

Chapter 5

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Tetralogy of Fallot (TOF) • TOF is the most common cause of cyanotic congenital heart disease*. □
 *however, at birth transposition of the great arteries is the more common lesion as patients with TOF generally present at around 1-2 months • It typically presents at around 1-2 months, although may not be picked up until the baby is 6 months old • TOF is a result of anterior malalignment of the aorticopulmonary septum. The four characteristic features are:

1. ventricular septal defect (VSD)
2. right ventricular hypertrophy
3. right ventricular outflow tract obstruction, pulmonary stenosis □ There is a single sound in Fallot's because of an absent P2.
4. overriding aorta • The severity of the right ventricular outflow tract obstruction determines the degree of cyanosis and clinical severity Other features • cyanosis • causes a right-to-left shunt • ejection systolic murmur due to pulmonary stenosis (the VSD doesn't usually cause a murmur) • a right-sided aortic arch is seen in 25% of patients • chest x-ray shows a 'boot-shaped' heart, ECG shows right ventricular hypertrophy

Notes & Notes for MRCP

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Management • surgical repair is often undertaken in two parts • cyanotic episodes may be helped by beta-blockers to reduce infundibular spasm The most common residual lesion in repaired tetralogy of Fallot is pulmonary regurgitation.

Ventricular septal defects (VSD) Overview • The second most common congenital heart defect □ bicuspid aortic valve is the most common congenital heart defect • They close spontaneously in around 50% of cases. • the most common site for a VSD □ Perimembranous □ Perimembranous VSDs account for 70-80% of VSDs and are situated between the inlet and outlet portions of the septum. Associations • Congenital VSDs: associated with: □ chromosomal disorders (e.g. Down's syndrome, Edward's syndrome, Patau syndrome) • Non-congenital causes include: □ Fetal alcohol

syndrome □ Intrauterine infection (e.g., TORCH) □ post myocardial infarction

Features • Pan-systolic murmur which is: □ louder in smaller defects □ usually loudest at the left lower sternal edge (LSE) • Mid-diastolic murmur over cardiac apex □ Due to increased flow through the mitral valve • systolic thrill • Loud pulmonic S2 (if pulmonary hypertension develops)

Investigations • Chest x-ray □ Enhanced pulmonary vascular markings □ Left atrial and ventricular enlargement • ECG □ The clue to diagnosis in the ECG finding □ Biventricular hypertrophy □ Biventricular hypertrophy is classically described as having biphasic QRS complexes in V2-5 – which is known as the Katz Wachtel phenomenon and is classic for VSD. • Doppler echocardiography: confirms diagnosis

Complications • Aortic regurgitation □ due to a poorly supported right coronary cusp resulting in cusp prolapse • Infective endocarditis • Eisenmenger's complex • Right heart failure • Pulmonary hypertension □ pregnancy is contraindicated in women with pulmonary hypertension as it carries a 30-50% risk of mortality.

Treatment • small to moderate defects often heal spontaneously

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

• Symptomatic and large VSDs □ Surgical (patch) repair • Heart-lung transplant or lung transplant with concurrent VSD repair if Eisenmenger's reaction has occurred

Atrial septal defect (ASD) • common congenital heart lesion □ VSD is more common

Types • Ostium secundum □ 70% of ASDs □ associated with Holt-Oram syndrome (tri-phalangeal thumbs) □ ECG: RBBB with RAD • Ostium primum □ present earlier than ostium secundum defects □ associated with abnormal AV valves □ the AV node is displaced posteriorly and inferiorly and atrial and/or AV nodal conduction is often delayed. □ ECG: RBBB with LAD, prolonged PR interval wide, fixed, split-second sound + right-axis deviation □ Ostium secundum wide, fixed, split-second sound + left-axis deviation □ Ostium primum

Features • Symptoms □ asymptomatic in youth □ often discovered on routine school health exams □ mild fatigue □ frequent respiratory infections □ Larger ones may lead to signs of right ventricular failure, such as shortness of breath and a parasternal heave. • Physical exam □ Mid-systolic ejection murmur (over the left second ICS) □ Due to □ Relative pulmonary stenosis due to an increase in stroke volume □ Soft mid-diastolic murmur (over the lower left sternal border) □ arises from increased flow across the tricuspid valve. □ loud S1 □ wide fixed-split S2 □ The most frequently tested knowledge □ splitting is fixed (does not vary with respiration) □ heaving cardiac impulse (LLSB) • Other features □ The grossly elevated DLco is secondary to the left-right shunt and increased pulmonary blood flow. In contrast, chronic pulmonary emboli will cause a low DLco. Predisposes patient to □ CHF □ 2nd/3rd decades of life □ Eisenmenger's syndrome □ pulmonary hypertension □ right ventricular hypertrophy □ reversal to a right-to-left shunt □ stroke □ due to paroxysmal embolus

