Example of essay on nursing: congestive heart failure

Experience, Failure

Introduction

Disease Process

Nursing Evidence based Practice

Nursing Care Plan

Conclusion

Introduction

Congestive Heart Failure is also known as heart failure. This happens when the heart becomes incapable of pumping blood efficiently to all organs in the body. Diagnosis is through physical examination. Patients often manifest with shortness of breath upon exertion and swelling in the both legs. Confirmation of the diagnosis is through echocardiographic evaluation. On many occasion congestive cardiac failure could be secondary to hypertension, type 11 diabetes or kidney disease (Goldman, 2011). Essentially, the significance of heart failure relates to ischemic heart disease, myocardial infarction; valvular heart disease and cardiac arrest. Besides, any situation in which situations where there is high output implicating ventricular systolic function heart failure is implicated. It is indicative of the heart's inability to appropriately control blood volume resulting in overload. Frequently, when conditions such as beriberi, thyrotoxicosis, Paget's disease, arteriovenous fistulae, Paget's disease or arteriovenous dysfunctions are present heart failure could occur (Strömberg & Mårtensson, 2013).

More importantly, heart failure has created very high health expenditure, mostly hospitalizations costs. There has been an estimated 2% of the total National Health Service budget in the United Kingdom, and above $35 billion in the United States. Significantly physical and mental health is severely

affected reducing the quality of life individuals affected by the disease. Besides, most hospitalizations among over age 65 are due to heart failure in developed nations. Current figures show that 20 million people are affected by heart failure internationally. In United States of America alone 5. 8 million people are affected (McMurray & Pfeffer, 2010). As such, the purpose of this assignment is to provide me with an opportunity to develop an in-depth understanding of a congestive cardiac failure and apply it to a clinical scenario.

Disease Process

Etiology/ Risk factors

According to the American Heart Association there is still some difficulty in specifically identifying causes for congestive heart failure due populations' cultural diversity, changing age prevalence and diagnosis challenges. However, studies conducted among adults in United States of America have identified cigarette smoking 16%; Ischemic heart disease 62%; hypertension 10%; diabetes mellitus3%; obesity 8% and valvular heart disease 2% (He, Ogden, Bazzano & Vupputuri, 2010).

Among Italians studies reveal other 5%; dilated cardiomyopathy 32%; ischemic heart disease 40%; hypertension 11%; valvular heart disease 12%. Rarer causes were discovered to be HIV cardiomyopathy; systemic lupus erythematosus; beta blocker pharmaceuticals; viral myocardosis; substance abuse and obstructive sleep apnea (He et. al, 2010)

Pathophysiologic changes at the cellular, tissue, and organ level

The pathophysiology of heart failure indicates that any underlying medical problem initiating inefficiency in myocardium function contributes to pathophysiologic changes at the cellular, tissue, and organ level. Consequently, a numbers of conditions can initiate the damage and consequences thereafter. Precisely, when myocardium is damaged reduction in force contraction develops as the first sign of inefficiency. This occurs from ventricular overloading. A healthy heart functions physiologically to compensate for increased ventricular filling with comparable exertion of force. This mechanism is called Frank–Starling law of the heart. A damaged myocardium is incapable of executing this function. Essentially, the ability to coordinate cross-link actin and myosin filaments when the heart muscle is overstretched become limited (Mueller, Scholer & Laule-Kilian, 2010).

Two other major pathophysiologic changes occurring at the cellular, tissue, and organ level are limited stroke volume and spare capacity. Stroke volume refers to the amount of blood the myocardium could release at each contraction. A reduction occurs because of increase in either end systole/ diastole. This mechanical change is directly associated with limited myocardial contractibility. Spare capacity relates to heart reserves. Myocardial insufficiency forces the heart to work much harder than usual. Consequently, spare capacity becomes limited (McMurray & Pfeffer, 2010). Subsequently, increased heart rate develops as a compensatory mechanism for the inability to manage systolic and diastolic rhythms. The myocardium enlarges which show significant increase in the heart’s size. Ventricles also

enlarge in an effort to help the failing heart. Eventually, the pathophysiologic effect manifests as limited cardiac output with increased heart pressure. Ultimately, arterial blood pressure drops; sympathetic stimulation is heightened; blood flow to organs is minimized; increased production of vasopressin creating fluid retention neuroendocrine hormones blood levels become chronically high and skeletal muscle atrophy due to poor blood supply. The major pathophysiology concerns are the effects of systolic and diastolic dysfunctions on organ systems in the body (Mueller et. al, 2010).

