LEFT HEART AND EXACERBATION OF LEFT HEART FAILURE

Concept Map

Aetiology: left heart failure is a severe fatal state in which the left side of the human heart fails to properly pump adequate amount of blood into the circulatory system. The left heart receives oxygenated blood from the lungs and pump out the rest throughout the system. In case of left side heart failure, the ability of the left heart to pump blood is decreased and the system does not receive proper amount of oxygen. This leads to fatigue. The increase of pressure within lung veins causes fluid accumulation, which in turn leads to pulmonary edema and dyspnoea.

Pathogenesis: the left side of the heart work harder to pump sufficient amount of blood. In diastolic dysfunction, the left ventricle fails to relax as the muscles become stiff. The heart, thus cannot fill with proper amount of blood while resting between individual beats. However, the left ventricle fails to contract normally in systolic failure. So, the heart fails to pump out blood into the circulation.

Clinical manifestation: produces frothy mucus, reduced production of urine, shortness of breath, fatigue, faintness, irregular pulse, palpitations, paroxysmal nocturnal dyspnea, and fluid retention.

Diagnosis: physical tests may demonstrate irregular heartbeats and raised breathing rate. Abnormality in heart motion could be felt while touching the wall of the chest. Crackling sounds can be heard on auscultation and breathing sounds could be decreased. Swollen legs are noticeable, depressions form, if pressed. Recommended tests are: blood test to check liver, kidney and thyroid function, chest X-ray, electrocardiogram, coronary angiography, ultrasound and heart stress test.

Treatment: treatment strategies need to be developed depend on the awareness of various compensatory mechanisms (peripheral edema, dyspnoea, loss of appetite and so on). Treatment goals are reducing symptoms, relieving stress on heart, improve the chances of survival. Besides, patients should be well informed about lifestyle modifications like controlling blood pressure, regular exercising, cessation of smoking and drinking, adherence with recommended medications, diet, proper follow-up with doctors. First-line therapy should include angiotensin receptor blockers, angiotensin converting enzymes. Palliative care is also recommended that not only manages symptoms but also helps in advanced and holistic care planning.

Prognosis: could be evaluated in different ways: cardiopulmonary prediction testing and clinical predictions. A prominent example in this context would be ‘BWH’ rule, which shows that patients with an elevated systolic pressure (more than 90mmHg), serum sodium more than 135 mmol/L, no alterations in ST-T waves, have comparatively less chance of developing major complications. However, patients’ responses also depend upon their gender, age and cause of heart failure.

Prevention of left heart failure: follow dietary guidelines, promote knowledge and awareness on recommended medications, continuous patient education and involvement in physical activities, level of hardness depend upon the age and gender.

Answer to question1.

The pathogenesis of an acute exacerbation of heart failure includes reduction of cardiac output to the circulatory system. The primary indications are often demonstrated because of the effects on pulmonary circulation. Due to systolic dysfunction of the heart, the expulsion fraction is reduced and leave an increased amount of blood inside the left ventricle (Marín-García, 2016). In case of left-sided heart failure, the heart fails to adequately pump the blood out into the entire system. The pumping action of the heart enables to move oxygenated blood from the lungs towards left atrium, left ventricle and then to the rest of the human system. The left ventricle of the heart provides majority of the pumping power of the heart, hence, it is comparatively larger than the other heart chambers of and crucial for normal functioning. During left-sided heart failure, the left side of the heart work harder to pump adequate amount of blood. There are majorly two types of mechanisms evident in left heart failure: diastolic and systolic failure (Garcia, 2008). In diastolic dysfunction, the left ventricle fails to normally relax as the associated muscles become stiff. The heart, thereby, cannot fill with blood in adequate amount while resting between individual beats. Whereas, the left ventricle fails to contract normally in systolic failure. Therefore, the heart fails to pump out blood into the circulation. The clinical manifestations such as: severe dyspnoea, atrial fibrillation, crackles sound, elevated blood pressure of Mrs. Brown, a 78years old female support the diagnosis of an exacerbation of heart failure. The abnormally increased volume of blood in the left ventricle increases pressure back to the left atrium and pulmonary veins. This in turn affects the normal alveolar drainage and favors movement of fluid to the parenchyma of the lungs from the capillaries. This also affect gaseous exchange. Thus, left-sided heart failure leads to severe dyspnea. During heart failure, heart attempts to compensate its lost pumping power by beating at a faster rate to maintain the blood flow throughout the system and causes atrial fibrillation. Probably, this is the same reason behind Mrs. Brown's elevated pulse rate. As heart fails, Mrs. Brown has developed clinically manifested symptoms as compensatory mechanisms, which take place while failing heart strives to maintain its proper functions. Elevated blood volume hampers the normal pressure of the blood flow and therefore, may clinically manifest elevated blood pressure. Inability of the heart to pump out adequate amount of blood pushes back the blood to the veins, which is taken through the lungs. This further increases pressure buildup within the blood vessels and fluid moves into the alveoli. Such fluid buildup inside the lungs generates crackles sound and is heard on auscultation. Normal S_PO2 or blood oxygen saturation level should be ranged between 94%-99% (Iwasaku

