Effect of Definition on Incidence of Postinfarction Pericarditis

Is It Time to Redefine Postinfarction Pericarditis?

Philip B. Oliva, MD; Stephen C. Hammill, MD; James V. Talano, MD

Pericarditis is possibly the most common cause of chest pain after an acute transmural myocardial infarction without reperfusion. It occurs in approximately 28% to 40% of fatal transmural infarctions, and a pericardial effusion is detectable by serial echocardiograms in 28% to 63% of patients with a nonfatal transmural infarction. Yet the clinically reported frequency of postinfarction pericarditis ranges between 7% and 41%. Such a wide range seems inconsistent with the narrower frequency range determined by pathological examination and serial echocardiograms.

Assuming that the wide frequency range of clinically diagnosed postinfarction pericarditis is due to a difference in the clinical definition among various studies, whereas the pathological and echocardiographic frequencies of postinfarction pericarditis and postinfarction pericardial effusion, respectively, are due to more precise, objective diagnostic criteria and observations, the present report reviews the clinical frequency of postinfarction pericarditis according to its definition (see below) and compares it with the available pathological and echocardiographic data.

Types of Postinfarction Pericarditis

Between 1936 and 1941, four articles emphasized an 80% to 85% incidence of acute localized—i.e., regional—postinfarction pericarditis, in contrast to a 15% to 20% incidence of the diffuse variety. Both types occurred during the first week after an acute myocardial infarction. In 1956, Dressler described a form of postinfarction pericarditis characterized by prolonged or recurrent positional pleuritic chest pain, pulmonary infiltrates, fever, an increased erythrocyte sedimentation rate, and/or a pericardial friction rub. This syndrome usually occurs 2 to 11 weeks after the infarction, although occasionally it is recognized during the first week. Although some authors subsequently have questioned the existence of Dressler’s syndrome, despite a 12% incidence of postinfarction heart muscle antibodies, general experience has established its incidence as 5% or less, a much lower incidence than the accepted, consistently earlier form.

Definition of Pericarditis

During the pre–World War II years, when a three- or four-lead ECG was standard, interest was focused on the ECG alterations associated with pericarditis. The diagnosis of pericarditis was based on the presence of a pericardial friction rub, a pericardial effusion, or autopsy evidence of pericardial injury. Symptoms were not used. Evolutionary changes of the ST segment and T waves were recognized and shown to be due to subepicardial inflammation and/or injury. It remained for Spodick to formally establish the characteristic four phases of repolarization changes due to pericarditis.

After World War II, a spate of reports appeared describing the clinical characteristics of acute rheumatic or nonspecific pericarditis. These articles emphasized the importance of recognizing positional pleuritic chest pain as evidence of pericarditis. Whereas 92% to 100% of patients with pericarditis had typical chest pain, only 47% to 74% had a pericardial friction rub.

With regard to postinfarction pericarditis, authors of late 20th-century textbooks on cardiology also advised avoiding reliance on a friction rub as the sole indicator of pericarditis. In 1966, Dr Charles K. Friedberg stated that “even when no rub is heard a highly probable diagnosis of acute pericarditis can be made on the basis of the typical pleuropurulent pain.” Six years later he added that positional pleuritic chest pain is “adequate to diagnose pericarditis whether or not a rub is audible.” In 1978, Dr J. Willis Hurst et al wrote that “pericarditis secondary to transmural infarction is much more common than the 15% incidence customarily quoted (by rub).” Moreover, they noted that if an “observer demands the presence of a pericardial friction rub,” the diagnosis of pericarditis may be “unrecognized in spite of typical pain.” Others warned that requiring a friction rub to diagnose postinfarction pericarditis will result in a “gross underestimate” of its incidence since “many patients have classic symptoms, but the friction rub does not occur or is missed because of its fleeting nature.”

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TABLE 1. Relation of Incidence of Postinfarction Pericarditis to Definition of Condition

