

Hypertension case study nursing



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1) CASE SUMMARY

Mr. MS is a 58-year-old Malay male who was previously diagnosed with hypertension, gout and triple vessel ischemic heart disease. He first presented with chest pain in March 2010 where he was diagnosed with ischemic heart disease. He was unable to complete an exercise stress test and an angiogram done in Hospital Sultanah Aminah found him to have triple vessel disease. He was told angioplasty was not possible due to the severity of the blocks and was counseled for CABG but he was not keen. Meanwhile, he has had angina attacks 2 to 3 times per week every week since his initial diagnosis for the last 3 months, usually relieved by sublingual GTN and was currently admitted for the 4th time for chest pain not relieved by GTN. ECG done 2 hours after onset of chest pain showed ST depression of 2mm at leads I, aVL, V3 – V6 and left axis deviation with no Q waves. Troponin T was positive (2.75 ng/ml) at 4 hours after onset and other cardiac enzymes were also raised significantly. He was diagnosed with NSTEMI and treated with aspirin 300mg, IV morphine 2.5 mg, sublingual GTN 3 tablets and subcutaneous clexane 60mg BD for 3 days as well as continuing his current medication regime of simvastatin, metoprolol, cardiprin, ISDN, amlodipine and GTN. Following admission, he was well in the ward with no recurrence of chest pain and did not develop any new complaints. He was discharged after 3 days of inpatient treatment with instructions to attend his follow-up appointment at the cardio clinic in HSAJB on the 16th of June 2010 to make an appointment for surgery. Following this episode of chest pain, which he says is the worst so far, he is now quite keen for CABG.

PATIENT'S DETAILS

I/C NUMBER: 510831015263 AGE: 58

SEX: Male DATE OF ADMISSION: 3/6/2010

R/N: 1348445

2) CLINICAL HISTORY

Chief Complaint

Chest pain for 1 day.

History of Present Illness

Mr. MS is a 58-year-old Malay male who was previously diagnosed with gout, hypertension and ischemic heart disease with triple vessel disease. He was awoken from sleep at about 10pm due to a central chest pain of sudden onset. He described the character of the pain as crushing in nature and radiated to his neck. This episode of chest pain was the most severe since he was first diagnosed with ischemic heart disease. The pain was associated with profuse sweating, body weakness and was not relieved by rest.

However, it was relieved by sublingual GTN, of which he has a supply of. His discomfort was made worst by exertion so he lay in bed to recover. Despite this, he had another episode of chest pain 30 minutes later. He took the sublingual GTN again but this time, the pain did not resolve. He was then brought to the emergency department of Hospital Batu Pahat by his son.

This is Mr. MS's fourth admission for chest pain since March 2010. Since his diagnosis of ischemic heart disease in March, he has experience angina

attacks two to three times per week, especially on exertion such as when straining while passing motion. During these attacks, he uses sublingual GTN to relieve his symptoms and normally feels much better after that. He only comes to the hospital when GTN does not work to relieve his symptoms.

Systemic Review

Mr. MS does not experience symptoms such as palpitations, dizziness, headache, nausea, vomiting, orthopnoea, paroxysmal nocturnal dyspnoea, epigastric pain, shortness of breath, fever, and had no syncopal episodes. He also does not have loss of appetite or loss of weight. Bowel and urinary habits are normal. His sleep has not been affected until this current episode whereby he was awoken by the chest pain.

Past Medical History

Mr. MS was diagnosed with hypertension 6 years ago when he had an episode of headache. He has been on medication since and was on regular follow-up with KK Rengit. He was diagnosed with gout 5 years ago when he had a left big toe swelling which resolved after some medication. He is not on long term medication for gout. Mr. MS was admitted for the first time 5 years ago in 2005 when he had bilateral renal calculi. He was subsequently referred to Hospital Sultanah Aminah for further management of this problem and it has since resolved and does not have follow-up anymore.

