Pet-related infections
what can you catch from your pet?

Pet-related infections range from self-limited skin conditions to life-threatening systemic illnesses. Some of the more frequent or serious infections are reviewed here.

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In the most recent survey of pet ownership in Australia (in 1994), there were just over 20 million people and 17.5 million pets living in this country. Two in every five households had a dog (3.1 million dogs in total) and one in every four households had a cat (2.5 million cats in total). There were also 6 million pet birds and 4.3 million pet fish.[1] As infections in these pets may occasionally spread to their owners, the taking of a 'pet history' can give medical practitioners important clues to the diagnosis of a variety of human infections.

This article reviews the clinical presentation, diagnosis and management of the common human infections that are related to pet animals. A more extensive list of pet-related infections, including those involving exotic and rural pets, is provided in Table 1.

Bacterial pet-related infections

Infections from dog and cat bites

Animal bite wounds are very common, and despite the fact that most (80%) bite wounds do not require medical care,[2] bites account for about 1% of hospital emergency department visits in Australia and the USA.[3,4] Dogs are responsible for about 80% of animal bite wounds, of which 2 to 20% become infected.[2,5] The incidence of infection, however, is up to 50% following a cat bite.[2] Cat bites are also more likely to cause serious infection as they are more likely than dog bites to penetrate into bones and joints.

IN SUMMARY

- **A history of contact with pet animals should be sought whenever there is an unusual manifestation of infection.**
- **Transmission of infections from pets to humans can be reduced by preventing disease in animals, treating unwell pets, not allowing pets access to beaches and playgrounds, removing animal faeces from backyards and public areas, washing hands after animal contact, and appropriately managing animal bites.**
- **Antibiotics are indicated for all cat bites, dog bites at high risk for infection and infected bites.**
- **Avoidance of toxoplasmosis is particularly important in pregnant women as congenital infection may result in fetal death, neurological impairment, chorioretinitis or cytopenias. Prevention includes avoidance of cat faeces or anything possibly contaminated with cat faeces, such as kitty litter, soil and raw vegetables, and not eating undercooked meat that could contain tissue cysts.**
- **Specific antimicrobial therapy is generally not indicated for toxoplasmosis. Exceptions include infections in immunocompromised patients, disseminated disease in immunocompetent patients, infection during pregnancy, and cases of chorioretinitis or congenital toxoplasmosis.**
- **Although there is no rabies in Australia, travellers should be aware that domestic dogs are the main reservoir worldwide of the rabies virus.**

continued

Table 1. Pet-related infections

Organism	Animals involved	Clinical disease in humans
Bacteria		
Pasteurella multocida	Cats, dogs	Skin, joint and bone infections, bacteraemia – from cat bites and scratches and dog bites
Capnocytophaga canimorsus	Dogs	Skin infection, bacteraemia, meningitis (especially in patients with asplenia) – from dog bites
Bartonella henselae	Cats	Cat scratch disease – lymphadenopathy
Chlamydophila psittaci	Birds	Psittacosis – atypical community-acquired pneumonia
Salmonella spp.	Snakes, lizards, dogs	Diarrhoea
Campylobacter spp.	Snakes, lizards, dogs, chickens	Diarrhoea
Mycobacterium marinum	Fish	Skin infection
Streptobacillis moniliformis	Rodents	Rat bite fever – skin infection, bacteraemia
Yersinia pestis	Cats, dogs	Bubonic (lymphadenopathy) or pneumonic plague – from cat and dog fleas
Yersinia enterocolitica and *Y. paratuberculosis*	Cats, dogs, rodents	Diarrhoea, abdominal pain, fever
Leptospirosis spp.	Dogs, livestock	Fever, hepatitis, jaundice, meningitis
Coxiella burnetii	Cattle, sheep, goats	Q fever – fever, hepatitis, pneumonia
Brucella spp.	Dogs, pigs, cows, goats	Fever, hepatitis, arthritis, spondylitis, lymphadenopathy
Erysipelothrix rhusiopathiae	Fish, pigs, horses	Skin infection
Staphylococcus spp. (including MRSA)	Dogs, cats, pigs	Skin infection
Viruses		
Herpes B virus	Monkeys	Skin infection and encephalitis
Lymphocytic choriomeningitis virus	Rodents	Meningitis
Lyssavirus	Dogs, cats, bats, monkeys	Rabies – encephalitis
Parapoxvirus	Sheep	Orf – localised vesicular skin rash
Fungi		
Microsporum canis and *Trichophyton mentagrophytes*	Cats, dogs, horses and rodents	Tinea capitis, corporis and barbae
Sporothrix schenckii	Cats	Skin lesions
Parasites		
Toxoplasma gondii	Cats	Lymphadenopathy, chorioretinitis, congenital infection, reactivation
Toxocara cati and *T. canis*	Cats, dogs	Visceral larva migrans – fever, wheezing, eosinophilia
Dipylidium caninum	Dogs	Asymptomatic, nonspecific intestinal symptoms
Ancylostoma caninum and *A. braziliensis*	Dogs	Cutaneous larva migrans – skin lesions
Ancylostoma caninum	Dogs	Eosinophilic enteritis
Giardia lamblia	Cats, dogs, rodents	Diarrhoea
Cryptosporidium parvum	Cats, dogs, rodents, livestock	Diarrhoea
Echinococcus granulosus	Dogs	Hydatid cysts