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>No. of Cases With AMI</th>
<th>No. (%) With Peri</th>
<th>Q-Wave AMI, %</th>
<th>Non-Q-Wave AMI, %</th>
<th>Rub</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>White</td>
<td>1926</td>
<td>62</td>
<td>8 (13)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Parkinson and Bedford</td>
<td>1928</td>
<td>100</td>
<td>7 (7)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Levine and Brown</td>
<td>1929</td>
<td>145</td>
<td>20 (14)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>White and Bland</td>
<td>1931</td>
<td>200</td>
<td>20 (10)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Master and Jaffe</td>
<td>1935</td>
<td>NS</td>
<td>2</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Blumer</td>
<td>1936</td>
<td>109</td>
<td>32 (29)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Vander Veen and Brown</td>
<td>1936</td>
<td>41</td>
<td>7 (17)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Bean</td>
<td>1938</td>
<td>176</td>
<td>24 (14)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Rosenbaum and Levine</td>
<td>1941</td>
<td>208</td>
<td>33 (16)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Wood</td>
<td>1968</td>
<td>NS</td>
<td>(10)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Thadani et al</td>
<td>1971</td>
<td>779</td>
<td>52 (7)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Niarchos and McKendrick</td>
<td>1973</td>
<td>195</td>
<td>22 (11)</td>
<td>100</td>
<td>0</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Barman et al</td>
<td>1973</td>
<td>1284</td>
<td>106 (8.25)</td>
<td>100</td>
<td>0</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Lichstein et al</td>
<td>1974</td>
<td>305</td>
<td>31 (10)</td>
<td>100</td>
<td>0</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Yan</td>
<td>1974</td>
<td>338</td>
<td>41 (10.6)</td>
<td>93</td>
<td>7</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Toole and Silverman</td>
<td>1975</td>
<td>554</td>
<td>40 (7)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Liem et al</td>
<td>1975</td>
<td>300</td>
<td>44 (15)</td>
<td>70</td>
<td>30</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>McLean et al</td>
<td>1975</td>
<td>1505</td>
<td>224 (14.9)</td>
<td>83</td>
<td>17</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Guillemin and Valere</td>
<td>1976</td>
<td>400</td>
<td>64 (16)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Sawaya et al</td>
<td>1980</td>
<td>261</td>
<td>38 (14.5)</td>
<td>100</td>
<td>0</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Hutter et al</td>
<td>1981</td>
<td>196</td>
<td>28 (14)</td>
<td>86</td>
<td>14</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Northcote et al</td>
<td>1984</td>
<td>80</td>
<td>23 (28)</td>
<td>NS</td>
<td>NS</td>
<td>+</td>
<td>5*</td>
</tr>
<tr>
<td>Kaplan et al</td>
<td>1985</td>
<td>43</td>
<td>12 (28)</td>
<td>79</td>
<td>21</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Dubois et al</td>
<td>1985</td>
<td>1264</td>
<td>297 (23.4)</td>
<td>86</td>
<td>14</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Galve et al</td>
<td>1986</td>
<td>121</td>
<td>35 (29)</td>
<td>91</td>
<td>9</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Krainin et al</td>
<td>1985</td>
<td>423</td>
<td>31 (7.3)</td>
<td>90</td>
<td>10</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Somolinos et al</td>
<td>1987</td>
<td>46</td>
<td>19 (41)</td>
<td>100</td>
<td>0</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Toffer et al</td>
<td>1989</td>
<td>703</td>
<td>141 (20)</td>
<td>70</td>
<td>30</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Oliva et al</td>
<td>1993</td>
<td>851</td>
<td>26 (30)</td>
<td>89</td>
<td>11</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

AMI indicates acute myocardial infarction; Peri, postinfarction pericarditis; and NS, not stated.
*All five patients with symptoms also had a friction rub.
†Exclusive of 115 patients who received lytic therapy.

pericarditis is "likely underestimated" because pericardial friction rubs are often evanescent or overlooked or because the pain may be "attributed to recurrent ischemia." Despite these recommendations by textbook authors to accept the diagnosis of postinfarction pericarditis if typical symptoms exist without a friction rub, authors publishing in medical journals have frequently required a pericardial friction rub to establish the diagnosis. Table 1 (References 47 through 69) discloses that all published articles in the English-language literature from 1926 through 1973 based the diagnosis of postinfarction pericarditis on a rub only. Not until 1974 was an article published that accepted typical pericardial pain and/or a pericardial friction rub to diagnose postinfarction pericarditis. From 1974 to 1993, 7 of 14 peer-reviewed articles and two letters to the Editor accepted classic symptoms and/or a friction rub.

If a friction rub alone is used to diagnose postinfarction pericarditis, the mean incidence of this condition among patients not receiving lytic therapy is 14%. If classic symptoms or a friction rub or both are used as diagnostic criteria, the mean incidence rises to 25% (Table 1). This frequency approximates the autopsy-observed frequency of postinfarction pericarditis of 28% to 40%.

