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Review Article

A Review of Some Protozoan Parasites Causing Infertility in Farm Animals

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The paper reviews some protozoan parasites responsible for infertility in livestock with specific emphasis on neosporosis, sarcocystosis, toxoplasmosis, and trichomoniasis. It highlights the transmission, pathogenesis, clinical signs, diagnosis, prevention, control, and treatment of the individual parasite. It is concluded that these parasites are often overlooked during investigation into causes of abortion in livestock, and they may, however, be responsible for some infertilities in this group of animals which may result in severe economic losses.

1. Introduction

Infertility is a major cause of economic losses and a major limitation to achievement of optimum efficiency in the livestock production system largely as a result of loss of milk production in the dairy sector and increase in culling rates. The causes of infertility can be numerous and complex [1]. They may relate to impairment in Graafian follicle development and maturation, onset of estrus, ovulation, successful coitus, fertilisation, implantation, and the development and delivery of the foetus and its membranes. Any temporary state interfering with these routines, such as disease conditions, malnutrition, inadequate herd management, hereditary and congenital factors, hormonal disturbances, or environmental changes, makes the animal infertile [2].

Parasitic infections are typically associated with poor and often marginalized communities in low-income countries [3]. Protozoan parasites are a significant cause of abortion and infertility in domestic ruminants. *Toxoplasma gondii* and *Sarcocystis* species are a common cause of extensive abortion in livestock and they both have a two-host life cycle. Carnivorous definitive hosts spread the infection through their feces and domestic ruminants are intermediate hosts. A similar, recently recognized protozoon, *Neospora* spp., has emerged as an important cause of reproductive disease, especially as an abortifacient in cattle. *Neospora* is presumed to also have a two-host life cycle, although the definitive host(s) has not been identified. The venereally transmitted *Tritrichomonas foetus* has also been reported to be an important cause of pregnancy loss in naturally bred cattle throughout the world [4].

2. Neosporosis

Neospora caninum is an obligate protozoan (Coccidian) parasite related to *Toxoplasma gondii* and was first associated with neuromuscular disease in dogs in the mid-1980s [5]. It is globally distributed and has been identified as a major cause of infertility in cattle resulting in considerable economic losses [6, 7]. The parasite has been reported to have a wide host range and infects all the major livestock species, as well as companion animals, and certain wildlife species [8, 9]. Clinical infections have also been reported in horses, goats, sheep, and deer. Direct production losses and treatment costs of *N. caninum* infection have been reported in the Maritime Provinces of Canada to be €1,921 annually [6]. Also, it has been estimated to result in an average annual cost of €249 with a maximum of €5,604 for a herd with 50 lactating cows [7]. Furthermore, the total annual losses due to *N. caninum* infections/abortions were estimated to range from

a median US $1.1 million in the New Zealand beef industry to an estimated median total of US $546.3 million impact per annum in the US dairy population [10].

Infection in cattle was first recognised in 1988 as causing abortion and infertility in cattle, and it is now recognised as an important cause of reproductive problems and abortion in cattle worldwide [11]. Economic implication of this infection in livestock is as a result of reduction in productivity of affected animal in terms of reproduction losses, decrease in milk production, premature weaning of calves, and weight losses [12].

2.1. Transmission and Pathogenesis. Dog is both the definitive host and intermediate host for the parasite [13]. Whilst dogs act as definitive hosts to *N. caninum* and can spread the infection in their faeces, their role in the horizontal transmission of the disease is not usually considered as important as vertical, congenital transmission from dam to calf during terminal stages of gestation or postnatally via milk [11, 14, 15]. Horizontal transmission from cow to cow does not take place [16]. Dogs can shed infectious oocysts for a variable period of time after infection and because the oocysts are very resistant to harsh environmental conditions, it is unclear how long they survive in the environment [17]. Intermediate hosts (cattle) ingest oocysts that are found in contaminated food and water [18, 19]. Infection spreads (as tachyzoites) to other tissues in the cow, for example, neural cells, macrophages, fibroblasts, vascular endothelial cells, hepatocytes, and, if pregnant, to the placenta, where damage to the placenta or transmission vertically through the placenta to the foetus can occur. Abortion may be a result of both the primary damage and the immune-mediated inflammatory response of the cow [20]. As a result of the immune response generated, the tachyzoites can transform into bradyzoites (a slowly dividing "dormant" stage or tissue cyst). They remain latent until the immune system of the cow is suppressed, when infection then can recrudesce [11]. Bradyzoites in tissue cysts can be consumed by dogs and then complete the life cycle of the parasite.

