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146 Infectious Complications of Bites

frontal, parietal, and occipital superior cerebral veins and the diploic veins, which communicate with the meningeal veins. Bacterial menin gitis is a common predisposing condition for septic thrombosis of the superior sagittal sinus. The diploic veins, which drain into the superior sagittal sinus, provide a route for the spread of infection from the meninges, especially in cases where there is purulent exudate near areas of the superior sagittal sinus. Infection can also spread to the superior sagittal sinus from nearby SDE or epidural abscess. Dehydration from vomiting, hypercoagulable states, and immunologic abnormalities, including the presence of circulating antiphospholipid antibodies, also contribute to cerebral venous sinus thrombosis. Thrombosis may extend from one sinus to another, and at autopsy, thrombi of different histologic ages can often be detected in several sinuses. Thrombosis of the superior sagittal sinus is often associated with thrombosis of supe rior cortical veins and small parenchymal hemorrhages.

The superior sagittal sinus drains into the transverse sinuses (Fig. 145-6). The transverse sinuses also receive venous drainage from small veins from both the middle ear and mastoid cells. The transverse sinus becomes the sigmoid sinus before draining into the internal jugu lar vein. Septic transverse/sigmoid sinus thrombosis can be a complica tion of acute and chronic otitis media or mastoiditis. Infection spreads from the mastoid air cells to the transverse sinus via the emissary veins or by direct invasion. The cavernous sinuses are inferior to the superior sagittal sinus at the base of the skull. The cavernous sinuses receive blood from the facial veins via the superior and inferior ophthalmic veins. Bacteria in the facial veins enter the cavernous sinus via these veins. Bacteria in the sphenoid and ethmoid sinuses can spread to the cavernous sinuses via the small emissary veins. The sphenoid and eth moid sinuses are the most common sites of primary infection resulting in septic cavernous sinus thrombosis. PART 5 Infectious Diseases ■ ■CLINICAL MANIFESTATIONS Septic thrombosis of the superior sagittal sinus presents with head ache, fever, nausea and vomiting, confusion, and focal or generalized seizures. There may be a rapid development of stupor and coma. Weakness of the lower extremities with bilateral Babinski’s signs or hemiparesis is often present. When superior sagittal sinus thrombosis occurs as a complication of bacterial meningitis, nuchal rigidity and Kernig’s and Brudzinski’s signs may be present. The oculomotor nerve, the trochlear nerve, the abducens nerve, the ophthalmic and maxillary branches of the trigeminal nerve, and the internal carotid artery all pass through the cavernous sinus (see Fig. 452-7). The symptoms of septic cavernous sinus thrombosis are fever, headache, frontal and retroorbital pain, and diplopia. The classic signs are ptosis, proptosis, chemosis, and extraocular dysmotil ity due to deficits of cranial nerves III, IV, and VI; hyperesthesia of the ophthalmic and

maxillary divisions of the fifth cranial nerve and a decreased corneal reflex may be detected. There may be evidence of dilated, tortuous retinal veins and papilledema. Headache and earache are the most frequent symptoms of trans verse sinus thrombosis. A transverse sinus thrombosis may also pres ent with otitis media, sixth nerve palsy, and retroorbital or facial pain (Gradenigo's syndrome). Sigmoid sinus and internal jugular vein thrombosis may present with neck pain. ■ ■DIAGNOSIS The diagnosis of septic venous sinus thrombosis is suggested by an absent flow void within the affected venous sinus on MRI and confirmed by contrast-enhanced magnetic resonance venography, CT venography, or the venous phase of cerebral angiography. The diagnosis of thrombophlebitis of intracerebral and meningeal veins is suggested by the presence of intracerebral hemorrhage but requires the venous phase of cerebral angiography for definitive diagnosis. TREATMENT Suppurative Thrombophlebitis Septic venous sinus thrombosis is treated with antibiotics, hydra tion, and removal of infected tissue and thrombus in septic lateral