Sensitivity of ECG Criteria for Regional Postinfarction Pericarditis and Relation of ECG Criteria to Definition of the Condition

Recently, through an autopsy examination of 70 patients with fatal left ventricular free-wall rupture, we learned that regional postinfarction pericarditis accompanied rupture in 94% of instances and was consistently associated with an atypical form of postinfarction...
TABLE 2. Effect of Lytic Therapy on Frequency of Postinfarction Pericarditis

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Non-Lytic Therapy Group, %</th>
<th>Lytic Therapy Group, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simoons et al76</td>
<td>1985</td>
<td>17</td>
<td>7</td>
</tr>
<tr>
<td>GISSI76</td>
<td>1986</td>
<td>12</td>
<td>6.4</td>
</tr>
<tr>
<td>ECS77</td>
<td>1988</td>
<td>11</td>
<td>6.3</td>
</tr>
<tr>
<td>AIMS78</td>
<td>1990</td>
<td>15</td>
<td>7</td>
</tr>
<tr>
<td>Oliva et al74</td>
<td>1993</td>
<td>30</td>
<td>15</td>
</tr>
</tbody>
</table>

GISSI indicates Gruppo Italiano per lo Studio della Streptocchina nell'Infarto Miocardico; ECS, European Cooperative Study Group; and AIMS, Anistreplase in Acute Myocardial Infarction Study.

T-wave evolution during the day, or more often several days, before rupture.71 These T-wave changes were similar to those recorded in dogs with localized postinfarction pericarditis in 1939.72 Two types of atypical T-wave evolution were observed clinically. Either the T waves remained persistently positive for 48 hours or longer after the onset of an acute myocardial infarction (type I), or initially inverted T waves gradually became positive deflections (type II). The sensitivity and specificity, as defined previously, of these atypical T-wave changes were 100% and 77%, respectively.70 The specificity rose to 96% if patients who had reinfarction, underwent cardiopulmonary resuscitation, or sustained a very small initial infarct as a consequence of early lytic therapy were excluded. The frequency of postinfarction pericarditis defined by classic symptoms and/or a rub was 30% in those patients who did not receive lytic therapy. There was a 2.5% frequency of unexplained atypical T-wave evolution ("false-positives"). One explanation for this 2.5% incidence of false-positives is that those patients may have had painless and acoustically silent postinfarction pericarditis. If so, the actual incidence of postinfarction pericarditis would be 32.5% in patients not receiving lytic therapy, in harmony with the autopsy range of 28% to 40%.

The sensitivity of these ECG alterations for postinfarction pericarditis was recently confirmed by an investigation performed at Northwestern University.73 We reviewed the serial ECGs of all patients from that institution with clinically recognized (by typical positional pleuritic chest pain and/or a friction rub) postinfarction pericarditis reported by Kaplan et al.9 All patients had one of the two types of atypical T-wave evolution,73 affirming the 100% sensitivity of these ECG changes to diagnose postinfarction pericarditis and also justifying the use of typical symptoms and/or a friction rub to diagnose this condition.

Effect of Lytic Therapy on the Incidence of Postinfarction Pericarditis

Since 1985, five reports74-78 have addressed the effect of lytic therapy on the incidence of postinfarction pericarditis (Table 2). All agree that the incidence is halved by lytic therapy, whether typical symptoms and/or a friction rub or a rub alone is used to define postinfarction pericarditis. We found that the incidence of postinfarction pericarditis as defined by typical symptoms and/or a rub was reduced from 30% to 15% by lytic therapy.74 Other reports75-78 using a rub alone found an incidence reduction from between 11% and 17% without lytic therapy to between 6% and 7% with lytic therapy. The absolute numbers are, of course, lower when a rub alone is used to define postinfarction pericarditis, for reasons discussed earlier. However, regardless of the definition, a 50% reduction of the incidence of postinfarction pericarditis by lytic therapy is observed.

Conclusions

Postinfarction pericarditis can be diagnosed when an accurate history is elicited from an informed cardiologist from an observant, articulate-patient. The requirement of a friction rub leads to a significant underestimation of the incidence of postinfarction pericarditis. Postinfarction pericarditis occurs in an average of 25% of instances of acute transmural myocardial infarction not treated with lytic therapy when typical symptoms and a pericardial friction rub are accepted as indicative of pericarditis, whereas the average incidence is only 14% when a friction rub alone is required.

Atypical T-wave evolution appears to be a very sensitive and reasonably specific ECG sign of regional postinfarction pericarditis. It is more sensitive than a friction rub and, when observed in a patient with chest pain after an acute myocardial infarction, provides an objective method of differentiating among the three causes of postinfarction chest pain.

Lytic therapy reduces the frequency of postinfarction pericarditis by 50% whether classic symptoms and a friction rub or a rub alone is used to define the condition. Since postinfarction pericarditis is almost always the consequence of a transmural infarction,78 its reduction by lytic therapy implies sparing of the pericardium and subjacent myocardium.

Acknowledgments

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Galve

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