Prehospital Management of Acute Myocardial Infarction: —History Taking, Physical Examinations, and Laboratory Procedures—

Kinji ISHIKAWA

Professor, First Department of Internal Medicine, Kinki University School of Medicine

Abstract: Thirty percent of deaths due to acute myocardial infarction occur before patients arrive at a hospital; thus prehospital care plays a critical role in the management of this disease. Accurate history taking is the most important step in this management, as about two-thirds of all cases of acute myocardial infarction show prodromal symptoms prior to onset. Characteristic symptoms are chest discomfort, including sensations of strangulation and pressure or pain in the chest. These are often accompanied by cold sweats, nausea, vomiting, and even fear of impending death. Acute myocardial infarction should be suspected if these symptoms continue for more than 30 minutes. Physical findings may vary from slight pallor of face and an expression of agony and cold sweating in mild cases, to cyanosis or even shock in severe cases. However, it is important to recognize that these signs and symptoms are often absent. When acute myocardial infarction is suspected, an electrocardiogram should be taken immediately; blood samples should be taken to assess the white blood cell count, serum creatine kinase activity, and myocardial troponin T levels; and an echocardiogram should be obtained. Ideally, these measures should be completed within 10 minutes after a patient's arrival at a hospital.

Key words: Acute myocardial infarction; Predromal unstable angina; Silent myocardial ischemia; Infarct size; Myocardial troponin T

Introduction

It is estimated that there are 1.5 million deaths from acute myocardial infarction in the U.S. each year — i.e., one every 20 seconds. According to the Ministry of Health and Welfare, there were 31,933 recorded deaths due to acute myocardial infarction in Japan in 1990. More recently, Japan's age-adjusted mortality rate from ischemic heart disease per 100,000 population was...
determined to be approximately one-sixth of the corresponding U.S. figure. Therefore, as the population of Japan is about half that of the U.S., the annual number of deaths from acute myocardial infarction in Japan could be approximately one-twelfth of the U.S. figure, or about 125,000. To reduce this high mortality, it is essential that the patients obtain early diagnosis and treatment.

Importance of Prehospital Care

The mortality rate for myocardial infarction markedly declined with the introduction of reperfusion therapies, such as thrombolysis and percutaneous transluminal coronary angioplasty (PTCA). Although the in-hospital mortality rate had been approximately 20% before reperfusion therapy, it declined to 10% or, by some accounts, to less than 5% after the introduction of reperfusion therapy. At present, therefore, the highest mortality from acute myocardial infarction occurs immediately following the onset of the disease: based on statistical figures from the 1960’s, it is estimated that 30% of patients die before they arrive at a hospital. And since the mortality rate after hospital arrival is on the decline, the proportion of patients dying before arrival must be increasing. Thus, prehospital care for the treatment of acute myocardial infarction, including quick diagnosis and initiation of appropriate measures following the onset of the disease, is crucially important.

History Taking

More than half of acute myocardial infarction cases can be diagnosed through history taking. Even though there are a variety of advanced laboratory examinations available today, an accurate history is fundamental in the diagnosis of this disease.

1. Prodromal symptoms

A third of patients with acute myocardial infarction have a history of stable angina, but experience a worsening of their anginal attacks 1 to 2 weeks prior to the onset of the infarction (worsening unstable angina). Another third do not have a history of angina, but experience anginal attacks for the first time 1 to 2 weeks prior to the infarction (de novo unstable angina). The remaining third have no history of angina, and their first episode of angina leads to myocardial infarction. Thus, unstable angina, which is indicative of rupture of the atheroma in a coronary artery and subsequent formation of a thrombus, occurs as a prodromal symptom in two-thirds of all acute myocardial infarction cases. Infarction occurs when the thrombus is large enough to occlude the lumen of a coronary artery. Consequently, patients with acute coronary syndrome should be encouraged to enter a hospital.

2. Chest symptoms

Symptoms of acute myocardial infarction include painful feelings of strangulation or squeezing and pressure in the chest, which have been described as sensations of “suffocation,” “stinging pain,” “being drilled,” or “burning.” These symptoms occur in an area between the back of the sternum and the left anterior chest, in an area about a size of a fist. In some cases, they appear not in the precordium but in the epigastric region and are sometimes misdiagnosed as gastric ulcer. They are also radiated to the pharyngeal region and jaw in some instances. Some patients experience weakness in the left upper extremity or numbness of its ulnar side. In addition, cold sweats, nausea, and vomiting are observed in approximately half of the patients. A fear of impending death is also a characteristic of the experience.

Symptoms last for 30 minutes or longer in most cases; they may also disappear temporarily and then reappear, repeating a waxing and waning cycle. This phenomenon is presumably caused by transient interruptions in coronary blood flow, which may be due to a cycle of growth and dissolution/reduction of a throm-
bus in a coronary artery, or to the appearance and disappearance of coronary spasms. Symptoms gradually diminish a few hours after onset and disappear within approximately half a day, leaving a mild sensation of pressure in the chest. In some cases, the symptoms completely disappear early after onset, which is indicative of dissolution of the occluding thrombus and the reestablishment of coronary blood flow (spontaneous recanalization).  

