

# Multisystem case

Business



The patient admits to a long history of cigarette smoking having decreased his smoking to half a pack daily since being diagnosed with emphysema five years ago. On initial assessment Mr.

Jones appeared stated age but fatigued, was alert and oriented to time, place and situation, pupils 3 equal and reactive. Blood pressure 156/94, heart rate 102, rhythm sinus tachycardia, heart tones includes S3 gallop, denies chest pain and peripheral pulses were palpable with weak bilateral post-tibial and dorsalis pedis.

Lung sounds were clear upper lobes with crackles in bilateral lower lobes, patient is dyspnic on exertion. Pulse oximeter 88% on room air, oxygen was applied at 2 L/min via nasal cannula which improved oxygen saturation to 94%. Abdomen obese had positive bowel sounds in all morning. Mr.

Jones reports a decrease in urinary frequency as well as amount, recalls urinating very small amount twice daily in the past two days. Skin was intact, has 3+ bilateral lower extremity edema. Mr.

Jones reports the following medication regime: Altace 5mg PO twice daily, Toprol XL 25mg PO daily, Aldactone 25mg PO daily, Advair Diskus 250/50 mcg inhaler twice daily and Spiriva 1 cap inhaled daily. Mr.

Jones reports that he has not taken his medications in the past 7 days because he did not have the money to purchase them. The ED physician orders the following tests: complete blood count, complete metabolic profile, thyroid level, lipid profile, homocysteine levels, Troponin I every 8 hrs x 3, BNP, HGBA1C, ABG, EKG, CXR, 2D Echo and a Cardiology consult.

Order for Lasix 40 mg IV every 8 hrs with the first dose to be given stat was written. A urethral catheter was inserted in order to maintain strict and 1500 ml fluid restriction was ordered. Chronic Disease Review: Congestive Heart Failure Definition According to Brashers heart failure affects 10% of the population over the age of 65 and is the most frequent reason for hospital admission in this age group (2008). HF encompasses several types of cardiac malfunction which results in insufficient blood supply to the body's tissues and organs.

The most common cause of HF results from left ventricular dysfunction which includes both systolic and diastolic heart failure. Right ventricular dysfunction (also known as cor pulmonale) in the absence of left ventricular dysfunction can be attributed to pulmonary disease such as emphysema which is also referred to as COPD. Signs and Symptoms Signs and symptoms of left heart failure result from pulmonary vascular congestion and insufficient perfusion to tissues and organs.

Brashers describes patient's experience fatigue, edema, decreased urine output, dyspnea, orthopnea, and frothy sputum (2008). Physical exam will reveal pulmonary edema, hypertension or hypotension, S3 gallop and possible evidence of acute or chronic CAD. Mr.

Jones did present fatigued with dyspnea on exertion, crackles bilaterally, he does have an audible S3 gallop, and blood pressure is 156/94. Edema of the bilateral lower extremities is evident at 3+ as well as Mr. Jones reports a 6 lb weight gain in the past week as well as decreased urine output.

Pathophysiology Systolic heart failure (SHE) is the inability of the heart to produce a cardiac output sufficient enough to perfuse major organs and tissues. Cardiac output depends on the heart rate and stroke volume. Stroke volume is affected by contractility, preload and afterload (Brashers, 2008).

Contractility is affected by disease processes such as myocardial infarction, myocarditis, cardiomyopathies, myocardial ischemias, and inflammatory, immune or neurohumoral changes.

According to Brashers preload increases due to excess in vascular volume which can result from intravenous fluid administration, renal failure and mitral valvular disease (2008). Frank-Starling's law states that increasing the ventricular end-diastolic volume will cause a stretching effect of the myocardium causing a stronger contraction which results in improved cardiac output; however prolonged increased preload will ultimately lead to decreased contractility as this myocardial stretching will cause sarcomere dysfunction.

Aortic valvular disease or hypertension is responsible for increases in hypertrophy. Brashers explain hypertrophy results in deposition of collagen between myocytes, causing ventricular remodeling consequently reducing the contractility properties of the myocardium resulting in a dilated and less compliant ventricle (2008).

The vicious (in this case) renin-angiotensin-aldosterone system gets activated by poor cardiac output and decreased renal perfusion. Our body's natural instinct to maintain homeostasis is stimulated.

Barrow receptors which detect a decrease in perfusion stimulate the sympathetic nervous system to cause further vasoconstriction and antidiuretic hormone is released by the hypothalamus causing the kidneys to hold on to fluid. Mr. Jones fits the above criteria exhibiting symptoms of dyspnea, edema, has the classic HF murmur S3 gallop, is hypertensive and reports decreased urine output with weight gain. Diastolic heart failure (DHF) is associated with delayed relaxation and increasing left ventricular rigidity which prevents adequate filling and decreases the ability to properly eject blood (Redderson, 2008).

