ESC CVD Prevention Guidelines 2012

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CASE STUDY

Acute coronary syndrome

Michael is a 46 year old man who has recently been discharged from hospital with a diagnosis of acute coronary syndrome.

He presented to the hospital initially complaining of 2 hour history of severe chest pain. Initially he thought it was indigestion and took some antacid. As there was no relief from the pain and it suddenly became worse and he also felt clammy and nauseated, his colleague brought him straight to A and E.

Previous history: Indigestion and some shortness of breath on exertion in recent months

Medications:	Nil
Allergies:	Nil known
Smoking status:	20/day for 25 years approx.
Alcohol history:	Drinks in excess of 35units/week as beer/wine.
Family History:	Father died suddenly 15 years previously of a heart attack. Mother is a diabetic.

Michael was admitted for investigations and treatment. His admission ECG showed ST elevation in inferior leads. His troponin levels were elevated. He was taken to the coronary angiogram lab and had a coronary angiogram performed. His right coronary artery (RCA) was 90% blocked with minor disease in the other coronary arteries. A drug eluting stent was inserted into the RCA. Michael spent 2 days in the coronary care unit before transfer to a ward and then discharged home.

The results of his tests were as follows:

ECG:	STEMI (Inferior leads) which resolved with q wave formation.
ECHO:	Area of dyskinesis in the right ventricular wall. Ejection Fraction 55%
Angiogram:	90% narrowing RCA and minor disease in the other arteries.
CXR:	Normal
Troponin:	0.56
Renal profile:	Normal
Liver Profile:	Normal
FBC:	Hb 14.2
Fasting Lipids:	Total Chol 6.2, Trigs 3.6, LDL 3.2, HDL 0.77mmol/L
Glucose:	6.3mmol/L

His discharge medications are:

Ramipril 1.25mg OD to increase to 2.5mgs after one week
Bisoproplol 1.25mg OD to increase to 2.5mgs after one week
Aspirin 75mg OD
Prasugrel 10mgs OD
Atorvastatin 80mgs OD

You are meeting Michael for the first time since his discharge. He is feeling a lot better, however, he is still shaken regarding his cardiac condition and what the future will hold. He is also confused about the need for so many tablets as he never took any before.

Today his BP is 152/96mmHg, heart rate is 78beats/min and regular. Weight 76kg and BMI 28kg/m^2

QUESTIONS:

1. What are the particular risk factors that caused Michael's heart disease?
2. Name two other risk factors that lead to the development of heart disease?
3. Outline the non-pharmacological measures that you would discuss with Michael in order to control his condition in line with ESC 2012 guidelines.
4. What steps outlined in the ESC 2012 guidelines would help you motivate Michael to quit smoking if he had not yet done so?
5. What concerns do you have about Michael's alcohol intake and what advice would you offer him?
6. Explain briefly the need for his new regime of medications and reasons for compliance.
7. What medications are proven to reduce cholesterol?
8. Explain briefly why such a high dose of a statin has been prescribed?
9. What blood tests would you repeat following commencement of this (statin) medication and why?
10. According to the new ESC 2012 guidelines what is the target for LDL cholesterol?
11. What is a normal blood sugar level and what follow up tests are required for Michael?
12. Today Michael's BP was initially high. What is the target BP for patients with CVD according to the ESC 2012 guidelines? What follow up would Michael need to monitor his BP if it remained high?

Q1.

Worldwide, cardiovascular disease (CVD) remains a significant cause of premature death.1 Health promotion in the primary and secondary prevention of CVD focuses on the modification of globally recognised risk factors. The WHO calculates that 80-90% of CVD deaths can be attributed to 1 or more major risk factors.[15]

Modifiable risk factors are:

- Smoking – Michael smokes 20 daily for 25 years
- Hypercholesterolaemia – Michael's total cholesterol is elevated at 6.2mmol/l, with raised triglycerides and LDL and low levels of cardioprotective HDL.
- Hypertension – Michael has grade 1 hypertension
- Obesity – Michael has a BMI of 28kg/m^2 with possible abdominal adiposity (central obesity).
- Alcohol consumption – Michael drinks >35 units per week
- Impaired fasting glucose/type 2 diabetes – Michael's fasting plasma glucose is 6.3mmol/l.

