Sarcocystosis

Sarcosporidiosis,
Equine Protozoal Myeloencephalitis
Pigeon Protozoal Encephalitis

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Importance

Sarcocystosis is caused by members of the genus *Sarcocystis*, a protozoal parasite that is widely distributed in mammals, birds and reptiles. These organisms cycle through two hosts: the sexual stage of the parasite is produced in the intestines of the definitive host, and asexual replication takes place in various tissues of the intermediate host. While the vast majority of infections seem to be subclinical, *Sarcocystis* spp. sporadically cause myositis, encephalitis and other diseases in animals or humans. Serious illnesses usually occur only in the intermediate host, though self-limited enteric signs have been seen in some intestinal infections. Why some infected individuals become ill but others remain healthy is uncertain, but the dose of the parasites is thought to be one factor.

Reports of sarcocystosis seem to have increased in the last 30 years, probably as the result of increased awareness. Important organisms in clinical cases include *S. neurona* (equine protozoal myeloencephalitis) in equids, sea otters and occasionally other mammals; *S. calchasi* (pigeon protozoal encephalitis) and *S. falcatula* in birds, and *S. nesbitti* in humans. However, many other species of *Sarcocystis* also infect vertebrates and cause some illnesses.

Etiology

Sarcocystosis is caused by members of the genus *Sarcocystis*, an intracellular protozoal parasite in the phylum Apicomplexa. Most species of *Sarcocystis* have never been associated with overt disease; others have caused clinical cases but also occur in many asymptomatic animals. A few illnesses have been given individual names. They include equine protozoal myeloencephalitis in horses, which is usually caused by *Sarcocystis neurona* (and less often by *Neospora hughesi*, a parasite not discussed here), and pigeon protozoal encephalitis, which is caused by *S. calchasi*.

While there are currently more than 150-200 named species of *Sarcocystis*, most of them were described on the basis of parasite morphology in a particular host, and the exact number of valid species is uncertain. For example, some new research suggests that *S. fayeri* and *S. bertrami*, which infect horses, are the same organism.

Species Affected

Sarcocystis spp. seem to be ubiquitous in mammals, marsupials, birds and reptiles. Whether these parasites can infect amphibians and fish is unclear. As of the late 1970s, amphibians and fish were not thought to be susceptible, and a few limited surveys seem to support this view. However, a paper from the 1940s described *Sarcocystis* in the muscles of Canadian speckled trout (*Salvelinus fontinalis*) and eel pout (*Zoarces angularis*).

Each species of *Sarcocystis* cycles between one or more definitive hosts and intermediate hosts, typically in a predator/ prey or scavenger/prey cycle. Animals are considered to be aberrant intermediate hosts when only immature parasites have been found in their tissues, making them incapable of infecting the definitive host. An animal can be a definitive host for one species of *Sarcocystis* and an intermediate host for another.

Definitive hosts

Carnivores and omnivores are the usual definitive hosts for *Sarcocystis*. In most cases, each parasite is thought to use one or a group of closely related hosts; however, a few organisms can develop in more distantly related species (e.g., cats and dogs). Some animals have been shown to be definitive hosts only by experimental inoculation, and may or may not be important in natural cycles.

Dogs can be definitive hosts for several species with livestock intermediate hosts, including *S. cruzi*. *S. miescheriana*, *S. tenella*, *S. arieticanis*, *S. capracanis*, *S. hircicanis*, *S. levinei* and *S. cameli*. Some of these organisms are also known to use other carnivores or omnivores, such as wolves, coyotes (*Canis latrans*), raccoon dogs (*Nyctereutes procyonoides*) foxes, hyenas, jackals or raccoons (*Procyon lotor*). Cats and other felids also serve as definitive hosts for species found in livestock, including *S. hirsuta*, *S. porcifelis* *S. moulei*, *S gigantea*, *S. medusiformis*, *S. fusiformis*,

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S. buffalonis and *S. sinensis;* as well as *S. muris, S. cymruensis* and some other species that infect rodents; and *S. leporum* and *S. cuniculi*, which occur in rabbits. Both cats and dogs can be definitive hosts for *S. wenzeli*, which infects poultry. The Virginia opossum (*Didelphis virginiana*) in North America and the white-eared opossum (*D. albiventris*) in South America are definitive hosts for S. *falcatula*, which causes disease in some birds, and *S. neurona*, which affects a variety of mammals. Nonhuman primates probably act as definitive hosts for some of the same parasites as humans (e.g., *S. suihominis, S. hominis*).

