Notifiable Australian zoonotic infections

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Zoonotic infection should be considered in any unwell patient with a history of exposure to animals, no matter how brief. Clinical features, diagnosis and treatment are outlined for eight zoonoses that are notifiable in Australia: anthrax, Australian bat lyssavirus infection, brucellosis, leptospirosis, psittacosis, Q fever, tularaemia and Hendra virus infection.

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Infectious diseases that are naturally transmitted from animals to humans (zoonoses) are responsible for approximately 60 to 70% of emerging human infectious diseases. New zoonoses that have emerged in Australia in the past decade include those caused by Australian bat lyssavirus, Hendra virus and Menangle virus. Many other zoonoses have emerged in other parts of the world, including severe acute respiratory syndrome, avian influenza and Middle Eastern respiratory syndrome coronavirus. The increasing ease and volume of international travel make it more likely that diseases such as these may reach Australia.

Zoonoses should be considered in the differential diagnosis in any unwell patient with a history of occupational or recreational exposure to animals. Animal contact may be brief, remote or forgotten, and patients should be asked specifically about occupation, hobbies, recreational activities, visits to farms or holiday homes and overseas travel. Certain occupations and recreational activities are classically associated with specific zoonoses, and knowledge of these associations is helpful when establishing a differential diagnosis.

Seven zoonoses are nationally notifiable in Australia and are reviewed here in an Australian context (Table 1). The most commonly reported of these is Q fever, with more than 300 cases notified in 2010, followed by leptospirosis, psittacosis and brucellosis.[1] Other notifiable zoonoses include anthrax, Australian bat lyssavirus (and other lyssaviruses) infection and tularaemia. In addition, Hendra virus infection is notifiable in both NSW and Queensland.

There are many other clinically important but non-notifiable zoonoses in Australia, and some of those most likely to be encountered in general practice are outlined at the end of this article.

ANTHRAX

Anthrax is caused by the spore-forming bacterium *Bacillus anthracis*. Spores can persist for long periods in the environment and are found in soil around the world. Animal infection typically occurs in cattle, sheep and goats that ingest spores during grazing. In Australia, anthrax occurs only sporadically in animals.[1] Human infection occurs after contact with contaminated animals or animal products.[2] Interest in anthrax has increased in the past decade because of its potential as a biological weapon.

Three anthrax syndromes are classically described: cutaneous, gastrointestinal and inhalational (Table 2). Recently, a fourth syndrome has emerged, known as injectional anthrax and characterised by severe soft tissue infections in injecting drug users.[2] Cutaneous anthrax accounts for about 95% of clinical cases globally and is the only form ever recorded in Australia (Figure 1). Three human cases were reported between 2000 and 2010.[1] Although fatalities in cutaneous anthrax are relatively uncommon, the case fatality rate in untreated gastrointestinal and inhalational anthrax can approach 100%.[2]

TABLE 1. NOTIFIABLE ZOONOSES IN AUSTRALIA*

Disease (causative organism)	Reservoirs in Australia	Risk groups	Incubation period	Mode of transmission	Human vaccine	Treatment†
Anthrax (*Bacillus anthracis*)	Cattle, sheep, goats	Veterinarians, agricultural, wildlife and livestock workers, handlers of processed hides, laboratory workers	1 to 60 days	Contact with contaminated animals or animal products, ingestion of contaminated food	Not available in Australia	Cutaneous: antibiotic choice depends on susceptibilities, treat for total of 60 days Severe forms: seek expert advice
Australian bat lyssavirus infection	Insectivorous bats, fruit bats (also known as flying foxes)	Veterinarians, animal handlers	<1 week to >1 year	Bite or scratch from infected bat	Rabies vaccine (PreP or PEP)‡	Wash wound with soap and water, PEP with HRIG and rabies vaccine (see flowchart on page 61)
Brucellosis (*Brucella* spp.)	Feral pigs	Feral pig hunters, laboratory workers	Weeks to months (average 14 to 21 days)	Consumption of unpasteurised dairy products, direct contact with infected animal secretions, inhalation	No vaccine	Doxycycline 100 mg orally 12-hourly for 6 weeks plus gentamicin 4 to 6 mg/kg intravenously initially, then according to blood levels for 7 days
Leptospirosis (*Leptospira* spp.)	Rats, small marsupials, cattle, pigs, horses	Banana and sugarcane workers, livestock workers, park rangers	2 to 30 days (average 7 to 10 days)	Direct contact with infected animal urine or tissues, contact with contaminated water or soil	Not available in Australia	Doxycycline 100 mg orally 12-hourly for 5 to 7 days
Psittacosis (*Chlamydophila psittaci*)	Birds	Bird owners, pet shop employees, veterinarians, poultry-processing workers, taxidermists	4 to 28 days (average 5 to 14 days)	Inhalation of dust containing dried faeces or secretions from infected birds, direct bird contact	No vaccine	Doxycycline 200 mg orally initial dose, followed by 100 mg daily for 14 days

