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Women and Heart Disease

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Most women believe that their most serious health risk is breast cancer. However, they are twice more likely to die of heart disease than they are of breast cancer. Statistics show that 1 in 2 women will die from heart disease or stroke compared with 1 in 25 women who will die of breast cancer.

Women with heart disease have different symptoms than men. Women have more subtle signs of heart disease. These may include abdominal discomfort, a feeling of heartburn and chest tightness. They may not have the classic symptoms of chest pain, left arm pain and shortness of breath. Studies have shown that women tend to wait longer to seek medical treatment after symptoms appear. Some of the symptoms such as nausea, dizziness and fatigue are more common in women on a regular basis. They may not associate these symptoms with heart problems.

There has been a lot of talk in the past few years about women with chest pain being blown off by their physicians or when they present to the

emergency room. This is changing with all the studies that have been done recently. Women deserve the same standard of care as men when they are having chest pain, or anginal symptoms. I have to admit, I have been guilty of this in the past, thinking they are having anxiety more than heart symptoms. My eyes were opened recently by a 22-year-old woman who presented to the emergency room where I work. Here is her story.

Mary Smith (name has been changed) presented to our ER at 2:00 am, accompanied by her husband. She was complaining of pain in her jaw and chest, nausea, and tingling in her left arm (fairly typical complaints of angina). Mary rated her pain as a 10 on a 1-10 pain scale. Symptoms began 25 minutes prior to arrival, while she was sleeping. She was anxious and crying. Mary's past medical history included asthma, for which she used albuterol and flovent inhalers as needed. She had a baby 3 months ago, delivered by caesarean section. She was a non-smoker, weighed 65 kg,

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Have a Heart

Sarah McLain RN, CLNC, CLCP

In the three decades since the performance of the first human heart transplant in December 1967, the procedure has changed from an experimental operation to an established treatment for advanced heart disease. Approximately 2,300 heart transplants are performed each year in the United States.

In 1981, combined heart and lung transplants began to be used to treat patients with conditions that severely damage both these organs. As of 1995, about 500 people in the United States and 2,000 worldwide have received heart-lung transplants.

There have been two main barriers to increasing the number of successful operations. In 1983, the first barrier to successful transplantations--rejection of the donor organ by the patient--was overcome. The drug cyclosporine was introduced to suppress rejection of a donor heart or heart-lung by the patient's body. Cyclosporine and other medications to control rejection have significantly improved the survival of transplant patients. About 80 percent of heart transplant patients survive 1 year or more. About 60 percent of heart-lung transplants live at least 1 year after surgery. Research is under way to develop even better ways to control transplant rejection and improve survival.

Organ availability is the second barrier to increasing the number of successful transplantations. Hospitals and organizations nationwide are trying to increase public awareness of this problem and improve organ distribution.

A transplant is considered when the heart is failing and does not respond to all other therapies, but health is otherwise good. The leading reasons why people receive heart transplants are:

- Cardiomyopathy--a weakening of the heart muscle.
- Severe coronary artery disease--in which the heart's blood vessels become blocked and the heart muscle is damaged.
- Birth defects of the heart.

Heart-lung transplants are performed on patients who will die from end-stage lung disease that also involves the heart. Alternative therapies for these patients have been tried or considered. Leading reasons people receive heart-lung transplants are:

- Severe pulmonary hypertension--a large increase in blood pressure in the vessels of the lungs that limits blood flow and delivery of oxygen to the rest of the body.
- A birth defect of the heart that results in Eisenmenger's complex--another name for acquired pulmonary hypertension.

Patients under age 60 are the most likely heart transplant candidates. Patients under age 45 are generally accepted for heart-lung transplants. In both cases, patients must be suffering from end-stage disease and be in good health otherwise. The doctor, patient, and family must address the following four basic questions to determine whether a transplant should be considered:

- Have all other therapies been tried or excluded?
- Is the patient likely to die without the transplant?

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and had no family history of cardiac disease. She was on oral contraceptives, which increases her risk for heart disease.

I triaged Mary as urgent, and spent about 5 minutes talking with her in triage to get her history, and checking her vital signs. Her blood pressure was 139/94, heart rate 95, respirations 28 and oxygen level was 100%.