Raptors and some other birds, such as corvids, are the definitive hosts for some organisms. In Europe, the Northern goshawk (*Accipiter gentilis*) and possibly the European sparrowhawk (*Accipiter nisus*) are definitive hosts for *S. calchasi*, which causes pigeon protozoal encephalitis. Other species of *Accipiter* might play this role in North America. Snakes are definitive hosts for some organisms such as *S. singaporensis*, which has rodent intermediate hosts, and *S. zamani*. However, their main clinical significance is as the probable hosts of *S. nesbitti*, which has caused several outbreaks in people.

Intermediate hosts

Intermediate hosts can be mammals, marsupials, birds, reptiles and possibly fish. They are often herbivores or omnivores, probably because they are common prey, but some organisms infect carnivores. Some species of *Sarcocystis* seem to be more host-specific than others.

Many mammals are known to be intermediate hosts or aberrant intermediate hosts for at least one species of *Sarcocystis*. Some domestic animal hosts and the organisms that can infect them include cattle (*S. hominis*, *S. cruzi*, *S. hirsuta*, *S. heydorni*), water buffalo (*S. fusiformis, S. buffalonis, S. levinei*, *S. sinensis*), sheep (*S. tenella*, *S. arieticanis*, *S. gigantea*, *S. medusiformis, S. microps*, *S. mihoensis*), goats (*S. capracanis*, *S. hircicanis*, *S. moulei*), pigs (*S. suihominis*, *S. miescheriana*, possibly *S. porcifelis*), horses and other equids (*S. neurona*. *S. fayeri/ S. bertrami)*, South American camelids (*S. aucheniae*, *S. masoni*), dromedary camels (*S. cameli*, *S. ippeni*), dogs (*S. canis, S. neurona)*, cats (*S. felis*, *S. neurona*) and rabbits (*S. leporum*. *S. cuniculi)*. There are also other *Sarcocystis* species or proposed species in some of these hosts.

Wild mammals can be infected with distinct species of *Sarcocystis*, but they also share some parasites with domestic animals (or humans). *S. miescheriana* circulates in wild boar in Europe, as well as in domestic and feral pigs; *S. tenella* has been found in chamois (*Rupicapra rupicapra*) as well as sheep; the cattle parasite *S. cruzi* can replicate in American bison (*Bison bison*) after experimental inoculation; white-tailed deer (*Odocoileus virginianus*), sheep and cattle can all be intermediate hosts for *S. odocoileocanis*; and *S. canis* or a closely-related species affected a bottlenose dolphin (*Tursiops aduncus*). Nonhuman primates have been proposed as intermediate hosts for *S. nesbitti*, which has caused outbreaks in humans and appears to use snakes as a definitive host. However, some sources suggest that this organism might normally circulate in an animal frequently preyed on by snakes, such as a rodent or small mammal.

S. neurona seems to infect a particularly diverse set of hosts. Most clinical cases occur in horses, but several outbreaks have been reported in sea otters (*Enhydra lutris*). Clinical cases thought or proven to be caused by this organism have also been seen in cats, dogs, a Canada lynx (*Felis canadensis*), mink (*Mustela vison*), a ferret (*Mustela putorius furo*), a fisher (*Martes pennanti*), raccoons, a striped skunk (*Mephitis mephitis*), red pandas (*Ailurus fulgens*), a white-nosed coati (*Nasua narica molaris*) a captive zebra (*Equus burchelli*), an immunosuppressed rhesus macaque (*Macaca mulatta*), Pacific harbor seals (*Phoca vitulina richards*i), harbor porpoises (*Phocoena phocoena*), a California sea lion (*Zalophus californianus*), a Pacific walrus (*Odobendus rosmarus divergens*) and other terrestrial or marine mammals. Some of these species are known to be intermediate hosts, but horses seem to be aberrant intermediate hosts in most cases. *S. neurona* has been reported in a few birds, though it might have been misidentified.

S. falcatula and *S. calchasi* use birds as intermediate hosts. *S. falcatula* seems to have a wide host range, including various birds in the orders Psittaciformes, Passeriformes, Columbiformes and Strigiformes. Grackles and cowbirds (*Molothrus ater*) are thought to be its usual intermediate hosts. Clinical cases have been reported in captive psittacines and a few wild raptors, such as bald eagles (*Haliaeetus leucocephalus*), a golden eagle (*Aquila chrysaetos*) and a great horned owl (*Bubo virginianus*). It can also cause disease in experimentally infected pigeons (*Columba livia*). *S. calchasi* has caused illnesses in various columbiform birds and psittacines. A limited study found no evidence that *S. calchasi* causes encephalitis in mammals. *S. horvathi* and *S. wenzeli* infect chickens, and *S. rileyi* infects ducks, but none of these organisms are thought to cause significant illnesses in poultry.

