Rat Bite Fever

Streptobacillus moniliformis Infection:
Streptobacillary Fever,
Streptobacillosis,
Streptobacilliosis,
Epidemic Arthritic Erythema,
Haverhill Fever

Spirillum minus Infection:
Sodoku,
Spirillary Fever

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Importance

Rat bite fever is a human illness that can be caused by *Streptobacillus moniliformis* and other zoonotic bacteria in the genus *Streptobacillus*, as well as the unrelated organism *Spirillum minus.* Although this disease is readily cured with antibiotics, complications such as endocarditis are possible and untreated infections are sometimes fatal. The causative organisms are primarily acquired from rodents, especially rats. At one time, rat bite fever was mainly a hazard of exposure to wild rats or laboratory rodents; however, control programs have largely eliminated *S. moniliformis* from the latter setting. Conversely, pet owners, pet shop employees and veterinary staff may be at risk with the increased popularity of rodent pets. There is also growing evidence that some cases might be acquired from other animal hosts.

Reports of illnesses in animals are generally sparse, though *S. moniliformis* is known to cause outbreaks in mice and may cause major economic losses if it affects laboratory colonies. There have also been sporadic reports of conditions such as pneumonia in felids, polyarthritis in turkeys, and rat bite fever-like illnesses in a dog and captive primates. Clinical cases in both humans and animals might be underdiagnosed, as the initial clinical signs are nonspecific, a diagnosis of rat bite fever requires specialized culture conditions or PCR tests, and the causative organisms usually respond to commonly administered empirical antibiotics.

Etiology

Rat-bite fever can be caused by several bacteria in the genus *Streptobacillus* and by the unrelated bacterium *Spirillum minus*. The two forms of disease in people are known, respectively, as streptobacillary rat bite fever (or Haverhill fever for orally acquired cases) and spirillary rat bite fever. Cases caused by *Streptobacillus* in animals are sometimes called streptobacillosis.

Members of the genus *Streptobacillus* are Gram negative, pleomorphic bacilli in the family Leptotrichiaceae. At one time, *Streptobacillus moniliformis* was thought to be the only member of this genus, and it was considered to be responsible for all clinical cases. However, several new *Streptobacillus* species have been identified in the last decade. They include *S. canis* and *S. ratti*, which have only been described from animals to date, and *S. notomytis* and *S. felis*, which have also been found in human rat bite fever cases. All of these organisms appear identical to *S. moniliformis*, even in most PCR tests, unless detailed genetic analysis is done. Another new organism, *S. hongkongensis*, differs genetically from all the other species, seems to be carried only in humans, and was recently renamed *Pseudostreptobacillus hongkongensis*. Guinea pig strains of *S. moniliformis*, which were isolated from abscesses and other diseases in these animals, were reclassified as a separate organism, *Caviibacter abscessus*, in 2016, and are no longer discussed in this factsheet.

Sp. minus is a short, thick, Gram negative spiral. It has never been cultivated in artificial media, and much about it, including its taxonomic relationships, is poorly understood.

Species Affected

Streptobacillus

The hosts for *Streptobacillus* spp. are currently difficult to define with certainty, given the recent recognition that there is more than one species in this genus and the difficulties in distinguishing these organisms. Rats are thought to be the reservoir hosts for *S. moniliformis*, which they usually carry asymptomatically. This organism has been documented in both *R. norvegicus*, the Norwegian rat, which is the ancestor of most laboratory and pet rats, and *Rattus rattus*, the black rat. However, finding other *Streptobacillus* species in black rats raises the possibility that *S. moniliformis* might have been misidentified in this host. Mice can be infected with *S. moniliformis*, either with or without clinical signs, and might sometimes maintain this organism for a time. How long it persists in mice is uncertain, with estimates varying from no persistence to 6 months.

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Information on the newer *Streptobacillus* species is still limited. *S. notomytis* is now known to be the organism, originally identified as *S. moniliformis*, that caused an outbreak of septicemia in spinifex hopping mice (*Notomys alexis*) at a zoo in the early 1980s. It has also been found in both asymptomatic and sick black rats. *S. ratti* has been detected in asymptomatic black rats. *S. felis* appears to be common in the oropharynx of healthy domestic cats, but caused cases of pneumonia in a domestic cat and a captive rusty-spotted cat (*Prionailurus rubiginosus*). *S. canis* was isolated from a wound infection in a dog. Dogs have been implicated in a few human cases of rat bite fever, presumably after eating or biting rodents, and an older report, published before *S. canis* was described, found nucleic acids of *S. moniliformis* in the mouths of some dogs. It remains to be determined whether some of these animals might have been colonized with *S. canis*.

