Bite-related and septic syndromes caused by cats and dogs

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Bite infections can contain a mix of anaerobes and aerobes from the patient's skin and the animal's oral cavity, including species of *Pasteurella*, *Streptococcus*, *Fusobacterium*, and *Capnocytophaga*. Domestic cat and dog bite wounds can produce substantial morbidity and often require specialised care techniques and specific antibiotic therapy. Bite wounds can be complicated by sepsis. Disseminated infections, particularly those caused by *Capnocytophaga canimorsus* and *Pasteurella multocida*, can lead to septic shock, meningitis, endocarditis, and other severe sequelae. An emerging syndrome in veterinary and human medicine is meticillin-resistant *Staphylococcus aureus* (MRSA) infections shared between pets and human handlers, particularly community-acquired MRSA disease involving the USA300 clone. Skin, soft-tissue, and surgical infections are the most common. MRSA-associated infections in pets are typically acquired from their owners and can potentially cycle between pets and their human acquaintances.

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Introduction

"Love the animals: God has given them the rudiments of thought and joy untroubled"

Fyodor Dostoyevsky

No relationship is as constant and ancient to mankind as that between human beings and their pets. Dogs were first domesticated about 15 000 years ago from wolves living in or near China.[1] The first domesticated cat originated in Cyprus almost 9500 years ago.[2] Today, about 75 million dogs and 88 million cats are owned in the USA alone, with pets being found in about 63% of American households.[3] In the UK, there are some 27 million domestic pets, inhabiting about 43% of British households.[4]

However, the closeness between pet owners and their animals comes with the potential for transmission of at least 30 infectious agents.[5] In 2001, more than 350 000 people were treated in US hospital emergency departments for non-fatal dog-bite-related injuries.[6] Annual direct medical costs related to dog-bite injuries are estimated to be approximately US$165 million.[7] Although certain zoonoses, such as bite-wound infections and cat-scratch disease are well recognised, other infections, such as device-related infections and community-acquired meticillin-resistant *Staphylococcus aureus* (MRSA) are newer and are increasing in prevalence. In this Review, we provide an overview of several of the most important infectious disease pathogens transmitted by dogs and cats to human beings, and in some cases, from human beings to their pets.

Bite-related injuries
Epidemiology
In the USA, dog and cat bites comprise roughly 1% of emergency room visits annually,[8,9] with similar numbers reported in Europe. Dog bites (figure 1) cost over $1 billion per year in the USA.[9] Roughly 60% of animal bites are related to dogs, with 10–20% attributed to cats. Cat bites are more common in women and the elderly.[10,11] Individuals aged under 20 years and men are associated with a higher incidence of injury.[12,13]

A common and controversial topic is the risk of bite injury posed by each specific dog breed. Pit bull terriers lead the list of most commonly reported aggressors, followed by Rottweiler and German shepherd breeds.[14] Larger dog breeds can cause more severe crush injuries due to the higher pressures exerted by their jaws, and can pose a greater risk of major organ or vessel injury than do smaller animals. Most bite exposures occur in young children, involve unrestrained dogs on the owner's property, and about 20% involve a non-neutered dog.[15,16] Risk is highest in young boys aged 5–9 years. Because of their small size and lack of insight about provocative behaviour, children might be more likely to provoke a bite by simple play, teasing, or abuse. Because of the child's height in relation to the dog's mouth, children often have bites to the face, neck, or head.[17]

Figure 1: **Dog-bite patient with extensive facial injuries**

Figure 2: **Septic arthritis of left first proximal interphalangeal joint**

	Aerobes	Anaerobes
Cat bites	*Pasteurella* spp, *Streptococcus* spp, *Staphylococcus* spp, *Moraxella* spp	*Fusobacterium* spp, *Bacteroides* spp, *Porphyromonas* spp
Dog bites	*Pasteurella* spp, *Streptococcus* spp, *Staphylococcus* spp, *Neisseria* spp	*Fusobacterium* spp, *Bacteroides* spp, *Porphyromonas* spp, *Prevotella* spp, *Capnocytophaga* spp

*Most infections include a mix of aerobes and anaerobes.

