Zoonoses Which Occur With Greater Incidence in Children

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The veterinarian frequently serves as a resource about diseases which affect both man and animal. It is his responsibility to be aware of their epidemiology and how their transmission to man can be avoided.

This paper will discuss those zoonoses that occur with a higher incidence in children. The following diseases will be briefly reviewed in terms of definition, incidence and etiology, epidemiology, the disease in animals and man, and prevention and control: California encephalitis, *Campylobacter* enteritis, Arizona disease, infant botulism, Dipylidiasis, Ancylostomiasis, Capillariasis, Cutaneous larva migrans, and Visceral larva migrans.

California encephalitis

California encephalitis (CE) is seen primarily in children less than 15 years of age.[1] The etiologic agent is the La Crosse virus which belongs to the California group of arboviruses. It is the only virus of this group known to cause disease in man.[2] The virus was isolated in California in 1945 from three children with encephalitis.[2] Since that time, no other isolations of this virus have occurred in California. In 1960, the La Crosse virus was isolated from a fatal case of encephalitis in Wisconsin.[2] Since then, cases have occurred regularly in the midwestern and eastern United States. In Indiana, it is frequently referred to as rural encephalitis and in rural areas it may be referred to as farmer's encephalitis.

CE is seen primarily in Ohio, Wisconsin, and Minnesota.[1] From 1960 to 1970, 509 cases were reported.[1] From 6 to 60 percent of rural workers in various parts of this country have antibodies to viruses to the California group. This disease is seen most frequently in the summer and early fall in forested, rural, or suburban areas.

The California group of viruses circulate in nature among rodents, lagomorphs, and mosquitoes (primarily *Aedes triseriatus*). The primary hosts are chipmunks (*Tamias striatus*) and squirrels *(Scirus).* Other vertebrate hosts generally do not develop a sufficient viremia to serve as reservoirs.[2] Transovarial transmission allows the virus to overwinter in eggs and larvae of *Aedes.* Horizontal transmission occurs via blood meals and possible venereal transmission between mosquitoes.[2] The disease in animals is most likely asymptomatic.[1] Man is purely an accidental host, being exposed when bitten by an infected mosquito. Areas of high risk for exposure and oviposition are sites which include any water-holding containers such as basal tree holes.

The disease in man ranges from a benign aseptic meningitis to a serious encephalitis. It is possible that many cases pass as a mild fever. Frequency of symptoms include: fever (95 percent), headache (86 percent), nuchal rigidity (55 percent), seizures (37 percent), and lethargy (28 percent).[2] In 35 children studied, eight had residual effects such as paresis or tremors.[2] Mortality has been estimated at not less than 5 percent.[3] It is clearly evident from these facts that California encephalitis should be regarded as an important zoonosis.

Control measures are aimed at avoiding mosquito bites, i.e. protective clothing and use of mosquito repellant. Mosquito-ridden areas, primarily oviposition sites should be avoided especially during times of peak mosquito activity; morning and late afternoon.

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Mosquito population in high risk locations, such as parks and suburban areas, may be decreased by filling or removing oviposition sites since *Aedes* has a limited flight range.[2]

Campylobacter enteritis

When many professionals think of a zoonotic food-borne illness, *Salmonella* often comes to mind. But another bacteria has received an increasing amount of attention in recent years, *Campylobacter jejuni*. According to some authors, it may soon surpass *Salmonella* in its zoonotic potential[4] as more and more animals are found to carry *C. jejuni* in their intestines. The incidence of campylobacteriosis in man has only recently been recognized. In 1977, a method was developed for culturing human feces for this bacteria which has made a definitive diagnosis possible.[5] One British study indicated a 7 percent isolation rate from people with diarrhea,[5] and another study demonstrated a 5.1 percent isolation rate in diarrheic children.[6] Clinically normal people generally do not carry the organism in their intestines if they live in areas with good hygiene.[4] In countries where poor hygienic measures exist, human carriers do occur.

