## Care, rationale and outcome in coronary care unit



Nurses are required to continue education and upgrading of skills to ensure their patients receive the best possible nursing care. Cardiac nursing is a dedicated nursing practice that gives focused and precise nursing interventions, that are governed by the best practice nursing standards using latest research based facts. Nurses need to have good technique and skill when performing health history and physical assessments to enable them to look after the person as a whole. When nursing patients, nurses need to understand the care they give and reasoning of why they deliver the cares in a certain way. A sound knowledge of assessment and observations help nurses plan, initiate and deliver health care. Without knowledge and rationales the nurse may not deliver cares in the correct manner or have the ability to know when to initiate them. Myocardial infarction is a common cause for admission into the Coronary Care Unit and this case study follows cares, rationales and outcomes in this setting.

Mr Smith (synonym for confidentiality) is a retired 58 year old man that was admitted to a Coronary Care Unit (CCU) via the Emergency Department (ED) of the Atherton Hospital. His admission diagnosis was an Anterior ST Elevated Myocardial Infarction (STEMI), which had already been treated with thrombolytic therapy. On the morning of his admission, he drove himself to the ED with chest pain. He presented with left sided chest pain that radiated to his left jaw and left arm which he scored 10/10 and described as " crushing". He was diaphoretic and hypertensive with nausea and vomiting. An ECG showed sinus bradycardia, rate of 60 bpm with hyperacute T waves in V2-V4, that progressed to ST Elevation. Thrombolytic therapy was administered 1 hour of his presenting to ED and within 2 hours of the initial

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chest pain that commenced at home. His ST segment was elevated approximately 8mm and continued to increase until 70 minutes post thrombolytic when he had 50% resolution of the ST elevation.

When he presented to the ED he was given oxygen, morphine, anginine, aspirin, clopidigrel and enoxaparin as first line pharmaceutical treatments. He was transferred that afternoon to Townsville. Mr Smith was not managed in Atherton due to the lack of cardiac catheter services and was transferred for a Percutaneous Coronary Intervention (PCI) the next day where he had a stent placed in his proximal area of his Left Anterior Descending Coronary Artery (LAD). Anterior MI's affect a large surface of the heart, thrombolytic therapy and PCI are the most effective way to treat them (Evans-Murray, 2008).

His medical history includes a previous STEMI and PCI in 1997,

hypercholesterolemia, depression, a ruptured bowel and neck injury from a Motor Vehicle Accident in 1977. Upon further questioning Mr Smith admitted to recently becoming " very short of breath" whilst mowing the lawn. His risk factors include ex-smoker ceasing in 1993, hypercholesteremia, and stress of brother dying 3 weeks previous. His current medications were aspirin 100mg daily, atorvastatin 20 mg daily and zoloft 200mg daily.

Upon arrival to a Townsville Coronary Care Unit (CCU), Mr Smith was pain free. He was connected to continuous cardiac monitoring and admission workup was attended, this includes admission paperwork, ECG, vital signs, mobile Chest x-ray and pathology tests. He was ordered and given stat doses of aspirin, clopidigrel and IV lasix. Mr Smith had an IVT running in his Left hand and an IVC in his Right hand.

During the next few days Mr Smith remained febrile 37. 6° with only a small elevation in white cell count (Huszar, 2007). Four days post infarction, Mr Smith became short of breath (SOB) in the shower and felt light headed; he was monitored in Sinus Rhythm with SaO2 of 95% on 3lpm via nasal cannula. On auscultation, crackles were heard in his lower bases. He was commenced on lasix 20mg daily. This was an indication that his Left Ventricle may not have been functioning adequately. An Echocardiogram was performed to see if the heart wall motion and valves were performing to their best ability (Kern, 2003). The report showed extensive akinesis of the septal, anterior and apex left ventricle wall. His Left Ventricle Ejection Fraction (LEVF) was 35%. Normal values for (LVEF) are 60-65% (Moser & Riegel, 2008). He was commenced on a Beta Blocker - Cavedilol 6. 25mg and Ramipril, which was commenced post PCI and decreased from 2. 5mg to 1. 25 mg. Use of these medications follow the criteria of the Reducing Risk in Heart Disease (Heart Foundation, 2007). He was sent to the cardiothoracic unit on day 5 with telemetry, to monitor for any changes in his cardiac condition (Jayasekara, 2009) and discharged two days later.

