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Myocardial Infarction Without Chest Pain

ABSTRACT & COMMENTARY

Source: Canto JG, et al. Clinical characteristics and mortality among patients with myocardial infarction presenting without chest pain. *JAMA* 2000;283:3223-3229.

The objective of this prospective observational study was to determine the frequency with which patients with myocardial infarction (MI) present without chest pain and to examine their clinical characteristics, subsequent management, and mortality compared to those with chest pain. The data were collected as part of the National Registry of Myocardial Infarction 2, which included a total of 434,877 patients with confirmed MI from 1674 hospitals in the United States. Chest pain was defined as any symptom of chest discomfort (including pressure) or arm, neck, or jaw pain.

Of all patients diagnosed with MI, 33% did not have chest pain on presentation to the hospital. MI patients without chest pain were older than those with chest pain (74.2 vs 66.9 years), with a higher proportion of women (49.0% vs 38.0%) and patients with diabetes mellitus (32.6% vs 25.4%) or prior heart failure (26.4% vs 12.3%). All of these differences were statistically significant.

Patients experiencing MI without chest pain were more likely to have a longer delay before hospital presentation (mean 7.9 vs 5.3 hours) and were less likely to be admitted with an initial diagnosis of MI (22.2% vs 50.3%). In addition, MI patients without chest pain were less likely to be treated with thrombolysis or primary angioplasty (25.3% vs 74.0%) and were less likely to receive aspirin, ß-blockers, or heparin. All of these differences were, again, statistically significant. The MI patients without chest pain had a 23.3% in-hospital mortality rate compared to 9.3% in patients with chest pain (adjusted odds ratio for mortality, 2.21 [95%, CI 2.17-2.26]).

■ COMMENT BY STEPHANIE B. ABBUHL, MD, FACEP

This study is a wake-up call to remind us that MI patients without chest pain are common and that we must be careful not to

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INSIDE

withhold standard treatments and to pay attention to the timeliness of diagnosis and therapy in this important group. The number of MI patients without chest pain was a remarkable proportion (one-third), and should remind us of the need to have a low threshold for considering the diagnosis of MI in patients with other complaints. Unfortunately, this study did not collect data about the exact nature of the other presenting complaints. Until the spectrum of other common presentations is defined, public health initiatives will need to emphasize a broader range of symptoms than only chest pain.

It was interesting to note that only one-third of the MI patients without chest pain were diabetics, challenging the common teaching that diabetes alone is the chief risk factor for MI without chest pain. MI patients without chest pain had greater than twice the in-hospital mortality than MI patients with chest pain. This finding is somewhat surprising and was true even after adjusting for differences in age, co-morbidities, and severity of presentation. Only 28% of the higher mortality could be attributed to the lower use of early standard treatments. This study, along with many others, again has demonstrated the lack of concordance between actual ischemia/infarction and the symptom of chest pain, and points to our difficulty in diagnosing and treating an illness that has many faces and can even, at times, be silent. ❖

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Cervical Injury: Identifying the Low-Risk Patient

ABSTRACT & COMMENTARY

Source: Hoffman JR, et al. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. *New Engl J Med* 2000;343:94-99.

This is a multicenter, prospective, observational trial designed to test a decision rule used to identify patients at low risk for blunt cervical injury in whom radiography can be safely excluded. The decision instrument required patients to satisfy the following criteria in order to be classified as low risk: 1) no midline cervical tenderness; 2) no focal neurologic deficit; 3) normal alertness; 4) no intoxication; and 5) no painful, distracting injuries. The instrument was applied prospectively in a large cohort of blunt trauma patients treated at 21 academic and non-academic centers. Radiographs were obtained at physician discretion. More than 34,000 patients were enrolled, of which 818 (2.4%) had cervical spine injury. The decision rule identified all but eight injuries (sensitivity, 99% [95%, CI 98-99.6%]; negative predictive value, 99.9% [95%, CI 99.8-100%]; specificity, 12.9%; positive predictive value, 1.9%). Only two of these patients had clinically significant fractures, and only one required surgical intervention. Using this decision rule, radiography could have been safely avoided in 12.6% of patients.

