

TOXOPLASMOSIS IN GOATS

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ABSTRACT

Natural and experimental Toxoplasma gondii infections in goats are discussed with attention given to etiology, pathogenesis, pathology and diagnosis of abortions in goats. Public health aspects of T. gondii infection in goats are also discussed.

INTRODUCTION

Toxoplasmosis is a common and widespread infection in man and many species of warm-blooded animals. Toxoplasma gondii is an obligate intracellular coccidian transmitted via 3 primary ways: congenital, carnivorar, and fecal-oral (Fig 1). Cats, including wild Felidae, are the only definitive hosts. Felidae excrete T. gondii nonsporulated (noninfectious) oocysts in their feces. Sporulation (development of infectious sporozoites inside the oocyst) may take 1 to several days after defecation, and is dependent on environmental conditions. Oocysts can survive for several months to a year under severe environmental conditions. They are remarkably resistant to most disinfectants (Dubey, 1977). After ingestion of sporulated oocysts in food or water by a warm-blooded animal the oocyst ruptures in the intestine, releasing 8 sporozoites. Sporozoites multiply intracellularly in the intestines and in associated lymph nodes, and tachyzoites (rapidly multiplying forms) are formed. Tachyzoites then spread to the rest of the body via blood and lymph and eventually encyst in the brain, skeletal and cardiac muscles, liver, and other organs. Encysted T. gondii are called bradyzoites (slowly multiplying forms). Tissue cysts are microscopic and may survive in tissues as long as the host lives.

After ingestion of infected tissues, proteolytic enzymes dissolve the cyst wall releasing bradyzoites which penetrate the

host's gut. After entry into host cells, bradyzoites transform into tachyzoites. In the host cell, the tachyzoites may undergo repeated divisions, ultimately encysting in tissues. The cycle of T. gondii is completed when tissue cysts are ingested by the cat. In the intestine of the cat, bradyzoites initiate a series of asexual generations (meronts) and the merozoites initiate the sexual cycle. After the male gamont fertilizes the female gamont, a wall forms around the fertilized female gamont to form the oocyst.

Transplacental infection can occur when a previously noninfected host becomes infected during pregnancy. Toxoplasma gondii multiplies in the placenta and then spreads to fetal tissues. Although transplacental infection can occur at any stage of gestation, the fetus is more severely affected if the dam becomes infected during the first half of gestation.

Toxoplasma gondii infection is also a common parasitic infection of goats. In addition to causing mortality in goats, T. gondii infection in goats may be a source of infection for man. This paper summarizes information on T. gondii infection in goats including its economic and public health aspects.

PREVALENCE

A recent compilation of world wide serologic surveys revealed infection rates of 0 to 100 percent (Dubey, 1986a). Infection rates increased with age (Tizard et al., 1977; Ruppanner et al., 1978; Chhabra et al., 1985). It is of interest that none of the 165 feral goats from Texas and South Australia had antibodies to T. gondii (Hein and Cargill, 1981; Dubey and Livingston, 1986). There are only a few reports of the isolation of T. gondii from the tissues of healthy goats (Dubey, 1986a). Hagiwara et al. (1978) isolated T. gondii from the tissues of 15 of 60 (25%) goats in Japan.

Distribution of T. gondii in caprine tissues and its public health significance. Toxoplasma gondii can persist in edible tissues of goats for perhaps the life of the goat. Dubey (1980) compared the distribution of T. gondii cysts in tissues of 10 naturally infected adult goats by bioassay in mice. Tissue cysts were found in the thigh muscles of 10, the hearts of 6, the diaphragms of 6, and the livers, kidneys and brains of 3 of 10 goats. These results were verified experimentally in 26 goats inoculated orally with oocysts of T. gondii (Dubey, 1981a, 1982). It was concluded that T. gondii encysted in livers, kidneys and muscles of goats and persisted in these tissues for a long time. The number of cysts per gram of tissue was greater in the liver and muscles than in the brain. Of particular interest are the results from 6 goats each fed only 10 or 100 T. gondii oocysts and killed between 335 and 441 days after inoculation. T. gondii tissue cysts were found in the thigh muscles, hearts, diaphragms and livers of the 6, brains of 5 and kidneys of 3 (Dubey, 1982). Similar results were obtained in goats congenitally infected with T. gondii (Dubey, 1981a).

Ingestion of raw or undercooked infected caprine tissues can result in T. gondii infection in humans and more importantly in cats (Fig I).

PUBLIC HEALTH SIGNIFICANCE OF TOXOPLASMA IN GOAT MILK

Toxoplasmosis has been reported in human beings (Riemann et al., 1975; Sacks et al., 1982) after drinking unpasteurized goat milk and T. gondii was found in milk of goats experimentally inoculated with T. gondii (Dubey, 1980). Therefore, goat milk should be pasteurized before human consumption. This is particularly important in infants since they are more susceptible to toxoplasmosis than adults. Although the stage of T. gondii present in milk is likely to be the tachyzoite (which is susceptible to digestive enzymes), T. gondii may survive longer in infants because the concentration of proteolytic enzymes in the digestive tract of an infant is less than that in the intestine of an adult. However, raw goat milk can be used to feed calves because cattle are more resistant to toxoplasmosis than other livestock species (Dubey, 1986b).