or cavernous sinus thrombosis. The choice of antimicrobial therapy is based on the bacteria responsible for the predisposing or asso ciated condition. Optimal duration of therapy is unknown, but antibiotics are usually continued for 6 weeks or until there is radiographic evidence of resolution of thrombosis. Anticoagulation with unfractionated or low-molecular-weight heparin is recom mended for aseptic venous sinus thrombosis and in the treatment of septic venous sinus thrombosis complicating bacterial meningitis in patients who have progressive neurologic deterioration despite antimicrobial therapy and intravenous fluids. The presence of a small intracerebral hemorrhage from septic thrombophlebitis is not an absolute contraindication to heparin therapy. Successful management of aseptic venous sinus thrombosis has been reported with surgical thrombectomy, catheter-directed urokinase therapy, and a combination of intrathrombus recombinant tissue plasmino gen activator (rtPA) and intravenous heparin, but there are not enough data to recommend these therapies in septic venous sinus thrombosis. ■ ■FURTHER READING Bodilsen J et al: European Society of Clinical Microbiology and Infec tious Diseases guidelines on diagnosis and treatment of brain abscess in children and adults. Clinical Microbiol Infect 30:66, 2024. Prosty C et al: Revisiting the evidence base for modern-day practice of the treatment of toxoplasmic encephalitis: A systematic review and meta-analysis. Clin Infect Dis 76:e1302, 2023. Ropper AH, Klein JP: Cerebral venous thrombosis. N Engl J Med 385:59, 2021. White AC et al: Diagnosis of Neurocysticercosis: 2017 Clinical Practice Guidelines by the Infectious Diseases Society of America (IDSA) and the American Society of Tropical Medicine and Hygiene (ASTMH). Clin Infect Dis 66:e49, 2018. Nongnooch Poowanawittayakom,

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Infectious Complications

of Bites The skin is an essential component of nonspecific immunity, protect ing the host from potential pathogens in the environment. Breaches in this protective barrier thus represent a form of immunocompromise that predisposes the patient to infection. Bites and scratches from animals and humans allow the inoculation of microorganisms past the skin's protective barrier into deeper, susceptible host tissues. Each year in the United States, millions of animal-bite wounds are sustained. The vast majority are inflicted by pet dogs and cats, which number >100 million; the annual incidence of dog and cat bites has been reported as 300 bites per 100,000 population. Other bite wounds are a consequence of encounters with animals in the wild or in occu pational

settings. While many of these wounds require minimal or no therapy, a significant number result in infection, which may be life-threatening. The microbiology of bite-wound infections in general reflects the oropharyngeal flora of the biting animal, although organ isms from the soil, the skin of the animal and the victim, and the animal's feces may also be involved. DOG BITES In the United States, dogs bite >4.7 million people each year and are responsible for 80% of all animal-bite wounds, an estimated 15–20% of which become infected. Each year, 800,000 Americans seek medical

