

Wake-Up Call

Part III—"Heart Attack Basics"

by

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It appears that Denise has suffered mild heart trauma, which may lead to a more severe heart attack if not treated. But wait ... isn't a heart attack when the heart stops beating? Not exactly.

Cardiac arrest is the term used when the heart muscle literally stops pumping blood. A heart attack, also known as a **myocardial infarction**, may lead to cardiac arrest, but it's defined as a sudden event where at least one of the **three major coronary arteries** (right coronary artery, left anterior descending coronary artery, and left circumflex artery) becomes partially or totally blocked, usually by a blood clot (**thrombus**). A more rare cause of coronary occlusion is an artery spasm that shuts down blood flow to the heart. This can occur with cocaine use and severe emotional stress. Other rare causes of heart attack include sickle cell crisis, allergic reactions, carbon monoxide poisoning, extreme hypoxia, and an unmet increased need for blood flow to the heart such as may occur during extreme physical exertion, shock, or hemorrhage.

Heart cells can live for about 20 minutes without oxygen. The loss of oxygen-rich blood to the heart cells during a heart attack leads to cell damage, which may be permanent and lead to **cell necrosis** (death), depending on the severity of the attack and the amount of heart tissue that the blocked artery supplies. **The area of infarction** is where cell necrosis occurs, if it does. Surrounding it is the **area of injury**, which may or may not suffer permanent damage. The outermost affected area is the **zone of ischemia**, which is weakened but regains function within two to three weeks.

Besides the possibility of cardiac arrest, other possible complications include the following: **cardiogenic shock** (where the heart is too weak to adequately pump blood), **pulmonary edema** (where a weakened heart causes blood backup and leakage of plasma into the lungs), irregular heart rhythm (**arrhythmia**), rupture of a heart wall or valve, or death.

It is a misconception that having a heart attack leads to chronic **coronary artery disease (CAD)**. In reality, CAD and accompanying **atherosclerosis** (hardened, narrowed arteries) is the number one cause of heart attacks. What causes CAD? The main culprit is **arteriosclerosis**, or plaque buildup in the coronary arteries. **Plaque** is a material composed mainly of lipids, cholesterol (lipoproteins), and calcium. **Cholesterol** (a type of lipid necessary for synthesis of hormones, vitamin D, and bile) is carried through the bloodstream by two main types of **lipoproteins: high-density lipoproteins (HDLs) or "good" cholesterol, and low-density lipoproteins (LDLs) or "bad" cholesterol**. Studies by the American Heart Association and the well-known NHLBI-supported Framingham Heart Study show that HDLs help prevent heart disease by transporting lipids and cholesterol from the arteries to the liver. LDLs, which contain more fat and less protein, are unstable and stick to artery walls to help contribute to plaque formation.

LDLs (**cholesterol-handling system**) produce toxins that form tiny lesions on the inner walls of arteries. These lesions attract triglycerides and other substances in the bloodstream. White blood cells

(**inflammatory system**) rush to the injury site, but cause the inner wall to become stickier and thus attract more LDLs. Platelets (**blood-clotting system**) collect at the lesion site, only to trap more lipids and white blood cells. Plaque build-up slowly occurs. (Note that cholesterol is not the sole cause of plaque formation.) Over time, some of the plaque can develop a thick, hard, calcified fibrous cap and is called **stable plaque**, yet causes the arteries to become narrower and harder (atherosclerosis). Other plaque can develop a large lipid and macrophage core, decreased smooth muscle cell content, and a thinner, softer, more unpredictable fibrous cap (due to increased metalloproteinase enzyme activity). This can rupture, producing a **thrombosis** (artery blockage), **cardiac ischemia**, and a heart attack can ensue.

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Part IV—"Call 911!"

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It was March. Emily was home for spring break and Denise was enjoying having her 19-year-old daughter around. Unfortunately, it was going to be hard to spend much time with her because it was that time of the year when weddings and other catered events were picking up again after the post-New Year's lull. Denise was feeling the pressure pile up again. She constantly felt fatigued and out of breath, but she attributed these to perimenopause.

