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Chapter 9

Notes & Notes for MRCP
By Dr. Yousif Abdallah Hamad Chapter 9

Infectious diseases

• haemolytic anaemia secondary to cold agglutins (IgM) • a maculopapular, pruritic rash develops in around 99% of patients who take ampicillin/amoxicillin whilst they have infectious mononucleosis Drug-induced rash is usually pruritic and is prolonged, in contrast to the viral rash of EBV infectious mononucleosis. Early infectious mononucleosis may present with a maculopapular generalized rash.It is nonpruritic and rapidly disappears. • Because leukocytosis is the rule in infectious mononucleosis, the presence of a normal or decreased WBC count should suggest an alternative diagnosis. • Lymphocytosis Relative lymphocytosis (≥ 60%) plus atypical lymphocytosis (≥ 10%) are the characteristic findings of EBV infectious mononucleosis. • presence of 50% lymphocytes with at least 10% atypical lymphocytes Atypical lymphocytes most commonly seen in patients who have infectious mononucleosis. Other causes drug reactions (phenytoin), stress, viral or bacterial infections, allergies, autoimmune diseases, thyroid problems malignancy. • ESR is most useful in differentiating group A streptococcal pharyngitis from EBV infectious mononucleosis. (ESR elevated with EBV infectious mononucleosis, not elevated in group A streptococcal pharyngitis). atypical lymphocytosis point towards a viral illness Diagnosis • heterophile antibody test (Monospot test) (immunoglobulin IgM to EBV) the initial screening test sensitivity 85% and specificity 100%. Cytomegalovirus is a herpesvirus that causes infectious mononucleosis with a negative monospot test. • EBV serological tests Definitive diagnosis should be obtained in patients with a mononucleosis-like illness and a negative finding on the Monospot test. Management is supportive and includes: • rest during the early stages, drink plenty of fluid, avoid alcohol • simple analgesia for any aches or pains • consensus guidance in the UK is to avoid playing contact sports for 8 weeks after having glandular fever to reduce the risk of splenic rupture • unfortunately on clinical appearances it is not possible to distinguish bacterial from viral or throat infections with any degree of reliability.

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• If the child has EBV infection, then the administration of Amoxicillin will give an erythematous rash. Non-vomiting patients can be treated with oral penicillin-v.' • Patients with EBV infectious mononucleosis who have positive throat cultures for group A streptococci should not be treated

because this represents colonization rather than infection • complicated EBV infectious mononucleosis : Short courses of corticosteroids are indicated for EBV infectious mononucleosis with: hemolytic anemia, thrombocytopenia, CNS involvement, or extreme tonsillar enlargement (impending airway obstruction). In which structure is the immune response most likely localized? Paracortex • immune response to the virus takes place through T-cell mediated immune responses, which take place in the lymphocyte-rich areas of the lymph node, namely the paracortex. • A biopsy of the lymph node of this patient would show reactive hyperplasia due to increased activity of the paracortex.

Parvovirus B19 Pathogen: Parvovirus B19 is a single-strand DNA virus. Transmission: particularly via airborne infection Pathology • Primarily infects progenitor cells of erythrocytes in bone marrow and endothelial cells • Attaches to P antigen on RBCs and endothelial cells → cell destruction Diseases • erythema infectiosum The most widely known clinical manifestation of parvovirus B19 is erythema infectiosum ('slapped cheek syndrome'), a mild viral illness of childhood characterised by a classic exanthema in which both cheeks appear bright red as though they had been slapped. • Aplastic crisis in patients with hemolytic anemias (e.g. sickle cell disease, thalassemias) • Parvovirus B19-associated arthritis most commonly in adults, particularly in women affect the small joints of the hands and feet. Knees or elbows are rarely involved. may mimic rheumatoid arthritis. Unlike rheumatoid arthritis, the post-infectious arthritis associated with parvovirus B19 does not cause permanent damage to bones or joints.

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• Pure red blood cell aplasia • The virus has a tropism for rapidly dividing erythrocyte precursors which they infect and destroy. Thus, no reticulocytes (immature erythrocytes) are available to replace aging or damaged erythrocytes as they are cleared by the reticuloendothelial system. This may not have any significant impact on otherwise healthy individuals, but can trigger an aplastic crisis - particularly in patients with haemoglobinopathies.

Leishmaniasis • Leishmaniasis is caused by the intracellular protozoa Leishmania, (intramacrophage protozoa) • transmitted to humans by phlebotomine sand flies. • There are four main clinical syndromes: cutaneous, muco-cutaneous, visceral (also known as kala-azar) and post kala-azar dermal leishmaniasis. Cutaneous leishmaniasis • caused by Leishmania tropica or Leishmania mexicana • crusted lesion at site of bite • present with ulcers or nodules. • usually heal spontaneously, but slowly, in immuncompetent individuals with resultant disfiguring scars. Mucocutaneous leishmaniasis • caused by Leishmania braziliensis • skin lesions may spread to involve mucosae of nose, pharynx etc • characterised by progressively destructive ulcerations of the mucosa extending from the nose and mouth to the pharynx and larynx, • are not self-healing. Visceral leishmaniasis (kala-azar) • mostly caused by Leishmania donovani • caused by the Leishmania donovani complex (L. donovani sensu stricto in East Africa and India, L. infantum in Europe, North Africa and Latin America). • incubation period of 2-6 months • patients present with persistent systemic infection (fever, sweating, rigor, malaise, loss of appetite and weight loss)

