Pericarditis and Pericardial Effusion in Acute ST-Elevation Myocardial Infarction in the Thrombolytic Era

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Abstract

Background: Myocardial infarction-associated pericarditis is a common cause of chest pain following MI, its frequency depending on how it is defined.

Objectives: To investigate the incidence of acute pericarditis and pericardial effusion in the acute phase of ST-elevation MI treated with thrombolytic therapy.

Methods: The study group comprised 159 consecutive patients fulfilling the criteria for acute MI who were admitted to our department during 18 months. Infarct-associated pericarditis was defined as the finding of a pericardial friction rub, a typical pleuropericardial pain, or both. All patients underwent physical examination of the cardiovascular system four times daily for 7 days, as well as daily electrocardiogram and echo Doppler examinations.

Results: Fourteen patients (8.8%) developed a friction rub and 11 patients (6.9%) had a mild pericardial effusion. Six patients (4.0%) had both a friction rub and pericardial effusion. Two patients had a friction rub for more than 7 days. Pleuropericardial chest pain was present in 31 patients (19.5%) but only 7 of them had a friction rub. The in-hospital mortality rate was 1.3% and no mortality was observed in the acute pericarditis group.

Conclusion: The incidence of signs associated with acute pericarditis was lower in MI patients treated with thrombolysis, compared with historical controls, when a friction rub and/or pericardial effusion was present. There was no significant reduction in the incidence of pleuropericardial chest pain.

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Early acute pericarditis, which may appear within a week after acute myocardial infarction, represents an important clinical event. Pericarditis is possibly the most common cause of chest pain after an acute ST-elevation myocardial infarction without reperfusion [1]. The frequency of post-infarction pericarditis ranges between 7 and 41% [2,3]. The wide range of frequency in reported post-infarction pericarditis is likely due to differences in the clinical definition of this syndrome in various studies. The diagnosis of infarct-associated pericarditis is usually made on the basis of friction rub accompanied by symptoms of classic positional chest pain. A pericardial friction rub has been reported to be the most specific non-invasive sign of pericardial inflammation [4]. Earlier studies have demonstrated that when a friction rub is considered the only diagnostic criterion for the diagnosis of pericarditis, the true incidence of pericarditis is likely to be underestimated because of its fleeting nature [5].

In this study we investigated the incidence of pericarditis in the acute phase of ST-elevation myocardial infarction treated with thrombolytic therapy. In addition, we address the question of how to most accurately define infarct-associated pericarditis and whether the definition affects the observed incidence of infarct-associated pericarditis.

Methods

Patients

We performed a prospective analysis of 159 consecutive patients admitted during an 18 month period to the coronary care unit within 12 hours of the onset of chest pain, fulfilling the criteria for acute ST-elevation MI, and treated with thrombolytic therapy. Infarct-associated pericarditis was defined as the finding of a pericardial friction rub, a typical pleuropericardial pain, or both.

Clinical evaluation

The diagnosis of MI was made if the patient had a new ST-elevation with new Q-waves (one or more in leads I, II, III, aVL, aVF and/or two or more in leads V1 to V6) on serial electrocardiograms, and cardiac enzyme elevation including creatinine kinase at least twice the normal value. All patients were monitored continuously in the coronary care unit and were systematically (four times daily) questioned about pleuropericardial chest pain, which was defined as respiratory chest pain radiating to the shoulder or the neck, worsening with inspiration or coughing, and improving when leaning forward or in the sitting position. Post-MI ischemia was excluded by the anamnesis, ECG and cardiac enzyme elevation. All patients were examined by careful auscultation at least four times a day for 7 days.

Pericardial rub is considered as a to-and-fro scratchy or grating noise heard in systole, mid-diastole and pre-systole, or one of these phases. The diagnosis of pericardial rub was made after confirmation by at least two cardiologists.

Erythrocyte sedimentation rate and serum C-reactive protein were not used as diagnostic criteria because of their low diagnostic specificity for pericarditis in the acute phase of acute MI, although they can be of value in non-infarct-associated pericarditis.

