ZOO NOTIC DERMATITIDES

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SUMMARY — Zoonoses are those diseases and infections which are naturally transmitted between vertebrate animals and man. Viruses, bacteria, fungi, protozoa, helminthes and arthropods can be transmitted directly or indirectly from animals to humans. A restricted number of agents are able to cause skin disease. The etiologic agent can be passively transmitted to the human host or can be actively inoculated in the skin with bites and scratches or arthropod bite. Although all humans are at risk for these skin diseases, some job categories are associated with greater exposure. Immunodeficient human patients are at particular risk of infection by zoonotic agents and, for them, the outcome can be easily fatal. Here we described the lesions caused by zoonotic agents able to induce dermatitis in humans.

Key words: zoonosis, skin dermatitis, dermatosis, viruses, bacteria, protozoa, fungi, helminthes, arthropods

Introduction

The World Health Organization defines zoonoses as “those diseases and infections which are naturally transmitted between vertebrate animals and man”. Viruses, bacteria, fungi protozoa, helminthes and arthropods can be transmitted directly or indirectly from animals to humans. Identifying the suggestive or pathognomonic clinical signs and lesions and the infecting organism allows for accurate diagnosis. Identifying the carrier may be simple or may be more difficult when the cause is a fairly common infection. Because of the increased trade between nations, and changes in animal habitats, there are often new zoonotic diseases. These may be found in animals transported from one nation to another, bringing with them new diseases. In some cases, changes in the environment lead to changes in the migratory habits of animal species, bringing new infections. A restricted number of agents are able to cause skin disease. The etiologic agent can be passively transmitted to the human host or can be actively inoculated in the skin with bites and scratches or arthropod bite. Although all humans are at risk for these skin diseases, some job categories are associated with greater exposure including butchers, abattoir and diagnostic laboratory prosectors, technicians, animal handlers and pet owners and, of course, veterinarians. Veterinarians have the great advantage of deep knowledge of the clinical pathological presentation of these conditions, the epidemiology and preventative measures. Unfortunately domestic and sylvatic animals may be asymptomatic or paucisymptomatic carriers of these sometimes fatal infectious agents. Immunodeficient human patients are at particular risk of infection by zoonotic agents and, for them, the outcome can be easily fatal.

Viruses

Cowpox (vaccinia)

This orthopoxvirus can be transmitted from cows, cats and rodents to humans. Lesions in animal and humans are characterized by the progressive formation of rubeolae, papules, pustules, ulcers and crusts. Histologically large intracytoplasmic eosinophilic viral inclusions may be observed associated with lipid droplets. Lesions are generally self-limiting, but atopic and immunocompromised patients are at risk of a cutaneous dissemination with a more severe course of the infectious illness; even with lethal outcome.

Buffalopox

Outbreaks of zoonotic buffalopox virus were reported from villages in India. In humans, pox lesions were observed on the hands, whereas in affected buffaloes and
In cows the lesions were noticed mainly on the teats and udder. Numerous virus strains were isolated from the skin scabs collected from infected humans and milk animals. A few children who had no contact with infected animals also showed clinical manifestations with disseminated lesions on the face, arm and buttocks, and thus were suspected of acquiring infection through their infected parents or other family members, indicating a possible man to man transmission.

**Monkeypox**

This is an *orthopoxvirus* disease in humans indistinguishable from smallpox. It is primarily observed in rhesus and cynomolgus monkeys. Signs in man include fever, sore throat, headache, and a vesiculopustular rash of peripheral distribution. Complications include bronchopneumonia.

**Yabapox**

In several primates, humans included, this *poxvirus* can cause subcutaneous histiocytomas) which usually regress spontaneously in 3 to 6 weeks.

**Tanapox**

In non human primates *tanapox virus* lesions occur primarily on the face, consisting of raised areas with a central scab. Papules ulcerate, scab and heal. In humans generally there is a single skin vesicle where is possible to histologically detect viral inclusions.

**Ecthyma contagiosum (orf)**

This virus *parapoxvirus* causes a papular cheilitis and stomatitis of sheep and goats that can be transmitted to humans. In humans, it generally manifests as a solitary skin lesion, although rarely it can have an unusual course or be accompanied by systemic symptoms or complications. It is usually diagnosed without difficulty when the lesions have the characteristic morphology and there is an appropriate history of contact with sheep. Unusual lesion locations, such as the perineum in children, have been described. Some masses may reach very large dimensions.

