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Reviews

Pericardial Involvement in Acute Myocardial Infarction in the Post-Thrombolytic Era: Clinical Meaning and Value

ERNESTO CORREALE, M.D., ALDO P. MAGGIONI, M.D.,* SALVATORE ROMANO, M.D., VINCENZO RICCIARDIELLO, M.D., ROSSANO BATTISTA, M.D., EUGENIO SANTORO, M.S.*

Ospedale Civile di Caserta and *Istituto di Ricerche Farmacologiche "Mario Negri," Milan, Italy

Summary: Pericardial involvement (PI) in acute myocardial infarction (AMI) is a complication usually considered benign and has therefore received less attention than those more severe. It may be easily missed because it presents few symptoms and signs, which in turn may be confused with those of AMI. Its pathophysiology, diagnosis, and pitfalls are discussed. The GISSI-1 trial revealed a marked reduction of PI in the group treated with thrombolysis. This unexpected finding was later confirmed by the GISSI-2 trial and by other studies, drawing attention to its meaning. Data from the GISSI as well as from other studies have been reviewed and seem to indicate that PI is associated with larger AMIs and with a significant increase in 6- and 12-month mortality. This may be attributed to the consequences of late remodeling of a large infarction. These findings lead to the conclusion that PI should be granted more attention, and that it might identify patients with a poorer long-term outcome.

Key words: pericarditis, acute myocardial infarction, site of infarction

Introduction

Thrombolysis in acute myocardial infarction (AMI) is a milestone in the progress of cardiology. Not only has it improved the outlook and outcome of patients with AMI, but it has also contributed to the understanding of its epidemiology and pathophysiology. Furthermore, large-scale clinical trials[1, 2] conducted in this field have provided data particularly useful in clinical medicine on events related to the AMI syndrome, such as paroxysmal ventricular fibrillation,[3] stroke,[4] and pericardial involvement (PI).[5] With respect to the latter, the GISSI–1 trial showed a marked reduction of PI in the streptokinase (SK)-treated group compared with the control group, namely 6.5 versus 12.2%. This finding has been confirmed by other trials, although its consequences have been overlooked or underestimated.[1, 2, 6]

Pathophysiology

Pericardial diseases are commonly grouped under the term pericarditis; this has been considered an improper generalization, particularly when trauma, drug toxicity, or AMI are the cause.[7] Pericardial involvement should be a more appropriate definition for those conditions. Recently, however, an inflammatory response has been found to be present also in these cases, and their pathophysiology seems to indicate a complex pathway.[8] A leukocyte-endothelial interaction and the activation of granulocytes have been deemed important elements of the inflammatory response induced by ischemia and reperfusion.[8]

Regardless of the etiology, the pericardium is known to react to any acute aggression in the same manner, namely, with exudation of fluid, fibrin, or cells, or any combination thereof.

The type of fluid or cells depends on the cause of the PI, and the final outcome varies accordingly.

The first response of the pericardium to any injury commonly is the appearance of fibrin on its surface. A fibrinous reaction of the pericardium is said to occur in virtually all patients with an AMI, even if its clinical manifestations may be absent. It may be localized over the transmural necrotic area, and, less commonly (10–20%), it may involve the pericardium more diffusely.[9] Constriction is rare and usually follows hemopericardium, especially after postmyocardial infarction syndrome.

Address for reprints:

Ernesto Correale, M.D.
GISSI Coordinating Center
Istituto di Ricerche Farmacologiche "Mario Negri"
Via Eritrea, 62
20157 Milano, Italy

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The intact pericardium does not react to serum or blood in its cavity; however, fibrous adhesions may occur: in the presence of lesions on the pericardium, in the presence of microorganisms, if the lipidic components of lysed erythrocytes are injected in the pericardial cavity. All these events are indeed uncommon in AMI.[10] Therefore, PI in AMI usually heals without leaving changes that influence the outcome per se.

The pathogenesis of fibrinous PI in AMI has not yet been well defined, but it has been attributed to damage to the microcirculation due to capillary microthrombosis, occlusive swelling, obstruction caused by platelet aggregation, and disruption of the integrity of the endothelial permeability barrier. All of these events may occur during AMI.[11]

Diagnosis and Pitfalls

The diagnosis of PI in AMI is based principally on the history and physical examination and much less on instrumental evidence.[12] For instance, although echocardiography provides important and unique contributions in the detection of pericardial adhesions, constrictions, and calcifications, it is not very useful in the PI following AMI, where these events are infrequent.

