Value of the History in Evaluating Patients for Early Myocardial Ischemia in Observation Chest Pain Centers

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The Percentage Shot Rather than the Forced Shot (Fig. 1)

Myocardial ischemia is usually manifested clinically as chest pain or chest discomfort. There are exceptions to this, such as presenting symptoms of shortness of breath, nausea, vomiting, weakness, profuse sweating, and so forth, but most of the time, the initial presentation is chest pain or chest discomfort. Myocardial ischemic chest pain may be abrupt, or it may be intermittent over a period of time (prodromal) (Fig. 2).

When acute, it may be abrupt and severe without letting up. The timing of the coronary occlusion appears obvious, but it may also be acute and intermittent, with the episodes increasing in frequency and each lasting for a longer time in a crescendo pattern. Here the timing of the coronary occlusion is less obvious and often misleading. Gauging the tempo of the ischemia allows the physician to diagnose the acuteness of the condition more accurately and to accelerate management of the patient accordingly. This finding is supported in the literature in the excellent study by Robert M. Califf, MD, et al: “From a series of Questions about the quality, severity, frequency, and course of the chest pain (angina) and an examination of the rest EKG, the experienced physician (Master Clinician) forms an overall clinical impression of the tempo of a patient’s coronary disease. A careful summarization of this clinical marker of ischemia in the form of an ‘angina score’ can prove a powerful prognostic tool and may aid clinicians in identifying high-risk patients who are candidates for aggressive therapeutic interventions. The angina score thus simulates the ‘Gestalt’ impression of the experienced physician.”

MARKERS OF MYOCARDIAL ISCHEMIA AND MYOCARDIAL INFARCTION

Serum Markers of Infarction

We know that serum troponin has been an invaluable marker of myocardial necrosis. In most cases, a large troponin rise indicates that significant myocardial damage has taken place, and therapy is often timed too late. With a small rise in troponin, only minimal damage may have taken place (Transient Ischemic Attack [TIA] of the heart). However, this is enough to confirm the myocardial infarction diagnosis and allows the physician to take the patient to the cardiac catheterization laboratory, where further delineation of the coronary artery problem can be undertaken. The latter finding underscores the importance of detecting minimal necrosis ahead of time to allow one to intervene and prevent further damage from taking place. If one were to detect the presence of ischemia before infarction, the outcome would be even better (Fig. 3).

Various serum ischemic markers are now being investigated. The inflammatory marker C-Reactive Protein (CRP), when it is positive in the serum of a chest pain patient before troponin becomes positive, may be just one of these ischemic markers; others include ischemic modified albumin (IMA). A positive cardiolyte nuclear scan (technetium...
sestamibi) in a chest pain patient without previous cardiac damage is also an indication of myocardial ischemia at an earlier stage (Table 1).

Clinical Markers of Ischemia
The presence of ischemia is often found in the history of a patient experiencing chest pain or chest discomfort. An astute diagnostician is able to obtain from the history alone a high index of suspicion of ischemic heart disease and to gauge the tempo of this ischemia in accelerating the patient’s medical care. Thus, intermittent chest discomfort can be interpreted as a clinical marker of myocardial ischemia and allows early treatment to take place. Discovering this ischemia comes at a time when the infarct can be averted (infarctus interruptus).4,5

EVIDENCE BASE FOR PRODROMAL BENEFIT
The data supporting prodromal benefit come from the Global Utilization Streptokinase TPA Outcomes (GUSTO) I study4 and the GUSTO IIB study,5 in which the benefit of prodromal symptoms captured as intermittent chest symptoms can be seen. In most cases, these symptoms have been described as new in onset, chest discomfort rather than chest pain (chest ache, chest pressure, chest burning, chest fullness, and so forth), coming and going, worsening with time, and more easily provoked.