Streptobacilli have been found sporadically in other species. There are a few reports linking *S. moniliformis* to diseases in turkeys and clinical cases in non-human primates, dogs, an owl and a koala (*Phascolarctos cinereus*). One study reported that turkeys, but not chickens, were susceptible to an isolate from an outbreak in these birds. Bacteria similar to *S. moniliformis* (described as ''*S. actinoides*") were found in the lungs of calves and sheep with pneumonia, although it is not certain they were the same organism. Organisms were also isolated from seminal vesicles in bulls. Gerbils and African squirrels are potential hosts for *S. moniliformis* or other streptobacilli, based on their (rare) association with human cases of rat bite fever. Animals that eat rodents, including ferrets, a weasel and a pig, might also be infected or colonized, as there is suggestive evidence linking them to a few human cases. In addition, unspecified *Streptobacillus* have been found in nonhuman primates, marine mammals (dolphins, sea lions), fish and birds (e.g., ducks).

Spirillum minus

Very little is known about *Sp. minus* infections in animals. Rats are thought to be the reservoir hosts for this organism, and carry it asymptomatically. *Sp. minus* is also reported to infect mice, and illnesses were reported in experimentally infected guinea pigs and rhesus macaques. The susceptibility of rabbits seems uncertain: some older research found that they develop clinical signs after inoculation, while other reports did not. One clinical case in a person occurred after a cat bite.

Zoonotic potential

The vast majority of rat bite fever cases have been attributed to *S. moniliformis*, but *S. notomytis*, *S. felis* and *Sp. minus* have also caused similar illnesses. It seems plausible that humans are also susceptible to other species of *Streptobacillus* such as *S. ratti* and *S. canis*.

Geographic Distribution

Streptobacillary rat bite fever has been documented on most continents. *S. moniliformis* seems to be cosmopolitan. There is little information yet on other species of *Streptobacillus*, which were mostly described in the last 5-10 years. As of 2021, *S. notomytis* has been found in Australia and parts of Asia and Europe, and is likely to be widespread. *S. felis* and *S. canis* were initially recognized in Europe, and *S. ratti* in Asia (Japan).

Human infections with *Spirillum minus* have been reported mainly from Asia, but occasional cases of rat bite fever have been attributed to this organism in North America, Europe and Africa. However, the limitations of diagnostic testing may cast doubt on some of these reports. For example, one early case of "spirillary rat bite fever" in Edinburgh occurred in a patient who also had organisms consistent with *S. moniliformis* in the blood. The case was attributed to *Sp. minus* (and the bacteria in the blood were dismissed as a secondary contaminant) because a few spirilla were recovered from the patient's enlarged lymph nodes, and because it was the only organism thought to cause rat bite fever at the time. Neither organism was found in another early case from the U.S., but the disease was attributed to *Sp. minus* because spirilla were seen in the blood of wild mice on the property where the patient was bitten.

Transmission

S. moniliformis appears to be a commensal organism in the nasopharyngeal flora of rats. It has also been found in the middle ear, salivary gland, larynx and upper trachea, and experimental infections have been established in rats by oronasal or parenteral inoculation. Proposed methods of transmission from rats to other animals include bites, aerosols and transmission on fomites, including food or water, that have been contaminated with secretions or excretions. Mice have been experimentally infected by intranasal, oral or parenteral inoculation, and by contact with rats. Oral colonization might be possible in carnivores that have bitten or eaten rodents, but how often this happens and how long the organisms might be carried is unclear.

Sp. minus is carried in asymptomatic rats, but there is little or no definitive information on its transmission. This organism has been detected in blood and possibly in conjunctival exudates. Whether it is shed in saliva, and under what conditions, is unclear. Rodents can be infected by inoculating them with contaminated blood or tissues. Some early experiments suggested that *Sp. minus* was not transmitted readily between mice by casual contact, or between guinea pigs in bites before they develop conjunctivitis.