Pericardial effusion, once considered rare in the PI after AMI,[13, 14] was discovered by echocardiography in only 25–41% of cases, and quantified as slight in 88% and moderate in 12% of these patients.[15, 16] Furthermore, it may not necessarily be due to the inflammation, for example, it may be due to noninflammatory hydropericardium with congestive failure.

Also the typical electrocardiographic (ECG) findings of acute pericarditis are not considered particularly useful in the diagnosis of PI, because they are usually masked by the AMI changes.[12]

Recently, however, an unusual evolution of the T waves has been reported when an AMI is complicated by PI. This evolution is expressed as either persistently positive T waves ≥48 h after AMI (67%) or premature gradual reversal of inverted T waves to positive deflections (33%).[17–21]

The true value of these findings needs to be confirmed as they may also occur after a cardiopulmonary resuscitation, a very small infarction, a new ischemia, or a reinfarction.

Pericardial involvement in AMI is not always easily detectable when the pain and the rub are neither typical nor clear. In these cases, the correct diagnosis is possible only if the probability of its occurrence is considered and when one keeps in mind that its symptoms and signs are transitory and often short-lived and should therefore be looked for with frequent controls, especially in the first 2 or 3 days; and that the transitory nature of a nontypical rub may not distinguish PI from the murmur of mitral regurgitation, as this also may disappear with the ischemic regression.

An important diagnostic symptom is the typical breath-related chest pain cutting deep inspiration.

This subjective symptom may become an objective sign when the patient, without mentioning it, manifests the inspiratory pain by an objective facial contraction. This could be defined a "symptom-sign," as it is an objective element with the same diagnostic value of the typical pericardial rub. It can confirm the presence of PI, prove that a nontypical rub mimicking a murmur is actually a rub, and, in the absence of a persistent rub, it can lead to more frequent auscultations in order to discover a transitory rub that would otherwise be missed. In our experience, this inspiratory finding is often the only manifestation of PI and should therefore be sought and evaluated with care.

Although PI is a well-known complication of AMI, it has not received due attention in clinical practice, particularly as a prognostic sign, probably because of its usually favorable in-hospital prognosis, unlike that of other early complications, such as residual ischemia, reinfarction, heart failure, or life-threatening arrhythmias.

Literature Review

A review of the literature shows a dearth of evidence on pericardial disease and particularly on PI in AMI.

In the studies carried out in the prethrombolytic period,[12, 22–24] the size of which generally was at most of the order of a few hundred patients, several questions remained unanswered and the significance of PI appeared on the whole unclear.

Prethrombolytic Era

In the early studies, by and large not specifically concerned with PI, its prevalence varied widely, ranging from 7.3 to 43%.[25]

This variation is due to the adoption of different diagnostic criteria, to the possibility of its being missed because of its short duration, and to the possible misinterpretation of its symptoms and signs.

If only studies which adopted explicit diagnostic criteria for PI are considered, namely, a rub looked for repeatedly and heard by at least two observers, the estimated incidence is around 11.5%.[12, 16, 22–24] In all the studies of the prethrombolytic era, the rub was found mostly in men within the second and third day and, in some studies, more commonly in anterior wall AMIs. More tachyarrhythmias were found in patients with PI14,[14, 22–24] and a greater occurrence of heart failure was reported by most,[12, 14 22, 24] if not by all.[23] Arrhythmias have been shown to be due to myocardial rather than pericardial involvement.[26]

The ejection fraction (EF) was found lower on admission in patients with PI. After 10 days it was still lower,[24] indicating a poorer ventricular function in patients with PI.

During the past 10 years, the literature on PI in AMI has continued to provide data on different aspects of the problem, such as the differential diagnosis of pericarditis and AMI,[18–29] the infrequency of large effusions in the PI of AMI and of the need for specific therapy,[30] the uncommon hemorrhagic nature of these pericardial effusions,[31] and the absence of con-

traindications to anticoagulant therapy when indicated for the treatment of the AMI.[30,32,33]

A striking and remarkably consistent finding of almost all the studies carried out in the prethrombolytic period on patients with PI has been higher Killip classes, greater myocardial damage, and poorer ventricular function, albeit unaccompanied by an increased in-hospital mortality.[12]