Clinical manifestations and complications with their underlying pathophysiology

Congestive heart failure symptoms are based on left and right sided changes. Significantly, these are taken into account since the left and right ventricles provide circulation to separate parts of the heart. It is important, however, to understand, that congestive heart failure does not pertain only to backward failure. Many exceptions to this rule exist. For example, in left to right division of heart failure left sided forward failure symptoms tend to produce overlapping manifestations with right sided backward failure. Frequently, right sided failure produces left sided decompensation. Ultimately, patients manifest both left and right sided symptoms (Mueller et. al, 2010).

Left- sided failure often manifest with respiratory symptoms including tachypnea, rales, cyanosis and pulmonary oedema. In addition symptoms related to ventricular failure can emerge. They include displaced apex beat; gallop rhythm; heart murmurs and backward failure. Backward left ventricular failure produces pulmonary vasculature congestion whereby the

patient experiences dyspnea and may resort to sleeping with pillows for comfort. Patients also complain of fatigue and easy exertion when trying to complete minimal tasks such as walking (Mueller et. al, 2010).

Right sided failure manifests as peripheral oedema, hepatomegaly and hepatojugular reflux. Right sided backward failure presents as systemic capillaries congestion, which occurs as bloating. This could be concentrated in the abdomen or generalized over the body. Often the condition is classified as anasarca or peripheral edema. Ankle edema occurs if the individual stands for a long period of time or at the sacrum when lying down. Jaundice and coagulation dysfunctions may develop when blood supply to the liver is diminished. Biventricular failure produces a combination of both left and right sided failure. A marked sign, however, is lung dullness upon finger percussion (Mueller et. al, 2010).

Two major treatment strategies

The two major treatment strategies focus on relieving symptoms and limiting progress of the disease from reaching a later stage. Reversible causes are also addressed in the first line intervention of relieving symptoms. Primarily, treatment intervention includes education pertaining to life style changes and drugs. In relieving symptoms measures are taken to address acute decompensation issues. These include attempts at re-establishing appropriate perfusion and oxygen to end organs. A combination of vasodilators such as nitroglycerine; furosemide (diuretic) and applications of non-invasive positive pressure ventilation are used (NIPPV) (McMurray & Pfeffer, 2010).

Treatment aimed at limiting the progression of congestive cardiac failure target chronic management by limiting acute decompensation effects and extensive cardiac remodeling, which occurs as a pathophysiological consequence. Cardiologists advocate that first-line therapy ought to be angiotensin-converting enzyme (ACE) inhibition because it improves myocardial function. ACE inhibitors include captopril, enalpril, ramipril and lisinopril (McMurray & Pfeffer, 2010).

Randomized trials show that there has been significant survival rate and quality of life improvement when these drugs are used initially in the treatment of heart failure.

Mortality rates were reduced and left ventricular dysfunction was greatly adjusted. Always a combination of vasodilators; beta blockers, oral loop diuretics, ACE inhibitors or angiotensin receptor blockers, vasodilators accompanied the first line therapy. When extensive cardiomyopathy is present aldosterone receptor antagonists is added to the drug regime (McMurray & Pfeffer, 2010).

Importantly, management of anemia is very important to the survival rate of congestive cardiac patients. With inability of the myocardium to pump blood into end tissue manifestations of poor blood supply to these areas would be evident as a direct result of heart congestive heart failure. Consequently, treatment with parenteral iron has revealed great improvement in survival rates as well as quality of life (McMurray & Pfeffer, 2010).