There is relatively little research on reptiles, but snakes are known to be infected with at least a few species (e.g., *S. pythonis*, *S. atrac*). *Sarcocystis* was also found in a gecko.

Zoonotic potential

Humans are definitive hosts for at least three organisms: *S. suihominis*, which uses members of the pig family as intermediate hosts, and *S. hominis* and *S. heydorn*i, which infect cattle. Other parasites, particularly those in nonhuman primates, might also infect people in some regions. Sporocysts of *S. cruzi*, which is thought to mature in the intestines of dogs, were found in the feces of one immunodeficient patient with diarrhea.

Humans can act as intermediate hosts or aberrant intermediate hosts for some *Sarcocystis* species, but many organisms found in human tissues have not been identified to the species level. *S. nesbitti*, which probably uses snakes as its definitive host, has caused a number of clinical cases in Malaysia.

A toxin associated with *S. fayeri* was suggested to be responsible for food poisoning associated with eating raw horsemeat in some countries.

Geographic Distribution

Sarcocystis spp. occur worldwide, but individual organisms can be limited by the range of their definitive or intermediate hosts. *S. neurona* and *S. falcatula* are not known to circulate outside the Americas, where opossums, their definitive hosts, reside. However, some evidence suggests that an organism related to *S. neurona* might be present in Europe. *S. calchasi* has been reported in Europe, North America and Japan, and is probably widespread.

Most cases of myositis in people have been acquired in Southeast Asia, especially Malaysia, and were caused by *S. nesbitti*. This or a related organism was recently identified in snakes in Australia, although no clinical cases have been described there. *S. hominis* and *S. suihominis*, which use humans as definitive hosts, are cosmopolitan.

Transmission and Life Cycle

Sarcocystis spp. have an indirect life cycle, which can only be completed with both an intermediate and a definitive host, typically a predator or scavenger and its prey. The parasites replicate asexually in the intermediate host, and form the sexual stage in the definitive host.

The definitive host becomes infected when it eats encysted parasites, which are called sarcocysts, in animal tissues. Each sarcocyst contains hundreds to thousands of bradyzoites, the infective form, which are released in the intestines. The parasite is not amplified in this host: after entering the intestinal wall, each bradyzoite develops directly into a microgamete (male) or macrogamete (female), which fuse to form oocysts. Mature oocysts, which contain two sporocysts, are shed in the feces; however the fragile oocyst wall often disintegrates during passage, releasing free sporocysts. The prepatent period seems to be around a week or two, and the definitive host may continue to excrete parasites for several months or more, with a few reports of oocyst shedding for as long as 1-2 years. Sporocysts are thought to remain viable for months in the environment under some conditions, including cool temperatures and freezing, but they can be killed by desiccation. One study found that *S. neurona* sporocysts survived for at least 34 months but less than 44 months at 4ºC (39ºF).

Intermediate hosts become infected when they ingest oocysts or sporocysts, often in food or water contaminated with the feces of the definitive host. Insects such as flies and cockroaches can be mechanical vectors. The ingested organisms cross the intestinal wall into the bloodstream. In many cases, they multiply in the walls of various small blood vessels before invading the muscles or neural tissues, where they multiply as merozoites or schizonts for several generations. These immature stages do not seem to be infectious. Merozoites eventually develop into bradyzoites, which are contained within the mature sarcocyst and usually take 2 months or more to form. Animals are considered to be aberrant intermediate hosts when the parasites do not develop to this stage. Most sarcocysts occur in skeletal or cardiac muscles, but they may also be found in smooth muscles, and occasionally in the central nervous system (CNS). They range in size from microscopic to visible, the latter about the shape and size of a grain of rice. Sarcocysts can persist for months to years, though many start to disintegrate after a few months. Parasites do not seem to pass from one intermediate host to another, even if it is eaten. Congenital infections can occur in the offspring of some hosts, including ruminants, horses and dogs, but this seems to be uncommon.