DHF occurring singly is described as pulmonary congestion with a normal cardiac output and stroke volume. Brashers describes DHF as the causative condition of 40% to 50% of all cases of left heart failure with a higher incidence in women (2008). Increased pressure at the end of diastole in the left ventricle is reflected back within the pulmonary circulation causing pulmonary congestion. Causes of DHF include myocardial ischemia, left ventricular hypertrophy induced by chronic hypertension, valvular diseases, cardiomyopathies and pericardial diseases.

During exercise individuals with DHF are not able to compensate for the increased demands, therefore the heart is not able to increase cardiac output since the left ventricle is hypokinetic. Signs and symptoms include dyspnea on exertion, fatigue, evidence of pulmonary edema, hypertension, coronary disease and valvular disease (Brashers, 2008).

Mr. Jones could have DHF as he does present with appropriate symptoms however his symptoms are more indicative of SHF and the test results will help differentiate between the two.

Right heart failure may result from left heart failure due to increased pressures in the left ventricle which reflects back into the pulmonary circulation. The right ventricle is not designed to cope with high pressures therefore it hypertrophies and fails. In the absence of left heart failure the cause is related to hypoxic pulmonary disease such as COPD, ARDS and cystic fibrosis causing pulmonary hypertension. Cardiac related conditions which affect contractility include pulmonic valvular disease, myocardial infarction, and cardiomyopathies (Brashers, 2008).

Signs and symptoms include decreased cardiac output during exercise; EKG shows right ventricular hypertrophy, jugular venous distension, peripheral edema and hepatosplenomegaly. Mr. Jones has a long history of smoking and he continues to smoke despite being diagnosed with emphysema five years ago. Tobacco use is the primary risk factor for developing COPD. Mr.

Jones's symptoms as well as his history and risk factors support a combination of left heart failure as well as right heart failure.

Diagnostic Screening and Evaluation: Radiographic ; Laboratory Diagnostic blood work is ordered in order to support the condition suspected and rule out other conditions that may mimic HF, as well as to determine the severity of HF. EKG will identify the heart rhythm, right ventricular hypertrophy as well as type natriuretic peptide which is secreted by the heart in order to maintain fluid balance, elevated levels support HF. CBC will rule out anemia  
<https://assignbuster.com/multisystem-case/>

and infectious processes. CMP will give us an indication of electrolyte status, renal, hepatic and pancreatic function.

Thyroid profile will show thyroid function, as it can also affect cardiac function, lipid profile will show good and bad cholesterol levels.

HGBAIC shows glycemic control for the past 3 months, ABG will demonstrate respiratory and metabolic status. Cardiac enzymes will determine if Mr. Jones is actively experiencing a myocardial infarction. CXR can support pulmonary congestion along with cardiomegally infiltrates, pleural effusion as well as differentiate COPD. A 2D Echo will show valvular function, kinesis of the myocardium as well as ejection fraction.

Cardiology consult is requested because cardiologists are specifically trained to treat HF. Mr. Jones's results were as follows: CBC, thyroid levels are normal; CMP shows elevated creatinine which indicates renal insufficiency. Lipid profile supports hyperlipidemia. Cardiac enzymes are negative, BNP is grossly elevated, and HGBAIC shows very poor glycemic control for the past three months.

ABG shows a compensated pH with a PaO<sub>2</sub> of 69. CXR reveals cardiomegally, and gross pulmonary congestion, EKG sinus tachycardia, right ventricular hypertrophy.

Echo cardiogram confirms an EF of 30% and right and left ventricular hypertrophy and mitral valve stenosis. In 2001 and again in 2005 the American College of Cardiology (ACC) and the American Heart Association (AHA) collaborated to create a frame work which allows providers to

understand the progression and HF. Heart failure progression is defined in four stages A, B, C, and D, beginning with at risk patients all the way to end-stage disease (Rasmusson, 2006). Treatment Interventions & Rationale Dr.

Heart reviews all the available information and places Mr.

Jones in Stage C class of HF. Mr. Jones exhibits acute decompensated symptoms such as dyspnea, lower extremity edema, pulmonary congestion, remodeling of the left ventricle from chronic hypertension resulting in structural changes. Also important to mention are the co-morbidities, such as Diabetes and COPD. According to Rasmusson treatment options must focus on reduction of morbidity and mortality.

Pharmacologic agents include ACE inhibitors, ARBs, beta-blockers and aldosterone antagonists, as well as iuretics and digoxin.

