Cardiac Case Study

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Cover Sheet

Christa: Introduction, Editing/compilation of paper, Pathophysiology

Team Khazakstan-- Ashley: Medications, Mollye: Labs/Diagnostics, Adria: Assessment

Team Alaska (Justine, Victoria, Molly, Kelsey): Careplan, Pathos, Diagnosis
Complex Cardiac Case Study

**Introduction**

Mr. Prince, a 68 year old man, came to the emergency department complaining of shortness of breath, inability to lay flat, and infrequent palpitations. Upon exam, it was found that he has recently gained 10 pounds, has 2+ pitting edema, and moist and gurgling breath sounds. A chest x-ray confirmed that he has pulmonary vascular congestion and left ventricular enlargement. His ABG showed acidosis. He is receiving oxygen via nasal cannula at 4L/min and is receiving furosemide. He is on continuous EKG monitoring, ABG's, and fluid and electrolyte labs. He is retired and has become less active in the last year and states that has not visited his PCP in several months. He has a history of asthma, hyperlipidemia, hypertension, stroke syndrome secondary to left internal carotid occlusion, GERD, congestive heart failure, anemia, polyps on colonoscopy, and 2 pack/day smoking until age 65.

**Pathophysiology**

**Asthma**

Asthma is “a chronic disorder of the airways that involves a complex interaction of airway obstruction, bronchial hyper-responsiveness and an underlying inflammation.” (McCance & Huether, 2010, p. 1283) It is a reaction which causes bronchiolar inflammation/swelling/constriction related to an increase of inflammatory mediators like histamine, prostaglandins, and leukotrienes; this along with increased mucous production leads to difficulty breathing, especially upon expiration, and lung hyperinflation can occur, causing damage to the lung tissue. Increased acetylcholine triggers the bronchial smooth muscle contraction, with mucous production. Asthma has found to have strong genetic ties, and is often found within families. Risk factors for asthma include family history, “allergen exposure, urban
residence, exposure to air pollution, cigarette smoke, recurrent respiratory viral infections, and obesity.” (McCance & Huether, 2010, p. 1283) Clinical manifestations may include chest constriction, wheezing, dyspnea, non-productive coughing, tachycardia, tachypnea, and even blood gas alterations (McCance & Huether, 2010). This patient is not experiencing asthma symptoms at this time. However, his GERD and history of cigarette smoking are likely to be triggers for an asthma attack.

**Coronary Artery Disease**

Coronary artery disease is a general diagnosis that includes “any vascular disorder that narrows or occludes the coronary arteries.” (McCance & Huether, 2010, p. 1160) This narrowing of the arteries decreases the flow of oxygenated blood that is able to feed the heart muscle its-self. The heart muscle cells will then be less effective, and may even die if the ischemia is severe enough; this in turn leads to myocardial infarction. Risk factors for developing CAD include advanced age, male gender, women after menopause, family history of CAD, dyslipidemia, hypertension, cigarette smoking, diabetes, obesity, sedentary lifestyle, and poor diet (McCance & Huether, 2010). This patient is a 68 year old male, who has been diagnosed with CAD for 16 years. He has a known history of cigarette smoking, and another diagnosis of hyperlipidemia, that also contributes to the development of his CAD. His hyperlipidemia likely began causing plaque build-up in his arteries, which led to decreased blood flow and oxygenation to his tissues. The years of cigarette smoking only made it more difficult to adequately nourish and oxygenate his cells, as the nicotine in cigarettes not only causes vasoconstriction, but inhaling smoke damages the lung tissue, and carbon monoxide replaces oxygen on the hemoglobin molecule; making even less oxygen available for the heart muscle cells.
Hyperlipidemia

Hyperlipidemia is a condition in which the blood levels of certain lipoproteins, or fats, rises above normal levels. The resulting increase in lipoproteins leads to fatty deposits in organs such as the “…heart, liver, and muscle.” (McCance & Huether, 2010, p. 69) Because the fats are carried in the bloodstream, deposits also form inside vessels throughout the body. This leads to a narrowing of the arteries, as fatty plaque takes up intravascular space. Hyperlipidemia increases the risk of CAD, heart attack, and stroke. A few risk factors for hyperlipidemia include a diet high in fats, obesity, sedentary lifestyle, family history of hyperlipidemia, and diabetes. This patient admits to being less active in the past few years since his retirement, which likely attributed to his hyperlipidemia. As mentioned earlier, because fatty plaques form inside blood vessels, hyperlipidemia plays a role in the development of CAD and congestive heart failure.