**Papular stomatitis**

This *parapoxvirus* causes papular mucocutaneous, and oral cavity lesions in cattle. A cutaneous form of bovine papular stomatitis was diagnosed in people involved in the handling of a bull. The cutaneous form of bovine papular stomatitis infection presents with gross lesions similar to the cutaneous form of contagious ecthyma (“orf”) or pseudocowpox (“milkers’ nodules”) infection. Because papular stomatitis in cattle occurs most often without evidence of readily observable lesions, unlike contagious ecthyma in sheep or pseudocowpox in cattle, the transmission to man in the cutaneous form could occur without apparent source. The mild clinical manifestations make the condition relatively minor; however, the occasional case may have more severe lesions.

**Foot-and-mouth disease**

This is a devastating disease of cattle, pigs and small ruminants caused by a resistant *aphthovirus*. All the cloven foot animals can be affected, as can elephants, hedgehogs and rodents. In cattle lesions include vesicular stomatitis and glossitis with abundant sialorrhea, followed by dissemination of the virus systemically with vesicular mammary and coronary band dermatitis, cytopathic effect in endocrine organs such as pituitary gland and pancreas leading to diabetes and hirsutism. High mortality can be observed in calves due to myocarditis. Lesions in small and sylvatic ruminants are milder in comparison with cattle. Pigs present with painful lesions on the snout and involving the foot coronary band often with hoof detachment. Humans may develop self-limiting mucocutaneous vesicles and may consequently transmit the disease to susceptible domestic animals and can carry the virus in the pharynx for 3-7 days.

**Bacteria**

**Anthrax**

This is a zoonotic illness caused by *Bacillus anthracis*. Herbivores are very sensitive and develop septicemia, becoming a very dangerous source of bacteria for humans. The bovine carcass tends to progress to precocious autolysis, there is splenomegaly and black blood loaded with bacilli. Sporadic human cases continue to be reported from many parts of the world. Most cases have occurred in agricultural laborers with a history of handling animal meat or skin of infected animals. The presentation in humans can be cutaneous, pulmonary and meningeal. The last two forms of the disease may have serious sequelae, especially the latter. The cutaneous form of illness often has a benign course and responds favorably to antimicrobial treatment. Personnel at risk can be vaccinated, as well as animals in endemic areas with soils particularly favorable to *B. anthracis* spores survival.
**Lyme borreliosis**

The most common vector-borne disease in the northern hemisphere is caused by bacteria belonging to the *Borrelia burgdorferi* complex.\(^{18}\) In Europe, the vector of the disease is the tick *Ixodes ricinus*, whereas in the United States of America, two primary tick vectors exist, namely: *I. scapularis* in the north-eastern and mid-western regions and *I. pacificus* on the west coast. Several species of small and medium-sized mammals and ground-feeding birds serve as reservoirs for the bacteria in endemic areas. Animals tend to be rather resistant to this agent. In nonhuman primates and humans the disease is multisystemic, causing dermatitis, meningonecephalitis, myocarditis and arthrosynovitis. The prognosis for patients with Lyme borreliosis is excellent, particularly when diagnosed and treated early in the course of infection.

**Brucellosis**

It is a common zoonotic disease of worldwide distribution, caused by *Brucella* spp., particularly *Brucella melitensis*. *Brucella* spp. is able to cause necro suppurrative endometritis and chori nitis in ruminants with fetal neutrophilic bronchopneumonia, hepatic granulomas and fibrinous polyserositis. Cattle are affected with *B. abortus*, small ruminants with *B. melitensis* and *B. ovis*, pigs with *B. suis* and dogs with *B. canis*. Skin complications are infrequent and affect less than 5% of human patients with brucellosis, although they may occasionally occur as a macropapular rash during the clinical course of the disease.\(^{19}\)

**Black plague**

*Yersinia pestis* is carried by rodents and transmitted to other animals and humans via fleas or direct contact. Now the few human cases resulted from contact with infected domestic cats, but considering that numerous species are susceptible, the hazard of transmission from other species remains. Cutaneous lesions in affected animals and humans include suppurrative dermatitis with satellite lymphadenitis. Multisystemic dis tribution is common with multiple abscesses and pneumonia with intral esional coccobacilli. In cats and humans aggregations of coccobacilli are particularly present in lymphoid tissue and sometimes in lung tissue (pneumonic plague). The most consistent histologic finding is necro suppurrative lymphadenitis. Invariably, *Y. pestis* bacteria are present in large numbers at affected sites. Orally infected cats have more numerous lesions in the lymph nodes of the head and neck regions. These experimentally induced cases of feline plague document that cats are unique among carnivores in exhibiting bubonic, pneumonic, and septicemic plague following exposure to *Y. pestis*. The lesions of the orally experimentally infected cats are consistent with those described for naturally occurring *Y. pestis* infections in cats and corroborate the contention that cats most commonly contract plague by eating *Y. pestis*-infected rodents and not via flea bite. The histopathology of *Y. pestis* disease in these cats is comparable to that described for human plague.\(^{20,21}\)