Associated condition

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

• Tricuspid atresia is the congenital cardiac disorder most commonly associated with an atrial septal defect. • Down syndrome • Fetal alcohol syndrome • Holt-Oram syndrome □ Autosomal

dominant disorder, which is also called hand-heart syndrome because affected children present with an ASD, a first degree heart block, and abnormalities of the upper limbs (e.g., absent radial bones). It affects approx. 1 in 100,000 children. ECG • Right bundle branch block • ostium primum ASD □ left axis deviation. • ostium secundum ASD □ right axis deviation. • first degree heart block □ prolongation of the PR interval □ due to delayed conduction through the atria or through the AV node • The QRS pattern typically is either an rSr' or an rsR' resulting from dilation and hypertrophy of the right ventricular outflow tract caused by volume overload of the right heart. prominent left precordium in a young patient with an ejection murmur in the second left intercostal space indicates □ ASD with pulmonary hypertension □ A prominent left precordium suggests that: □ the right ventricle was dilated during childhood □ RV working against a high pressure

Patent ductus arteriosus Overview • acyanotic congenital heart defect • connection between the pulmonary trunk and descending aorta • more common in premature babies, born at high altitude or maternal rubella infection in the first trimester Features • left subclavicular thrill • continuous 'machinery' murmur at the left upper sternal edge with late systolic accentuation • large volume, bounding, collapsing pulse • wide pulse pressure • heaving apex beat Management • indomethacin closes the connection in the majority of cases • if associated with another congenital heart defect amenable to surgery then prostaglandin E1 is useful to keep the duct open until after surgical repair

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Patent foramen ovale (PFO) • PFO is present in around 20% of the population. • It may allow embolus (e.g. from DVT) to pass from right side of the heart to the left side leading to a stroke - 'a paradoxical embolus' • There also appears to be an association between migraine and PFO. □ Some studies have reported improvement in migraine symptoms following closure of the PFO • right heart catheter: left to right shunting of oxygenated blood at level of the atrium. □ oxygen saturation data show a step-up in the saturations between the vena cava and the right atrium.

Paradoxical embolisation • For a right-sided thrombus (e.g. DVT) to cause a left-sided embolism (e.g. stroke) it must obviously pass from the right-to-left side of the heart. • The following cardiac lesions may cause such events □ patent foramen ovale - present in around 20% of the population □ atrial septal defect - a much less common cause Blue toe syndrome • 80% of digital ischaemias have an emboli originating from the heart and so an urgent echocardiogram is crucial to prevent further and more severe events occurring. • sudden onset of a cold, painful, and cyanotic big toe. the next steps □ Therapeutic heparin and urgent echocardiogram

Eisenmenger's syndrome Definition • Eisenmenger's syndrome describes the reversal of a left-to-right shunt in a congenital heart defect due to pulmonary hypertension. • This occurs when an uncorrected left-to-right leads to remodeling of the pulmonary microvasculature, eventually

causing obstruction to pulmonary blood and pulmonary hypertension. Associated with • ventricular septal defect □ Although patients with tetralogy of Fallot have, by definition, a ventricular septal defect they do not go on to develop Eisenmenger's syndrome • atrial septal defect • patent ductus arteriosus Features • original murmur may disappear • cyanosis • clubbing • right ventricular failure • polycythemia • haemoptysis, embolism Management • heart-lung transplantation is required Eisenmenger's syndrome - the reversal of a left-to-right shunt

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Ebstein's anomaly Definition • Ebstein's anomaly is a congenital heart defect characterised by low insertion of the tricuspid valve resulting in a large atrium and small ventricle. It is sometimes referred to as 'atrialisation' of the right ventricle. Causes • Ebstein's anomaly may be caused by exposure to lithium in-utero Features • hypoplastic (atrialised) RV, • apical displacement of the septal and posterior tricuspid valve leaflets, • ASD, • Right bundle branch block pattern on ECG. Associations • tricuspid incompetence (pan-systolic murmur, giant V waves in JVP) • Wolff-Parkinson White syndrome occurs in around 15% of the patients. The presence of delta waves and short PR interval is indicative of WPW. When correlated with past surgical history (repair of atrial septal defect and tricuspid valve abnormalities as a child), Ebstein's anomaly is the most likely diagnosis.