(*occasionally patients may report increased appetite with paradoxical weight loss) • parasitic infection of the blood and reticulo-endothelial system lymphadenopathy, massive splenomegaly and hepatomegaly • grey skin - 'kala-azar' means black sickness • investigations pancytopaenia secondary to hypersplenism There is also often marked polyclonal hypergammaglobulinaemia. Visualisation of the parasite (amastigote form) from lymph nodes, bone marrow or spleen is used as a confirmatory test. PCR can be used to detect the parasite in the blood.

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 Anti-leishmanial antibodies can be detected, but they remain positive up to several years after cure and therefore cannot be used to detect relapse. • Treatment First line antimonials are sodium stibogluconate and meglumine antimoniate. Adverse effects influde cardiac arrhythmias and acute pancreatitis. Amphotericin B is increasingly being used. Post kala-azar dermal leishmaniasis • a complication of visceral leishmaniasis • characterised by a macular, maculo-papular or nodular rash
• frequently observed after treatment.It can also occur in immunosuppressed individuals. • highly infectious.

Leptospirosis (Also known as Weil's disease*) • *the term Weil's disease is sometimes reserved for the most severe form If the infection causes jaundice, kidney failure and bleeding, it is then known as Weil's disease. If it affects the lung and causes pulmonary haemorrhage, then it is known as severe pulmonary haemorrhage syndrome. • leptospirosis is commonly seen in questions referring to sewage workers, farmers, vets or people who work in abattoir. • It is caused by the spirochaete Leptospira interrogans (serogroup L icterohaemorrhagiae), • classically being spread by contact with infected rat urine. • Weil's disease should always be considered in high-risk patients with hepato-renal failure Features • fever • flu-like symptoms • renal failure (seen in 50% of patients) • jaundice • headache, may herald the onset of meningitis • subconjunctival haemorrhage • Haemorrhagic tendencies with purpura or petechiae • Enlargement of liver and spleen. • Presentation with heart failure is uncommon but has been described in severe leptospirosis. Management • high-dose benzylpenicillin or doxycycline • other options: cefotaxime or ceftriaxone.

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Lyme disease Aetiology • Lyme disease is caused by the spirochaete Borrelia burgdorferi and is spread by ticks of the genus Ixodes Ixodes ricinus is predominantly responsible for its transmission in Europe. Ixodes pacificus and Ixodes scapularis are the ticks responsible for transmission of in the USA. Features Early features • erythema chronicum migrans (small papule often at site of the tick bite which develops into a larger annular lesion with central clearing, 'bulls-eye'. Occurs in 70% of patients) Erythema migrans is often the presenting sign of Lyme disease • systemic symptoms: malaise, fever, arthralgia Later features • CVS: heart block, myocarditis •

neurological: (Neuroborreliosis): cranial nerve palsies, meningitis • polyarthritis Investigation • serology: antibodies to Borrelia burgdorferi (ELISA test for antibodies to Borrelia burgdorferi) Serological tests are the most appropriate first line investigation for diagnosing Lyme disease. ELISA tests are preferred to Western blots as they are more sensitive. Management • Early disease: doxycycline is the drug of choice for 2 – 3 weeks Amoxicillin is an alternative if doxycycline is contraindicated (e.g. pregnancy) • Disseminated disease: ceftriaxone if disseminated disease • Jarisch-Herxheimer reaction is sometimes seen after initiating therapy: fever, rash, tachycardia after first dose of antibiotic (more commonly seen in syphilis, another spirochaetal disease) MRCPUK-part-1-September 2013 exam: H/O returning from a camping holiday in the New Forest. C/O lethargy, arthralgia, rash consistent with erythema chronicum migrans. What is the most appropriate test to perform given the likely diagnosis? ELISA test for antibodies to Borrelia burgdorferi

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Lymphadenopathy There are many causes of generalised lymphadenopathy Infective • infectious mononucleosis • HIV, including seroconversion illness • eczema with secondary infection • rubella • toxoplasmosis • CMV • tuberculosis • roseola infantum Neoplastic • leukaemia • lymphoma Others • autoimmune conditions: SLE, rheumatoid arthritis • graft versus host disease • sarcoidosis • drugs: phenytoin and to a lesser extent allopurinol, isoniazid

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Malaria Malaria: Falciparum • P. falciparum typically presents within the first three months of return from an endemic area. In the slide shown, the blood film shows ring forms within erythrocytes; some erythrocytes contain two to three parasites per cell - typical of falciparum; other forms of malaria seldom have more than one parasite per red cell.

