ZOONOTIC 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.[1,2,3,4] 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.[5,6,7,8] 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.[9]

Buffalopox

Outbreaks of zoonotic buffalopox virus were reported from villages in India.[10] In humans, pox lesions were observed on the hands, whereas in affected buffaloes and

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.[11] 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.[12]

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.[13] 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 mucocutanous vescicles and may consequently transmit the disease to susceptible domestic animals and can carry the virus in the pharynx for 3-7 days.[14]

Bacteria

Anthrax

This is a zoonotic illness caused by *Bacillus anthracis*.[15,16,17] 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 necrosuppurative endometritis and chorionitis 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 haz ard of transmission from other species remains. Cutaneous lesions in affected animals and humans include suppurative dermatitis with satellite lymphadenitis. Multisystemic distribution is common with multiple abscesses and pneumonia with intralesional 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 necrosuppurative 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 typhoidal pneumonic, 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 suppurative 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 intralesional branching bacteria in several domestic and sylvatic species. Humans can be also 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 fleabites. 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.[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.[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.[30]

Listeriosis

Listeria monocytogenes causes septicemia with disseminated microabscesses, rhombencephalitis with malacia and microabscesses and lymphoplasmacytic leptomeningitis, chorionitis 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.[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.[33,34,35]

Leprosy

Mycobacterium leprae[36], 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 (chimpanzees [*Pan troglodytes*], sooty mangabey monkeys [*Cercocebus atys*] and cynomolgus macaques [*Macaca fascicularis*]), thus qualifying leprosy as a zoonosis. In contrast to human leprosy, murine leprosy (*Mycobacterium lepremurium*) is not a zoonosis.

Dermatophylosis

Dermatophylus 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 suppurative cutaneous lesions are described. [36a]

Glanders (farcy)

Burkholderia mallei is able to cause, mainly in horse and humans but also in other species, granulomatous pneumonia (pulmonary form) suppurative, 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-

dence 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 Pasteurellae, 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 schenkii*. 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 intracellularly 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]).[2] 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 *Archaeopsylla erinacei*.[47,48,49,50,51] *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.[52]

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 brasiliense*, *A. caninum*) are the most common causative parasites. The adults are hematophagic 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. [53,54] 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.[55] *Baylisascaris procyonis*, a roundworm infection of raccoons, is emerging as an important helminthic 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.[56,57]

Zoonotic Agents Aransmitted by Arthropods ("Arbozoonoses")

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

Bacteria

Ehrlichioses

Human ehrlichioses[58] 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 phagocytophila* (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 cane 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 phagocytophila* 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 diseases[61] 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 (fičvre boutonneus). 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 fičvre 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 forth 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 it is caused by *Rickettsia akari* and it is a benign disease, mainly of adults, transmitted to humans by the mite *Liporyssoides sanguine*). 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 septicemia in small ruminants, it 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, chiclero 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, chiclero) 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 sever 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*[63, 64], 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*, *Panstrongylus megistus* and *Rhodnius prolixus*. 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 trypamastigotes, 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 (trypomastigote) 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. cruzy 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 nonpurulent 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 iliac 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.[65] 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..[66,67,68]

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 otters, 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.F. Jubb, Peter C. Kennedy & Nigel Palmer, Academic Press.