Our chest pain protocol allows the ER nurse to obtain an EKG and portable chest x-ray, insert a saline lock and draw labs work, and apply oxygen by nasal canula prior to the doctor seeing the patient. Upon putting her on the cardiac monitor, I noticed her ST segments were very elevated on lead II (as an experienced ER nurse, this clued me in that maybe this was cardiac). I applied oxygen at 2 liters via a nasal canula. As I was doing the EKG, Dr. Lyons came in. She had seen the monitor pattern in the nurse's station, and came in to see what was going on in room 2. Expecting to see a patient in his or her 50's or 60's, imagine the look on her face when she saw this was a young woman of 22 years. As the EKG printed, she looked over my shoulder. We looked at each other in disbelief. Mary's EKG was very abnormal. The computer read out said, "Sinus rhythm, **POSSIBLE ACUTE ANTERIOR INFART**, Lateral ST elevation, **CONSIDER ACUTE INFARCTION.**"

Dr. Lyons spoke with Mary, and got a brief history. She told her that it looked like she was having a heart attack. As she was talking, I continued to work, drawing her lab work, and putting in two intravenous lines. Dr. Lyons stepped out of the room to call the cardiologist. She had asked me to give her 4 baby aspirin, and nitroglycerin sublingual x3 every 5 minutes until her pain was gone, or her blood pressure was below 100 systolic. Thinking we may have to give her thrombolytics I left the room to get everything I would need. There is a tackle box in our cardiac room that has everything in it.

Mary's pain was a 4/10 after 2 nitroglycerin. The x-ray technician came in to do a portable chest x-ray. Dr. Lyons spoke with the cardiologist, and he told her to hold off on giving thrombolytics, and he would be right in. He wanted

heparin and loproressor and nitroglycerin only at this point. I gave her a bolus of heparin (blood thinner), and then hung a continual drip. Lopressor is given to lower her heart rate, to decrease the oxygen consumption. I gave her two doses, and it brought her heart rate in the 70's. I also began a nitroglycerin drip, and gave her some morphine to decrease the pain. Her blood work all came back within normal limits. I didn't expect her cardiac enzymes to be elevated because her pain had just started. It usually takes a few hours before they go up. We also did a urine pregnancy test, and a drug screen, since cocaine use can cause a heart attack. These were both negative.

Mary's pain was down to a 2/10 by the time the cardiologist got in to see her at 2:48. I repeated the EKG, and her ST elevation had improved. The decision was made to transfer her to a larger hospital that had the capability of doing interventional cardiac catheterization, and/or open-heart surgery if needed. Mary's vital signs remained stable during her stay in our ER. I called an ambulance, gave report to the receiving hospital, and she left us at 3:30.

After she left, and I had a chance to think over what had just happened, I was very happy with how everything had gone. As a legal nurse consultant, I am always thinking of legal issues. That made me think of how this could have been a completely different scenario. I work in a 75-bed hospital. At night there are only 2 RN's on in the ER. When a patient comes in at night, the admitting clerk calls back to the nurse's station and tells us we have a patient. One of us goes out to talk with the patient and decide if they need to come right back to a treatment room. I think of other nights, when the ER is full, and she may not have been brought right back to the ER. Luckily we were not real busy, and I was able to bring her right in. With a heart attack, time is muscle. The longer the heart muscle is deprived of oxygen, the more permanent damage is done. I feel that Mary got the best possible care that we could have given her. Unfortunately, we don't have cardiac catheterization capability 24 hours a day, so she did need to be transferred to another facility. Had it been during the day shift, we probably would have gotten her to the cardiac catheterization

lab within 45 minutes. I checked on her a few days later, and found out that she had 1 stent placed, and had done fine. This patient will always help to remind me that cardiac disease spares no one, and you have to treat everyone with cardiac symptoms as heart related until proved otherwise.

Have a Heart

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- Is the person in generally good health other than the heart or heart and lung disease?
- Can the patient adhere to the lifestyle changes--including complex drug treatments and frequent examinations--required after a transplant?