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Feature of severe malaria • schizonts on a blood film • parasitaemia > 2% • hypoglycaemia • acidosis • temperature > 39 C • severe anaemia • complications as below • Complications cerebral malaria: seizures, coma acute renal failure: blackwater fever, secondary to intravascular haemolysis, mechanism unknown acute respiratory distress syndrome (ARDS) (Respiratory rate 30 per minute) hypoglycaemia disseminated intravascular coagulation (DIC) Uncomplicated falciparum malaria • strains resistant to chloroquine are prevalent in certain areas of Asia and Africa • the 2010 WHO guidelines recommend artemisinin-based combination therapies (ACTs) as first-line therapy • examples include artemether plus lumefantrine, artesunate plus amodiaquine, artesunate plus mefloquine, artesunate plus sulfadoxine-pyrimethamine, dihydroartemisinin plus piperaquine Severe falciparum malaria • a parasite counts of more than 2% will usually need parenteral treatment irrespective of clinical state • Hyperparasitemia, where more than 5% of the red blood cells are infected by malaria parasites In 2010, WHO defined hyperparasitemia as >2%/100 000/µL in low intensity transmission areas or >5% or 250 000/µL in areas of high stable

malaria transmission intensity. • intravenous artesunate is now recommended by WHO in preference to intravenous quinine I.V quinine is reserved for severe or cerebral malaria (most deaths from M. falciparum occur in first 96 hours of starting treatment). The initial dose should NOT be reduced in those severely ill with renal/hepatic impairment. High doses of quinine in pregnancy are teratogenic in the first trimester. However in malaria, the benefit of treatment outweighs the risk. WHO Guidelines (2006) recommend artemisinins are first line in the second and third trimester. In the first trimester, both artesunate and quinine are considered treatment options. Hypoglycaemia is an important side effect of quinine Quinine ↑ insulin secretion and the sensitivity of cells to insulin hypoglycaemia Malaria itself can cause hypoglycaemia too, so blood glucose should be monitored every 2 h. • if parasite count > 10% then exchange transfusion should be considered • shock may indicate coexistent bacterial septicaemia - malaria rarely causes haemodynamic collapse Malaria: non-falciparum

• P. vivax: The most common cause of non-falciparum malaria is Plasmodium vivax, with Plasmodium ovale and Plasmodium malariae accounting for the other cases. The incubation period of P. vivax can go up to six months or more with malaria being caused by hypnozoites. P. falciparum incubation is normally around six days though it can go till 14 days or more. The Duffy antigen on RBCs acts as a receptor for P. vivax. facilitate the entry of P. vivax in to RBCs. Duffy negative individuals are therefore resistant to this strain West Africans lack the Duffy blood group and therefore P. ovale replaces P. vivax in this region. • P. ovale: it is quite rare The incubation period is similar to that of P. vivax but on the thick film the parasites are more compact and smaller. On the thin film the red blood cells appear oval with ragged ends. • P. malariae: it is rare. Its incubation could go up to14 days like P. falciparum. The thick film will show a few compact rings or small neat schizonts or small round gametocytes with yellow-brown pigment. The thin film will show red blood cells in band forms. • Plasmodium vivax is often found in Central America and the Indian Subcontinent whilst Plasmodium ovale typically comes from Africa • Both P. vivax and P. ovale have a liver hypnozoite stage which can cause repeated relapses. May present six months after return from an endemic area Features • fever, Plasmodium vivax/ovale: cyclical fever every 48 hours. Plasmodium malariae: cyclical fever every 72 hours • headache, • splenomegaly • Plasmodium malariae: is associated with nephrotic syndrome Investigations • Plasmodium ovale, all stages of the parasite and not just trophozoites and gametocytes are visible in the peripheral blood. • In P. falciparum malaria, only trophozoite-ring forms and gametocytes are usually seen. Treatment • non-falciparum malarias are almost always chloroquine sensitive • patients with ovale or vivax malaria should be given primaquine following acute treatment with chloroquine to destroy liver hypnozoites and prevent relapse. all individuals should be screened for glucose-6-phosphate dehydrogenase (G6PD) deficiency, as primaquine may cause haemolysis in those without the enzyme.

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fast-acting intermediate-acting slow-acting high-efficacy blood schizonticides that may be effective as monotherapy low-efficacy schizonticides that normally need to be administered in combination. Artemesinin Mepacrine Pyrimethamine Doxycycline is also a very slow-acting antimalarial. Pyrimethamine Quinine Mefloquine • used in the treatment of uncomplicated malaria, particularly for chloroquine-resistant P. falciparum. • It acts on both the erythrocytic and hepatic phases of infection. • It inhibits dihydrofolate reductase in the parasite thus preventing the biosynthesis of purines and pyrimidines, and thereby halting the processes of DNA replication, cell division and reproduction. • It is normally used alongside a sulfonamide. Malaria: prophylaxis • around 75% of malaria in patients returning from endemic countries are caused by the potentially fatal Plasmodium falciparum protozoa.
• The majority of patients who develop malaria did not take prophylaxis. • It should also be remembered that UK citizens who originate from malaria endemic areas quickly lose their innate immunity. Drug Side-effects + notes Atovaquone + proguanil (Malarone) GI upset 1 - 2 days 7 days Chloroquine Headache Contraindicated in epilepsy Taken weekly Doxycycline Photosensitivity Oesophagitis Mefloquine (Lariam) Dizziness Neuropsychiatric disturbance Contraindicated in epilepsy and mental illnesses Taken weekly Proguanil (Paludrine) Proguanil + chloroquine See above 1 week 4 weeks Notes & Notes for MRCP
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Time to begin before travel Time to end after travel 1 week 4 weeks 1 - 2 days 4 weeks 2 - 3 weeks 4 weeks 1 week 4 weeks