Table: **Major pathogens isolated from dog and cat bites*[17]**

Most exposures in adults are to the hand, followed by bites to the face, scalp, neck, and thigh or leg.[18]

Cat bites are most likely to involve the upper extremities and face,[19] but are less destructive and life threatening. However, cats' narrow sharp teeth inflict deeper puncture wounds than dogs, and tend to carry a higher risk of infection and soft-tissue abscess. If occurring close to joints or bones, such bites might be complicated by septic arthritis (figure 2). Patients presenting more than 8 h after a bite injury often have infected wounds, with cat bites progressing to infection more rapidly than dog bites.

Microbiology

Most bite infections contain a mix of aerobes and anaerobes from both the skin of the patient and the mouth of the animal (table). Some of the common pathogens in cat bites include *Streptococcus* spp (including *Streptococcus pyogenes*), *Staphylococcus* spp (including MRSA), *Fusobacterium* spp, *Bacteroides* spp, *Porphyromonas* spp, *Moraxella* spp, and most commonly *Pasteurella multocida*. Like cat bites, the canine pathogens involved are usually a mix of aerobes and anaerobes. Commonly found pathogens include *Pasteurella canis*, *Streptococcus* spp, *Staphylococcus* spp, *Fusobacterium* spp, *Bacteroides* spp, and *Capnocytophaga canimorsus*.[17]

Severe infections caused by *C canimorsus* have been described in chronic alcohol users and in asplenic and immunocompromised patients. In a series of 39 cases of *C canimorsus* sepsis, Pers and colleagues[20] found that 56% of cases were related to dog bites, and 10% were due to licking. Case mortality was 31%.

Clinical manifestations

Severe infections related to dog and cat bites can occur in about 20% of all cases.[21] Depending on the predominant organism, infection can become systemic with bacteraemia or other serious sequelae. Because of the number of small enclosed compartments and fascial planes, numerous small nerves, and several bones and joints adjacent to the surface, hands are the most common bite site to develop infection and long-term morbidity. They are also the most likely site to develop rapidly spreading and permanently disabling damage. By contrast with other sites, 30–40% of hand bites become infected. Cat bites are more likely to penetrate deeply, particularly into bones and joints, leaving only a small skin opening from which fluids can drain. Therefore, deep abscesses (spread along fascial planes) and osteomyelitis are more common with cat bites.[21–23]

Bites to the head and neck are particularly severe in infants and children. Dogs can create depressed or open skull fractures, severe scalp and intracranial bleeding, and severely disfiguring damage to the face or damage to major vessels and nerves in the neck and thoracic inlet. Such damage is often associated with high mortality. Cats can scratch or bite the face. However, head and neck injuries caused by cats are much less serious.[24,25]

Management

Proper treatment of cat and dog bites should incorporate treatment of the immediate injury (superficial and possibly in deeper tissue) and management of the risk of acute infection (panel). Although most therapy for cat and dog bites tends to be empirical, wound swabs (preferably deep) for Gram stain and aerobic and anaerobic cultures should be obtained for each infected wound before therapy. Gram stains tend to be specific indicators of infection, but have low sensitivity.[28] Careful assessment of wound characteristics is crucial for management, because deep wounds are at greater risk for abscess formation after primary closure. Prompt and generous irrigation with tap water or normal saline helps to dislodge foreign matter and bacteria inoculated into the wound during the bite, and can reduce the transmission of rabies virus. Careful debridement of devitalised tissues, including probing for embedded teeth or fragments, is crucial. A radiograph should be obtained of the site of injury to rule out presence of a foreign body, bony involvement, or fracture. Additional diagnostic testing such as CT or MRI should be used if clinically indicated. In the case of bites to the hand, orthopaedic specialist involvement is important, along with subsequent elevation, immobilisation, and hand physiotherapy. A plastic surgeon should be consulted in the case of most head and neck bites. Because of an ample vascular supply, face-related wounds can be associated with a lower risk of infection and can be closed for cosmesis. Neurosurgical consultation might be indicated in children with possible cranial injury.