Disinfection

Sporocysts/ oocysts (the forms shed in feces) can be destroyed by heating to more than 60ºC (140ºF) for 1 minute, 55°C(131ºF) for 15 minutes, or 50°C (122ºF) for one hour, but can survive freezing. *S. miescheriana* sarcocysts in pork can be destroyed by holding the meat at 60°C for 20 minutes, 70ºC (158ºF) for 15 minutes or 100°C (212ºF) for 5 minutes, or by freezing it at -4ºC (25ºF) for 2 days or -20ºC (-4ºF) for 24 hours.

A number of commonly used disinfectants (e.g., 1% iodine, 10% formalin, 12% phenol, 2% chlorhexidine) did not kill *S. neurona* sporocysts, but 5.25% sodium hydroxide (bleach) for one hour was effective.

Infections in Animals

Incubation Period

Little is known about the incubation period in most intermediate hosts. Some clinical signs can appear within the first few weeks, while others (e.g., encephalitis in birds infected with *S. calchasi*) have an incubation period of 2 months or more in the same animal. Reported incubation periods for cases of equine protozoal myeloencephalitis range from 10-12 days to several years.

Clinical Signs

Any clinical signs in the definitive host seem to be mild and transient. Self-limited diarrhea or other enteric signs have been seen in some mammals or birds given high parasite doses in the laboratory, but clinical signs are rarely documented in naturally infected animals.

Most intermediate hosts also seem to be asymptomatic, even when parasites are found in tissues at necropsy. Clinical cases are usually the result of damage to the CNS, muscles and/or heart, and sometimes the liver or lungs, though other organs and tissues can be affected.

Equine protozoal myeloencephalitis and other diseases caused by S. neurona

Equine protozoal myeloencephalitis is a sporadic illness seen in some horses infected with *S. neurona*. Often, only a single animal becomes ill. Most cases begin insidiously, though an acute onset is also possible. Affected animals

develop various focal or multifocal CNS signs, such as head tilt, facial paralysis, visual defects, dysphagia, signs of upper respiratory dysfunction, behavioral abnormalities, asymmetrical or symmetrical weakness, ataxia of one or more limbs, and occasionally seizures. Stumbling and frequent interference between limbs can be an early sign. There may also be discrete areas of spontaneous sweating or loss of reflexes and skin sensation. Most horses initially have normal mentation. CNS lesions can lead to muscle atrophy, and asymmetrical gait deficits with focal muscle atrophy are said to be particularly suggestive of this disease. In many cases, the clinical signs in untreated animals gradually become more severe; however, cases can also progress quickly to recumbency and death. Some animals stabilize for a time before relapsing days to weeks later.

S. neurona has been found or implicated in cases of meningoencephalitis in other species of mammals. There are also occasional reports of myositis, myocarditis, disseminated infections or liver disease in these hosts, either with or without CNS signs. A few animals had concurrent respiratory signs or lesions (e.g., dyspnea, interstitial pneumonia, lung mass), and retinochoroiditis accompanied neurological signs in at least one sea otter. A few unusual syndromes have also been reported. One dog had pustular dermatitis associated with *S. neurona* in the skin, and a ferret developed rhinitis with large numbers of organisms in the nasal turbinates, shortly after it was given a live canine distemper vaccine. A captive coati had only nonspecific signs (anorexia, lethargy, progressive weakness), which were associated with disseminated sarcocystosis affecting the gastrointestinal tract, urinary bladder and other sites, but no myositis, myocarditis or encephalitis. Most clinical cases affected a single animal, but several epidemics were reported in sea otters, and one outbreak occurred in mink.

Syndromes caused by other species of Sarcocystis in mammals

Experimentally infected animals that are given large doses of various *Sarcocystis* species can become ill, and sometimes die. Fever and nonspecific signs of illness were the major signs in some animals, but myositis, abortions, pneumonia/dyspnea and anemia have also been seen. In addition, some cattle had diarrhea, hemorrhages, icterus or neurological signs (hyperexcitability). Clinical cases in naturally infected animals, which are probably exposed to lower doses of the parasite, seem to be infrequent or rare. The syndromes reported in various domestic animals and captive or free-living wildlife are similar to those caused by *S. neurona*, and have included encephalomyelitis, myositis, liver disease (elevations in liver enzymes, with or without overt signs of hepatitis), respiratory disease (pneumonia, dyspnea), cardiomyopathy and acute nonspecific illnesses or sudden death caused by disseminated sarcocystosis. However, abortions and/or stillbirths have also been associated with sarcocystosis in some species, particularly domestic ruminants. One group of feedlot cattle failed to thrive after recovery from acute sarcocystosis and eventually died of cachexia.