Mr. MS was diagnosed with ischemic heart disease in March 2010 when he presented with chest pain for the first time. Following his recovery, he underwent a stress test in Hospital Batu Pahat but according to him, was

unable to complete the procedure due to chest discomfort. He was referred to the cardiology unit in Hospital Sultanah Aminah for further management where an angiogram was performed and he was told to have triple vessel disease. He was also told that angioplasty was not possible due to the severity of the blocks. He was recommended to have Coronary Artery Bypass Grafting (CABG) but as of yet, no appointment has been made as he was still unsure of going through with the procedure. Following this episode of chest pain, Mr. MS has decided that going for the CABG is the only thing that will keep him alive.

His current medications include:

Tab Simvastatin 20mg OD

Tab Metoprolol 75mg BD

Tab Cardiprin 100mg OD

Tab Isosorbide Dinitrate (ISDN) 5mg TDS

Tab Amlodipine 10mg OD

Sublingual Glyceryl Trinitrate (GTN) PRN

He is compliant to his medication regime.

Mr. MS is not known to have diabetes or hyperlipidemia. He also does not have any known food or drug allergies.

Family History

Mr. MS is the 3rd of 9 siblings. His father had hypertension and passed away a long time ago due to unknown causes. His mother and other siblings are healthy. None of them have hypertension, diabetes, ischemic heart disease or malignancy.

Social History

He lives in a kampung in Rengit with his wife and 5 children. Mr. MS does not smoke nor consume alcohol. He works in a palm oil plantation. The distance from his house to Hospital Batu Pahat is about half an hour. On further enquiry, Mr. MS says that the cost of the CABG is about RM1000, which he can afford.

3) FINDINGS ON CLINICAL EXAMINATION

(Mr. MS was examined by me 9 hours after onset of chest pain)

Mr. MS was alert, conscious, and communicative. He was not in obvious pain or respiratory distress. He was lying down comfortably on his bed. There were no tendon xanthomata, xanthelasma, pallor, corneal arcus or pedal edema. His JVP was not raised. His clinical parameters are:

Blood Pressure : 158/94 mmHg

Heart Rate : 94 beats per minute. Regular rhythm

Respiratory Rate : 20 breaths per minute

Temperature : 37°C

SpO2 : 97% under room air

On examination of the precordium, the apex beat was located at the 5th intercostal space on the midclavicular line and was normal in character. Parasternal heave was not felt and there were no thrills. First and second heart sounds were heard. There were no murmurs or added heart sounds.

On examination of the chest, there was no deformity and chest expansion was equal on both sides. Percussion and tactile vocal fremitus was normal and equal on both sides. On auscultation, vesicular breath sounds were heard throughout all lung fields with good air entry. There was no wheezing or crepitations heard.

On examination of the abdomen, it was soft and non-tender. There were no masses felt. Bowel sounds were heard and normal.

4) PROVISIONAL AND DIFFERENTIAL DIAGNOSES WITH REASONING

Provisional Diagnosis

Acute myocardial infarction with underlying triple vessel ischemic heart disease and hypertension

With a history of diagnosed triple vessel ischemic heart disease with multiple episodes of angina attacks since the initial diagnosis, it is highly likely that Mr. MS is presenting with an acute coronary event and this should be a priority until proven otherwise. This is evidenced by the presentation of central, crushing chest pain of sudden onset that radiated to the neck and associated with profuse sweating and body weakness which is classical of a myocardial infarction. Mr. MS will require immediate investigations such as an electrocardiogram and cardiac enzymes to differentiate the acute

coronary syndromes so that the appropriate management may be instituted for him e. g. if he has an ST-segment elevation myocardial infarction (STEMI), he will require myocardium-saving thrombolytic therapy to disrupt the ischemic event. As Mr. MS did not present with features such as acute shortness of breath, loss of consciousness and severe palpitations, it seems that he does not have complications of acute myocardial infarction but these developments should be watched out for throughout his admission as complications may arise later.

