Infectious or parasitic diseases transmitted to man by animals or remains of animals

General consideration

Many infectious diseases can be transmitted by occupational exposure to animals or their excreta. This chapter covers **leptospirosis, tularaemia, Lyme disease, psittacosis, avian influenza, Q fever** and **erysipeloid**.

The main occupational groups at risk for these infections are: farmers, mainly animal breeders, abattoir workers, butchers, meat packers, agricultural engineers, laboratory technicians, workers engaged in the preparation of animal skin, forestry workers, and veterinary workers.

Some human diseases which are undoubtedly transmitted by infected animals are too rare to be examined here. They will be recognized as occupational illnesses only by individual assessment in each case, or as a complication of an occupational accident, particularly following bites or stings (rabies, pasteurellosis, malaria, etc.).

Leptospirosis

1. Definition and causal agent

Leptospirosis is caused by the *Leptospira interrogans* complex. Leptospira are thin, highly motile spirochaetes with hooked ends. The organisms are microscopically demonstrable by dark-field illumination and silver staining. There are 19 serological groups.

2. Transmission of infection

2.1 Exposure

Rodents, especially rats, are the most important reservoir. *Rattus norvegicus* and *Mus musculans* carry a broad spectrum of serotypes, though other animals, including cattle, pigs, goats, dogs, foxes and voles, can be infected. The organism may exist in the animal host without causing pathological change. Exposure to urine of these animals is the commonest type of human risk.

2.2 Occupational groups at risk

People working in a rat-infested environment or where there is infected material or water are at greatest risk. These include agricultural workers (especially those working in rice fields), sewer workers, miners, veterinarians, abattoir workers, fish handlers and military personnel.

3. Clinical disease

3.1. Presenting features

The incubation period of 2 to 20 days depends on the host response and the quantity of micro-organisms. In the mild form there may only be a low-grade fever, but the severe form associated (Weil's disease) is characterised by jaundice, renal failure and haemorrhages. Cardiovascular collapse may occur.

The disease has two phases: a septicaemic phase, which lasts 4 to 7 days with fevers, headaches, myalgia and conjunctival infection, whilst renal failure and jaundice (10%-20%) are uncommon but serious features. A second immune phase lasts four to 30 days. The immune phase coincides with the disappearance of the spirochaete from most tissues. Uveitis, rashes, meningitis, encephalitis and myelitis may occur. Liver and kidney abnormalities continue from the first phase.

3.2 Laboratory diagnosis

A history of exposure to rat-infested environments is very helpful in differentiating leptospirosis from other pyrexial illnesses. Leucocytosis with neutrophilia (and in 40% of cases a thrombocytopaenia) with increased plasma fibrinogen levels are supplemented by abnormal urinalysis and the demonstration of leptospira spp. in the urine. Liver function tests may be abnormal. After the second week of the disease, isolation of the organism becomes less likely but serology is then important. Macroscopic slide agglutination is a good screening test supplemented by microscope-type specific agglutination tests, where titres of 1:100 are sufficient to indicate a previous infection.

3.3 Prognosis

Recovery is the rule but older patients and those with severe renal, haematological and hepatic change may succumb. Renal dialysis has greatly reduced previous mortality figures and the long-term follow-up of dialysed patients indicates a good recovery of renal functions.

Exposure criteria:

Acute infections and their complications:

Minimum intensity and duration of exposure: not applicable

Maximum latent period: three weeks

Tularaemia

1. Definition and aetiological agent

Tularaemia is caused by *Franciscella tularensis.* A number of animal species may be infected by this gram-negative bacillus and humans can easily be contaminated, most often through direct contact. The disease is usually characterized by skin lesions with regional ganglion hypertrophy.

2. Transmission of infection

2.1 Exposure

Many animals are natural carriers of *F. tularensis.* They include, particularly, hares, rabbits, squirrels, marmots, musk rats, foxes, mice, rats, quails and pheasants. Humans are very sensitive to tularaemia; the bacteria most often enter directly through the skin, even where there is no existing skin lesion, and only rarely through insect vectors (ticks). The bacillus can also penetrate the body through the mucous membranes, the digestive tract or the respiratory tract.