Three facts lead to the hypothesis that PI in AMI may be a sign of greater myocardial damage and therefore of poorer outcome. First, PI in AMI is considered a sign of a transmural infarction;[34] in fact, PI has been found at autopsy in patients with a transmural infarction,[35] and in only 5.6% (3/53) of patients with subendocardial infarctions.[36] Second, all the studies of the prethrombolytic period showed indirect signs of greater myocardial damage and poorer ventricular function in patients with PI, namely, pump failure, arrhythmias, and low EF. This was subsequently confirmed by the large trials of the thrombolytic era.[12,14,22–24] Third, large scale trials in patients treated with and without thrombolytic drugs not only proved that thrombolytic drugs reduce mortality and ventricular dysfunction, but also that they reduce the incidence of PI by 50%.[1,2,6]

Despite these three facts, no increase in mortality has been observed in patients with PI.[12,22,23] This paradox was so striking that the importance and usefulness of a longer follow-up was suggested by Guillevin and Valere.[23] To our knowledge, in the prethrombolytic era no follow-up study was carried out in a reasonable number of patients to establish the long-term outcome of those presenting with this apparently innocent complication.

Only one study of the prethrombolytic period had a 6- and 12-month follow-up of 703 patients, showing a borderline statistically significant difference in hospital mortality with the exception of a small subgroup of patients with non-Q AMI. At 12 months, the mortality rate of patients with and without PI was 18 and 12%, respectively ($p = 0.055$), 17 versus 14% in the Q-wave AMIs, and 28 versus 8% ($p < 0.01$) in the non-Q wave AMIs.[24] Larger numbers were needed for statistically significant results.

Thrombolytic Era

The large trials on thrombolytic therapy conducted on thousands of patients have contributed to our knowledge with new and interesting data. These trials have confirmed many results that had been reported previously on fewer patients and without a control group. A new and unexpected finding has been PI reduction by over 50% among patients treated with thrombolytic drugs.

Two studies[5,33] focused on this striking finding of the thrombolytic trials. Wall *et al.*[33] analyzed the data of an uncontrolled series of 810 patients to evaluate the incidence and outcome of PI. All patients were treated with thrombolytic therapy for their AMI. Pericardial involvement was diagnosed in the presence of a pericardial rub. Its incidence was low (5%), being about half the rate reported in the studies on patients without thrombolysis and similar to that found in the group of thrombolyzed patients in the large trials. The comparison of patients with versus those without PI showed that the former had a lower EF (45 vs. 51%, $p = 0.002$), a higher frequency of pump failure (83 vs. 57%), a higher frequency of anterior wall location of the AMI (53% of cases, $p = 0.002$), a higher frequency of triple-vessel disease, a more common involvement of the left anterior descending coronary artery, more severe coronary disease (33 vs. 17% had triple-vessel or left main disease), and a higher in-hospital mortality (15 vs. 6%, $p = 0.056$). When the left ventricular damage was evaluated by global EF, its degree predicted the presence of a rub.

All these findings confirmed those of the previous studies, indicating a greater extension of the infarction and a poorer ventricular function in patients with PI.

An interesting finding in this study, confirmed by others,[37] was the absence of hemopericardium and tamponade in all patients, regardless of their receiving aggressive treatment with concomitant anticoagulant and antiplatelet therapy. This observation is useful in clinical practice, as it documents that hemopericardium and tamponade during AMI are uncommon events and indicates that a pericardial rub in the course of an AMI is not an absolute contraindication to anticoagulation with heparin. The possibility of echocardiographic control makes this kind of therapy even safer.

The GISSI investigators[5] revisited the GISSI-1 and GISSI-2 data on PI in AMI to describe the epidemiology of PI in patients treated with and without thrombolysis, to ascertain whether PI is a marker of infarct size, and to establish whether pericardial involvement is an independent prognostic risk factor for in-hospital and long-term mortality (Table I).

The GISSI-1 trial enrolled 11,806 patients with AMI, treated with and without streptokinase (SK) within 12 h of the event, to evaluate its efficacy primarily on in-hospital, 6- and 12-month mortality.

The GISSI-2 trial enrolled 12,381 patients treated with SK or recombinant tissue plasminogen activator (rt-PA) within 6 h of the event. Its main end point was to evaluate differences in survival and ventricular function during hospital stay and after 6 months in patients with AMI treated within 6 h of the event with either SK or rt-PA. In both studies the occurrence of PI had to be reported in the clinical record form.