Patients who do not meet the above considerations or who have additional problems--other severe diseases, active infections, or severe obesity--are not good candidates for a transplant.

A transplanted heart functions differently from the old one. Because the nerves leading to the heart are cut during the operation, the transplanted heart beats faster (about 100 to 110 beats per minute) than the normal heart (70 beats per minute). The new heart also responds more slowly to exercise and doesn't increase its rate as quickly as before.

A patient's prognosis depends on many factors, including age, general health, and response to the transplant. Recent figures show that 73 percent of heart transplant patients live at least 3 years after surgery. Nearly 85 percent of patients return to work or other activities they like. Many patients enjoy swimming, cycling, running, or other sports.

Approximately 70 percent of commercial insurance companies and 92 percent of Blue Cross/Blue Shield plans cover heart-lung transplants. Medicaid coverage for heart-lung transplants is available in 20 states. According to the UNOS, estimated first year charges for a heart-lung transplant is \$246,000, and annual follow-up charges are \$18,400.

To learn more about transplantation, or to become an organ donor, please visit the American Heart Association website at: www.americanheart.org

Cardiac Chest Pain: Keys to Accurate Diagnosis

Maggie Driscoll RN, BSN, CCRN, CLNC

Achieving an accurate diagnosis in the chest pain patient is difficult due to the multiple non-cardiac causes of chest pain (i.e., dissecting aortic aneurysm, gastrointestinal disease, pulmonary embolism, skeletal chest wall pain). The Physician Insurers Association of America (PIAA) Data Sharing Project collects and analyzes claim information from professional liability carriers. In a 1996 study on Acute Myocardial Infarction (AMI) cases, PIAA determined that the majority of cases studied involved errors in diagnosis.

The American Heart Association defines ACS or acute coronary syndrome as “any group of clinical symptoms compatible with acute myocardial ischemia.” Acute myocardial ischemia is chest pain due to a lack of blood supply to the heart muscle that results from coronary artery disease (also called coronary heart disease). The 3 syndromes range from unstable angina to an acute full muscle-wall thickness myocardial infarction:

1. Unstable angina

Blood flow to the coronary artery, reduced by fatty plaque, is further compromised by platelet clumping that forms after plaque rupture. Symptoms may include new onset of chest pain or chest pain that now occurs at rest.

2. Non-Q-wave infarction

Incomplete and usually brief coronary artery occlusion related to blood clot formation at the site of plaque rupture. These patients are at high risk for developing a full muscle wall thickness or “Q-wave” myocardial infarction.

3. Q-wave infarction

The blood clot, or thrombus, completely occludes the coronary artery for a prolonged period, causing permanent damage to the heart muscle.

Each of the three syndromes is associated with specific treatment interventions and prognosis. Rapid recognition of ACS is imperative in reducing mortality rates among chest pain patients.

Diagnosis of acute coronary syndromes (ACS) is based on a thorough history and physical examination, serial electrocardiogram (EKG) studies, and lab tests to detect myocardial injury. All hospitals, clinics and physician offices should have policies that guide the care and treatment of a patient complaining of chest

pain. Patients at higher risk for cardiac-related ischemic chest pain typically exhibit the following characteristics:

History

- Prior history of angina, MI
- Positive family history of CAD
- Smoking
- Hypertension
- Obesity
- Diabetes mellitus
- High cholesterol or impaired cholesterol function
- Male age > 60, Female > 70
- Cocaine use

Presenting Symptoms

- Midsternal chest pressure, squeezing, tightness or burning usually lasting several minutes.
- Pain radiating to arms, shoulders, jaw or between shoulder blades
- Chest or arm pain or discomfort as chief symptom reproducing prior documented angina
- Shortness of breath
- Nausea and/or vomiting
- Diaphoresis (sweating)
- General feeling of anxiety or impending doom

Physical Exam

- Abnormal heart sounds, such as an S3 or murmur of mitral regurgitation
- Abnormal lung sounds, such as crackles or wheezing
- Skin that is pale or bluish in color

Not all patients will exhibit the “classic symptoms” of an AMI. It is well known in the medical community through study and clinical experience that women and diabetics have atypical chest pain such as feelings of “indigestion” or may not have any pain at all, but only experience nausea or diaphoresis. According to the PIAA study, two most common incorrect impressions of cardiac chest pain were diagnosed as gastrointestinal disorders and musculoskeletal complaints.