**Tularemia**

*Francisella tularensis* is able to affect rodents, lagomorphs, and other sylvatic and domestic species. Lesions include septicemia with disseminated pyogranulomas, pneumonia, enteritis and lymphadenitis. Approximately 200 cases of tularemia are reported in the United States per year and present with various forms such as, glandular, oculoglandular, ulceroglandular, oropharyngeal, and typhoidial pneu monia, occasionally with pyogranulomatous dermatitis.\(^{22,23,24}\)

**Staphylococcosis**

*Staphylococcus aureus* is a common agent of suppurative and gangrenous mastitis in cattle and causes suppurative cutaneous lesions in lagomorphs, chickens and pets. *S. aureus* from animals has been associated with suppurrative dermatitis with folliculitis, furunculosis and impetigo, especially in children below five years of age.\(^{25}\)

**Nocardiosis**

*Nocardia asteroides* is able to cause pyogranulomatous cutaneous, multisystemic pyogranulomas and empyema with intral esional branching bacteria in several domestic and sylvatic species. Humans can also be affected by this type of lesions and occasionally is possible to observe a primary cutaneous form secondary to cat scratch.\(^{26}\)

**Bartonellosis**

*Bartonella henselae* is able to cause histiocytic lymphadenitis in cats and is transmitted by scratches and flea bites. The skin lesions, ranging from 0.2 to 4 cm in diameter, may be nodular or eroding. They occur in series of crops, usually on the limbs and face, which may persist for months to years and may be accompanied by pain and fever. Although lesions may resemble those of Kaposi's sarcoma, they are distinguishable on biopsy. It usually occurs in immunocompromised people but it is not exclusive to them. Immunocompetent individuals with *B. henselae* in-
fected wounds due to trauma, burning and arthropod bite occasionally may present with this condition.\textsuperscript{27,28}

**Erysipelas**

*Erysipelothrix rhusiopathiae* causes septicemia, vegetative endocarditis, polyarthritis and thrombotic dermatitis in pigs and lambs. Infection in man is occupationally related, occurring principally as a result of contact with animals, their products or wastes.\textsuperscript{29} Human infection can take one of three forms: a mild cutaneous infection known as erysipeloid, a diffuse cutaneous form and a serious, although rare, systemic complication with septicemia and endocarditis. Infection still occurs in specific environments and infection by the organism may be underdiagnosed because of the resemblance it bears to other infections and the problems that may be encountered in isolation and identification. Diagnosis of erysipeloid can be difficult if not recognized clinically, as culture is lengthy and the organism resides deep in the skin.\textsuperscript{30}

**Listeriosis**

*Listeria monocytogenes* causes septicemia with disseminated microabscesses, rhombencephalitis with malacia and microabscesses and lymphoplasmacytic leptomenigitis, chorioitits and fetal septicemia in ruminants, primates and, sporadically, infects dogs and chickens where neutrophilic myocarditis can be observed. Dermatitis can be observed in humans. The condition appears as papular or pustular lesions on the arms or hands, and is most often acquired as an occupational hazard from infected animals. Most cases are mild and resolve successfully. However, listeric infections are potentially fatal, and as the initial cause of the lesion may be unknown.\textsuperscript{31,32}

**Fish tank mycobacteriosis**

*Mycobacterium* spp. infections are increasingly reported in fish fanciers who keep an aquarium. The three main species that have been implicated in mycobacteriosis in fish are *M. marinum*, *M. fortuitum* and *M. chelonae*. Most of the cases of human mycobacteriosis are caused by *M. marinum*, although all can cause disease in humans. Inoculation occurs on abraded skin after an incubation period of 2 to 3 weeks. Clinically papules, nodule, ulcers or verrucous plaques develop. These may progress into sporotrichoid lesions or into deeper infections involving tendons and bone. Histopathologic examination is characterized by a nonspecific inflammatory infiltrate in the acute phase. In chronic lesions the histopathologic pattern is a tuberculosis-like granuloma.\textsuperscript{33,34,35}