Common signs in horses with myositis caused by *S. fayeri/ S. bertrami* include stiffness, muscle pain and/or gait abnormalities, which may be accompanied by muscle atrophy, weight loss and a decreased appetite. Dysphagia may be seen occasionally (i.e., when the muscles of the esophagus are also affected). Myositis caused by other organisms in dogs, cats and South American camelids appears similar. Other signs of organ dysfunction, including dyspnea or signs of liver injury, occurred concurrently in some animals, and dogs sometimes had nonspecific clinical signs (e.g., lethargy, anorexia) before the onset of myositis. Myositis can have a prolonged course, and some cases have been fatal. In ruminants, eosinophilic myositis is usually an incidental finding detected during meat inspection in asymptomatic animals.

Sarcocystosis in birds

S. falcatula usually causes acute respiratory disease, encephalitis and/or myositis in psittacine birds. Most birds with respiratory signs or encephalitis are severely ill, and these syndromes are often fatal. Sudden death or a rapidly progressive illness is common in respiratory cases. Myositis is characterized by profound muscle weakness, and tends to have a more prolonged or chronic course. Some birds with myositis have recovered, though it may take weeks to months of treatment and supportive care. *S. falcatula* has also caused encephalitis and/or rapidly progressive respiratory signs, which were usually fatal, in wild raptors.

S. calchasi usually causes neurological signs (e.g., torticollis, opisthotonos, muscle tremors, ataxia, paralysis) in pigeons. Most cases are fatal, and survivors may have residual neurological deficits. There are also reports of hepatitis and sudden death, with no evidence for myositis or encephalitis, in naturally infected pigeons. Experimentally infected pigeons had a biphasic disease, with mild diarrhea and nonspecific signs of illness a few weeks after inoculation, and encephalitis and myositis in some birds at approximately 2 months. Similar early and late signs occurred in experimentally infected cockatiels (*Nymphicus hollandicus*). Neurological signs were reported in a group of naturally-infected captive psittacines. The signs in this outbreak were severe, and appeared to progress more quickly than in some pigeons.

Post Mortem Lesions

Mammalian intermediate hosts

Sarcocysts can be found in skeletal and cardiac muscles either as an incidental finding or in clinical cases. In cases of myositis, the parasites are usually accompanied by granulomatous inflammation. Sarcocysts are cylindrical and whitish, range in size from a few micrometers to a few centimeters, are oriented along the length of the muscle fiber, and may or may not be visible to the naked eye. Visible sarcocysts often resemble a grain of rice. Gray to greenish, well demarcated muscle discoloration is a common finding in asymptomatic ruminants with microscopic sarcocysts. In pigs and cattle, sarcocysts are found most often in the myocardium, diaphragm and esophagus. The tongue is also a common site in sheep.

Other tissues and organs, particularly the CNS and liver, may also be damaged by the parasites. The lesions vary with the organs affected, and may include organ enlargement, inflammation, and signs such as icterus or hemorrhages. Hemorrhages tend to occur mostly in the serosa of visceral organs, myocardium and/or skeletal muscles. Gross CNS lesions, if any, are usually limited to focal discoloration, hemorrhages or malacia. Some animals have microscopic evidence of encephalitis with no apparent gross lesions.

Avian intermediate hosts

S. calchasi and *S. falcatula* can also cause meningoencephalitis with few or no gross lesions. Thickened and opaque meninges may be seen in some birds. Lesions might be more likely in *S. calchasi*-infected pigeons during the early stages of the illness. Moderate to severe hepatomegaly is common in experimentally infected pigeons at this time. Some birds also had necrotic foci in the liver or diffuse pale hepatocellular necrosis, an enlarged spleen and clear gelatinous fluid in the coelomic cavity. Birds with respiratory disease caused by *S. falcatula* can have pulmonary edema and/or congestion, hemorrhages and fibrin deposits in the lungs, and increased opacity of the air sacs. The liver and spleen may be enlarged.