Hypertension

Hypertension is the result of complicated interactions between genetic and environmental factors that increase vascular tone and blood volume; causing sustained increase in blood pressure. Hypertension is classified as a blood pressure of 140/90 or above. The increased pressure on the artery walls can damage blood vessels and other organs in the body. Over time, hypertension affects the body in many ways, and is “…associated with increased risk for target organ disease events, such as myocardial infarction, kidney disease, and stroke.” (McCance & Huether, 2010, p. 1149) Hypertension is often called the “silent killer” because it is frequently asymptomatic until it becomes severe, and target organ disease occurs. A patient with severe hypertension may experience a variety of symptoms secondary to effects on blood vessels such as fatigue, reduced activity tolerance, dizziness, palpitations, angina, and dyspnea. This patient did express palpitations as a symptom upon ED admission. Hypertension only makes the heart have to work
harder; this fatigues the heart, and with time will cause thickening of the heart wall. This in turn leads to decreased cardiac output, due to inadequate ventricular filling. His hypertension likely played into the development of his congestive heart failure.

**Stroke Syndrome**

A Stroke Syndrome, also known as a Cerebrovascular Accident, is a common indication of cerebrovascular disease and presents as “a sudden, non-convulsive focal neurologic deficit (McCance & Huether, 2010, p.600).” A stroke may be thrombotic, embolic, hemorrhagic, or lacunar. Thrombotic strokes occur when thrombi formed in arteries that supply the brain cause an occlusion of the vessel. They are often a result of atherosclerosis and inflammation of the arterial walls. Embolic strokes occur when a fragment from a thrombus outside of the brain breaks off and travels to the brain obstructing a vessel and causing ischemia. A hemorrhagic stroke occurs when a mass of blood has formed and brain tissue is compressed or ruptured. This is often related to hypertension. A lacunar stroke is a very small stroke (less than 1cm diameter) that occurs mainly in the basal ganglia, internal capsules, and pons. This patient had a cerebral infarction as a result of a left internal carotid occlusion, meaning that a thrombus that blocked the vessel and caused an area of the brain to lose blood supply. Risk factors for stroke that are relevant to this patient are hypertension, smoking, and hyperlipidemia. Hypertension is a significant risk factor for thrombi and emboli formation because years of high blood pressure cause damage to the vessel walls. Smoking has been found to double the risk of stroke (McCance & Huether, 2010, p.601). Clinical manifestations of stroke depend on the type and severity of the stroke, but may include dizziness, confusion, trouble speaking, paralysis of the face, arm, or leg, headache, and vision problems.
Gastric Esophageal Reflux

GERD occurs when chyme from the stomach, enters the esophagus. Gastric hydrochloric acid and pepsin secretions that reflux into the lower esophagus cause esophageal irritation and inflammation. The degree of inflammation depends on the amount and composition of the gastric reflux and on the ability of the esophagus to clear the acidic contents. Normally, “…acid is neutralized and cleared from the esophagus by peristaltic action within 1 to 3 minutes, and sphincter tone is restored” (McCance & Huether, 2010, p. 1458). Often, the lower esophageal sphincter is incompetent and lets the gastric contents move into the esophagus. Certain foods such as caffeine and chocolate and drugs such as anti-cholinergic can decrease the effectiveness of the lower esophageal sphincter.

Risk factors for GERD may include a hiatal hernia, an incompetent lower esophageal sphincter, decreased esophageal clearance, obesity, cigarette smoking, and decreased gastric emptying.

Common signs of GERD are heartburn, dyspepsia (pain in the upper abdomen), hyper-salivation, and chest pain that may mimic angina (burning, squeezing pain that may radiate to the back, neck, jaw, or arms). Although the patient did not complain of any current GERD symptoms, he does have a history of asthma, which may be triggered by inflammation and irritation that occur as a result of GERD.