CLINICAL INFECTIONS

Since Munday and Mason (1979) first reported on an outbreak of toxoplasmic abortion in goats in Tasmania, Australia, reproductive failures in goats associated with T. gondii have been reported from the U.S.A., New Zealand, France, Victoria, Australia, India, Greece, and Costa Rica (for review see Dubey, 1986a).

Toxoplasma gondii can cause early embryonic death and resorption, fetal death, mummification, abortion, stillbirth, and neonatal death, depending upon the stage of gestation at the time of primary Toxoplasma infection in the doe. Clinical effects are more severe in the fetus infected in the first half than in the second half of gestation (Dubey, 1981a). Toxoplasmic abortion can occur in does of all ages. Abortion probably occurs mainly in does that acquire infection during pregnancy although abortion may be repeated in the next gestation (Dubey, 1981a, 1982). The does are generally clinically normal at the time of abortion.

The economic impact of caprine abortion due to toxoplasmosis is not known but may be high because (1) experimentally, T. gondii induces more fatalities in caprine fetuses than any other species of livestock studied; (2) the disease is usually sporadic; (3) only a minor proportion of aborted kids are submitted for diagnosis; (4) those submitted may be inadequately examined; (5) unsuitable material may be sent for diagnosis; (6) toxoplasmosis generally does not produce clinical disease in the doe at the time of abortion so that this disease does not alarm the farmer as much as bacterial and viral infections. Nurse and Lenghaus (1986) recently reported a large outbreak of toxoplasmosis in a flock of 80 Angora goats in Australia. Forty-two (52.5%) does had dead fetuses or stillborn kids.

Toxoplasmosis occasionally cause fatalities in adult goats. These naturally infected goats had encephalitis, nephritis and hepatitis, necrotizing abomasitis, enteritis and cystitis (Hartley and Seaman, 1981; Mehdi et al., 1983).

PATHOGENESIS

After the ingestion of oocysts, T. gondii multiplies in the intestinal submucosa and associated lymph nodes. Data on comparative recovery of T. gondii in various tissues of goats inoculated orally with oocysts suggest that many organisms are killed in the mesenteric lymph nodes. Parasitemia occurs during the first week and T. gondii is disseminated quickly to several caprine tissues via the lymph and blood. T. gondii encysts during the second week, frequently in visceral tissues and muscles. Goats may have fever, anorexia, diarrhea, respiratory distress and may die of enteritis and encephalitis (Dubey et al., 1980; Dubey 1980; Dubey, 1981 a-d).

In a susceptible pregnant doe, parasitemia occurs during the first 2 weeks after inoculation and T. gondii infects the fetal tissues approximately 14 days after the inoculation of the doe. T. gondii continues to multiply in the placenta longer than in other tissues of the doe. However, even in the placenta, the multiplication of T. gondii is not extensive. Nonspecific abortion may occur before invasion of the fetal tissues, probably due to fever. T. gondii invades and encysts in several tissues of the caprine fetus, particularly in the skeletal muscles. Abortion due to toxoplasmosis can occur at any time 21 days after the doe has been inoculated. The pathogenesis of toxoplasmic abortion is not clear. Some fetuses from does with heavily necrosed placentas are not infected whereas others become infected. Hormonal deregulation may be involved but has not been documented.

EPIZOOTIOLOGY

The ingestion of T. gondii oocysts appears to be the only source of postnatal infection for goats. Infection is related to chance exposure to oocysts because not all of the goats housed together become infected (Dubey et al., 1986). In most cases, infection probably occurs when goats begin to eat hay. Cats are usually present around domestic goats. They may contaminate hay, grain and water in the barn with T. gondii oocysts.

Nothing is known of the breed susceptibility to toxoplasmosis in goats. Although T. gondii can be found in goat semen (Dubey and Sharma, 1980), it is doubtful if venereal transmission is important in causing abortion in goats because the doe is likely to become infected and develop immunity before implantation of the ovum.

DIAGNOSIS

Rapid diagnosis of infective abortions in goats is important so that future breeding decisions can be made.

Gross and microscopic examinations, cytology, serology and animal inoculations can be used to diagnose abortion due to toxoplasmosis. T. gondii produces characteristic lesions in the goat placenta (Dubey, 1981b). The main lesion is necrosis of the fetal cotyledons; the intercotyledonary areas are essentially normal. The characteristic lesions consist of white flecks or multiple white/yellowish areas of discoloration up to 1 cm in

diameter. These foci may be sparse or dense and may occur in any plane of the cotyledon. Foci may be confluent and not all cotyledons are affected to the same degree. It is, therefore, necessary to wash the cotyledons thoroughly in 0.85% NaCl solution to expose deeper lesions that may otherwise be overlooked. Unlike other abortifacients, T. gondii does not produce generalized placentitis.

Microscopically, the lesion is necrosis of the trophoblasts and lamina propria of the villi (Munday and Mason, 1979). Older lesions may be mineralized. Tachyzoites are present in small numbers, individually and in clumps, especially around the necrotic areas.