**Leprosy**

*Mycobacterium leprae*, the etiological agent of leprosy in humans, causes a chronic granulomatous disease that affects primarily the skin and peripheral nerves, and secondarily some internal organs such as the testis and the eye; viscera are seldom involved. Depending on host resistance, leprosy may present as a benign disease (tuberculoid leprosy) or as a malignant disease (lepromatous leprosy), with a spectrum of intermediate stages appearing between the two. Immunity against leprosy depends on the cell-mediated immunity of the host, and this is severely compromised in the malignant (lepromatous) form of leprosy. Although culture of *M. leprae* has never been achieved in artificial media, the bacterium may be grown in several experimental animals, including the armadillo, non-human primates, and to a certain extent, rodents. Naturally acquired leprosy has been reported in wild nine-banded armadillos (*Dasypus novemcinctus*) and in three species of non-human primates (*Pan troglodytes*, sooty mangabeys *Cercocebus atys* and cynomolgus macaques *Macaca fascicularis*), thus qualifying leprosy as a zoonosis. In contrast to human leprosy, murine leprosy (*Mycobacterium lepromurium*) is not a zoonosis.

**Dermatophylosis**

*Dermatophylos congolensis* causes dermatitis and epidermitis in several species. Lesions are characterized by superficial unctuous crusts conglomerating the coat. Histologically there is mild superficial dermatitis with prominent crusting neutrophilic epidermitis and numerous linear (railroad track-like) bacterial colonies. In humans pustular desquamative dermatitis and nodular suppurrative cutaneous lesions are described.\textsuperscript{36a}

**Glanders (farcy)**

*Burkholderia mallei* is able to cause, mainly in horse and humans but also in other species, granulomatous pneumonia (pulmonary form) suppurrative, granulomatous and ulcerative dermatitis (cutaneous form) and can spread multisystemically forming abscesses and granulomas. The granulomas are characterized by central necrosis with karyorhexis. When lesions heal a prominent starry scar remains. The disease is fatal if untreated.

**Meliodosis**

Caused by *Pseudomonas pseudomallei* and also called pseudoglanders, or Whitmore’s disease, occurs in wild rodents, goats, pigs, sheep and primates. There is no evi-
idence that animals are important reservoirs, except in the transfer of the agent to new foci. Skin lesions with fistulous tracks and multisystemic abscesses can occur in animals and humans.

**Rat bite fever**

Caused by *Streptobacillus moniliformis* and *Spirillum minus*. Humans are infected by bite of infected rodent or via contaminated milk or food and may exhibit rash on extremities, often soles and palms. Complications lead to septicemia and multisystemic inflammatory lesions.

**Pasteurellosis**

All animals and birds may be colonized by *Pasteurella*, and human infection occurs by wound infection from bites or scratches. *P. multocida* and the renamed *Mannheimia haemolytica* can cause acute pneumonia or septicemic disease in many species. *P. multocida* may cause chronic infection of upper respiratory and middle ear especially in the rabbit. Local inflammation occurs around the bite or scratch, possibly leading to abscess formation with systemic symptoms.

**Capnocytophagiasis**

*Capnocytophaga canimorsus* can lead to cellulitis, septicemia, meningitis, endocarditis, septic arthritis, and disseminated intravascular coagulation. The organism appears to have an affinity for the eye, causing angular blepharitis and severe keratitis. 36b

**Fungi**

**Dermatophytoses**

Dermatophytoses are superficial skin infections due to dermatophytes which are filamentous fungi. Some dermatophytes can be hosted by domestic animals. *Microsporum canis, Trichophyton (T.) mentagrophytes* variety *mentagrophytes*, *T. verrucosum* and *T. equinum* are zoophilic species capable of infecting humans. The major lesions observed on the affected animals are multifocal to extensive alopecia and/or circumscribed thick hairless skin patches affecting the head, neck, flanks and limbs. Infections can be persistent. Ringworm of cattle is nearly exclusively caused by *Trichophyton verrucosum*. This skin disease is worldwide present in cattle and is responsible for high economic losses in cattle farming. Dermatophytic lesions include neutrophilic superficial and sometimes deep dermatitis with hyperkeratosis predisposing to secondary bacterial infections. Eosinophils can be seen in cattle, dogs, cats and humans. Folliculitis and pyogranulomatous furunculosis often develop in severe dermatophyte in which host and parasite are poorly adapted. 37,38,39,40,41,42