The Role of the 12-Lead EKG

According to the American Heart Association the 12-Lead EKG “stands at the center of decision-making for the care of the patient with ACS.” A twelve-lead EKG should be interpreted by a qualified clinician on all patients suspected as having an AMI, preferably

within 10 minutes of arrival in the ED. Based on American College of Cardiology and American Heart Association Practice Guidelines, each patient should be classified into one of three groups based on EKG findings:

1. ST-segment elevation or new or presumably new Left Bundle Branch Block
2. ST depression or dynamic T-wave inversion
3. Non-diagnostic or normal EKG.

EKG findings will further guide treatment protocols. Initial EKG’s in the AMI patient are normal or non-diagnostic in 55% of all patients admitted to the ED with chest pain. For this reason serial EKG’s and serum cardiac markers should be performed on patients who meet criteria for unstable or new-onset angina. Today, these patients are often admitted to chest pain observation units and monitored over the next several hours for EKG changes, positive serum cardiac markers, and arrhythmias.

Serum Cardiac Markers

Enzymes (termed serum cardiac markers) released by injured or ischemic heart muscle can be measured in a variety of ways. Each marker rises, peaks and falls at different intervals from the time of chest pain onset. Accurately identifying the onset of symptoms can be helpful in analyzing the results of the cardiac enzymes released. The cardiac enzymes, CK-MB and Troponin I, commonly used to identify AMI do not rise until 3-8 hours after onset of symptoms. A patient complaining of chest pain that occurred less than 6 hours ago would need additional cardiac enzyme testing to be able to accurately diagnose AMI. However, when the patient history is unreliable, unobtainable or when pre-infarction angina is suspected, the timing of onset of chest pain should be set at the arrival of the patient to the ED. If the first marker is negative, the American College of Emergency Physicians recommends repeating serum CK-MB and Troponin I testing at intervals of 8-12 hours. AMI can be reliably ruled-out if markers are negative 12-24 hours after onset of pain. Myoglobin, an enzyme non-specific for myocardial damage, should not be exclusively relied upon to identify or exclude the diagnosis of AMI. The failure to diagnose AMI is a missed opportunity to preserve heart muscle function and potentially save a life.

Heart Attacks & Worker's Compensation

Robert Morrison, RN BSN

After several years in industrial safety, corporate risk management and Worker's Comp case management I have become accustomed to hearing employees claim Worker's Compensation benefits for a wide variety of personal ailments. Some days it seems that every malady they experience comes under the label of "work-related." While employers may roll their eyes and insurance adjusters get out their "Denied" stamps, there are some situations that do indeed entitle an employee to such benefits. In the spirit of this month's heart theme we take a brief look at the employee who suffers a myocardial infarction (MI) while at work.

First, look at the basic premise of Worker's Compensation. This was a system developed to assist the worker who suffered illnesses or injuries arising out of, or in the course of, their employment. "Arising out of" refers to the specific activity performed by the employee and the causative relationship between it and the injury or illness. This deals with establishing the specific cause(s) of the damage. "In the course of" deals with when and where the injury or illness occurred. This is the basic foundation of Worker's Compensation, and could be the subject of several articles by itself. Each state gives specific definitions and criteria that control the program in that state. This article explores some of the different ways that states deal with the issue of MI's as compensable damages, as evidenced by personal experience as well as newspaper, journal, and newsletter articles published over the past two years.

The basic issue is whether the MI was caused by the employee's job. Again, each state will have legislation and case law that reflects their own values and requirements. Nebraska and Iowa, for example, are very different in their approach. Nebraska takes the employee's entire medical history into account to decide where the wear and tear took place. Plaintiff attorneys seem to agree that this makes Nebraska a tough state to deal with. Iowa has historically been more liberal. For example, case law states that continuous

exertion required by the employment after the onset of symptoms can be ruled a causative factor, regardless of the employee's medical history.

