

Cardiac nursing case study myocardial infarction



Cardiac nursing is a specialized care of patients suffering from various conditions of the cardiovascular system. It can be either problems of the Coronary artery or the vascular system. Coronary artery disease is caused by atherosclerosis; a progression of fatty tissues building up a plaque on the walls of the artery (Zafari, 2011). The plaque causes the narrowing of the artery which slows or stops blood flow to the heart. Once the blood flow is slowed or stopped the patient may experience chest pain, shortness of breath and heart attacks; medically termed as Myocardial Infarction (MI). In many people, atherosclerosis can remain silent (causing no symptoms or health problems) for years or decades. Atherosclerosis can commence as early as the teenage years, but symptoms or health problems usually do not occur until later in life when the arterial narrowing becomes severe.

Another cause of decreased blood supply is artery spasm (Riaz, 2010). Sometimes a coronary artery momentarily contracts or goes into spasm. When this happens the artery narrows and blood flow to the heart muscle decreases or stops. A spasm can occur in normal-appearing blood vessels as well as in vessels partly blocked by atherosclerosis. A severe spasm can cause a Myocardial infarction.

Myocardial Infarction (MI) is the death of some mass of the heart muscle caused by inadequate blood supply (Aehert, 2009). The heart muscle is permanently damaged (Bolooki & Askari, 2010). Some of the causative factors of myocardial infarction include smoking, large amounts of fatty food consumption, lack of exercise and drug use. Smoking causes MI by decreasing oxygen to the heart, increasing blood pressure and heart rate,

increasing blood clotting and damaging to cells that line coronary arteries and other blood vessels.

Diagnosis of myocardial Infarction is done through a twelve lead Electrocardiography and a series of biomarkers; also known as cardiac enzymes. Troponin I and Troponin T are the most specific biomarkers. These are quite expensive and not available in Labasa hospital. Creatinine kinase (CK) and CK-MB are essential for confirming the diagnosis. An elevation in the serum concentration occurs 4 to 6 hours from the onset of symptoms, some patient may have a rise as long as after 12 hours (Ryan, 2008). This assignment is a case study of a client who is admitted in the Coronary Care Unit with Myocardial Infarction.

Mr. X is a 48 year old Indian male admitted to the Coronary Care Unit on the 18th of April 2011 at 7pm. He presented to the Emergency room at 4pm with the chief complaint of left sided chest pain since three hours prior to presenting to hospital. The pain was crushing in nature, associated with sweating, shortness of breath and had a sense of impending doom. A twelve lead Electrocardiograph (ECG) showed changes are distinctive ST elevations in leads I, aVL, V2, V3, V4, V5 and V6. These changes are consistent of Anterolateral Myocardial Infarction. Laboratory investigations done showed a normal full blood count, normal levels of Urea, Creatinine, Sodium, Potassium, chloride, urates and liver enzymes. Mr. X's cholesterol and triglyceride levels were markedly elevated. The cardiac enzymes were slightly elevated initially but rose drastically after six hours.

Mr. X is tall and slim built, 180cm and weighs 77kg. He was admitted two years ago with stroke. He is a known case of hypertension and attending medical clinic. His parents are hypertensive. In his social history patient is a Hindu priest by profession. He denies any use of alcohol or kava. He is a smoker, claims to be smoking 4 to 5 rolls of cigarette per day. He is married with four children. He is sexually active and does not do any form of exercises.

Objective Data: Mr X is tall and slim built. Vital signs; BP: 140/93 of Hg, pulse rate of 63 per minute, temperature of 36 degrees Celsius and respiratory rate of 22 per minute. Skin was warm to touch, no cyanosis. Level of consciousness; alert and oriented to time, place and people. Lung fields are clear and air entry is normal bilaterally, breath sounds were normal.

Cardiovascular system; no visible pulsation, thrills or heaves detected. S1 and S2 heard. Peripheral pulses felt bilaterally, no edema or cyanosis seen. Gastrointestinal tract; no difficulty in swallowing, abdomen is soft and non tender on palpation, no mass felt. Genitourinary tract: no abnormalities detected. Musculoskeletal system: normal muscle mass, good tone and gait. Power on the right side 3/5 and on the left side it was 5/5, it is the residual effect of the stroke he had two years ago.