**Malassezia**

In dogs the incidence of dermatitis and otitis resulting from overgrowth of *Malassezia pachydermatis* is prominent. Lesions in dogs are often present in the lower cervical area and chests are grossly unctuous and histologically are characterized by hyperplastic superficial perivascular dermatitis irregular epidermal hyperplasia and spongiform pustules.43

*M. pachydermatis* has been implicated as a pathogen in neonatal intensive care units in which health care workers have introduced infection to human neonates from their family pets. 44

**Sporothricosis**

All forms of sporothricosis are caused by a single species, *Sporothrix schenckii*. In the great majority of cases the fungus gains entrance into the body through trauma to the skin with some kind of plant materials such as thorns or splinters. Zoonotic transmission is also possible and several animals are implicated. Sporothricosis is a ubiquitous mycosis characterized by nodular lesions of the cutaneous or subcutaneous tissues and adjacent lymphatics that usually suppurate and ulcerate. Histologically lesions are pyogranulomatous with multinucleated giant cells. Spread of the organism and granulomatous involvement of numerous viscera is common. Periodic acid-Schiff-positive yeast-like cells can be demonstrated intracellulary in giant cells and extracellularly scattered throughout the tissues. Morphology of the cells varies, with some nonbudding cells resembling *Cryptococcus neoformans* and others cigar-shaped resembling *Sporothrix schenckii*. Indirect immunohistochemistry may be necessary for the final diagnosis.45 In humans, secondary spread to the articular surface and bone or dissemination to the central nervous system, genitourinary tract or lungs is also possible. This kind of transmission is most frequently a professional hazard of people dealing with animals and pets, particularly cat owners.46

**Blastomycosis**

The dimorphic fungus *Blastomyces dermatitidis* when it disseminates can cause subcutaneous abscesses as well as pyogranulomatous lesions in several organs, in particular pneumonia, in animals and humans.
Arthropods

Different pest arthropods of animals may be transmissible to man and produce human dermatoses. The movement and cutaneous penetration habits of external parasites often cause crusted papules, severe itching and dermatitis or may damage their hosts by blood-sucking or by sensitizing them to their saliva. Farm animals may favor the proliferation of external parasites such as ticks (Ixodes ricinus [mammals], Argas reflexus [birds]), fleas (Ceratophyllus gallinae, Ceratophyllus columbae [birds], Spilopsyllus cuniculi [rabbit]), mites (Sarcoptes scabiei var. bovis, Sarcoptes scabiei var. suis, Dermanyssus gallinae [birds], Cheyletiella parasitovorax [rabbit]). Small animals can be responsible of the spread of other arthropod species such as the mites Sarcoptes scabiei var. canis, Notoedres cati, Cheyletiella yasguri, Cheyletiella blakei, fleas such as Ctenocephalides canis, Ctenocephalides felis and ticks. Hedgehogs, which are kept as pets may carry the flea Archaeopylla erinacei. Dermanyssus gallinae mites live in narrow openings and cracks close to the bird housing during the daytime. At night they attack the birds on whose blood they live. When their host disappears, they may attack humans, notably when their breeding places are in or near houses. Mite bites result in urticarial and itchy papular and vesicular skin eruptions.

Nematodes

Larva migrans

Larbish, cutaneous larva migrans or creeping eruption, is a serpiginous cutaneous eruption caused by skin penetration of infective larva from various animal nematodes. Hookworms (Ancylostoma braziliense, A. caninum) are the most common causative parasites. The adults are hematophagous and live in the small intestine of dogs and cats where their ova are deposited in the animal feces. In sandy and shady soil, when temperature and moisture are elevated, the ova hatch and mature into infective larva. Infection occurs when humans have contact with the infected soil. Larvae penetrate the exposed skin of the body, commonly around the feet, hands and buttocks. In humans, the larvae are not able to complete their natural cycle and remain trapped in the upper dermis of the skin. The disease is widespread in tropical or subtropical regions, especially along the coast on sandy beaches. The diagnosis is easy for the patient who is returning from a tropical or subtropical climate and gives a history of beach exposure. The characteristic skin lesion is a fissure or erythematous cord that is displaced a few millimeters each day in a serpiginous track. Scabies, the larva currens syndrome due to Strongyloides stercoralis, must be distinguished from other creeping eruptions and subcutaneous swelling lesions caused by other nematodes or myiasis. Medical treatments are justified because it shortens the duration of the natural evolution of the disease. A little known hazard of similar infections exists among veterinarians and laboratory workers exposed to Strongyloides larvae from horses located in temperate climates. Continued exposure may lead to a state of hypersensitivity to the parasitic protein resulting in severe hyperimmune reactions. Baylisascaris procyonis, a roundworm infection of raccoons, is emerging as an important helmintic zoonosis, principally affecting young children. Raccoons have increasingly become peridomestic animals living in close proximity to human residences. They cause a visceral granulomatous tract and lesions and they can be fatal, but skin lesions are not described as are not described for the dog ascarid Toxocara canis larvae.