3. Silent myocardial infarction

Twenty to sixty percent of myocardial infarctions are detected by chance on an electrocardiogram taken at a periodic medical check-up, or they may be discovered at autopsy. In half of these cases, information about symptoms pointing to the possible presence of myocardial infarction can be obtained by history taking. On the other hand, no such symptoms are observed in the remaining half of these cases. Those with a history of angina, hypertension, or diabetes are more likely to have such silent myocardial infarctions.

4. Other symptoms

Fainting may occur due to bradycardia caused by complete atrioventricular block, which is most frequently observed with inferior infarctions. Fainting can also be caused by malignant ventricular arrhythmias (ventricular fibrillation or ventricular tachycardia). Indeed, ventricular fibrillation accounts for a large fraction of the deaths from acute myocardial infarction occurring within 30 minutes of disease onset. Cardiogenic shock may occur when the size of the infarct is large and there is an episode of left ventricular failure, leading to a decrease in blood pressure (Table 1). If so, peripheral circulatory dysfunction and disturbed consciousness are observed; breathing difficulty and cyanosis are noted in cases where the left ventricular failure results in pulmonary congestion.

### Physical Findings

1. General condition

Although no physical abnormalities are observed in mild cases, the patients often look pale and show an expression of anguish. Patients with shock also experience cold sweats, decreased skin temperature, cyanosis, and disturbed consciousness. Body temperature does not rise on the first day after disease onset, but gradually increases thereafter. If a rise in temperature is noticed immediately following disease onset, other disorders, such as acute pericarditis, should be suspected. Tachycardia is frequently observed due to increased sympathetic nerve activity. Some patients, however, may present with sinus bradycardia due to increased vagal tone, or bradycardia resulting from atrioventricular block. Elevated blood pressure due to increased sympathetic nerve activ-
It is also seen in mild cases. Because decreased blood pressure is observed primarily in severe cases, hypotension at the first medical examination suggests a poor prognosis (Table 2).

2. Pulmonary auscultation

Bubbling rales are heard in patients with pulmonary congestion. Careful auscultation should be performed posteriorly to both the left and right lower lung fields while the patient sits still. The Killip classification (Table 3), which is based on the magnitude of the rales heard, is important for classification of the severity of a myocardial infarction.

3. Cardiac auscultation

The first sound is attenuated on the first day after the onset of acute myocardial infarction. In patients with left ventricular diastolic dysfunction, the third sound is often audible. Papillary muscle dysfunction is suspected when a loud systolic murmur is heard in the area between the left sternal edge in the 4th intercostal space and the apex. Patients with papillary muscle rupture or perforation of the interventricular septum experience thrills. These can be readily diagnosed using echocardiography or Doppler examination. Pericardial friction rubs are heard in 6% to 30% of patients with acute myocardial infarction; detection requires careful auscultation, however, as they are transient and localized.

4. Other findings

Palpation should always be performed on the left and right dorsal pedis arteries of patients with myocardial infarction. The inability to locate these arteries indicates the presence of arteriosclerosis obliterans, which is one of the important factors influencing patient prognosis.

Laboratory Examinations

When acute myocardial infarction is suspected, the tests listed in Table 4 should be performed immediately.

(1) Electrocardiogram (ECG): ECG is the most important examination tool. For that reason, one should strive continuously to optimize one’s ability to use ECG for diagnosing acute myocardial infarction.
(2) **Echocardiography**: Echocardiography is the second most useful examination tool. When abnormal left ventricular wall motion detected by echocardiography matches the extent of the ST segment elevation on an ECG, myocardial infarction is almost certain.

(3) **Leukocytes**: The white blood cell count is an important indicator because it increases within 2 hours after disease onset. One drawback, however, is that it can also increase in diseases other than myocardial infarction.

(4) **Serum creatine kinase activity (CK)**: Elevation of serum CK levels is seen 3 to 4 hours after disease onset, immediately following the increase in the leukocyte count. As with white blood cell counts, determination of serum CK levels takes time because blood samples must be sent to the laboratory for analysis.

(5) **Myocardial troponin T**: Blood levels of myocardial troponin T can be readily detected using a commercially available testing kit. This enables a diagnosis of myocardial infarction to be made within 15 minutes, merely by applying a drop of blood to test paper at the bedside.

**Differential Diagnosis**

Differential diagnosis is sometimes necessary to distinguish chest symptoms caused by acute myocardial infarction from those caused by hypertension, valvular diseases, myocardial diseases, aortic dissection, pulmonary embolism, pneumothorax, gastrointestinal disease, intercostal neuralgia or herpes zoster. Differential diagnosis is also necessary in cases of cardiac neuralgia or chest pain syndrome. In most cases, acute myocardial infarction can be differentiated by careful analysis of the symptoms with the aid of an ECG and a troponin T test.

**Conclusion**

If you see a patient who complains of chest symptoms, acute myocardial infarction should be suspected, and in-depth questioning should be conducted. When you suspect the disease, ECG should be performed immediately, after which blood tests for myocardial troponin T, white blood cell counts, and myocardial CK activity should be performed. Echocardiography should then be carried out while waiting for the results of the blood tests. If the diagnosis is confirmed, treatment measures, such as intravenous thrombolysis, may be indicated. When acute myocardial infarction is suspected, or the diagnosis cannot be confirmed, the patient should be transferred to a hospital with comprehensive disease management capabilities.

**REFERENCES**