attention for dog bites; of those injured, 386,000 require treatment in an emergency department, with >1000 emergency department visits each day and ~43 deaths per year. Most dog bites are provoked and are inflicted by the victim's pet or by a dog known to the victim. These bites are frequently sustained during efforts to break up a dogfight. Children are more likely than adults to sustain canine bites, with the highest inci dence of 6 bites per 1000 population among boys 5–9 years old. Victims are more often male than female, and bites most often involve an upper extremity. Among children <4 years old, two-thirds of all these injuries involve the head or neck. Infection typically manifests 8–24 h after the bite as pain at the site of injury with cellulitis accompanied by purulent, sometimes foul-smelling discharge. Septic arthritis and osteomyelitis may develop if a canine tooth penetrates synovium or bone. Systemic manifestations (e.g., fever, lymphadenopathy, and lymphangitis) also may occur. The microbiology of dog-bite wound infections is usu ally mixed and includes Pasteurella species, β-hemolytic streptococci, Staphylococcus species (including methicillin-resistant Staphylococcus aureus [MRSA] and Staphylococcus intermedius), Neisseria species (commonly Neisseria weaveri, formerly known as CDC group M-5), Eikenella corrodens, and Capnocytophaga canimorsus. Many wounds also include anaerobic bacteria such as Bacteroides, Fusobacterium, Prevotella, and Porphyromonas species. While most infections resulting from dog-bite injuries are localized to the area of injury, many of the microorganisms involved are capable of causing systemic infection, including bacteremia, meningitis, brain abscess, endocarditis, and chorioamnionitis. These infections are par ticularly likely in hosts with edema or compromised lymphatic drain age in the involved extremity (e.g., after a bite on the arm in a woman who has undergone mastectomy) and in patients who are immu nocompromised by medication or disease (e.g., glucocorticoid use, systemic lupus erythematosus, acute leukemia, or hepatic cirrhosis). In addition, dog bites and scratches may result in systemic illnesses such as rabies (Chap. 214) and tetanus (Chap. 157). Infection with Capnocytophaga canimorsus (and other Capnocyto phaga species) following dog-bite wounds (or licking of preexisting wounds) may result in fulminant sepsis, disseminated intravascular coagulation, and renal failure, particularly in hosts who have impaired hepatic function, who have undergone splenectomy, or who are immu nosuppressed. This thin gram-negative rod is difficult to culture on most solid media but grows in a variety of liquid media. It may require up to 14 days of incubation to grow on blood cultures. The bacteria are occasionally seen within polymorphonuclear leukocytes on Wrightstained smears of peripheral blood from septic patients. Tularemia (Chap. 175) also has been reported to follow dog bites. CAT BITES Although less common than dog bites, cat bites and scratches result in infection in more than half of all cases. Because the cat's narrow, sharp canine teeth penetrate deeply into tissue, cat bites are more likely than dog bites to cause septic arthritis and osteomyelitis; the development of these conditions is particularly likely when punctures are located over or near a joint, especially in the hand. Women sustain cat bites more frequently than do men. These bites most often involve the hands and arms. Both bites and scratches from cats are prone to infec tion from organisms in the cat's oropharynx. Pasteurella

multocida, a normal component of the feline oral flora, is a small gram-negative coccobacillus implicated in the majority of cat-bite wound infections. Like that of dog-bite wound infections, however, the microflora of cat-bite wound infections is usually mixed. However, the median time from bite to the appearance of signs and symptoms of wound infection is much shorter when compared to dog bites. Other microorganisms causing infection after cat bites are similar to those causing dog-bite wound infections. The same risk factors for systemic infection following dog-bite wounds apply to cat-bite wounds. Pasteurella infections tend to advance rapidly, often within hours, causing severe inflammation accompanied by purulent drainage with adenitis; Pasteurella may also be spread by respiratory droplets from animals, resulting in pneumonia or bacteremia. Like dog-bite wounds, cat-bite wounds may result in

the transmission of rabies or in the development of tetanus. Infection with Bartonella henselae causes cat-scratch disease (Chap. 177) and is an important late consequence of cat bites and scratches. Tularemia (Chap. 175) also has been reported to follow cat bites. Occasionally, sporotrichosis (Chap. 225) has been associated with scratches or bites by animals, especially domestic cats.