The source of infection to man usually is not found but reservoirs have been identified. A small percentage, approximately 5 percent, of human campylobacteriosis is acquired from dogs.[4] The usual history is a recent acquisition of a puppy with diarrhea and subsequent diarrhea in the owner. The true pathogenicity of *C. jejuni* of dogs remains controversial, as many surveys show no significant difference in the number of isolates between normal and diarrheic animals.[6] The duration of *Campylobacter* excretion in dogs and cats is unknown. Only four cases of campylobacteriosis have been associated with cats.[6] The rate of infection in non-diarrheic cats is low and they are most likely to be infected when associated with an unhygienic environment. Outbreaks of the disease have been associated with the consumption of unpasteurized or improperly pasteurized milk. Mastitis has been experimentally produced by the inoculation of just 2.6 colony-forming units into the udder of cows.[6] Although *C. jejuni* does not grow in milk, only a small number (i.e. 500 organisms in 180 ml. of milk) were needed to produce clinical illness in human volunteers.[4] The organism is effectively killed by "flash" pasteurization (HIST—72.5°C for 15 seconds), the most common method of pasteurization used. A recent study indicates a strain of *C. jejuni* from humans which is able to survive 60°C for one minute and 50°C for at least four minutes.[7] *C. jejuni* has been suggested as a cause of enteritis in cattle but further studies are needed as normal healthy cattle carry *C. jejuni* in their intestines.

There have been only a few reports of human disease acquired from porcine sources but muscle swabs from 22 of 58 swine carcasses yielded *C. jejuni*.

Ferrets have been implicated as a possible reservoir for *C. jejuni* as one study indicated a 61 percent infection rate in a ferret colony. *C. jejuni* has been isolated from ferrets with proliferative enteritis. Experimentally infected animals have been shown to shed the bacteria for up to 16 weeks and clinical disease has developed under conditions of experimental stress. Traditional treatment with erythromycin has been ineffective in eliminating *C. jejuni* from clinically normal carriers. All of these facts should be kept in mind since the ferret has become a popular pet in recent years.

Healthy birds of all kinds, including poultry, wild birds, migrating waterfowl, etc, have a high carrier rate of *C. jejuni*. Birds, particularly domestic poultry, are being implicated as a major reservoir for *C. jejuni* in developed countries. One source believes that contaminated food products, especially inadequately cooked chicken and, only rarely, contaminated red meat, are probably the most frequent source of *C. jejuni*. It has been found in up to 75 percent of broiler carcasses after slaughter, and it may survive processing and refrigeration for several days and longer if frozen.[5] Cooking with the proper heat readily destroys *C. jejuni* at temperatures of 60°C for 15 minutes.

As stated previously, most cases of campylobacteriosis are of unknown origin but with the development of enrichment media, more sources may be properly identified.

Campylobacter jejuni usually creates a self-limiting illness that lasts approximately one week and is seen more frequently in children.[1] It generally occurs as sporadic outbreaks. The average incubation period is two to five days. Symptoms include fever, malaise, and diarrhea accompanied by abdominal pain, nausea, and vomiting. The severity of symptoms is variable and on occasion exploratory abdominal surgery has been performed be-

cause of the severity of abdominal pain.[5] Transmission between humans is unlikely because of *C. jejuni*'s senstivity to oxygen.

Prevention of human infection centers around good hygienic measures. Raw meat, especially poultry, and cooked foods should be kept separated and hands should be washed after handling raw meat. Meat should be thoroughly cooked. Exposure from milk can be prevented by drinking only properly pasteurized milk. Veterinarians should investigate those cases of enteritis which occur simultaneously in owners and their pets. Feces of animals should be cultured. Owners should be referred to their physician and the physician advised of culture results. Affected animals can be treated with erythromycin at 40 mg/kg daily for five days. If vomiting occurs, tylosin can be substituted at 45 mg/kg for five days. Positive dogs should be re-cultured after treatment. Children should be instructed to wash their hands after handling animals and diarrheic animals should be isolated from children.

Arizona disease

Arizona disease is a food-borne illness caused by the *Arizona* group of Enterobacteriaceae. The bacteria involved is *Arizona hinshawii,* a lactose fermenter. The incidence of the disease in man and animals is not known since many laboratories routinely discard lactose fermenting Enterobacters.[1]

In nature, animals are the primary reservoirs of the *Arizona* group of bacteria. *A. hinshawii* has been found in both healthy and diseased animals. Carriers help maintain the organism in turkey flocks where transmission occurs through eggs. The organism has been associated with a septicemic disease in young chickens and turkey poults. The organism is frequently found in the intestines of normal reptiles and they may possibly be the main reservoir.[1] The source of infection to man is usually contaminated foods, primarily eggs, egg products, and improperly cooked turkey meat.