Nursing Evidence based Practice

Aronow, Rich and Ahmed (2012) conducted an observational research pertaining to In-Hospital Cardiology Consultation and Evidence- Based Care

for Nursing Home Residents with Heart Failure. Their objective was to discover ‘ the association between cardiology consultation and evidence-based care for nursing home (NH) residents with heart failure (HF)’ (Aronow, Rich & Ahmed, 2012, p 2). These researchers sampled 646 nursing home residents discharged from 106 hospitals located in Alabama with a diagnosis of congestive heart failure during 1998-2001(Aronow et. al, 2012).

Left Ventricular Ejection Fraction (LVEF) Assessment according to these researchers is used for determining application of evidence based therapy. It was discovered that nursing home patients did not receive adequate cardiology consultation neither Left Ventricular Ejection Fraction (LVEF) Assessment. Consequently, evidence based care for congestive heart failure care was not administered to them (Aronow et. al, 2012).

The conclusions emerging from this study indicates that congestive cardiac nursing home (NH) residents happen to be a distinct subset of heart failure patients. There is very limited evidence to guide to their therapy due to insufficient Left Ventricular Ejection Fraction (LVEF) Assessment. Consequently, clinicians functioning in nursing home environments caring for congestive heart failure residents ought to individualize therapy. They also cited that this may be the explanation for residents in nursing homes noncompliance with Therapuetic interventions (Aronow et. al, 2012).

Indirectly the nursing evidence based practice implication is that congestive heart failure patients ought to be thoroughly screened before appropriate therapeutic interventions could be prescribed. Nurses respond to doctors’ orders. If doctors have no evidence to prescribe care then nurses are left at the same disadvantage when designing interventions for congestive cardiac

patients in nursing homes. Individualized interventions ought to be considered. It would appear that due to the high cost of health care in America nursing homes in trying to cut cost has limited Left Ventricular Ejection Fraction (LVEF) Assessment. Precisely, nursing home residents are in long term care and they deserve evidence based interventions. The researchers concluded that maybe health care administration estimate that patient with congestive heart failure will die anyway; hence, the reason that they are in a nursing home (Aronow et. al, 2012).

However, quality of life is the important event in the little life left of these residents. In my opinion nurses should advocate for evidence based interventions at all cost for residents in long term care facilities. Medicare does not pay for long term care. Many of these residents have to give up their property among numerous other amenities of life to be in a nursing home. It is only right that they enjoy the best quality of life form the sacrifices they have made for their country as United States Citizens.

M. S is a 68 year old woman arrived at the Emergency Room brought in by the ambulance. The client complained of shortness of breath for about one week. It became worse today why she called 911. She experienced persistent coughing, especially, during the night.

There were no known allergies. Medications she was taking were clonidine; digoxin and Insulin 70/30. Besides she gave a history of Diabetes Mellitus type 2. She admitted to having hypertension and heart problems (Congestive heart Failure Case study).

Physical examinations revealed dusky nailbeds; upright sitting; difficulty in talking; skin cool and moist; ankle edema present+++ and rales in the

middle right as well as bilateral lobes. Vital signs read BP 218/110; HR 120; RR 34 and T 38. 1. Laboratory values Dig 0. 6; Na 135; K 3. 9; Hct 33%; ABGs (Creat 1. 9); ph7. 48; BUN 10; paCo 30; paO2 80; Sao2 95% and Hco3 24.

CXR Bilateral pulmonary congestion; 12 Lead EKGSinus tachycardia with occasional PVCS and Old Anterior MI (Congestive heart Failure Case study).

Nursing diagnoses relevant to this case study are ineffective breathing pattern; readiness for enhanced fluid balance; readiness for enhanced comfort

Nursing Care Plan

Conclusion

The foregoing scholarly exposition highlighted congestive heart failure from a pathophysiological perspective with the purpose of obtaining an opportunity to develop in-depth understanding of the disease and apply it to a clinical scenario. Treatment modalities were explored and a case study scenario articulated along with a supporting nursing care plan

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