Cardiac manifestations of genetic disorders

Peripheral vascular disease • is a marker for increased risk of cardiovascular events even when it is asymptomatic. • the femoropopliteal artery, the most common site of peripheral arterial disease. □ paresthesia, intermittent claudication in calf and foot and palpable femoral pulses but absent pedal pulses Risk factors • age □ about 20% of people aged over 60 years have some degree of peripheral arterial disease. • male gender • Smoking • Diabetes • hypertension • coronary artery disease. Feature • intermittent claudication (leg pain while walking) (The most common initial symptom). • Critical limb ischaemia : ischaemic pain, ulceration, tissue loss and/or gangrene.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

Investigations • measuring the ankle brachial pressure index □ Calculate the index in each leg by dividing the highest ankle pressure by the highest arm pressure. • Imaging before considering revascularization □ duplex ultrasound (first-line imaging) □ contrast-enhanced magnetic resonance angiography (after duplex ultrasound) □ computed tomography angiography (if contrast-enhanced magnetic resonance angiography is contraindicated or not tolerated.) Treatment Mild symptoms: • exercise programme □ 2 hours of supervised exercise a week for a 3-month period □ encouraging people to exercise to the point of maximal pain. • management of cardiovascular risk factors (for example, with aspirin or statins) • vasoactive drug treatment (for

example, with naftidrofuryl oxalate). • Which drug might help improve pain-free walking distance? □ Naftidrofuryl □ Indicated only when exercise has not led to satisfactory improvement and the person refuse angioplasty or bypass surgery. □ discontinue naftidrofuryl oxalate if there has been no symptomatic benefit after 3–6 months. • Vasoactive drugs have limited benefit in treating intermittent claudication. • There is modest evidence for the use of drugs such as naftidrofuryl and pentoxifylline, but little benefit from cinnarizine or inositol nicotinate. • Simvastatin may be prescribed for patients with peripheral vascular disease who have elevated cholesterol levels, but there is no data on improvements in walking distance. severe symptoms: • endovascular treatment (such as angioplasty or stenting), bypass surgery, pain management and/or amputation.

Differential diagnosis of foot ulcers Venous ulcers Arterial ulcer Diabetic ulcer Neuropathic ulcer Location □ Gaiter region (above the ankle) □ Plantar pressure points of the foot (over the head of the metatarsal bones or the heel) Mechanism □ Chronic local venous hypertension → tissue ischemia Wound features □ Irregular borders □ Exudative □ Superficial □ Hyperkeratotic borders □ Deep Pain □ Mild □ Severe □ Absent Additional features □ Varicose veins □ Oedema □ Stasis dermatitis • Treatment of venous ulceration: □ control of oedema, treating any infection, and compression. □ Compressive dressings or devices should not be applied if the arterial circulation is impaired, and ankle-brachial pressure index is needed before application of compression

Rheumatic fever Definition • an autoimmune process following infection with group A streptococci. Overview • Type II hypersensitivity is seen in rheumatic fever. • Myocarditis is the most common cause of death during the acute phase of rheumatic fever. Diagnosis: based on: • Evidence of recent streptococcal infection accompanied by: □ 2 major criteria □ 1 major with 2 minor criteria • Evidence of recent streptococcal infection □ ASOT > 200iu/mL □ history of scarlet fever □ positive throat swab □ increase in DNase B titre Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

□ Pressure points of the foot and shin (e.g., lateral malleolus, tips of the toes) □ Vessel occlusion → tissue ischemia □ Diabetic microvasculopathy and neuropathy → impaired tissue sustenance □ Punched-out appearance □ No exudation □ Pale, shiny, cold, hairless surrounding skin □ Nail dystrophy □ Absent pulses □ Charcot joints □ Absent ankle reflex □ Impaired sensation (esp. vibration) □ Claw toes

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology Jones Criteria • Major criteria

1. erythema marginatum
2. Sydenham's chorea
3. polyarthrititis
4. carditis (endo-, myo- or peri-)
5. subcutaneous nodules □ Pea-sized, firm and non-tender. □ characteristically seen on the extensor surfaces of joints such as knees and elbows and also over the spine. • Minor criteria