People usually become infected with *Streptobacillus* spp. and *Sp. minus* after bites or scratches from rats or other animals, but there are reports of clinical cases after other forms of contact, including handling a rat, being exposed to its urine, kissing it and sharing food. There are also some cases of rat bite fever with no obvious exposure to rodents or other hosts. Haverhill fever results from eating or drinking food or water that has been contaminated with rat excrement. Person-to-person transmission of zoonotic *Streptobacillus* or *Sp. minus* has not been reported.

Disinfection

S. moniliformis is susceptible to various disinfectants including 70% ethanol, sodium hypochlorite, accelerated hydrogen peroxide and quaternary ammonium compounds. *Sp. minus* has not been cultured in artificial media and its disinfectant susceptibility is unknown.

Infections in Animals

Incubation Period

Experiments in mice suggest that clinical signs develop within a week or two after exposure to *S. moniliformis*. Guinea pigs inoculated with clinical specimens containing *Sp. minus* became symptomatic in approximately 1-2 weeks in one experiment and 14-18 days in another. This organism was found in the blood of guinea pigs in 5-37 days, or the blood of mice in 5-30 days.

Clinical Signs

Streptobacillus moniliformis

Rats usually carry *S. moniliformis* asymptomatically, but this organism has occasionally been reported as a secondary invader in subcutaneous abscesses, bronchopneumonia, chronic pneumonia, conjunctivitis, otitis media and other conditions. Mice are more likely to become ill, and may develop septic lymphadenitis, arthritis, various other purulent lesions, and acute or subacute septicemia. Septic lymphadenitis tends to affect the ventral cervical lymph nodes at first, but can spread to other superficial lymph nodes. Mice with septicemia from *S. moniliformis* may die unexpectedly with few or no clinical signs or experience a brief, nonspecific fatal illness. Conjunctivitis, photophobia, cyanosis, diarrhea, anemia, hemoglobinuria and emaciation may also be seen. Dermatitis, characterized by brown crusts over the mammae of nursing females, was reported in one outbreak. Pregnant mice may abort or give birth to stillborn pups. Reported sequelae of infection in mice include chronic arthritis, with swelling of the limbs or tail; deformities, ankylosis, or spontaneous amputation of the limbs or tail; and spinal column involvement, which may lead to posterior paralysis, kyphosis and priapism.

Rare clinical cases have been attributed to *S. moniliformis* in other species, with varying levels of evidence for its involvement. This organism was isolated from an abscess in one dog, and found in another dog with a fatal illness characterized by anorexia, diarrhea, vomiting, arthritis in the hind legs, and lesions of pneumonia and endocarditis. Septic arthritis and endocarditis were described in two naturally infected nonhuman primates, and experimentally infected rhesus macaques can develop a febrile illness. Pleuritis caused by *S. moniliformis* was reported in a koala, and pneumonia in calves. Several outbreaks have been seen in turkeys, with clinical signs that included polyarthritis, synovitis, tendon sheath swelling and joint lesions. Some of these infections were fatal. A tawny owl with infected feet was reported in the U.K.

Other species of *Streptobacillus* have been documented in only a few clinical cases, to date. Sudden death was the main syndrome during an outbreak of septicemia caused by *S. notomytis* in spinifex hopping mice at a zoo. Although this organism has been found in asymptomatic black rats, it also caused an outbreak of bilateral, high-grade, purulent otitis media and otitis interna among black rats in a zoo exhibit. The otitis resulted in various neurological signs including disorientation, torticollis, stall walking and ataxia in some animals. Sudden death was also seen. *S. felis* was found in a cat with acute bronchopneumonia, endocarditis and myocarditis, which was found dead on a farm. This organism also caused pneumonia in a rusty-spotted cat in a zoo. The latter animal had respiratory distress, bilateral blepharitis, weakness, and anorexia, and was euthanized due to disease progression. However, *S. felis* also appears to be common in the oropharynx of healthy domestic cats. *S. canis* was isolated from a superficial wound infection in a dog. *S. ratti* has only been found in asymptomatic black rats, to date.