Two other issues debated when considering compensation for MI's are workplace stress and laws of presumption. It is without argument that the workplace can be a source of varying and significant stress. This can come from the employer-deadlines, difficult product specs, individual workloads, etc, or from other employees- ill humor, pranks that cause problems, or violence. Three types of workplace stress have been described and have drawn this writer's notice. *Physical-mental* stress is when a physical injury results in a mental condition, i.e. an extremely difficult or incomplete rehabilitation. *Mental-physical* stress is the type this article is most concerned with. This is when mental stress results in a physical injury, such as intense stress in the workplace resulting in an MI or other medical emergency. *Mental-mental* is when a mental stress results in a mental condition, such as Post-Traumatic Stress Disorder following an extremely disturbing event on the job.

Different states have attempted to regulate the issue of workplace stress. Some require the employee to show that they were exposed to a higher level of stress than the average worker in a similar situation or job. Some only require the employee to show they experienced a higher level of stress than the general public. That leaves a lot of room for argument either way. Also debatable is whether the perceived stress level increased just before the cardiac event, and whether medical conditions pre-dating employment should be considered. Again, case law creates a wide variety of approaches to this issue. Some occupations are inherently more stressful than the general employee population. Others are "average" in their stress levels, but with occasional spikes. It isn't enough to say, "Well, he would have had that heart attack whether he was at work or at home." You have to consider all of the sources of possible stress specific to the employee's situation when you are trying to understand the overall stress level. Another thing to consider is what this writer calls the laws of presumption. There

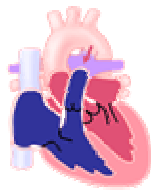
are several states that have passed laws stating that cardiac events, strokes, hypertension, communicable diseases, and other conditions that occur on the job are presumed to be work-related. This is often aimed at specific occupations such as firefighters, law enforcement officers and select others. This writer has also seen this included in union contracts. This legal presumption may override any discussions about the employee's medical history or off-the-job stressors or personal habits such as smoking. In this situation the employer may have the burden of proving that the MI was NOT caused by the job.

In general, most states seem to presume that if the injury occurred on the employer's premises during the employee's regular working hours then the injury is compensable. Again, the burden of proof is on the employer to refute. This presumption has been the focus of much of this writer's professional time and effort. There are any number of activities that the employee may engage in during the course of the workday. Personal care, breaks and lunch times, and other activities not directly related to the job can result in injuries. While this is beyond the scope of this article, one can see how this presumption affects the issue of heart attacks in the workplace.

These are a few of the issues involved in determining the compensability of cardiac emergencies at the workplace. The specific actions needed by the prosecuting and defense teams are determined by enacted legislation and case law in the appropriate state. It takes both healthcare and legal experience to provide the client with the tools they need to successfully deal with this problem.

Abbreviations

MI-myocardial infarction or heart attack
 AMI-Acute myocardial infarction
 ACS-acute coronary syndrome
 EKG-electrocardiogram
 CAD-coronary artery disease
 PDA-patent ductus arteriosus
 CHF-congestive heart failure
 CPR-cardiopulmonary resuscitation
 CVD-cardiovascular disease
 EMS-emergency medical services
 AED-automated external defibrillator



What is a Patent Ductus Arteriosus?

Ductus arteriosus is a pathway between the aorta and the pulmonary artery and is

Part of the fetal circulation in the fetus. In utero, the fetal lungs receive oxygen from the mother via the placenta. The ductus arteriosus diverts ~97 % of the blood to the fetal organs allowing only ~ 3.5 % of the blood circulating to become part of the pulmonary blood flow to the lungs. The closing of the ductus is carefully choreographed because as the lungs expand at delivery causing a decrease in the resistance in the pulmonary blood vessels and vessels relax. As the vessels relax they dilate allowing more blood to flow to the lungs. As this relaxation and dilatation is taking place the muscles of ductus arteriosus are stimulated. The muscle stimulation is caused by the increase in oxygen levels in the muscle and is the trigger for the ductus to begin to close. But sometimes this carefully choreographed maneuver fails and the ductus remains partially open or completely open. When this happens the condition is known as Patent ductus arteriosus.