Infections resulting from dog and cat bites, as well as cat scratches, are usually polymicrobial. Organisms include aerobic bacteria such as *Streptococcus* spp., *Staphylococcus* spp., *Pasteurella multocida* and *Capnocytophaga canimorsus*, and anaerobic bacteria such as *Actinomyces* spp., *Bacteroides* spp. and *Fusobacterium* spp.

Infection with *C. canimorsus*, a Gram-negative bacillus, following a dog bite or, less commonly, a cat bite may result in meningitis, endocarditis or bacteraemia with septic shock. Asplenic individuals are at particular risk of fulminant sepsis, with a fatality rate of 25%.[5] The organism is susceptible to most antibiotics, including penicillin.

P. multocida, a Gram-negative coccobacillus, is associated with more than 50% of infected cat bites.[2] Infection usually develops within hours of the bite, with acute onset of erythema, pain and swelling. Penicillin-based antibiotics such as amoxycillin and amoxycillin–clavulanic acid are the drugs of choice. Dicloxacillin, cephalexin, clindamycin and erythromycin have poor activity against this organism.

The management of animal bites is outlined in Table 2.[2,4,6,7] Antibiotics are indicated for all cat bites, dog bites at high risk for infection (Table 3) and infected bites. Amoxycillin–clavulanate 875 mg/125 mg twice daily is the oral antibiotic of choice. In the case of penicillin allergy, metronidazole (Flagyl, Metrogyl, Metronide) 400 mg twice daily with doxycycline 100 mg daily, sulfamethoxazole–trimethoprim (Bactrim, Resprim, Septrin) 160 mg/800 mg twice daily or ciprofloxacin 500 mg twice daily is recommended.[8] Patients who have not been fully immunised against tetanus (fewer than three doses received in a course) should receive tetanus immunoglobulin and tetanus vaccine (ADT Booster). Those who have been fully vaccinated but have not received a booster in the last five years should be given tetanus vaccine.

Cat scratch disease

Bartonella henselae, a Gram-negative coccobacillus, was identified as the cause of cat scratch disease (CSD) in 1992.[9] Cats are the main reservoir of this bacterium, and usually have asymptomatic bacteraemia. Kittens (less than 12 months of age) are more likely than older cats to be bacteraemic, and therefore are a greater risk of infection to humans. Although fleas are responsible for transmission between cats, humans are usually infected by a cat scratch or bite. Painful lymphadenopathy develops one to three weeks after a distal scratch and usually persists for two to three months. Suppuration of the lymph node

Table 2. Steps in the management of dog and cat bite wounds[2,4,6,7]