Spirillum minus

Rats usually carry *Sp. minus* asymptomatically. A few older studies described experimental infections in rodents; however, they used very crude preparations (e.g., tissue isolates from a rat bite fever patient, or blood containing spirilla from rats or sick guinea pigs), and it is difficult to be sure that *Sp. minus* was the causative organism. Guinea pigs became ill after inoculation with organisms from rats or tissues from a person with rat bite fever. The clinical signs included fever, conjunctivitis, keratitis, lymphadenopathy, weight loss and hair loss. Some infections were fatal. The same inocula did not cause illness in rats or mice, although spirilla were found in the blood. A febrile illness was reported in experimentally infected rhesus macaques. One study found that rabbits developed edematous, indurated, inflammatory lesions at the inoculation site, followed by regional lymphadenopathy and edema of the face (eyelids, lips, nose, base of the ears) and genitals. In other studies, rabbits did not become infected.

Post Mortem Lesions

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Septic lymphadenitis in laboratory mice can involve multiple subcutaneous lymph nodes, but the ventral cervical lymph nodes are usually the first to be affected. Mice with acute septicemia may die with few lesions, but other animals can have typical septicemic lesions such as multifocal, suppurative, embolic, interstitial nephritis, focal necrosis of the spleen and liver, splenomegaly and lymphadenopathy. In other cases in mice, the predominant finding is septic polyarthritis with numerous subcutaneous and periarticular abscesses. Fibrosis of the joints, joint deformation and spontaneous amputation of the limbs and tails may be seen in some animals. Spinifex hopping mice with septicemia from *S. notomytis* primarily had liver lesions including hepatomegaly, necrosis and microabscesses.

Lesions reported in other species vary but range from wound infections to pneumonia, arthritis/ polyarthritis, endocarditis, myocarditis and septicemic lesions. Bilateral

high-grade (purulent) otitis media and interna was the only lesion in black rats infected by *S. notomytis* at a zoo, though some infections were fatal.

Diagnostic Tests

Streptobacillus spp.

Infections with *Streptobacillus* can be diagnosed by isolation of the organism, molecular techniques or serology Most of these tests were developed to monitor colonies of laboratory animals for *S. moniliformis* and diagnose outbreaks in these animals, but some are also appropriate for clinical cases in other species.

Streptobacillus spp. can be difficult to culture from clinical specimens, and the laboratory should be informed that a member of this genus is suspected. These organisms are fastidious, must be grown in media enriched with serum, blood or ascitic fluid, and are readily overgrown by other bacteria. *S. moniliformis*, at least, is also known to be inhibited by sodium polyanethol sulphonate (SPS), an anticoagulant that is often used in automatic blood culture systems. A recent study suggests that this limitation can be overcome by the use of larger blood volumes (10ml). On solid media, *Streptobacillus* spp. usually appear as tiny, smooth, grayish colonies; however, most species other than *S. canis* readily form cell wall deficient L-forms, which have a ‘‘fried egg’’ colony appearance similar to *Mycoplasma*. Microscopic examination reveals Gram negative, pleomorphic bacteria which can occur as single rods (occasionally with lateral bulbar swellings) or coccobacilli, or in long, unbranched filamentous or fusiform chains, sometimes forming loops or curls. The form can vary with the medium and the age of the culture, and clumps of *S. moniliformis* may look like proteinaceous debris in some specimens. *S. moniliformis* does not always stain well with Gram stains. Alternatives include carbol fuchsin or Giemsa. Staining with acridine orange and examination under a fluorescent microscope may also aid visualization.

Organisms can be identified to the genus level by conventional biochemical and carbohydrate fermentation analysis, PCR, sequencing of the 16S rRNA gene or gas-liquid chromatographic analysis of the fatty acid profile. PCR can also be used directly on clinical specimens. *S. moniliformis* PCR tests can also detect other *Streptobacillus* species such as *S. felis*, *S. notomytis* and *S. ratti*. The latter organisms are indistinguishable from *S. moniliformis* in some of these assays; however, one recently described PCR test is stated to specifically distinguish *S. moniliformis* from the other organisms, though it amplifies all of the species. Specialized genetic techniques only available at research laboratories must be used for further species identification. The presence of *Leptotrichia* spp. can cause false positives in a least some PCR tests.

Serological tests are mainly used to monitor SPF laboratory animal colonies for *S. moniliformis*. Agglutination and complement fixation tests were used in the past, but they have generally been replaced by ELISAs and indirect immunofluorescence. False positive reactions in the ELISA test can be recognized by immunoblotting (Western blotting) and PCR. Some laboratory animals may not develop antibodies after infection.