et al., 2008). But, clinical report showed Mrs. Brown's S_PO2 level was 85%, which is far below the normal range. Oxygen saturation shows how much oxygen is carried by the blood within the system. In case of a heart failure, the heart fails to pump out adequate amount of blood. Hence, over time, inadequate amount of oxygen affects individual organs inside the system that also include the lungs. This further lowers the oxygen saturation level.

Answer to question2.

Two high priority nursing strategies to mange Mrs Brown would be to control cardiac output and supporting her with adequate ventilation and tissue oxygenation.

Control cardiac output

Strategies	Rationale
Evaluate heart rate, apical pulse, note dysrhythmia	Tachycardia is present to compensate reduced ventricular contractility. Atrial fibrillation is a common dysrhythmia related to heart failure.
Palpate peripheral pulse	Reduced cardiac output could be identified by popliteal, radial pulses that are irregular to palpation (Selby & Trupp, 2009).
Monitor blood pressure	Blood pressure may be increased due to an increase in systemic vascular resistance. Sometimes, body fails to compensate and intense hypotension may take place.
Inspect cyanosis, skin color	If peripheral perfusion reduces, pallor is noticeable, which is secondary to improper cardiac output. Cyanosis may take place in refractory heart failure.

Adequate ventilation and tissue oxygenation

Strategies	Rationale
Auscultate breathing sound, note wheezes and crackles	Shows pulmonary congestion, accumulation of secretions.
Ensure bed rest, elevated bed-head to approximately 30degrees, support arms with	Lessens oxygen demand, improves lung inflation.

pillow	
Administer supplemented oxygen, as directed by the doctor	Increases concentration of alveolar oxygen that reduces tissue hypoxemia.
Monitor arterial blood gases	In pulmonary edema, hypoxemia could be severe and compensatory changes are evident in heart failure.

Answer to question3.

a) Mrs. Brown was administered with furosimide and glyceryl trinitrate. Furosimide is a potent loop diuretic that inhibits reabsorption of water in the nephrone by obstructing the action of socium potassium chloride cotransporter in the loop of Henle. Furosimide reduces intravascular volume that decreases the pressure in central veins, pulmonary vasular and left and right heart flling preassure (Sica, 2007). Venous capacitance raises and the intrapulmonary fluid returns back to circulation. This indicates administration with furosimide in heart failure is highly significant.

Glyceryl trinitrate activates guanylate cyclase that stimulates cyclic guanosine 3`, 5`-monophosphate synthesis, which activates protein kinase dependent phosphorylation in smooth muscle cells. This ultimately, give rise to myosin light chai dephosphorylation in the smooth muscle fiber (Gupta et al., 2013). It further supports calcium ion release and thereby, vasodilation and relaxation of smooth muscles. As glyceryl trinitrate diminishes pulmonary vascular resistance and helps in dilation of veins and arteries, it is significant to use in treating heart failure.

b) Adverse effects of furosemide include ringing in ears, loss of hearing, dark urine, itching, pain in upper stomach radiating to back, vomiting and nausea. Adverse effects of glyceryl trinitrate include dizziness, headache, vomitting, low blood pressure, diarhoea, soreness, and itching (Andersen, 2009). Hence, nurses caring for Mrs. Brown should monitor vital signs to avoid any kinds of side-effects while administering furosemide and glyceryl trinitrate. Besides, both the medicines were amdinisterd intravenously. Hence, the respective care professionals should monitor the presence of inflmmation in the site of insertion. If overllooked, patient may feel pain or it may cause severe infection. The nurses should also check for the presence of any allergic response while administering with these medicines.

C) Nurses should measure respiratory rate, pulse rate blood pressure, presence of crackling sound on auscultation. If the measures demonstrate normal range, it would indicate that the patient has responded well to the administered drug.

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