6. raised ESR or CRP
7. pyrexia
8. arthralgia (not if arthritis a major criteria)
9. prolonged PR interval Histology • Aschoff bodies are foci of chronic inflammation seen histologically in the myocarditis of acute rheumatic fever. □ Anitschkow cells are reactive histiocytes with wavy, slender, caterpillar-like nuclei seen in Aschoff bodies of acute rheumatic fever. Erythema marginatum is seen in around 10% of children with rheumatic fever. It is rare in adults Rheumatic fever major criteria: J♥NES • Joints - polyarthritis; • ♥-carditis; • Nodules (subcutaneous); • Erythema marginatum; • Sydenham's chorea.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Infective endocarditis (IE) The most common cause of endocarditis: • Staphylococcus aureus is now the most common cause of infective endocarditis • Staphylococcus epidermidis if < 2 months post valve surgery. Definition • an infection of the endocardium, the inner layer of the heart and valves. Pathophysiology • Damaged valvular endothelium → adherence of platelets and fibrin → sterile vegetation (microthrombus) → bacteremia → bacterial colonization of vegetation → valve destruction with loss of function Risk factors • previous episode of endocarditis: The strongest risk factor for developing infective endocarditis. • previously normal valves (50%, typically acute presentation) • rheumatic valve disease (30%) • prosthetic valves • congenital heart defects • intravenous drug users (IVDUs, e.g. Typically causing tricuspid lesion) • hemodialysis • Hypertrophic cardiomyopathy.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology Types Acute Endocarditis Subacute Endocarditis Larger vegetations Smaller vegetations Attacks previously normal valves Attacks damaged or abnormal valves Destructive; 50% mortality rate despite treatment Less destructive; most patients recover with treatment High-virulence organisms, especially S aureus Low-virulence organisms, especially the viridans streptococci S mutans and S sanguinis The likelihood of infection • The higher the valvular pressure, the greater the likelihood of infection. Thus, mitral > aortic > tricuspid > pulmonary. The exception to this rule is IV-related infective endocarditis; in this case, the tricuspid valve is the most commonly involved because it is the first valve encountered after venous injection. • If the valve is already abnormal, then the likelihood of infection is greater and will be most likely on the aortic valve (High-pressure systems create more blood turbulence and permit inoculation of the valve). • Diseases that affect the mitral valve, such as mitral valve prolapse and mitral regurgitation, are the most common valvular diseases. So the mitral valve is the valve most frequently affected by endocarditis. The exception is IV drug use. In these patients, the tricuspid valve is the most frequently involved valve Causes Streptococcus bovis endocarditis is associated with colorectal cancer • Staphylococcus aureus (coagulase positive) : the most common causative organism of IE (especially acute presentation, IVDUs). □ Staphylococcus aureus endocarditis is an aggressive disease frequently associated with valve destruction and abscess formation. • Staphylococcus epidermidis (coagulase negative) most commonly associated with prosthetic valves < 2 months post operative. •

Streptococcus viridans: commonly causing subacute bacterial endocarditis. The two most notable viridans streptococci are Streptococcus mitis and Streptococcus sanguinis. They are both commonly found in the mouth and in particular dental plaque so endocarditis caused by these organisms is linked with poor dental hygiene or following a dental procedure • Streptococcus gallolyticus (formerly Streptococcus bovis) is associated with colorectal cancer □ colonoscopy should be done. • Bacteroides is the most likely organism following bowel resection, though S. bovis is also seen. Management is metronidazole. • Candida endocarditis: Risk factors: Intravenous drug abuse, immunodeficiency states and indwelling catheters. The aortic valve is the most common valve to be involved. Treatment with Valve replacement followed by amphotericin B for 6 weeks. • Non-infective (sterile vegetations) □ systemic lupus erythematosus (Libman-Sacks), commonly result in mitral regurgitation. □ malignancy: marantic endocarditis • Culture negative causes □ prior antibiotic therapy

