Scrub Typhus Department of Infectious Diseases, The Third Affiliated Hospital Li Gang

Definition Acute febrile infectious disease Caused by Rickettsia tsutsugamushi Transmitted by the bite of chiggers Source of infection: Rats Characterized by fever, eschar, rash, and lymphadenopathy

Etiology Intracellular organism Propagated in mice, some cells Antigenically diverse microorganism Common antigen with Proteus OX-K Low resistance

Epidemiology % Source of infection: Rats % Vector: Mite The life cycle: ovum, larva, pupa, naiad and imago Only its larva (chigger) feed on humans

Epidemiology Proliferate in warm, wet environments Both a vector and a reservoir % Route of transmission: bite of chigger % Susceptibility: universal % Immunity: long-lasting immunity against the homologous strains; Partial immunity against the other heterologous strains

Epidemiology % Other epidemiological features: Geographic distribution: Asian-pacific region Sporadic Seasonal distribution: rainy seasons between May and November

Characteristic manifestations $ Eschar and ulceration: Characteristic sign. Seen in 36.9ˆ 98| of patients. Generally located in warm, wet, intense smelled areas.

Characteristic manifestations 2. Lymphadenopathy: Enlarged markedly regional lymph nodes near the eschar. Generalized lymphadenopathy. Painful, movable, not purulent.

Characteristic manifestations 3. Rash: Appears on the 4th to the 6th day. Beginning on the trunk, spread to the extremities. Maculopapular, congestive, no itching. Lasting 3ˆ 7 days. Seen in 35ˆ 100| of patients.

Characteristic manifestations 4. Splenomegaly and hepatomegaly Splenomegaly : 30ˆ 50| of patients. Hepatomegaly: 10ˆ 20| of patients.

J Complications Pneumonia, myocarditis, DIC, hepatitis.

Diagnosis $ Epidemiologic data History of exposure to endemic areas. Rainy season.

Diagnosis 2. Clinical features Abrupt onset of fever. Accompanied with chill, lymphadenopathy, rash, enlargement of spleen and liver. Most characteristic sign: eschar. Therapeutic diagnosis.

Diagnosis 3. Lab findings A. Routine lab data: WBC ! liver enzyme values ! proteinuria. B. Serological tests `$ Weil-Felix reaction

Diagnosis `$ Weil-Felix reaction: Positive result obtained from the 4th day. Higher than 1:160 is suggestive. Available and inexpensive. About 50| of patients have diagnostic titers.

Diagnosis a$ Complement fixation test and indirect immunofluorescent antibody test: More sensitive and specific than Weil- Felix reaction. Fourfold or greater increase in convalescence. Rarely used because of the difficult in preparing antigens.

Diagnosis C. Isolation of organism Blood inoculating into mice. Organism found in mononuclear cells. Specific test to confirm the diagnosis. Can't give a rapid diagnosis.

Diagnosis D. Molecular biologic assays Nucleic acid hybridization and PCR. Great potential for sensitive and specific detection of nucleic acid. Being evaluated. Limitation of technique and facilities conditions.

Differential Diagnosis Other rickettsial diseases, typhoid fever, leptospirosis, malaria, dengue, septicemia, influenza Prognosis Fatality rate: 9ˆ 60| without treatment. < 5| effective treatment.

Treatment Chloramphenicol, tetracycline, doxycycline Chloramphenicol: 1.5ˆ 2g daily, divided into 3ˆ4 aliquots. Fever abates within 48 hours. Total course: 10ˆ14 days. Side effects: suppression of bone marrow and aplastic anemia.

Prevention $ Exterminating the source of infection. $ Reducing and controlling the vectors. $ Individual protection: Avoiding contact with mites: the best method.

*Cysticercosis

Definition and introduction One of parasitic diseases. Caused by the infection with the tissue larvae of Taenia solium. Acquired by ingestion of Taenia solium eggs in contaminated foods. Humans: definite host and intermediate host.

Definition and Introduction Human infected with Taenia solium in two forms: intestinal Taenia solium and cysticercosis. Cysticercosis has greater clinical significance. Cysticercosis: human tissue infection with the intermediate cyst forms of pork tapeworm. Cysticercus located in the subcutaneous tissue, muscle, brain.

Etiology and Pathogenesis $Taenia solium eggs passed out from patients. a$ Eggs taken in by the fecal-oral route. b$ Eggs digested by gastric juice to liberate oncosphere. c$ Oncosphere penetrates the intestinal wall into blood circulation. d$Located in subcutaneous tissue, muscle, brain. e$Scolex appears and develops into cysticercus.