Children are affected more frequently than adults. The usual clinical picture is a gastroenteritis similar to that caused by *Salmonella* spp. Cases of septicemia have been reported.

Control measures are directed toward the proper handling of fowl and fowl products, i.e. thorough cooking, good sanitation, and food hygiene.

Infant botulism

Traditionally botulism has been linked with eating foods contaminated with *Clostridium botulinum* and its powerful neurotoxin. Since the mid-1970s, evidence has been accumulating to indicate that botulism occurs in infants without exposure to foods traditionally linked to the disease, i.e. foods stored under anaerobic conditions where *C. botulinum* multiplies and produces its toxin. The disease in infants has not been associated with the ingestion of pre-formed toxin but with the ingestion of spores.

Infant botulism is seen primarily in 3 to 26-week old infants.[1] From January 1975 to July 1978, 62 cases were reported.[9] Dr. V. R. Dowell of the Center for Disease Control believes that the incidence is even higher than reported cases as infant botulism is not always recognized. He estimates that approximately 250 cases occur yearly.[9]

The source of Clostridial spores for these infants is not clearly understood. *C. botulinum* Types A, D, E, and F are found almost universally. No absolute epidemiologic association has been found between animal and human botulism. *C. botulinum* spores have been isolated from fecal material of animals. The microorganism has been isolated from the intestinal tract and liver of cattle dying from other causes, and the intestines and liver of healthy dogs. In light of these facts, it is possible that animals may be carriers of *C. botulinum* and may serve as agents to spread it.

Cases have occurred in infants fed only breast milk or only formula, but usually the infants had exposure to food other than formulas or breast milk. Of 508 cases investigated by the Center for Disease Control before 1978, only six potential sources have been identified.[9] Vacuum cleaner dust was one source of the spore (Type A), while yard soil yielded the organism (Type B) in another case. In the three other cases, opened jars of honey were contaminated with Type B *Clostridium botulinum.* All three of these infants had been fed honey.

In a California survey, 29.2 percent of hospitalized patients had previously been fed honey.[10] This survey also revealed that the patients had not been significantly more exposed than controls to cereals, fruits, or vegetables either canned or frozen. In food items tested, only honey was postive for *C. botulinum.* It has been estimated that honey accounts for 35

percent of all known hospitalized cases.[10] Evidence suggests that 10 to 15 percent of all honey is contaminated with *C. botulinum* spores.[11] Since only one-third of patients were exposed to honey and because the organism is found so widely in the environment, it is likely that multiple routes of exposure exist, including airborne.[11] In view of these facts, it is likely that exposure to the spores for both adults and infants is not a rare event.

The important question remains as to why infants are susceptible to the ingestion of spores when adults are not. Incidence of the disease peaks when infants are being weaned or shifted from pure formula to infant foods which may alter intestinal flora in some manner allowing for growth of *C. botulinum* and production of its toxin. Distinct differences exist in the intestinal bacterial flora between breast-fed and formula-fed infants which may influence the status of *C. botulinum.* It is known that some species of Enterobacteriaceae and other *Clostridia* spp. can inhibit the growth of *C. botulinum.*[10] Further studies are needed on the flora of breast and formula fed babies to determine its role in the development of infant botulism. As infants age, the intestinal flora changes and this change may play a role in their susceptibility to the spores.

Disease manifestations range from inapparent to a fulminating illness, which may lead to respiratory arrest. The first symptom is usually constipation and it has been speculated as to whether this is a possible cause of proliferation of *C. botulinum* or an effect of the disease. Constipation is followed within hours to weeks by lethargy, weakness, and neurologic symptoms, i.e. hypotonia, hyporeflexia, and multiple cranial nerve dysfunctions. Any afebrile infant with poor suckling or swallowing reflexes, weakness, poor head control, hypotonia, ptosis, mydriasis, ophthalmagia, and normal CSF fluid should be considered a candidate for infant botulism.[12] In the past, it may have been diagnosed as infant polyneuropathy. In half of the reported cases in 1976, respiratory arrest occurred following hospitalization and two infants died.[10] In general, newborns are less severely affected than adults and most respond to supportive treatment.[11] Penicillin has been shown to have little effect on the course of the disease despite evidence that the pathogenesis involves spore germination with elaboration of toxin.[1] Interestingly, *C. botulinum* and its toxin were found in the stool of affected patients but not in the serum.[1]