Differential Diagnosis

Pulmonary embolism

Pulmonary embolism is a possibility that can be considered when a patient presents with an acute chest pain that is accompanied by shortness of breath, hemoptysis, tachypnea, fever and even cyanosis and collapse in severe cases. Furthermore, the chest pain is of a pleuritic nature, of which it is worsened on breathing, and a pleural rub can be heard on auscultation of the chest. However, Mr. MS did not present in such a way. At the same time, Mr. MS did not have risk factors such as a deep vein thrombosis, prolonged immobilization or recent surgery. It is still highly likely that Mr. MS has suffered an acute myocardial infarction, and an ECG would help to differentiate between the two as pulmonary embolism might show the classic S1Q3T3 pattern of right axis deviation or right bundle branch block. Either way, the diagnosis should be made quickly so treatment may be instituted before his condition becomes worse or complications develop.

Aortic dissection

Aortic dissection presents as an acute onset chest pain that is tearing in nature, and often radiates to the back. It is often confused with myocardial infarction due to its presentation but differences include the lack of profuse sweating, signs of heart pump dysfunction and a normal ECG. Risk factors are usually uncontrolled hypertension, connective tissue disorders or chest trauma. Mr. MS has hypertension, but is under control, and does not have the other risk factors. A diagnosis of myocardial infarction should be the priority as thrombolytic therapy is vital, but if there is any reason to doubt that diagnosis, then further investigations should be performed.

5) IDENTIFY AND PRIORITISE THE PROBLEMS**1. Acute chest pain**

Mr. MS has acute chest pain with features very suggestive of a classical picture of myocardial infarction as he presents with crushing central chest pain that radiates to the neck and associated with profuse sweating and weakness. Given that he is known to have triple vessel ischemic heart disease and that he has suffered many angina attacks since his initial diagnosis, it is highly likely that he is having an acute myocardial infarction. Without further a due, he needs an electrocardiogram (ECG) and cardiac enzymes tested to distinguish between the different acute coronary syndromes so that the appropriate treatment protocols may be initiated for him as soon as possible to disrupt the ongoing ischemia. As Mr. MS is having severe chest pain that may overstimulate his sympathetic system and cause further ischemia, he will require immediate supportive therapy such as effective pain medication and oxygen therapy.

2. Triple vessel ischemic heart disease awaiting CABG

Mr. MS was diagnosed with triple vessel ischemic heart disease when he first presented with chest pain in March 2010 and has since experienced many episodes of angina. Given his diagnosis and disease pattern, he is at a very high risk of developing a severe acute coronary event that may prove fatal if the infarction is too extensive or if complications develop. As percutaneous revascularization with a stent or balloon was not possible for him, he will require a CABG to both relieve his symptoms and reduce his mortality risks in the long term. He was unsure of going ahead with the operation previously, therefore no appointment date was given for surgery. However, now that he has changed his mind, every effort should be made by both the doctors in charge of him here in Hospital Batu Pahat and in the cardiology unit of Hospital Sultanah Aminah to arrange for his surgery as soon as possible, given the circumstances of his condition.

3. Compliance to medication

Mr. MS is on several medications for his triple vessel ischemic heart disease and will require revascularization surgery soon in order to decrease his mortality risks. However, waiting for a CABG in the government setting may take some time, even under dire circumstances due to the nature of the system. Therefore, it is extremely crucial that Mr. MS is compliant to his medication regime while awaiting a CABG to prevent another episode of infarction. He should be counseled to fully understand this and the situation of his ischemic heart disease. It is also the responsibility of his doctors to ensure that he is taking the right combination of medications with the aim to

prevent another acute cardiac event. Meanwhile, a sufficient supply of sublingual GTN should be provided for Mr. MS in cases of angina attacks at home. He should come to the hospital immediately if GTN fails to relieve his symptoms.

4. Regular screening for comorbid diseases

Mr. MS has not been diagnosed with diabetes or hyperlipidemia previously but these diseases are strong risk factors for the long term implications of his ischemic heart disease. Therefore, Mr. MS should be screened regularly e. g. twice yearly during his follow-up appointments. Early detection of diabetes is necessary so that treatment can start as soon as detected in order to prevent his ischemic heart disease from becoming worse than it already is. As for his lipid control, if his lipid profile is found to be outside the normal limits, the dosage of his medication can be increased as necessary. Following his CABG, he will need to maintain a healthy lifestyle of a good, well-balanced, low-salt and low-fat diet and regular exercise within his limits.