OTHER ANIMAL BITES Infections have been attributed to bites from many animal species. Often these bites are sustained as a consequence of occupational exposure (farmers, laboratory workers, veterinarians) or recreational exposure (hunters and trappers, wilderness campers, owners of exotic pets). Generally, the microflora of bite wounds reflects the oral flora of the biting animal. Most members of the cat family, including feral cats, harbor P. multocida. Bite wounds from aquatic animals such as alliga tors or piranhas may contain Aeromonas hydrophila. Shark, moray eel, and barracuda bites, like other injuries sustained in saltwater, are often associated with infections with marine Vibrio species. Venomous snakebites (Chap. 471) result in severe inflammatory responses and tis sue necrosis—conditions that render these injuries prone to infection. The snake’s oral flora includes many species of aerobes and anaerobes, such as Pseudomonas aeruginosa, Serratia marcescens, Proteus species, Staphylococcus epidermidis, Salmonella species, Bacteroides fragilis, and Clostridium species. Bites from nonhuman primates are highly suscep tible to infection with pathogens similar to those isolated from human bites (see below). Bites from Old World monkeys (Macaca) may also result in the transmission of B virus (Macacine herpesvirus 1, Herpes virus simiae, Cercopithecine herpesvirus), a cause of serious infection of the human central nervous system. Actinobacillus lignieresii has often been reported in infected wounds of humans bitten by horses, pigs, and sheep. Bites of seals, walruses, and polar bears may cause a chronic sup purative infection known as seal finger, which is probably due to one or more species of Mycoplasma, including Mycoplasma phocacerebrale, colonizing these animals. CHAPTER 146 Infectious Complications of Bites Small rodents, including rats, mice, and gerbils, as well as animals that prey on rodents may transmit Streptobacillus moniliformis (a microaerophilic, pleomorphic gram-negative rod) or Spirillum minor (a spirochete); these organisms cause a clinical illness known as rat-bite fever. The vast majority of cases in the United States are streptobacil lary, whereas Spirillum infection occurs mainly in Asia. In the United States, the risk of rodent bites is usually greatest among laboratory workers or inhabitants of rodent-infested dwellings (particularly children). Rat-bite fever is distinguished from acute bitewound infection by its typical manifestation after the initial wound has healed. Streptobacillary disease follows an incubation period of 3–10 days. Fever, chills, myalgias, headache, and severe migratory arthralgias are usually followed by a maculopapular rash, which char acteristically involves the palms and soles and may become

confluent or purpuric. Complications include endocarditis, myocarditis, menin gitis, pneumonia, and abscesses in many organs. Haverhill fever is an S. moniliformis infection acquired from contaminated milk or drinking water and has similar manifestations. Streptobacillary rat-bite fever was frequently fatal in the preantibiotic era. The differential diagnosis includes Rocky Mountain spotted fever, Lyme disease, leptospirosis, and secondary syphilis. The diagnosis is made by direct observation of the causative organisms in tissue or blood, by culture of the organisms on enriched media, or by serologic testing with specific agglutinins. Spirillum infection (referred to in Japan as sodoku) causes pain and purple swelling at the site of the initial bite, with associated lymphan gitis and regional lymphadenopathy, after an incubation period of 1–4 weeks. The systemic illness includes fever, chills, and headache. The original lesion may eventually progress to an eschar. The infection is diagnosed by direct visualization of the spirochetes in blood or tissue or by animal inoculation. HUMAN BITES Human bites may be self-inflicted; may be sustained by medical per sonnel caring for patients; or may take place during fights, domestic abuse, or sexual activity. The risk of infection in human-bite wounds

depends on the depth of the wound. The risk of wound infection ranges from about 2% in superficial wounds to over 25% in deep bite wounds such as clenched-fist injuries. Human-bite wounds become infected more frequently (~10–15% of the time) than do bites inflicted by other animals. These infections reflect the diverse oral microflora of humans, which includes multiple species of aerobic and anaerobic bac teria. Common aerobic isolates include viridans streptococci, S. aureus, E. corrodens (which is particularly common in clenched-fist injury; see below), and Haemophilus influenzae. Anaerobic species, includ ing Fusobacterium nucleatum and Prevotella, Porphyromonas, and Peptostreptococcus species, are isolated from 50% of wound infections due to human bites; many of these isolates produce β-lactamases. The oral flora of hospitalized and debilitated patients often includes Entero bacteriaceae in addition to the usual organisms. Hepatitis B, hepatitis C, herpes simplex virus infection, syphilis, tuberculosis, actinomycosis, and tetanus have been reported to be transmitted by human bites; it is biologically possible to transmit HIV through human bites, although the risk is quite low. In general, postexposure prophylaxis should be considered for bites involving severe trauma with extensive tissue dam age and the presence of blood in saliva. There is essentially no risk of transmission if the skin is intact.