■ COMMENT BY MICHAEL A. GIBBS, MD, FACEP

Missed cervical spine injury can have catastrophic results for both patients and physicians. Early identification of these injuries should be an absolute priority. While a philosophy of liberal radiographic imaging will accomplish this goal, it comes at the expense of a large number of negative radiographs, delays in evaluation, unnecessary exposure to radiation, and staggering cost. Using a decision rule to identify the low-risk patient in whom radiographs can be excluded safely makes good clinical sense. While several prior studies have proposed using this strategy, none have had the statistical power to validate the conclusions. The results of the

National Emergency X-ray Utilization Study (NEXUS) represent a milestone of academic accomplishment in emergency medicine. A trial of this size is unlikely to be duplicated, and the proposed decision rule will rapidly become an integral part of our practice. The clinician should keep in mind that several of these criteria are subjective. "A little bit" of cervical tenderness should be considered a positive finding, as should "a couple of beers." "Distracting injury" is perhaps the most subjective of these criteria, and will vary widely from patient to patient. These criteria should be used, but use them with care and attention to detail. ❖

TTP Now Associated with Clopidogrel

ABSTRACT & COMMENTARY

Source: Bennett CL, et al. Thrombotic thrombocytopenic purpura associated with clopidogrel. *N Engl J Med* 2000;342:1773-1777.

Beyond aspirin, several new oral antiplatelet agents have arrived on the market in the United States. Two thienopyridine derivatives, ticlopidine and clopidogrel, have been utilized for secondary prevention of cerebral ischemia and stroke in aspirin failures or in those patients who are intolerant to aspirin (perhaps their most common use to date); they also are used as aspirin alternatives in acute coronary syndromes and peripheral vascular disease.[1,2] Until recently, thrombotic thrombocytopenic purpura (TTP) had been associated with ticlopidine use (with a frequency of up to 1 case per 5000 patients), but not with clopidogrel, despite the structural similarity of the two agents.[3]

In this study, active surveillance on the part of blood bank medical directors, hematologists, and the manufacturers of clopidogrel was conducted for two years. This resulted in the detection of 11 cases of TTP which were associated with clopidogrel. Inclusion criteria required previous or current clopidogrel administration when the diagnosis of TTP was made; 10 of 11 patients stopped taking the drug when the syndrome developed, while the other had discontinued clopidogrel three weeks before the onset of TTP. Only one patient had been on ticlopidine previously, and that was two years prior to onset of TTP, without any history of TTP before the index case.

There was one fatality from TTP; eight others had complete recovery after discontinuation of clopidogrel and plasma exchange therapy, and two had relapses up to seven months after the onset of the syndrome, with recovery after further plasma exchange. All but one case of TTP occurred within two weeks of clopidogrel therapy initiation. Five of 11 patients with clopidogrel-associated TTP also were taking "statin" cholesterol-lowering agents (either simvastatin or atorvastatin)—the authors acknowledge that TTP has been reported previously in one patient on simvastatin. The mechanism by which clopidogrel might cause TTP was deemed unclear.

■ COMMENT BY RICHARD A. HARRIGAN, MD, FAAEM

As emergency physicians, we see patients every day who are on a wide variety of medications, most of which we did not prescribe for them, and may not commonly prescribe at all. Ticlopidine and clopidogrel are examples of two drugs we see, yet rarely prescribe. However, because toxicology is more within our bailiwick than that of any other specialty, we must be aware of the adverse effect profile of drugs both in overdose and nonoverdose situations. The symptoms of patients presenting to the ED always must be viewed in light of what medications they currently are taking. Thus, this report of an association between clopidogrel and TTP should be kept in mind. TTP is a life-threatening disease characterized by thrombocytopenia, microangiopathic hemolytic anemia, fever, renal dysfunction, and neurologic changes; any of these components of the syndrome may contribute to the patient's chief complaint, or may be discovered unexpectedly on laboratory analysis.