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• Which drug? In certain parts of South-East Asia there is widespread chloroquine resistance. Chemoprophylaxis using atovaquone + proguanil (Malarone), mefloquine (Lariam) or doxycycline is therefore recommended. Doxycycline prophylaxis is the safest option with less resistance in many parts of the world compared to the other options available. Atovaquone and proguanil are used for prophylaxis especially where there are high levels of resistance against most of the other drugs. Proguanil should not be used alone as malaria could develop resistance to it. • Pregnant women Pregnant women should be advised to avoid travelling to regions where malaria is endemic. Diagnosis can also be difficult as parasites may not be detectable in the blood film due to placental sequestration. However, if travel cannot be avoided: chloroquine can be taken proguanil: folate supplementation (5mg od) should be given Malarone (atovaquone + proguanil): the BNF advises to avoid these drugs unless essential. If taken then folate supplementation should be given mefloquine: caution advised doxycycline is contraindicated • Children It is again advisable to avoid travel to malaria endemic regions with children if avoidable. However, if travel is essential then children should take malarial prophylaxis as they are more at risk of serious complications. diethyltoluamide (DEET) 20-50% can be used in children over 2 months of age doxycycline is only licensed in the UK for children over the age of 12 years MRCPUK-part-1-May 2013 exam: H/O vivax malaria treated initially with chloroquine then later given primaquine. What is the benefit of the primaquine? Destroy liver hypnozoites and prevent relapse MRCPUK-part-1-May 2014 exam: A 25-year-old man with a history of epilepsy presents for advice regarding malarial prophylaxis. Next month he plans to travel to Vietnam. What is the most appropriate medication to prevent him developing malaria? Atovaquone + proguanil

Measles Overview • RNA paramyxovirus • spread by droplets • infective from prodrome until 4 days after rash starts • incubation period = 10-14 days Features • prodrome: irritable, conjunctivitis, fever Patients present with the three C's: cough, coryza, and conjunctivitis. Rash usually develops on the head and torso, typically sparing the wrists and hands. • Koplik spots (before rash): white spots ('grain of salt') on buccal mucosa Koplik's spots are small, irregular, bright red spots with blue-white centres, occurring on the inside of the cheek next to the premolars.Seen only in measles, they are diagnostic.

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 The spots usually occur briefly after the fever begins and a couple of days before the generalised rash appears. Not infrequently, the spots disappear as the eruption develops. • rash: starts behind ears then to whole body, discrete maculopapular rash becoming blotchy & confluent Koplik spots Complications • encephalitis: typically occurs 1-2 weeks following the onset of the illness) • subacute sclerosing panencephalitis: very rare, may present 5-10 years following the illness • febrile convulsions • giant cell pneumonia • keratoconjunctivitis, corneal ulceration • diarrhoea • increased incidence of appendicitis • myocarditis The rash typically starts behind the ears and then spreads to the whole body

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Management of contacts • if a child not immunized against measles comes into contact with measles then MMR should be offered (vaccine-induced measles antibody develops more rapidly than that following natural infection) • this should be given within 72 hours

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Rubella • also known as german measles. • RNA virus , part of the togavirus family rubella has positive single-stranded RNA. rubeola virus (measles) contains negative single-stranded RNA • affects unimmunized children and presents with a rash that begins at the head and moves down with postauricular lymphadenopathy. • A positive rubella haemagglutination inhibition (HAI) combined with a negative rubella IgM is consistent with:

1. Early acute infection with rubella The IgM may take several days to rise and the test should be repeated one to two weeks later.
2. Previous vaccination, or
3. Previous rubella infection.

Parotitis Causes • Bacterial parotitis Commonly unilateral more common in older patients. The most common bacterial cause of parotitis is Staphylococcus aureus. The risk is increased by agents that have an atropine-like action, including medications prescribed to reduce excess respiratory secretions. A ductal stone, with consequent pooling of infected secretions, should be

excluded, and ultrasound is an appropriate investigation to perform for this. Antibiotics should be selected that cover typical mouth flora. • Viral parotitis Mumps parotitis is usually bilateral Parotitis, orchitis, aseptic meningitis, and pancreatitis are symptoms of mumps virus infection. • autoimmune disease, Sjogren's syndrome. • Bulimia nervosa

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Parotid swelling • causes of bilateral parotid swelling include: Infection with viruses, including mumps, parainfluenza virus type 3, Coxsackie viruses and influenza A virus Metabolic diseases, such as: diabetes mellitus uraemia

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 Drugs, such as: phenylbutazone thiouracil • Other conditions associated with chronic parotid swelling include: Alcoholic liver disease Sarcoidosis Sjögren syndrome Lymphoma Infection with HIV