Human bites are categorized as either occlusional injuries, which are inflicted by actual biting, or clenched-fist injuries, which are sustained when the fist of one individual strikes the teeth of another, causing traumatic laceration of the hand. For several reasons, clenched-fist injuries, which are sometimes referred to as “fight bite” and which are more common than occlusional injuries, result in particularly serious infections. The deep spaces of the hand, includ ing the bones, joints, and tendons, are frequently inoculated with organisms in the course of such injuries. The clenched position of the fist during injury, followed by extension of the hand, may further promote the introduction of bacteria as contaminated tendons retract beneath the skin’s surface Moreover, medical attention is often sought only after frank infection develops. Patients with clenched-fist injury should undergo careful physical examination of the area, including the extensor tendons. PART 5 Infectious Diseases APPROACH TO THE PATIENT Animal or Human Bites A careful history should be elicited, including the type of biting animal, the type of attack (provoked or unprovoked), and the amount of time elapsed since injury. Local and regional publichealth authorities should be contacted to determine whether an individual species could be rabid and/or to locate and observe the biting animal when rabies prophylaxis may be indicated (Chap. 214). Suspicious human-bite

wounds should provoke careful ques tioning regarding domestic or child abuse. Details on antibiotic allergies, immunosuppression, splenectomy, liver disease, mastec tomy, and immunization history should be obtained. The wound should be inspected carefully for evidence of infection, including redness, exudate, and foul odor. The type of wound (puncture, laceration, or scratch); the depth of penetration; and the pos sible involvement of joints, tendons, nerves, and bones should be assessed. It is often useful to include a diagram or photograph of the wound in the medical record. In addition, a general physical examination should be conducted and should include an assess ment of vital signs as well as an evaluation for evidence of lym phangitis, lymphadenopathy, dermatologic lesions, and functional limitations. Injuries to the hand warrant consultation with a hand surgeon for the assessment of tendon, nerve, and muscular dam age. Radiographs should be obtained in penetrating wounds to evaluate the evidence of fracture or retained foreign body such as a tooth fragment. Culture and Gram's staining of all infected wounds are essential; anaerobic cultures should be undertaken if abscesses, devitalized tissue, or foul-smelling exudate is present. A small-tipped swab may be used to culture deep punctures or small lacerations. It is also reasonable to culture samples from

apparently uninfected wounds due to bites inflicted by animals other than dogs and cats, since the microorganisms causing dis ease are less predictable in these cases. The microbiology labora tory should be notified if fastidious organisms such as E. corrodens are under consideration in human bites. The white blood cell count should be determined, and the blood cultured if systemic infection is suspected. TREATMENT Bite-Wound Infections WOUND MANAGEMENT Wound closure is controversial in bite injuries. Many authori ties prefer not to attempt primary closure of wounds that are or may become infected, choosing instead to irrigate these wounds copiously, debride devitalized tissue, remove foreign bodies, and approximate the wound edges. All abscesses should be drained. Delayed primary closure may be undertaken after the risk of infec tion is over. Small uninfected wounds may be allowed to close by secondary intention. Puncture wounds due to cat bites should be left unsutured because of the high rate at which they become infected. Facial wounds are usually sutured after thorough cleaning and irrigation because of the importance of a good cosmetic result in this area and because anatomic factors such as an excellent blood supply and the absence of dependent edema lessen the risk of infec tion. In general, wounds >12 h old (for bites to the arm or leg) or

> 24 h old (for bites to the face) should not be closed primarily and may require prophylactic antibiotics (see below). ANTIBIOTIC THERAPY Established Infection Antibiotics should be administered for all established bite-wound infections and should be chosen in light of the most likely potential pathogens, as indicated by the biting species and by Gram's stain and culture results (Table 146-1). For dog and cat bites, antibiotics should be effective against S. aureus, Pasteurella species, C. canimorsus, streptococci, and oral anaerobes. For human bites, agents with activity against S. aureus, H. influenzae, and β-lactamase-positive oral anaerobes should be used. The combination of an extended-spectrum penicillin with a β-lactamase inhibitor (amoxicillin/clavulanic acid, ampicillin/sulbactam) appears to offer the most reliable coverage for these pathogens. Third-generation cephalosporins (ceftriaxone, cefpo doxime) also offer