Diagnostic Tests

Definitive hosts can be identified by detecting sporocysts (approx. 10x15 µm) and/ or oocysts with fecal flotation techniques, including flotation/ centrifugation. Sucrose or salt solutions with specific gravity ≥ 1.15, such as saturated sodium chloride, Sheather's, sodium nitrate, magnesium sulfate or zinc sulfate, can be used. In a wet mount from a float, the organisms can be found just beneath the coverslip. The oocyst wall can be difficult to see unless the preparation is stained. Mucosal scrapings, taken at necropsy, are more likely to reveal *Sarcocystis* than fecal samples if the infection is light. Sporocysts and oocysts from different species of *Sarcocystis* are morphologically indistinguishable.

Clinical cases in intermediate hosts may occasionally be diagnosed with a biopsy, but this is uncommonly done and the parasites can be missed even at sites of muscle swelling. More often, they are found in the tissues at necropsy. Two specialized methods sometimes used to visualize *Sarcocystis* spp. are muscle squash preparations and a tissue digestion technique (e.g., with pepsin or trypsin), which releases free bradyzoites. Tissue digestion is a particularly sensitive method if sarcocysts are present; however, the immature stages of *Sarcocystis* are destroyed by the enzymes. *Sarcocystis* can be stained with hematoxylin and eosin, periodic acid-Schiff, Giemsa and other stains. Organisms cannot always be observed in the CNS of mammals or birds with encephalitis, but their nucleic acids can usually be detected by PCR. In birds, *S. calchasi* and *S. falcatula* may also be found in the muscles at this time. *S. falcatula* sarcocysts are reported to persist longer in leg (quadriceps) muscles than pectoral muscles.

Body fluids are not examined routinely for *Sarcocystis*; however, merozoites were unexpectedly found in the cerebrospinal fluid (CSF) of a cat with encephalitis, and in the blood of another cat with disseminated disease and pneumonia. A PCR test was developed for the antemortem diagnosis of horses with equine protozoal myeloencephalitis, using CSF samples, but its sensitivity is poor. The different species of *Sarcocystis* have traditionally been distinguished by sarcocyst morphology, but genetic techniques are increasingly used in research. Antibodies to *Sarcocystis* spp. can distinguish this parasite from other protozoa such as *Toxoplasma gondii* or *Neospora* spp.

Serology, using CSF samples, is useful for diagnosing equine protozoal myeloencephalitis in live horses. This should be evaluated as the ratio of CSF/serum antibodies, to avoid misdiagnosis caused by the passive transfer of antibodies across the blood-brain barrier or minor contamination of CSF samples with blood. Quantitative tests include immunofluorescent antibody (IFA) assays and ELISAs. A semiquantitative immunoblot may also be available in some laboratories. A direct agglutination test (SAT) can detect antibodies to *S. neurona* but is not quantitative. There are also reports of using an IFA test to diagnose myositis or encephalitis caused by *S. falcatula* in birds. (Respiratory disease is usually fatal before the antibodies develop.) Serological tests often cross-react with other species of *Sarcocystis*, but there is little or no cross-reactivity with other pathogenic protozoa, such as *T. gondii*.

Treatment

Equine protozoal myeloencephalitis can be treated with toltrazuril, ponazuril or diclazuril, or a sulfonamide combined with either pyrimethamine or trimethoprim. Pyrimethamine is more effective against protozoa than trimethoprim. Supportive care, including anti-inflammatory drugs, may also be necessary.

Treatments in other mammals and birds have not been standardized, but similar antiprotozoal agents including ponazuril, decoquinate, pyrimethamine/ sulfonamide or trimethoprim/ sulfonamide have been tried, with some apparent success, in dogs, cats, marine mammals, birds and other species. Some animals with myositis have recovered after supportive treatment, antibiotics (e.g., clindamycin) and steroids. Whether the various antiprotozoal drugs affected the outcome of the disease was not always clear, particularly in cases of myositis. Treatment often fails in respiratory disease or encephalitis caused by *S. falcatula* due to their severity and rapid course.

Control

Disease reporting

Veterinarians who suspect a clinical case is caused by *Sarcocystis* should follow their national and/or local guidelines for disease reporting. Some states in the U.S. (e.g., Alaska) require that that equine protozoal myeloencephalitis be reported to state authorities within a specified period.

Prevention

Prevention generally depends on reducing exposure to oocysts and sporocysts in the environment. Horses should be provided with water sources unlikely to be contaminated by opossum feces. It may also be helpful to feed them off the ground. In cases where contamination is suspected, steam cleaning might be used to decontaminate the horse's environment, and feed could be heat treated. Food sources that may attract opossums (e.g., grains, bags of birdseed, fallen fruit) should be sealed in metal containers or removed. A partially buried fence and electric fences may discourage wildlife from entering paddocks. Opossums might occasionally be trapped and relocated, though this is likely to be impractical or ineffective where these animals are common. A vaccine was marketed for a short time in the U.S. but it did not seem to be effective and was withdrawn. Similar measures, based on reducing exposure to feces from a parasite's definitive hosts, may be used to protect other intermediate hosts.