6) PLAN OF INVESTIGATION, JUSTIFICATIONS FOR THE SELECTION OF TESTS OR PROCEDURES, AND INTERPRETATION OF RESULTS

1. Electrocardiogram (ECG)

To look for any changes that may indicate an ongoing ischemic event, such as ST elevation or depression and T wave inversion in order to support the diagnosis of an acute myocardial infarction so appropriate treatment can be started. Differentiation of ST segment elevation or depression is also crucial

in initiating treatment as thrombolytic therapy is only indicated for ST-elevation myocardial infarction.

Results: ECG on admission (2 hours after onset) shows sinus rhythm with ST depression at leads I, aVL, V3 – V6 with left axis deviation. T wave was present and normal.

Interpretation: The ST depression in the leads above indicate an ischemic event at the anterolateral sections of the heart. The lack of ST elevation concludes a diagnosis of either unstable angina or NSTEMI, depending on the levels of cardiac enzymes. There is no sign of old infarction.

2. Cardiac Enzymes

To look for elevated levels of cardiac enzymes such as troponin T, creatinine kinase (CK), lactate dehydrogenase (LDH) and aspartate transaminase (AST) that will indicate myocardium ischemia and necrosis. If elevated, a diagnosis of NSTEMI can be made in accordance with the ECG changes. However, cardiac enzymes when done too early after onset may not show any rise in levels 1. This does not mean that necrosis has not taken place and the test should be repeated once more at 6 hours after onset 1.

Results: Troponin T (4 hours after onset) – 2. 75ng/ml ↑

(12 hours after onset) (60 hours after onset) Normal Range (U/L)

CK – 997 ↑ 263 ↑ <175

LDH – 392 ↑ 518 ↑ 114 – 241

AST – 139 ↑ 59 ↑ <37

Interpretation: Troponin T is elevated indicating myocardial infarction and necrosis has taken place, and combined with the features on ECG, a diagnosis of NSTEMI is made regarding Mr. MS's current episode of chest pain.

3. Full Blood Count

To look for signs of infection or anemia which could have precipitated the acute coronary event, to check platelet levels as thrombolytic or anticoagulation therapy will be started for Mr. MS, and as baseline for monitoring as anticoagulation therapy with heparin may cause thrombocytopenia.

Result: TWBC – 13.7 $\times 10^9/L$ ↑ (neutrophils – 59.2%, lymphocytes – 35.5%)

Hemoglobin – 13.7 g/dL

Platelets – 357 $\times 10^9/L$

Interpretation: The total white cell count is raised, but that could be due to the reaction of the body towards the acute stressing event of the myocardial infarction. Hemoglobin is normal indicating no anemia and platelets are normal, therefore there is no contraindication to start anticoagulation therapy.

4. Prothrombin Time, INR, Activated Partial Thromboplastin Time (PT/INR/APTT)

To obtain a baseline of the coagulation profile before starting any anticoagulation or thrombolytic therapy.

Result: PT – 14. 3s INR – 1. 28 APTT – 42. 6s

Interpretation: PT/INR/APTT is within normal range. There is no contraindication to anticoagulation or thrombolytic therapy if necessary.

5. Renal Profile

To assess the renal functions as the patient has a history of hypertension. Also, it is necessary to check renal functions before drugs such as ACE-inhibitors are started.

Result: Urea – 5. 7mmol/L Sodium – 135mmol/L

Potassium – 4. 1mmol/L Creatinine – 129µmol/L

Interpretation: Mr. MS's renal functions are normal. There is no evidence of renal disease and drugs such as ACE-inhibitors may be added to his medication regime if required.