Ticlopidine and clopidogrel act by inhibiting adenosine diphosphate (ADP)-induced platelet aggregation. Ticlopidine has suffered in comparison with clopidogrel, due to the following characteristics: 1) delayed antithrombotic effect; 2) twice daily dosing; and 3) more adverse effects (gastrointestinal and hematologic).[2] Clopidogrel has a more rapid onset of platelet inhibition, once-a-day dosing, and (until now) a better safety profile, which has led many to favor its use over ticlopidine.[2-4] This report casts the comparative safety of clopidogrel in doubt. The accompanying editorial congratulates the authors' aggressive approach to soliciting these adverse effects;[3] rather than publishing only reported adverse effects, their active surveillance endeavor led to the gathering of more cases. The association of ticlopidine with TTP, coupled with the structural similarity of the two agents, should spark an investigation (as it did), rather than relying on passive surveillance to detect serious adverse effects.[3] ❖

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Cranial CT for Minor Head Trauma

ABSTRACT & COMMENTARY

Source: Haydel MJ, et al. Indications for computed tomography in patients with minor head injury. *New Engl J Med* 2000;343:100-105.

This was a prospective, multicenter study designed to develop and validate a set of clinical criteria that could be used to identify patients with minor head injury (MHI) who do not require cranial computed tomography (CT). In the first phase of the study, the authors recorded clinical findings in 520 consecutive patients with MHI, defined by a loss of consciousness (LOC) and, on arrival to the emergency department (ED), a Glasgow Coma Scale (GCS) score of 15 and a normal neurologic examination. All patients underwent cranial CT. Using recursive partitioning, a set of clinical criteria was derived to identify all patients who had abnormalities on CT. In the second phase, the sensitivity and specificity of these criteria were evaluated in a cohort of 909 patients.

Of the 520 patients in the first phase, 36 (6.9%) had positive scans. All patients with positive CT scans had one or more of seven clinical findings: headache, vomiting, age older than 60 years, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicles, or seizures. Among the 909 patients in the second phase, 57 (6.3%) had positive scans. In this group, the sensitivity of the seven findings combined was 100% (95%, CI 95-100%). All patients with positive CT scans had at least one of these findings.

■ **COMMENT BY MICHAEL A. GIBBS, MD, FACEP**

Each year, more than two million Americans present to the ED following head trauma. Current recommendations suggest that neuroimaging be performed in the vast majority of these patients. In the population with MHI (i.e., history of LOC or amnesia, GCS = 15), the yield of this conservative approach is quite low: Between 6% and 9% will have a "positive" CT, and less than 1% will require neurosurgical intervention.[1-3]

In keeping with other recently developed clinical decision rules, Haydel and colleagues have developed a straightforward set of criteria that can be applied at the bedside. While prospective validation at other centers would be helpful, the high negative predictive value (100%) of this rule for excluding intracranial abnormalities in this patient population is impressive. ❖

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Special Feature

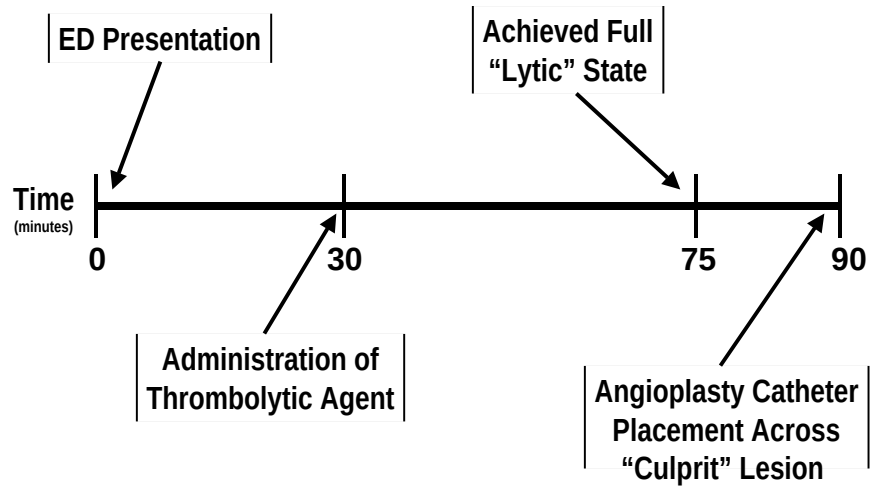
Reperfusion Therapy in Acute Myocardial Infarction: Thrombolysis vs. Primary Angioplasty