> substantial coverage when given in conjunc tion with a drug that provides anaerobic coverage (clindamycin or metronidazole). The choice of antibiotics for penicillin-allergic patients (particularly those in whom immediate-type hypersensi tivity makes the use of cephalosporins hazardous) is more difficult and is based primarily on in vitro sensitivity since data on clinical efficacy are inadequate. The combination of an antibiotic active against gram-positive cocci and anaerobes (such as clindamy cin or metronidazole) with trimethoprim-sulfamethoxazole or a fluoroquinolone, which is active against many of the other potential pathogens, would appear reasonable. Moxifloxacin, a fluoroquinolone with anaerobic coverage, can also be considered as a single agent. In vitro data suggest that azithromycin alone provides coverage against most commonly isolated bite-wound pathogens; however, this agent has variable activity against

P. multocida, E. corrodens, and fusobacteria and thus should be avoided unless no alternative agent is available. Empirical use of agents active against MRSA should be considered in high-risk situations while culture results are awaited. Antibiotics are generally given for about 5–7 days and for no more than 14 days, but the response to therapy must be carefully monitored. Failure to respond should prompt a consideration of diagnostic alternatives and surgical evaluation for possible drain age or debridement. Complications such as osteomyelitis or septic arthritis mandate a longer duration of therapy.

TABLE 146-1 Management of Wound Infections Following Animal and Human Bites COMMONLY ISOLATED PATHOGENS PREFERRED ANTIBIOTIC(S)a BITING SPECIES Dog Staphylococcus aureus, Pasteurella spp. (mainly

P. multocida and

P. canis), anaerobes, Capnocytophaga canimorsus Amoxicillin/clavulanate (875/125 mg PO q12h) or ampicillin/sulbactam

(3.0 g IV q6h) or Ceftriaxone 2 g IV once daily plus metronidazole 500 mg q8h Cat P. multocida, S. aureus, anaerobes Amoxicillin/clavulanate, ampicillin/sulbactam, or ceftriaxone plus metronidazole as above Human, occlusional Viridans streptococci, S. aureus, Haemophilus influenzae, anaerobes Eikenella corrodense Amoxicillin/clavulanate plus TMX-SMX if consider including MRSA coverage or

ampicillin/sulbactam

or ceftriaxone plus metronidazole (consider adding vancomycin if MRSA coverage required) Monkey As for human bite As for human bite As for human bite Always For macaque monkeys, consider B virus prophylaxis with acyclovir. Snake Snake oral flora including Pseudomonas, Morganella spp., E. coli, group D streptococci, Salmonella spp., anaerobic organisms including Bacteroides fragilis, Clostridium spp. Piperacillin/tazobactam

3.375 g IV q6–8h Rodent Rat bite fever; Streptobacillus moniliformis, Spirillum minus, Streptobacillus notomytis, Leptospira spp., P. multocida Penicillin VK (500 mg PO qid) or ceftriaxone