2.2. Clinical Signs. Infection due to *N. caninum* is common but seldom results in clinical disease [15]. However, it can result in abortion which can occur at any stage of pregnancy but mostly occurs between 3 and 9 months of pregnancy (particularly 5 to 6 months), with moderate rotting (autolysis) of the foetus. Immunologically competent foetus may be born alive but is congenitally infected, although still-birth may occur. Thus, foetuses may be resorbed, mummified, autolysed, still-born, born alive with clinical signs, or born clinically normal but chronically infected and may manifest nervous symptoms [21]. Such animals may remain malnourished leading to increased mortality, hydrocephalus, exophthalmos, underweight, and difficulty in growth [22]. Congenitally infected heifers may run the risk of abortion, especially in the first gestation and continue to pass the infection to their offspring, while horizontally infected heifers may or may not pass the infection on to their offsprings [4].

2.3. Diagnosis. Clinical signs are of little help in the diagnosis of neosporosis. Diagnosis can be made by combination of serology, for example, immunofluorescent antibody tests (IFAT) and enzyme linked immunosorbent assay (ELISA), with immune histochemistry and histopathology of aborted foetus [23]. The most promising specimens for the diagnosis of neosporosis are heart, liver, placenta, and body fluids or blood serum; examination of several tissues gives a higher diagnostic rate; the fetal brain is the most consistently affected organ and has the most characteristic lesion. Polymerase chain reaction (PCR) is also an important tool in the diagnosis of neosporosis, efficiency of which is dependent on the laboratory, stage of the autolysis of the foetus, and the sampling procedures [22, 23].

2.4. Prevention/Control. In herds free of *N. caninum*, prevention of the introduction of the infection through standard biosecurity measures is the primary goal [11], whereas, in *N. caninum*-infected herds, control programs are based on decreasing the vertical transmission in a herd by reduction of the number of seropositive cattle and/or decreasing the risk of horizontal transmission of *N. caninum* principally by controlling the definitive host population as a source of oocyst contamination [24, 25].

2.5. Treatment. There is no safe and effective treatment for bovine neosporosis. Drug therapy is not usually advocated because it is usually prophylactic involving long-term administration which can result in unacceptable milk and meat residues and withdrawal problems [15, 26].

3. Sarcocystosis

Sarcocystosis is a disease of birds and mammals caused by several species of single-celled coccidian protozoa in the group called *Sarcocystis*. The Greek word *sarco* refers to flesh or muscle, with sarcocyst referring literally to a cyst in muscle. *Sarcocystis* is a genus of Coccidian protozoan parasites that is associated with the presence of muscle cysts which are usually grossly evident, in striated muscle of an intermediate host species [27]. The muscle cyst stage in the intermediate host is relatively benign. However, the early developmental stages in other tissues of the intermediate host can prove that fatal. *Sarcocystis* species occasionally cause abortion in cattle [28]. Three species have been reported in bovine muscle; they are *Sarcocystis cruzi* with canids as definitive hosts, *Sarcocystis hirsuta* with felids as definitive hosts, and *Sarcocystis hominis* with primates as definitive hosts [29, 30]. Abortions are usually sporadic, but they may occur as "storms." *Sarcocystis* is spread by carnivores (wild and domestic) and cattle become infected by ingesting feed or forages contaminated by carnivore faeces. Abortions occur at any stage of gestation. Infection due to *Sarcocystis* results in economic losses as a result of weight loss, anorexia, decreased milk production, anemia, and prostration often culminating in death [31].