Consultation with local public-health authorities should be strongly considered for any serious domestic pet bite, particularly in the case of a stray animal, if the attack was unprovoked, if the pet cannot be apprehended, or if the pet's rabies vaccine status is unknown. The circumstances of the bite exposure and the animal's behaviour and whereabouts should be carefully documented.

Rabies prophylaxis should be considered, on the basis of exposure risk and local epidemiological information. In addition, the need for tetanus vaccine or booster for the patient should be addressed. Re-examination should be scheduled at 48 h.[24,29] The possibility of subsequent post-traumatic stress disorder should not be under-estimated and might warrant education of the patient and anticipatory guidance.

Prophylactic antibiotics are advised unless the bite is very superficial and easily cleansed. Treatment should be directed toward the pathogens that colonise the oral cavity of the aggressor animal. Because *Pasteurella* spp are usually not susceptible to dicloxacillin, cephalexin, clindamycin, or erythromycin, these antibiotics should not be used as monotherapy in the treatment of domestic animal bites. Amoxicillin–clavulanic acid provides excellent coverage against *P multocida*, *Capnocytophaga* spp, anaerobes, and susceptible *S aureus*, and is a mainstay of oral prophylaxis. Doxycycline with or without metronidazole is a suitable combination to cover these organisms in penicillin-allergic patients. Alternatives include clindamycin with a fluoroquinolone, or clindamycin plus trimethoprim–sulfamethoxazole (co-trimoxazole) in children. Ceftriaxone can be used in pregnancy, and cefuroxime and cefpodoxime are oral alternatives that also are generally safe (class B) to use. If community-acquired MRSA incidence is high in a region, this data should inform the selection of appropriate antibiotics. Doxycycline is an effective oral alternative for coverage of MRSA, as is co-trimoxazole, which is more appropriately for use in children. Clindamycin might be used if the community strain of MRSA is not likely to carry mutations for inducible clindamycin resistance.[24]

Established infection is likely to require admission to hospital for surgical debridement and drainage. Cultures of purulent fluid should be obtained before initiating new antibiotics to ensure appropriate antibiotic choice and to narrow subsequent therapy decisions. Wound swabs are relatively uninformative due to the likelihood of culturing contaminating organisms. Suitable antibiotics for inpatients include a β-lactam–β-lactamase inhibitor combination, such as ampicillin–sulbactam, piperacillin–tazobactam, or ticarcillin–clavulanic acid. Ceftriaxone, aztreonam, or a fluoroquinolone with excellent Gram-negative coverage plus metronidazole are suitable alternatives, as is monotherapy with a carbapenem such as ertapenem, meropenem, doripenem, or imipenem–cilastatin.[30]

Panel: Management and treatment of dog and cat bites[21,23,26,27]

Cultures
Gram stain, aerobic, and anaerobic cultures are indicated if abscess, severe cellulitis, devitalised tissue, or sepsis present

Irrigation
Normal saline irrigation copiously with high-pressure jet from syringe

Debridement
Debride necrotic tissue and remove any foreign bodies

Imaging
Plain radiographs, MRI, or CT if fracture or bone penetration, to rule out osteomyelitis

Wound closure
Primary wound closure is not usually indicated

Antimicrobial therapy
Prophylactic antibiotics in selected cases. Coverage based on patient type and specific animal involved

Hospitalisation
Indications include fever, sepsis, spreading cellulitis, substantial oedema or crush injury, loss of function, immunocompromised status, or noncompliance

Immunisations
Tetanus booster if original three-dose series has been given but none in the past year. Give primary series and tetanus immunoglobulin if the patient was never vaccinated Human diploid rabies vaccine (days 0, 3, 7, 14, and 28) with rabies immunoglobulin may be required (assess exposure risk)