Prevention of infections in the definitive host (e.g., dogs and cats) is based on avoiding diets that contain raw or undercooked animal tissues. Freezing raw meat (see Prevention - Humans) might also be effective.

Morbidity and Mortality

Exposure to *Sarcocystis* spp. is common in asymptomatic livestock (e.g., ruminants, horses, South American camelids, camels), dogs, cats, and some wildlife, with antibodies often reported in 10-50%, and occasionally up to 100%, of a study population. Management factors such as sanitation and contact with the definitive host (including livestock exposure to herding dogs) can influence exposure. Pigs reared in confinement are less likely to have antibodies to *Sarcocystis*; however, some authors have speculated that a lack of immunity might also increase the risk of disease if these pigs are later exposed to a source of organisms.

Clinical cases are thought to be related to the dose of parasites, and seem to be uncommon in most species. While sarcocystosis often affects only a single animal, there can be outbreaks after common source exposure. Many of these incidents are fairly small (e.g., 10% of a sheep flock, or 0.3% of the mink on a farm), but there have been serious and extensive outbreaks of *S. neurona* encephalitis in sea otters off the West Coast of the U.S., possibly when large numbers of oocysts were washed into the ocean. High mortality rates (e.g., 92% in alpacas, up to 50% in young pigs) are sometimes reported in experiments where animals are given large doses of oocysts. Severe or fatal illnesses have also been reported in some naturally infected animals.

Equine protozoal myeloencephalitis is one of the most commonly reported forms of sarcocystosis. It is a sporadic illness that affects only a small percentage of the horses infected with S. neurona. The average incidence in the U.S. is 14 cases per 10,000 horses per year, while approximately 30-60% of the horses in the U.S. are seropositive. This disease usually affects a single animal, but it can appear in clusters. Morbidity may occasionally exceed 25%. Clinical cases are most common in horses during the first few years of life. One study also found an increased incidence in horses > 13 years of age. Stressors (e.g., heavy exercise, transport, parturition) and immunosuppression are thought to increase the risk of illness. Approximately 60-75% of sick horses improve with treatment, but fewer recover fully. Up to 30% return to their original level of performance. Relapses are possible. Some other species also have significant exposure to S. neurona though clinical cases are rarely reported. One study found antibodies to this organism in 5-10% of farm cats, and up to 40% of the cats on some farms.

S. falcatula can be a significant cause of mortality, especially in outdoor-housed birds. Large numbers of birds have been affected in some outbreaks. Most clinical cases occur in Old World psittacine birds, which did not evolve around the definitive hosts, rather than New World psittacines. In New World psittacines, clinical cases are more likely to occur in nestlings. Cases of respiratory disease, which usually progresses rapidly, and encephalitis are often fatal, and attempts to treat these birds have generally been unsuccessful. Birds with myositis have sometimes recovered with supportive care.

Intestinal sarcocystosis is thought to be asymptomatic in most cases, and transient and self-limited if clinical signs occur.

Infections in Humans

Incubation Period

In some human volunteers, intestinal signs occurred as soon as 3-6 hours after eating sarcocysts, then recurred 14-18 days later. The incubation period for myositis caused by *S. nesbitti* can be as long as 1-2 months, but an estimate from one outbreak suggests it is probably around 9-13 days in many cases.

Clinical Signs

To date, the two major syndromes reported in humans are myositis, where people act as intermediate hosts, and enteritis when they are the definitive host.

Myositis

Most clinical descriptions of myositis are based on outbreaks caused by *S. nesbitti* in Southeast Asia. Some people infected with this organism had nonspecific early signs, such as fever, malaise, headache, myalgia, arthralgia, coughing, and episodes of weakness or fatigue. The onset of myositis is characterized by painful or tender muscle swelling (which may include subcutaneous nodules in some patients), generalized muscle weakness and/or fatigue, and fever. The muscle groups affected vary, and included the muscles of the face and jaw in some outbreaks. Muscle atrophy is possible in the later stages. Many patients also have eosinophilia. Cutaneous signs such as transient pruritic rashes, pruritus without a rash, and urticaria have been seen occasionally. Lymphadenopathy seemed to be uncommon in some outbreaks. Most cases of myositis seem to end within

a few weeks to a few months, but some patients have had persistent or recurrent symptoms for a year or more. In a few cases, the signs lasted for several years.