Emily could sense that her mother was tense and out of sorts, so she planned a relaxing evening for her parents and offered to cook mushroom lasagna, her mother's favorite dish. All was going well until dessert, when Emily noticed her mother's face growing paler by the minute. Suddenly, just like that night back in October, Denise began to have severe trouble breathing and her heart began racing. The room began to spin and, without warning, she fainted on the dining room floor.

"Oh my God! Dad, call 911!"

"Uh oh. Oh! Oh no! Denise. Denise! Do you read me? I'm in the middle of a heart attack!! I know it. I can feel it! That plaque in your left anterior descending coronary artery just ruptured. Now everything is going crazy. Everyone in the whole body seems to be swimming by. High levels of fibrinogen, C-reactive protein (CRP), and interleukin-18 (IL-18-inflammatory markers present in the bloodstream when there's unstable plaque) are combining with your high blood serum cholesterol. BAD things are happening, Denise. Really, really BAD!

Plaque ruptures. Platelets stick to the exposed lipid core at the site of rupture. The blood clot grows...too big. Oh too big. Is it going to break? Say it isn't going to break. Not thrombosis, please....

.... It's been 10 minutes since my heart cells supplied by the blocked artery have been without oxygen. If something isn't done soon, my cells are going to die. Necrosis! I never thought I could say that word. They say a heart attack can take over four to six hours. This first hour is horrible—the most critical period. Parts of the blood clot may break loose, travel in the blood, and stick in some tiny little blood vessel. My God, it could get in a coronary artery or the brain! An embolism. I need help! Now... NOW. HELP!!

I've got to get my self in hand. It's the only way in a crisis. Right? Right! Why didn't Denise go to her doctor to complain about her chronic breathlessness, fatigue, and nausea? All this stress elevated her blood pressure and further increased her risk for a heart attack. Alright, so she didn't know that she had a mutation in her LDL receptor gene. How could she know that LDL was not being efficiently removed from her blood? Whatever. At least she should have known her LDL blood levels were very high. So were

her levels of lipoprotein (LP a). This stuff increases heart disease risk. Why didn't anyone warn her?

Sure, I know I'm involved. I'm taking it personally. Wouldn't you? But maybe, just maybe, if Denise had been more aware of the symptoms of heart disease she would have sought help. I happen to know that heart attacks are the number one cause of death in the U.S. More people die from cardiovascular disease (including heart attacks, atherosclerosis, and hypertension) each year than the next six leading causes of death combined, including cancer and automobile accidents. It's an epidemic that people need to be educated about. So get it. I'm here to tell you. Denise. If you won't listen to me, who will you listen to?

Questions:

1. Why is the first hour of a heart attack the most critical?
2. What do fibrinogen, C-reactive protein (CRP), and interleukin-18 (IL-18) indicate?
3. What is the cause of Denise's breathlessness, fatigue, and nausea?
4. What are platelets and what do they have to do with Denise's heart problem?
5. What is an embolism and what is its connection to thrombosis?
6. What does LDL have to do with heart attacks?
7. How does hypertension develop and what does it have to do with a high risk of heart attacks?

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Part V—"Emergency Room"

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The doctor spoke calmly to Jeremy in the waiting room. "Mr. Belmore, your wife is in no immediate danger but she has suffered a heart attack to her left ventricle. She's in the emergency room right now, with the aid of an oxygen mask. We noticed some scar tissue, meaning that some prior heart trauma occurred as well. Is this your wife's first attack?"

"Yeah. I'm actually the one who has been diagnosed with heart disease in the house, and I'm the one with a family history. I don't understand. Where did this come from? Denise is conscious of her weight, and she's healthier than I am. She's the one who usually looks out for me and my daughter."

"Well, from her records, your wife hasn't had her blood pressure and cholesterol tested in a few years. Unfortunately, they were highly elevated, which greatly increased her risk of heart disease. Although she looked fit on the outside, blood work would have revealed hidden dangers. Tell me, had your wife been feeling out of sorts these past few months?"

"She has always been an on-the-go person and tends to worry a lot. Her job is pretty stressful. I did notice that these past few months she seemed more tired than usual and acted almost asthmatic. But, don't heart attack victims experience chest pain? Denise has never complained of that."