Diagnosis

The microbiological laboratory should be contacted for advice on collection, preparation and transport of specimens when anthrax is suspected.[3] The clinical context should be provided along with the specimen. The diagnosis of anthrax is usually made on culture, with confirmation by immunohistochemistry or polymerase chain reaction (PCR) testing. *B. anthracis* can be isolated from numerous clinical specimens, including blood, skin lesion exudates, sputum and faeces.[3]

Management

Patients with inhalational, gastrointestinal and injectional anthrax require complex management and should be referred immediately to a specialist tertiary hospital. Those with cutaneous anthrax can be managed in an outpatient setting in conjunction with specialist advice. Antibiotic treatment is recommended to decrease the likelihood of systemic disease, and a prolonged course of oral antibiotics is recommended.[4]

Strategies for preventing anthrax in workers in high-risk occupations include sterilisation of at-risk products and the use of protective clothing. An anthrax vaccine for livestock is available in Australia. When cases of anthrax are confirmed in animals, management includes vaccination and quarantining, burial of carcasses and disinfection of contaminated sites. No human anthrax vaccine is currently registered in Australia, but vaccines made and approved for use in the USA and UK may be imported for use in Australia under exceptional circumstances.[3]

TABLE 1. NOTIFIABLE ZOONOSES IN AUSTRALIA* continued

Disease (causative organism)	Reservoirs in Australia	Risk groups	Incubation period	Mode of transmission	Human vaccine	Treatment†
Q fever (*Coxiella burnetii*)	Cattle, sheep, goats, marsupials, cats, dogs	Abattoir workers, cattle, livestock and dairy farmers, farm workers, shearers, veterinarians, animal transporters	14 to 60 days (average 14 to 21 days)	Inhalation or direct contact with dust, aerosols or animal products	Q-Vax should be considered in at-risk adults§	Acute infection: doxycycline 100 mg orally 12-hourly for 14 days Chronic disease: seek expert advice
Tularaemia (*Francisella tularensis*)	Small mammals (e.g. possums)	Laboratory workers, farmers, veterinarians, bushwalkers	3 to 8 days (average 3 to 5 days)	Contact with infected animals, arthropod bites (e.g. ticks), ingestion or inhalation of contaminated products	No vaccine	Gentamicin 4 to 6 mg/kg initially (severe sepsis 7 mg/kg), then dose according to blood levels for 10 days
Hendra virus infection (notifiable only in NSW and Queensland)	Horses, fruit bats (flying foxes)¶	Horse handlers, trainers, farmers	5 to 21 days	Exposure to infected horse secretions, tissue or body fluids	No vaccine	No specific treatment

ABBREVIATIONS: HRIG = human rabies immunoglobulin; PEP = post-exposure prophylaxis; PreP = pre-exposure prophylaxis.

* Annual reports on nationally notifiable diseases, summarising data collected by the National Notifiable Diseases Surveillance System, appear in *Communicable Diseases Intelligence* (www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi3601a15.htm).

† Treatments listed here are first-line regimens for nonpregnant adults. More details, including treatment options for children and pregnant women, can be found in *Therapeutic Guidelines: antibiotic. Version 14*.[4]

‡ For detailed information about PreP and PEP for Australian bat lyssavirus, see *Australian Immunisation Handbook. 10th ed*.[7]

§ Pre-vaccination screening with both *Coxiella burnetii* serology and intradermal skin testing is required to exclude previous exposure to the organism, as these patients may develop significant adverse reactions to the vaccine.

¶ Although fruit bats have been identified as the primary host for Hendra virus, there has been no evidence of bat-to-human transmission.

AUSTRALIAN BAT LYSSAVIRUS AND RABIES

Rabies is a widespread zoonotic disease that is responsible for more than 25,000 deaths per year worldwide.[5] It is present in all continents except Antarctica, with more than 95% of cases seen in Africa and Asia.[5] Australian bat lyssavirus (ABLV) is one of 12 known species within the genus *Lyssavirus*, and is the only lyssavirus known to occur in Australia.[6,7] As the clinical disease caused by rabies virus is indistinguishable from that caused by other lyssaviruses, the term 'rabies' is used to refer to disease caused by any of the known lyssavirus species.