3.1. Transmission and Pathogenesis. *Sarcocystis* spp. normally develop in 2-host cycles consisting of an intermediate host (prey) and the final host (predator). Species-specific prey-predator cycles have been demonstrated, for example, cattle-dog (*S. cruzi*), cattle-cat (*S. hirsuta*), and opossum-psittacine (*S. falcatula*) [26]. Definitive hosts which are carnivores become infected by feeding on preys whose tissues contain encysted *Sarcocystis* protozoa and subsequently shed the infectious sporocysts in their faeces. The intermediate host ingests the sporocysts, which develop into sarcocysts in the muscle of the host [32].

3.2. Clinical Signs. Most animals infected with *Sarcocystis* spp. are asymptomatic, and the parasites are seen mainly as an incidental finding at necropsy. However, clinical cases are occasionally reported, particularly in the intermediate host, but often depend on the number of sporocysts ingested and the species of infecting *Sarcocystis*. Clinical signs reported in heavily infected cattle include fever, anorexia, wasting, decreased milk production, diarrhoea, muscle spasms, weakness, hyperexcitability, pneumonia, haemorrhages, anaemia, icterus, prostration, and death. These signs may last several days to several weeks. When pregnant mammals ingest sporocysts, they may abort or give birth to a still-born foetus. Definitive hosts typically do not show any clinical signs of sarcocystosis [33].

3.3. Diagnosis. The diagnosis is usually done postmortem by examination of the skeletal muscles. The cysts are tan to white resembling parallel grains of rice, which may be observed throughout the breast muscle tissue and other muscles, including the heart. The affected tissue often produces a gritty sound when cut with a knife. In some species, the cysts may be visible to the naked eye (ducks, mice, rabbits, and sheep), but, in most species, microscopic examination is required, which includes peptic digestion, muscle squash, squeezing methods, and indirect fluorescent antibody test Antemortem diagnosis may be made with the use of dermal sensitivity test or complement fixation tests [34].

3.4. Prevention/Control. Dogs and other carnivores should not be allowed access to raw meat, offal, or dead animals as these may harbor cysts of *sarcocystosis*. Dogs and cats should not be allowed access to buildings where livestock feed or animals are kept [26, 33]. It has been reported that good sanitation is important in the control of this disease in domestic and captive animals. Freezing meat prior to cooking can also help to kill the organisms. Condemned carcasses as a result of sarcocystosis should be disposed of by deep burial to avoid consumption by scavenging animals.

3.5. Treatment. There is no effective treatment for cattle infected with *Sarcocystis* spp. However, anticoccidial drugs like amprolium and salinomycine have been used to prevent severe illness and death in experimentally infected calves [35, 36]. In addition, anti-inflammatory medications or vitamin E may be given, and supportive care may be necessary [37].

4. Toxoplasmosis

Toxoplasma gondii is a systemic coccidian, a universal parasite, a protozoan, and a member of the suborder *Eimeriina* [30]. It is a tissue protozoan infecting humans and warm blooded animals. It has three life stages, namely, tachyzoites, bradyzoites, and oocyst containing sporozoites, and is of zoonotic importance of worldwide distribution [38, 39]. A primary infection in pregnant animals is capable of establishing a placental and foetal infection, which may result in foetal death and resorption, abortion, or stillbirth [38]. The definitive host for this parasite is the cat and has, therefore, not developed resistance to the parasite [40]. In the definitive host for *T. gondii*, the organism undergoes both sexual and asexual life cycles. Other mammals and birds are intermediate hosts in which *T. gondii* undergoes only the asexual life cycle [41]. Infection with this parasite has been associated with a variety of mental health and brain problems in humans, including schizophrenia, neurosis, and brain cancer [42].