Echocardiography

M-mode, two-dimensional and Doppler echocardiography (Hewlett Packard Sonos 1000, USA) were performed daily during the first 7 days after the myocardial infarction, and classical views were recorded on videotape for subsequent analysis. An epicardial pericardial separation that persisted throughout the cardiac cycle

MI = myocardial infarction

ECG = electrocardiogram

(d-pattern) was considered diagnostic for pericardial effusion [6]. In order to quantify the size of the effusion, measurements were obtained at the level of the tip of the mitral valve as described by Weitzman et al. [7]. Total effusion was graded as mild (<10 mm), moderate (10–20 mm), or severe (> 20 mm).

Statistical analysis

Chi-square test was used for statistical analysis for comparison of the two groups (with and without diagnosis of infarct-associated pericarditis).

Results

The characteristics of the 159 patients included in the study group are presented in Table 1. Eleven patients (6.9%) had a mild pericardial effusion, of whom 4 had anterior wall MI and 7 had diaphragmatic MI. Six patients had both a friction rub and pericardial effusion, with three having diaphragmatic and three anterior wall MI. Two patients had a friction rub for more than 7 days. Pleuropericardial chest pain was present in 31 patients (19.5%), but only 7 of them had a friction rub.

The in-hospital mortality rate for the 159 patients was 1.3% and there was no mortality observed in the acute pericarditis group. There was no cardiac tamponade or rupture. There were no significant statistical differences in the incidence of pericarditis between the different locations of MI. The incidence of pericarditis and pericardial effusion was significantly higher in females (P = 0.019 and 0.015 respectively). In our study group we did not observe a higher incidence of pericarditis or pericardial effusion in different age groups. Moreover, subgroup analysis of patients with an additional diagnosis – such as hypertension, hyperlipidemia, renal failure or previous MI – did not reveal a higher incidence of pericarditis or pericardial effusion. Patients with diabetes mellitus type 2 had a higher incidence of pericarditis (P = 0.011), but not of pericardial effusion [Table 2].

Discussion

Previous studies observed a 25% mean incidence of infarct-associated pericarditis if the symptoms of classic positional chest pain, a rub, or both were used as diagnostic criteria, whereas pericarditis detected by a friction rub alone was found in 14% of the patients. The use of thrombolytic therapy in acute MI was accompanied by a 50% reduction in the incidence of infarct-associated pericarditis regardless of its definition [1]. In the present study we found the incidence of infarct-associated pericarditis to be 23.9% when the symptoms of positional chest pain, rub, or both were used as diagnostic criteria; however, if only friction rub is considered, the incidence was 8.8%.

All patients were treated with thrombolytic agents. Although our findings in the patients treated with thrombolytic therapy are compatible with the results of earlier studies – 50% reduction in the incidence of infarct-associated pericarditis according to friction rub alone – we did not find a 50% decrease in the incidence according to typical pericardial chest pain. We believe that the diagnosis of infarct-associated pericarditis according to positional chest pain is likely to lead to an overestimation of its incidence. The incidence of acute pericarditis defined by pericardial effusion, occurrence of a friction rub, or both, was lower in patients treated with thrombolytic therapy for acute ST-elevation MI, compared to literature reports in the pre-thrombolytic era. The patients were carefully examined every 6 hours for a friction rub during 7 days; however, we still cannot exclude a friction rub that was present in some of the patients for 1 or 2 hours and therefore our finding of friction rub in 14 patients (8.8%) is the minimal incidence. When pleuropericardial pain is considered, there is no significant reduction in the incidence of pericarditis.

Table 1. Patients' characteristics

	No. (%)
Patients	159
Gender	
Males	134 (84.3)
Females	25 (15.7)
Age (yr)	
Range	31-89
Median	57
MI	
Anterior	70 (44)
Inferior	82 (51.6)
Lateral	7 (4.4)
Old MI	34 (21.4)
Dyslipidemia	78 (49.1)
Smoking	94 (59.1)
Hypertension	57 (35.8)
Diabetes	35 (22)
Renal failure	7 (4.4)

The total number of female and diabetic patients was too small to draw definite conclusions, however a tendency towards higher occurrence of pericarditis was noted. The incidence of pericarditis in these subgroups deserves further study.