In GISSI-1, the incidence rate of PI in the treated group compared with the control group was 6.5 versus 12.2%, respectively. The 12.2% of the control group confirmed the av-

TABLE I Incidence of pericardial involvement

	Prethrombolytic era	Thrombolytic era	
		GISSI-1	GISSI-2
No thrombolysis	12.7%	12.2%	—
Thrombolysis		6.5%	5.6% SK—6.3% rt-PA

Abbreviations: SK = streptokinase, rt-PA = recombinant tissue plasminogen activator.

erage value (11.5%) found in studies conducted in the prethrombolytic era. The 6.5% found in those treated with thrombolysis confirmed the average 5% found in previous small, uncontrolled studies on thrombolytic therapy.[33]

The GISSI-2 data confirmed these findings, with 5.6 and 6.3% PI rates in patients treated with SK or tPA, respectively.

The overall GISSI data also confirmed the early occurrence of PI with a peak at Days 1 and 2, and lower rates in patients treated early after the beginning of symptoms.

The findings of these trials, indicating PI to be a sign of a greater AMI size,[5] are the significant increasing trend in PI for peak creatine kinase increases from less than fivefold the normal upper limit to 5 to 10, 10 to 20, and over 20 fold; the increasing incidence of PI with the increase in the number of leads with ST elevation, a marker of a larger infarction; the greater incidence of PI in anterior and multisite AMI; the higher incidence of PI in inferior wall AMI with ST depression in the precordial leads, a marker of a larger infarction, compared with that of the inferior wall AMI without ST depression; the very low incidence of PI in patients with ST depression at entry (GISSI-1) and in those with non-Q-wave AMI (GISSI-2); the greater incidence of PI in patients with Killip class 2 to 4 at entry, in those with clinical signs of heart failure during the hospital stay, and in those with reduced left ventricular EF and greater akinetic-dyskinetic score.

It is interesting that the GISSI data confirmed the absence of a significant difference in hospital mortality in patients with and without PI (11.2 vs. 11.5%, respectively, in patients treated with SK and 14.4 vs. 13.2% in the control group of GISSI-1; 9.4 vs. 9% in GISSI-2, where all patients were treated either with SK or rt-PA) (Table II)

However, the 6-month and 1-year follow-up of these two large GISSI trials proved that the incidence of late mortality is significantly higher in patients with PI: 20.3 vs. 13.1% in the SK group and 28 vs. 18.8% in the control group of GISSI-1; 14.2 vs. 12.4% in the GISSI-2 trial.[5]

The absence of a significant difference in hospital mortality between patients with and without PI is not surprising, since during hospital stay these critical patients are followed very closely and are immediately and properly treated at the first appearance of arrhythmias or signs of left ventricular dysfunction.

This does not always happen after discharge, as the myocardial changes underlying life-threatening arrhythmias and ventricular remodeling and failure require time, and their gradual occurrence may be missed by the patient who may adapt to the gradual onset of his symptoms. This may lead to a point of poor or no response to therapy.

The univariate and multivariate analysis of the GISSI-1 data indicate PI as an independent prognostic index of mortality, but this was not confirmed when more specific indices of myocardial involvement and function were considered in the multivariate analysis in GISSI-2.[5]

This may be explained by remembering that PI in AMI is characterized by fibrin deposition that usually heals completely, does not contribute per se to an unfavorable evolution, and is therefore a sign and not a cause of greater AMI size.

TABLE II Hospital and late mortality

	Thrombolysis		Control	
	PI (%)	No PI (%)	PI (%)	No PI (%)
Hospital mortality				
GISSI-1	11.2	11.5	14.4	13.2
GISSI-2	9.4	9	—	—
Late mortality				
GISSI-1	20.3	13.1	28	18
GISSI-2	14.2	12.4	—	—

Abbreviation: PI = pericardial involvement.

Conclusions

Studies on PI following AMI lead to the following considerations: PI in AMI is a fibrinous pericarditis that usually heals without consequences; bedside symptoms and signs are the best evidence of PI available today; new ECG signs of PI have been described and, if confirmed, may be useful; although an effusion may occur, it rarely leads to tamponade; PI does not contraindicate thrombolytic and anticoagulation therapy with heparin; thrombolysis not only reduces in-hospital and long-term mortality but also reduces the occurrence of PI by 50%; PI in AMI is not an independent risk factor albeit a marker of a larger infarction, with a greater incidence of ventricular dysfunction and of late mortality, and therefore of poorer late outcome; and PI in AMI is an expense-free, easily detectable bedside finding.

Clinical Implications

Pericardial involvement in AMI should be looked for during the first days after an AMI because patients with this complication are those with a larger infarction, greater incidence of left ventricular dysfunction and of late mortality, and because pharmacologic treatments now available may avoid or delay the gradual postinfarction ventricular remodeling occurring after large AMI and possibly causing late lethal arrhythmias, failure, and death.

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