- Take the patient's history, including immunocompromise or any other predisposition to serious infection
- Examine the wound, including vascular, tendon and nerve integrity and involvement of joints or bones
- Culture clinically infected wounds – inform the laboratory of the nature of the wound as standard laboratory techniques may fail to isolate more unusual organisms
- Irrigate the wound with sterile normal saline
- X-ray the wound area when fracture, foreign body or joint penetration is suspected
- Debride devitalised issues, drain abscesses and remove foreign bodies
- Primary wound closure is generally not recommended, and is particularly not recommended for bites that are more than 24 hours old, on the hand or clinically infected; facial wounds may be an exception in order to avoid scarring
- Initiate antibiotic therapy for all cat bites, dog bites at high risk for infection (see Table 3) and infected dog bites
- If the patient has not been immunised for tetanus within the last five years, immunise with tetanus toxoid; also administer tetanus immunoglobulin if there is no history of primary vaccination (i.e. if a course of three doses of vaccine has never been received)
- Consider rabies vaccination if the bite occurred in an endemic region
- Instruct the patient regarding elevation and immobilisation of the injured area
- Follow up the patient within 24 hours or if signs of infection occur
- Refer the patient to hospital if any of the following are present: significant hand bites, severe cellulitis, evidence of systemic sepsis, infection unresponsive to oral antibiotics, penetration into joints/tendons, open fractures, airway compromise, immunocompromise or head injuries

Table 3. Dog bites considered at high risk for infection

- Wounds on the hand or wrist
- Deep puncture wounds
- Wounds requiring surgical debridement
- Wounds after primary closure
- Wounds near a prosthetic joint
- Wounds involving a limb with venous or lymphatic compromise
- Wounds in immunocompromised individuals, including those with alcoholism, cirrhosis, asplenia, diabetes mellitus, rheumatoid arthritis or undergoing corticosteroid therapy

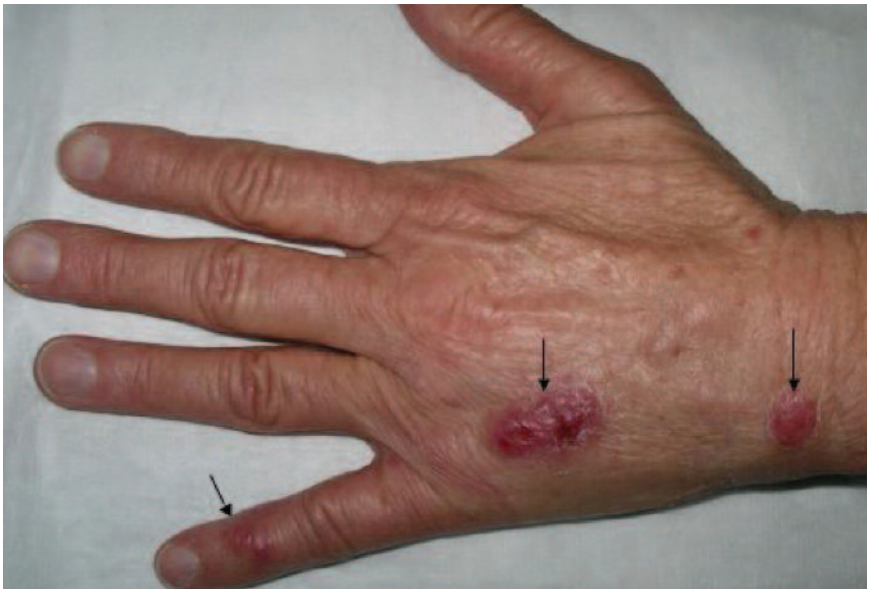


Figure 1. *Mycobacterium marinum* infection in a woman with a history of cleaning her fishpond. The initial lesion developed on the fifth finger, and subsequently spread up the hand (arrows) and along the arm.

may occur. Atypical features include prolonged fever, malaise, arthralgia, hepatosplenomegaly, encephalopathy, neuroretinitis and endocarditis. In immunocompromised individuals, *B. henselae* may also cause bacillary angiomatosis (vascular proliferation of the skin) and peliosis hepatis (vascular proliferation of sinusoidal hepatic capillaries).

Culture of *B. henselae* from lymph nodes or blood is rare. Molecular techniques for detection are more sensitive but may not be readily available. Demonstration of antibodies to the organism is the main diagnostic tool. Fine needle biopsy may be required to exclude malignancy or mycobacterial infection. Needle aspiration is recommended if suppuration occurs.