IV Aquatic animal (alligator, piranha, shark, moray eel, barracuda) Aeromonas hydrophila, marine Vibrio spp. (Vibrio vulnificus) Third-generation cephalosporin (e.g., ceftriaxone, 1 g IV q24h) plus doxycycline (100 mg PO bid) aAntibiotic choices should be based on culture data when available. These suggestions for empirical therapy need to be tailored to individual circumstances and local conditions. MRSA empirical coverage is based on individual risk factors. IV regimens should be used for hospitalized patients. A single IV dose of antibiotics may be given to patients who will be discharged after initial management. bAvoid monotherapy for aerobic coverage due to poor activity against Pasteurella spp. Risk of Clostridioides difficile infection. cCat bite may leave small external wounds but deep puncture wounds. dProphylactic antibiotics are suggested for severe or extensive wounds, facial wounds, and crush injuries; when bone or joint may be involved; delayed wound care >8 h; and when comorbidity is present (see text). Prophylactic antibiotic duration is generally between 3 and 5 days. eEikenella corrodens is resistant to penicillinase-resistant penicillin, first and second generation cephalosporins, clindamycin, metronidazole, and aminoglycosides in vitro. Abbreviations: DS, double-strength; TMP-SMX, trimethoprim-sulfamethoxazole. Management of C. canimorsus bacteremia requires an initial treatment with a 2-week course of IV antibiotics such as penicillin G (2 million units IV every 4 h) or IV ampicillin/sulbactam (3.0 g every 6 h) along with supportive measures. Once the patient is improved, then a switch to oral antibiotics can be considered. Alter native agents for the treatment of C. canimorsus infection include cephalosporins or carbapenems. Serious infection with P. multocida (e.g., pneumonia, sepsis, or meningitis) also should be treated with IV penicillin G. Alternative agents include a second- or thirdgeneration cephalosporin or ciprofloxacin. Penicillin resistance is uncommon. Bites by venomous snakes (Chap. 471) may not require antibi otic treatment. Because it is often difficult to distinguish signs of infection from tissue damage caused by the envenomation, many authorities continue to recommend treatment directed against the snake’s oral flora—i.e., the administration of broadly active agents such as ceftriaxone (1–2 g IV every 24 h) or ampicillin/sulbactam (3.0 g IV every 6 h). Seal finger appears to respond to doxycycline (100 mg twice daily for a duration guided by the response to therapy).

ALTERNATIVE IN PENICILLIN-ALLERGIC PATIENT PROPHYLAXIS ADVISED FOR EARLY UNINFECTED WOUNDS OTHER CONSIDERATIONS Clindamycinb or metronidazole plus either TMP-SMX

(1 DS tablet PO bid) or ciprofloxacin (500 mg PO bid) Sometimesc Consider rabies prophylaxis. Clindamycin or metronidazole plus TMP-SMX as above or fluoroquinolone Usuallyd Consider rabies prophylaxis. Carefully evaluate for joint/bone penetration. TMP-SMX plus metronidazole Always Clindamycin or metronidazole plus a fluoroquinolone Evidence does not support the benefit. Can consider in regions with high rates of infection such as Brazil. Administer antivenin for venomous snakebite. Tetanus prophylaxis. CHAPTER 146 Doxycycline (100 mg PO bid) Sometimes Infectious Complications of Bites Clindamycin or metronidazole plus levofloxacin (750 mg PO qd) plus doxycycline Always Obtain prompt surgical consultation, as risk for necrotizing infection is high with Aeromonas and Vibrio spp. Presumptive or Prophylactic Therapy The use of antibiotics for patients presenting early (within 8 h) after bite injury is controver sial. Although symptomatic infection frequently will not yet have manifested at this point, many early wounds will harbor pathogens, and many will become infected. Studies of antibiotic prophylaxis for wound infections are limited and have often included only small numbers of cases in which various types of wounds have been man aged according to various protocols. A meta-analysis of eight ran domized trials of prophylactic antibiotics in patients with dog-bite wounds demonstrated a reduction in the rate of infection by 50% with prophylaxis. However, in the absence of sound clinical trials, many clinicians

base the decision to treat bite wounds with empirical antibiotics on the species of the biting animal; the location, severity, and extent of the bite wound; and the existence of comorbid condi tions in the host. All human- and monkey-bite wounds should be treated presumptively because of the high rate of infection. Most catbite wounds, particularly those involving the hand, should consider prophylactic antibiotics. Other factors favoring treatment for bite wounds include severe injury, as in crush wounds; potential bone or joint involvement; involvement of the hands or genital region; host

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