Dogs are responsible for transmission in more than 99% of human deaths from lyssaviruses worldwide and are the dominant reservoir in Africa and Asia.[5] Other mammals are potential reservoirs, particularly bats, cats and monkeys. ABLV has only been identified in fruit bats (flying foxes) and insectivorous bats, and it is assumed that any bat in Australia could potentially carry the virus.[7] Since the discovery of ABLV in 1996, three human cases of ABLV infection have been reported, all of which occurred after close contact with an infected bat. All three patients presented with a rabies-like illness, which was fatal in all cases.

The incubation period of lyssavirus infection in humans is highly variable. The clinical illness typically occurs in two phases: a prodromal phase lasting up to 10 days with nonspecific symptoms such as fever, headache, myalgia, nausea and anorexia; and a central nervous system (CNS) phase where a range of neurological

TABLE 2. ANTHRAX: THE CLASSIC CLINICAL SYNDROMES

Clinical syndrome	Mode of transmission	Clinical features	Incubation period
Cutaneous	Introduction of pathogenic spores through a cut or abrasion	Skin lesion with central necrosis (eschar) surrounded by oedema (see Figure 1), ± fever, regional lymphadenopathy	1 to 14 days (average 3 to 5 days)
Gastrointestinal	Ingestion of contaminated food (typically under-cooked meat)	Oropharyngeal ulceration, regional lymphadenopathy, oedema, fever, nausea and vomiting, bloody diarrhoea, prostration and shock	1 to 10 days (average 2 to 5 days)
Inhalational	Inhalation of anthrax spores	Flu-like symptoms initially, rapidly progressive fever, dyspnoea, diaphoresis and shock; widened mediastinum on chest x-ray	1 to 60 days (average 4 to 6 days)

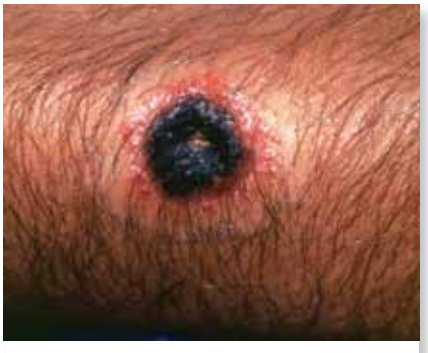


Figure 1. Cutaneous anthrax lesion showing central necrosis (eschar) and surrounding oedema.

and psychological symptoms may be seen. Local paraesthesia at or near the site of the wound may be present early in the illness. Classical signs in late disease include aerophobia, hydrophobia, autonomic dysfunction (e.g. hypersalivation), agitation and alternating periods of lucidity and confusion. MRI may show features of encephalitis.[5] Any patient with symptoms suggestive of rabies should be transferred to hospital immediately.

Diagnosis

The diagnosis can be made by identification of viral antigen in neural tissue, skin snips (from the nape of the neck), saliva or cerebrospinal fluid (CSF); or from isolation of viral RNA from saliva, CSF or CNS tissue.[4] In unvaccinated people, antibody detection from serum or CSF can also be diagnostic.[4] Rabies is almost universally fatal in unvaccinated patients.

Management

Rabies vaccination is effective against ABLV, and appropriate timely post-exposure prophylaxis (PEP) with rabies vaccine prevents clinical disease.[6] PEP for ABLV and rabies virus includes wound management plus administration of vaccine and human rabies immunoglobulin (HRIG). Local treatment should be undertaken as soon as possible after injury and involves thorough washing of the wound with soap and water, followed by application of a virucidal preparation such as povidone–iodine solution.[5,7] Rabies vaccine should be administered, with or without HRIG, depending on the category and source of exposure (see the box and flowchart on page 61).

Pre-exposure (PreP) rabies vaccination is also available in Australia. The recommended schedule consists of three doses of vaccine, given intramuscularly on days 0, 7 and 21 to 28. PreP is recommended for those at risk of bites or scratches from Australian bats, such as veterinarians, bat handlers and wildlife officers, and should be considered for travellers and expatriates who will spend time in rabies-enzootic areas.[7]

Post-exposure treatment for Australian bat lyssavirus and rabies exposures is available free across Australia. National guidelines on rabies and other lyssavirus exposures and infections can be found on the Australian Government Department of Health and Ageing website (www.health.gov.au/internet/main/publishing.nsf/Content/cdna-song-abvl-rabies.htm). Each state or territory has its own system for disease notification and supply of rabies vaccine and HRIG (see Table 3 for contact details).

BRUCELLOSIS

Brucellosis, caused by bacteria of the genus *Brucella*, is one of the most common zoonotic infections globally. Most human cases are caused by the three species *Brucella abortus*, *B. melitensis* and *B. suis*.[8] Animal reservoirs include pigs, cattle, sheep and goats. Infected animals excrete large numbers of organisms in body fluids, and brucellosis can be transmitted to humans through direct contact of broken skin with infected animal secretions and carcasses, ingestion of unpasteurised milk and dairy products or inhalation of infected aerosols.[8,9] Laboratory-acquired infections can occur in individuals handling cultures from patients with unrecognised brucellosis.