Orf Orf is generally a condition found in sheep and goats although it can be transmitted to humans. It is caused by the parapox virus. In animals • 'scabby' lesions around the mouth and nose In humans • generally affects the hands and arms • initially small, raised, red-blue papules • later may increase in size to 2-3 cm and become flat-topped and haemorrhagic

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Pelvic inflammatory disease(PID) Definition • infection and inflammation of the female pelvic organs including the uterus, fallopian tubes, ovaries and the surrounding peritoneum. • It is usually the result of ascending infection from the endocervix Causative organisms • Chlamydia trachomatis - the most common cause • Neisseria gonorrhoeae • Mycoplasma genitalium • Mycoplasma hominis one of the most frequently isolated mycoplasma in the genital tract. It is an opportunistic pathogen which may cause pelvic inflammatory disease in immunocompromised patients. Clindamycin is used in the treatment Features • lower abdominal pain • fever • deep dyspareunia • dysuria and menstrual irregularities may occur • vaginal or cervical discharge • cervical excitation Investigation • screen for Chlamydia and Gonorrhoea

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Management • due to the difficulty in making an accurate diagnosis, and the potential complications of untreated PID, consensus guidelines recommend having a low threshold for treatment • Consensus guidelines recommend treatment once a diagnosis of pelvic inflammatory disease is suspected, rather than waiting for the results of swabs • oral ofloxacin + oral metronidazole or intramuscular ceftriaxone + oral doxycycline

- oral metronidazole • RCOG guidelines suggest that in mild cases of PID intrauterine contraceptive devices may be left in. The more recent BASHH guidelines suggest that the

evidence is limited but that ' Removal of the IUD should be considered and may be associated with better short term clinical outcomes' Complications • infertility - the risk may be as high as 10-20% after a single episode • chronic pelvic pain • ectopic pregnancy • Fitz-Hugh-Curtis syndrome is a rare complication of pelvic inflammatory disease, resulting in liver capsule inflammation. It is most often caused by untreated sexually transmitted infections including Chlamydia trachomatis and Neisseria gonorrhoeae. a patient may present with septic shock secondary to the untreated liver capsule infection.

Psittacosis (ornithosis) • Chlamydia psittaci is endemic in birds including psittacine birds, canaries, finches, pigeons and poultry. • Pet owners, vets and zoo keepers are most at risk. It is rare in children. • Person to person transmission occurs especially in a hospital environment. • Sputum Gram stain reveals a few leucocytes and no predominant bacteria. • There are few signs and few laboratory/x ray findings. • Positive serology is with complement-fixing antibodies. • It is treated with tetracycline.

Pyogenic liver abscess • The most common organisms found in pyogenic liver abscesses are Staphylococcus aureus in children and Escherichia coli in adults. Management • amoxicillin + ciprofloxacin + metronidazole • if penicillin allergic: ciprofloxacin + clindamycin January 2018 exam: What is the most appropriate antibiotic therapy to accompany drainage of liver abscess? Amoxicillin + ciprofloxacin + metronidazole

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Pyrexia of unknown origin indium labelled leukocyte study: • useful for detecting occult abscesses in patients with pyrexia of unknown origin where conventional scans have failed to detect a source of infection. Definition • Defined as a prolonged fever of > 3 weeks which resists diagnosis after a week in hospital Neoplasia • lymphoma • hypernephroma • preleukaemia • atrial myxoma Infections • abscess • TB Connective tissue disorders

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Q fever Q fever - Coxiella burnetti Overview • Q fever is a zoonotic disease caused by Coxiella burnetii an obligate gram-negative intracellular bacterium. • The organism is very resistant to drying. • does not grow on standard culture media. Transmission • The organism is usually inhaled from infected dust (animal products) • acquired through contact with animals. Cattle, sheep and goats are the primary reservoirs of C. burnetii. • drinking unpasteurised milk from infected cows. Risk factors • It is not notifiable, but can occur in outbreaks in farming communities and in abattoirs. and therefore an occupational history is very important. Features: • high fevers, chills, sweats • severe headache, (typically retrobulbar) • general malaise, myalgia, • confusion, • sore throat, , • non-productive cough, • nausea, vomiting, diarrhoea, abdominal pain • chest pain. • Between 30% and 50% of patients with a symptomatic infection will develop pneumonia. • may be complicated by immune complex-mediated glomerulonephritis

• Chronic infection can manifest as hepatitis, osteomyelitis or endocarditis. • In Q fever endocarditis: the aortic valve is involved in over 80% of cases. A murmur is not always present, but augmentation of an existing murmur may occur. Low-grade fever (or no fever), signs of heart failure, hepatosplenomegaly, clubbing, arterial emboli, leukocytoclastic vasculitic rash. Diagnosis: • Confirmed by serological testing for C. burnetii. phase I antibody titre to Coxiella burnetti (IgG and/or IgA) greater than 1:200 is virtually diagnostic of Q fever. • chest X-ray might show multilobar consolidation. • Anaemia • Thrombocytopenia • Elevated ESR • Hypergammaglobulinaemia • liver function tests abnormal in the majority of patients and some will develop hepatitis. • Microscopic haematuria may be present. Treatment : • Most patients will recover within a few months with no treatment. • Doxycycline is the treatment of choice for acute Q fever. OR prolonged courses of tetracyclines. Prognosis • Only 1–2% of people with acute Q fever die of the disease. • Chronic Q fever Endocarditis with negative culture findings and seropositivity is the main clinical presentation of chronic Q fever, usually occurring in patients with preexisting cardiac disease including valve defects, rheumatic heart disease, and prosthetic valves.

