Transient Constrictive Pericarditis: Causes and Natural History

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OBJECTIVES
This study was designed to elucidate the causes and natural history of transient constrictive pericarditis (CP).

BACKGROUND
In some patients with acute CP, the symptoms and constrictive physiologic features resolve with medical therapy alone, a phenomenon that has been labeled "transient constrictive pericarditis." No large studies have examined the causes or natural history of transient CP.

METHODS
Review of the Mayo Clinic echocardiogram database identified 212 patients who had echocardiographic findings of CP from 1988 through 1999. Demographic, clinical, and echocardiographic findings were identified in all patients. In 36 of these patients, follow-up echocardiograms showed resolution of the constrictive hemodynamics without pericardiectomy.

RESULTS
The average age of the patients was 49 ± 21 years, and 72% were men. The causes for the CP were diverse, the most common being prior cardiovascular surgery (25%). In a subset of 22 patients who were followed serially during the course of their illness, resolution of the constrictive physiologic features occurred at an average of 8.3 weeks after diagnosis.

CONCLUSIONS
A subset of patients with CP experience resolution of the disorder without requiring pericardiectomy. (J Am Coll Cardiol 2004;43:271–5) © 2004 by the American College of Cardiology Foundation

Classic constrictive pericarditis (CP), a progressive and debilitating condition, is characterized by pericardial fibrosis, with or without calcification, which results in chronic refractory congestive heart failure and for which pericardiectomy is often required. Until relatively recently, the development of constrictive physiology was presumed to be irreversible. However, in recent years there have been reports describing a transient form of CP that resolves without surgical intervention. The development of constrictive hemodynamics and subsequent resolution with medical therapy (transient CP) was first described by Sagrista-Sauleda et al. (1) in 1987 in a group of 16 patients with idiopathic pericarditis. Since then, it has been described in the setting of collagen vascular disease (2), purulent bacterial pericarditis (2,3), and chemotherapy (4). To elucidate the causes and natural history of transient CP, we report our experience with 36 patients with this disorder.

Patients were excluded from the study if they had previous pericardiectomy (including partial pericardiectomy or pericardial window) at another institution, if no follow-up echocardiogram was available, or if clinical findings were not consistent with constrictive physiology.

Echocardiographic diagnosis of CP. The diagnosis of CP can be elusive, even after extensive investigation. Two-dimensional echocardiography with Doppler is one of the initial tests performed when CP is clinically suspected and oftentimes is diagnostic. However, it should be noted that even a carefully performed echocardiogram may be inconclusive in differentiating CP from restrictive cardiomyopathy, and in these cases additional testing, including cardiac catheterization and even surgical exploration, may be required to establish the diagnosis. The two-dimensional and M-mode echocardiographic criteria for CP include abnormal ventricular septal motion (5,6), respiratory variation in ventricular size (7), and presence of a dilated inferior vena cava (7). The Doppler findings in CP, as described by Hatle et al. (8), are both sensitive and specific (9). They reflect the inspiratory decreases and expiratory increases in left ventricular filling (with reciprocal changes in right ventricular filling) that occur in CP as a result of the relatively fixed cardiac volume. These patients have an expiratory increase in transmitial inflow velocity of 25% or more compared with inspiration. Other characteristic Doppler findings in CP are expiratory decreases in hepatic vein diastolic forward flow and increases in hepatic vein diastolic flow reversals.
RESULTS

During the period 1988 through 1999, 36 patients met the inclusion criteria for the diagnosis of transient CP. The majority were men (72%), and the average age was 49.0 ± 21 years (range 7 to 89). Thirty-three patients (92%) had at least one clinical symptom (Table 1). More than half of the patients (53%) experienced chest pain, and 44% described dyspnea.

A follow-up echocardiogram documenting resolution of the constrictive physiologic features was available in all patients (Figs. 1 to 3). The various causes of transient constriction and their frequency, along with the time to echocardiographic resolution of the constrictive features, are shown in Table 2.

In a subset of 22 patients who were followed throughout the course of their illness at our institution, resolution of symptoms and constrictive physiologic features occurred after a mean of 8.3 weeks. Table 3 shows the various treatments used. Most patients (86%) received some form of medical therapy, more than half (56%) having been treated with nonsteroidal anti-inflammatory drugs (NSAIDs). After a mean of 2.3 years, there have been no recurrences of constrictive physiology or clinical symptoms.

A pericardial effusion (PE) was documented in 24 patients (67%), 8 of whom underwent pericardiocentesis. In all patients who had pericardiocentesis, echocardiographic features of constrictive physiology persisted after pericardiocentesis and thus verified the presence of effusive-CP. Additionally, in the 17 patients who had chest computed tomography or magnetic resonance imaging, 10 (59%) showed increased pericardial thickening. Two of these patients had follow-up pericardial imaging, and in both cases the pericardial thickness was normal (Fig. 4).