Zoonotic Agents Transmitted by Arthropods (“Arbozoonoses”)

Some zoonotic agents able to cause skin lesions are transmitted almost exclusively by arthropods (“arbozoonoses”).

Bacteria

Ehrlichioses

Human ehrlichioses are tick-borne infections caused by bacteria in the genus Ehrlichia. Human monocytic ehrlichiosis is caused by Ehrlichia chaffeensis and human granulocytic ehrlichiosis is caused by an agent similar to Anaplasma phagocytophilum (former Ehrlichia equi). E. chaffeensis infects mononuclear phagocytes and is transmitted by Lone Star ticks (Amblyomma americanum) found in the south central and eastern United States. The agent of human granulocytic ehrlichiosis infects mostly neutrophils, it transmitted by Ixodes species ticks, and occurs mostly in the upper midwest and northeast United States, but also in central and south America, Europe and Africa. The presentation of both ehrlichioses is similar with fever, headache, myalgias, leukopenia, thrombocytopenia, and elevated liver enzyme activities, the diagnostic methods are distinct. Occasional severe complications include meningoencephalitis, adult respiratory distress syndrome, shock, and opportunistic infections. Immunocompromised patients are at high risk for death. Organisms can be visualized within...
histiocytes, although morulae can be rarely present within lymphocytes. An adverse outcome is associated with delayed diagnosis and therapy; thus, empirical treatment is advocated. Treatment with doxycycline usually results in prompt defervescence and cure. *Anaplasma phagocytophilum* is transmitted by *Ixodes* ticks and causes high fever, depression, anorexia, limb edema, petechiation, icterus, ataxia, and stiffness in gait, lymph node hyperplasia in horses.59,60

**Rickettsioses**

Rickettsial diseases61 have a diversity of epidemiologic characteristics reflective of the variety of ecologic situations in which the obligate intracellular bacteria are transmitted to humans. For the spotted fever group rickettsiae, *Rickettsia typhi*, *R. tsutsugamushi*, *Coxiella burnetii*, and the human ehrlichial agent, humans are a dead-end host who plays no role in the maintenance of the organism in nature. All rickettsioses exist as zoonoses. Moreover, all rickettsiae are found in infected arthropods, which generally serve as the natural hosts and can transmit the infection to the next generation of ticks, mites, chiggers, or fleas. The microscopic pathology of the rickettsial diseases is characteristic. The rickettsiae multiply within the endothelial cells lining the small blood vessels. Endothelial proliferation and perivascular infiltration lead to leakage and thrombosis. Such vascular lesions in the skin produce the rash; identical lesions in the meninges probably account for the headache. Thromboses and necrosis of arteriolar walls lead to the rupture of vessels and consequent petechial or large hemorrhages. Degenerative changes in muscles frequently occur in Rocky Mountain spotted fever (*Rickettsia rickettsii* carried by the tick *Dermacentor andersoni*), but are rare in other rickettsial diseases. Eastern tick-borne rickettsioses include North Asian tick-borne rickettsiosis, Queensland tick typhus, African tick typhus, and Mediterranean spotted fever (fièvre boutonneuse). The causative agents belong to the spotted fever group of rickettsiae. North Asian tick-borne rickettsiosis, caused by *R. sibirica*, occurs in Armenia, Central Asia, Siberia, and Mongolia; Queensland tick typhus, caused by *R. australis*, occurs in Australia. Fièvre boutonneuse, the prototype of the group, caused by *R. conorii*, occurs throughout the African continent, in India, and in areas of Europe and the Mideast adjacent to the Mediterranean, Black, and Caspian seas. It often is named after the area in which it occurs (Mediterranean spotted fever, Marseilles fever, Indian tick typhus). The epidemiology of these tick-borne rickettsioses resembles that of spotted fever in the Western Hemisphere. Ixodid ticks and wild animals maintain the rickettsiae in nature; if humans intrude accidentally into the cycle, they become infected. In certain areas, the cycle of fièvre boutonneuse involves domiciliary environments, with the brown dog tick, *Rhipicephalus sanguineus*, as the dominant vector. Transovarial transmission of rickettsiae occurs in various ticks. After an incubation period of one week, fever, malaise, headache, and conjunctival hyperemia develop. A small buttonlike ulcer 2 to 5 mm in diameter with a black center appears (an eschar, or, in fièvre boutonneuse, tache noire). Usually there is regional or satellite lymphadenopathy. On about the fourth day of fever, a red maculopapular rash appears on the forearms and extends to most of the body, including the palms and soles. Fever lasts into the second week. Complications are rare, and death is rare except among aged or debilitated patients. However, the disease should not be ignored; a fulminant form of vasculitis can occur. Rickettsialpox is caused by *Rickettsia akari* and it is a benign disease, mainly of adults, transmitted to humans by the mite *Liponyssoides sanguineus*. The mouse (*Mus musculus*) is a definitive host. It is an urban disease. The primary, or herald lesion, occurs at the site of the bite after one-two weeks. The lesion forms a black eschar and inflammation is intense in the deeper layer of the dermis, where there is actual necrosis. There is a septicemia and ensuing generalized rash. The rash is vesicular papular. The vesicles are small and they appear to be embedded in the papule. *Coxiella burnetii*, which causes chorionitis and fetal septicaemia in small ruminants, is a resistant and important zoonotic agent (Query fever, Q fever), but is not associated with a cutaneous exanthema.