Rabies Overview • Rabies is a viral disease that causes an acute encephalitis. • The rabies virus is classed as a RNA rhabdovirus and has a bullet shaped capsid. • It is commonly transmitted by bat, raccoon and skunk bites. • Following a bite the virus travels up the nerve axons towards the central nervous system in a retrograde fashion. Features • prodrome: headache, fever, agitation • hydrophobia: water-provoking muscle spasms • hypersalivation • Negri bodies: cytoplasmic inclusion bodies found in infected neurons

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There is now considered to be 'no risk' of developing rabies following an animal bite in the UK and the majority of developed countries. Following an animal bite in at risk countries: • if an individual is already immunised then 2 further doses of vaccine should be given • if not previously immunised then human rabies immunoglobulin (HRIG) should be given along with a full course of vaccination • Lyssaviruses such as rabies cannot cross intact skin and humans are regarded as an end-host (outside of transplantation-associated transmission). Therefore, only standard infection-prevention precautions such as gloves and gowns are required.

Scabies Scabies should be suspected in any sexually active young person who presents with generalised pruritus without any specific signs. Overview • Scabies is caused by the mite Sarcoptes scabiei and is spread by prolonged skin contact. • It typically affects children and young adults. Pathophysiology • The scabies mite burrows into the skin, laying its eggs in the stratum corneum. • The intense pruritus associated with scabies is due to a delayed type IV hypersensitivity reaction to mites/eggs which occurs about 30 days after the initial infection. Features • widespread pruritus •

linear burrows on the side of fingers, interdigital webs and flexor aspects of the wrist The tiny erythematous burrows in the web spaces of the fingers are almost pathognomonic • in infants the face and scalp may also be affected • secondary features are seen due to scratching: excoriation, infection Investigation • Skin scrapings demonstrate Sarcoptes scabiei Management • first-line is permethrin 5% • second-line is malathion 0.5% • Application should be repeated seven days after initial treatment to kill any mites hatched from eggs in that time • give appropriate guidance on use (see below) • pruritus persists for up to 4-6 weeks post eradication Patient guidance on treatment (from Clinical Knowledge Summaries) • avoid close physical contact with others until treatment is complete • all household and close physical contacts should be treated at the same time, even if asymptomatic Re-infection most likely means Other household members were not treated

• launder, iron or tumble dry clothing, bedding, towels, etc., on the first day of treatment to kill off mites. Patients should be given the following instructions: • The BNF advises to apply the insecticide to all areas, including the face and scalp, contrary to the manufacturer's recommendation. • apply the insecticide cream or liquid to cool, dry skin • pay close attention to areas between fingers and toes, under nails, armpit area, creases of the skin such as at the wrist and elbow • allow to dry and leave on the skin for 8-12 hours for permethrin, or for 24 hours for malathion, before washing off • reapply if insecticide is removed during the treatment period, e.g. If wash hands, change nappy, etc • repeat treatment 7 days later Crusted (Norwegian) scabies • Crusted scabies is seen in patients with suppressed immunity, especially HIV. • The crusted skin will be teeming with hundreds of thousands of organisms. • Ivermectin is the treatment of choice and isolation is essential

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Helminths Nematodes (roundworms) Worm Notes Treatment Larvae are present in soil and gain access to the body by penetrating the skin Strongyloides stercoralis Features include diarrhoea, abdominal pain, papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks, larva currens: pruritic, linear, urticarial rash, if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered Enterobius vermicularis (pinworm) asymptomatic in 90% of cases, possible features include perianal itching, particularly at night; girls may have vulval symptoms Diagnosis may be made by the applying sticky plastic tape to the perianal area and sending it to the laboratory for microscopy to see the eggs Larvae penetrate skin of feet; gastrointestinal infection → anaemia Thin-shelled ova Ancylostoma duodenale, Ne cator americanus(h ookworms) Loa loa Transmission by deer fly and mango fly Causes red itchy swellings below the skin called 'Calabar swellings', may be observed when crossing conjunctivae Typically develops after eating raw pork. Features include fever, periorbital oedema and myositis (larvae encyst in muscle) Trichinella spiralis Causes 'river blindness'. Spread by female blackflies Onchocerca volvulus Features include blindness, hyperpigmented skin and

possible allergic reaction to microfilaria Transmission by female mosquito Wuchereria bancrofti Causes blockage of lymphatics → elephantiasis Transmitted through ingestion of infective eggs. Toxocara canis (dog roundworm) Features include visceral larva migrans and retinal granulomas VISCious dogs → blindness Notes & Notes for MRCP
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Ivermectin and - bendazoles are used -bendazoles -bendazoles Diethylcarbamazine -bendazoles Ivermectin rIVERblindness = IVERmectin Diethylcarbamazine Diethylcarbamazine