Spirillum minus

Spirillum minus cannot be cultured in artificial media. Detection of this organism has relied on finding organisms with the typical morphology in darkfield or phase contrast preparations, or after Giemsa, Wright or silver staining. *Sp. minus* is a short, spiral-shaped, Gram-negative (or Gram variable) rod that is 0.2-0.5 µm by 3-5 µm and reported to have 2-3 coils (although some sources report more) and bipolar tufts of flagella. If microscopy is unsuccessful, inoculation into mice, guinea pigs or *Sp. minus*-free rats has been used for diagnosis in human cases. Spirochetes may be found in the blood of these animals after 5–15 days, using dark-field microscopy. Because *Sp. minus* cannot be cultured, no serological or molecular (PCR) tests are available.

Treatment

There are few published reports of treatment in animals. As with rat bite fever in humans, penicillins may be a good option (in species that do not have adverse reactions to these drugs), but a number of other antibiotics are also expected to be effective. During one outbreak in mice, breeding animals were treated with ampicillin in the drinking water, together with tetracycline to prevent the survival of penicillin-resistant L-forms. Although most of the mice recovered, some later relapsed and died of septicemia. Another group reported that streptomycin was more effective than penicillin in experimentally infected mice with arthritis, although penicillin was also used successfully. A dog with an abscess attributed to *S. moniliformis* recovered after treatment with 'strepto-penicillin' antibiotics. The organism in the latter case was described as having resistance to many antibiotics during *in vitro* testing. Incision and drainage of abscesses or other adjunct treatment may also be needed.

Control

Disease reporting

Clinical cases from *Streptobacillus* spp. or *Sp. minus* in pets and livestock are unlikely to be reportable; however, veterinarians should remain aware of any national and/or local guidelines, as well as any guidelines for infected laboratory animals. State guidelines should be checked in the U.S.

Prevention

Most colonies of laboratory rodents used in research have been cleared of *S. moniliformis*, and are monitored regularly for this organism. It is also possible to generate cesarean derived, barrier maintained SPF breeding colonies for rodent pets, though these animals are often conventionally bred. Captive rodents, including pets, should be protected from contact with wild rats or other animals that may carry *S. moniliformis* or *Sp. minus*.

Morbidity and Mortality

S. moniliformis appears to be relatively common in rats, and has been estimated to occur in 50-100% of wild Norway and black rats. What proportion of these infections may be other species, such as *S. ratti* or *S. notomytis*, is not known. *S. moniliformis* was also found in 10-100% of laboratory rats at one time, but it has become rare in these animals with the advent of cesarean derived, barrier maintained SPF colonies. However, organisms can still be found in conventionally bred rats, including some pets. Data on *Sp. minus* are sparse, but up to 25% of the wild rats in some countries are thought to carry this organism.

Clinical cases seem to be uncommon in rats, though it is possible that some illnesses are overlooked due to lack of awareness or limited diagnostic testing. Mice are more likely to become ill, and some outbreaks caused by *S. moniliformis* are severe, with morbidity and mortality rates that can approach 100%. However, susceptibility can differ between strains of mice, and certain experimentally infected inbred mice can become bacteremic but remain asymptomatic.

Relatively little is known about *Streptobacillus* spp. in cats, dogs or other species. *S. felis* appears to be common in the oropharynx of healthy domestic cats, and one study found *S. moniliformis* DNA in the mouth of 15% of dogs that had contact with rats. (It is possible that the latter organism might have been another species, such as *S. canis*.) A few clinical cases have been reported in dogs, cats, zoo animals, turkeys and livestock. Whether these cases are actually rare is uncertain, given the difficulties in diagnosis and the broad susceptibility of these organisms to antibiotics, which are often given empirically.

Infections in Humans

Incubation Period

The incubation period for streptobacillary rat bite fever ranges from 2 days to more than 3 weeks, but most cases seem to develop in less than 7-10 days. In one case series, septic arthritis appeared in 4 days to 7 weeks.

The reported incubation period for spirillary rat bite fever ranges from one day to a month, with some sources suggesting that it might sometimes be as long as 4 months. Most cases are thought to appear more than 10 days after a bite. The U.S. Centers for Disease Control and Prevention (CDC) estimates an incubation period of 1-3 weeks.

Clinical Signs

Streptobacillary rat bite fever

Most descriptions of streptobacillary rat bite fever are based on patients infected with *S. moniliformis;* however, cases caused by *S. notomytis* and *S. felis* appear to be similar.