References

1. VON ESSEN SG, McCURDY SA. Health and safety risks in production agriculture. West J Med 1998; 169(4):214-20.
2. BECK W. [Farm animals as disease vectors of parasitic epizoonoses and zoophilic dermatophytes and their importance in dermatology] Hautarzt 1999; 50(9):621-8.
3. BIN ZAKARIA M, LERCHE NW, CHOMEL BB, KASS PH. Accidental injuries associated with nonhuman primate exposure at two regional primate research centers (USA): 1988-1993. Lab Anim Sci 1996; 46(3):298-304.
4. PARISH LC, SCHWARTZMAN RM. Zoonoses of dermatological interest. Semin Dermatol 1993; 12(1):57-64.
5. STEINBORN A, ESSBAUER S, MARSCH WCh. [Human cowpox/catpox infection] Dtsch Med Wochenschr 2003;128(12):607-10.
6. LEWIS-JONES S. The zoonotic poxviruses. Dermatol Nurs 2002; 14(2):79-82, 85-6.
7. AMER M, EL-GHARIB I, RASHED A, FARAG F, EMARA M. Human cowpox infection in Sharkia Governorate, Egypt. Int J Dermatol 2001; 40(1):14-7.
8. BLACKFORD S, ROBERTS DL, THOMAS PD. Br J Dermatol 1993;129(5):628-9.
9. CZERNY CP, EIS-HUBINGER AM, MAYR A, SCHNEWEIS KE, PFEIFFB. Animal poxviruses transmitted from cat to man: current event with lethal end. Zentralbl Veterinarmed B 1991; 38(6):421-31.
10. KOLHAPURE RM, DEOLANKAR RP, TUPE CD, RAUT CG, BASU A, DAMA BM, PAWAR SD, JOSHI MV, PADBIDRI VS, GOVERDHAN MK, BANERJEE K. Investigation of buffalopox outbreaks in Maharashtra State during 1992-1996. Indian J Med Res 1997;106:441-6.
11. STEAD JW, HENRY CM, SIMPSON RH. Rare case of autoinoculation of orf. Br J Gen Pract 1992; 42(362):395-6.
12. GUREL MS, OZARDALI I, BITIREN M, SAN I, ZEREN H. Giant orf on the nose. Eur J Dermatol 2002;12(2):183-5.
13. BOWMAN KF, BARBERY RT, SWANGO LJ, SCHNURRENBERGER PR. Cutaneous form of bovine papular stomatitis in man. JAMA 198; 246(24):2813-8.
14. BAUER K. Foot- and-mouth disease as zoonosis. Arch Virol Suppl 1997;13:95-7.
15. KUMAR A, KANUNGO R, BHATTACHARYA S, BADRINATH S, DUTTA TK, SWAMINATHAN RP. Human anthrax in India: urgent need for effective prevention. J Commun Dis 2000; 32(4):240.
16. WITKOWSKI JA, PARISH LC. The story of anthrax from antiquity to the present: a biological weapon of nature and humans. Clin Dermatol 2002; 20(4):336-42.
17. TAYLOR JP, DIMMITT DC, EZZELL JW, WHITFORD H. Indigenous human cutaneous anthrax in Texas. South Med J 1993; 86(1):1-4.
18. GERN L, FALCO RC. Lyme disease. Rev Sci Tech 2000; 19(1):121-35.
19. MILIONIS H, CHRISTOU L, ELISAF M. Cutaneous manifestations in brucellosis: case report and review of the literature. Infection 2000; 28(2):124-6.
20. WATSON RP, BLANCHARD TW, MENSE MG, GASPER PW. Histopathology of experimental plague in cats. Vet Pathol 2001; 38(2):165-72.
21. Orloski KA, Eidson M. Yersinia pestis infection in three dogs. J Am Vet Med Assoc 1995; 207(3):316-8.

22. YOUNG LS, SHERMAN IL. Tularemia in the United States: recent trends and a major epidemic in 1968. J Infect Dis 1969; 119(1):109-10.

23. YOUNG LS, BICKNESS DS, ARCHER BG, CLINTON JM, LEAVENS LJ, FEELEY JC, BRACHMAN PS. Tularemia epidemia: Vermont, 1968. Forty-seven cases linked to contact with muskrats. N Engl J Med 1969; 5;280(23):1253- 60.

24. GILL V, CUNHA BA. Tularemia pneumonia. Semin Respir Infect 1997; (1):61-7.

25. RAO PN, NAIDU AS, RAO PR, RAJYALAKSHMI K. Prevalence of staphylococcal zoonosis in pyogenic skin infections. Zentralbl Bakteriol Mikrobiol Hyg [A] 1987; 265(1-2):218-26.

26. ASTUDILLO L, DAHAN S, ESCOURROU G, SAILLER L, CARREIRO M, OLLIER S, ARLET P. Cat scratch responsible for primary cutaneous Nocardia asteroides in an immunocompetent patient. Br J Dermatol 2001; 145(4):684-5.

27. KARAKAS M, BABA M, AKSUNGUR VL, HOMAN S, MEMISOGLU HR, UGUZ A. Bacillary angiomatosis on a region of burned skin in a immunocompetent patient. Br J Dermatol 2000; 143(3):609-11.

28. MARGILETH AM. Recent Advances in Diagnosis and Treatment of Cat Scratch Disease. Curr Infect Dis Rep 2000; 2(2):141-146.