Congestive Heart Failure

Congestive heart failure is a complex disease which essentially involves impaired cardiac filling and/or pumping. CHF can be caused by so many factors, a few include high cholesterol, anemia, infections, hypothyroidism, dysrhythmias, bacterial endocarditis, pulmonary diseases, nutritional deficiencies, hypovolemia, past MI, hypertension, weight, and age. There are different types of heart failure, which include systolic and diastolic failure. Systolic involves the left ventricle and
the loss of ability to pump blood effectively throughout the body. Over time, this leads to left ventricular dilation and hypertrophy (Lewis, 2011, p. 798). In diastolic failure, there is increased filling pressure in the ventricles due to difficulty of the muscle to relax and stretch properly for ventricular filling of blood. This ultimately leads to venous engorgement throughout the body because of the backup of blood. Patients may have either types or a mixture of the two. Clinical manifestations of CHF include pulmonary edema with possible cyanosis, fatigue, edema in the extremities, tachycardia, dyspnea, nocturia, skin changes, behavioral changes, chest pain and weight changes due to fluid build-up (Lewis, 2011, p. 802). Complications of CHF can include pleural effusion, dysrhythmias, left ventricular thrombus, hepatomegaly and renal failure. Patients experiencing left-sided heart failure may show signs such as “dyspnea, orthopnea, cough of frothy sputum, fatigue, decreased urine output, and edema.” (McCance & Huether, 2010, 1193) This patient reported being short of breath, and having difficulty breathing while laying down. These are signs of left-sided heart failure, as pulmonary edema and pleural effusion of the lungs occurs; filling the alveolar space with fluid, instead of oxygenated air. His heart failure certainly has many contributing factors, among those are hypertension, hyperlipidemia, and CAD.

**Anemia**

Anemia is a deficiency in the number of red blood cells, quantity of hemoglobin molecules available, and hematocrit levels. Iron deficiency anemia can be caused by blood loss or decreased production of RBCs. Anemia generally occurs due to “impaired erythrocyte production, blood loss, increased erythrocyte destruction, or a combination of these three.”(McCance & Huether, p. 989). Many older adults have anemia due to nutrition deficiencies. Deficient levels of iron and folic acid can cause decreased production RBCs,
leading to anemia. Due to decreased RBCs, which transport oxygen on the hemoglobin molecules, a patient can experience tissue hypoxia. Clinical manifestations depend on the severity of the disorder. Mild anemia shows itself with heart palpitations, dyspnea with exertion, and mild fatigue. Moderate anemia can cause increased palpitations, dyspnea, and fatigue. Severe anemia will show itself in many ways including pallor, jaundice, pruritis, blurred vision, retinal hemorrhage, jaundice of the sclera, tachycardia, claudication, angina, HF, MI, tachypnea, dyspnea at rest, depression, irritability, vertigo, anorexia, hepatomegaly, splenomegaly, difficulty swallowing, sore mouth, bone pain, sensitivity to cold, weight loss, and lethargy. Blood lab values will be drawn, in order to determine the severity of one’s anemia. In this particular patient, a decrease in available oxygen, due to anemia, likely contributed to ischemic heart muscle cells; only making the effects of hyperlipidemia and CAD worse.

**Assessment**

Mr. Prince, a 68-year-old Caucasian male, presented to the ED with reports of dyspnea that has persisted for the last two to three days. He has a difficult time sleeping meanwhile lying flat because he feels as though he is “suffocating;” he finds relief when sitting upright. The patient also reports having infrequent “palpitations.” Other respiratory symptoms and fever were denied, further leading us to believe the symptoms were related to heart failure.

Bibasilar lungs sounds were loud and wet with faint breath sounds at the mid-axillary lines on either side. Also, bronchial sounds were wet and “gurgling.” These findings are consistent with those of pulmonary edema found in left-sided heart failure (Williams & Wilkins, 2012, p.302). The patient’s respirations are elevated at 28/min and are irregular & “choppy.” His body struggles to meet oxygen demands due to pulmonary edema which makes it difficult for
appropriate gas exchange to occur. Also, dilution of the blood makes oxygen scarcer and the body has to compensate in order to make up for the difference.