As B. abortus was eradicated from Australian cattle herds in 1989, and *B. melitensis* has never been reported in Australian sheep or goats, all human infections with these species in Australia are related to overseas travel.[1] *B. suis* is the only brucellosis species reported in Australia, mainly in feral pigs in Queensland. In 2010,

LYSSAVIRUS EXPOSURE CATEGORIES*

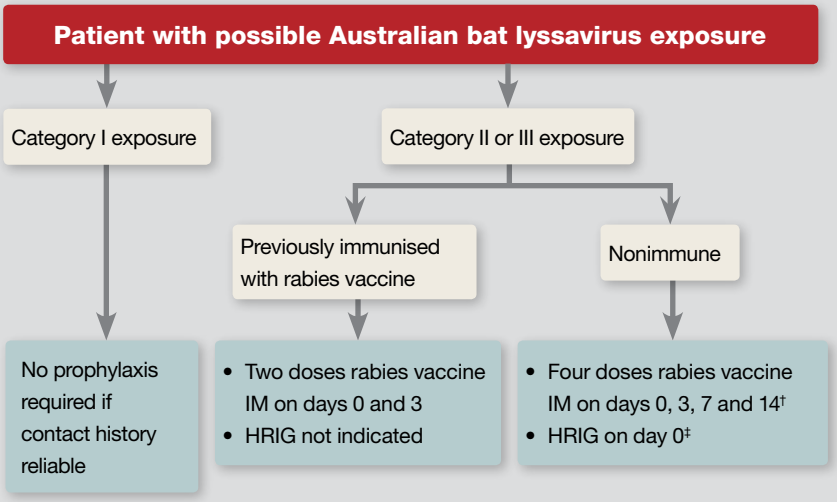
Category I: Touching or feeding animals, licks on intact skin, contact of intact skin with blood, urine or faeces or contact with an animal that has been dead for longer than four hours

Category II: Nibbling of uncovered skin, minor scratches or abrasions without bleeding

Category III: Single or multiple transdermal bites or scratches, licks on broken skin, contamination of mucous membranes with saliva from licks

* Modified from World Health Organization (WHO). WHO expert consultation on rabies: second report. (WHO technical report series ; no. 982). Geneva: WHO; 2013.[5]

VACCINE AND HRIG USE AFTER POTENTIAL EXPOSURE TO AUSTRALIAN BAT LYSSAVIRUS*



ABBREVIATIONS: HRIG = human rabies immunoglobulin. IM = intramuscular.

* Modified from Australian Technical Advisory Group on Immunisation, Australian Government Department of Health and Ageing. Australian immunisation handbook. 10th ed. Canberra: Australian Government; 2013.[7]

† The recommended number of post-exposure doses of rabies vaccine for Australian bat lyssavirus was reduced from five doses to four doses in the 10th edition of the *Australian Immunisation Handbook* (2013).[7]

‡ When HRIG is indicated, a dose of 20 IU/kg should be administered, with as much as possible infiltrated in and around the wounds, and any remainder injected intramuscularly at a site away from the rabies vaccine injection site. HRIG should be given only to patients with no documented history of previous vaccination, or those who received their first dose of vaccine within the previous seven days.

there were 21 notified cases of brucellosis in Australia, 16 of which occurred in Queensland.[1] The most common risk factor for human *B. suis* infection in Australia is exposure to infected feral pigs through recreational or occupational hunting or butchering.[1,10]

Brucellosis is a systemic bacterial illness with a wide spectrum of clinical manifestations and may present as an acute, subacute or chronic infection. The most common symptom is fever, seen in more than 90% of patients.[10] Arthralgia, myalgia, back pain, sweats, chills, fatigue, headache and malaise are frequently reported. Hepatomegaly and splenomegaly are common, and lymphadenopathy is seen in approximately 10% of patients. Complications can arise in almost any organ system: osteoarticular manifestations and epididymo-orchitis are the most common. Endocarditis is uncommon (1% of patients), but is the most serious complication and accounts for most of the 5% mortality rate of human brucellosis.[8] Risk of complications is increased when there is a delay in diagnosis. Common laboratory abnormalities include anaemia, leukopenia, thrombocytopenia and elevation of C-reactive protein (CRP) level and erythrocyte sedimentation rate (ESR).[8,9]

Diagnosis

Diagnosis is by culture of *Brucella* species from clinical specimens. Sensitivity of blood cultures ranges from 30 to 90% depending on the timing and administration of antibiotics, and blood cultures are frequently positive in uncomplicated relapsing disease.[8] *Brucella* species can also be cultured from pus, tissue samples, bone marrow, CSF and pleural, joint or ascitic fluid. Serological tests are useful in patients who do not live in endemic areas.

