

038 - Pages 926-950

- [038](#)

038

Pages 926-950

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Risk factors The highest prevalence of myocardial infarction is 72 hours post operation. Patients with diabetes may not have chest pain due to autonomic dysfunction. Investigations • ECG (best initial test) • Cardiac troponin levels: Measure as soon as possible and repeat after 1-6 hours. • Transthoracic echocardiography: if the diagnosis is unclear. Findings: □ Wall motion abnormalities □ Decreased LV function Myocardial infarction: management

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

- Glyceryl trinitrate □ Sublingual glyceryl trinitrate and intravenous morphine + metoclopramide should be given to help relieve the symptoms. □ ongoing pain despite the use of sublingual GTN is suggestive of continuing myocardial ischaemia/infarction □ IV GTN • Aspirin 300mg. □ the initial drug therapy □ Aspirin 300mg should be given to all patients (unless contraindicated). □ It is safe in the post-surgical patient with no signs of bleeding at three days post operation. □ A second antiplatelet is normally given, usually ticagrelor, clopidogrel or prasugrel (all are antagonists of the P2Y12 adenosine diphosphate receptor). □ (Aspirin + ticagrelor) is better than (aspirin + clopidogrel) □ ticagrelor was associated with a 13% relative reduction in cardiovascular events versus a conventional clopidogrel based regimen. □ This has driven use of ticagrelor in place of clopidogrel in major guidelines on antiplatelet therapy post STEMI. □ A loading dose of 180 mg stat is recommended at the time of diagnosis of STEMI. □ This was also associated with increased risk of bleeding events when compared to aspirin and clopidogrel. □ NICE do not recommend giving other antiplatelet agents (i.e. Clopidogrel) outside of hospital. The dose of clopidogrel is 300 mg in ACS. • Other treatments that may be given include bivalirudin (a direct thrombin inhibitor, usually given alongside aspirin + clopidogrel) and a form of heparin (either low-molecular weight or unfractionated). □ Heparin in Non-STEMI (has no benefit in ST elevation MI). • do not routinely give oxygen, only give if sats < 94%* • *NICE suggest the following in terms of oxygen therapy: □ do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to: □ people with oxygen saturation (SpO2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO2 of 94-98% □ people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO2 of 88-92% until blood gas analysis is

available. □ ESC guidelines 2017 state that: routine oxygen is not recommended when SaO₂ is ≥ 90%. • perform an ECG as soon as possible but do not delay transfer to hospital. A normal ECG does not exclude ACS • percutaneous coronary intervention (PCI) □ is the first-line and the gold-standard treatment management to revascularise the myocardium. □ but is not available in all centres. Thrombolysis should be performed in patients without access to primary PCI □ offer primary PCI to patients who present within 12 hours of onset of symptoms, if it can be delivered within 120 minutes of the time when fibrinolysis could have been given. A practical example may be a patient who presents with a STEMI to a small district general hospital (DGH) which does not have facilities for PCI. If they cannot be transferred to a larger hospital for PCI within 120 minutes then fibrinolysis should be given. If the patient's ECG taken 90 minutes after fibrinolysis failed to show resolution of the ST elevation then they would then require transfer for PCI.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Percutaneous coronary intervention (PCI) • PCI is a technique used to restore myocardial perfusion in patients with ischaemic heart disease, both in patients with stable angina and acute coronary syndromes. • Stents are implanted in around 95% of patients - it is now rare for just balloon angioplasty to be performed • Following stent insertion migration and proliferation of smooth muscle cells and fibroblasts occur to the treated segment. The stent struts eventually become covered by endothelium. Until this happens there is an increased risk of platelet aggregation leading to thrombosis. • Following insertion, the most important factor in preventing stent thrombosis is antiplatelet therapy. Aspirin should be continued indefinitely. The length of clopidogrel treatment depends on the type of stent, reason for insertion and consultant preference • How long should he continue dual antiplatelet therapy following stent insertion? □ 12 months □ When dual therapy is maintained for less than 12 months, early cessation of clopidogrel is associated with an increased risk of further ischaemic events. □ Thrombosis of a drug-eluting stent is associated with high morbidity (42%) and mortality (71%). For this reason, dual antiplatelet therapy (usually aspirin and clopidogrel) is continued for at least twelve months following the insertion of this type of stent. • Elective surgery should be postponed for twelve months when it is considered safe to stop clopidogrel and continue with aspirin. Complications: Two main complications may occur

1. Stent thrombosis: □ due to platelet aggregation as above. □ Occurs in 1-2% of patients, most commonly in the first month. □ Usually presents with acute myocardial infarction □ Treated by primary angioplasty.
2. Restenosis: □ due to excessive tissue proliferation around stent. □ Occurs in around 5-20% of patients, most commonly in the first 3-6 months. □ Usually presents with the recurrence of angina symptoms. □ Risk factors include diabetes, renal impairment and stents in venous bypass grafts □ In patients with type-2 diabetes, uncoated coronary stents are liable to re-stenosis at a rate of 40-50% by the end of a 6-month