By William J. Brady, MD

Re-establishing perfusion in the infarct-related coronary artery with the use of thrombolytic therapy (in essence re-opening the infarct-related artery) increases the opportunity for salvage of ischemic myocardium and, consequently, reduces morbidity and mortality. Thrombolytic therapy unequivocally improves survival in patients presenting with ST segment elevation (STE) acute myocardial infarction (AMI). Numerous, large thrombolytic investigations have supported this statement; a meta-analysis of nine major investigations of thrombolytic therapy in patients with AMI demonstrated approximately a 20% reduction in short-term mortality with the use of such agents compared to placebo. When all patient groups were pooled, a reduc-

Figure

Thrombolytic Agent vs. PTCA—Time Issues in the Treatment of AMI



Thrombolytic Agent vs. PTCA—Time Issues in the Treatment of AMI. The patient presents to the ED at time "0." In the thrombolytic scenario, the patient receives drug at 30 minutes, yet does not reach a full "lytic" state for an additional 45 minutes; cumulative time is now 75 minutes from presentation. In the primary PTCA scenario, the patient is transferred to the catheterization laboratory with placement of the angioplasty catheter across the culprit lesion by 90 minutes from ED presentation.

tion of 18 deaths per 1000 patients treated was found.

While thrombolytic therapy has widespread availability and a proven ability to improve coronary flow, limit infarct size, and improve survival in AMI patients, many individuals with acute infarction are not considered suitable candidates for such treatment. Patients with absolute contraindications to thrombolytic therapy, certain relative contraindications cardiogenic shock, and unstable angina may be ineligible to receive thrombolytic therapy. The benefits of administering prompt reperfusion therapy to these patients, as well as other limitations of thrombolytic therapy, have led many clinicians to advocate percutaneous transluminal coronary angioplasty (PTCA) as the primary therapy and treatment of choice for AMI. Primary PTCA has many theoretical advantages over thrombolysis, including an increased number of eligible patients; a lower risk of intracranial bleeding; a significantly higher initial reperfusion rate; an earlier definition of coronary anatomy, with rapid triage to surgical intervention; and risk stratification allowing safe, early hospital discharge.

Results of Comparative Trials

Several trials of varying sizes comparing primary PTCA with thrombolysis have been reported in the past 10 years. Despite a clear and consistent benefit of primary PTCA in restoring patency of the infarct-related artery, differences in mortality in the individual trials were difficult to evaluate because of the relatively small sample sizes in the studies. More recent studies, however, suggest that PTCA is a superior therapy for the AMI patient. The PAMI trial enrolled 395 patients who were randomly assigned to primary PTCA vs. t-PA.[1] Compared with standard-dose t-PA, primary PTCA reduced the combined occurrence of nonfatal reinfarction or death, was associated with a lower rate of intracranial hemorrhage, and resulted in similar left ventricular function. The results of the Netherlands trial indicated that primary angioplasty was associated with a higher rate of patency of the infarct-related artery, a less severe residual stenotic lesion, better left ventricular function, and less recurrent myocardial ischemia and infarction than in patients receiving streptokinase.[2]