Septic syndromes

Sepsis can be a severe complication of bite wounds, particularly those infected with *C canimorsus*, *P multocida*, *Staphylococcus* spp (including MRSA), and *Streptococcus* spp. Meningitis, endocarditis, and peritonitis can also complicate bite-wound infections. Several other species, including *Bacteroides*, *Fusobacterium*, *Neisseria*, and *Prevotella*, might also produce bite-wound sepsis in individuals with leukaemia and lupus, and in those receiving chronic steroids. The clinical picture in these subgroups follows the same pattern as in other disseminated infections. Because the overwhelming proportion of non-staphylococcal or non-streptococcal septicaemic infections are due to *Pasteurella* spp and *C canimorsus*, we will focus on these two syndromes. Reviews of the less common pathogens have been published elsewhere.[31]

Capnocytophaga canimorsus
Epidemiology
Initially identified as Centers for Disease Control group dysgonic fermenter 2 (DF-2), *C canimorsus* was first isolated in 1976 from the blood and spinal fluid of a dog-bite patient and later became known as the "dog-bite organism".[32] In 1989, Brenner and colleagues[33] proposed naming the group *C canimorsus* due to its association with dog bites. *C canimorsus* (DF-2) strains are oxidase and catalase positive. The organism has been isolated as

normal flora in dogs, cats, and from patients after dog or cat bites. A separate group of capnocytophaga, known as dysgonic fermenter 1 (DF-1), originates from the human oral cavity and are oxidase and catalase negative. They cause disseminated infections only in severely immunocompromised patients.[34] The *Capnocytophaga* genus consists of nine species. All are common to human and canine flora, but only *C canimorsus* causes severe infections in human beings.

The spectrum of *C canimorsus* infections can range from cellulitis to meningitis and endocarditis.[35,36] After the first report in 1976, 160 more cases have been published, including gangrene, sepsis, meningitis, and endocarditis. Most cases are associated with an underlying immune disorder, including splenectomy, chronic alcohol use, or cirrhosis. However, no identifiable risk factor has been found in 40% of cases.[37]

Capnocytophaga spp do not produce endotoxin and infection is of relatively low incidence in those without immunodeficiency. Its pathogenicity is not fully understood, but production of a substance that inhibits neutrophil motility has been described by Shurin and colleagues,[38] and seems to be associated with moderate resistance to phagocytosis. Furthermore, gliding motility might facilitate migration of the organism into the vascular space. Serum bactericidal resistance has been recognised in blood isolates, which is thought to result from a mutated lipopolysaccharide structure of the cell wall that inhibits phagocytosis.[39–41]

Severe sepsis caused by *C canimorsus* is extremely rare. Most infections occur in individuals older than 40 years. Almost 80% report some form of canine exposure, 58% report a bite, and 20% report exposure without a bite or scratch (such as licking of broken skin).[20] Mortality from sepsis can range from 25–30% to as high as 60% in patients with septic shock. 60% of those who develop septic shock die within 30 days.[20,42]

Clinical manifestations

The panorama of clinical features ranges from mild to fulminant, and includes fever, chills, myalgia, vomiting, diarrhoea, abdominal pain, malaise, dyspnoea, mental confusion, and headache. After an incubation period of 1–7 days, patients can experience an abrupt onset of malaise, abdominal pain that might mimic an acute abdominal syndrome, confusion, shortness of breath, and rapid progression to severe septic shock. On physical examination, patients present with a petechial rash on the trunk, lower extremities, and mucous membranes. The rash might evolve from purpuric lesions to gangrene. The clinical manifestations of sepsis are secondary to a profound inflammatory response leading to microvascular injury of the endothelium, resulting in disseminated intravascular coagulation, acute respiratory distress, gangrene, and organ damage. Patients with severe manifestations might develop septic shock that can progress to multiorgan failure and death.[42–44] Individuals

aged more than 50 years are at greatest risk. Endocarditis caused by *C canimorsus* is not extensively reported in the literature; however, there are many reports of bacteraemia and sepsis in which a septic focus was never found. As with other fastidious Gram-negative infections, the published record of cases might underestimate the true incidence of *C canimorsus* infection. Clearly, myocarditis, as well as culture-negative, acute, or subacute endocarditis, might occur. Classical findings that are associated with endocarditis, such as heart murmur, fever, and increased C-reactive protein, might be unapparent at presentation. In addition, infection might occur in individuals without previous cardiac pathology. A history of a recent dog bite in a patient presenting with clinical features of endocarditis should raise suspicion for *C canimorsus* infection.[45]