29. BROOKE CJ, RILEY TV. Erysipelothrix rhusiopathiae: bacteriology, epidemiology and clinical manifestations of an occupational pathogen. J Med Microbiol 1999; 48(9):789-99.

30. ROBSON JM, McDOUGALL R, VAN DER VALK S, WAITE SD, SULLIVAN JJ. Erysipelothrix rhusiopathiae: an uncommon but ever present zoonosis. Pathology 1998; 30(4):391-4.

31. McLAUCHLIN J, LOW JC. Primary cutaneous listeriosis in adults: an occupational disease of veterinarians and farmers. Vet Rec 1994;135(26):615-7.

32. HAUSERMANN P. [Cutaneous listeriosis] Schweiz Rundsch Med Prax 2001; 90(19):851-2.

33. KIESCH N. [Aquariums and mycobacterioses] Rev Med Brux 2000; 21(4):255-6.

34. KELLY R. Mycobacterium marinum infection from a tropical fish tank. Treatment with trimethoprim and sulphamethoxazole. Med J Aust; 30;2(18):681-2.

35. BLACK H, RUSH-MUNRO FM, WOODS G. Mycobacterium marinum infections acquired from tropical fish tanks. Australas J Dermatol 1971; 12(3):155-64.

36. ROJAS-ESPINOSA O, LOVIK M. Mycobacterium leprae and Mycobacterium lepraemurium infections in domestic and wild animals. Rev Sci Tech 2001; 20(1):219-51.

36a. ALBRECHT R, HOROWITZ S, GILBERT E, HONG R, RICHARD J, CONNOR.DH. Dermatophilus congolensis chronic nodular disease in man. Pediatrics 1974; 53(6):907-12.

36b. LE MOAL G, LANDRON C, GROLLIER G, ROBERT R, BURUCOA C. Meningitis due to Capnocytophaga canimorsus after receipt of a dog bite: case report and review of the literature. Clin Infect Dis 2003; 36(3):42-6

37. DEKIO S, JIDOI J. Tinea corporis due to Trichophyton verrucosum: report of a patient from the San'in District. J Dermatol 1994; 21(5):347-51.

38. MORIELLO KA, DeBOER DJ. Feline dermatophytosis. Recent advances and recommendations for therapy. Vet Clin North Am Small Anim Pract 1995; 25(4):901-21.

39. SPIEWAK R. Zoophilic and geophilic fungi as a cause of skin disease in farmers. Ann Agric Environ Med 1998;5(2):97-102.

40. SPIEWAK R, SZOSTAK W. Zoophilic and geophilic dermatophytoses among farmers and non-farmers in Eastern Poland. Ann Agric Environ Med 2000;7(2):125-9.

41. WABACHA JK, GITAU GK, BEBORA LC, BWANGA CO, WAMURI ZM, MBITHI PM. Occurrence of dermatomycosis (ringworm) due to Trichophyton verrucosum in dairy calves and its spread to animal attendants. J S Afr Vet Assoc 1998; 69(4):172-3.

42. LATEUR N. [Dermatophytoses due to domestic animals] Rev Med Brux 2000; 21(4):237-41.

43. MORRIS DO. Malassezia dermatitis and otitis. Vet Clin North Am Small Anim Pract 1999; 29(6):1303-10.

44. CHANG HJ, MILLER HL, WATKINS N, ARDUINO MJ, ASHFORD DA, MIDGLEY G, AGUERO SM, PINTO-POWELL R, VON REYN CF, EDWARDS W, MCNEIL MM, JARVIS WR. An epidemic of Malassezia pachydermatis in an intensive care nursery associated with colonization of health care workers' pet dogs. N Engl J Med 1998; 338(11):706-11.

45. WENKER CJ, KAUFMAN L, BACCIARINI LN, ROBERT N. Sporotrichosis in a nine-banded armadillo (Dasypus novemcinctus). J Zoo Wildl Med 1998; 29(4):474-8.

46. OLIVEIRA-NETO MP, MATTOS M, LAZERA M, REIS RS, CHICARINO-COELHO JM. Zoonotic sporothricosis transmitted by cats in Rio de Janeiro, Brazil. A case report. Dermatol Online J 2002; 8(2):5.