**Protozoa**

**Leishmaniasis**

Members of the genus *Leishmania*62 infect many vertebrates, including humans, dogs, and rodents. The life cycles of members of the genus involve a vertebrate host (e.g., the human) and a vector (the sand fly *Phlebotomus*) that transmits the protozoa between vertebrate hosts. In the vector the parasite takes on a characteristic morphological form known as the promastigote and it reproduces asexually in the vector’s intestine. When the vector bites the vertebrate host, promastigotes are injected into the vertebrate host. The promastigotes enter cells of the vertebrate host and change into a form called the amastigote. The amastigote reproduces in the host’s cells, and when the cell eventually dies the amastigotes are released and infect other cells. Several species of *Leishmania* are patho-
genic for man: *L. donovani* causes visceral leishmaniasis (Kala-azar, black disease, dumdum fever); *L. tropica* (*L. t. major*, *L. t. minor* and *L. ethiopica*) cause cutaneous leishmaniasis (oriental sore, Delhi ulcer, Aleppo, Delhi or Baghdad boil); and *L. braziliensis* (also, *L. mexicana* and *L. peruviana*) are etiologic agents of mucocutaneous leishmaniasis (espundia, Uta, chichlero ulcer). In cutaneous leishmaniasis (Oriental sore, Delhi ulcer, Baghdad boil), the organism (*L. tropica*) multiplies locally, producing of a papule, 1-2 weeks, or as long as 1-2 months, after the bite. The papule gradually grows to form a relatively painless ulcer. The center of the ulcer encrusts while satellite papules develop at the periphery. The ulcer heals in 2-10 months, even if untreated but leaves a disfiguring scar. The disease may disseminate in the case of depressed immune function.). The initial symptoms of mucocutaneous leishmaniasis (espundia, Uta, chichlero) are the same as those of cutaneous leishmaniasis, except that in this disease the organism can spread and the lesions involve oral, pharyngeal and nasal tissues and lead to their destruction and hence severe deformity. The organisms responsible are *L. braziliensis*, *L. mexicana* and *L. peruviana*. The pathogenesis of leishmaniasis is due to an immune reaction to the organism, particularly cell-mediated immunity. Laboratory examination reveals a marked leukopenia with relative monocytosis and lymphocytosis, anemia and thrombocytopenia. IgM and IgG levels are extremely increased due to both specific antibodies and polyclonal activation.