"That's a good question. The simple answer is that women's heart disease symptoms can be subtler than men's and are often overlooked. Take a look at the charts on the wall over there and you'll see what I mean. Patients may experience all, some, or none of those symptoms. It is even possible to have a silent heart attack."

Women's Symptoms

Angina (chest pain may radiate into jaw and down left shoulder and arm)

Breathlessness (especially at night)

Chronic fatigue (usually overwhelming)

Dizziness or even blackouts

Edema or swelling, especially in the ankles

Fluttering (rapid heartbeat) and pallor

Gastric upset (nausea) and sweating

Men's Symptoms

1. Sudden *immense* pressure or pain in the chest center (may persist or occur on and off)

2. Pain that radiates from chest center to neck, shoulders, and arms

3. Dizziness, nausea, sweating

4. Sudden onset of rapid heartbeat

The doctor continued, "This is a pamphlet that gives you some background on cardiovascular disease and the factors that go into them. You'll notice that some of these are things you can't change. We call them "non-modifiable." They include your gender, age and your hereditary background; we're all stuck with these. Then there are the "modifiable" factors, things like smoking, stress, and a high fat diet. When more than one factor is present, risk further increases. Once Denise is better I think you both need some time together to consider how you might change your lifestyle."

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Part VI—"The Aftermath"

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"Well, it's been four hours since the chaos began here in Denise's heart. I'm pooped! Here's the way I see it. A bunch of my cells are dead. So now there's an inflammatory response of neutrophils and monocytes and an elevated body temperature. Enzyme levels in the bloodstream are up. I don't know one enzyme from the other. They're all just proteins to me. But here's what I heard the doctors say—I mean it, they really use these big words: Creatine phosphokinase (CPK) has become elevated and will peak within 12 to 24 hours since the attack and with luck it'll return to normal within 48 to 72 hours. Its isoenzyme, CK-MB, is also elevated. CK-MB2 undergoes a change to CK-MB when released into the bloodstream. The ratio of CK-MB2 to CK-MB1 is more than 1.5 for heart attack patients, which is a benchmark doctors use to diagnose myocardial infarction within 6 hours of symptom onset. The blood level of aspartate aminotransferase (AST or GOT) has become elevated due to cell injury, will peak in 24 to 48 hours, and will return to normal in five days. In contrast to the rapid rise and decline of these enzymes, lactate dehydrogenase (LDH) will begin to elevate within a day of the attack onset and will persist at high levels for 10 to 20 days.

Cardiac troponins T and I (which help me contract) will remain elevated in the blood for 10 to 15 days after myocardial injury. This means that if the doctors find that the troponins levels are up, they can really be sure the heart has been injured. Well, that's sure to be what happened to me. So now what have I got to look forward to? Some rest and healing time. With luck, four to six weeks from now, Denise's body will have deposited collagen fibers and scar tissue at the plaque rupture site. Some more collateral vessels will have been built. But for me, things will never be the same. Any of my heart tissue that died from oxygen starvation will be lost and replaced with scar tissue ... unless doctors can find a way to regenerate it. Geesh, I never thought this would happen to me. Denise is so young...."

Assignment:

Denise is back home and on cholesterol-lowering medication and is learning how to better handle stress. Your assignment is to help Denise and her family research the key measures in preventing heart disease, or in Denise's case, another heart attack. Answer the following questions briefly and directly. You may include a table if desired. The sources cited in the References for this case are good sites to utilize.

1. Heart-Healthy Diet
 - a. What foods/nutrients should be limited and specifically what foods/nutrients are beneficial and why? (Example: what are the benefits of folic acid, monounsaturated fats, omega 3 fats, etc? Why are saturated fats bad?)

2. Lifestyle Changes
 - a. What activities are hazardous to heart health and what are some solutions? (Example: handle stress with stress management, not overeating.)
 - b. What are the benefits of exercise concerning heart health?
3. Aspirin
 - a. How can aspirin help in preventing heart disease?
4. Draw a diagram of the changes in blood enzyme and troponin levels that occur before, during and after a heart attack.
5. Create a pamphlet that the doctor could give to Denise about altering her life style. It should include information on smoking, cholesterol, blood pressure, obesity, diabetes, physical activity, diet, and stress.

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