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

□ Coxiella burnetii (Q fever agent), typically associated with exposure to animals (sheep and cattle). □ Bartonella (from cats) □ Brucella □ Chlamydia psittaci (from birds). □ HACEK: (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella) □ slow-growing, Gram negative bacteria □ These are normal flora of the upper respiratory tract □ constitute 5-10% cases of endocarditis; □ they require prolonged incubation in enriched media and increased carbon dioxide tension. □ The human bite injury and gram-negative culture make Eikenella corrodens the most likely causative organism. □ third-generation cephalosporin (Ceftriaxone) is effective against enteric gram-negative rods, including HACEK organisms Associations Most common cause Generally Staphylococcus aureus prosthetic valves < 2 months post operative Staphylococcus epidermidis IV drug use Staphylococcus aureus Recent dental procedure Streptococcus viridans : (Streptococcus mitis and Streptococcus sanguinis). Colorectal cancer Streptococcus gallolyticus (formerly Streptococcus bovis) Diagnosis Infective endocarditis: Modified Duke criteria • Infective endocarditis diagnosed if □ pathological criteria positive, or □ 2 major criteria, or □ 1 major and 3 minor criteria, or □ 5 minor criteria • Pathological criteria □ Positive histology or microbiology of pathological material obtained at autopsy or cardiac surgery (valve tissue, vegetations, embolic fragments or intracardiac abscess content) • Major criteria □ Positive blood cultures □ two positive blood cultures showing typical organisms consistent with infective endocarditis, such as Streptococcus viridans and the HACEK group, or □ persistent bacteraemia from two blood cultures taken > 12 hours apart or three or more positive blood cultures where the pathogen is less specific such as Staph aureus and Staph epidermidis, or □ positive serology for Coxiella burnetii, Bartonella species or Chlamydia psittaci, or □ positive molecular assays for specific gene targets □ Evidence of endocardial involvement □ positive echocardiogram (oscillating structures, abscess formation, new valvular regurgitation or dehiscence of prosthetic valves).

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology • Minor criteria

1. predisposing heart condition or intravenous drug use
 2. microbiological evidence does not meet major criteria
 3. fever > 38 C
 4. vascular phenomena: major emboli, splenomegaly, clubbing, splinter haemorrhages, Janeway lesions, petechiae or purpura
 5. immunological phenomena: glomerulonephritis, Osler's nodes, Roth spots
- Classical symptoms of infective endocarditis can be remembered using the mnemonic FROM JANE: fever, Roth spots, Osler nodes, murmur, Janeway lesions, anaemia, nail-bed haemorrhage, emboli.
 Note for Osler nodes: Osler nodes and Janeway lesions are similar in appearance, yet Osler nodes are painful and Janeway lesions are painless. Peripheral signs associated with infective endocarditis

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Investigation • Transthoracic echocardiography (TTE) is the initial test of choice for all patients with suspected IE. • How should the blood samples be drawn to maximise the chances of obtaining positive cultures? □ Draw three samples of blood from different venepuncture sites with the first separated from the last by at least one hour over 24 hours Aortic valve endocarditis can cause aortic root abscess which can cause damage to the AV node resulting in prolongation of the PR interval on ECG. Management Current antibiotic guidelines (source: British National Formulary) Scenario Suggested antibiotic therapy Initial blind therapy • Native valve: □ Not allergic to penicillin , no MRSA , not severe sepsis: □ Amoxicillin + gentamicin □ Allergic to penicillin , MRSA , or severe sepsis: □ vancomycin + gentamicin • prosthetic valve: □ vancomycin + rifampicin + gentamicin Staphylococci endocarditis • Native valve: □ Not allergic to penicillin , no MRSA , not severe sepsis: □ Flucloxacillin □ Allergic to penicillin , MRSA , or severe sepsis: □ vancomycin + rifampicin • Prosthetic valve: □ Not allergic to penicillin , no MRSA , not severe sepsis: □ Flucloxacillin + rifampicin + gentamicin □ Allergic to penicillin , MRSA , or severe sepsis: □ vancomycin + rifampicin + gentamicin Streptococci endocarditis • Native valve and Prosthetic valve : □ not allergic to penicillin □ Benzylpenicillin ± gentamicin □ Allergic to penicillin: □ vancomycin + gentamicin IV amoxicillin is the empirical treatment of choice in native valve endocarditis The most useful laboratory test used to monitor the treatment of infective endocarditis is serial C reactive protein estimation.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology • length of treatment: □ 6 weeks of intravenous therapy is generally accepted as the length of treatment needed. Indications for surgery • organisms that are difficult to eradicate by medical therapy as such fungi, brucella, coxiella, pseudomonas aeruginosa, vancomycin-resistant enterococci • persistent bacteraemia despite appropriate antibiotic therapy • extension of infection to a extravalvular site • early prosthetic valve endocarditis (within 2 months) • dehiscence or obstruction of a prosthetic valve. • large (more than 10 mm) vegetations Prophylaxis • NICE recommends the following procedures do not require prophylaxis: □ dental procedures □ upper and lower gastrointestinal tract procedures □ genitourinary tract; this includes urological, gynaecological and obstetric procedures and childbirth □ upper and lower respiratory tract; this