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

□ Drug eluting stents have been shown to reduce the relative risk of re-stenosis by around 80%, but only where dual anti-platelet therapy with clopidogrel and aspirin is continued for at least 1 year. Types of stent • bare-metal stent (BMS) • drug-eluting stents (DES): stent coated with paclitaxel or rapamycin which inhibit local tissue growth. Whilst this reduces restenosis rates the stent thrombosis rates are increased as the process of stent endothelialisation is slowed Thrombolysis Thrombolysis is no longer indicated except in the context of STEMI where PCI is not available within 90 minutes of first medical contact. • ECG criteria for thrombolysis within 24 hours of typical pain include: □ ST elevation of more than 1 mm in in two adjacent limb leads. □ ST elevation more than 2 mm in in two adjacent anterior chest leads. □ new left bundle branch block. • Pre-hospital thrombolysis is indicated if the time from the initial call to arrival at hospital is likely to be over 30 minutes. □ When primary percutaneous coronary intervention cannot be provided within 120 minutes of ECG diagnosis, patients with STEMI should receive immediate (prehospital or admission) thrombolytic therapy □ (NICE) recommends using intravenous bolus (reteplase or tenecteplase) rather than an infusion for pre-hospital thrombolysis • Thrombolitics □ tissue plasminogen activator (tPA) has been shown to offer clear mortality benefits over streptokinase □ streptokinase □ mechanism of action □ Combining with plasminogen to form a complex □ Streptokinase forms a 1:1 complex with plasminogen that induces structural changes in the protein that activates it without direct cleavage of the Arg-Val bond. it is not specific for fibrin-bound plasminogen. □ alteplase □ Unlike streptokinase, alteplase activates plasminogen bound to fibrin without activating unbound plasminogen proteins. □ it is not associated with hypotension or allergic reactions like streptokinase. □ It has a much shorter half-life of only 3-4 minutes compared to 18 minutes for streptokinase. □ tenecteplase □ easier to administer □ has been shown to have non-inferior efficacy to alteplase with a similar adverse effect profile • ECG should be performed 90 minutes following thrombolysis to assess whether there has been a greater than 50% resolution in the ST elevation □ if there has not been adequate resolution then rescue PCI is superior to repeat thrombolysis □ for patients successfully treated with thrombolysis PCI has been shown to be beneficial. The optimal timing of this is still under investigation • Contraindications to thrombolysis include: □ Gastrointestinal (GI) bleeding in the preceding three weeks. □ Heavy vaginal bleeding □ Ischaemic stroke in last six months □ Previous history of hemorrhagic stroke □ Uncontrolled severe hypertension

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

□ Prolonged cardiopulmonary resuscitation (CPR) (more than half an hour). □ Known or suspected aortic dissection □ Known bleeding disorder □ Major surgery or serious trauma within two weeks. □ Lumbar puncture in the preceding week. • Relative contraindications □ Proliferative diabetic retinopathy, □ allergy and □ oral anticoagulants • Risk factors for bleeding □ Advancing age □ Renal impairment □ Low body weight and □ Known bleeding problems. Management of hyperglycaemia in acute coronary syndromes • the most appropriate treatment for his glycaemic control □ Commence intravenous insulin infusion and stop metformin □ metformin □ increased risk of lactic acidosis. • Nice in 2011 recommends using a dose-adjusted insulin infusion with regular monitoring of blood glucose levels to glucose below 11.0 mmol/l • The Diabetes and Insulin-Glucose Infusion in Acute Myocardial Infarction (DIGAMI) study demonstrated significant reductions in mortality in subjects with diabetes and myocardial infarction (MI) treated with IV insulin infusion (followed by three months of sc insulin) compared with conventional therapy with their oral

hypoglycaemic agents. □ intensive insulin therapy (an intravenous infusion of insulin and glucose with or without potassium, sometimes referred to as 'DIGAMI') regimes are not recommended routinely MRCPUK-part-1-january-2018: What is the mode of action of alteplase? □ Plasminogen activator □ Induce conversion of plasminogen to plasmin leading to the dissolution of a fibrin clot. Myocardial infarction: complications

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Myocardial infarction complications Complication Notes Cardiac arrhythmia Occurs within the first few days after MI. Important cause of death before reaching the hospital and within the first 24 hours post-MI. Ventricular fibrillation is the most common cause of death following a MI. Atrioventricular block is more common following inferior myocardial infarctions. LV failure and pulmonary oedema Can occur 2° to LV infarction, VSD, free wall rupture, papillary muscle rupture with mitral regurgitation. Post infarction fibrinous pericarditis 1–3 days: common around 10% of patients. friction rub Papillary muscle rupture (leads to acute mitral regurgitation). 2–7 days: posteromedial papillary muscle rupture. ↑ risk due to single blood supply from posterior descending artery. More common with infero-posterior infarction. Suddenly develops pulmonary oedema and a loud systolic murmur at the apex which radiated into the axilla with associated pulmonary oedema. often require emergency surgical repair. Interventricular septal rupture 3–5 days: macrophage-mediated degradation → VSD → ↑ O₂ saturation and pressure in RV. acute heart failure associated with a pan-systolic murmur. An echocardiogram is diagnostic and will exclude acute mitral regurgitation which presents in a similar fashion. Urgent surgical correction is needed. Ventricular pseudoaneurysm formation 3–14 days: free wall rupture contained by adherent pericardium or scar tissue; low cardiac output, risk of arrhythmia, embolus from mural thrombus. True ventricular aneurysm 2 weeks to several months: outward bulge with contraction (“dyskinesia”), associated with fibrosis. typically associated with persistent ST elevation and left ventricular failure. Thrombus may form within the aneurysm increasing the risk of stroke. Patients are therefore anticoagulated. Ventricular free wall rupture 5–14 days: present with acute heart failure secondary to cardiac tamponade (raised JVP, pulsus paradoxus, diminished heart sounds). LV hypertrophy and previous MI protect against free wall rupture. Urgent pericardiocentesis and thoracotomy are required. Dressler syndrome Several weeks: autoimmune phenomenon resulting in fibrinous pericarditis. characterised by a combination of fever, pleuritic pain, pericardial effusion, friction rub on auscultation and a raised ESR. Treated with NSAIDs. Chronic heart failure The most important factor predicting outcomes post-STEMI is the presence of new systolic heart failure. Primary prevention drugs which have evidence for the reduction of risk of developing a cardiac event? • Angiotensin converting enzyme inhibitor □ The most appropriate treatment to reduce cardiovascular risk should focus on adequate blood pressure control □ ↓ BP is most important than control of DM and lipids in CV risk reduction • Aspirin • Metformin □ treatment of overweight, diabetic patients with metformin, lowers the relative risk of (MI) by 40%, as opposed to treatment with sulphonylureas or insulin. • Statins