Signs and Symptoms Related to Defect Size

The signs and/or symptoms of Patent ductus arteriosus (PDA) is dependent on the size of the ductus (opening) and the

Patent Ductus Arteriosus

Jan Aken RN IBCLC

amount of blood flow through the opening. If the PDA is small the infant may not present with any difficulties, but a heart murmur may be heard on auscultation. The flow of blood through the PDA may put the infant at risk for infections. A premature infant with heart failure and a murmur is the classic diagnosis of PDA. But some physicians fail to diagnosis the condition and treat the infection. Some of these infants develop an even more serious infection known as endocarditis, which is an inflammatory condition of the innermost part of the heart. If the PDA is large the signs and symptoms one might see would be fast breathing, some difficulty in breathing, tiring more easily, poor growth, along with frequent respiratory infections.

Statistics

Approximately 40,000 babies are born each year in America with heart defects, according to the American Heart Association, 2003 statistics. Of these, about nine percent have PDA

Risk Factors:

2. Premature infants
3. Maternal rubella during pregnancy
4. Delivery in a high altitude area of the country.
5. Family history of PDA
6. Female 2:1 Male

Treatment Depends on the Severity:

1. Pulmonary support
2. Oxygen to correct hypoxemia
3. Fluid restriction

1. Correct anemia
2. Measures to correct the Congestive Heart Failure
3. Antibiotic therapy for prevention of endocarditis
4. Medications

The medication use will be dependent on the clinical status of the infant.

- a Prostaglandins are prescribed to keep the ductus patent until the surgery is done.
- b Prostaglandin inhibitors (e.g. NSAID) are used to close the ductus arteriosus if the surgical ligation is not necessary.

Prognosis:

1. Prognosis is excellent for uncomplicated PDA.
2. If the infant has more heart anomalies then the PDA, this will increase the morbidity.
3. Natural or spontaneous closures are excellent in those younger than 3 months but in those older than 3 months are rare.
4. Spontaneous closure in the premature infants group is 75%.
5. Postoperative results are best if closed while the patient is younger than 3 years.

Medical/Legal Problems

Cases that may come to the attention of the attorney.

1. Failure to diagnose PDA .
2. Failure to use antibiotics.

CPR-Why Learn it?

Pattie Patterson RN, LNCC, LCP

Cardiovascular disease (CVD) is the number one killer in the United States, and women are now more at risk than men, according to the American Heart Association, as it accounts for one in five deaths of women.

This year an estimated 1.2 million Americans will have a coronary attack or recurrent attack. About 502,000 of these people who experience a coronary attack in a given year will die from it-340,000 of them in an emergency room or before they reach the hospital. That's two-thirds of all deaths from CVD.

A heart attack occurs when the blood supply to part of the heart muscle is severely reduced or stopped because one or more of the heart's arteries is blocked. If the blood supply is cut off for more than a few minutes, the heart muscle

suffers permanent injury in that area. Depending on the severity of the injury, it can disable or even kill the person. This is why it's so important for everyone to learn CPR. CPR or cardiopulmonary resuscitation is often the first line of defense when someone is having a heart attack. Even if you have been trained in CPR before, you should keep your certification current, as the proper technique changes periodically.

For instance, we used to initiate CPR for one minute before calling for the EMS (dialing 911). Now, unless it is being performed on an infant or child, you call the EMS first, BEFORE you initiate CPR. The rationale for this is that unlike infants or children, the cause of the heart attack in adults is usually due to a disease process that needs to be treated as

soon as possible. With infants and children, it is usually because of preventable accidents.

Brain damage can start to occur in only 4 to 6 minutes after the heart stops pumping. Death may be prevented if the victim receives immediate CPR and defibrillation within a few minutes. If there is no one to initiate CPR, the person's chances of survival decreased by 7 to 10 percent for every minute that CPR is not initiated. When bystanders provide immediate CPR and the victim is defibrillated within 3-5 minutes the survival rate can be as high as 74 percent. Many communities also have automated external defibrillators, which lay persons can be trained to use in most local CPR classes. So, go sign up for a class today!

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