Other syndromes, including self-limited cardiac abnormalities or glomerulonephritis, have been seen in a few people with *S. nesbitti* myositis, but were not definitively linked to the parasitic infection. One source mentions a death caused by myocarditis, in an isolated case of sarcocystosis in a healthy woman.

Intestinal sarcocystosis

Many or most intestinal infections, which are usually caused by *S. suihominis* or *S. hominis*, are thought to be asymptomatic. However, some people may have enteric signs such as diffuse abdominal tenderness, diarrhea, nausea and/ or vomiting, and a low-grade fever, chills or sweats in some cases. As with other intestinal illnesses, dehydration is possible. Intestinal sarcocystosis is thought to be transient and self-limited in most cases.

Some reports suggest that *S. fayeri* or another organism may produce a toxin that can cause a form of food poisoning (nausea, vomiting, diarrhea) associated with eating raw horsemeat.

Diagnostic Tests

As in animals, intestinal infections can be diagnosed by detecting oocysts or sporocysts in the feces with flotation techniques. Direct smears are also used in people, and include the Kato thick smear, which examines a larger fecal sample and is more sensitive than other methods. The prepatent period for *S. hominis* and *S. suihominis* is thought to be roughly 2 weeks, and parasites might not be found in the early stages.

In cases of myositis, sarcocysts may be detected in the muscles with a muscle biopsy. However, the parasites are unevenly distributed, and may be absent or few in number. A complete blood count, to reveal eosinophilia, may be helpful. PCR tests have been used in some outbreaks, but their sensitivity may be low: parasites were found by visual examination in some PCR-negative samples.

Serological tests are not widely available in human diagnostic laboratories. One study found that paired titers were not useful in the diagnosis of myositis in travelers or residents during an outbreak in Malaysia.

Treatment

Intestinal sarcocystosis may not be treated, as infections are usually self-limiting. Some patients with symptomatic myositis have been given antiparasitic drugs such as metronidazole, trimethoprim/ sulfonamide, pyrimethamine/ sulfonamide or albendazole. The value of these agents is still unclear; in some cases, it was uncertain whether the drug was effective or the disease improved on its own. Supportive care may include anti-inflammatory agents.

Prevention

Intestinal infections, can be preventing by avoiding raw or undercooked meat in the diet. Freezing the meat to -20°C for 48 hours or -4°C for 48 hours appears to destroy the parasites.

Myositis can result from fecal contamination of water, fruits, vegetables, and other sources of sporocysts in the environment. Water that might be contaminated should be boiled or filtered; Sarcocystis can persist in water treated with chemical decontaminants, including chlorine. Food that may have been contaminated should be washed thoroughly, and cooked if removal of the organisms is uncertain.

Morbidity and Mortality

The incidence of sarcocystosis can range from < 1% in some locations to ≥ 20% in others. Intestinal infections are more prevalent in cultures where raw meat is commonly eaten. Both the dose of parasites and host factors seem to influence the severity of the signs, even in healthy people. Many or most intestinal infections are probably asymptomatic. *Sarcocystis* spp. do not multiply in the definitive host, and any clinical signs are thought to be self-limited and transient in most cases. People can probably be reinfected with the same organism when acting as the definitive host.

A few hundred clinical cases of myositis have been reported in humans worldwide, as of 2019, with the vast majority seen during outbreaks in Malaysia. Approximately 20% of the people in this region are seropositive, and most infections might be subclinical or mild. Clinical cases usually occurred in travelers, or in one case, in both travelers and residents at a conference. Many patients were young and otherwise healthy. Myositis often seems to be self-limiting. The symptoms were generally milder and ended sooner in Malaysian residents, suggesting that immunity from previous exposures might help limit parasite replication. Myositis has rarely been described in humans outside Malaysia, although some cases might be attributed to other causes. Sarcocysts are occasionally found in the muscles of asymptomatic individuals as an incidental finding.

Internet Resources

Centers for Disease Control and Prevention, U.S. (CDC). Sarcocystosis

Food and Agriculture Organization of the United Nations [FAO] - Manual on Meat Inspection for Developing Countries

The Merck Manual

The Merck Veterinary Manual

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*Link defunct