The circumstances under which this microorganism causes meningitis in human beings is not clear. Although septic shock attributable to infection with *C canimorsus* has frequently been described, the occurrence of meningitis seems rare. Apart from a possible bite wound found at physical examination, no other symptom is known to discriminate between this rare form of meningitis and meningitis caused by other microorganisms. The detection of small Gram-negative bacilli in the cerebrospinal fluid supports the diagnosis; however, no bacteria were initially found in about a third of reported cases. *C canimorsus* meningitis should be suspected if signs and symptoms of meningitis coincide with a history of a recent dog or cat bite.[34]

A few cases of fatal acute haemorrhagic adrenal insufficiency (Waterhouse–Friderichsen syndrome) caused by *C canimorsus* infection have also been described. Gangrene and purpura fulminans might occur as a consequence of widespread inflammation, endothelial damage, hypoperfusion, and disseminated intravascular coagulation.[43,44,46]

Management

Diagnosis is usually based on clinical history because the bacteria are extremely difficult to grow. Up to 14 days of incubation might be necessary to detect growth on typical media. Peripheral blood smear and observation of abundant intracytoplasmic fusiform rods within neutrophils allows a presumptive diagnosis in the correct setting.[42,47] *Capnocytophaga* spp infection responds well to penicillin and β-lactam–β-lactamase inhibitor combinations. Other active agents include clindamycin, linezolid, tetracycline, carbapenems, and chloramphenicol. Antibiotics with variable activity include erythromycin, rifampicin, quinolones, metronidazole, vancomycin, latamoxef, aztreonam, penicillins, and cephalosporins. Polymyxin B and E, fusidic acid, fosfomycin, aminoglycosides, and trimethoprim have limited activity. Because of the relatively aggressive nature of *C canimorsus* and the difficulty in obtaining a laboratory diagnosis, therapy should be started as early as possible.[40,42]
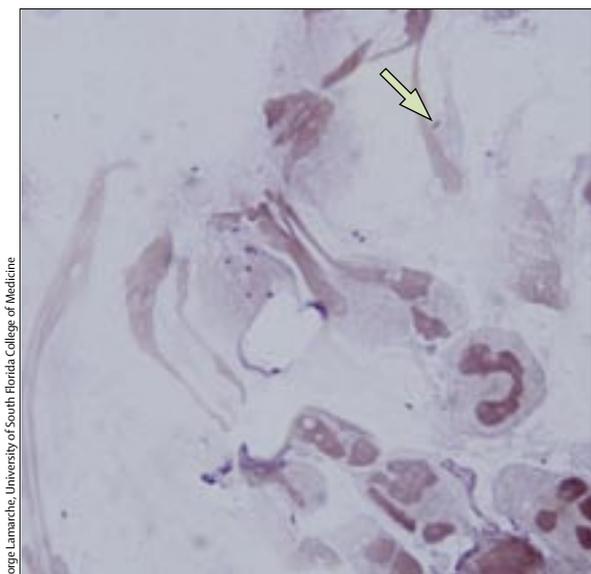
Figure 3: Gram-positive, non-spore-forming bacilli consistent with *Pasteurella multocida* from a peritoneal dialysis patient with *Pasteurella*-associated peritonitis

Pasteurella spp
Epidemiology
Pasteurella spp are Gram-positive, facultative anaerobic, non-spore-forming bacilli that can be seen in pairs or short chains (figure 3). Most strains are positive for catalase, oxidase, indole, sucrose, and decarboxylate ornithine. Potential virulence factors include capsular lipopolysaccharide, a cytotoxin, and iron acquisition proteins. The subtypes associated with human infections include *P multocida* subsp *multocida*, *P canis*, *P multocida* subsp *septica*, *P stomatis*, and *P dagmatis*.[48,49] *P multocida* can be found as part of the normal flora of the upper respiratory tract of some mammals, particularly cats. Most human infections are caused by dog or cat bites, although licks from these animals have also been associated with infection.[50–53] Exposures involving other types of animal contact and some with no known animal contact have been described.[54]