6. Chest X-Ray

Provides information of the left heart function by looking for cardiomegaly or increased pulmonary markings as Mr. MS has ischemic heart disease and hypertension. However, Mr. MS has no complaints suggestive of failure. Therefore, this chest radiograph may be used as a baseline for comparisons with future radiographs.

Result: There is no cardiomegaly and no features of pulmonary congestion.

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Interpretation: Mr. MS's left heart function is normal, as the lack of failure symptoms suggests.

7. Fasting Blood Glucose

To screen Mr. MS for diabetes, which is an important co-morbid condition and risk factor for his ischemic heart disease and will require early treatment if detected.

Result: 6.4 mmol/L

Interpretation: The fasting blood glucose level is normal but is on the high end of the normal range. This could be the result of the body's acute stress response to the myocardial infarction. Mr. MS should be screened regularly in future for diabetes given his high risk.

8. Fasting Lipid Profile

Despite the fact that Mr. MS was not previously diagnosed with hyperlipidemia, he is taking medication (simvastatin) as it is recommended for patients with ischemic heart disease. However, his lipids should be checked regularly to detect a rise in the cholesterol and LDL levels so that it can be managed appropriately.

Results: Total Cholesterol – 4.4 mmol/L HDL – 1.3 mmol/L

Triglycerides – 1.5 mmol/L LDL – 2.4 mmol/L

Interpretation: His fasting lipid profile is normal. This indicates that Mr. MS requires no adjustment to his medication for lipid control.

7) WORKING DIAGNOSIS AND PLAN OF MANAGEMENT ON ADMISSION

Working Diagnosis

Non ST-segment elevation myocardial infarction (NSTEMI) with underlying triple vessel ischemic heart disease and hypertension

Plan of management at the emergency department

Sublingual GTN 1 tablet stat

Tablet aspirin 300mg stat

Oxygen therapy 3L/min via nasal prong

Intravenous morphine 2. 5mg stat

Intravenous drip 1 pint Normal Saline

Plan of management on admission to the ward

Subcutaneous clexane 60mg BD for 3 days

Sublingual GTN 1 tablet PRN

Oxygen therapy 3L/min via nasal prong

Encourage oral intake

Daily ECG and if chest pain recurs

Vital signs and SpO2 monitoring 2 hourly

Tablet simvastatin 20mg ON

Tablet metoprolol 75mg BD

Tablet cardiprin 100mg OD

Tablet isosorbide dinitrate (ISDN) 5mg TDS

Tablet amlodipine 10mg OD

To inform staff nurse or house officer immediately if symptoms recur

8) SUMMARY OF INPATIENT PROGRESS (INCLUDING MAJOR EVENTS, CHANGE OF DIAGNOSIS OR MANAGEMENT AND OUTCOMES)

In the emergency department, Mr. MS was relatively stable on presentation despite the acute coronary event. He was given sublingual GTN 3 times in total in the emergency department before his symptoms were relieved. Upon admission to the ward, he was started on subcutaneous clexane and was continued on his current medication regime. Throughout Mr. MS's stay in the ward, he did not have any recurrences of chest pain and did not develop any complications of his NSTEMI. He was relatively comfortable in the ward, did not have any new complaints, slept well at night and was able to tolerate well orally. His vital signs were also stable throughout his stay. Daily ECG showed resolving ST changes with no new evolving changes. He was discharged after 3 days of inpatient treatment whereby he completed three days of subcutaneous clexane. ECG taken on discharge showed T wave inversion in leads I and aVL, ST depression in leads V4 to V6 and Q waves in leads V4 to V6. On discharge, he was given appointment at the specialist

clinic in Hospital Batu Pahat to review his renal profile, fasting blood glucose and fasting lipid profile in 3 months time. He was also told to continue his current medication regime and his follow-up appointment with the cardiology clinic in Hospital Sultanah Aminah on the 16th of June 2010 to fix a date for his CABG.

9) DISCHARGE PLAN, COUNSELLING AND MOCK PRESCRIPTION

Discharge Plan

Continue current medication regime.