Most cases in immunocompetent individuals are self-limited, with antibiotics not affecting the natural history of disease. There is, however, some evidence that azithromycin (Zedd, Zithromax) 500 mg on day 1, 250 mg days 2 to 5) is beneficial in reducing lymphadenopathy in large, bulky disease.[10] Antibiotics are recommended for systemic disease (e.g. endocarditis, liver or neural infection), bacillary angiomatosis and peliosis hepatis.

Psittacosis

The main reservoir for *Chlamydophila psittaci* is birds, particularly parrots, parakeets and cockatiels.[11] Psittacosis in birds may manifest as ruffled feathers, anorexia, nasal discharge or diarrhoea. Some birds are chronic asymptomatic carriers. Humans are infected via the respiratory route by inhalation of dried faeces or contaminated dust, or by direct contact with contaminated feathers or other items and subsequent inoculation of mucocutaneous membranes of the respiratory tract (i.e. conjunctiva, nasal and oral mucosa). Less commonly, transmission may occur via exposure to birth products from other infected animals, such as dogs, cats, sheep and horses.

Psittacosis in humans ranges from mild influenza-like illness to life-threatening disease with respiratory failure and systemic manifestations. The incubation period is one to four weeks. Typically there is an acute onset of fever and a prominent headache, followed by a dry cough. Other symptoms include diarrhoea, altered mental status and a sore throat. Chest x-ray findings are usually more marked than the chest examination findings. While lobar consolidation is most common, interstitial opacities or no abnormality may be seen.

Presumptive diagnosis is based on the clinical features in combination with a history of bird contact. A significant rise in antibody titre between acute and convalescent serum confirms the diagnosis. The treatment of choice is doxycycline 100 mg twice daily for 10 to 14 days. Macrolide antibiotics are an alternative.[8]

Maintaining a clean and hygienic environment for the birds, seeking veterinary advice when birds are unwell and wearing masks when cleaning birdcages may reduce the risk of infection.

Fish tank granuloma

Mycobacterium marinum is found in fresh and salt water and is associated with infections in pet fish owners.[12] The bacterium causes chronic, slowly progressive skin lesions – nodules, plaques or ulcers – most commonly on the hands following mild trauma (Figure 1). Lymphatic spread, manifested by cutaneous nodules along the lines of lymphatic drainage, is a classical feature. Dissemination is rare and usually only occurs in immunocompromised individuals.

Diagnosis is often difficult, usually requiring a skin biopsy for mycobacterial culture and histological examination for granulomas and acid-fast bacilli. Several months of treatment with doxycycline, clarithromycin or sulfamethoxazole–trimethoprim is recommended.[8]

Gastroenteritis

Species of the genera *Salmonella* and *Campylobacter* have been reported in animals such as cats, dogs, chickens and reptiles. Animal infection may be asymptomatic. Although not common, transmission of these organisms between

pets and humans (presumably through faecal–oral contact) may occur. Both *Salmonella* spp. and *Campylobacter* spp. cause inflammatory diarrhoea in humans, which in most cases is self-limiting without antibiotics.

Diagnosis is by stool culture. Antibiotics are indicated in severe or prolonged cases of *Campylobacter* spp. and severely ill, bacteraemic or immunocompromised cases of *Salmonella* spp. Antibiotic therapy should be guided by sensitivity testing. Resistance to fluoroquinolone antibiotics is an emerging problem.

Viral pet-related infections

Rabies

Rabies is responsible for more than 35,000 deaths per year worldwide.[13] The rabies virus (lyssavirus genotype 1), a single-stranded RNA virus, is transmitted by bites and scratches, and by salivary contamination of mucosa or skin wounds. Domestic dogs are the major reservoir worldwide and the main vector in Asia.[5] However, cats can also be infected, and bats and many other wild animals are also vectors. Australia, most of Scandinavia and several islands, including New Zealand, the British Isles, Japan and the South Pacific Islands, are free of endemic rabies. Australian Bat Lyssavirus (lyssavirus genotype 7), however, is present in Australian flying foxes and bats, and has been responsible for two deaths in Australia from an illness similar to rabies following exposure to bats.[14]

The incubation period for rabies is usually 20 to 90 days but ranges from four days to more than 19 years.[13] There are two clinical syndromes, both of which are invariably fatal. Furious rabies is associated with hydrophobia and aerophobia. Attempts to drink or air blown across the face causes a jerky respiratory spasm, and there may be associated convulsions and cardiorespiratory arrest. Hallucinations, aggressive behaviour and autonomic dysfunction also occur. Paralytic rabies causes an ascending flaccid paralysis, eventually resulting in death due to paralysis of the respiratory muscles.