Management

Management of brucellosis usually requires specialist input. Australian guidelines recommend combination antibiotic therapy for at least six weeks.[4] Relapse is common (up to 10% of patients who complete six weeks of treatment), and patients with relapsed disease or organ complications may require a longer duration of therapy and consideration of alternative combination regimens.[4,9]

Those at risk of brucellosis, including hunters and butcherers of feral pigs, can take precautions to prevent infection. These include covering all cuts or abrasions with waterproof dressings, wearing gloves, overalls and eye protection when slaughtering animals or handling carcasses, thorough hand washing and use of disinfectant.[11]

LEPTOSPIROSIS

Leptospirosis is one of the most widespread zoonotic diseases in the world. It is caused by pathogenic spirochaetes of the genus *Leptospira*. Animal reservoirs include rats, bandicoots, possums, kangaroos and farm animals such as dairy cows.[12] Infected animals chronically secrete *Leptospira* organisms in their urine and contaminate the environment. Human infection occurs through direct contact with infected animal urine or tissues, or indirectly through contact of broken skin or mucous membranes with contaminated water or soil. At-risk groups in Australia include agricultural (banana and sugarcane) and livestock

TABLE 3. DISEASE NOTIFICATIONS AND SUPPLY OF LYSSAVIRUS POST-EXPOSURE PROPHYLAXIS IN AUSTRALIA: CONTACT DETAILS

State/ territory	Department	Phone number, website
ACT	Communicable Disease Control (CDC), Health Protection Service, ACT Government	02 6205 2155, 02 9962 4155 (24-hour emergency line) www.health.act.gov.au/health-services/population-health/health-protection-service/communicable-diseases
NSW	Public Health Units, NSW Health	1300 066 055 www.health.nsw.gov.au/Infectious/Pages/default.aspx
NT	Centre for Disease Control, Department of Health, NT Government	08 8922 8044 www.health.nt.gov.au/Centre_for_Disease_Control/Notifiable_Diseases/index.aspx
QLD	Public Health Units, Queensland Health	1343 2584 or individual public health units www.health.qld.gov.au/publichealthact/notifiable
SA	Communicable Disease Control Branch, SA Health	1300 232 272 www.sahealth.sa.gov.au/wps/wcm/connect/public+content/sa+health+internet/clinical+resources/health+notifications/notifiable+disease+reporting/notifiable+disease+reporting
TAS	Communicable Diseases Prevention Unit, Department of Health and Human Services, Tasmania	1800 671 738 www.dhhs.tas.gov.au/peh/infectious_diseases
VIC	Communicable Disease Prevention and Control Unit, Department of Health, Victoria	1300 651 160 http://ideas.health.vic.gov.au/notifying.asp www.health.vic.gov.au/immunisation/vaccine-order-forms.htm
WA	Communicable Disease Control (CDC) Directorate or Metropolitan and Regional Population Health Units	08 9328 0553 (CDC Directorate) or individual population health units www.health.wa.gov.au/circularsnew/circular.cfm?Circ_ID=12573 www.public.health.wa.gov.au/3/284/3/notifiable-comm.pm

workers and park rangers. In recent years, recreational exposures have been recognised as a common source of infection, especially in international travellers.[13]

In Australia, leptospirosis is most commonly seen in central west and far north Queensland, and to a lesser degree in New South Wales and Victoria.[1] In 2010, a total of 131 cases of leptospirosis were reported in Australia, with Queensland accounting for more than 60% of these notifications.[1] Disease incidence peaks in summer and autumn in temperate climates, and during the rainy season in the tropics as heavy rains and flooding provide ideal conditions for disease transmission.

Leptospirosis infection is frequently subclinical, and most symptomatic patients present with a self-limiting anicteric febrile illness. Common symptoms include fever, chills, headache and myalgia. Weil's disease (icteric leptospirosis), a severe form of leptospirosis, is classically associated with *Leptospira icterohaemorrhagiae* infection and is rare in Australia. Features include jaundice, renal failure and pulmonary haemorrhage.[14]

Diagnosis

In Australia, most leptospirosis cases are diagnosed on the basis of serology. Microscopic agglutination testing is the reference standard and should be performed on paired acute and convalescent serum samples where possible.[14] PCR of serum samples can enable earlier diagnosis, before development of antibodies.[15] Culture of blood and CSF can be attempted but samples must be transported at room temperature, specialised culture media are required and results take several weeks. Blood cultures are most likely to be positive if samples are taken during the first 10 days of illness, whereas urinary excretion is seen from the second week onwards and may continue for many weeks.