Wounds infected by *S. moniliformis* usually heal without complications, often before the first signs of rat bite fever appear. This illness usually begins abruptly, with a fever and chills. Other common symptoms include severe myalgia and joint pain, headache, nausea and vomiting. Infants and young children can develop severe diarrhea, which may lead to dehydration and weight loss. Most patients also have a maculopapular, purpuric or petechial rash. The rash occurs most often on the extremities, especially the hands and feet, but it can sometimes involve the entire body. Hemorrhagic vesicles, pustules and papules, which are very tender, may also be seen. Many cases of rat bite fever resolve spontaneously within two weeks, but complications and deaths are possible in untreated cases.

At least half and perhaps as many as 75% of all patients develop polyarthritis or polyarthralgia, the most common complication. It often appears within a week of the initial signs, commonly affecting the knees, ankles, shoulders, elbows, wrists and/or hands. It is sometimes migratory, and can persist for months or even several years, with periods of remission and exacerbation. Most patients have nonsuppurative (sterile) arthritis, which might be caused by an immunological mechanism. Septic arthritis, which often but not always involves multiple joints, is reported infrequently, and can be caused by *S. notomytis* and *S. felis*, as well as *S. moniliformis*. It might be more common in people with joint abnormalities such as osteoarthritis. Septic and nonsuppurative streptobacillary arthritis are generally similar in appearance, and can be difficult to distinguish. However, septic arthritis is sometimes seen in patients who have relatively few of the typical signs of rat bite fever.

Other complications seem to be rare, but can be diverse, with reports of osteomyelitis, tenosynovitis, anemia, endocarditis, pericarditis, myocarditis, hepatitis, kidney dysfunction, systemic vasculitis, prostatitis, pancreatitis, meningitis, pneumonia, sepsis and focal organ abscesses, as well as one case of ammonitis (infection of the amniotic fluid). Endocarditis is often, but not always, seen in patients with damaged heart valves. Osteomyelitis at sites such as the vertebrae may lead to additional complications, including neurological signs.

Most patients with streptobacillary rat bite fever respond well to antibiotics, but prolonged migratory polyarthralgias, fatigue and slow resolution of the rash are possible. One person infected with *S. felis* had a partial response to initial treatment with amoxicillin/clavulanic acid but an unusually prolonged course of persistent, disabling myalgia, in addition to typical rat bite fever symptoms. The myalgia eventually resolved after additional antibiotics. Most deaths occur in infants and in patients who develop sepsis or endocarditis. Long-term complications were not reported in patients with septic arthritis after treatment.

Haverhill fever

Haverhill fever is very similar to streptobacillary rate bite fever, but pharyngitis and vomiting are more pronounced. Some patients have had severe arthralgia and frequent relapses.

Spirillary rat bite fever

Spirillary rat bite fever resembles streptobacillary rat bite fever, but an indurated, painful and often ulcerated skin lesion is usually seen at the site of the bite, and is often accompanied by regional lymphadenopathy. The skin lesion may appear when the fever develops, if the wound initially healed without complications. Patients with spirillary rat bite fever may experience febrile relapses separated by afebrile periods. These episodes may recur several times over 1-3 months, but rarely continue for more than a year. Although skin rashes are less common than in streptobacillary rat bite fever, some patients have a distinctive rash consisting of large violaceous or reddish macules. Erythematous plaques or urticaria may also be seen, especially near the site of the bite. Arthritis is uncommon, but other complications resemble those seen in streptobacillary rat bite fever (e.g., endocarditis, myocarditis, hepatitis and meningitis). Untreated cases can be fatal.

Diagnostic Tests

Streptobacillary rat bite fever

Streptobacillary rat bite fever in humans is usually diagnosed by culture and/or PCR, as in animals. Organisms or nucleic acids may be found in blood, other body fluids, affected tissues (e.g. abscesses, pustules) or the wound. They may be detected in synovial fluid in cases of septic arthritis, but in most patients, the joint fluid is sterile. Some clinical cases caused by *S. felis* and *S. notomytis* were diagnosed with PCR tests and/or culture that suggested *S. moniliformis*, followed by genetic analysis that revealed a different species of *Streptobacillus*.