The infarct-associated pericarditis appeared within a week after acute myocardial infarction. The diagnosis is important because infarct-associated pericarditis can be confused with pain resulting from ischemic events or extension of the infarction. Pericarditis usually occurs only when the acute MI is transmural [4,8]. The presence of transmural necrosis is believed to be a prerequisite for the development of pericardial inflammation [9]. According to this explanation, infarct-associated pericarditis occurs more frequently among patients with Q-wave MI, however it is also present in small numbers of those with non-Q-wave MI [10]. It is likely that Q-wave infarcts mainly represent transmural infarcts, whereas non-Q wave infarcts only occasionally do [11]. In our study, no patients with ST-elevation MI developed non-Q wave infarctions.

Table 2. Results of MI-associated pericarditis in patients with pleuropericardial pain and friction rub

Variable	MI-associated pericarditis (%)	No pericarditis (%)	*P*
Total	38 (23.9)	121 (76.1)	
Age (mean)	59.9	57.9	NS
Gender			
Male	27 (20.1)	107 (79.9)	0.01
Female	11 (44)	14 (56)	
Diabetes	14 (40)	21 (60)	0.011
Hypertension	15 (26.3)	42 (73.7)	NS
Smoking	18 (19.1)	76 (80.9)	0.091
Dyslipidemia	15 (19.2)	63 (80.8)	NS
MI			
Anterior	19 (27.1)	51 (72.9)	
Inferior	19 (23.2)	63 (76.8)	NS
Lateral	0 (0)	7 (100)	

The incidence of infarct-associated pericarditis depends on its definition. The most important clinical signs of infarct-associated pericarditis are pleuropericardial chest pain and a friction rub. Some authors considered the presence of a pericardial rub necessary for diagnosis [4,8,12,13]; others [14] warned that requiring a friction rub to diagnose post-infarction pericarditis will result in a gross underestimation of its incidence, since many patients have typical pleuropericardial pain but friction rub does not occur or is missed because of its fleeting nature.

The typical diagnostic ECG changes of acute pericarditis are extremely rare in early post-infarction pericarditis [4]. Nonetheless, Oliva et al. [15] described two types of T-wave evolutions characteristic of infarct-associated pericarditis – T-waves that remain persistently positive 48 hours or more after infarction, and the pattern of premature reversal of initially inverted T waves to positive deflections. Nagahama and co-workers [16] observed PQ-segment depression in a minority of patients with infarct-associated pericarditis. In our patients no characteristic pattern of ECG changes could be found.

Echocardiographic examination is very useful for the detection of pericardial effusion, but its detection should not be used as a criterion for diagnosis [17]. The hemodynamic mechanism is the major factor contributing to the occurrence of pericardial effusion in the absence of a pericardial rub, whereas an increase in capillary permeability and excessive fluid exudation through the irritated epicardial surface are mechanisms associated with pericardial effusion with a pericardial rub in the early phase of acute MI [18]. Although congestive heart failure is significantly associated with pericardial effusion, it is not observed more frequently in patients with pericarditis [2,17,19]. According to several studies, the incidence of pericardial effusion detected by serial echocardiographic studies ranged from 25 to 63% of patients with transmural infarction not receiving thrombolytic treatment [2,17,19,20]. Widimsky and Gregor [21] reported a 43% incidence of pericardial effusion after Q-wave myocardial infarction, which was similar for the patients treated and those not treated with thrombolysis. In our study we found a 6.9% incidence of pericardial effusion in patients treated with thrombolytics. This reduction in the incidence of pericardial effusion may be explained by the use of thrombolytic agents, which allows early reperfusion and prevents progression to transmural necrosis. These factors are consistent with an improvement in the hemodynamic state and a reduction in the epicardial irritation and fluid exudation.

According to the MILIS Study [10] the patients with MI-associated pericarditis who did not receive thrombolytic treatment sustained more serious myocardial damage compared to those without pericarditis, as evidenced by a larger infarct size (cardiac enzymes), a lower left ventricular ejection fraction and a greater incidence of congestive heart failure. Pericarditis was reported more commonly following anterior myocardial infarction [8,22]; however, other investigators [4,13] did not find that infarct location predicts the development of pericarditis. Neither in our study did we find any significant difference in the incidence of pericarditis among the patients with anterior or inferior wall MI.

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