A systematic approach should be taken when attending to health history and physical assessment. Throughout the assessment, skin temperature, body odour, mood and appearance are observed. Patients need to feel comfortable with nurses so Mr Smith had the physical assessment explained to him and the reasons for performing it. (Brown, 2007) Mr Smith's physical assessment was completed in the morning prior to his PCI. He seemed relaxed with a jovial manner but at times did appear nervous. He was of a clean well kept appearance and looked younger than his 58 years.

Neuro – intact. Orientated to time, person and place, GCS – 15 and PEARLA. He had a good memory of the event.

Cardiovascular – monitored in sinus rhythm with frequent PVC's and runs of bigeminy. ECG attached. Febrile- low grade 37. 4 °, Pulse – 70 bpm, blood pressure 102/69, no peripheral oedema. Jugular venous pressure was approximately 4 cm's. Initially I could not palpate the apical pulse but when patient positioned onto his left side it was felt 5th ICS MCL. The reason it is felt is due to the apex of the heart comes into contact with the chest wall (Marieb & Hoehn, 2010) No thrills or heaves heard. Mr Smith was warm to touch but not diaphoretic. Upon auscultation of the carotid arteries no bruits were heard. Normal S1 and S2 heart sounds were heard upon auscultation. Good radial, carotid and femoral pulses, Normal 2+ – according to pulse volume scale (Lewis, 2007). Mr Smith did look pale and his haemoglobin was 121g/L.

Respiratory – rate of 18 per minute. Sao2 – 94% on 2lpm via Nasal cannula. Inspection of the thorax area revealed equal shape, size and symmetry of chest with nil use of accessory muscles. Trachea was midline. Lips and nail beds showed no signs of cyanosis. Diaphragmatic excursion was equal at 4 cms. Anterior, lateral and posterior areas revealed equal air entry, bilaterally in high and mid thoracic zones. Basal zones of thorax areas were bilaterally dull. No adventious sounds heard. Chest X-ray noted that some consolidation in bilateral bases which corresponds to the decreased air entry heard in the bases (Wang, Baumann, Slutsky, Gruber, & Jean, 2010).

Gastrointestinal – revealed an old scar midline under the umbilicus from previous MVA. Bowel sounds heard in all 4 quadrants. Abdomen was soft with no distension.

Mr Smith's upper and lower limbs and nail beds showed no signs of cyanosis or clubbing, ulceration or varicose veins. Capillary refill was normal – less than 3 seconds in all limbs. Range of motions and strength were bilaterally equal and normal in all 4 limbs. Dorsalis pedis and posterior tibial veins were felt on palpation and scored 2+ bilaterally (Lewis, 2007).

Acute coronary syndrome is a common cause of death. Myocardial infarction can have a good mortality rate if treated early. Treatment can be as basic as oxygen, ECG, observations, nitroglycerine through to thrombolytic therapy or a rescue angiogram/angioplasty (Overbaugh, 2009). One is not more important than the other and the patient's prognosis is the main concern.

Patient's complain of chest pain due to myocardial oxygen demand and supply mismatching. The coronary arteries supply the myocardium with blood supply, if the supply is interrupted by a clot, spasm or atherosclerotic plaque the myocardial oxygen requirement (demand) is not met which causes myocardial cells to " starve" for oxygen supply. This causes the depolarization of the cells to be interrupted and changes will occur on the ECG. (Woods, 1995) Ischemia is shown on the ECG by ST segment elevation. This is primarily an emergency situation as the first 6 hours post infarction is when myocardial damage becomes irreversible (Thelan, 1994). In this time many interventions can be attended to resupply the myocardium with oxygen enriched blood supply. Oxygen is administered for at least the first 48 hours post MI so that tissue hypoxia does not become evident. At times chest pain can be relieved by applying oxygen.(Swearingen & Keen, 2001)