Mr. Prince had a tympanic temperature of 99°F which is not of concern right now. His blood pressure was elevated at 188/76 and his apical pulse was at 132 bpm and irregular. These findings correspond to the increased workload of the heart. With blood backing up the heart has to work harder to circulate blood, especially since the ventricles are stretched. The patient’s breath sounds made it difficult to auscultate the heart; however, a distinct murmur could be heard at the 2nd intercostal space on the right. Murmurs are heard due to regurgitation (Williams & Wilkins, 2012, p.248). Also, it is difficult to make out for sure but he may have a S3 gallop which is common with left-sided heart failure (Williams & Wilkins, 2012, p.302).

Mr. Prince is 6’1” and weighs 210 lbs. He reports his normal weight being at 200 lbs. No time frame was given regarding the weight gain. Weight gain is typical of heart failure (Williams & Wilkins, 2012, p.304). Mr. Prince has pitting edema that extends up from his ankles at 2+ to about the pre-patellar level at 1+. Dependent edema is a sign of right-sided heart failure. Also a sign of right-sided heart failure is hepatomegaly. The patient has 1+ enlargement of his liver beyond his right rib margin. His skin is normal but has a “grayish” tone. Since he is not clearly cyanotic the discoloration may be related to the patient’s anemia. His capillary refill is greater than 2 seconds which is delayed but there is no clubbing to suggest that it has been a long term issue. The patient’s poor capillary refill is related to poor cardiac output; his discoloration may also be related to this (Williams & Wilkins, 2012, p.302).

Diagnostic results confirm that there is left ventricular enlargement as well as pulmonary vascular congestion as seen in heart failure (Williams & Wilkins, 2012, p.248). Also, there appears to be more fluid in the right than in the left portion of the heart. Atrial fibrillation and
erratic ventricular response were found in Mr. Prince’s electrocardiogram. Atrial response was recorded at 132/min and ventricular response was recorded at 52-68/minute. In a minute recording there were six premature ventricular ectopy. The atrial fibrillation, erratic ventricular response, and ventricular ectopy are related to the heart being damaged. With these things we are concerned about there being clot formation since abnormal pumping causes blood to be stagnant.

Lab results show that sodium, potassium, and chloride are low. Low levels of sodium and potassium are due to fluid being displaced into tissues (Williams & Wilkins, 2012, p.303). Blood urea nitrogen and creatinine levels are elevated; this is due to poor kidney perfusion. Mr. Prince’s blood sugar levels are elevated which happens when the body is under stress. Also, arterial blood glasses reveal that there is uncompensated respiratory acidosis; this is what we would expect of someone with tachypnea.

Information that pertains to Mr. Prince’s current situation includes a history of coronary artery disease, hyperlipidemia, and hypertension. These are all conditions that affect the vascular system and its ability to work and adjust to meet metabolic needs, contributing to the heart being overworked (American Heart Association, 2012). He has a history of gastric esophageal reflux disease, benign polyps on colonoscopy, and asthma as well; however, these are of little interest at this time. Initially his history of asthma would be of interest since he presented to the ED with shortness of breath but that was quickly ruled out as a cause for his complaint.

Other points of interest are Mr. Prince’s history of smoking (2 pack/day until the age of 65), his age (68 years old), the fact that he is a male, his inactive lifestyle, and stress regarding his condition. These things increase his risk for developing heart problems as well as other health problems (American Heart Association, 2012). Also, the patient reports having had congestive
heart failure previously. Patient’s with this background require careful maintenance and close monitoring to prevent further damage.

**Labs/Diagnostics**

The patient most likely has left and right sided heart failure. The first indicator is the patient’s report of shortness of breath over the past 2-3 days that prevents him from lying down to sleep. This rules out diseases and illnesses such as pneumonia, bronchitis, and asthma attack, especially since he has not experienced any symptoms of fever or other respiratory illnesses. Outside of direct respiratory issues, cardiac issues are known to cause shortness of breath; the second indicator that further confirms this is the patient’s report of ‘palpitations.’ Thirdly, in the physical assessment, it is noted that the patient’s weight is 10 pounds more than his usual weight. This is a third indicator for heart failure, leading to the need for a chest x-ray, electrocardiogram (ECG), chemistry lab, and arterial blood gases (ABG) for further assessment and clarity on what is going wrong.