DISCUSSION

In 1980, Hancock (10) described two forms of pericardial constriction, one being elastic and the other more analogous to a rigid shell. The fibroelastic form, he proposed, represented the acute or subacute phase of constriction. He noted that patients with subacute fibroelastic constriction present with more subtle signs and symptoms, rather than the classic findings seen in chronic CP. In many patients who are identified in the course of the initial pericardial inflammatory response, pericardial inflammation continues and pericardial fibrosis and calcification subsequently develop, leading to chronic (rigid) CP. However, as shown by this and other studies (1–4), in some patients the pericardial inflammation resolves without progressing to chronic CP. Thus, the subacute form of CP appears to be a reversible step in the progression to chronic CP.

Transient CP was originally described in the English literature by Sagrista-Sauleda et al. (1) in 1987. They reported the development of objective evidence of constrictive physiology occurring in 16 of 177 patients (9%) with effusive acute idiopathic pericarditis, with subsequent resolution with medical therapy and observation. The interval between the first echocardiogram showing PE and noninvasive recordings demonstrating CP ranged from 5 to 30 days (mean 11 days). The time to normalization of the noninvasive recordings for all patients ranged from 7 days to 58 months (mean 9.4 months). Seven patients in the study who were followed prospectively had resolution of the constrictive physiologic features after a mean of 2.7 months (range 12 days to 10 months). At a mean follow-up of 31 months, none of their patients had had a recurrence of constriction. The authors suggested that the mechanism responsible for the findings in these patients was a transiently thickened and inelastic pericardium resulting from edema, fibrin deposition, or inflammation. They also noted a characteristic three-phase evolutionary pattern of constriction. The first phase was at least a moderate amount of PE without evidence of constriction. The second phase was constrictive physiology. In this phase, the PE had resolved in 6 of the patients and had decreased in size in the remaining 10 patients. The third phase was normalization of...

Table 1. Symptoms in 36 Patients With Transient Constrictive Pericarditis

<table>
<thead>
<tr>
<th>Symptom</th>
<th>No. (%)</th>
</tr>
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<tbody>
<tr>
<td>Chest pain</td>
<td>19 (53)</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>16 (44)</td>
</tr>
<tr>
<td>Fever</td>
<td>6 (17)</td>
</tr>
<tr>
<td>Edema</td>
<td>4 (11)</td>
</tr>
<tr>
<td>Cough</td>
<td>2 (6)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>2 (6)</td>
</tr>
<tr>
<td>None</td>
<td>3 (8)</td>
</tr>
</tbody>
</table>

Figure 1. (A) Echocardiographic findings in constrictive pericarditis. Parasternal long-axis view of a 39-year-old man who presented one month after a motor vehicle accident with dyspnea, showing markedly increased pericardial thickness (arrows). (B) Follow-up echocardiogram performed one month later, showing near-normal pericardial thickness (arrows). Ao = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.
hemodynamics with no evidence of recurrence of constric-
tion at a mean follow-up of 31 months.

In 1993, Oh et al. (2), using Doppler echocardiography,
demonstrated four instances of transient CP in conditions
other than acute idiopathic pericarditis. Two were thought
to be immune mediated, one was attributed to acute viral
pericarditis, and one was a result of purulent bacterial
pericarditis. With treatment of the underlying problem,
the constrictive hemodynamics resolved in all four cases. This
study extended the initial findings of Sagrista-Sauleda et al.
(1) and showed that transient CP has causes other than
idiopathic pericarditis.

Figure 5 compares the etiologic factors in our patients
with those responsible for CP requiring pericardiectomy in
a recent study from the Mayo Clinic (11). As can be seen,
the causes and their frequency of occurrence are quite
similar in the two studies, with the notable exception of
radiation-induced disease. In none of the patients in our
study was radiation-induced disease a cause of the constrict-
tive disorder; nor does the literature cite any cases of
transient CP resulting from radiation therapy. Given the
progressive, unremittent, fibrotic nature of postirradiation
CP and the lack of medical therapy for radiation-induced
disease, it seems unlikely that transient constrictive physi-
ology would occur in this subset of patients. However, a
prospective study of a population of patients with radiation-
induced disease would be necessary to prove this hypothesis.