A course of three doses of rabies vaccine (Merieux Inactivated Rabies Vaccine, Rabipur) is recommended for travellers who will be spending prolonged periods (i.e. more than a month) in rabies endemic areas. In Australia, the vaccine is also recommended for people who are at risk of being bitten or scratched by bats (i.e. bat handlers, wildlife workers and veterinarians).

Following a potential rabies exposure, washing of the wound is very important. It should be flushed for several minutes with running water, and washed with soapy water and disinfectants (povidone–iodine [Betadine Preparations] or 40 to 70% alcohol).[6] Primary wound closure should be avoided. For those people not previously vaccinated, postexposure prophylaxis consisting of rabies immunoglobulin (Imogam Rabies [Pasteurized]), half infiltrated around the wound and the remaining intramuscularly in the deltoid, and a series of five doses of rabies vaccine over a month (days 0, 3, 7, 14 and 28) should be given as soon as possible. In Australia, this procedure is recommended for any person who is bitten or scratched by a bat. For those people previously vaccinated, two booster doses of the vaccine (days 0 and 3) should be given.

Fungal pet-related infections

Tinea (ringworm)

Dermatophytes – fungi requiring keratin, i.e. skin, hair and nails, for growth – may be anthropophilic (primarily cause infection in humans), zoophilic (primarily cause infection in animals) or geophilic (primarily occur on hairs, feathers or nails in the environment).[15] In humans, zoophilic dermatophytes cause more intense inflammatory lesions than anthropophilic dermatophytes, but are also more likely to resolve spontaneously. The two main zoophilic dermatophytes that infect humans are *Microsporum canis* and *Trichophyton mentagrophytes*, and they are acquired through direct contact with animals such as dogs, cats, horses and rabbits.

Zoophilic dermatophytes cause tinea capitis (superficial infection of the scalp, eyelashes and eyebrows), tinea barbae (infection of the beard area) and tinea corporis (infection of the trunk, limbs or face). *M. canis* is the primary causative agent in Australia of tinea capitis and corporis.[16] Tinea barbae is mainly caused by *Trichophyton* spp. but can be caused by *M. canis*. Tinea capitis is most commonly seen in children aged 3 to 7 years, and may vary from mild scaling to widespread alopecia to highly inflammatory lesions called kerions. Tinea corporis typically presents as dry, circular scaling lesions with more marked erythema at the margin. Zoophilic infections may be pustular.

Tinea is diagnosed by collecting infected hair (including the root) or epidermal scales from the advancing edge of the lesion, using a sterile scalpel blade to scrape from the centre to the edge of the lesion, and examining the specimens microscopically for the presence of fungal hyphae. Confirmation by culture may take up to one month.

Tinea capitis and tinea barbae require systemic therapy – terbinafine (Lamisil Tablets, Tamsil, Terbihexal, Zabel) 250 mg daily or griseofulvin (Grisovin) 500 mg daily for four to eight weeks. Oral azoles, such as itraconazole (Sporanox) 100 mg/day, are an alternative. Ketoconazole shampoo (Hexal Konazol 2% Shampoo, Nizoral Treatment, Sebizole Shampoo) may also be used in combination with oral therapy. While tinea corporis often resolves spontaneously in several months, topical therapy (terbinafine 1% [Lamisil Cream, SolvEasy Tinea Cream], clotrimazole 1% [Clonea, Clotreme Anti-Fungal Cream, Clozole Topical Cream 1%, Topizol], ketoconazole 2% [DaktaGOLD, Nizoral Cream] or miconazole 2% [Daktarin, Eulactol Antifungal Spray, Resolve Solution]) can speed resolution.[17] In severe cases oral terbinafine or itraconazole may be

indicated.[17] Infected individuals should avoid sharing headgear, combs and brushes.

