing countries (where it is the leading cause of pericarditis), in immigrants (especially from Africa and Eastern Europe), and in immunosuppressed patients.[4,7,27] Tuberculous pericarditis occurs in $\approx$1% to 2% of patients with tuberculosis and may present clinically in 3 forms: pericardial effusion (80% of cases), constrictive pericarditis (5% of cases), or effusive-constrictive pericarditis (15% of cases).[28] Pericardial constriction may be a delayed complication reported even today with a variable rate ranging from 20% to 60% of all cases, depending on the stage of the disease, and despite medical therapy.[4,7,27,28] However, this disorder may be increasing among immigrants from underdeveloped nations and patients with human immunodeficiency virus infection. Other forms of bacterial pericarditis are now rare. Purulent pericarditis is rare in the modern antibiotic era and accounts for <1% of unselected cases with acute pericarditis.[27] Nevertheless, for bacterial pericarditis, the risk of developing constrictive pericarditis is generally considered high.

Unfortunately, there are no contemporary prospective data on the risk of developing constrictive pericarditis after acute pericarditis in developed countries, and the incidence of the complication after idiopathic acute pericarditis is not well assessed. The constrictive evolution is a feared complication for either the clinician or the patients, especially when the disease does not respond to conventional therapy, the course is incessant or recurrent, or the etiology is unknown.

This study on a large sample size of patients with a long-term follow-up attempts to quantify the risk of developing constrictive pericarditis after a first attack of acute pericarditis with a risk assessment according to the etiology.

Overall, the complication is not common (1.8% in the overall population of acute pericarditis), but the risk is correlated with the etiology. Such evolution is rare (<0.5%) in idiopathic or viral acute pericarditis but is not negligible in other specific etiologies and is very high for bacterial causes: 2.8% for connective tissue disease or pericardial injury syndrome, 4.0% for neoplastic pericarditis, 20% for tuberculous pericarditis, and 33% for purulent pericarditis. In the observed study population, the symptoms of constrictive pericarditis were permanent and often progressive in chronic cases unless the constrictive pericarditis was surgically treated with pericardiectomy, whereas constriction was transient or reversible in 15% of patients with acute pericarditis. The clinical course with response to anti-inflammatory therapies in these patients implied the presence of constriction due to inflammation that resolved after standard treatment for acute pericarditis. On this basis, for patients with newly diagnosed constrictive pericarditis and without evidence of chronic constriction, a trial of conservative management with anti-inflammatory agents for 2 to 3 months is warranted rather than pericardiectomy.

Patients with idiopathic forms and a recurrent course did not show a constrictive evolution; these data are consistent with the previous observation that idiopathic recurrent pericarditis does not evolve to constrictive pericarditis[19] and that the risk of developing constrictive pericarditis also in the setting of recurrent pericarditis may be correlated with the etiology and not the number of recurrences. Such findings are interesting and suggest that the evolution toward constrictive pericarditis may follow a direct pathway correlated with the response to the specific etiologic agent.

The distinction between recurrent pericarditis and acute pericarditis directly evolving into constrictive pericarditis is clinically important. In idiopathic recurrent pericarditis, the clinical picture is characterized by attacks of pain, fever, and increased C-reactive protein that resolve, with normalization of C-reactive protein and disappearance of symptoms, but then recur after a free interval; this course is generally benign.[5,19] On the other hand, the 2 cases of idiopathic acute pericarditis that we observed evolved directly into constrictive pericarditis with an incessant course, with pain and fever at the beginning and with constriction developing after several months and becoming the dominant clinical feature.

In conclusion, constrictive pericarditis is a rare complication of viral or idiopathic acute pericarditis, whereas nonidiopathic etiologies (especially bacterial) are correlated with an increased risk of complications and constrictive pericarditis. Specific features, such as incessant course, large pericardial effusions, and failure of empirical anti-inflammatory therapy, should warrant a close follow-up because they may represent potential risk factors for the evolution toward constrictive pericarditis. In contrast, a recurrent course has a lower risk of constriction.

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Disclosures

None.

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CLINICAL PERSPECTIVE

Constrictive pericarditis (CP) is a rare but dreaded possible complication of acute pericarditis. This risk is evident after tuberculous pericarditis in particular, whereas it is not well established after idiopathic and viral acute pericarditis, the most common causes of acute pericarditis in developed countries. In a prospective cohort study of 500 consecutive cases of acute pericarditis, after a mean follow-up of 60 months, CP developed in 9 of 500 patients (1.8%): 2 of 416 patients with idiopathic/viral pericarditis (0.48%) versus 7 of 84 patients with a nonviral/nonidiopathic etiology (8.3%). The incidence rate of CP was 0.76 cases per 1000 person-years for idiopathic/viral pericarditis, 4.40 cases per 1000 person-years for connective tissue disease/pericardial injury syndrome, 6.33 cases per 1000 person-years for neoplastic pericarditis, 31.65 cases for 1000 person-years for tuberculous pericarditis, and 52.74 cases per 1000 person-years for purulent pericarditis. After a long follow-up, the overall number of cases of CP was relatively small (<2%). Patients and clinicians should be aware that CP is a rare complication of viral or idiopathic acute pericarditis. In contrast, nonidiopathic etiologies (especially bacterial) are correlated with an increased risk of complications and CP. Warning signs of a possible evolution toward CP are (1) an incessant course of the disease, (2) a nonidiopathic etiology, (3) large pericardial effusions, and (4) failure of empirical anti-inflammatory therapy. In contrast, a recurrent course has a lower risk of constriction.