Etiology and Pathogenesis Cysticercus: %Bladder-like, fluid-filled cyst. %Containing an invaginated scolex. % Surrounded by fibrous capsule. % Multiple, 0.5ˆ2cm in size.

Etiology and Pathogenesis Cysticercus: % Location in subcutaneous tissue and skeletal muscle to produce minimal, if any, symptoms; % Location in brain to have serious effects; % Location in substantial to be able to occupy the space to produce the relevant sign; % Location in ventriculi to produce cysticercus racemosus.

Epidemiology %Geographic distribution: Latin America, East Europe, southeast Asia, Africa. % Source of infection: patient. % Transmission: fecal-oral route. % Susceptibility: universal.

Clinical Manifestation % Depend on 2 factors: `$The location and number of infecting cysts; a$If the inflammation exists. % Brain cysticercosis: found in 60ˆ92% of the total cases. % Subcutaneous nodule: found in 2/3 of the total cases.

Clinical Manifestation $ Brain cysticercosis The incubation period: within 5 years. `$ Cerebral cysticercosis % Epilepsy: Caused by cysticerci located in the cortex near to the motorium. Always the first and the only symptom.

Clinical Manifestation % Epilepsy: % Multifocal and unstable seizure. % 2/3 of the grand mal begin with a local spasm. %Petit mal includes sensory and motorial obstruction.

Clinical Manifestation % Neurosis: %May be the only manifestation in patients with cryptogenic cysticercosis. %Intracranial hypertension symptoms: vomiting, headache, visual disturbances.

Clinical Manifestation a$ Ventricular cysticercosis % 10|of brain cysticercosis. % Caused by acute obstruction of CSF circulation. % Manifest as the valve syndrome (Brun's syndrome ) with intermittent positional severe headache, vomiting, shock.

Clinical Manifestation b$ Subarachnoid cysticercosis % 10|of brain cysticercosis. % Chronic, intermittent meningitis. % 3/4 have increased intracranial pressure. c$ Mixed form More serious neuropsychic symptoms.

Clinical Manifestation $ Ocular cysticercosis % 1.8|of patients with cysticercosis. % Single eye involved. % Eye pain, decreasing vision, retinal detachment.

Clinical Manifestation $ Subcutaneous or muscle cysticercosis % 2/3 of the patients have nodules. % Number of nodules: 1ˆ 1000. %More frequently felt on body and head. %Generally no symptoms

Diagnosis $ Definitive diagnosis: biopsy of tissue cyst. % Clinical diagnosis: & History of residence in an endemic area; & Clinical manifestation; & Plain film, CT, MRI; & Suggestive laboratory finding: detection of specific IgG in serum or CSF.

Treatment $ Albendazole 1g daily, 2 aliquots, 10 days, repeat it 2 weeks later. $ Praziquantel 3g daily, 3 aliquots, 3 days.

Treatment Notice when treatment: % Patients should be hospitalized during the drug therapy. % Epilepsy should be controlled at the same time. % Increased intracranial pressure should be decreased.

Treatment Notice when treatment: % Patients with cysticercosis of the eye should not receive drug therapy until the eye disease has been controlled surgically. % Surgery preferential for CSF obstruction. % Serious side effects easily occurred in patients with psychiatric changes.

Prevention % Avoid consuming undercooked pork and contaminated food. % Careful personal hygiene. % High level of community sanitation.

rat 2 naiad larva imago pupa ovum

The life cycle of mite

human body rickettsemia rickettsia vasculitis eschar local site mite rats

Pathogenesis

Pathology Basic lesion: inflammation of the walls of the small blood vessels Pathologic findings: eschar, lymphadenopathy, rash, enlargement of spleen and liver Serious pathologic manifestations: myocarditis, meningoencephalitis, pneumonia, interstitial nephritis

Clinical manifestations Incubation period: 4ˆ 21 days Sudden onset, High fever: remittent fever accompanied by chill, headache, malaise, prostration, poor appetite.

Clinical manifestations Signs of multiorgan damage: Meningoencephalitis: delirium, convulsion, coma, and neck stiffness. Interstitial pneumonia: cough, chest pain, breathlessness.

Clinical manifestations Signs of multiorgan damage: myocarditis: gallop rhythm, poor quality heart sounds, systolic murmurs. hepatitis: jaundice, hemorrhage. Natural course: 2ˆ 3 weeks.

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