In a substudy of the GUSTO IIb trial, the authors randomly assigned 1138 patients with AMI to either primary PTCA or accelerated t-PA.[3] The composite end point of the study included death, nonfatal reinfarction, and nonfatal disabling stroke, all occurring within 30 days of the AMI. Of those patients assigned to primary PTCA therapy, 83% were candidates for such treatment and underwent angioplasty 1.9 hours after emergency department (ED) arrival for a total elapsed time from chest pain onset to therapy of 3.8 hours. Ninety-eight percent of the patients assigned to thrombolytic therapy received t-PA 1.2 hours after hospital arrival. The occurrence of the composite end point was encountered significantly less often in the PTCA group (9.6%) compared to the t-PA group (13.7%) at 30 days. When the individual components of the composite end point at 30 days were considered separately, the incidence of death (5.7% vs 7%), reinfarction (4.5% vs 6.5%), and stroke (0.2% vs 0.9%) occurred at statistically similar rates in both treatment groups—PTCA and t-PA—respectively. Additional work in the form of a meta-analysis by Weaver and colleagues reviewed 10 major studies comparing thrombolysis to primary PTCA in more than 2600 patients.[4] The 30-day mortality was found to be significantly lower in the PTCA group (4.4%) than in patients treated with thrombolytics (6.5%). Primary PTCA also was associated with a significant reduction in total stroke rate and hemorrhagic strokes.

The longer-term results of primary PTCA, however, are less clear. The GUSTO IIb study showed no overall mortality advantage of primary PTCA at six months.[3]

Conversely, two-year follow-up from the PAMI trial found a significant reduction in hospital readmission, recurrent ischemia, target vessel revascularization, and reinfarction, with a trend toward a reduction in mortality in the PTCA group, compared to treatment with thrombolysis.[1]

Time Is Important

It is widely accepted that the early restoration of perfusion in the AMI patient limits myocardial damage, preserves left ventricular function, and reduces mortality; such restoration may be accomplished by either thrombolytics or PTCA. The rapid application of reperfusion therapy is a must in the patient with STE AMI. Emergency and cardiovascular physicians must consider many factors regarding early reperfusion treatment decisions when managing the AMI patient. While primary angioplasty may offer improved outcome over thrombolysis, PTCA must be applied early without prolonged delay. It must be stressed that PTCA should be initiated within 90 minutes of arrival at the hospital ED.[5,6] *(See Figure on pg. 29.)* If the time required to mobilize staff and arrange for PTCA is prolonged (i.e., greater than 90 minutes to balloon catheter inflation across the culprit coronary lesion), then thrombolysis is preferred, assuming the patient is a candidate for thrombolysis. Delays beyond this time period are unacceptable if the patient originally was a thrombolytic candidate. Prior agreement between the ED and the cardiovascular physicians at institutions with angioplasty capability must be obtained so that PTCA consideration will not introduce further delays in thrombolytic drug administration. Such cooperation has been shown to limit additional delays in the administration of thrombolytic agents in AMI patients who are considered for PTCA.

The emergency physician must consider several related issues. First, the literature base to answer this question is somewhat heterogeneous (e.g., differing therapies, study sites, outcome measures), making absolute, all-encompassing recommendations impossible and thus providing fuel for further debate. Second, the question of technical expertise should be considered. In the GUSTO-IIb trial, the vast majority of physicians performed at least 75 procedures per year;[3] these results may not generalize to smaller-volume centers with less-experienced operators. Third, the urgent transfer of a thrombolytic-eligible AMI patient to another institution for primary PTCA is not recommended until thrombolytic therapy is initiated; the delay in restoring perfusion in such a patient is not acceptable in most instances. If the patient is an acceptable candidate for thrombolysis, the thrombolytic agent should be started before or during transport to the receiving hospital. ❖

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CME Questions

19. In a patient with AMI, primary PTCA is likely to be a superior therapy when compared with thrombolysis if:
a. the patient has no history of coronary artery disease.
b. primary PTCA can be performed within 120 minutes of patient arrival.
c. the patient can undergo primary PTCA within 90 minutes of arrival.
d. the balloon catheter can be placed across the culprit lesion within 90 minutes of arrival.