<table>
<thead>
<tr>
<th>Lab/Diagnostic</th>
<th>Normal Ranges</th>
<th>Patient’s Range</th>
<th>Indication</th>
</tr>
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<tbody>
<tr>
<td>ECG</td>
<td>Rhythm: Regular Rate: 60-100 beats per minute P Waves: 0.06-0.12 seconds PR Interval: 0.12-0.20 seconds QRS Complex: P wave, PR Interval</td>
<td>Atrial fibrillation (SA node &gt;100 times/minute) with atrial response at 132/minute. Irregular rhythm at 52-68/minute 6 ventricular ectopy in 1 minute.</td>
<td>Atrial fibrillation, irregular ventricular response and premature ventricular ectopy are all within the category of dyshytmias; common causes are heart failure, myocardial ischemia &amp; cardiomyopathy (Lewis, Dirksen, Heitkemper, Bucher, &amp; Camera, 2011, p.823).</td>
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</table>
<0.12 seconds
SA node: 60-100
times/minute
AV node: 40-60
times/minute

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<tr>
<th>Sodium</th>
<th>135-145 mEq/L</th>
<th>130 mEq/L</th>
<th>Low</th>
<th>With heart failure, backed up fluid leaks out into the intracellular spaces, pulling sodium and potassium with it (Lewis, Dirksen, Heitkemper, Bucher &amp; Camera, 2011, p. 798).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td>3.5-5.0 or 5.3 mEq/L (depending on sources or workplace)</td>
<td>2.8 mEq/L</td>
<td>Low</td>
<td>Chloride levels are known to drop with hypokalemia and hyponatremia. Also, patients with heart failure may develop hypochloremia because serum chloride levels are diluted by excess fluid in the body (Williams &amp; Wilkins, 2011, p. 185).</td>
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<tr>
<td>Chloride</td>
<td>95-105 mEq/L</td>
<td>92 mEq/L</td>
<td>Low</td>
<td>These are both waste products and indicate decreased kidney function.</td>
</tr>
<tr>
<td>BUN</td>
<td>5-20 mg%</td>
<td>31 mg%</td>
<td>High</td>
<td>This is expected because reduced kidney perfusion related to poor cardiac output (CO) leads to decreased filtration and diuresis.</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.5-1.1 mg%</td>
<td>1.6 mg%</td>
<td>High</td>
<td>Blood sugar levels become increased when the body is under stress. It could also be assumed that this patient has used his albuterol frequently over the past 2 days to try and treat his shortness of breath. A side effect of albuterol use is hyperglycemia (Deglin, Vallerand, &amp; Sanoski, 2011, p. 127).</td>
</tr>
<tr>
<td>Blood Sugar</td>
<td>80-120 mg%</td>
<td>180 mg%</td>
<td>High</td>
<td>These results reveal uncompensated respiratory acidosis, which would be expected from someone who is experiencing shortness of breath. Also, the lower the pH of the blood, the lower the affinity of red blood cells to oxygen, which means their carrying capacity is decreased and the patient has to breath with more effort and vigor to get the needed oxygen for his body (Lewis, Dirksen, Heitkemper, Bucher &amp; Camera, 2011, p. 501).</td>
</tr>
<tr>
<td>ABG</td>
<td>pH: 7.35-7.45 CO₂: 34-45 mm Hg HCO₃⁻: 22-26 mmol/L PO₂: 80-100 mm Hg arterial 38-42 mm Hg venous. O₂ Saturation: &gt;95%</td>
<td>pH: 7.30 (Acidotic) CO₂: 51 mm Hg (High: acidotic) HCO₃⁻: 22 mmol/L (Normal) PO₂: 70 mm Hg arterial &amp; 30 mm Hg venous. (Low) O₂ Saturation: 82% (Low)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Two labs that were not addressed in the case study, but would be beneficial to assess are BNP and troponin levels for the purpose of assessing the level of damage of cardiac tissue. (Lewis, Dirksen, Heitkemper, Bucher & Camera, 2011, p.727). Even though chest pain is a classic sign for a myocardial infarction (MI), it is known for some patients to not experience any pain at all but rather ‘discomfort,’ weakness, shortness of breath, or dysrhythmias (Lewis, Dirksen, Heitkemper, Bucher & Camera, 2011, p.779). It is clear that this patient is experiencing heart failure; an MI may be the underlying cause and would be an important piece of information to know.