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Myocardial infarction: secondary prevention All patients should be offered the following drugs: • dual antiplatelet therapy (aspirin plus a second antiplatelet agent) • ACE inhibitor • beta-blocker • statin Some selected lifestyle points: • Diet: □ advise a Mediterranean style diet, switch butter and cheese for plant oil-based products. □ Do not recommend omega-3 supplements or eating oily fish • Exercise: □ advise 20-30 mins a day until patients are 'slightly breathless' • Sexual activity □ may resume 4 weeks after an uncomplicated MI. □ Reassure patients that sex does not increase their likelihood of a further MI. □ PDE5 inhibitors (e.g, sildenafil) may be used 6 months after a MI. □ They should however be avoided in patient prescribed either nitrates or nicorandil Clopidogrel • STEMI: □ the European Society of Cardiology recommend dual antiplatelets for 12 months. In the UK this means aspirin + clopidogrel • Non-ST segment elevation myocardial infarction (NSTEMI): □ following the NICE 2013 guidelines, clopidogrel should be given for the first 12 months Aldosterone antagonists • patients who have had an acute MI and who have symptoms and/or signs of heart failure and left ventricular systolic dysfunction, treatment with an aldosterone antagonist licensed for post-MI treatment (e.g. eplerenone) should be initiated within 3-14 days of the MI, preferably after ACE inhibitor therapy

Hyperlipidaemia: management See endocrinology

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Heart failure Definition • structural or functional impairment of ventricular filling and/or ejection of blood. Types • Heart failure with reduced ejection fraction (HFrEF) □ Reduced contractility → systolic ventricular dysfunction → decreased left ventricular ejection fraction (LVEF) → decreased cardiac output □ Causes include: □ Damage and loss of myocytes (e.g., following myocardial infarction, coronary artery disease, dilated cardiomyopathy) □ Cardiac arrhythmias □ High-output cardiac failure □ A state of heart failure characterized by increased cardiac output and lowered systemic vascular resistance. May be caused by arteriovenous fistulas, renal disease, anemia, beriberi, or Graves' disease. • Heart failure with preserved ejection fraction (HFpEF) □ Decreased ventricular compliance → diastolic ventricular dysfunction → reduced ventricular filling and increased diastolic pressure → decreased cardiac output (while the left ventricular ejection fraction remains normal) □ Causes include: □ Increased stiffness of the ventricle (e.g., long-standing arterial hypertension with ventricular wall hypertrophy, restrictive cardiomyopathy) □ Impaired relaxation of the ventricle (e.g., constrictive pericarditis, pericardial tamponade) NYHA classification • The New York Heart Association (NYHA) classification is widely used to classify the severity of heart failure: □ NYHA Class I □ no symptoms □ no limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations □ NYHA Class II □ mild symptoms □ slight limitation of physical activity: comfortable at rest but ordinary activity results in fatigue, palpitations or dyspnoea □ NYHA Class III □ moderate symptoms □ marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms □ NYHA Class IV □ severe symptoms □ unable to carry out any physical activity without discomfort: symptoms of heart failure are present even at rest with increased discomfort with any physical activity

Diagnosis (NICE 2010) • Patient with previous myocardial infarction □ arrange echocardiogram within 2 weeks □ if transthoracic doppler 2D echocardiography imaging is poor (eg: in obese) □ consider other imaging methods, such as: □ radionuclide angiography, □ cardiac magnetic resonance imaging or □ trans-oesophageal Doppler 2D echocardiography. • No previous myocardial infarction □ measure serum natriuretic peptides (BNP) □ if levels are 'high' (> 400) arrange echocardiogram within 2 weeks □ if levels are 'raised' (100-400) arrange echocardiogram within 6 weeks □ 40% of patients with raised BNP will have left ventricular systolic dysfunction on echo. the remaining will have other cardiac abnormalities. □ if levels are 'normal' (< 100) heart failure is unlikely (investigate for other causes) B-type natriuretic peptide (BNP) • Source □ produced mainly by the left ventricular myocardium in response to strain. • Effect □ The net effect of these peptides is: □ ↓BP (due to the decrease in systemic vascular resistance) and, thus, afterload on the heart. □ ↓cardiac output (due to an overall decrease in central venous pressure) and preload as a result of the reduction in blood volume that follows natriuresis and diuresis. • Uses □ normal level rules out acute heart failure in the emergency setting □ Very high levels are associated with a poor prognosis. • Excretion □ Less than 5% of BNP is cleared renally whereas NT-proBNP is reliant solely on the kidney for excretion and hence it is unreliable in patients with coexistent renal dysfunction. BNP NTproBNP High levels