Non-modifiable risk factors include age, gender and family history (Michael's father had a fatal myocardial infarction).

Q2.

Two other risk factors that could lead to the development of heart disease are.[1]

Diet and exercise

1) Sub-optimal intakes of fruit, vegetables, fish and fibre. Excess salt, saturated and trans-fats in the diet.
2) A sedentary lifestyle or irregular and inconsistent exercise.

Psychosocial risk factors

1) Low socio-economic status
2) Work and family stressors
3) Depression and anxiety
4) Social isolation
5) Hostility
6) Type 'D' personality.

Q3.

1. All patients should unequivocally be encouraged and supported to quit smoking[(1,7,15)].
2. Fruit, Vegetables, Fibre and Fish have cardioprotective benefits.
   4-6 portions of fruit and vegetables and 30-45grams of fibre daily are recommended.
   Fish consumption of 1-2 servings per week have been shown to reduce CHD mortality by an impressive 36%[(1,15)].
3. A diet low in saturated and trans-fats [(8,15)].
   High fat diets contribute to obesity levels and cardiovascular and metabolic ill-health.
   Trans-fats offer no nutritional benefit, yet are commonplace in vegetable oils, fast foods and baking.
4. Reduce salt intake to <5grams daily.
   Processed foods, bacon and ham have a high salt content. Michael should be advised that 1g of salt is equivalent to 0.4g of sodium as this misunderstanding could lead to excess salt intake. Advise Michael to avoid salt alternatives due to raised potassium content. Although potassium is beneficial for blood pressure control, excess intake could be arrhythmogenic.[10] Suitable substitutes are herbs and spices.
5. Limit alcohol to 2 glasses a day (for males).[1] Caution Michael re: substituting alcohol with soft drinks due to the association with obesity and diabetes development.
6. Increase regular exercise – to optimise cardiovascular fitness, reduce weight, control BP, reduce risk of type 2 diabetes, improve HDL cholesterol and reduce LDL levels. Encourage participation in moderate intensity exercise for 30 minutes daily or a minimum of 2.5 hours weekly. Low intensity exercise eg. walking may be advised initially if Michael has been sedentary[1,3,7]
7. Manage weight – aim for a BMI of 18.5-24.9kg/m^2.1 Obesity is becoming a worldwide crisis. Michael's BMI of 28 qualifies him as obese. A waist circumference measurement of >94cm for males indicates central obesity, which has implications for type 2 diabetes development.[1,12] Diet and exercise advice to manage weight will also benefit BP control, glucose and lipid levels.
8. Offer Michael tips to manage stressors in his work-life balance.[1] Diet, exercise, peer support, adequate sleep and reduced alcohol dependence are important areas to consider.

Q4.

Smoking is prothrombotic and contributes significantly to premature death rates. Amounts of tobacco and duration of use are important factors contributing to irreversible plaque formation[1,2]

ESC guidelines (2012) indicate that smoking cessation is the most important singular factor in secondary prevention, with significant reductions in mortality evident even after 6 months cessation.

Unfortunately, many patients often revert to smoking once the acute phase of their MI has passe.[2)]

Nurses must actively discourage smoking. Brief motivational interventions using the 5 As strategy should be used at each patient contact opportunity[1,3,6)].]]

5 As	Patient contact	Health promotion
Ask	If the patient is a smoker, ask about amount and duration. If non-smoker, advise to avoid passive smoking	
Advise	Reiterate harmful effects of smoking, encourage total abstinence, reduction in cigarettes does not offer comparative benefits	Give oral, visual and written advice
Assess	Readiness to quit. Is the patient at the pre-contemplative or contemplative stage?	Explore advantages and disadvantages of quitting
Assist	Set date for quitting. Provide supportive, individualised care and counselling.	Re: managing withdrawal symptoms, NRT and patches and access to smoking cessation groups.
Arrange	Follow-up visit to praise/ encourage /offer support if patient is lapsing	Acknowledge success. Reflect on difficult areas.

NOVARTIS

Q5.