Acute Myocardial infarction (MI) occurs as a result of thrombotic occlusion of the coronary artery and causes irreversible cell injury and necrosis (Woods, Froelicher, Motzer & Bridges, 2005). There are 4 major risk factors which are responsible for this heart attack; Hypertension also called as High Blood Pressure, Hypercholesterolemia, Diabetes, and Smoking (Riaz, 2010).

Anterolateral MI is the result of occlusion of the left anterior descending

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artery and occlusion of coronary branches supplying the lateral wall of the left ventricle. Three criteria for diagnosing MI as identified by World Health Organization standards include patient history of severe and prolonged chest pain, electrocardiography changes and serial enzymes (Woods. et al., 2005). More heart attacks occur between 4: 00 A. M. and 10: 00 A. M. because of the higher blood levels of adrenaline released from the adrenal glands during the morning hours due to increased adrenaline which contributes to rupture of cholesterol plaques (Kulick & Lee, 2011). Electrocardiography allows identification of regions of the heart affected.

During the initial phase of Myocardial infarction, Mr. X experienced chestpain, shortness of breath, profuse sweating and had a sense of impending doom. The presence of chest pain is a hallmark symptom in the diagnosis of acute MI (Woods. et al., 2005). The pain Mr. X initially experienced was due to coronary artery (responsible for supplying blood to the heart) being blocked, injury is caused to the heart muscle due to deprivation of blood and oxygen to the heart muscle. which causes chestpain and chest pressure sensation (Kulick & Lee, 2011). The shortness of breath is caused if the left ventricle is affected by the infarction, the cardiac output will be reduced (Woods, Froelicher, Motzer & Bridges, 2005). The heart is unable to pump enough supply of blood to the body (Lindberg, 2011). The sense of impending doom is caused by the release of adrenaline and other catecholamines as a component of the compensation mechanism, also the real fear of death is present due to the nature of disease and preconceived ideas (Kulick & Lee, 2011) Profuse sweating is caused by Sympathetic activation (Zafari, 2011).

The immediate treatment received by Mr. X was 600 µg Glycerol trinitrate (GTN) sublingually to ease the pain. GTN is a vasoactive agent which is administered to reduce myocardial oxygen consumption, which decreases ischemia and relieves pain (Clayton, Stock & Harroun, 2007). He was also given Tablet Aspirin 300mg orally which is an antiplatelet; it reduces platelet aggregation and prevents thrombus formation thus reducing the risk of MI (Clayton, Stock & Harroun, 2007).

Mr. X was placed on a semi-fowlers position and given five litres per minute oxygen through a hudsons mask to assist in breathing. Mr. X was relieved from his anxiety when he was explained about his condition and treatment. He was also advised on the importance of taking complete bedrest and cooperating with the ongoing treatment. Mr. X was stabilized in the Emergency room for three hours before transferring to the Coronary Care unit (CCU). Once the diagnosis is confirmed the primary objective is to open the blocked artery and restore blood flow; this process is termed as reperfusion. Assessing from the presentation and clinical manifestation of Mr. X, he was a possible candidate for Streptokinase infusion but it was contraindicated since Mr. X had a previous history of stroke with residual effects. Streptokinase can cause symptomatic intracranial haemorrhage in patients with previous history of stroke (Saver, 2011).

Once the artery is open, patients becomes pain free since the damage to the heart muscle ceases. By minimizing the extent of heart muscle damage, early reperfusion preserves the pumping function of the heart (Kulick & Lee, 2011). In order to achieve early reperfusion Mr. X was placed in a restful and comfortable environment and commenced on treatment. He was also

oriented to the Coronary Care Unit. Upon admission Mr. X was assessed by the nurse on duty and a list of problems were identified. He was having pain related to ischaemic myocardial tissue, anxiety and fear due to the change in health status and he was at risk of decreased cardiac output related to altered cardiac rate and rhythm. The expected outcome after medical care and nursing therapy is to relieve pain, Mr. X will verbalize reduced anxiety and fear as well as maintain adequate cardiac output.

The planning and implementation of therapy included administration of Injection Morphine 2.5 mg intravenously to relieve pain when tab Glyceryl trinitrate was unable to relieve pain. Mr. X was instructed to report all chestpain. He was encouraged to verbalize all fear and concerns. A cardiac monitor was attached to monitor the rate and rhythm of the heart. His vital signs were assessed every two hours for the first six hours then every four hours. He was monitored for signs of reperfusion: return of ST segment to baseline and reperfusion dysarrhythmias, example PVCs, bradycardia and heart block.