includes ear, nose and throat procedures and bronchoscopy • Prophylaxis is only recommended in those patients who are at highest risk of adverse outcomes on the development of endocarditis. These patient groups include: □ Prosthetic cardiac valve or prosthetic material used for cardiac valve repair □ Previous endocarditis □ Unrepaired cyanotic congenital heart disease including palliative shunts and conduits □ Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first six months after the procedure □ Repaired congenital heart disease with residual defects (persisting leaks or abnormal flow) at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialisation) □ Cardiac transplantation recipients who develop cardiac valve abnormalities. Prognosis

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Poor prognostic factors • Staph aureus infection (□ Acute endocarditis) □ (Streptococcus viridans □ Subacute bacterial endocarditis has a better prognosis.) • Prosthetic valve (especially 'early', acquired during surgery) • Culture negative endocarditis • Low complement levels • Infection of the aortic rather than mitral valve • Associated rhythm disturbance. • Heart failure: the most common cause of death from infective endocarditis • Intravenous drug abuse (often left and right sided disease) • Old age • Insulin dependent diabetes mellitus • Severe co-morbidities. Mortality according to organism • Staphylococci - 30% • Bowel organisms - 15% • Streptococci - 5%

Myocarditis The short prodromal illness coupled with the development of biventricular heart failure, tachycardia, T-wave inversion and elevated troponin is most consistent with viral myocarditis. The features, including the mild flu-like illness, are consistent with Coxsackie B. Pathology •

Lymphocytic infiltrate with focal necrosis of myocardial tissue Causes • In 50% of cases, no cause can be identified; hence, myocarditis is commonly idiopathic. • In patients with an identified cause: □ the most commonly implicated etiology is viral (similar to pericarditis), of which enteroviruses, notably Coxsackie B, are the most common. • Viral: □ The most common in adults: □ Parvovirus B19 □ Human herpes virus 6 □ Other Viral Causes □ Coxsackie B virus □ most common in children □ results in dilated cardiomyopathy. □ Adenovirus, HIV, Hepatitis C, Influenza virus, Epstein-Barr virus • Bacteria: diphtheria, clostridia • Spirochaetes: Lyme disease (most commonly presents as heart block). • protozoa □ Chagas' disease, □ caused by Trypanosoma cruzi, a common pathogen in South America □ Chagas disease myocarditis results in dilated cardiomyopathy. □ Toxoplasmosis • Noninfectious

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology

□ Autoimmune (e.g., systemic lupus erythematosus, sarcoidosis, dermatomyositis, polymyositis), Vasculitis (e.g., Kawasaki disease) □ Toxins (e.g., carbon monoxide poisoning, black widow venom), Cocaine. □ Medication (e.g., sulfonamides), chemotherapy (e.g., anthracycline, doxorubicin) □ Radiation therapy Presentation • usually young patient with a history of viral prodrome 2 to 3 weeks prior to the onset (fever, arthralgia, myalgia, upper respiratory tract infections) • typically

present with symptoms of heart failure (dyspnea, orthopnea, and leg swelling). • chest pain, due to involvement of the pericardium. • Palpitations, typically sinus tachycardia. Investigations • Markedly raised troponin. • ↑ ESR (and CRP) • ECG: □ sinus tachycardia or ventricular arrhythmias □ nonspecific ST changes □ diffuse ST elevation in those with pericardial involvement (perimyocarditis). • Echocardiography: global systolic dysfunction Differential diagnosis • Acute coronary syndrome □ differentiating factors: ECG changes (NSTEMI and STEMI) with increased troponins Treatment • Supportive, usually similar to heart failure. • NSAIDs should be avoided in the acute phase of acute myocarditis as it may impair healing.