“ 400 pg/ml (116 pmol/litre) 2000 pg/ml (236 pmol/litre) Raised levels 100-400 pg/ml (29-116 pmol/litre) 400-2000 pg/ml (47-236 pmol/litre) Normal levels < 100 pg/ml (29 pmol/litre) < 400 pg/ml (47 pmol/litre) Diagnosis of acute heart failure (Nice guidelines 2014): • In people presenting with new suspected acute heart failure: □ rule out the diagnosis of heart failure if : □ BNP less than 100 ng/litre □ NT- proBNP less than 300 ng/litre. □ new suspected acute heart failure with raised natriuretic peptide levels □ perform transthoracic Doppler 2D echocardiography (within 48 hours of admission)

Cardiology

Factors, which alter the BNP level: Increase BNP levels Decrease BNP levels • Left ventricular hypertrophy • Aortic stenosis, • Hypertension • Ischaemia • Tachycardia • Right ventricular overload • Hypoxaemia (including pulmonary embolism) • GFR < 60 ml/min • Sepsis • COPD, Cor pulmonale • Diabetes • Age > 70 • Liver cirrhosis • Hyperaldosteronism • Cushing's syndrome • Stable angina, Acute coronary syndromes • Atrial fibrillation (AF) • Obesity • Diuretics • ACE inhibitors • Beta-blockers • Angiotensin 2 receptor blockers • Aldosterone antagonists Mechanism of central sleep apnea (CSA) in HF: • Which mechanism is responsible for the patient's polysomnography findings in heart failure? □ Increased sensitivity to carbon dioxide and stimulation of the vagal receptors. □ increased sensitivity to PaCO₂ is a protective mechanism from

hypercapnia due to heart failure. □ HF □ ↑ duration of circulation of blood gases from the lungs to the brain. □ When these blood gases reach the brain, the increased sensitivity to PaCO₂ □ higher-than-normal response of hyperventilation □ ↓ PaCO₂ lower than the apneic threshold. □ As soon as the brain detects low PaCO₂ it will cease ventilation with apnea (central) so PaCO₂ can rise again. □ As soon as the PaCO₂ rises again and reaches the brain (longer than normal due to heart failure), it will cause another episode of hyperventilation. □ supine position □ ↑ venous return □ pulmonary congestion □ activate vagal receptors □ hyperventilation. Hyponatraemia in patients with CHF • Water restriction is the first-line and mainstay of therapy • Stopping furosemide will not be possible for a patient who has decompensated heart failure. • Similarly, administration of hypertonic saline is only indicated if there is neurological manifestation of hyponatremia. • Moreover hypertonic or isotonic saline administration will be poorly tolerated in a volumeoverloaded patient. • associated with the worst prognosis

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Investigations • Chest x-ray: Features of pulmonary oedema on a chest x-ray may include: □ interstitial oedema □ bat's wing appearance □ upper lobe diversion (increased blood flow to the superior parts of the lung) □ Kerley B lines □ pleural effusion □ cardiomegaly may be seen if there is cardiogenic cause Typical CXR signs associated with heart failure The most common cause of flash pulmonary oedema is myocardial ischaemia. Bilateral renal artery stenosis is a less common cause of flash pulmonary oedema. Pharmacological management Acute heart failure management • Initial pharmacological treatment □ intravenous diuretics • Initial non-pharmacological treatment □ cardiogenic pulmonary oedema with severe dyspnoea and acidaemia consider starting non-invasive ventilation □ in this case it is the most useful next step , before diuretics . The effect of the diuresis comes much later and has a modest overall contribution in managing the symptoms of shortness of breath. □ Consider invasive ventilation in acute heart failure that, despite treatment, is leading to or is complicated by: respiratory failure or reduced consciousness or physical exhaustion.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

In a person presenting with acute heart failure who is already taking beta-blockers: • continue the beta-blocker treatment unless they have a heart rate less than 50 beats per minute, second or third degree atrioventricular block, or shock. • restart beta-blockers once their condition has been stabilised – for example, when intravenous diuretics are no longer needed. In a person presenting with acute heart failure who is already taking frusemide 80 mg: • in a patient with evidence of decompensated heart failure and fluid overload. The most appropriate initial management is to Increase furosemide and relieve the symptoms of fluid overload – pulmonary and peripheral oedema. Chronic management • 4 drugs have been shown to improve mortality in patients with chronic heart failure:

1. ACE inhibitors

2. spironolactone
3. beta-blockers
4. hydralazine with nitrates • No long-term reduction in mortality has been demonstrated for loop diuretics such as furosemide. • In patients with symptoms of heart failure not controlled on ACE inhibitors alone, switching to the combination of ARB and neprilysin inhibitor can further improve symptoms and quality of life. □ e.g: combination of sacubitril and valsartan reduced cardiovascular death and heart failure hospitalisations by 20%. • NICE issued updated guidelines on management in 2010, key points include: □ first-line treatment for all patients is both an ACE-inhibitor and a beta-blocker □ With the persisting symptoms despite 80 mg of furosemide, guidelines would initially suggest the addition of an ACE inhibitor. □ Although beta-blockers would be of further benefit in this patient, it is important first to establish him on ACEi and then introduce betablockers like carvedilol, metoprolol or bisoprolol in a small dose and gradually increase. □ second-line treatment is now either an aldosterone antagonist, angiotensin II receptor blocker or a hydralazine in combination with a nitrate □ if symptoms persist cardiac resynchronisation therapy or digoxin should be considered □ digoxin has also not been proven to reduce mortality in patients with heart failure. □ It may however improve symptoms due to its inotropic properties. □ Digoxin is strongly indicated if there is coexistent atrial fibrillation □ There is no evidence that increasing a dose of digoxin above 62.5 µg in a patient in sinus rhythm would have any added benefit. □ diuretics should be given for fluid overload □ offer annual influenza vaccine □ offer one-off pneumococcal vaccine □ adults usually require just one dose but those with asplenia, splenic dysfunction or chronic kidney disease need a booster every 5 years Drugs that improve prognosis are beta blockers, ACE inhibitors, ARNIs, aldosterone antagonists, and hydralazine with nitrate.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