Worm Notes Treatment Ascaris lumbricoides(giant roundworm) • the most common nematode parasite of humans. • Eggs are visible in faeces • large roundworm, growing up to 35 cm in length • result of pneumonitis caused by the worm's migration through the lungs • May cause intestinal obstruction and occasional migrate to lung (Loffler's syndrome) • biliary/pancreatic duct obstruction. Cestodes (tapeworms) Worm Notes Treatment • Responsible for hydatid disease • Transmission through ingestion of eggs in dog faeces. • Definite host is dog, which ingests hydatid cysts from sheep, who act as an intermediate host. • Often seen in farmers. • Features include liver cysts and anaphylaxis if cyst ruptures (e.g. during surgical removal) • the most appropriate next step in diagnosis? ELISA testing for Echinococcus Echinococcus granulosus Taenia solium Often transmitted after eating undercooked pork. Causes cysticercosis and neurocysticercosis, mass lesions in the brain 'swiss cheese appearance' May cause biliary obstruction Triclabendazole Fasciola hepatica (the liver fluke) Trematodes (flukes) Worm Notes Treatment Hosted by snails, which release cercariae that penetrate skin. Causes 'swimmer's itch' - frequency, haematuria. Risk factor for squamous cell bladder cancer Schistosoma haematobium Caused by undercooked crabmeat, results in secondary bacterial infection of lungs Paragonimus westermani Clonorchis sinensis Caused by undercooked fish Features include biliary tract inflammation. Known risk factor for cholangiocarcinoma Notes & Notes for MRCP
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-bendazoles Piperazine is the treatment of choice in patients presenting with bowel obstruction; mebendazole may be used to treat other infections. • bendazoles alone (For smaller cysts) • albendazole combined with surgical excision. • (for larger cysts) -bendazoles Praziquantel Praziquantel Praziquantel

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Infectious diseases

Schistosomiasis Schistosoma haematobium causes haematuria Schistosomiasis, or bilharzia, is a parasitic flatworm infection. Types • Schistosoma mansoni and Schistosoma intercalatum: intestinal schistosomiasis • Schistosoma haematobium: urinary schistosomiasis This typically presents as a 'swimmer's itch' in patients who have recently returned from Africa. Schistosoma haematobium is a risk factor for squamous cell bladder cancer Features • frequency • haematuria • bladder calcification Management • single oral dose of praziquantel • Praziquantel is the treatment of choice for all Schistosoma species. • CNS involvement S. japonicum Praziquantel 60 mg/kg per day for 6 days and prednisolone 1 mg/kg per day Praziquantel 60 mg/kg per day

for six days is recommend for S. japonicum with a maximum dose of 5 grams per day with prednisolone 1 mg/kg. S. mansoni and S. haematobium. Praziquantel 40 mg/kg per day for three days is recommended for S. mansoni and S. haematobium. Since some of the pathology in neuroschistosomiasis is secondary to hypersensitivity reactions there is need to use a steroid, in this case prednisolone 1 mg/kg per day. There is no consensus about when it should be started or stopped. Complications: • S. mansoni Eggs can migrate to liver through the portal venous system where they can elicit a granulomatous fibrosing reaction venous blockade Portal venous hypertension varicies and upper GIT bleeding. • S. haematobium leads to granulomatous inflammation, ulceration of the vesicle and ureteral walls. Subsequent fibrosis can cause bladder neck obstruction, hydroureter and hydronephrosis. These changes can cause a chronic renal impairment and predispose to secondary bacterial infection as well as squamous cell carcinoma. • all schistosome species can result in immune complex deposition in the kidneys leading to a proteinuria and nephrotic syndrome. • S. japonicum: is prevalent in China, Indonesia, Thailand and the Philippines mainly. It is the commonest cause of Schistosoma encephalitis. Its eggs are smaller unlike those of S. masoni and S. haematobiumwhich are more likely to cause spinal cord schistosomiasis because of their larger size and spikes which do not enable them get to the brain hence the infection in the spinal cord.

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Strongyloides stercoralis • Strongyloides stercoralis is a human parasitic nematode worm. The larvae are present in soil and gain access to the body by penetrating the skin. Infection with Strongyloides stercoralis causes strongyloidiasis. Features • diarrhoea • abdominal pain/bloating • papulovesicular lesions where the skin has been penetrated by infective larvae e.g. soles of feet and buttocks • larva currens: pruritic, linear, urticarial rash • if the larvae migrate to the lungs a pneumonitis similar to Loeffler's syndrome may be triggered Treatment • ivermectin and albendazole are used

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Tape worms • Tape worms are made up of repeated segments called proglottids. These are often present in faeces and are useful diagnostically Cysticercosis • caused by Taenia solium (from pork) and Taenia saginata (from beef) • These may affect any tissue in the body but are commonest in subcutaneous tissues and (CNS) patient with a palpable nodule who has an epileptic seizure • management: niclosamide Hydatid disease • caused by the dog tapeworm Echinococcus granulosus • life-cycle involves dogs ingesting hydatid cysts from sheep liver • often seen in farmers • may cause liver cysts • management: albendazole