DVLA: cardiovascular disorders Group 1 car and motorcycle Group 2 bus and lorry Angina □ Must not drive when symptoms occur at rest, with emotion or at the wheel. □ Need not notify the DVLA. Acute coronary syndromes (ACS) □ After successful coronary angioplasty: can drive after 1 week. □ If no successful coronary angioplasty, drive after 4 weeks □ Need not notify the DVLA Coronary artery bypass graft (CABG) □ Can drive after 4 weeks □ Need not notify the DVLA Arrhythmia □ Can drive if arrhythmia is controlled for at least 4 weeks. □ may need to notify the DVLA. Successful catheter ablation □ May drive after 2 days □ Need not notify the DVLA Pacemaker implant □ Can drive after 1 week □ Need not notify the DVLA CRT pacemaker □ Can drive after 4 weeks □ Must notify the DVLA □ Can drive after 6 weeks □ Must notify the DVLA Implantable cardioverter defibrillator (ICD) □ Can drive 6 months □ May need to notify the DVLA. Hypertension □ May drive and need not notify the DVLA □ Must not drive and must notify the DVLA if resting BP is consistently: 180 mm Hg or higher systolic and/or 100 mm Hg or more diastolic. Heart failure □ Asymptomatic: May drive and need not notify the DVLA. □ Symptomatic: Must not drive but need not notify the DVLA. □ Left ventricular assist device implanted: Can drive after 3 months. Need not notify the DVLA. Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

□ Must not drive and must notify the DVLA when symptoms occur. □ Driving may be relicensed if no angina for at least 6 weeks. □ Can drive after 6 weeks □ Must notify the DVLA □ Can drive after 3 months □ Must notify the DVLA □ Can drive if arrhythmia is controlled for at least 3 months □ Must notify the DVLA □ For arrhythmia causing incapacity: can drive after 6 weeks. □ For arrhythmia NOT causing incapacity: can drive after 2 weeks. □ Must notify the DVLA □ Can drive after 6 weeks □ Must notify the DVLA □ Permanent bar □ Must notify the DVLA □ Asymptomatic: May drive and need not notify the DVLA. □ Symptomatic: Must not drive and must notify the DVLA. Relicensing would require LV ejection fraction at least 40% □ Left ventricular assist device implanted: Licence will be refused permanently. Must notify the DVLA.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 5

Cardiology ICD means: • cannot drive a group 1 vehicle for 6 months • Loss of a group 2 HGV license, regardless of the circumstances DVLA advice post MI: • if successfully treated by angioplasty □ cannot drive for 1 week • If does not undergo angioplasty □ cannot drive for 4 weeks DVLA: cardiovascular disorders Acute coronary syndrome • if successfully treated by angioplasty □ cannot drive for 1 week • If does not undergo angioplasty □ cannot drive for 4 weeks Coronary artery bypass graft (CABG) • Group 1 car: 4 weeks off driving • Group 2 bus and lorry: Must not

drive and must notify the DVLA. pacemaker insertion: 1 week off driving implantable cardioverter-defibrillator (ICD):

- if implanted for sustained ventricular arrhythmia: cease driving for 6 months.
- If implanted prophylactically then cease driving for 1 month.
- for Group 2 drivers $\square$ permanent bar

Heart failure : LVEF of $< 40\%$ bars him from driving a lorry, even if he becomes asymptomatic with treatment successful catheter ablation for an arrhythmia: 2 days off driving

Dextrocardia Definition • The heart is located on the right side of the chest. Epidemiology • No ethnic or gender-related predilection Associations • Situs inversus totalis (reversal in the position of other organs) • Kartagener syndrome: classic triad of situs inversus (reversal in the position of the abdominal organs), recurrent sinusitis, and bronchiectasis • When dextrocardia is associated with a normal position of other thoracoabdominal structures, it is called situs solitus. ECG Features • Right axis deviation • Negative P wave and QRS complex in lead I. • Upright p wave in AVL • Reverse R wave progression across the precordium; the R wave is tallest in V1 and progressively decreases in amplitude in leads V2 to V6. The diagnosis may be confirmed by obtaining right-sided chest leads that demonstrate the normal progression of R wave amplitude.

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad

Characteristic changes of dextrocardia include a negative P wave and QRS complex in lead I, since atrial and ventricular depolarization begin on the left and spread to the right. There is also reverse R wave progression across the precordium; the R wave is tallest in V1, and progressively decreases in amplitude in leads V2 to V6. Differential diagnosis • Cardiac dextroposition $\square$ Dextrocardia also involves a change in the orientation of the heart with its base to the apex axis being directed to the right, in contrast to the normal heart orientation where the apex is directed to the left. This change in orientation differentiates it from cardiac dextroposition, where the heart is displaced to the right side as a result of extracardiac causes, such as a diaphragmatic hernia, right pneumonectomy, or right lung hypoplasia. Prognosis • Isolated dextrocardia is a benign condition often diagnosed incidentally. • Typically, patients have a normal life expectancy if no cardiac anomalies are present.