2.2 Occupational groups exposed

Gamekeepers, foresters; those involved in animal rearing, slaughtering and transport; handling of rabbits, hares and other small furry animals; preparation of animal skins; laboratory work involving contact with rabbits and small rodents.

3. Clinical picture

3.1 Presenting features

The incubation period is usually three to five days. The clinical forms depend mainly on the path of infection, but all involve fever, asthenia, joint and muscle pain and headache. The most common clinical form combines ulceration at the point of infection with regional adenopathy. The eyes, lungs and digestive tract may be affected, depending on the path of infection.

3.2 Diagnosis

Isolation of bacteria from lesions. Serological tests to identify antibodies.

Exposure criteria:

Minimum induction period: a few hours

Maximum latent period: 15 days.

Lyme disease

1. Definition and aetiological agent

Lyme arthritis is caused by the spirochaete *Borellia burgdorferi* and is transmitted to humans through tick bites. It is characterized by chronic migratory erythema sometimes accompanied by joint or neurological disorders.

2. Transmission of infection

2.1 Exposure

Dogs and a number of wild species including deer may carry the bacteria. Some species of tick are responsible for transmitting the disease to humans.

2.2 Occupational groups exposed

All forestry work in areas where the disease is endemic.

3. Clinical picture

3.1 Symptoms

Chronic migratory erythema appears 3 to 20 days following the tick bite. Skin lesions may be accompanied by general signs, arthralgia and myalgia. Encephalitis, myocarditis and arthritis may develop.

3.2 Diagnosis

Isolation of bacteria (difficult).

Serological tests for specific antibodies.

Exposure criteria:

Minimum induction period: 3 days;

Maximum latency period: one month for chronic migratory erythema, Six months for late-appearing sequelae.

Psittacosis

1. Definition and aetiological agent

Ornithosis is caused by *Chlamydia psittaci.* Infection is most often characterized by acute respiratory disease.

2. Transmission of infection

2.1 Exposure

C. psittaci is carried by domestic and wild birds. For humans, the infection is airborne from a bird-contaminated environment.

2.2 Occupational groups exposed

Work involving contact with birds, poultry or their excreta.

3. Clinical picture

3.1 Symptoms

Following an incubation period of usually one to two weeks, infection is usually characterized by an acute, febrile pneumonia. Asymptomatic forms may also be observed.

3.2 Diagnosis

Intracellular isolation of the bacteria is difficult. A number of serological tests can be carried out to identify antibodies. However, cross reactions may be observed between *C. psittaci, C trachomatis* and *C. pneumoniae.* In addition, early treatment with tetracyclines can reduce antibodies.

Exposure criteria:

Minimum induction period: 48 hours

Maximum latent period: 21 days.

Avian influenza

1. Definition and causal agent

Avian influenza is caused by the Orthomyxoviridae virus *Influenza A*. Virus subtypes are identified by their glycoprotein haemagglutinin (H) and neuraminidase (N) antigens. At present 15 H subtypes (H1-H15) and nine neuraminidase subtypes (N1-N9) have been recognised. The virus primarily infects birds but more rarely other species, including pigs and humans. To date, all outbreaks of the highly pathogenic form of avian influenza have been caused by viruses of the H5 and H7 subtypes. Avian H5N1 is a strain with pandemic potential, since it might ultimately adapt into a strain that is contagious among humans.

2. Transmission of infection

2.1 Exposure

Wild waterfowl introduce avian influenza viruses, in their low pathogenic form, to poultry flocks, but do not carry or directly spread highly pathogenic viruses. Other bird species, including domestic poultry, develop disease when infected with avian influenza viruses. All evidence to date indicates that close contact with dead or sick birds is the principal source of human infection with the H5N1 virus.

2.2 Occupational Groups at risk

Workers engaged in slaughtering, plucking, butchering and preparing for consumption infected birds. Swimming in water where the carcasses of dead infected birds have been discarded or which may have been contaminated by faeces from infected ducks or other birds might be another source of exposure.