Treatment goal is to block neurohormones preventing the cycle of decreasing contractility, increasing preload and afterload, and relieving pulmonary congestion (2006). In the acute treatment phase emphasis is placed on stabilizing hemodynamics, correcting fluid volume, determination of etiology, and reversing conditions that can be reversed. Long term care includes vital patient and family education, appropriate titration of pharmacologic agents, salt restriction, and possibly cardiac resynchronization therapy (CRT).

CRT refers to bi-ventricular pacing which allows synchronization of the left ventricle consequently improving the ejection fraction (EF) which is normally 55%-70% (Brashers, 2008). Patients with an EF .



12 seconds are at a high risk for arrhythmias and sudden cardiac death therefore an implantable cardiac defibrillator is recommended (Rasmusson, 2006). Treatment for DHF focuses on improving ventricular relaxation, and prolonging diastolic filling times in order to reduce diastolic pressure.

Inotropic drugs are not although digoxin may be used in patients with atrial fibrillation in order to achieve rate control (Brashers, 2008). Prevention Mr. Jones presented to the ED with exacerbation of HF related to non-compliance with medication regime.

In this case patient education is of up-most importance. Hospitalization prevention is important due to the exorbitant costs. Patient education will focus on medication compliance, and signs and symptoms of exacerbations. Having the ability to recognize early symptoms will allow Mr.

Jones to visit his physician, which could adjust the medication regime, impose a fluid and salt restrictions, as well as provide aggressive diuresis at the office in order to prevent a hospital admission. A social worker consult should be arranged in order to provide assistance with indigent issues.

Chronic Disease Review: COPD or Emphysema Chronic obstructive pulmonary disease includes pathologic lung changes consistent with emphysema or chronic bronchitis (Brashers, 2008). There is a permanent enlargement of gas exchange airways in conjunction with destruction of alveolar walls with out apparent fibrosis.

Loss of elastic recoil is the causative factor of airflow limitation. The major cause of COPD is cigarette smoking even though childhood respiratory

infections and air pollution are known to be contributing actors (Brashers, 2008). Signs and Symptom Clinical manifestations of COPD include, dyspnea, wheezing, and prolonged expiration.

Individuals will have a classic barrel chest appearance. Late in the course of COPD patients will experience chronic hypoventilation, polycythemia and cor pulmonale also known as right heart failure.

Fatigue, weight loss, poor appetite as well as sleep disturbance may occur. Mr. Jones does have dyspnea, however it is unclear if it is related to the HF or COPD, nonetheless his history and test results do support a combination of conditions all exacerbated by his non-compliance with the prescribed medication regime (Kara, 2005) Pathophysiology The irreversible process begins with destruction of the alveolar septa consequently increasing the volume of air in the acinus.

Pollutant particles stimulate inflammation resulting in alveolar destruction and loss of elastic recoil of the bronchi.

This destruction produces bullae and blebs which are not effective in gas exchange resulting in hypoxemia due to ventilation - perfusion mismatching. The loss of elastic recoil reduces the volume of air that can be expired making expiration difficult and causing air to become trapped in the lungs. Hyperexpansion is the result of trapped air, which stresses the muscles of respiration, therefore late in the course of disease hyperventilation and hypercapnia develops (Brashers, 2008).

In non-smokers and individuals who develop the diseases before the age of 40 the causative factor is a rare genetic condition, which involves a deficiency of  $\alpha_1$ -antitrypsin which does not inhibit proteolysis in the lung tissue (Kara, 2005).

Diagnostic Screening and Evaluation: Radiographic & Laboratory Pulmonary function testing, arterial blood gas, high-resolution computed tomography and chest x-ray are used for diagnoses. Pulmonary function measurements, such as vital capacity (VC) and particularly forced expiratory volume (FEV1) are helpful in determining the stage of the disease.

In 1998 the Global recommendations for COPD management based on the latest scientific evidence available to date (Kara, 2005). COPD can be classified into four stages Stage I - Stage ' V, from mild where the individual is not even aware the condition exists to very severe where essentially respiratory failure is present based on the results of FEV1 and patient's symptoms. Mr.

Jones appears to be in Stage II to Stage III due to fatigue, shortness of breath and recent exacerbation. Again important to mention is that Mr. Jones has multiple processes involved therefore his symptoms are caused by a cumulative effect.

Treatment Interventions & Rationale Treatment for COPD is based on primary prevention, relieving symptoms, improvement of overall health status and exercise tolerance, diligent treatment of exacerbations and complications (Kara, 2005). Educating on smoking cessation and ensuring immunizations are up to date is crucial.