Vital signs are attended to frequently in CCU, usually hourly, which enables nurses to see any changes in hemodynamic monitoring. Complications of infarctions are heart failure and arrhythmias, due to the large area of heart wall damaged. When Mr Smith suddenly became SOB and adventious breath sounds were heard on auscultation, it alerted medical staff that his left side of the heart was congested and not efficiently pumping. Early indications of Left ventricular failure are shortness of breath (SOB) and intolerance of beta blockers, nitrates, or ACE inhibitors. Mr Smith showed signs of SOB and lightheadedness, which may be due to Ramipril ( ACE inhibitor) that was then decreased in dose (Schell & Puntillo, 2006).

Continuous cardiac monitoring enables nurses to keep constant checks on heart rates and rhythms, it gives nurses the ability to act on any life threatening rhythms immediately or enables them with the knowledge of impending problems that could arise (Drew, 2004). Premature Ventricular Contractions (PVC), Ventricular Tachycardia (VT) or Ventricular Fibrillation(VF) are the most likely rythyms to be noted due to the scarring or necrotic myocardial tissue (Aehlert & elnstruction Corp., 2011). Mr Smith was noted to have occasional PVC's that became more frequent until he was https://assignbuster.com/care-rationale-and-outcome-in-coronary-care-unit/ monitored in bigeminy, which can lead to runs of VT (Huszar, 2007). Monitored patient's can be observed in pulseless VT/ VF via the central monitor at the nurses' station and can be immediately defibrillated, whereas if a ward patient collapses a monitor needs to be attached before the heart rhythm can be established and treatment given (McDonough, 2009).

ST Segment monitoring shows significant changes in monitoring that can indicate ischemia or infarction. Central monitors should have regular nurse surveillance, will alarm if there is a significant change to the ST segment. Changes occur with or without complaints of chest pain or shortness of breath, indicating myocardial oxygen mismatch (Smith, 2008). Patient's need to advised to tell staff of chest pain whilst being monitored. Some patients assume nursing staff know from the monitor when they are experiencing chest pain. (Swearingen & Keen, 2001)

An ECG can be performed to show any significant changes of the heart. Mr Smith showed ST segment changes in his anterior /septal (V3 &V4 position) aspect of his left ventricle. This area is supplied by the Left Anterior Descending Coronary Artery. Treatment does not differ depending on which area of the heart is affected. All areas require oxygen supply. While in hospital Mr Smith was ordered serial ECG's, these are taken daily to show any changes. Expected changes expected post MI are the development of a pathological Q wave. Q waves indicate the necrosis of myocardial tissue and specifically in V1 to V4 indicates anteroseptal infarction (Dubin, 2000)

Mr Smith was initially given morphine, an opioid that relieves pain by decreasing myocardial oxygen demand by decreasing the Autonomic Nervous System and decreasing anxiety (Lewis, 2007). Nitro-glycerine, was ordered as a smooth muscle relaxant that vasodilates the vessels to restore blood supply if the mismatch is due to a coronary spasm(Yassin, 2007). Aspirin is given daily indefinitely as it is a antiplatelet aggregation inhibitor that Hung, 2008 states is " proven for secondary prevention of myocardial infarction, stroke and cardiovascular death in both men and women". He also discusses the combined use of clopidigrel and aspirin to reduce subacute stent thrombosis after PCI's (Hung, 2008).

Thrombolytic therapy is given within the first 6 hours of chest pain.(Levin, 2008) Tenecteplase 90mg was given. Thrombolytic Therapy is given to dispel the clot and allow blood flow to the affected area. It can take up to 90 minutes for full resolution to occur (Goldberger, 2010). There are certain considerations that medical staff must ensure prior to administration of this therapy, these include an absence of CVA/TIA's or surgery in the last 12 weeks (Gibson, 2009). Once administered ECG's are taken in 15-30 min intervals to see changes of ST segment, showing that myocardial blood supply and depolarization being restored.