Management

Patients with severe illness require inpatient management, preferably in a tertiary hospital. For patients with mild disease, doxycycline is the drug of choice, and treatment in consultation with an infectious diseases physician is recommended.

At-risk groups should be educated on minimising exposure through the use of protective clothing (e.g. fully covered shoes and gloves), hand washing and covering cuts and grazes with waterproof dressings. Previous leptospirosis exposure does not provide cross-protective immunity, and re-infection with other serovars is possible. Chemoprophylaxis with doxycycline (200 mg weekly) can be given to people with sporadic exposure to high-risk environments, such as travellers, but the benefit is unclear.[13]

PSITTACOSIS

Psittacosis (also known as ornithosis and parrot fever) is a systemic zoonosis caused by *Chlamydophila psittaci*. The major zoonotic reservoir is birds, including budgerigars, cockatoos, parakeets, chickens, seabirds, waterfowl, pigeons and doves.[16] Infected birds are typically asymptomatic carriers, but some can develop clinical illness, with ruffled feathers, anorexia, nasal discharge, diarrhoea and conjunctivitis.[16,17] Human infection with these bacteria occurs through direct bird contact or inhalation of aerosolised organisms in dried faeces or secretions from infected birds.

Notification rates of psittacosis in Australia are relatively low, and in 2010, 56 cases were reported, most in NSW and Victoria.[1] The disease is seen mainly in adults, with a median patient age of 54 years reported over the past five years.[1]

Occupational or recreational exposure to birds is the main risk factor for psittacosis. Pet bird ownership is common in Australia (about 15% of the population).[17] Although most cases are sporadic, outbreaks can occur, mostly related to infection in commercial flocks. A history of bird contact is not always elicited, and outbreaks involving wild birds have been reported where activities such as gardening and lawn mowing were risk factors.[16]

Clinical illness in humans ranges from asymptomatic infection to fulminant sepsis with multiorgan failure, which may occasionally be fatal despite appropriate treatment.[16,17] Common presenting symptoms include fever, headache, myalgias, diaphoresis and cough, and onset may be abrupt.[16] Headache may be prominent and severe and in about 10% of patients, may be associated with neck stiffness, photophobia and altered consciousness.[16] Fever and abnormalities on chest examination are common, and chest x-ray is abnormal in approximately 80% of patients, most frequently with consolidation affecting a single lobe of the lungs (Figure 2).[16] Non-specific findings on initial blood tests include toxic granulation or 'left shift' in neutrophils without marked neutrophilia, deranged liver enzymes and elevated CRP level. Fulminant psittacosis with multiorgan failure is uncommon but can lead to adult respiratory distress syndrome, renal failure, disseminated intravascular coagulation and haemophagocytic syndrome.[16] Other uncommon complications of psittacosis include encephalitis, endocarditis, myocarditis and reactive arthritis.

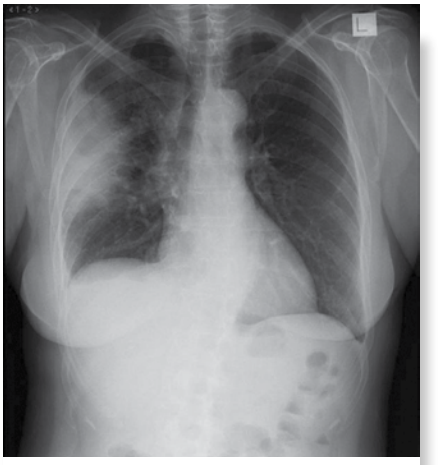


Figure 2. Chest x-ray in a woman with psittacosis showing focal consolidation in the right upper lobe of the lungs. The patient presented with progressive respiratory failure one week after exposure to an unwell native bird and subsequently required intensive care admission. She was treated with intravenous azithromycin followed by oral doxycyline, and made a complete recovery after several weeks.

Diagnosis

Diagnosis of psittacosis is generally serological, based on either a fourfold rise in antibody titre in paired acute and convalescent phase sera (taken two weeks apart), or a single high IgM acute phase titre in the setting of a clinically compatible illness.[16,17] Culture of the organism from respiratory secretions is generally not attempted because of the danger to laboratory workers. PCR techniques can be used but are not routinely available outside reference laboratories.

Management

Doxycycline is the antibiotic of choice for psittacosis and treatment duration is usually two weeks.[4] Macrolides such as clarithromycin, azithromycin and roxithromycin have good in vitro activity and are appropriate agents in pregnant women and children up to the age of eight years.[16]

Infected birds should be treated with antibiotics under veterinary guidance. Cleaning of potentially contaminated areas is necessary, as *C. psittaci* can persist in the environment. The use of protective equipment while undertaking this process, including gloves, an N95 mask, eyewear and a surgical cap, is recommended.