Alcohol has atherosclerotic effects, contributing to hypertension, CVA and cardiomyopathy. Alcohol excess can promote obesity and malnutrition and is associated with depression and cancer.[-7]

Michael over-indulges in alcohol (>35 units/week). The potential for chronic ill-health exists, given his current lifestyle choices. Alcohol use should be discussed at primary care level and brief motivational interventions employed. Health promotion should focus on education and self-monitoring.

Instruct Michael regarding the daily alcohol limits (2 drinks/day for men) and the long-term health implications of excess consumption.[1,7] It may be pertinent to encourage Michael to acknowledge triggers for his drinking habits. NICE (2010) advise the FRAMES formula[(16)]:

- FEEDBACK on the problem
- RESPONSIBILITY for change
- ADVICE when required
- MENU of choices to enable change
- EMPATHY
- SELF-EFFICACY

Q6.

ACEi (Ramipril) are used extensively in primary and secondary CVD prevention. ACEi reduces post-MI mortality and has anti-hypertensive properties.[1]

Longterm ACEi therapy is recommended in patients with large wall motion abnormalities. Michael has an area of dyskinesis in his right ventricular wall, although left ventricular systolic function is preserved, given his ejection fraction of 55%.[-2]

ACEi preserves renal function in diabetic patients and reduces incidence of microalbuminuria and proteinuria.[1] This may be relevant for Michael given his impaired fasting glucose and with a first degree relative with diabetes.

ACEi can precipitate hyperkalaemia.[10] Therefore, check BP and electrolytes after 2 weeks. ACEi induced coughs may be avoided by changing to an ARB.

Betablockers in the post-MI period reduce longterm mortality, with greatest benefit achieved in patients at moderate-high risk.

Bisoprolol has anti-arrhythmic and anti-hypertensive properties.[1] This is advantageous for Michael given his diagnosis of inferior MI in the setting of possible hypertension (152/96mmHg).

Betablockers display anti-anginal qualities by reducing cardiac workload and cardiac O_2 consumption.

Administration in a once-daily dose will also facilitate compliance where polypharmacy is an issue.

Aspirin and Prasugrel – Dual anti-platelet therapy is advised post MI.

Aspirin has shown positive results in secondary prevention and is recommended lifelong. It maintains the patency of the affected artery and reduces the extent of a re-infarction.[-2,3] A maintenance dose of 75mgs has similar benefits to higher doses without the risk of GI bleeding.

Prasugrel at 10mgs is recommended in the acute phase post-MI and where the patient has had angioplasty. Michael was stented to his RCA. Treatment is advised for 12 months.[-2]

Poor compliance to longterm drug treatments is a very legitimate concern in secondary prevention. Nurses need to be vigilant in exploring reasons for non-adherence and to continue to educate, support and empower patients to accept their diagnosis.[14,15]

Q7.

Raised plasma cholesterol, LDL and triglycerides levels and low cardioprotective HDL levels are recognised risk factors in CHD. Reducing LDL cholesterol by 1mmol can reduce CHD risk by 21%.[4]

Statins are first-line therapy for dyslipidaemia. ESC (2012) guidelines suggest that statins arrest atherosclerotic progression. Early studies have proven the efficacy of statins – the Scandinavian Simvastatin Survival Study 1994 documented notable reductions in total and LDL cholesterol with reductions in morbidity and mortality.[5]

Where statin intolerance develops, other drugs may be used – fibrates, Niacin or Ezetrol. Certain high-risk patients may require a combination of these therapies.

Q8.

Guidelines advocate intensive and immediate treatment (80mgs Atorvastatin) in the post-MI period.[1]

Statins inhibit cholesterol synthesis, thereby lowering levels of atherogenic LDLs.[1,4] An optimal target LDL of <1.8mmol/l is suggested as this has been attributed to a lower risk of recurrence of cardiovascular events.[1,2]

The use of the maximum tolerated dose of a statin must be balanced against the potential risk of adverse effects eg. Myositis or liver dysfunction.

Q9.