The animal reservoir of a dermatophyte infection should be identified by careful inspection. *M. canis*, but not *T. mentagophytes*, can be detected by fluorescence of infected hairs under a Wood's lamp. Good hygiene and fungicidal sprays and washes are recommended to control the infections in animals.

Parasitic pet-related infections

Toxoplasmosis

Toxoplasmosis is a protozoan infection, with cats the definitive host (i.e the host in which the parasite reproduces sexually).[18] Oocysts containing sporozoites are produced during the sexual cycle of *Toxoplasma gondii* reproduction in the small intestine of cats, and are excreted in the faeces. Cats may shed millions of oocysts in their faeces for up to three weeks after infection. The oocysts contaminate food and water and are ingested by a variety of other animals (such as farm animals, mice, birds and lizards). Once ingested, the oocysts rupture to release sporozoites that then convert into tachyzoites (the rapidly dividing asexual life cycle form of *T. gondii* in the acute phase of infection) in the host's small intestine. The tachyzoites disseminate to the host's organs and form tissue cysts containing bradyzoites (the slowly growing asexual life cycle form of *T. gondii*). Cats become infected or reinfected when they eat small animals infected with tissues cysts. Because of immunity, reinfection is rarely followed by reshedding of oocysts.[19]

People are infected in several ways:

- ingestion of food or water contaminated by cat faeces containing *T. gondii* oocysts
- ingestion of *T. gondii* oocysts directly from hands or objects contaminated by cat faeces
- ingestion of undercooked meat containing *T. gondii* tissue cysts
- transmission of tachyzoites via the placenta to the fetus following primary maternal infection.

In the USA, 22.5% of the population over 12 years of age has serological evidence of infection with toxoplasmosis; in other populations the incidence may be up to 95%.[19] Primary infection is asymptomatic in most (80 to 90%) healthy people, but may present as an acute febrile illness with lymphadenopathy or as chorioretinitis with acute unilateral visual loss. Immunocompromised individuals, such as those with HIV infection, are at risk of reactivation of latent infection, most commonly with life-threatening cerebral lesions or sight-threatening chorioretinitis. Congenital infection most frequently

occurs when the mother is acutely infected during the third trimester (30 to 75% risk of fetal infection). Maternal infection in the first trimester is less likely to infect the fetus, but if it does the damage is more severe.[20] Congenital infection may result in fetal death, neurological impairment, chorioretinitis or cytopenias.

Conversion from seronegative to IgM/IgG-positive is the basis of diagnosis. *T. gondii*-specific IgM antibodies may be long-lasting (unlike IgM antibodies in most other infections) and so determining whether an infection is acute may be difficult unless previous negative serology is available. This is of particular importance in pregnant women. Additional testing, such as IgG avidity or molecular techniques may be useful. (The IgG avidity test helps discriminate between past and recently acquired infection. Following antigen challenge the IgG antibodies produced initially bind weakly to the antigen – low avidity; as the immune response develops there is maturation of IgG antibody response and the avidity increases progressively over weeks or months – high avidity.

In most circumstances, specific antimicrobial therapy is not indicated in toxoplasmosis. Exceptions include infections in immunocompromised patients, disseminated disease in immunocompetent patients, infection during pregnancy, and cases of chorioretinitis or congenital toxoplasmosis. Pyrimethamine (Daraprim) plus sulfadiazine (Sulfadiazine Tablets) is the usual therapy, or spiramycin in pregnancy. (Spiramycin is only available in Australia under the Special Access Scheme.)

Prevention of toxoplasmosis, which is particularly important in pregnant women, includes washing raw vegetables before eating, avoiding eating raw or undercooked meat, wearing gloves while gardening, washing hands after gardening or cooking with raw meat or vegetables, and avoiding contact with kitty litter or at least wearing gloves while doing so. Infection of cats can be prevented by not feeding them raw meat and keeping them inside or contained to prevent them from preying on infected wild animals. Pregnant women may consider avoiding contact with cats altogether.