MRCPUK-part-1-jan-2018: In a patient with significant heart failure on maximum medical therapy (ramipril 10 mg OD, furosemide 80 mg OD, bisoprolol 10 mg OD and spironolactone 25 mg OD). Despite this, they have continued to deteriorate but criteria for cardiac resynchronisation therapy (CRT) are not achieved. What is the most appropriate next step to improve mortality? □ Ivabradine □ acts as an inhibitor of the If current within the myocardium. This current, particularly present in the sino-atrial and atrio-ventricular nodes, acts as the cardiac pacemaker. □ By inhibiting this current, ivabradine reduces the heart rate without impacting the force of cardiac contraction. □ This has been shown to reduce heart failure hospitalisation and mortality in patients already on maximum medical therapy. □ Due to its mechanism, ivabradine is only effective in patients in sinus rhythm. If known case of heart failure – on β-blocker – presented with acute pulmonary oedema □ Increase diuretics, stop β-blockers and restart β-blockers when his lungs are dry. A significant benefit from using IV iron in patients with heart failure and iron deficiency was demonstrated in a study history of heart failure + iron deficiency. the first step □ correcting iron deficiency Non- pharmacological management • Cardiac resynchronisation therapy (CRT) (biventricular pacing): criteria for resynchronisation therapy recommended by NICE guidance

1. They are in sinus rhythm + □ either with a QRS duration of ≥ 150 ms estimated by ECG (LBBB) □ or with a QRS duration of 120-149 ms estimated by ECG and mechanical

dyssynchrony that is confirmed by echocardiography

2. They have a left ventricular ejection fraction of $\leq 35\%$.
3. They are receiving optimal pharmacological therapy. • Benefit : Improved symptoms and reduced hospitalisation in NYHA class III patients • Investigations : the most useful investigation in predicting symptomatic response to cardiac resynchronisation therapy is ☐ transthoracic echocardiogram and ECG (The echo will show asynchronous contraction of the LV and RV and subsequently reduced ejection fraction). • Complications: When a CRT device is implanted the left ventricular lead is inserted in the coronary sinus. To obtain access to the coronary sinus a catheter with an aggressive tip is used. There is a 1% risk of causing dissection/perforation to the coronary sinus which can lead to cardiac tamponade. Implantable cardioverter defibrillator (ICD) • Where there is no LBBB and QRS is between 120-149 ms, ICD is the recommended option according to NICE guidelines. This is because of the risk of VT on account of the low ejection fraction, ($<35\%$), and symptomatic heart failure.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Exercise training • improves symptoms but not hospitalisation/mortality Tocolysis-associated pulmonary oedema • Tocolytics are medications administered for the suppression of premature contractions. • Acute pulmonary oedema can occur with administration of β_2 agonists for tocolysis in up to 5–15% of cases. • It usually occurs after 24 h of administration of these agents. • The chest X-ray reveals pulmonary infiltrates and normal heart size. • Concomitant use of corticosteroids that are often administered for lung maturation have also been implicated as risk factor for development of tocolysis-associated pulmonary oedema. • Treatment involves stopping the tocolytics, oxygen and careful volume control. • Differential: ☐ Peripartum cardiomyopathy: ☐ typically presents in the last month of pregnancy and up-to 6 months postpartum. ☐ cardiomegaly on chest X-ray. NICE management summary

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Other management options If heart failure is caused or worsened by other conditions, these should be managed appropriately: • Revascularisation (e.g. coronary artery bypass grafting) • Valve surgery (e.g. aortic valve replacement) • Implantable cardiac defibrillator (ICD): inserted if EF $<30\%$ for prevention of fatal arrhythmias • Cardiac resynchronisation therapy + defibrillator (CRT-D): a biventricular pacemaker for EF $<30\%$ + QRS >130 m/sec to re-synchronise left and right ventricular contraction to improve EF • Cardiac transplantation is rare and strict criteria must be met for consideration. ☐ By five years following cardiac transplantation, nearly all patients have some degree of small coronary vascular narrowing (Coronary arteriopathy). Potentially harmful drugs to avoid in heart failure Drug to avoid Notes Non-steroidal anti-inflammatory drugs (NSAIDs) • May cause sodium and water retention, peripheral vasoconstriction, worsen heart failure, and decrease renal function. • Acute renal failure may be more likely when these agents are used in combination with an ACE inhibitor (ACEI) / angiotensin receptor blocker (ARB) and/or diuretic. Non-

dihydropyridine calcium channel blockers –verapamil and diltiazem1 • Negative inotropic effect may further depress cardiac function. Risk is greatest with verapamil, then diltiazem and least risk with dihydropyridines, but use with caution • Non-dihydropyridine calcium channel blockers are contraindicated in systolic heart failure , but may be useful in heart failure with preserved ejection fraction where slowing heart rate can increase filling time Tricyclic antidepressants May prolong QT interval and cause arrhythmias as well as hypotension from alpha-blocking effects Thiazolidinediones (e.g. pioglitazone) May cause fluid retention and heart failure by increasing renal sodium reabsorption Corticosteroids May worsen heart failure due to sodium and water retention (mineralocorticoid effect) Clozapine May cause cardiomyopathy and myocarditis Oncology treatments such as anthracyclines (doxorubicin, daunorubicin), trastuzumab may cause heart failure Tumour necrosis factor antagonists (e.g. infliximab, etanercept) May cause heart failure Moxonidine (centrally acting antihypertensive) Contraindicated in heart failure. Associated with increased mortality in heart failure