Third edition Notes & Notes For MRCP part 1 & 11 By Dr. Yousif Abdallah Hamad Nephrology Updated

Notes & Notes for MRCP

By Dr. Yousif Abdallah Hamad Chapter 6

Nephrology

Renal anatomy The tables below show the anatomical relations of the kidneys: Right kidney Direct contact Layer of peritoneum in-between Right suprarenal gland Duodenum Colon Liver Distal part of small intestine Left kidney Direct contact Layer of peritoneum in-between Left suprarenal gland Pancreas Colon Stomach Spleen Distal part of small intestine Renal physiology Diagram showing the basic physiologic mechanisms of the kidney Renal blood flow (RBF) • Renal blood flow is 20-25% of cardiac output • The 'Fick principle' can be used to estimate RBF through clearance. • Sympathetic stimuli produce vasoconstriction and RBF should be increased in response to hypoxia.

- Renal cortical blood flow > medullary blood flow (i.e. tubular cells more prone to ischaemia)
- Glomerular filtration rate and renal blood flow increase by about 50% in pregnancy leading to decreased BUN and creatinine on laboratory examination.
- What is the effect of decrease in hematocrit on renal function?
- Decreased Renal Blood Flow □ the relationships between Renal Blood Flow (RBF), Renal Plasma Flow (RPF), Hematocrit (Hct), and Glomerular Filtration Rate (GFR):

Notes & Notes for MRCP

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□ $RBF = RPF / (1 - Hct)$. □ Assuming that the GFR is stable, this equation suggests that a decrease in Hct would lead to an decrease in RBF. Renal tubular functions

- Sodium, glucose, bicarbonate and amino acids are absorbed at the proximal tubule level
- Sodium reabsorption is mostly through active transport in the loop of Henle with only a modest reabsorption facilitated by aldosterone.
- Ammonia is secreted by the distal tubule
- Regulation of water secretion is by the distal tubule and the collecting ducts under the influence of vasopressin □ increase permeability to water.
- The relative hyperosmolality of the medulla is maintained by a counter-current mechanism and is responsible for the flux of water across the renal tubule
- descending loop of Henle is permeable to water but impermeable to solutes, due to the presence of aquaporin 1 in its tubular wall □ water moves to medullary space □ hypertonic filtrate
- ascending loop of Henle is impermeable to water (because of a lack of aquaporin, a common transporter protein for water channels in all cells except the walls of the ascending loop of Henle) but permeable to solutes, but here Na^+ , Cl^- , and K^+ are actively transported into the medullary space, making the filtrate hypotonic
- What is the renal cellular mechanism that prevents a sodium load intake from drastically increasing plasma osmolality?
- Movement of aquaporin channels to the apical surface of collecting duct cells □ An increase in sodium intake will cause an increase in plasma osmolality, triggering the release of antidiuretic hormone (ADH), a.k.a. vasopressin. □ The immediate effect of ADH (occurs over minutes) □ movement of aquaporin channels to the apical surface of collecting duct cells. □ the long-term effect of ADH (occurs over days) □ Increase in aquaporin gene expression by collecting duct cells.

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Nephrology Renal Physiology of Pregnancy

- Kidneys size increase by 1 to 1.5 cm during pregnancy.
- Kidney volume increases by up to 30%, primarily due to an increase in renal vascular and interstitial volume.
- Glomerular filtration rate (GFR) and renal blood flow rise markedly during pregnancy, resulting in a physiologic fall in the serum creatinine concentration. A serum creatinine of 1.0 mg/dL in a pregnant woman probably reflects significant renal insufficiency. □ The glomerular filtration rate increases 50% with subsequent decrease in serum creatinine, urea, and uric acid values.
- mechanisms contribute to decreased vascular resistance, increased renal plasma flow, and increased GFR during pregnancy: □ Reduced vascular responsiveness to vasopressors such as angiotensin 2, norepinephrine, and antidiuretic hormone. □ Additionally, the ovarian hormone and vasodilator relaxin is a key mediator of enhanced nitric oxide signaling in pregnancy.
- The best method to estimate GFR in pregnancy is by 24-hour urine collection for creatinine clearance. □ Completeness of the collection should be confirmed by checking the 24-hour creatinine excretion (10 to 15 mg creatinine/day per kg body weight is consistent with a complete collection). □ Estimating equations, such as the Modification of Diet in Renal Disease

Study (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equations, are not accurate in pregnancy. □ Physiologic ureteral dilatation (hydronephrosis and hydroureter) is common during pregnancy, and results from:

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