Commentary by Francis L. Counselman, MD, Associate Editor in Chief

Never Too Young to Have a Heart Attack

Case

A 21-year-old woman presented to the ED for evaluation of severe chest pain radiating to her left arm, and associated shortness of breath, nausea, and vomiting. She stated that the pain started 2 hours earlier while she was resting and had become progressively worse. She denied any history of similar symptoms. The patient denied fever, chills, or cough. She stated that she was otherwise in good health and did not take any medication on a regular basis. Regarding her social history, she admitted to smoking one pack of cigarettes a day and drinking alcohol on occasion.

On physical examination, the patient appeared uncomfortable. Her vital signs were: blood pressure, 136/86 mm Hg; heart rate, 102 beats/min; respiratory rate, 22 breaths/min; and temperature, 98.6°F. Oxygen saturation was 97% on room air. The head, eyes, ears, nose, and throat examination was unremarkable. Auscultation of the lungs revealed clear breath sounds bilaterally. The heart examination revealed tachycardia, but with regular rhythm and without murmurs, rubs, or gallops. The abdomen was soft and nontender. No lower extremity examination was documented.

The patient was seen by a physician assistant (PA) in the ED. An electrocardiogram (ECG), complete blood count (CBC), basic metabolic profile (BMP), troponin level, chest X-ray (CXR), and urine pregnancy test were ordered. The patient was given intravenous (IV) fluids and prochlorperazine 10 mg IV. The ECG and CXR were interpreted as normal. The urine pregnancy test was negative, and the remaining blood test results were within normal limits.

The PA believed the patient suffered from gastroenteritis, coupled with anxiety. He discharged the patient home with instructions to drink clear liquids for 24 hours, and take the prescribed prochlorperazine tablets as needed for continued nausea and vomiting.

At home, the patient continued to experience increasingly severe chest pain, shortness of breath, and vomiting. The next morning, she could no longer tolerate the pain and returned to the same ED via emergency medical services.

The patient's history and physical examination remained unchanged from her presentation 16 hours earlier. At this ED visit, the patient was seen by an emergency physician (EP) who, concerned the patient had suffered an ischemic coronary event, ordered repeat ECG, CBC, BMP, and troponin evaluation. The EP also contacted cardiology services, but the cardiologist did not see the patient for several hours. When the cardiologist evaluated the patient and interpreted the ECG, he was concerned for an ST-segment elevation myocardial infarction (STEMI), and activated the catheterization lab.

Unfortunately, the patient had significant myocardial damage, with a resulting ejection fraction of only 10%. She was judged to be a candidate for heart transplantation, and received a left ventricular assist device (LVAD) as a bridge until a suitable donor heart could be identified. One month after implantation of the LVAD, the patient experienced an ischemic stroke that resulted in dense left-side weakness, leaving her confined to bed.

The patient sued the PA, the EP, the hospital, and the cardiologist for failing to identify and treat the acute STEMI in a timely manner. The plaintiff claimed the STEMI began at her first presentation to the ED, and that it should have been diagnosed and treated at that time. The plaintiff further argued that she should at least have been monitored and undergone repeat testing (ie, ECG and troponin level evaluation) at the first visit, stating that if she had received proper treatment, she would not have required an LVAD and therefore would not have had a stroke. The patient also alleged that at the second ED visit, there was a significant time delay before she was taken to the catheterization lab, which resulted in additional myocardial injury.

The defendants argued the patient was appropriately evaluated and treated at the first presentation, and that there was no evidence to suggest an MI. The EP argued that the delay in the patient's care at the second visit was not his fault. All of the parties involved negotiated a settlement in the amount of $6 million in favor of the plaintiff.

Discussion

Myocardial infarction in adults younger than age 45 years is relatively rare, comprising only 2% to 10% of

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all MIs.[1,2] The percentage of MI in patients younger than age 25 years must be even smaller, but no good data are available. In fact, age 40 years and younger is usually an exclusion criteria in many of the multicenter studies involving MI. Women are relatively spared from coronary artery disease (CAD) before menopause, thanks to the cardioprotective effects of estrogen. Young women who do experience an MI usually will have cardiovascular risk factors, especially smoking.

Risk Factors for MI in Young Patients

Cigarette Smoking. When examining common risk factors in young patients who had an MI (defined as patients younger than age 45 years), cigarette smoking is the most common risk factor.[1,2] Between 76% and 91% of young patients with an MI are smokers, compared to only 40% incidence in older patients.[1] It is thought that cigarette smoking produces endothelial dysfunction and can precipitate coronary spasm.[1]

Nonatherosclerotic Etiology. Interestingly, several studies of MI in young patients found a higher incidence of nonatherosclerotic causes of MI in women compared to men.[2] One explanation for this finding is that women experience vasospastic syndromes and hypercoagulable states, secondary to oral contraceptive use or hereditary coagulation disorders.[2] It has also been shown that young women have more active platelets following an MI and experience plaque erosions, rather than the plaque ruptures that occur in men and older women.[2,3]

Hyperlipidemia. Hyperlipidemia is an additional risk factor for MI in the younger adult patient population. In one study of young patients who had an MI, hyperlipidemia was the most important risk factor, in the absence of other obvious risk factors.[1,4] In fact, some researchers think hyperlipidemia may be a more reliable predictor of MI in patients aged 30 to 39 years than in older patients.[1,5] Unfortunately, many of these young adults are not aware that they have hyperlipidemia until they experience a complication such as an acute coronary syndrome. With respect to the patient in this case, it is not clear from the published report whether or not she had hyperlipidemia.

Family History. Another risk factor for MI in younger patients is a positive family history of CAD in a first-degree relative younger than age 55 years.[1] Siblings of a young patient who experienced an MI have up to a 10-fold increase for developing CAD.[1] It is currently not known why a positive family history increases the risk of MI in younger patients, but it may be related to inherited disorders of lipid metabolism, blood coagulation, or other genetic factors.[1]

Drug Abuse. Finally, drug abuse must be considered in young patients presenting with an MI. The use of cocaine, methamphetamine, marijuana, and K2 (synthetic marijuana) have all been associated with MI, especially in young patients,[6-9] who typically do not have cardiac risk factors and do not show evidence of atherosclerotic disease on cardiac catheterization. As for the patient in this case, we do not know if she used any illicit drugs prior to presentation.

Summary

This case underscores the importance of not excluding MI in the differential diagnosis based simply on age or sex. While MI is uncommon in a 21-year-old woman, it can and does occur. In young patients presenting with

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chest pain, it is important to obtain a thorough history, including smoking, family history of MI, hyperlipidemia, and illicit drug use. While MI may be low on the differential diagnosis, it still needs to be considered.

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