There are usually no specific gross lesions in the fetus, although brain may have scattered chalky white areas of necrosis and mineralization. Nonsuppurative encephalitis is the most consistent lesion in fetal caprine toxoplasmosis. Myocarditis, pneumonitis, hepatitis and necrosis of the mesenteric lymph nodes and spleen may be present. It is more important to recognize the lesions in the placenta and fetal brain than to search for T. gondii because only a few T. gondii may be present. Moreover, tachyzoites of T. gondii are often difficult to recognize among degenerative host cells.

Serologic examination of the fetus and doe can aid in a diagnosis. Specific antibodies in presuckling kids are diagnostic of transplacental toxoplasmosis (Dubey et al., 1985, 1986). However, the absence of T. gondii antibodies does not preclude the possibility of infection because development of antibodies depends on fetal age at the time of infection and the on time lapse between infection and examination. Both blood serum and body fluids can be used to detect T. gondii antibodies. It is important that serologic examination of kids is performed before colostrum is given. This is because the amount of antibodies in colostrum is several times greater than that in serum and may cause high neonatal titers which may persist up to 12 weeks.

The presence of high T. gondii antibody titers in serum of does is not necessarily diagnostic of recent infection, because titers may remain high into the next breeding season (Dubey et al., 1985). However, if specific antibodies cannot be detected, then abortion is not due to toxoplasmosis since antibodies are highest before abortion.

Numerous serologic tests have been used (Dubey, 1986a). Detection of a significant amount of antibody in the fetus by any test is indicative of transplacental toxoplasmosis because maternal antibody is not known to cross the caprine placenta.

The isolation of T. gondii from placental and fetal tissues by mouse inoculation can confirm the diagnosis and is particularly helpful in recovering organisms from autolysed tissues that may be unsuitable for histologic or serologic examinations. Placental cotyledons and fetal brain are the best tissues for isolating T. gondii (Dubey, 1977). If neither

tissue is available, muscles from the limbs can be used. Material used for isolation studies should be refrigerated but not frozen because T. gondii in tissues are reduced or killed by freezing.

PREVENTION AND CONTROL

It would be desirable to have a vaccine for the immunization of goats to prevent abortion since goats can develop a protective immunity to toxoplasmosis (Dubey, 1981c, d). No vaccine is yet available, but research is in progress. Fetal membranes and dead fetuses should not be handled with bare hands to prevent infections in people handling them and should be buried or incinerated to prevent infection of felids and other animals on the farm. Ideally, cats should not be allowed near pregnant goats but, in practice, it is difficult to do. Cats should be neutered to curtail breeding on farms because most cats become infected with T. gondii soon after weaning and, once they have shed oocysts, they acquire immunity to reinfection. Grain should be kept covered to prevent oocyst contamination. Goat milk should be pasteurized or boiled before human consumption. All tissues and meat products from goats should be heated to 70 C before human or animal consumption.

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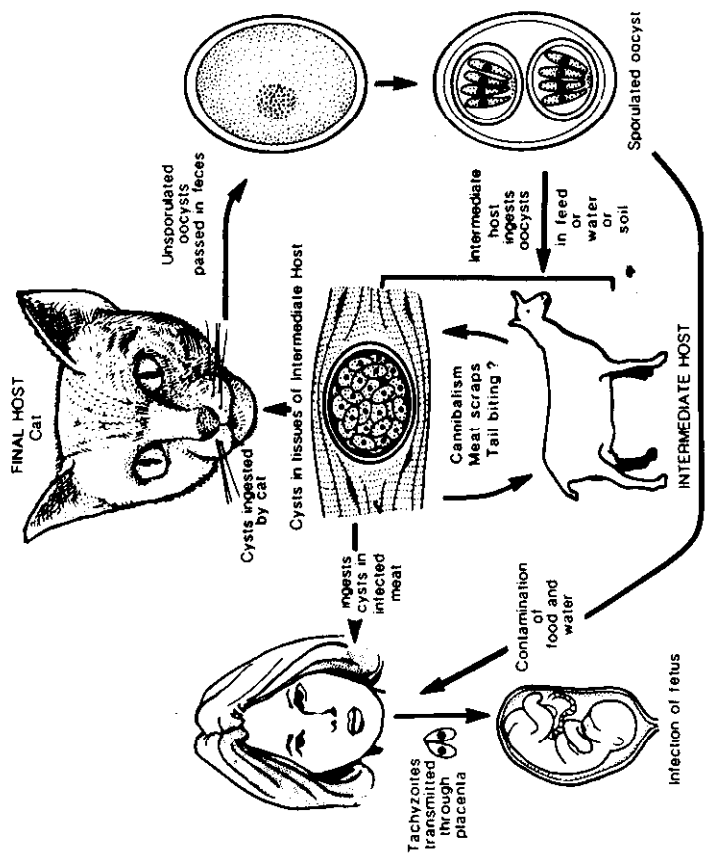


Figure 1. Life cycle of *Toxoplasma gondii*.

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