Q FEVER

Q fever (short for 'query' fever) is caused by the bacterium *Coxiella burnetii*. It was first described in Australian abattoir workers in the 1930s, but is now recognised to occur in most places in the world except New Zealand.[18,19] The primary reservoirs in Australia are cattle, sheep and goats, although many mammalian hosts can become infected.

C. burnetii is extremely infectious, and very few organisms are required to cause disease. Infection occurs via inhalation of contaminated dust, or direct contact with infected animals, infected tissues or excreta, particularly placental tissue and birth fluids.[18] The at-risk populations for Q fever include abattoir workers, cattle and livestock farmers, dairy farmers, shearers, farm workers, veterinarians and animal transporters.[18] Occasionally, people without direct animal contact can be infected via inhalation. Rates of notified cases in

TABLE 4. COMMON CLINICALLY SIGNIFICANT NON-NOTIFIABLE ZOONOSES IN AUSTRALIA

Zoonosis	Causative organism	Animal reservoirs	Risk groups
Cat scratch disease	*Bartonella henselae*	Cats (particularly kittens)	Cat owners
Fisk tank granuloma (see Figure 3)	*Mycobacterium marinum*	Fresh and salt water	Pet fish owners
Hydatid disease	*Echinococcus granulosus*	Dogs, dingoes, foxes (definitive hosts), sheep, wallabies, kangaroos (intermediate hosts)	Farmers, rural residents
Toxocariasis	*Toxocara canis/cati*	Dogs, cats	Children
Toxoplasmosis	*Toxoplasma gondii*	Cats (definitive host), farm animals, mice, birds, lizards (intermediate hosts)	Cat owners, immunocompromised individuals

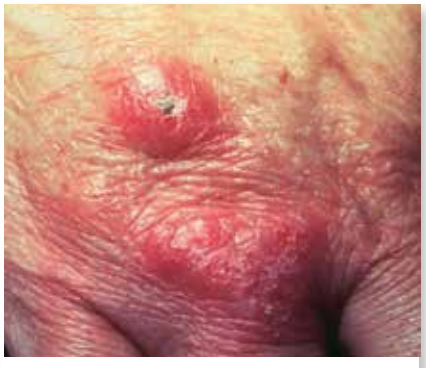


Figure 3. Nodular lesions of fish tank granuloma (caused by *Mycobacterium marinum*). The diagnosis was confirmed by skin biopsy and mycobacterial PCR, and the patient was treated with doxycyline 100 mg twice daily for three months, with complete resolution.

Australia have dropped significantly since the introduction of vaccination for at-risk groups in 2001; in 2010, 323 cases were notified nationwide.[1]

A detailed review of the clinical features of Q fever, and its investigation, diagnosis and management can be found in the April 2013 issue of *Medicine Today*.[18]

TULARAEMIA

Tularaemia is a zoonosis that affects a wide range of wildlife species, including mammals, birds and arthropods.[20] It is caused by the bacterium *Francisella tularensis*, with three subspecies recognised in human disease: *tularensis*, *holarctica* and *novicida*. Modes of transmission for tularaemia include arthropod bites, bites from infected animals, inhalation of infectious aerosols and exposure to contaminated food or water. Clinical presentation depends on route of transmission and two syndromes are typically described: ulceroglandular (with skin and lymph node involvement; about 75% of patients) and typhoidal. Only two cases of tularaemia have been reported in Australia in the past 10 years, both caused by *F. tularensis* subspecies *holarctica*. Both cases were of the ulceroglandular type and were reported in Tasmanian residents who were bitten by possums.[20]

HENDRA VIRUS INFECTION

Hendra virus was first isolated in 1994, following an outbreak in 21 horses and two humans in the Brisbane suburb of Hendra.[21] Fruit bats (flying foxes) have been identified as the natural reservoir of Hendra virus, but there is no evidence of bat-to-human transmission. All human cases have been related to direct close contact with infected horses, including autopsy.[22] Human-to-human transmission has not been reported. Seven human cases of Hendra virus infection have been recorded in Australia, with four deaths.

Clinical illness in humans typically begins with an influenza-like illness, which may progress to encephalitis or pneumonitis with multiorgan failure.[22] Diagnostic methods include virus isolation, immunohistochemistry, serology and PCR testing. There is no specific treatment for Hendra virus infection, and no human vaccine is currently available.

NON-NOTIFIABLE ZOONOSES

Many non-notifiable zoonoses are also clinically important. Some of those most likely to be encountered in Australian general practice are outlined in Table 4.