Toxocariasis

The adult worms of *Toxocara canis* (dog roundworm) and *Toxocara cati* (cat roundworm) live in the small intestine of dogs and cats, respectively.[21]

Eggs of the dog roundworm are passed in the animal faeces and undergo a period of development in the soil (about two to five weeks) before they become infective. Eggs ingested by a dog hatch into larvae in the small intestine, penetrate the gut wall and enter the circulation. In most dogs, the larval maturation process is arrested as the larvae become encysted in body tissues. In young puppies, however, the larvae migrate into the lungs, pass up the trachea, down the oesophagus and subsequently into the intestine where they develop into adult worms. In pregnant bitches with encysted larvae, the dormant larvae resume development and migrate across the placenta to the fetus, and also into mammary tissue and hence the milk. After the birth of the puppies, the larvae continue their maturation process, migrating from the lungs to the gastrointestinal tract via the trachea, and maturing in the puppies' intestinal tracts. Bitches then become reinfected while caring for their puppies. The main sources of eggs, therefore, are puppies aged younger than 3 months and lactating female dogs.

The cat roundworm has a life cycle similar to that of the dog roundworm except that vertical transmission is due to lactation more than transplacental transmission.

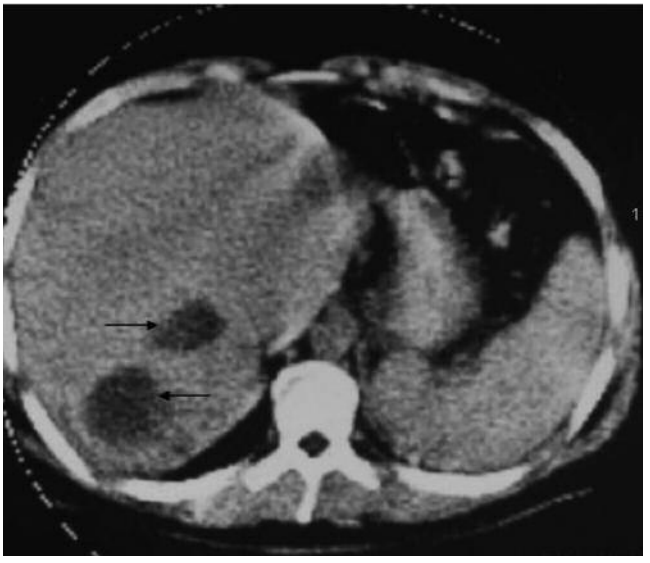
Humans are incidental hosts for *Toxocara* spp. and the larvae are unable to mature fully. Children are most at risk of toxocariasis, commonly becoming infected when playing in sandboxes and playgrounds, or eating dirt contaminated with *Toxocara* spp. eggs, which may remain infective in the soil for years. Direct contact between humans and infected dogs does not play a role in disease transmission.

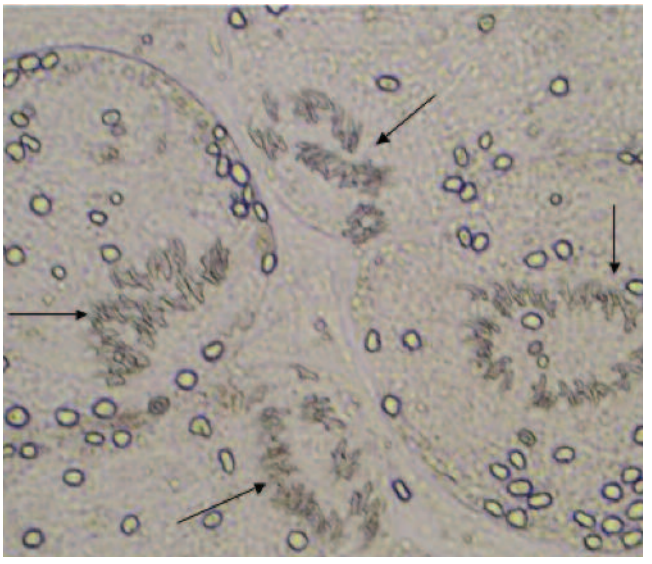
Most human infections are asymptomatic. Disease results from the host's inflammatory response to the larvae, particularly those in the process of dying. The two main manifestations of toxocariasis are visceral larva migrans (VLM) and ocular larva migrans (OLM). VLM usually affects children aged younger than 5 years. Common features include fever, hepatosplenomegaly, hepatitis and respiratory symptoms such as bronchospasm or asthma, in association with marked eosinophilia. OLM tends to affect older children (5 to 10 years of age), and causes visual impairment due to migration of the larvae across the retina. VLM should be suspected in any paediatric patient with an unexplained fever and marked eosinophilia, and is supported by the presence of *Toxocara* spp. antibodies. OLM is diagnosed mainly on findings during ophthalmological examination, as serology tends to be less sensitive.