Mr. X was on complete bed rest for the first three days of admission with restrictions on having visitors. This was implemented to permit Mr. X to have maximum rest and to allow the healing process of the heart to take place. Complete bedrest reduces the workload of the heart (Woods, Froelicher, Motzer & Bridges, 2005). During admission he was served with low sodium and low fat and low cholesterol diet. Extra sodium in the body which is not eliminated from the body increases the blood pressure and increase the work of the heart. Thus Mr. X needs low sodium diet because he is already a hypertensive patient. Saturated fat increase blood cholesterol, Mr. X needed

a fat free diet for the reason that his blood tests reflected an elevated cholesterol level.

His Daily medication included Tablet Aspirin 150mg once daily, Tab Glyceryl trinitrite 600 µg p. r. n, Tablet Isorsobide dinitrite 10mg three times a day, tablet Simvastatin 20mg nocte and tablet Enalpril 5mg daily. Isorsobide dinitrite is a peripheral and coronary vasodilator responsible for increasing blood flow and improving collateral circulation, reducing preload and afterload, decreasing myocardial oxygen consumption and increasing cardiac output (Clayton, Stock & Harroun, 2007). Simvastatin from a group of drugs called HMG CoA reductase inhibitors, or “ statins.” It reduces levels of “ bad” cholesterol (low-density lipoprotein, or LDL) and triglycerides in the blood, while increasing levels of “ good” cholesterol (high-density lipoprotein, or HDL) (Clayton, Stock & Harroun, 2007).

On the the second day of admission Mr. X was assessed to be having slight chest pain, according to him it was vague therefore did not need intervention. He was relieved from profuse sweating. The nursing intervention for the day included oxygen administration level reduced to four litres per minute via hudsons mask. He continued to be monitored on the cardiac monitor and his vital signs were checked every four hours. He was able to tolerate his meals and medications without developing any complications.

Assessment on the third day revealed that there was no need for oxygen administration and was pain free. It was identified that Mr. X is out of immediate danger. Mr. Xs cardiac enzyme was repeated; it reflected

improvement as it was reduced from the one obtained after six hours of chest pain. On the fourth day of admission Mr. X was permitted to mobilize out of bed. He was visited by the Physiotherapist who advised him on need for exercise. A Dietitian counselled him on the importance of having a low sodium, low fat diet. On the fifth day of admission Mr. X was transferred to Mens Medical Ward since he was out of the danger phase of Myocardial infarction. In Mens Medical ward he continued to receive collaborative care in moving towards rehabilitation after Myocardial infarction. Cardiac rehabilitation can improve the heart's ability to function, lower the heart rate, and reduce the risk of dying or developing complications from heart disease (Aroesty & Kannam, 2009). Mr. X had another huge challenge to quit smoking as it a major contributor towards heart diseases. Cigarette smoking doubles the risk of developing coronary heart disease, and smoking cessation can rapidly reduce this risk (Aroesty & Kannam, 2009).

Mr. X was discharged from hospital on the sixth day. He was asked to attend medical clinic after six weeks. He was advised to quit smoking and commence on a balance diet; keeping in consideration his dietary needs and modification. He was also advised to commence on a daily routine of exercises. He was given medications to take home and encouraged to be compliance in taking his medications. His discharge medications included Tablet Aspirin 150mg daily, tablet Enalapril 5mg daily, tablet Simvastatin 20mg nocte, Tablet Isosorbide dinitrate 10mg three times a day and Tablet Glyceryl trinitrate 600µg p. r. n. Mr. X was advised to seek medical help if he experiences any chest pain which is not relieved by taking three tablet

Glyceryl trinitrate at five minute intervals. Angina can present in many ways especially by chest pain or discomfort (Maxwell, 2008).

In conclusion, Cardiac nurses need comprehensive understanding of the complex field of cardiovascular medicine and the ever changing advances in cardiac care technology. The skill to multitask and quickly evaluate cardiac symptoms and resolve problems is essential. Knowledge of proper cardiac health rehabilitation steps and the ability to effectively communicate the information to the cardiac patient and his family members is essential. Exceptional critical care nursing skills are required.