Clinical manifestations
Pasteurella spp can cause serious infections, including necrotising fasciitis, septic arthritis, osteomyelitis, and less commonly, sepsis, septic shock, and meningitis.[51,52,55] Severe infection (ie, sepsis and septic shock) can be seen in infants, pregnant women, patients on chronic steroids, HIV-positive individuals, organ-transplant recipients, and other immunocompromised patients.[52,55,56] Bacteraemia occurs in 25–50% of patients with pneumonia, meningitis, and septic arthritis due to *P multocida*. Many patients with bacteraemia have evidence of notable liver disease.[52,57,58] However, rare cases of bacteraemia have also been described in previously healthy individuals. In such cases, the mortality remains substantial at 25%.[52]

Pasteurella meningitis is a disease of the extremes of life, particularly infants younger than 1 year and adults older than 60 years.[52,59] Results of cerebrospinal-fluid examination are similar to other bacterial meningitides, with increased white blood cell count, high protein concentration, and low glucose concentration. Gram stain of the cerebrospinal fluid is positive in 80% of patients, but the organisms can be confused with *Haemophilus influenzae* or *Neisseria meningitidis*.

Endocarditis is a rare complication of *P multocida* infection that can involve native or prosthetic valves.[60,61] Peritonitis due to *P multocida* can occur in the setting of continuous ambulatory peritoneal dialysis (figure 3).[58]

Management
A clinical history of a dog or cat bite should prompt the diagnosis. Risk of disseminated disease and septic shock should be suspected in patients with risk factors such as liver disease. Pasteurella is not a difficult organism to isolate and identify because the bacterium grows well on various commercial media, including chocolate and sheep-blood agar. However, it does not grow well on certain selective enteric media. Recovery from wounds, blood cultures, peritoneum, or other typical sites of oxidase-positive, Gram-negative bacilli that are strongly indole positive but fail to grow on MacConkey agar is usually sufficient to identify the organism.[62]

Like *C canimorsus*, *P multocida* is not susceptible to many oral antibiotics typically given for skin and soft-tissue infections, including dicloxacillin, cephalexin, and clindamycin. In addition, many strains are resistant to erythromycin (although susceptible to azithromycin). In most cases, treatment with betalactam antibiotics, such as penicillin or ampicillin, is effective. However, penicillin-resistant *Pasteurella* spp have been reported.[63] Other options include use of second-generation and third-generation cephalosporins (eg, cefuroxime, cefpodoxime), and doxycycline or fluoroquinolones in penicillin-allergic patients.[52]

Community-associated meticillin-resistant *Staphylococcus aureus* infections
Epidemiology
MRSA, long recognised as a human pathogen, is notorious for causing a wide variety of disease syndromes. MRSA has long been recognised as a pathogen associated with hospital or health-care settings.[64] Community-acquired MRSA strains have been increasing in prevalence over the past decade. These organisms and the most common genotype, USA300, are distinct from traditional hospital-acquired strains in that they are easily transmitted in the household, are a frequent cause of skin and soft-tissue infections, are generally susceptible to most antibiotics other than β-lactams, and produce the Panton-Valentine leukocidin fusion protein.[65]

As community-acquired strains increase in prevalence, a growing body of clinical evidence has documented

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Figure 4: **MRSA-associated infection of the left forearm of a 15-year-old patient (A) whose cat had developed recurrent MRSA culture-positive skin lesions of the perineal area (B)**