The results of this study suggest that patients with
symptoms and physiologic features consistent with CP can
be managed with a trial of medical therapy before surgery is
considered. It should be emphasized that the patients
reported here were all diagnosed in the acute phase of their
illness and that these results may not apply to patients who
present with a longer duration of symptoms. The mean time
to recovery in patients who were followed serially was 2.1
months in this study and 2.7 months in the study by
Sagrista-Sauleda et al. (1). All the patients in our study who
were followed serially experienced resolution of echocardio-
graphic findings of constriction in <6 months. Therefore, a
trial of medical therapy is recommended with the expecta-
tion of an average recovery time of three months. Obviously,
patients with worsening symptoms or clinical deterioration

Figure 2. (A) Doppler findings in constrictive pericarditis. Pulsed-wave Doppler recording of the mitral inflow velocity in the patient described in Figure 1, showing a marked increase in the mitral inflow E velocity (single arrowhead) with expiration compared with inspiration (double arrowhead). (B) Follow-up echocardiogram one month later, showing no significant increase in the mitral inflow E velocity with expiration, consistent with resolution of constrictive hemodynamics. I = inspiration; E = expiration.

Figure 3. (A) Doppler findings in constrictive pericarditis. Pulsed-wave Doppler recording from the hepatic vein in the patient described in Figure 1, showing increased diastolic flow reversal with expiration (arrowhead). (B) Follow-up echocardiogram performed one month later, showing an absence of increased diastolic flow reversals with expiration (double arrowhead), consistent with resolution of constrictive hemodynamics. I = inspiration; E = expiration.
not responsive to medical therapy should be considered for early surgical treatment.

As noted earlier, the majority of patients received some form of therapy. More than half (56%) were treated with NSAIDs, and other patients were treated with steroids, antibiotics, chemotherapy, or diuretics. Five patients (14%) received no therapy. Given the retrospective nature and small numbers of patients in this study, it is impossible to know whether resolution of symptoms was more rapid with medical therapy compared with no therapy. However, a reasonable approach would be to utilize medical therapy directed at treating the patient's symptoms while waiting for the constrictive physiologic features to resolve (i.e., NSAIDs for chest pain, antibiotics for infection, diuretics for fluid retention, and so forth).

This study has several important limitations, including its retrospective design. The patients were not followed at uniform intervals; that is, follow-up examinations and echocardiograms were performed at the discretion of the treating physician, and in some patients echocardiograms were not obtained at initial presentation. In addition, it is quite likely that this disorder occurs more often than reported in this study, as a number of patients who were treated for transient constrictive physiology and symptoms have not returned for a follow-up examination or echocardiography to document possible resolution of the abnormal findings. Finally, it is possible that some patients who would have experienced a transient course of constriction were treated with pericardectomy.

### Table 2. Causes of Transient Constrictive Pericarditis in 36 Patients

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. (%)</th>
<th>Resolution (Weeks)</th>
</tr>
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<tbody>
<tr>
<td>Postpericardiotomy</td>
<td>9 (25)</td>
<td>17.4</td>
</tr>
<tr>
<td>Pericarditis, viral</td>
<td>7 (19)</td>
<td>9.8</td>
</tr>
<tr>
<td>Pericarditis, bacterial</td>
<td>4 (11)</td>
<td>18.9</td>
</tr>
<tr>
<td>Idiopathic</td>
<td>8 (22)</td>
<td>14.4</td>
</tr>
<tr>
<td>Collagen vascular disease</td>
<td>5 (14)</td>
<td>27.6</td>
</tr>
<tr>
<td>Trauma</td>
<td>1 (3)</td>
<td>16.0</td>
</tr>
<tr>
<td>Malignancy</td>
<td>1 (3)</td>
<td>8.0</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>1 (3)</td>
<td>112.0</td>
</tr>
</tbody>
</table>

### Table 3. Therapy Used for Treatment of Transient Constrictive Pericarditis in 36 Patients

<table>
<thead>
<tr>
<th>Therapy</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti-inflammatory agents</td>
<td>20 (56)</td>
</tr>
<tr>
<td>Steroids</td>
<td>16 (44)</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>4 (11)</td>
</tr>
<tr>
<td>ACEI, diuretics</td>
<td>2 (6)</td>
</tr>
<tr>
<td>Chemotherapy</td>
<td>1 (3)</td>
</tr>
<tr>
<td>None</td>
<td>5 (14)</td>
</tr>
</tbody>
</table>

ACEI = angiotensin-converting enzyme inhibitor.

In conclusion, this study, the largest to date of this patient population, shows that transient constrictive physiologic features, as seen by Doppler echocardiography, can occur in almost any condition that can cause chronic CP, with the possible exception of radiation therapy. If the constrictive findings are going to resolve, they do so at an average time of three months. The results of this study suggest that patients who have constrictive features early in the course of their illness and are hemodynamically stable should be considered for a trial of conservative therapy before pericardectomy is pursued.

![Figure 4](image)
REFERENCES