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology Prognosis • Prognosis is poor overall, with approximately 50% of people with heart failure dying within five years of diagnosis • Factors indicating worse prognosis in heart failure □ High BNP/NT-pro-BNP □ Anaemia □ Hyponatraemia □ Increased uric acid. Mechanical support with the insertion of an intra-aortic balloon pump (IABP) in patient with Cardiogenic shock • In case with hypotension and cardiogenic shock, what is the most appropriate intervention after failure of an inotropic support treatment? □ Intra-aortic balloon counter pulsation (IABCP) to support cardiac output. • An intra-aortic balloon pump is inserted under echocardiographic guidance. At which point of the ECG should balloon inflation be timed? □ Middle of the T wave □ Balloon inflation is timed with diastole once closure of the aortic valve has occurred; this corresponds to the middle of the T wave. • What is the contraindication to placement of an intra-aortic balloon pump? □ For blood to be ejected antegrade to perfuse the tissues and retrograde to perfuse the coronaries, the aortic valve must be closed and competent. Aortic regurgitation is therefore a contraindication to placement of an intra-aortic balloon pump.

Hypertrophic obstructive cardiomyopathy (HOCM) HOCM is the most common cause of sudden cardiac death in the young • (HOCM) is an autosomal dominant disorder of muscle tissue caused by defects in the genes encoding contractile proteins. • The most common defects involve a mutation in the gene encoding β -myosin heavy chain protein or myosin binding protein C. • Mutations to various proteins including beta-myosin, alpha-tropomyosin and troponin T have been identified. • type of mutation □ Frame-shift mutation • The estimated prevalence is 1 in 500. • Septal hypertrophy causes left ventricular outflow obstruction. • It is an important cause of sudden death in apparently healthy individuals.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Features Symptoms and signs are similar to those of aortic stenosis, except that the character of the pulse in HOCM is jerky • often asymptomatic • dyspnea (the most common presenting

symptom) • angina, • syncope • sudden death (most commonly due to ventricular arrhythmias), arrhythmias, heart failure • jerky pulse, • large 'a' waves, • double apex beat • ejection systolic murmur: increases with Valsalva manoeuvre and decreases on squatting □ Diastolic decrescendo murmur of aortic regurgitation (10% of patients) Associations • Friedreich's ataxia • Wolff-Parkinson White Echo - mnemonic - MR SAM ASH • mitral regurgitation (MR) • systolic anterior motion (SAM) of the anterior mitral valve leaflet • asymmetric hypertrophy (ASH) ECG • left ventricular hypertrophy • progressive T wave inversion • deep Q waves • right or left axis deviation • PR prolongation • atrial fibrillation may occasionally be seen • Right bundle branch block □ the most ECG FINDING which support a diagnosis of HOCM □ RBBB is correlated with anterior, anteroseptal and mid-septal myocardial fibrosis in HOCM.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

ECG showing typical changes of HOCM including LVH and T wave inversion Dagger-like Q waves

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

This ECG shows the typical pattern of apical HCM: • Large precordial voltages. • Giant T wave inversions in the precordial leads • Inverted T waves are also seen in the inferior and lateral leads. Type of cardiomyopathy Selected points Hypertrophic obstructive cardiomyopathy • Leading cause of sudden cardiac death in young athletes • Usually due to a mutation in the gene encoding β -myosin heavy chain protein • Common cause of sudden death • Echo findings include: □ MR, □ systolic anterior motion (SAM) of the anterior mitral valve □ asymmetric septal hypertrophy Arrhythmogenic right ventricular dysplasia • Right ventricular myocardium is replaced by fatty and fibrofatty tissue • Around 50% of patients have a mutation of one of the several genes which encode components of desmosome • ECG abnormalities in V1-3, □ typically T wave inversion. □ An epsilon wave is found in about 50% of those with ARV - this is best described as a terminal notch in the QRS complex Management • Amiodarone • Beta-blockers or verapamil for symptoms • Cardioverter defibrillator • Dual chamber pacemaker • Endocarditis prophylaxis