In a substudy of the GUSTO I study, 196 patients were treated at St. Agnes HealthCare (Baltimore, MD); 32 patients in this group were found to have minimal myocardial damage. Nineteen of these 32 patients actually had their infarction aborted, showing no rise in their cardiac enzymes. The mortality of these patients over 30-day, 1-year, and 5-year periods was significantly less than reported in the remaining 164 patients (Table 2). Interestingly, 80% of these 32 patients had presented with intermittent chest discomfort (prodromal symptoms), whereas in the remainder of the patients, the ratio of abrupt to prodromal was 50:50. The incidence of pro-
dromal presentation previously described in the literature is between 40% and 60%.

Perhaps the most surprising finding in this study was the fact that the median time to thrombolytic therapy was 2.72 hours, considerably more than the 1 hour regarded as necessary for good results (1.2% mortality), as seen in the Myocardial Infarction Triage Intervention (MITI) trial. The authors concluded that patients with intermittent symptoms were having intermittent opening and closing of the coronary vessel, and this was protecting the myocardium (preconditioning benefit). The timing of the coronary occlusion appears to be difficult to assess in patients with intermittent symptoms (prodromal), thus providing the rationale for a more aggressive approach in such patients (Fig. 4).

In the GUSTO IIB study, this intermittent chest pain feature was captured in approximately 40% of patients with acute myocardial infarction and in 60% of patients with unstable angina. In most cases, these intermittent symptoms occurred over a 2-week period. It was not surprising to find that the median time for these symptoms was 2 days for patients with acute myocardial infarction and 3 days for patients with unstable angina. However, the mortality was twice as high in the myocardial infarction category (6% vs. 3%). This finding brings up the question whether capturing prodromal patients in the unstable angina stage before the infarction could significantly reduce mortality.

**Other Studies**

Similar findings of prodromal benefit have been found in the In-Time Study in a substudy of 450 patients treated in the United States and Canada. In the Early trial, patients with prodromal symptoms (intermittent) were found to have increased platelet activity, possibly explaining the benefit that is gained from antiplatelet inhibitors. Prodromal patients in studies from Maseri have also been found to exhibit more inflammation. Thus, elevated CRP levels may be used to confirm the clinical impression of ischemia in patients who have not yet had myocardial necrosis as evidenced by positive troponin levels. Discovering the presence of prodromal symptoms in a chest pain patient has allowed the astute clinician to diagnose ischemia before myocardial infarction takes place (Fig. 5). Thus, the importance of this discovery and taking action cannot be overstressed.

<table>
<thead>
<tr>
<th>TABLE 1. Serum Markers</th>
<th>CPK-MB</th>
<th>Troponin</th>
<th>CRP, IMA, Cardiolyte</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usual myocardial infarction (large)</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Microinfarction (small)</td>
<td></td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Ischemia only</td>
<td></td>
<td></td>
<td>+</td>
</tr>
</tbody>
</table>

IMA indicates ischemic modified albumin.

| TABLE 2. Data for Patients Having Aborted MI, Minor Myocardial Damage, and Extensive Myocardial Damage based on CPK-MB |
|--------------------------------------------|--------|----------------|----------------|----------------|
| CK-MB MAXIMA                              | No. Patients | No. (%) with Prodromal Symptoms | Time to Treatment | Deaths: 24 h to 30 d | Deaths: 30 d to 1 y | Total deaths at 5 y |
| Minimal <40 U/L                           | 32     | 26 (81.3%)     | 2.18 hours      | 0 (0%)          | 1 (3.1%)         | 4 (13.8%)           |
| Extensive ≥ 40 U/L                        | 164    | 84 (51.2%)     | 2.67 hours      | 13 (8.0%)       | 8 (4.9%)         | 40 (27.2%)          |
| Aborted MIs                               | 19     | 16 (84.2%)     | 2.72 hours      | 0 (0%)          | 0 (0%)          | 1 (5.9%)            |

*Threatening MIs that are aborted represent the best of EHAC and are seen predominantly in acute MI patients with prodromal symptom presentation. A corollary to this is than an awareness program for prodromal symptom recognition of a heart attack becomes a necessary link to this improvement in acute heart attack care. The EHAC awareness program is based on this approach.

MI indicates myocardial infarction.
The lost art of meticulous in-depth history-taking in chest pain patients needs to be revised, perfected, and well executed to take advantage of the benefits from this early heart attack approach.