Infant botulism has been suggested as a possible etiologic agent of SIDS (sudden infant death syndrome).[9] The neurotoxin produced by *Clostridium botulinum* can lead to respiratory arrest which may be the cause of SIDS. SIDS victims have been found to have similar post-mortem lesions as monkeys exposed to the neurotoxin.[9]

Intrathoracic petechiae seen with SIDS have been regarded as a sign of airway obstruction before death which could be due to the neurotoxin's paralysis of cranial nerves supplying laryngeal muscles. SIDS and infant botulism also share a similar age distribution. A study involving 280 infants, who had died suddenly, found that 10 infants had the organism and/or the toxin in their feces.[11] In contrast, of 160 normal infants, only one was positive for *C. botulinum* in the stool.[9]

To avoid exposure to *C. botulinum,* the Center for Disease Control recommends not feeding honey to infants less than one year of age. Raw foods should be washed and peeled before cooking to decrese the number of spores associated with them. Since the spores are relatively heat resistant and can survive boiling for several hours, home cooking and canning of foods may not destroy the spores. Commercially prepared baby foods are prepared in such a manner as to destroy the spores.

Dipylidiasis

Dipylidiasis is a worldwide infection caused by the tapeworm *Dipylidium caninum,* a common intestinal cestode of dogs. The definitive hosts are dogs, cats, wild cats, civets, hyenas, jackals, dingoes, foxes, and man. The dog flea, *Ctenocephalides canis,* and the cat flea, *C. felis,* are the most frequent intermediate hosts. Infrequently, the flea of man, *Pulex irritans,* and the dog louse, *Trichodectes,* can serve as intermediate hosts.

Gravid proglottids are excreted in the feces of the definitive host and eventually break down, releasing eggs, which are ingested by a flea larva. In the flea, the eggs develop into cysticercoid forms which, when ingested by a definitive host, usually the dog, develop into adult tapeworms in the intestinal tract. Man is infected accidentally by ingestion of the infected intermediate host.

In animals, infections are generally asymptomatic. With heavy loads, the passage of seg-

ments may create anal irritation and itching and the owner may see the animal scooting. Heavy infections may also create vague gastrointestinal symptoms.

Man is relatively resistant to infection by this parasite when one considers the frequency with which it occurs in animals.[1] In humans, this tapeworm is usually seen in infants and young children with close exposure to pets. The symptoms and their severity depend on the parasite load and individual susceptibility. Digestive disorders may be present, such as diarrhea, abdominal pain, and increased appetite. Passage of proglottids may be the only symptom and is usually the one most noticed by parents.

Control of infection in both man and animals centers around the elimination of fleas. If infections occur in dogs, they may be treated with praziquantel at a single dose of 5mg/kg.[14] This drug has nearly a 100 percent efficacy against *Dipylidium caninum.*[14] Children should be instructed to wash their hands after contact with family pets to avoid ingestion of infected fleas.

Ancylostomiasis

Ancylostomiasis, an infection with hookworms, is usually caused in man by *Ancylostoma duodenale* and *Necator americanus.* Occasionally, animal hookworms, primarily *Ancylostoma ceylanicum,* may infect humans. *A. ceylanicum* is a parasite of domestic cats, wild cats, and dogs found in tropical areas of the world, such as Asia, Africa, and Brazil.

Adult nematodes live in the small intestine of the host producing eggs which are passed out into the environment with the feces. Under proper environmental conditions (i.e. wet sandy soil, 20–30°C., and lack of direct sunlight) the eggs embryonate to first-stage larvae. Within a week the infective third-stage larvae develop. Humans may become infected by ingestion or through the skin.

The symptoms of hookworm infection in animals are dependent on factors such as age, nutritional condition, and parasite load. Infection is seen more often in young animals. Mild infections may be asymptomatic. Heavier infections produce gastrointestinal symptoms and anemia because of blood loss. Eosinophilia may be present. Death may follow pre-natal infections, where sudden maturation of a large number of parasites may occur.

In man, infection with *A. ceylanicum* is usually asymptomatic because of a light parasite burden. Usually this parasite is found in the intestinal tract in combination with the agents of human hookworm disease.[1] Because of the latter fact, *A. ceylanicum* is not considered a major zoonotic agent.