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Trypanosomiasis • Two main form of this protozoal disease are recognised: 1. African trypanosomiasis (sleeping sickness) and 2. American trypanosomiasis (Chagas' disease)

1. African trypanosomiasis, or sleeping sickness Two forms of African trypanosomiasis, or sleeping sickness, are seen:

1. Trypanosoma brucei gambiense in West Africa West African trypanosomiasis has a slower course. Symptoms start several weeks or even months after the tsetse fly bite.
2. Trypanosoma brucei rhodesiense in East Africa. Trypanosoma rhodesiense tends to follow a more acute course. progression is more rapid - starting within days of infection. Death may occur within weeks or months. Rash is a more prominent feature and lymphadenopathy is less frequently present. Both types are spread by the tsetse fly. Clinical features include: Trypanosoma chancre - painless subcutaneous nodule at site of infection

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Infectious diseases intermittent fever enlargement of posterior cervical lymph nodes later: central nervous system involvement e.g. somnolence, headaches, mood changes, meningoencephalitis The reversal of the sleep wake cycle is typical and can be accompanied by behavioural changes. Stages The first stage of disease is haematolymphatic spread and is accompanied by fever, and lymphadenopathy (discrete, rubbery, non-tender nodes). A rash sometimes occurs and mild hepatosplenomegaly may develop. The second stage is the meningoencephalitic stage. This occurs months or years after the acquisition of infection. Manifestations include personality change and progressive indifference with daytime somnolence. Extrapyramidal signs and ataxia are common. Management early disease: IV pentamidine or suramin later disease or central nervous system involvement: IV melarsoprol 2. American trypanosomiasis, or Chagas' disease caused by the protozoan Trypanosoma cruzi. Transmitted by triatomine bug bite. Features: acute phase: asymptomatic (95%) chagoma (an erythematous nodule at site of infection) periorbital oedema Chronic Chagas' disease mainly affects the heart, gastrointestinal tract and CNS. Cardiac feature myocarditis may lead to dilated cardiomyopathy (with apical atophy) and arrhythmias. Cardiac involvement is the leading cause of death in patients with Chagas' disease GIT feature: Mega-oesophagus (causing dysphagia) Mega-colon (causing constipation) CNS feature meningoencephalitis Management treatment is most effective in the acute phase using azole or nitroderivatives such as benznidazole or nifurtimox chronic disease management involves treating the complications e.g., heart failure.

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Nematodes • most common cause of cutaneous larva migrans Ancylostoma braziliense • commonest cause of visceral larva migrans Toxocara canis Ancylostoma braziliense • most common cause of cutaneous larva migrans • common in Central and Southern America • The infection is acquired by direct contact with dog or cat faeces - often acquired when sunbathing on contaminated sand, etc. The larvae burrow in the dermo-epidermal junction. • Symptoms include pruritus and a raised, serpiginous erythematous rash that migrates at a rate of up to 1 cm/day. • Treatment i The disease is self-limiting but the duration of disease varies considerably Oral ivermectin in a single dose of 200 µg/kg body weight is the main treatment. Other treatment options include oral albendazole or topical thiabendazole. Strongyloides stercoralis • acquired

percutaneously (e.g. walking barefoot) • causes pruritus and larva currens - this has a similar appearance to cutaneous larva migrans but moves through the skin at a far greater rate • abdo pain, diarrhoea, pneumonitis • may cause Gram negative septicaemia due carrying of bacteria into bloodstream • eosinophilia sometimes seen • management: thiabendazole, albendazole. Ivermectin also used, particularly in chronic infections Toxocara canis • commonly acquired by ingesting eggs from soil contaminated by dog faeces • commonest cause of visceral larva migrans • other features: eye granulomas, liver/lung involvement cutaneous larva migrans cutaneous larva migrans

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Infectious diseases

Filariasis • Manifestations of filariasis Remember 3 L's: Lymphatic filariasis (caused by Wuchereria bancrofti and Brugia malayi) Loiasis (caused by Loa loa) Light (light, sight, blindness - river blindness caused by Onchocerca volvulus) Tropical eosinophilia: Tropical eosinophilia is an allergic reaction to microfilaria of Wuchereria bancrofti. Characteristic features include: myalgia; fatigue; weight loss; cough and dyspnoea with wheeze; fever; current or previous residence in an area endemic for filariasis (southern Asia, Africa, India, South America); lymphadenopathy; marked peripheral blood eosinophilia high titres of anti-filarial antibodies. The chest x ray shows bilateral reticulonodular shadowing. This condition is commonly accompanied by false positive serological tests for syphilis and high titres of cold agglutinins. There is typically a rapid response to treatment with diethylcarbamazine. • Diagnosis finger prick test identifying microfilariae on Giemsa stained, thin and thick blood film smears, "Filariasis fills the blood at night." To remember that Microfilaria can be demonstrated in peripheral smear only at night. W. bancrofti, whose vector is a mosquito; night is the preferred time for blood collection. Loa loa's vector is the deer fly; daytime collection is preferred. • Which immune mechanisms does the body employ against the live filarial worms ? Antibody-dependent cell-mediated cytotoxicity

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