MRSA colonisation in domestic animals, often implying direct acquisition of *S aureus* infection from their human owners.[66–69] MRSA colonisation has been documented in companion animals such as horses, dogs, and cats, and these animals have been viewed as potential reservoirs of infection.[70–72] Paradoxically, although *S aureus* is the predominant staphylococcal strain in horses and human beings, *Staphylococcus intermedius* is the overwhelming species in cats and dogs, with *S aureus* comprising less than 10% of feline and canine animal strains.[73]

The recognition of *S aureus* as a potential coloniser of domestic animals was first documented in 1959, when Mann[74] reported isolating *S aureus* from the nares of 23 of 100 dogs and concluded that, "the common house pet can serve as an important reservoir or carrier of staphylococcal infective [sic] for man." This observation was reinforced by Live and Nichols[75] in 1961. At the dawn of the MRSA era, Live and Nichols[76] wrote that, "'antibiotic-resistant staphylococci' have become established in veterinary hospitals, which might act as a source of the organisms for human beings associated with them, as well as for hospitalised animals." In the early 1980s, experimental transmission of *S aureus* from

dogs to kennel attendants was also reported.[77] In 1988, Scott and colleagues[71] reported the first published case of domestic animal transmission of MRSA strains in a UK rehabilitation geriatric unit from a ward cat that was believed to be heavily colonised from the environment. Screening of patients and staff revealed that 38% of nursing staff were colonised. The outbreak was promptly aborted when appropriate infection-control measures were instituted and the cat was removed from the ward. 6 years later, Cefai and colleagues[72] reported a second UK case of MRSA associated with a domestic pet. An epidemic strain (designated EMRSA1) of MRSA was isolated from a patient in an intensive-care unit. The strain was traced to a nurse and his spouse, who was also a nurse working in another ward. Despite a decolonisation attempt, both nurses were then linked to a second outbreak in the unit 6 months later. The intensive-care nurse later revealed that his dog had developed an eye infection for several weeks. Swabs of the pet confirmed the presence of EMRSA1. Simultaneous decolonisation of the entire household (both nurses and the dog) was ultimately successful.

Since the initial reports of MRSA transmission between human beings and pets,[72] more sophisticated procedures for typing bacterial pathogens have become available. DNA fingerprinting techniques have further documented the transmission of MRSA strains between human beings and household pets. Simoons-Smit and colleguese[78] used genetic fingerprinting to analyse eight non-resistant strains of *S aureus* obtained from a patient with recurrent *S aureus* infections, her relatives, and her household pets. The index patient suffered from frequently relapsing pyoderma associated with congenital bullous ichthyosiform erythroderma. *S aureus* strains were isolated from the patient, her mother, boyfriend, and from the cat and dog. All individuals and the two pets were then prescribed a 5-day eradication protocol consisting of nasal mupirocin, topical povidone–iodine shampoo and chlorhexidine gluconate body wash, and oral cephadrine as an antibiotic agent. Swabs of the axilla of the index patient and the armpit of the cat were still positive after the 5-day protocol. Genetic analysis of all specimens showed indistinguishable patterns among all eight strains (96·1% [SD 1·2]).

In a similar investigation, Manian[70] used pulse-field gel electrophoresis to isolated a strain of mupirocin-resistant MRSA from the nares of a healthy dog whose owner had experienced repeated episodes of soft-tissue staphylococcal infections. The chromosomal pattern was identical for the human and canine strains. In 2006, Vitale and colleagues[69] isolated MRSA from the skin lesions of a 3-year-old domestic short-haired cat with flea allergy and pyoderma. Identification of MRSA was shown by detection of penicillin binding protein 2a and by pulse-field gel electrophoresis, and the isolate was confirmed as a USA300 clone. This was the first confirmed case of USA300 community-acquired MRSA isolated from a household pet.[69]