Capillariasis

Capillaria hepatica is a common parasite of rodents and occasionally other mammals. It is found on all continents primarily among synanthropic and wild rodents. A high rate of infection in rodents has been found in certain areas of the United States, Canada, Korea, and the Phillippines.[15] In some areas, the infection rate in rats may be as high as 50 to 80 percent.[1]

As stated previously, the primary reservoirs of this parasite are rodents. The adult parasite lives in the liver, producing eggs. When the rodents are eaten by carnivores, the eggs are passed out in the feces of the carnivores. The eggs may survive for months in moist soil. In approximately two months under favorable conditions, infective larvae develop which may then be ingested by rodents. The larvae migrate to the liver and mature into adults in one month. Infections in rodents are usually asymptomatic but with very heavy infections symptoms of liver disease may be seen.

Human infection occurs when contaminated soil is ingested. Carnivores serve to spread the parasite. Soil may also become contaminated by death and decomposition of rodents.

The disease in man is rare. Approximately 12 cases have occurred during the past 60 years.[15] Most cases were in younger children in impoverished areas who had a history of eating dirt. It can be a serious disease which, if left untreated, may be fatal. Symptoms of liver involvement are present, i.e. fever, nausea, vomiting, abdominal distension, and peripheral edema. Splenomegaly and pneumonitis may be found. Clinical pathology may reveal eosinophilia and anemia. Death may occur because of secondary bacterial infections. Post mortem lesions consist of granulomas and necrotic areas in the liver. A study done in Czechoslovakia on post mortem liver lesions supports the notion that subclinical cases occur.[1] Liver biopsy is needed to establish a definitive diagnosis as the finding of *Capillaria* eggs in the feces does not indicate

infection but only the ingestion of liver from infected animals.

Prevention of human infection depends on control of rodent population and good hygienic measures.

Cutaneous larva migrans

Cutaneous larva migrans, (CLM, linear dermatitis, creeping eruptions) "is an acute but occasionally chronic clinical syndrome resulting from the invasion and migration of larval parasites in the skin of an abnormal host."[15] Any nematode or insect which normally invades tissue may cause CLM, but the primary etiologic agent is the third-stage larvae of *Ancylostoma braziliense,* an intestinal nematode of dogs, cats, and wild animals. Occasionally, CLM may be caused by other animal ancyclostomids.

The adult parasite lives in the small intestine of the host and produces eggs which pass out with the feces. With proper environmental conditions, embryonation occurs within 24 to 48 hours to form a first-stage larva which is susceptible to low temperatures and dryness. In two to four weeks, a third-stage infectious larva is formed.[1] Human infection occurs when third-stage larvae penetrate the skin. Infection with *A. braziliense* in animals produces symptoms similar to that discussed previously for *A. ceylanicum.*

CLM is seen worldwide but most often in tropical and subtropical areas. In temperate climates, the incidence is increased during the summer. The true incidence of the disease is not known.[15] Children are most frequently exposed to contaminated areas especially when playing with sand. In humans, the infective larvae do not routinely pass through the epidermis. Papules form at the entry site and vesicles form along tunnels the larvae create as they burrow. An intense tissue reaction may occur creating pruritus. Secondary bacterial infection may develop because of self-inflicted trauma. Lesions are seen most often on the feet, legs, and hands. The larvae can remain alive and travel in the skin for several weeks. The eventual fates of the migrating larvae are unknown, i.e. as to whether they are destroyed, encapsulated, or migrate to other tissues. Healing usually occurs spontaneously.

Avoidance of infection in man depends upon prevention of environmental contamination and prevention and treatment of hookworm infection in animals. Attempts should be made to limit the population of stray animals. Dogs and cats should be kept off of, or supervised well while on, sandy beaches, parks, and play areas to prevent fecal contamination. Proper disposal of dog and cat feces and enforcement of leash laws would decrease the spread of infetive larvae.