47. BECK W. [Animal mite-induced epizoonoses and their significance in dermatology] Hautarzt 1996; 47(10):744-8.

48. TSIANAKAS P, POLACK B, PINQUIER L, LEVY KLOTZ B, PROST-SQUARCIONI C. [Cheyletiella dermatitis: an uncommon cause of vesiculobullous eruption] Ann Dermatol Venereol 2000;127(10):826-9

49. WALTON SF, CHOY JL, BONSON A, VALLE A, MCBROOM J, TAPLIN D, ARLIAN L, MATHEWS JD, CURRIE B, KEMP DJ. Genetically distinct dog-derived and human-derived Sarcoptes scabiei in scabies-endemic communities in northern Australia. Am J Trop Med Hyg 1999;61(4):542-7

50. BECK W, CLARK HH. [Differential diagnosis of medically relevant flea species and their significance in dermatology] Hautarzt 1997;48(10):714-9. Dodd K. Skin lesions associated with Cheyletiella yasguri infestation. J Ir Med Assoc 1970;63(401):413-4.

51. TAYLOR RM. Cheyletiella parasitivorax infestation of a cat and associated skin lesions of man. Aust Vet J 1969; 45(9):435.

52. PRINS M, GO IH, VAN DOOREN-GREEBE RJ. [Parasitic pruritus: bird mite zoonosis] Ned Tijdschr Geneeskd 1996;140(51): 2550-2.

53. CHABASSE D, LE CLEC'H C, DE GENTILE L, VERRET JL. [Larva migrans] Sante 1995; 5(6):341-5.

54. PROCIV P. Pathogenesis of human hookworm infection: insights from a 'new' zoonosis. Chem Immunol 1997;66:62-98.

55. ROECKEL IE, LYONS ET. Cutaneous larva migrans, an occupational disease. Ann Clin Lab Sci 1977; 7 (5):405-10.

56. SORVILLO F, ASH L R, BERLIN O.G.W.,*YATABE J, DEGIORGIO G, MORSE SA. An Emerging Helminthic Zoonosis. Emerging infectious diseases. April 2002; 8 (4).

57. WISEMAN RA.Toxocariasis in man and animals. Vet Rec 1969; 84(9):214-6.

58. DUMLER JS, BAKKEN JS. Human ehrlichioses: newly recognized infections transmitted by ticks. Annu Rev Med 1998;49:201-13

59. MADIGAN JE, PUSTERLA N. Ehrlichial diseases. Vet Clin North Am Equine Pract 2000;16(3):487-99.

60. CHANG YF, NOVOSEL V, DUBOVI E, WONG SJ, CHU FK, CHANG CF, DEL PIERO F, SHIN S, LEIN DH. Experimental infection of the human granulocytic ehrlichiosis agent in horses. Vet Parasitol 1998; 78(2):137-45.

61. WALKER DH, FISHBEIN DB. Epidemiology of rickettsial diseases. Eur J Epidemiol 1991; 7(3):237-45.

62. KUMARI S, RAM VJ. Visceral Leishmaniasis: Clinical Features, Pathology, Diagnosis and Chemotherapeutic Developments. Drug News Perspect 2002;15(7):417-431.

63. PRATA A. Clinical and epidemiological aspects of Chagas disease. Lancet Infect Dis 2001; 1(2):92-100.

64. RACCURT CP. Trypanosoma cruzi in French Guinea: review of accumulated data since 1940. Med Trop 1996;56: 79-87.

65. CHAUVE CM. Importance in France of the infestation by Dirofilaria (Nochtiella) repens in dogs. Parassitologia 1997; 39(4):393-5.

66. HERZBERG AJ, BOYD PR, GUTIERREZY. Subcutaneous dirofilariasis in Collier County, Florida, USA. Am J Surg Pathol 1995; 19(8):934-9.

67. GUTIERREZ Y. Diagnostic features of zoonotic filariae in tissue sections. Hum Pathol 1984; 15(6):514-25.

68. DEGARDIN P, SIMONART JM. Dirofilariasis, a rare, usually imported dermatosis. Dermatology 1996; 192 (4):398-9.

69. SATO H, KAMIYA H, HANADA K. Five confirmed human cases of gnathostomiasis nipponica recently found in northern Japan. J Parasitol 1992; 78 (6):1006-10

70. CHANG JH, LIN OS, YEH KT. Subcutaneous sparganosis—a case report and a review of human sparganosis in Taiwan. Kaohsiung J Med Sci 1999; 15(9):567-71.