TEST	RATIONALE	ACTION
Lipids	4-6 weeks after initiation of statin, a reduction in total and LDL cholesterol should be evident.[1,2]	Query compliance. Atorvastatin can be taken in the morning. Reiterate lifestyle advice. If target LDL achieved, consider reducing statin dose
Liver tests	To monitor for hepatic impairment.[1]	If elevated, discontinue/change statin. Monitor for drug interactions e.g. avoid grapefruit, certain antibiotics (Clarithromycin)
CK (Creatine Kinase)	If patient reports new myalgia. To monitor for development of myopathy. Avoid progression to rhabdomyolysis.[4]	If elevated, discontinue drug. Check for drug interactions.
Thyroid function	TFTs if not already completed to rule out secondary dyslipidaemia.[4]	If TFTs indicate hypothyroidism, commence Eltroxin.

Q10.

Asymptomatic individuals with elevated total and LDL cholesterol are risk assessed for the probability of developing CVD over a 10 year period. Lifestyle (diet and exercise)

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modification is strongly advised to achieve a target TC of <5mmol/l.

Moderate risk individuals should aim for LDL cholesterol of <3mmol/l.

Control is tightened for patients with high risks to <2.5mmol/l. This may be achieved by intensive lifestyle advice +/ – statin addition.

Very high risk patients (previous cardiac event, with multi-morbidities or risk factors), should strive for a LDL of <1.8mmol/l or at least a 50% reduction in baseline LDL.[1]

Q11.

In Ireland, diabetes has increased by 13% since 1980s[17] and is predicted to rise by 37% from 2005-2015.[13] Hyperglycaemia increases risks of cardiovascular and microvascular (retinopathy, nephropathy and neuropathy) complications.

In asymptomatic patients, a diagnosis is confirmed by 2 abnormal results, either fasting plasma glucose or HbA_{1c}.[13]

Oral glucose tolerance testing (OGTT) is recommended where fasting levels exceed 5.6mmol/l or HbA_{1c} is 5.7-6.4%. Target HbA_{1c} is <7.0%.[1] The following table classifies Michael's potential results.

DIAGNOSIS	RESULT
Impaired fasting glucose	Fasting glucose of 5.6 – 6.9 mmol/l on 2 separate occasions.
Impaired glucose tolerance	Post OGTT – 2 hour glucose 7.8 – 11.0 mmol/l.
Type 2 diabetes	Post OGTT – 2 hour glucose of >11.1mmol/l.

Michael needs assessment for detection of symptoms associated with type 2 diabetes. He is at risk of metabolic syndrome and insulin resistance.[11,12] having risk factors of hypertension, dyslipidaemia, raised BMI, established heart disease, first degree relative with diabetes and impaired fasting glucose.

Management of risk factors through diet and exercise can reduce HbA_{1c} by 2%.[13] Metformin, if tolerated, may need to be introduced as a first-line drug therapy. This drug is associated with reducing cardiovascular morbidity.[13]

Q12.

Michael has Grade 1 hypertension with BP of 152/96mmHg, however, this may simply be white coat hypertension.

Guidelines suggest a target BP for patients with CVD of 130-139/80-85mmHg. Lower targets have not consistently been supported in research, except for patients with CVAs.

Persistently elevated BP needs to be confirmed by means of either[1]

1. Ambulatory BP monitoring – targets of 125-130/80mmHg.
2. Home BP monitoring – target of 130-135/85mmHg.

If hypertension is confirmed, Michael needs advice regarding lifestyle modification, namely, smoking cessation, weight management, alcohol moderation, regular exercise, low-salt, low-fat diet with improved intake of fruit and vegetables (Dietary Approaches to Stop Hypertension (DASH) diet).[9] Potassium in fruit and vegetables will help to reduce BP however, given that Michael is on ACEi also, it may be prudent to monitor serum electrolytes, to reduce risk of hyperkalaemia.[10]

Michael's anti-hypertensive medications, Ramipril and Bisoprolol can be titrated to maximum tolerated doses. Combination therapy is more beneficial for BP control than simply maximising the dose of one drug.[1]

Conduct an ECG to rule out evidence of left ventricular hypertrophy (LVH) which is an independent CV risk.

Assess renal damage by checking urine to measure creatinine and glomerular filtration rate (GFR) and to detect microalbuminuria or proteinuria.

Ensure Michael's compliance with therapy by clarifying any issues that may arise and offer support and counselling, as required, in the post-MI period.

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