<https://assignbuster.com/multisystem-case/>

Acute and chronic symptoms will be managed with bronchodilators such as ipratropium and B2-agonists, in severe cases the use of methylxanthines, intravenous, inhaled or oral steroids and home oxygen may be required. Adequate nutrition is also very important.

Lastly pulmonary rehabilitation is aimed at improving functional capacity and quality of life (Kara, 2005). Mr. Jones exacerbation of symptoms is related to medication non-compliance and continued smoking. Nebulizer treatments with DuoNeb (albuterol/ ipratropium) every 4 hrs and prn via nebulizer and steroids would decrease the severity of symptoms.

In the event that tachycardia persists a switch to Xopenex/ ipratropium would be beneficial, as Xopenex has less incidence of inducing tachycardia. Supplemental oxygen was applied upon pulse oximetry reading.

Antibiotic therapy would be considered if there were signs of infectious processes. Prevention focuses on patient education and medication compliance. Assessment of Mr. Jones's cognitive status is important to facilitate educational conversation that he would understand.

Indigent support in order to encourage medication compliance is important although realistically not always available. Chronic Disease Review: Rheumatic Fever Rheumatic fever is an inflammatory disease caused by the group A  $\beta$ -hemolytic streptococcus, characterized by inflammation of the joints, nervous system and heart.

When not appropriately treated, rheumatic fever will cause scarring and deformity of cardiac structures (Brashers, 2008). Signs and Symptoms

Rheumatic fever often exhibits symptoms that are common to other conditions such as nausea, vomiting, abdominal pain, fever, arthralgia, lymphadenopathy and epistaxis and fever. According to Kara the American Heart Association and the World Health Organization developed the following criteria for diagnosis purposes: carditis, erythema marginatum, acute migratory polyarthritis and chorea (2005).

Pathophysiology Rheumatic fever occurs as a consequence to a pharyngeal infection by group A  $\beta$ -hemolytic streptococcus which causes an abnormal humoral and cell-mediated immune response.

Brashers explains; the immune response cross-reacts with molecularly similar self-antigens on brain, muscle, heart and joints resulting in an inflammation that may subside before treatment; however damage to the heart valves remains. Individuals with CHF and pericarditis suffered significant damage.

Endocardial inflammation can cause swelling of the valve leaflets and aggregation of clumps of vegetations containing platelets and fibrin become deposited on valvular tissues causing stenotic valves. If the inflammation is able to penetrate the myocardium it may cause cardiomegaly and left heart failure due to fibrin deposits also known as Aschoff bodies (Brashers, 2008). According to the test results Mr. Jones has mitral valve stenosis, cardiomegaly and HF.

Unfortunately Mr. Jones had rheumatic fever as a child which may be the causative factor of his HF.

Diagnostic Screening and In the acute phase rheumatic fever is diagnosed based on clinical symptoms plus by positive throat culture for group A  $\beta$ -hemolytic streptococci, antistreptolysin O antibody titers ; 250 Todd units, elevated values of anti-DNase B, antihyaluronidase, antistreptozyme, WBC, ESR, and CRP (Kara, 2005). Treatment Interventions ; Rationale Therapy is focused on eradicating the streptococcal infection through appropriate antibiotic therapy. Other pharmacologic agents include NSAIDs, cardiac glycosides, corticosteroids, and diuretics (Brashers, 2008).

In the event that there is significant hemodynamic instability related to damaged valves than surgical intervention may be required. Conclusion In conclusion Mr. Jones presents with an intricate combination of symptoms requiring a systematic approach with focus on alleviating symptoms, and educating the patient on preventative measures. Is the history of rheumatic fever a key factor in the patient's extremely poor condition? It may very well be a component of Mr. Jones's issues.

Risk factors such as poor glycemic control, smoking, and poor nutritional status in addition to the co-morbidities associated with COPD, Diabetes, hypertension, hyperlipidemia and HF certainly add to the severity of the situation.

Serious consideration needs to be given to quality of life issues, at this point a Living Will and Code Status should be discussed with Mr. Jones. Frequent hospitalizations and chronic conditions can place a big burden on resources as well as emotional well being. Mr. Jones may require rehab prior to

discharge home, or even decide to try a long term placement facility for better medical management of his condition.

Mr.

Jones may require a surgical consult for valve replacement and/or a cardiology consult for a bi-ventricular pacer / automated implantable cardiac defibrillator. Much emphasis needs to be placed on education; most importantly smoking cessation, identifying early signs and symptoms of HF and COPD exacerbation and medical regime compliance. As nurses we have to quickly identify educational opportunities and provide our patients the necessary tools for them to actively accomplish positive and therapeutic change.