CONCLUSION

Zoonoses are an important group of diseases that should be considered in the differential diagnosis in any unwell patient with a history of occupational or recreational exposure to animals. The risk of transmission from animals to humans can be reduced by preventing disease in animals and using precautions when dealing with potentially contaminated animal secretions or products. MT

REFERENCES

A list of references is included in the website version (www.medicinetoday.com.au) and the iPad app version of this article.

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Notifiable Australian zoonotic infections

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REFERENCES

1. NNDSS Annual Report Writing Group; Milton A, Stirzaker S, Trungove M, et al. Australia's notifiable disease status, 2010: annual report of the National Notifiable Diseases Surveillance System. Commun Dis Intell Q Rep 2012; 36: 1-69. Available online at: www.health.gov.au/internet/main/publishing.nsf/Content/cda-cdi3601a15.htm (accessed September 2013).
2. Hicks CW, Sweeney DA, Cui X, et al. An overview of anthrax infection including the recently identified form of disease in injection drug users. Intensive Care Med 2012; 38: 1092-1104.
3. Australian Government Department of Health and Ageing. Anthrax: public health response plan for Australia. 2nd ed. Canberra: Commonwealth of Australia; 2012. Available online at: www.health.gov.au/internet/publications/publishing.nsf/Content/ohp-anthrax-toc (accessed September 2013).
4. Antibiotic Expert Group. Therapeutic guidelines: antibiotic. Version 14. Melbourne: Therapeutic Guidelines Limited; 2010.
5. World Health Organization (WHO). WHO expert consultation on rabies: second report. (WHO technical report series; no. 982). Geneva: WHO; 2013. Available online at: www.who.int/rabies/en (accessed September 2013).
6. Calisher CH, Ellison JA. The other rabies viruses: the emergence and importance of lyssaviruses from bats and other vertebrates. Travel Med Infect Dis 2012; 10: 69-79.
7. Australian Technical Advisory Group on Immunisation, Australian Government Department of Health and Ageing. Australian immunisation handbook. 10th ed. Canberra: Australian Government; 2013. Available online at: www.health.gov.au/internet/immunise/publishing.nsf/Content/Handbook10-home (accessed September 2013).
8. Franco MP, Mulder M, Gilman RH, Smits HL. Human brucellosis. Lancet Infect Dis 2007; 7: 775-786.
9. Dean AS, Crump L, Greter H, et al. Clinical manifestations of human brucellosis: a systematic review and meta-analysis. PLoS Negl Trop Dis 2012; 6: e1929.
10. Robson JM, Harrison MW, Wood RN, et al. Brucellosis: re-emergence and changing epidemiology in Queensland. Med J Aust 1993; 159: 153-158.
11. Massey PD, Polkinghorne BG, Durrheim DN, Lower T, Speare R. Blood, guts and knife cuts: reducing the risk of swine brucellosis in feral pig hunters in north-west New South Wales, Australia. Rural Remote Health 2011; 11: 1793.
12. Slack A. Leptospirosis. Aust Fam Physician 2010; 39: 495-498.
13. Leshem E, Meltzer E, Schwartz E. Travel-associated zoonotic bacterial diseases. Curr Opin Infect Dis 2001; 24: 457-463.
14. Lim VKE. Leptospirosis: a re-emerging infection. Malaysian J Pathol 2011; 33: 1-5.
15. WHO/FAO/OIE Collaborating Centre for Reference and Research on Leptospirosis. National leptospirosis surveillance report number 20. Brisbane: Queensland Government; 2011. Available online at: www.health.qld.gov.au/qhcss/qhss/lepto/documents/11-surveillance-report.pdf (accessed September 2013).
16. Stewardson AJ, Grayson ML. Psittacosis. Infect Dis Clin N Am 2010; 24: 7-25.
17. Gorman J, Cook A, Ferguson C, et al. Pet birds and risk of respiratory disease in Australia: a review. Aust N Z J Public Health 2009; 33: 167-172.
18. Chaudhuri A, Robson J. Q fever queries and answers. Med Today 2013; 14(4): 54-59.
19. Raoult D, Marrie TJ, Mege JL. Natural history and pathophysiology of Q fever. Lancet Infect Dis 2005; 5: 219-226.
20. Jackson J, McGregor A, Cooley L, et al. Francisella tularensis subspecies holarctica, Tasmania, Australia, 2011. Emerg Infect Dis 2012; 18: 1484-1486.
21. Murray K, Selleck P, Hooper P, et al. A morbillivirus that caused fatal disease in horses and humans. Science 1995; 268: 94-97.
22. Mahalingam S, Herrero LJ, Playford EG, et al. Hendra virus: an emerging paramyxovirus in Australia. Lancet Infect Dis 2012; 12: 799-807.