Treatment with corticosteroids and antihistamines is aimed at suppressing the inflammatory response. The role of anthelmintics, such as albendazole (Eskazole, Zentel), is less clear. Control of infection involves preventing dogs and cats from defaecating in areas where children play, covering sandpits at night, periodic anthelmintic treatment of pets and taking care to wash hands after contacting dogs and cats.

Hydatid disease

Hydatid disease, caused by the tapeworm *Echinococcus granulosus*, is most commonly found in rural areas where farming practices involve both dogs and sheep. In the usual lifecycle of *E. granulosus*, dogs are the definitive host and herbivores such as sheep and cattle are intermediate hosts.[22] Other wildlife have been reported to maintain the infection in certain





Figures 2a and b. Hydatid disease. a (left). CT scan of liver showing two hydatid cysts (arrows). b (right). Photomicrograph (1000X) of the contents of a surgical specimen of a hydatid cyst, demonstrating four protoscoleces (larval tapeworms), with the distinctive hooklets (arrows) by which they will attach themselves to the host's tissues. The protoscoleces are the infective forms that develop into adult worms in the intestine of the dog.

areas, such as dingoes and kangaroos in Australia. Adult worms live in the intestines of dogs, and their eggs are excreted in the faeces, contaminating the environment. Herbivores become infected by eating contaminated pasture, the eggs hatch in the gastrointestinal tract, and the larvae enter the circulation and disseminate to various organs where they develop into cysts. Thousands of larval tapeworms, or protoscolices, are produced in each cyst by asexual budding. Dogs ingest the cysts, usually as a result of feeding on raw offal, and the protoscolices develop into adult tapeworms that eventually produce eggs to complete the life cycle.

Humans are infected by inadvertently ingesting eggs, usually as a result of close dog contact, and are considered incidental, 'dead end' hosts. In humans, cysts most commonly form in the liver, followed by the lungs; they may, however, form in any organ, including the brain, bone and spleen. The size of the cyst depends on the organ involved; liver cysts generally range from 2 to 10 cm in diameter, but may be even larger. Many infections are asymptomatic and the incubation period can be many years. Symptoms are usually a result of either physical compression by an enlarging cyst or allergic or inflammatory reactions as a result of cyst rupture. Diagnosis is based on a relevant exposure history, typical findings on CT scan (Figure 2a) and/or ultrasonography and positive hydatid serology, and is confirmed by microscopic examination of the cyst contents (Figure 2b). Hydatid serology is most sensitive for active liver lesions, and less sensitive for calcified, small, extrahepatic lesions.

Asymptomatic calcified (inactive) lesions do not need to be treated. Other lesions are treated by either surgical excision or percutaneous aspiration techniques, in combination with antiparasitic therapy (albendazole with or without praziquantel [Biltricide]). The risk of anaphylaxis or disseminated infection as a result of spillage of viable cyst contents during these procedures is reduced by prior treatment with albendazole.

Prevention of hydatid disease in endemic areas involves avoiding feeding dogs raw offal, periodic deworming of dogs, reducing fox and wild dog populations, and hand washing after handling dogs.

Conclusion

A history of contact with pet animals should be sought whenever there is an unusual manifestation of infection. The risk of transmission of infections from pets to humans can be reduced by preventing disease in animals (e.g. maintaining hygienic living conditions and regular deworming and treating of fleas), treating unwell pets, not allowing pets access to beaches and playgrounds, removing animal faeces from backyards and public areas such as footpaths and parks as well as regular cleaning of litter trays, washing hands after animal contact, and appropriately managing animal bites. Pregnant women should avoid contact with kitty litter, or even cats altogether.

Immunocompromised individuals should be careful to avoid animal bites and scratches. If such wounds do occur, they should be treated promptly to reduce the risk of serious systemic infection.

Collaboration with a veterinarian may be required in order to prevent and treat infections in pets. MT

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