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Beta-blockers • Generally first-line agents □ increase diastolic filling and decrease contractility □ Reduces provokable gradient disopyramide • If β -blockers alone are ineffective, disopyramide, may be added (Class IA antiarrhythmic drug) • anticholinergic side-effects include dryeyes and mouth, urinary hesitancy or retention, and constipation. • QTc interval should be monitored during dose up-titration and the dose reduced if it exceeds 480 ms. • Disopyramide should be avoided in patients with glaucoma, prostatism, and in patients taking other drugs that prolong the QT interval, such as amiodarone and sotalol. Verapamil • Verapamil (starting dose 40 mg three times daily to maximum 480 mg daily) can be used when β -blockers are contraindicated or ineffective, • close

monitoring is required in patients with severe obstruction (≥ 100 mm Hg) or elevated pulmonary artery systolic pressures, as it can provoke pulmonary oedema. • Verapamil should however be avoided in HOCM patients with coexistent Wolff Parkinson White as it may precipitate VT or VF. Implantable cardioverter defibrillators (ICD) implantation □ prevention of sudden cardiac death • recommended in patients who have survived a cardiac arrest due to VT or VF or who have spontaneous sustained VT causing syncope or haemodynamic compromise Invasive treatment (myomectomy or alcohol septal ablation) (ESC Guidelines 2014) • Left ventricular outflow tract obstruction (LVOTO) is defined as a peak instantaneous Doppler LV outflow tract gradient of ≥ 30 mm Hg, but the threshold for invasive treatment is usually considered to be ≥ 50 mm Hg. • Septal reduction therapy is recommended in patients with LVOT gradient of ≥ 50 mm Hg, who are in NYHA functional Class III-IV, despite maximum tolerated medical therapy. • The most commonly performed surgical procedure used to treat LVOTO is ventricular septal myectomy (Morrow procedure). • Pre-operative determinants of a good long-term outcome are age < 50 years, left atrial size < 46 mm, absence of atrial fibrillation and male gender. • surgery VS septal alcohol ablation (SAA) □ both procedures improve functional status with a similar procedural mortality. □ Septal alcohol ablation is associated with a higher risk of AV block, requiring permanent pacemaker implantation and larger residual LV outflow tract gradients. □ In contrast to myectomy, most patients develop right-, rather than left bundle branch block after SAA. Drugs to avoid • nitrates • ACE-inhibitors • Inotropes : Digoxin Poor prognostic factors, which are predictive of sudden cardiac death

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- syncope • family history of sudden death • Maximum left ventricular wall thickness greater than 3 cm • young age at presentation • non-sustained ventricular tachycardia on 24 or 48-hour Holter monitoring • Abnormal blood pressure changes on exercise (Blood pressure drop during peak exercise on stress testing). Screening of HOCM
- Current guidelines suggest that a resting ECG and TTE (transthoracic ECHO) are the most effective screening strategies for relatives of patients with HOCM. • Genetic testing is not recommended as a first line screening tool given varying rates of penetrance.

Dilated cardiomyopathy (DCM) Overview • Most common cardiomyopathy • Sex: ♂ $>$ ♀ (approx. 3:1) • dilated heart leading to systolic (+/- diastolic) dysfunction • all 4 chambers affected but LV more so than RV Features • arrhythmias, • emboli □ cardio-embolic stroke, • mitral regurgitation • absence of congenital, valvular or ischaemic heart disease Causes • Common causes □ Idiopathic (approx. 50%) □ alcohol: may improve with thiamine □ postpartum □ hypertension • Other causes □ genetic inherited dilated cardiomyopathy: □ around third of DCM patients □ the majority of defects are inherited in an autosomal dominant fashion although other patterns of inheritance are seen □ infections e.g. Coxsackie B, HIV, diphtheria, parasitic □ endocrine e.g. Hyperthyroidism □ neuromuscular e.g. Duchenne muscular dystrophy □ nutritional e.g. Kwashiorkor, pellagra, thiamine/selenium deficiency □ Selenium deficiency is one of the reversible causes of dilated cardiomyopathy. □ drugs e.g. Doxorubicin □ Infiltrative (may also lead to restrictive cardiomyopathy) e.g. Haemochromatosis, Sarcoidosis Diagnosis □ Echocardiogram • The echo may show: □ Reduced left ventricular ejection fraction, □ myocardial dyssynchrony (myocardial

segments contract at different points in time), □ thinning of the left ventricular wall □ dilated left ventricle.

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Type of cardiomyopathy Selected causes/points Dilated cardiomyopathy Classic causes include • alcohol • Cocksackie B virus • wet beri beri • doxorubicin Restrictive cardiomyopathy Classic causes include • amyloidosis • post-radiotherapy • Loeffler's endocarditis Becker's muscular dystrophy • X-linked recessive disorder resulting from a mutation in the dystrophin gene. • The clinical picture is similar to that of Duchenne's muscular dystrophy but it is much milder. • Patients usually present between the ages of 5 and 15 years, though presentation may not be until the fourth or fifth decade. • Patients may present with heart failure secondary to dilated cardiomyopathy rather than the classic proximal muscle weakness.

Restrictive cardiomyopathy Causes • amyloidosis (e.g. secondary to myeloma) - most common cause in UK □ Cardiac involvement is the most common cause of death in patients with amyloidosis associated with an immunocyte dyscrasia - typically as restrictive cardiomyopathy □ Transthyretin gene mutations can lead to restrictive cardiomyopathy from amyloid deposition in the heart. □ Diagnosis is confirmed by myocardial biopsy, which shows amyloid infiltration when stained with Congo Red. □ myocardial biopsy, which when stained with Congo Red will show "apple green birefringence" amyloid under polarized light. • haemochromatosis • Loeffler's syndrome • sarcoidosis • scleroderma • Radiotherapy • Systemic sclerosis • Carcinoid syndrome.