Visceral larva migrans

Visceral larva migrans (VLM) is an accidental infection due to juvenile nematodes migrating in an abnormal host. Recent literature reviews define two forms of VLM, visceral larva migrans and a separate entity when migration occurs through the eye, i.e. ocular larva migrans.[17] The usual etiologic agent is *Toxocara canis,* the common dog roundworm. Even though *Toxocara cati* is a frequent parasite in cats, it only occasionally causes VLM. In a study of soil samples, the eggs of *T. cati* were found less frequently in the soil than the eggs of *T. canis.*[17]

Toxocara canis is found worldwide. It is an extremely common parasite. One study from humane shelters and commercial breeding colonies indicated that nearly all dogs are infected with the nematode at some point in their lives.[17]

The adult parasite lives in the intestine of dogs and other wild canines. The eggs are passed in the feces and are extremely resistant to environmental conditions and may survive for months or years.[1] Under proper conditions, the egg develops into an infective second-stage larva in approximately two weeks. Transmission between dogs can occur in several ways: cannibalism, carrion eating (i.e. eating smaller mammalian paratenic hosts that have the larvae in their tissues), eating foods contaminated by eggs, coprophagia, intrauterine transmission, and mammary transmission. When an animal less than three to four weeks of age ingests infective larvae, they penetrate the intestinal wall and migrate to the lungs via the bloodstream. After a molt they travel through pulmonary capillaries to the bronchial tree, up to the trachea and epiglottis where they are swallowed and pass to the intestines. After two molts, the larvae reach the adult stage and begin producing eggs. Dogs greater than six months of age are less likely to have patent infections and therefore fewer nematodes are found in the intestines. An exception to this is seen in bitches

which may develop a patent infection after ingesting larvae from the feces of her pups.

In older dogs, the larvae reach the lungs but then migrate to the heart via pulmonary veins. After that, they migrate to other organs where they are encysted and their development stopped. This phenomenon is known as "somatic migration and age resistance." The encysted juveniles can then be activated at a later time and transmitted to fetuses via the placenta or milk. Somatic migration allows *T. canis* to live in an animal in a state which cannot be readily diagnosed. This may explain why *T. canis* remains so prevalent in the canine population despite effective anthelmintics. One study demonstrated that age resistance was not a function of previous exposure and therefore may not be due to a classical immunologic response.[18]

The efficiency of the life cycle in dogs is primarily due to transplacental migration of larvae from infected bitches to their fetuses during the last trimester of pregnancy. It is believed that most bitches harbor the larvae in their tissues for years, which can serve as a source of infection for successive litters.[19] At birth, third-stage larvae may already be found in the lungs of infected pups and eggs may be shed by the pup as early as 23 days after birth.[19] By the time a puppy is four weeks old, a female worm can be producing 200,000 eggs daily.[17] In nursing bitches, infective larvae may be found in the milk up to the 32nd day of lactation.[20]

Most pups are probably infected early in life due to the efficiency of pre-natal and neonatal transfer of the larvae from the bitch to the pups. Infection rates in pups two to six months of age have been estimated at greater than 50 percent. In contrast, dogs at least one year of age have an infection rate of less than 20 percent.[20]

Puppies and kittens with acute infections demonstrate mainly digestive disorders, i.e. diarrhea, abdominal distension, and lethargy. If infected with a high number of parasites pre-natally, animals may die early in life. Adults show few symptoms because they are usually infected with a low number of parasites.

Human infections occur by ingesting infective larvae from contaminated soil, hands, or fomites. But since the eggs are not directly infective, it is unlikely that direct contact with a pet creates a hazard. In view of the prevalence of this parasite and the number of pets in American households, the chance of exposure is high. People without pets may be exposed by contact with contaminated soil. One-third of soil samples from city parks were found to be contaminated with nematode eggs.[17] But exposure to a contaminated environment is not the only factor necessary to acquire the disease. People in high risk occupations, i.e. veterinarians, veterinary technicians, and kennel workers, do not have a higher incidence of the disease. Ingestion of infective larvae is necessary to acquire the disease and this is most likely to occur in children with a history of geophagia.

The incidence of VLM in man is not clear at this time in part because it is not a reportable disease. It is difficult to diagnose with biopsies because the larvae may be widely dispersed in tissues and until the late 1970s serologic testing was not available. From 1950 to 1970, 245 cases of ocular infections were recorded worldwide.[1] During the first year after a serologic test was available, 2,606 serum specimens were received at the Center for Disease Control and of those 782 had significant titers.[20] Approximately 2 percent of apparently healthy people have a positive skin test for *Toxocara* and up to 13 percent have antibody titers compatible with recent infections.[17] The reliability of the skin test for *Toxocara* has been debated.[12] In a group ophthalmologic practice in the Atlanta area, 41 sero-positive cases of ocular larva migrans were diagnosed in an 18-month period, which represented 37 percent of all retinal diseases diagnosed in that pediatric population.[20] Serologic tests are important in differentiating OLM from retinoblastoma to avoid unncessary enucleation of the eye, although some eyes with OLM are eventually enucleated.