Recent analysis of pet-associated MRSA strains has shown evidence of a possible role of pets in the transmission of fluoroquinolone-resistant strains to human beings. Lin and Davies[79] identified strains of multidrug-resistant *S aureus* isolated from dogs and analysed their relation to strains of human origin by use of multilocus sequence typing and analysis with the eBURST programme. Two canine strains were identified as sequence type 239, an epidemic MRSA strain common in human isolates, and both showed high-level resistance to the fluoroquinolones. The investigators concluded that the treatment of infections in animals with fluoroquinolones might contribute to the emergence of fluoroquinolone-resistant strains in pets that might then be transferred to human beings. In a more in-depth investigation of MRSA susceptibility patterns among domestic animals, Morris and colleagues[80] did a retrospective review of 139 *S aureus* isolates cultured from cats and dogs treated at the University of Pennsylvania Veterinary Hospital (Philadelphia, PA, USA), and analysed antibiotic susceptibility profiles for each isolate. 35% (39 of 111) of *S aureus* strains were MRSA, and among the 39 MRSA strains, susceptibility was modest for clindamycin (28%), erythromycin (15%), and fluoroquinolone agents (10%), suggesting that MRSA strains in pets are likely to be multidrug resistant. The most reliable oral drugs from the panel were chloramphenicol (90%) and co-trimoxazole (97%).

Clinical manifestations

Scientific reports of the clinical manifestations of MRSA infections on cats or dogs in the veterinary literature are uncommon, but a few case reports have been published. Tomlin and colleagues[81] reported a case series of 11 dogs with MRSA infection. Eight of the 11 cases were associated with surgical procedures, most of which were orthopaedic procedures, and three were related to atopic dermatitis. Susceptibility data revealed that all MRSA strains were vancomycin susceptible, but that some were resistant to macrolides and fluoroquinolones. Most of the strains were susceptible to co-trimoxazole and tetracyclines. Of the eight surgical cases, six dogs were returned to the operating room to manage their infections. Oral antibiotic therapy with culture and susceptibility data improved or resolved MRSA infections in nine of 11 dogs (one dog was euthanised and one was lost to follow-up). MRSA-related skin infections of pets seem to occur in various manifestations, including simple dermatitis, pustular disease, and even perineal cellulitis, and can be easily spread to owners (figure 4).

Management

Specific therapy for pet-associated MRSA infections is similar to regimens used in most community-acquired MRSA syndromes. Contact with an asymptomatic pet is not a risk factor for sensitive or immunocompromised patients for acquiring *S aureus*, because most household pets are not likely to be MRSA colonised. Since most infections are skin and soft-tissue related, mild-to-moderate infections can be treated with oral anti-staphylococcal agents. Oral co-trimoxazole, doxycycline or minocycline, or clindamycin are the mainstays of therapy. Oral linezolid is often used an alternative for more severe infections. A second oral antibiotic should be added if streptococci are suspected and co-trimoxazole or doxycycline have been given because these drugs have relatively poor coverage against *S pyogenes*. For more complicated suppurative or systemic infections, parenteral therapy with a glycopeptide antibiotic (vancomycin or teicoplanin), daptomycin, linezolid, tigecycline, or quinupristin–dalfopristin should be considered.

Although the transmission of pathogenic strains of MRSA between human beings and domestic animals has been well documented, their drug susceptibility profile analysed, and their role in pet-associated and human infections further studied, much more remains to be learned about MRSA and pet-associated human infections. Future research needs to improve understanding of the dog–MRSA host–pathogen relations, to enhance recognition of the mechanisms of canine–human transmission, and to elucidate the virulence factors in domestic pet-associated strains.

Conclusion

Pet owners are often unaware of the potential for transmission of life-threatening pathogens from their canine and feline companions. Bite injuries are a major cause of injury in the USA and Europe each year, particularly in children. Bites to the hands, forearms, neck, and head have the potential for the highest morbidity. Common pathogens in these injuries include oral anaerobes, *P multocida*, *C canimorsus*, and MRSA. Treatment of cat and dog bites should include wound assessment, deep culture, radiography, debridement, wound management, and rabies prophylaxis if applicable. Antibiotic therapy should be directed towards the anticipated pathogens. Health-care providers are at the forefront of protecting the vital relationships between people and their pets. Clinicians must continue to promote loving pet ownership, take an adequate pet history, and be aware that associated diseases are preventable via recognition, education, and simple precautions.

Review

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