Follow-up appointment with specialist clinic, Hospital Batu Pahat in 3 months time to review renal profile, fasting blood glucose, and fasting lipid profile.

Follow-up appointment with cardiology clinic, Hospital Sultanah Aminah on 16/6/2010 to fix a date for CABG.

Suggested Additional Discharge Plan:

Add Tab. Clopidogrel 75mg OD 1. Consider adding Tab. Trimetazidine 35mg BD 2.

Counseling

Advised compliance to medications to prevent further recurrence of chest pain.

Advised to return immediately to the hospital if Mr. MS suffers from chest pain that is not relieved by GTN or other worrying symptoms.

Advised to watch a healthy lifestyle in order to prevent other comorbid conditions such as hyperlipidemia and diabetes. At the same time, healthy living confers benefits and reduces mortality risks of ischemic heart disease.

Mock Prescription

Tab. Simvastatin 20mg ON x 3/12

Tab. Metoprolol 75mg BD x 3/12

Tab. Cardiprin 100mg OD x 3/12

Tab. Isosorbide Dinitrate (ISDN) 5mg TDS x 3/12

Tab. Amlodipine 10mg OD x 3/12

S/L GTN 1 tablet PRN x 3/12

10) REFERRAL LETTER (IF APPLICABLE)

Cardiologist,

Cardiology Clinic,

Hospital Sultanah Aminah, Johor Bahru. 5th June 2010

Mr. MS (IC. 510831015263)

Date of admission: 3rd June 2010, Date of discharge: 5th June 2010

Problem: Triple Vessel Ischemic Heart Disease for CABG

Dear doctor,

Mr. MS is a 58-year-old gentleman who is under your follow-up for triple vessel ischemic heart disease. He presented to us with acute chest pain not relieved by GTN. He was diagnosed with NSTEMI as ECG on admission showed ST depression (2mm) in leads I, aVL, V3 – V6, and cardiac enzymes were positive. He was admitted for 3 days during which he was treated with subcutaneous clexane 60mg BD for 3 days and continued his current medications of simvastatin 20mg ON, metoprolol 75md BD, cardiprin 100mg OD, ISDN 5mg TDS, amlodipine 10mg OD and GTN 1 tablet PRN. During his stay, he did not have recurrence of chest pain and was relatively comfortable throughout. We discharged him with instructions to attend your clinic as scheduled on the 16th of June 2010. It is our understanding that he was recommended CABG but was not keen initially. However, that has changed. Please review his condition during his follow-up with you and if possible, to fix a date for CABG. Thank you very much for your attention.

Yours sincerely,

11) LEARNING ISSUES IN THE 8 IMU OUTCOMES

1. Critical thinking and research

Mr. MS is on many medications for his ischemic heart disease. However, he suffered frequent recurrent angina attacks despite this. The worry is that after discharge, he may continue to suffer from angina attacks, or worse still, another myocardial infarction. This issue explores the benefits of adding the anti-anginal medication trimetazidine to his medication regime.

Trimetazidine is a relatively new anti-anginal medication which works to prevent ischemia by counteracting the major metabolic disorders occurring

within the ischemic cell such as acidosis, disturbance of transmembrane ion exchange and the production of free radicals. In the systematic review by Ciapponi et al 2, 23 studies were included to determine the efficacy and tolerability of trimetazidine in patients with stable angina. Trimetazidine in these studies were compared with either monotherapy versus placebo or another anti-anginal agent, or in combination therapy versus regimes without trimetazidine. The two main outcomes measured were the frequency of angina attacks and the frequency of GTN use. Trimetazidine was shown to reduce angina attacks per week by 40% independent of whether it was given in mono or combination therapy, but confidence intervals were wide. This study also showed that GTN use was reduced by a mean of 1.47, a finding that supports the efficacy of adding trimetazidine to anti-anginal therapy. This study also showed that trimetazidine appeared to be better tolerated by patients in terms of adverse effects when compared to other medications in combination therapy from the dropout rate of some trials. However, this information is limited by the lack of trials directly comparing the safety profile of trimetazidine versus beta blockers, calcium channel blockers, or nitrates. Furthermore, there is little information in the literature about trimetazidine and its effect on mortality, cardiovascular events, or quality of life. The authors recommend long term trials comparing trimetazidine with other anti-anginal agents using clinically important outcomes such as the above. In conclusion, there appears to be some benefit in adding trimetazidine to Mr. MS's medication regime in a bid to reduce the frequency of his angina attacks, but there is no evidence to suggest that it confers better protection in terms of long term mortality, cardiovascular events, and quality of life.