Cardiac markers are Pathology tests that also give evidence of myocardial damage. When cardiac cells are damaged the membrane walls leak these substances into the blood stream (Aehlert & elnstruction Corp., 2011). Myoglobin, Creatine kinase (CK), Troponin T and Troponin I are myocardial specific and along with ST elevation can be evident of a STEMI. Ëarly in ischaemia the ST segment may lose the ST-T wave slope and appear straight. Then as the T wave broadens and the ST segment rises, the segment loses it's concave form and becomes upwardly convex with https://assignbuster.com/care-rationale-and-outcome-in-coronary-care-unit/ elevations" (Moser & Riegel, 2008). Non STEMI do not have a significant change on the ECG only cardiac markers alter. These markers usually peak between 15-24 hours post infarction and remain elevated for 2-3days (Huszar, 2007) Creatine Kinase has normal value of 45-250 U/L and Mr Smiths on admission was 4290 U/L decreasing to 800 U/L, 2 days post. Troponin T normal values are ≤0. 03ug/L but Mr Smiths ranged from 14. 20ug/L at 2200hours on the day of MI, to 4. 39ug/L 2 days later. Serial pathology tests are taken usually every 6 hours for the first 24 hours.

Mr Smith was taken for a PCI the day after his MI. He had a stent put in his proximal area of his Left anterior descending coronary artery (LAD) in the Cardiac Catheter Lab. Mr Smiths had a PCI even though his blood supply looked like it had been reinstated, the stent will prevent clot formation again and reocclussion (Cannon, 2010). He was then transferred back to CCU and remained RIB overnight. He had a femoseal deployed into his groin to occlude the opening of the femoral vessel used for this procedure. Nurses need to do regular neurovascular and pedal pulse observations to check for bleeding or vessel occlusion (Shoulders-Odom, 2008).

Mr Smith needed to be educated on his procedure pre and post operative. He has previously been for this procedure but needed re-education. It must be a daunting experience to be given twilight sedation whist having the PCI. Mr Smith's last procedure was 13 years ago which would see many new techniques being practiced that he was not familiar with. His post op education included the importance of keeping his affected leg still and care of his affected groin.(Moser & Riegel, 2008) Myocardial Infarction education can be given to him at the same time but this is information that needs to be https://assignbuster.com/care-rationale-and-outcome-in-coronary-care-unit/ reiterated continually during his hospitalization(Lewis, 2007). He and his family need to be aware of the risk of reinfarction especially in the next 2 weeks post MI as the heart muscle is still weak and irritable and increase in activity can cause another MI. This is the time that patients start to resume their normal daily activities after hospitalization and are at the most risk. (Douglas, 2010)

Documentation is very important and needs to be filled out correctly as it is a legal document (Lewis, 2007). The CCU's clinical pathway for infarction indicated strict rest in bed with commode privileges for the first 48 hours, this decreases the need for myocardial oxygen. This is difficult for active patients but it needs to be strictly followed. Due to immobility other medical complications can arise, pneumonia and decreased gas exchange, deep vein thrombosis or emboli are common. To prevent these patients are encouraged to attend to hourly Deep Breathing Exercises (DBE), leg exercises and triflow. Patients can also be sat in an upright position which increases venous return (Thelan, 1994). Anticoagulants prevent clot formation therefore Mr Smith was administered daily Clexane 90mg post PCI until discharge and administered Abciximab (Reopro) for 12 hours post PCI. To test the adequacy of anticoagulants, INR and APPT are taken to check patient's dose is therapeutic. Problems with administering the anticoagulant after thrombolytic therapy is bleeding (Yassin, 2007). Mr Smith was noted to have large traces of blood in his urinalysis and was sent for a Pelvic Ultrasound to be sure there was no other complications, the ultrasound was NAD.

Prior to discharge Mr Smith was educated on his new regime of medications and the importance of medication compliance to decrease his risks of further cardiac complications (Albert, 2008).

Nurses if experienced and up to date with current research and practices can work alongside medical staff and initiate nursing cares that are in the best interest of patients. Coronary Care Units must have confident and competent nurses to run the ward as most times they make significant decisions on implementation of nursing care. When Doctors have confidence in the nurse looking after their patients they will respect and listen to nurses opinions because they know they are educated and empowered with knowledge.