**American trypanosomiasis (Chagas’ disease)**

Chagas’ disease is caused by the protozoan hemoflagellate, *Trypanosoma cruzi*, which is scattered irregularly in Central and South America, extending from parts of Mexico to Argentina. Rare cases have been reported in Texas, California and Maryland. The organism is transmitted to mammalian host by many species of kissing (riduvid) bugs such as *Triatoma infestans*, *T. sordida*, *Pintstrongylus megistus* and *Rhodnius prolisus*. Transmission takes place during the feeding of the bug that normally bites in the facial area (hence the name, kissing bug) and defecates during feeding. The metacyclic trypanastigotes, contained in the fecal material, gain access to the mammalian tissue through the wound which is often rubbed by the individual that is bitten. Depending on its host environment, the organism occurs in three different forms: the trypanosomal (tryponastigote) form in mammalian blood, the crithidial (epimastigote) form in the insect intestine and the leishmanial (amastigote) form found intracellularly or in pseudocysts in mammalian and lacks a prominent flagellum. More than one hundred mammalian species of wild and domestic animals including cattle, pigs, cats, dogs, rats, armadillo, raccoon and opossum are naturally infected by *T. cruzi* and serve as a reservoir. In animals *T. cruzi* causes severe progressive lymphocytic and plasmacytic fibrosing myocarditis leading to systemic passive congestion. Chagas’ disease can be divided into three stages: the primary lesion, the acute stage, and the chronic stage. The primary lesion, chagoma, appearing at the site of infection, within a few hours of a bite, consists of a slightly raised, flat non-purulent erythematous plaque surrounded by a variable area of firm edema and satellite lymphadenopathy. It is usually found on the face, eyelids, cheek, lips or the conjunctiva, but may occur on the abdomen or limbs. Infection in the eyelid, resulting in a unilateral conjunctivitis and orbital edema (Ramana’s sign), is the most common finding. Other manifestations of the acute phase are cervical, axillary and iadic adenitis, hepatomegaly, erythematous rash and acute myocarditis. Meningo-encephalitis can occur in children. The chronic stage includes myocarditis, aneurysma, megaesophagus and megacolon.

**Uncommon Cutaneous Zoonoses due to Helminthes**

**Dirofilariasis**

The presence of *Dirofilaria repens* has been identified in worldwide (dogs, foxes, cats, lions). Adult worms in animals cause mild skin focal lesions but embolized microfilariae can induce multifocal necrosis of the skin. In humans, subcutaneous nodules can be caused by *D. tenuis*, a parasite from raccoons, *D. repens*, from dogs and cats, *D. ursi*, from bears, and *Onchocerca spp.* from horses or cattle. Nodules that on microscopic examination are seen to be lymph nodes may harbor *Brugia spp.*

**Gnathostomiasis**

This uncommon disease, even in endemic areas of Southeast Asia, most often results from ingestion of the third-stage larvae of the nematode *Gnathostoma spinigerum*, although several other species also cause human disease. Definitive hosts for *Gnathostoma* species include dogs, cats, tigers, leopards, lions, minks, opossums, raccoons, and others, in which the adult worms live in a fibrous cyst in the gastric wall. Eggs leave an aperture in the tumor that opens on the stomach lumen and pass into water in the feces. Eggs develop into larvae, which hatch, and then are ingested by the first intermediate host, minute crustaceans of
the genus *Cyclops*. The copepods are ingested by the second intermediate hosts or definitive hosts. The larvae may be found in raw or undercooked meat such as freshwater fish, chicken, snails, frogs, pigs) or in contaminated water. Rarely, larvae penetrate the skin of those exposed to such meat or water, as well. Any organ system can be involved, but the most common manifestation of infection is localized, intermittent, migratory swelling in the skin and subcutaneous tissues. Such swelling may be painful, pruritic, and/or erythematous. With *Angiostrongylus cantonensis*, *Gnathostoma* species are a common cause of parasitic eosinophilic meningitis, resulting from their random migration into the central nervous system (CNS). Typically, infection is associated with peripheral eosinophilia.69

**Sparganosis**

Human infection with the sparganum stage of the species of the tapeworm *Spirometra* results from drinking pond, lake, or stream water containing procercoid-infected *Cyclops*, eating a raw infected frog, snake, or small mammal, or applying plerocercoid-infected flesh of frogs, snakes, or possible warm-blooded animals as a poultice on an inflamed eye or finger. As spargana are known to develop in pigs, human infection may occasionally be acquired by eating raw pork. In the subcutis sparganum froms a and often times it will migrate through the tissue, hence “creeping tumor” with granulomatous and eosinophilic dermatitis and sinus tract. When the larva dies, its remnants will cause great inflammation in the area and become very painful.70

**Note**

The information present in this manuscript regarding the pathology of the diseases in animals is based primarily on the author’s experience. Additional information, including numerous references, can be obtained from the three volume book “Pathology of Domestic Animals”, Fourth edition Edited by K.V. Jubb, Peter C. Kennedy & Nigel Palmer, Academic Press.

**References**

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