2. Self directed life-long learning and information management

Beta blockers, calcium channel blockers and nitrates have long been the mainstay of anti-anginal medication. However, ranolazine has recently been approved as an anti-anginal agent ³. This issue explores its benefits.

Ranolazine was approved by the US Food and Drug Administration (FDA) on 31st January 2006, the first new drug approved for the treatment of chronic angina in over a decade ³ and a new class of anti-anginal agent in almost 25 years ⁴. Ranolazine is an orally active piperazine derivative. Its mechanism of action is not fully understood, but it is known to inhibit myocardial fatty acid oxidation, resulting in preferential glucose oxidation leading to a decrease need for oxygen for a given level in the glucose pathway that translates into an anti-ischemic action whereby there is increased oxygen efficiency in the myocardium ⁴. It is metabolized in the liver and excreted in the urine. Therefore, it is contraindicated for patients with any form of liver impairment ⁴. It also shows drug interactions with drugs such as digoxin and simvastatin ⁴. The first large placebo-controlled trial to establish a dose-response anti-anginal relationship of ranolazine monotherapy was the MARISA trial ⁵ in 2004 by Chaitman et al. They demonstrated that in patients taking ranolazine, there was a significant dose-related increase in the exercise capacity of patients known to have exercise restrictions from angina ⁵. The follow up CARISA trial combined the use of ranolazine with beta blockers or calcium channel blockers and found that there was a significant increase of exercise duration independent of background anti-anginal therapy ⁶. They also recorded a significantly reduced use of GTN by the

patients in the ranolazine group 6. The ERICA trial was a double-blind study of 565 patients to determine the benefits of ranolazine in chronic angina in patients already on maximum recommended doses of amlodipine 7. The primary endpoints of their study were the number of angina attacks and GTN consumption per week. They also addressed safety issues of ranolazine via assessment of side effects and ECG. They found that ranolazine significantly reduced the frequency of angina attacks and GTN use per week and also found that ranolazine appeared to have a more pronounced effect in patients with more frequent angina attacks 7. They also reported that ranolazine was well tolerated. It was these results of the ERICA trial that satisfied the FDA that ranolazine offered some benefit over the current standard therapies 4. However, Anderson et al raised the issue of side effects of ranolazine as dose-related prolongation of the QT interval on ECG but could not associate this with any incidences of ventricular arrhythmia 8. Due to these findings, FDA approval of the use of ranolazine is limited to patients who have not responded ideally to other drugs and to be used in combination therapy 4. However, the later MERLIN-TIMI 36 trial 9 found that the addition of ranolazine to combination therapy did not significantly improve mortality or cardiovascular events. At the same time though, they showed that ranolazine did not increase the risk of all-cause mortality or symptomatic documented arrhythmias, thus supporting ranolazine's safety profile 9. This new information, together with that of the CARISA extension study 10 that showed ranolazine's effect in improving glycemic control in diabetic patients included in the study, prompted its developer, CV Therapeutics to apply to the FDA to change the indications of ranolazine to that of a first-line anti-anginal agent with benefits in HbA1c and arrhythmia reduction. The FDA

approved this in November 2008. In conclusion, the addition of ranolazine to Mr. MS's medication regime may confer some benefit in reducing the number of angina attacks, but seems similar as the use of trimetazidine. However, ranolazine is not available in our setting.

3. Disease prevention and health promotion

Mr. MS has just suffered a NSTEMI. He also has a history of frequent angina attacks. What are his risks of developing another serious cardia