3. Clinical Disease

3.1 Symptoms

In general, human infection with these viruses has resulted in mild symptoms and very little severe illness. There is one notable exception: the highly pathogenic H5N1 virus. In many patients, the disease caused by the H5N1 subtype follows an aggressive clinical course, with rapid deterioration and high fatality. Initial symptoms include high fever, with a temperature above 38°C, and influenza-like symptoms. Diarrhoea, vomiting, abdominal pain, chest pain, and bleeding from the nose and gums have also been reported as early symptoms in some patients. The spectrum of clinical symptoms may, however, be broader, and not all confirmed patients have presented with respiratory symptoms. In some cases acute encephalitis, in the absence of any respiratory disease, has occurred.

3.2 Diagnosis

Common laboratory abnormalities, include leucopenia (mainly lymphopenia), thrombocytopenia, elevated serum aminotransferases, and evidence of disseminated intravascular coagulation. Isolation and identification of the causative virus. Tests for serum antibodies against H and N antigens are available for some virus subtypes.

3.3 Prognosis

Depending on the type of infection, from very good to fatal. Infection with the H5N1 strain has a poorer prognosis.

Exposure criteria:

Minimum intensity and duration of exposure: unknown.

Maximum latent period: 2 to 17 days (median 7 days).

Q fever

1. Definition and aetiological agent

Q fever is caused by the rickettsia *Coxiella burnetii.* In humans, infection is often benign and unnoticeable, but it can cause intermittent fever and in some cases endocarditis and hepatitis.

2. Transmission of infection

2.1 Exposure

There are two main routes by which humans can acquire infection with C.burnetii.

(i) *via* infected domestic animals, particularly cattle and sheep. Humans can be infected from contaminated placentas, aborted matter, secretions, viscera, etc.
(ii) *via* infected wild or domestic animals, through tick bites. This route appears to be much rarer.

2.2 Occupational groups exposed

The disease mainly affects sheep and cattle farmers, abattoir workers, veterinary surgeons and laboratory staff working with the bacteria.

3. Clinical picture

3.1 Symptoms

In humans, the disease is most often benign and goes unnoticed. Less often it may cause the sudden onset of an intermittent fever accompanied by general signs. In the acute phase, acute febrile pneumonia and gastro-intestinal disorders may occur. Longer term complications include endocarditis and hepatic complications.

3.2 Biological diagnosis

Isolating the bacteria is difficult. Diagnosis is mainly based on serological tests for specific IgG and IgM antibodies.

Exposure criteria:

Minimum induction period: one week;

Maximum latent period: three weeks.

Erysipeloid

1. Definition and aetiological agent

Erysipeloid is caused by *Erysipelothrix rhusiopathiae*. The bacteria are found in a number of species of domestic and wild animals, particularly mammals, birds, and aquatic animals. Humans can be infected through direct contact. Infection is characterized by skin lesions and is generally benign.

2. Transmission of infection

2.1 Exposure

A number of species of mammals and birds carry *E. rhusiopathiae*. Pigs are the most commonly infected (swine erysipelas). Humans contract the disease through contact with carriers or sick animals, when handling products of animal origin or objects contaminated by the animals. The path of infection is often through wounds or skin abrasions.

2.2 Occupational groups exposed

Gamekeepers, foresters; farmers, veterinary surgeons, abattoir, tripe and meat-processing workers; pig, cattle, poultry, game and other farmers; fishermen and fish-market workers; processing and conserving of food products of animal origin.

3. Clinical picture

3.1 Symptoms

Most often erythematous and oedematous skin lesions on the hands and fingers, following a wound. They may be accompanied by problems in the joints but the development of the disease is usually benign. In exceptional cases, cardiac disorders and septicaemia have been observed.

3.2 Diagnosis

Diagnosis is basically clinical. It may be confirmed by isolating and identifying bacteria from the lesion.

Exposure criteria:

Minimum induction period: a few hours;

Maximum latent period: seven days.