As early as 1922, the possibility of infection by *Toxocara* in man was suggested, but it was not until 1952 that the larvae were definitely found by biopsy.[17] The clinical manifestations of VLM vary widely depending on the number of larvae present, frequency of infection, where the larvae are found, and the host immune response. It is seen primarily in children from one to four years of age with a history of pica. After the eggs are ingested, they hatch in the intestine and migrate to the liver via the portal system. As the larvae migrate, they create mechanical damage which may be accentuated by the host's immune re-

sponse. Symptoms seen indicating liver involvement include abdominal pain, vomiting, diarrhea, and hepatomegaly. An eosinophilia of up to 80 percent may be seen.[2]

From the liver, the larvae then migrate to the lungs via the venous system, creating coughing, wheezing, and pulmonary infiltrates. Eosinophilia may continue to be present. While migration is occurring, hypergammaglobulinemia is often seen. If frequent reinfections occur, pulmonary and hepatic infections can occur simultaneously, increasing the severity of the illness. After leaving the lungs, the larvae enter the systemic circulation and may be found anywhere in the body. Granulomas may form or the larvae may incite very little reaction. The disease in children is usually self-limiting if the source of infection is removed, but death can occur when extensive migration occurs through vital organs such as the brain or myocardium.[17]

In 1950, *Toxocara* larva remnants were found in 24 of 46 eyes enucleated because of presumed retinoblastomas (the most common eye tumor in children).[17] Ocular larva migrans is primarily a granulomatous retinal lesion and involvement of the anterior segment of the eye is uncommon. With the above in mind, it is unclear how many retinal lesions have been mistakenly diagnosed as retinoblastomas when OLM was the real reason.

Although OLM is a specific type of VLM, it shows distinct differences. Patients with OLM rarely show evidence of systemic involvement. Affected children tend to be older with no history of pica. Titers are consistently lower in cases of OLM. Without pica, it is possible that the number of larvae ingested are lower, reducing the immune response. But in the eye itself, one larva can cause a marked immune response leading to a high antibody titer in the vitreous body.

From the above discussion, it is clear that VLM is a serious zoonosis and efforts should be made to decrease its incidence.

Fecal contamination of the environment by pets is a major health problem. A total of 3,500 tons of dog feces is deposited daily in the environment.[19] Owners should be encouraged and required to pick up the feces of their pets. Enforced leash laws would decrease the distance a pet may roam. Controlling the stray population would, of course, also decrease the spread of feces. Children should be advised of pica and be taught to wash their hands after playing.

The public, particularly pet owners and veterinarians, need to be educated about the public health threats of *T. canis*. A survey has revealed that many veterinarians do not view it as a significant public health problem.[21] It has been found that only 33 of 100 veterinarians believe that it is a threat.[20] In addition, they interviewed families of OLM patients, 80 percent of whom utilized the service of a veterinarian. The majority of people indicated that the only disease they thought they could get from pets was rabies.

The most important method in preventing human infection with *T. canis* is elimination of the roundworms in pet animals. The recommended age (from the AVMA) of the first treatment for roundworms is two weeks.[20] Of 100 veterinarians randomly questioned from an AAHA directory, only 7 percent said they routinely treated animals less than three weeks of age.[20] Repeated treatment of the young is needed as continual exposure occurs through the milk and possibly through the environment. Other researchers recommend treating both the bitch and pups at 2, 4, 6, and 8 weeks after parturition.[20] Of veterinairans they surveyed, only 15 percent of them advised examination and treatment of nursing bitches.[20] Weaned pups can be treated two times, 10 to 14 days apart.[20] Dogs acquired from animal shelters or pet stores should be checked for roundworms and treated accordingly. Older animals can be treated as indicated by routine fecal exam. Several drugs are available that are effective against roundworms, i.e. piperazine, pyrantel pamoate, and dichlorvos.

Until recently, no drugs were available in preventing pre-natal transmission. Researchers have found that fenbendazole is effective in decreasing the number of *T. canis* in newborns by 89 percent when the bitch was treated from the 40th day of gestation to 14 days post partum at a dose of 50 mg/kg per day.[22] Fenbendazole has just been approved for use in the dog. Because the treatment did not eliminate *T. canis,* they recommended treatment at 2, 4, 6, and 8 weeks as mentioned previously.

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