“Medicine is learned at the bedside
Not in the classroom”
—Sir William Osler

“I recollect the wisdom of the gray haired clinician who informed me that 90% of the evaluation of a patient with heart disease was concluded when the history was completed.”—Paul Dudley White, MD

“A doctor who cannot take a good history and
A patient who can not give one

**ASTUTE CLINICIANS**

Astute clinicians over the years have emphasized the importance of history-taking in chest pain patients having an acute myocardial infarction. One of these—Bernard Lown, MD, from Brigham and Women’s Hospital in Boston, Massachusetts—is quoted in the *Journal for Myocardial Ischemia*: “To me the art of history taking is a wonderful exercise in communication. The crucial point here is that when you talk to a patient, you are constantly listening, not only with your ears but also with all your powers of observation. You are listening between the lines and you are also carefully watching the patient when he isn’t speaking. You are looking at the eyes, the face, and the body language. You’re aware what angina feels like and when you are, rarely do you make a wrong diagnosis. People tell me that angina is difficult to diagnose. I don’t believe that.” In this article, he points out how the binary questioning of the computer (predicted to replace the history) can often throw you off the
mark, but the astute physician will go back to a question and re-examine it (Appendices A, B).

The Master clinician Thomas J. Ryan, MD, is quoted as saying, “The call for the development of chest pain clinics makes sense if they are used to capture persons who are having prodromal symptoms prior to an ischemic event rather than using them to manage persons who already had an acute ischemic event.”

Other Evidence

Henry McIntosh, MD, refers to a study by Master, Dack, and Jaffe in 1944 describing the prodromal symptoms of angina pectoris in 115 of 260 patients (44%), in which there was a footnote to an article in 1914 describing prodromal symptoms in unstable angina patients. Thus, the historical information we have on prodromal symptoms before myocardial infarction has been described in the literature for almost 100 years.

McIntosh and Bahr submitted a questionnaire to cardiologists throughout the United States to investigate the occurrence and the importance to cardiologists of finding prodromal symptoms as a way of detecting myocardial ischemia early in its course before myocardial infarction took place. They found that 81% of the cardiologists felt that these waxing and waning symptoms were important enough to be discovered, but 80% of them felt that the general public was unaware of this importance. This discovery in itself is a mandate for change.

Eighty-four percent of the cardiologists felt that chest pain and chest discomfort should be considered risk factors for heart attack. To date, this has not been the case. The risk factors that we have are more for the progression of coronary artery disease than for identifying the heart attack in progress.

CHEST PAIN OR DISCOMFORT AS A RISK FACTOR

In the Chain of Survival of the American Heart Association, the first link is cardiac access, and it applies to emergency cases. However, patients who are having mild and stuttering chest symptoms do not perceive this to be emergency enough to go to the emergency room. Recognizing that chest pain is a risk factor will go a long way in recognizing that it is the chest pressure or chest discomfort that is the real risk factor, and that detection can bring about prevention of the heart attack.

USING THE HISTORY AS A CLINICAL MARKER OF ISCHEMIA

When we constantly report that the median time from symptom onset to treatment in patients with acute myocardial infarction remains more than 2.5 hours, we have only ourselves to blame. We need to convey to the public the importance of detecting and preventing impending heart attack at an earlier stage. To improve performance further, we need not only to inform the public (Early Heart Attack Care [EHAC]) but also to provide a user-friendly check-out system at the nearest available hospital (chest pain center). When these are coupled, the EHAC knowledge can become a tool for saving lives through early intervention (Fig. 6).

OBSERVATIONAL CARE

It is true that many chest discomfort patients will turn out not to have ischemic heart disease, but this fact should not stop us from encouraging such patients coming into the hospital earlier to have such symptoms checked out: it is better to be safe than sorry. This is especially true with recent developments in chest pain centers. Chest pain centers in most community hospitals now have an observational unit for patients with low to moderate risk of ischemic heart disease to be checked out. These are not patients who are crashing with severe, prolonged chest pain, but patients who may be having mild chest symptoms that potentially could result in crashing.