Pathophysiology: • Proliferation of connective tissue → ↓ elasticity of myocardium → ↓ ventricular compliance → ↓ diastolic filling → atrial congestion → atrial enlargement and severe diastolic dysfunction Features • Physical examination reveals right heart failure with a raised JVP, characteristically showing a prominent deep Y descent • Heart size is often normal. • S 4 heart sound, due to ventricular noncompliance. • Pericardial effusion is common, but rarely causes tamponade • The most characteristic ECG finding of restrictive cardiomyopathy is diffusely diminished voltages • Echocardiography findings □ small thick ventricles and a thick interatrial septum due to amyloid deposits, which have a 'granular sparkling' appearance □ Amyloid deposits in the heart produce generalized thickening of the myocardium (as opposed to asymmetrical septal hypertrophy commonly seen in hypertrophic cardiomyopathy) and diastolic dysfunction. □ impaired relaxation in the diastolic phase. □ bright speckled appearance. Differential diagnosis • constrictive pericarditis □ Features are very similar in constrictive pericarditis, but in constrictive pericarditis: □ the apex is frequently non-palpable due to the thick pericardium □ chest X-ray may show pericardial calcifications Features suggesting restrictive cardiomyopathy rather than constrictive pericarditis • prominent apical pulse • absence of pericardial calcification on CXR • heart may be enlarged • ECG abnormalities e.g. bundle branch block, Q waves Clinical Features of Constrictive Pericarditis and Restrictive Cardiomyopathy Clinical Features Constrictive Pericarditis Restrictive Cardiomyopathy History Prior history of pericarditis or condition that causes pericardial disease Systemic examination

- Heart sounds Pericardial knock, high-frequency sound Presence of loud diastolic filling sound S3, Low-frequency sound Murmurs No murmurs Murmurs of mitral and tricuspid insufficiency apical pulse apex is frequently non-palpable due to the thick pericardium Prior chest radiograph Pericardial calcification Normal results of prior chest radiograph Management • Cardiac transplant Notes & Notes for MRCP By Dr. Yousif Abdallah Hamad

History of systemic disease (eg, amyloidosis, hemochromatosis) prominent apical pulse

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Peripartum cardiomyopathy (PCM) • biventricular heart failure during the third trimester. • the aetiology: unknown, although both myocarditis and low levels of dietary selenium have been postulated as causes. Management • similar to the management of heart failure in any other situation with vasodilators, diuretics and beta blockade. ACE inhibition is reserved for the post-partum period. □ sodium restriction, □ diuretics to optimise the volume status, □ digoxin and afterload-reducing agents. □ Hydralazine • For patients presenting with PCM, defined as left ventricular systolic dysfunction 1 month prior to delivery or 5 months postpartum, volume status should first be managed with diuretics after liaison with obstetricians. Beta-blockers should be added once the patient's volume status is optimised. • Anticoagulation □ Patients with PCM are at risk of thromboembolism due to both hypercoagulable state of pregnancy and stasis of blood in the left ventricle. Therefore, anticoagulation with heparin is recommended. Type of cardiomyopathy Selected points Peripartum cardiomyopathy • Typical develops between last month of pregnancy and 5 months postpartum • More common in older women, greater parity and multiple gestations Takotsubo cardiomyopathy • 'Stress'-induced cardiomyopathy e.g. patient just found out family member dies then develops chest pain and features of heart failure • Transient, apical ballooning of the myocardium • Treatment is supportive

Takotsubo cardiomyopathy Definition: • Takotsubo cardiomyopathy is a type of non-ischaemic cardiomyopathy associated with a transient, apical ballooning of the myocardium. • acute, stress-induced, reversible dysfunction of the left ventricle Epidemiology: • especially postmenopausal women > 60 years Pathophysiology: • emotional/physical stress → massive catecholamine discharge → cardiotoxicity, multivessel spasms and dysfunction → myocardial stunning Features • chest pain • features of heart failure • ST elevation • normal coronary angiogram Treatment • supportive Prognosis: • spontaneous recovery if stressors are avoided

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Congenital heart disease: types Congenital heart disease • cyanotic: TGA most common at birth, Fallot's most common overall • acyanotic: VSD most common cause Acyanotic - most common causes • ventricular septal defects (VSD) - most common, accounts for 30% • atrial septal defect

(ASD) 10%. • patent ductus arteriosus (PDA) • coarctation of the aorta • aortic valve stenosis VSDs are more common than ASDs. However, in adult patients ASDs are the more common new diagnosis as they generally presents later Cyanotic - most common causes • tetralogy of Fallot □ There is a single sound in Fallot's because of an absent P2. □ A Blalock shunt (anastomosis of subclavian artery to pulmonary artery) used to be performed for Fallot's tetralogy and leads to a weak left radial pulse. • transposition of the great arteries (TGA) □ Fallot's is more common than TGA. However, at birth TGA is the more common lesion as patients with Fallot's generally presenting at around 1-2 months □ TGA is usually treated by prostaglandins in order to keep the ductus arteriosus patent (from pulmonary artery to the descending aorta), so some oxygenated blood can reach systemic circulation. • tricuspid atresia • pulmonary valve stenosis • Total anomalous pulmonary venous connection (TAPVC) □ TAPVC consists of an abnormality of blood flow in which all four pulmonary veins drain into systemic veins or the right atrium with or without pulmonary venous obstruction. □ Systemic and pulmonary venous blood mix in the right atrium. Other notes • Aortic regurgitation may be a feature of osteogenesis imperfecta. • Ebstein's anomaly is associated with maternal LiCO₃ use if exposed in the first trimester. • The majority of cases of neonates with complete heart block may be caused by autoimmune disease, particularly anti-RO antibodies in the mother. • Left ventricle (LV) hypoplasia occurs when the left sided chambers fail to develop and blood enters the systemic circulation from the right ventricle via the pulmonary artery and a patent ductus arteriosus.