20. All of the following are considered low-risk criteria for cervical spine fracture, *except*:
a. no evidence of intoxication/normal level of alertness.
b. lack of a painful, distracting injury.
c. age younger than 50 years.
d. absence of posterior midline cervical tenderness.
e. absence of a neurologic deficit.

21. In a large, prospective study comparing MI patients with and without chest pain, all of the following statements are true *except*:
a. Of all the patients with MI, 33% did not have chest pain on presentation to the hospital.
b. MI patients without chest pain had greater than twice the in-hospital mortality than patients with chest pain.
c. MI patients without chest pain were less likely to receive thrombolysis or primary angioplasty than those with chest pain.
d. Almost all of the MI patients without chest pain had diabetes.

22. Thrombotic thrombocytopenic purpura is characterized by all of the following *except*:
a. renal dysfunction.
b. fever.
c. microangiopathic hemolytic anemia.
d. low platelet count.
e. pulmonary fibrosis.

23. Clopidogrel:
a. inhibits platelet function via cyclooxygenase inhibition.
b. is dosed twice daily.
c. has been associated with thrombotic thrombocytopenic purpura.
d. has a slower onset of therapeutic action than does ticlopidine.

24. Which of the following is the correct group of characteristics in the study by Haydel et al for predicting the need for cranial CT in minor head trauma (i.e., having at least one of the following)?
a. Headache, vomiting, age older than 60, intoxication, short-term memory deficit, evidence of trauma above the clavicles, or seizures
b. Headache, nausea, age older than 60, intoxication, short-term memory deficit, evidence of trauma above the clavicles, or seizures
c. Headache, vomiting, age older than 20, intoxication, short-term memory deficit, evidence of trauma above the clavicles, or seizures
d. Headache, nausea, age older than 20, intoxication, short-term memory deficit, evidence of trauma above the clavicles, or seizures

25. Which of the following demographic characteristics was more common in MI patients presenting without chest pain?
a. Female gender
b. Older age
c. Having diabetes
d. All of the above

What is Beat X?

By Ken Grauer, MD

Figure. Simultaneous rhythm strip recording of leads I and II. What is beat X?

Clinical Scenario: The Figure shows a rhythm strip with *simultaneously* recorded leads I and II. What is beat X? What is unusual about this beat? Why is the PR interval of the following beat prolonged?

Interpretation: The underlying rhythm is sinus bradycardia and arrhythmia. Beat X is a premature ventrucular contraction (PVC). Although the amplitude of this beat is greatly reduced (and easy to overlook) in lead I, confirmation of its true etiology is readily apparent from inspection of simultaneously recorded lead II, where this beat is obviously wide and very different in appearance from normal sinus complexes. QRS amplitude in a given lead may be null (or almost so, as in this case) when the mean vector of a beat is oriented perpendicular to the lead being monitored.

The second unusual aspect of beat X is that there is no compensatory pause following this beat, as usually occurs because the premature ventricular complex renders the AV node refractory to conduction of the next sinus impulse. Instead, the R-R interval containing the PVC in this tracing is barely longer than the R-R interval of the underlying sinus rhythm. Such PVCs are said to be "interpolated." The final finding of interest is the presence of *concealed* conduction, which produces PR interval prolongation in the beat following the PVC (seen best in lead II). The term concealed conduction is used when an ECG finding is seen that is not explained by the surface ECG. Instead, one has to postulate that the reason the PR interval of the third sinus beat is prolonged reflects the greater amount of time needed for the atrial impulse to penetrate an AV node rendered partially refractory from the preceding ventricular beat. ❖

In Future Issues:

Combination of Glycoprotein IIb/IIIa Inhibitors and Thrombolysis in Acute MI