The success of the observation unit in sorting out such patients has been documented in peer-reviewed articles reporting that 80% of patients seen in the observation
unit can be discharged home without an inappropriate admission to the hospital. This not only is cost-effective but also saves on telemetry bed usage. The observation unit has also been able to show that observed patients can be safely discharged, in that the missed myocardial infarction has decreased from 4% to less than 0.5%. Thus, observation units in the chest pain center allow the hospital to have a more aggressive cardiac outreach program (EHAC) to alert the public about these early symptoms that need to be checked out.

CONCLUSIONS

With the availability of the observation unit in the chest pain center, the hospital now has the know-how machinery that can deal with the false-positive clinical patients and pick out the true patients with scant myocardial ischemia. Putting emphasis on the clinical history (prodromal symptoms) as a marker of ischemia before infarction will greatly complement this observational practice. Efforts to develop these history-taking skills from master clinicians in cardiology and emergency medicine should be encouraged in these observation units.

Best Possible Scenario

The scenario of a young person coming in with stuttering chest discomfort; clinically being diagnosed as having acute myocardial ischemia without necrosis; being stabilized with antiplatelet and antithrombin medication; and being taken to the cardiac catheterization laboratory, where the culprit lesion is identified and corrected, brings out the best in heart attack care.

Having this patient go home in less than 2 days with his life and his muscle, with his coronary vessel opened, and with more motivation to reduce his risk factors is the hallmark of this developing strategy in chest pain centers. This paradigm shift in the emerging chest pain centers is what is needed to reduce significantly the high mortality seen with the heart attack problem in communities throughout the United States. The strategy of the chest pain center approach is
based on this paradigm shift to a community coronary care unit and making these units universally available throughout the 5000 hospital communities in the United States.

Strangely enough, we have all seen this best possible scenario, but presently it occurs infrequently. We need to find a way to do this more often and more consistently. We can help do this with a return to a full appreciation of the value of history-taking in diagnosing and ascertaining the tempo of the myocardial ischemia before it becomes myocardial infarction. Once it is recognized, we can clearly see the relationship of the vulnerable atherosclerotic plaque to the vulnerable patient before myocardial infarction sets in (Fig. 7).

**Early Heart Attack Care Going Public**

Lastly, history-taking, as discussed, must not remain only in the hands of physicians but extend to nurses and all health care workers and even to the public at large now that the machinery to sort out such patients (observation unit) for early chest symptoms (scant ischemia) exists in chest pain centers in community hospitals throughout the United States. Cardiopulmonary resuscitation knowledge became successful when it went

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**FIGURE 7.** Relationship of vulnerable plaque to the vulnerable patient before myocardial infarction sets in.

**FIGURE 8.** EHAC awareness.

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public; equally important is the EHAC awareness knowledge in going public (Fig. 8). 16,17

REFERENCES


APPENDIX A. Does the Patient With Chest Pain Have Ischemia?

<table>
<thead>
<tr>
<th>Likely</th>
<th>Unlikely</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central chest pain/discomfort</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Diffuse</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Pin-point</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Radiates left shoulder and inner aspect of left arm</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Neck pain with radiation outer left arm</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Responds to nitroglycerin within 5 min</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Does not respond within 5 min</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Same as previous MI pain</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Extreme weakness with attack</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Cold perspiration</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Short of breath (especially females)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Previous epigastric; responsive to antacid</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

MI indicates myocardial infarction.
APPENDIX B. Does the Patient Have Ischemia Before Infarction? Go Deeper Into Possible Prodromal Symptoms?

Have you ever had this same pain or chest discomfort before?
Recently?
When did this take place? How long ago?
Was it different?
Less painful?
More of a pressure or an ache, fullness or burning sensation?
How long did it last?
What relieved it? Did it come back?
What brought it back?
What made it worse?
Did it go away, then come back?
Has it been getting more frequent?
Has it been lasting longer?
Did you tell anyone?
Spouse, friend, call a doctor, call 911?
Were you reluctant to tell someone?
What did you think was happening?
Were you anxious?
What were you concerned about when you were having this?
What was on your mind?
Did you have chores to finish before checking out these symptoms?