Inoculation into rodents was used for diagnosis in the past, but other techniques (e.g., PCR) are now preferred if culture is unsuccessful. Serological tests used in the past (e.g., slide agglutination) were not considered to be reliable, and there are currently no validated serological tests for diagnosis in humans.

Spirillary rat bite fever

Spirillary rat bite fever is usually diagnosed by identifying spirilla consistent with *Sp. minus* in blood, exudates or tissues, including lymph node aspirates, the bite wound or erythematous plaques, as described in animals. If microscopy is unsuccessful, blood or wound aspirates can be inoculated into mice, guinea pigs or *Sp. minus*-free rats for diagnosis.

Treatment

The organisms that cause rat bite fever are susceptible to a number of antibiotics. Treatment of uncomplicated cases results in a shorter clinical course and may prevent severe complications. Penicillin is considered to be the treatment of choice for both *S. moniliformis* and *Sp. minus*, but streptomycin, tetracycline, doxycycline, some cephalosporins (e.g., ceftriaxone), carbapenems and other agents have also been used, and additional drugs have demonstrated *in vitro* susceptibility. Penicillin-resistant strains of *S. moniliformis* seem to be rare, although they have been reported. The choice of drug also depends on penetration into affected tissues, e.g., the joint in suppurative arthritis. Combinations of antibiotics have been recommended for *S. moniliformis* endocarditis, and prolonged treatment may be necessary. Some patients may also need adjunct treatments, such as arthroscopy, arthrotomy or joint lavage in some cases of septic arthritis.

Prevention

The risk of rat bite fever can be reduced by avoiding exposure to rodents, especially wild rats. Specific information on wild rodent control around households is available from the various sources, such as the CDC in the U.S. (see Internet Resources, below). SPF rodents, rather than conventional animals, should be used in laboratories or, if available, when breeding pets. All captive rodents, including pets, should be housed in areas free of wild rodents. Hand-to-mouth contact should be avoided when handling a rodent or cleaning its cage, and the hands should be washed after contact. Proper handling techniques can help prevent bites when handling rodents. Gloves and other PPE may also be helpful in some situations. Any bite wounds or scratches should be cleaned promptly and thoroughly. People who become ill after being bitten by a rodent should report their exposure history to ensure that rat bite fever is considered in the differential diagnosis.

To decrease the risk of Haverhill fever, food and water storage should be designed to prevent contamination with rodent excrement, and potentially contaminated water and food sources should be avoided. Pasteurization of milk and sterilization of drinking water are also helpful.

Morbidity and Mortality

Rat bite fever seems to be an uncommon disease; however, its incidence is uncertain and it may be underdiagnosed. Higher risk groups include the owners of pet rats, pet shop personnel and veterinarians, as well as people who are exposed to wild rats for various reasons, including household infestations. Laboratory workers were at elevated risk of rat bite fever at one time, but laboratories now mainly use SPF rats and mice. Human infections have occasionally been linked to bites from other animals, including African squirrels, a gerbil, a cat, dogs, non-human primates, ferrets, a weasel and a pig. The relative incidence of cases caused by *S. moniliformis* and other agents is currently uncertain. The latter cases were mostly diagnosed because a patient had persistent signs or there were other reasons to pursue advanced diagnostics.

Rat bite fever tends to be a sporadic disease, but it can also occur in outbreaks, especially when people are exposed to contaminated food or water. Large outbreaks of Haverhill fever were reported in Haverhill, MA in 1926; in Chester, USA in 1925; and at a boarding school in Essex, U.K. in 1983. The first two outbreaks were associated with contaminated, unpasteurized milk products, and the third was linked to contaminated water from a spring.

Rat bite fever is readily treated with antibiotics in most cases. Untreated *S. moniliformis* infections are estimated to be fatal in approximately 7-13% of cases, and untreated *Sp. minus* infections in approximately 7-10%. Most deaths occur in infants and in patients who develop cardiac complications (e.g., endocarditis) or sepsis. While endocarditis and pericarditis are rare, the case fatality rate in these conditions is estimated to be around 40-50%, with most deaths occurring in people who did not receive antibiotics effective against this organism.

Internet Resources

Centers for Disease Control and Prevention (CDC). Rat bite fever information

CDC. Information on wild rodent control

Public Health Agency of Canada. Pathogen Safety Data Sheets

The Merck Manual

The Merck Veterinary Manual

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