4.1. Transmission and Pathogenesis. The sexual cycle is initiated when a susceptible cat ingests oocysts or tissue cysts. In the latter case, bradyzoites excyst from the tissue, penetrate the epithelial cells of the small intestine, and disseminate to other tissues, such as striated muscle and the brain, where they encyst and are resistant to attacks by the host's immune system [41, 42]. Bradyzoites in animal tissues are a source of toxoplasmosis in humans. In the enteroepithelial cells of the cat, the oocysts undergo gametogony, and the gametocytes are released into the lumen of the intestine and pass out in the faeces. Sporulation occurs in the environment within 1 to 5 days, with each oocyst containing 8 infectious sporozoites. Four days after ingesting tissue cysts, the cat is capable of shedding millions of oocysts in the faeces which can survive for 12–18 months in the environment, depending on climactic conditions, and they serve as an important source of infection for grazing animals [43, 44]. Oocysts may be shed in smaller numbers for a few days or not at all, 20 days after a cat ingests oocysts or tachyzoites [41].

4.2. Clinical Signs. Clinical signs develop as a result of inflammation in infected tissues. The course of the disease varies a great deal between animal species and between age groups being more of a problem of the young. If organism replication is attenuated by immune response, tissue cysts may develop containing the more slowly dividing bradyzoites. Bradyzoites can later be reactivated under conditions of immunosuppression to divide rapidly as tachyzoites, potentially resulting in clinical disease. Bradyzoites can persist in affected tissues for the lifetime of the host. Infection of ewes during early pregnancy may result in embryonic death and resorption due to the immature nature of the foetal immune system. Infection during mid-pregnancy may result in either foetal death followed by mummification or foetal retardation due to compromised placental nutrition and foetal infection. Characteristic lesions on the infected placenta are white spots visible to the naked eye due to areas of necrosis, resulting in

the impaired function of the placenta [45]. Late pregnancy infection may result in abortion of freshly dead lambs or birth of weak lambs with high neonatal mortality rates [43].

4.3. Prevention/Control. Measures should be taken to limit breeding of cats and their prevention from gaining access to livestock feed and pasture. The coccidiostat drug, decoquinate, can be fed to pregnant ewes as an aid to prevention of abortions due to toxoplasmosis. Despite the current knowledge of immunology, pathology, and genetics related to the parasite, a safe vaccine for prevention of the infection in both humans and animals does not exist [46]. However, a live, attenuated vaccine, containing the S48 strains (Toxovax), has been used to vaccinate sheep. The vaccine should be given at least three weeks before mating. One dose of the vaccine is enough to protect sheep for a period of 18 months [47]. T-263 strain of *T. gondii,* ME-49 strain, and 60Co irradiated tachyzoites of the Beverley strain of *T. gondii* have been used to control oocyst shedding in cats [48–50].

4.4. Treatment. In the face of a late abortion storm, injectable or oral sulphonamide antibiotics can be used to reduce losses, a combination of sulfamethazine and pyrimethamine has also been reported to give good results.

5. Trichomoniasis

Trichomoniasis is a venereal disease of cattle, characterized primarily by early pregnancy loss and, occasionally, by abortion and pyometra leading to significant infertility. It is worldwide in distribution and has been reported to possibly be the third most common cause of abortion in cattle (after Brucellosis and Leptospirosis) [51]. The causative agent, *Tritrichomonas foetus*, is a flagellated protozoan parasite transmitted from infected, asymptomatic bulls to heifers or cows at the time of coitus [52]. The infection has been reported in dogs [53] and in cats as a cause of large-bowel diarrhea [54, 55]. Furthermore, *Tritrichomonas suis* has been reported to be a gastrointestinal commensal of pig [53].

5.1. Aetiology and Pathogenesis. The parasite has characteristic three anterior flagellae about the same length as the body of the parasite and an undulating membrane [4]. Younger bulls are less liable to become persistent carriers than older bulls because the crypts of the penile integument and prepucial mucosa are less developed in young than older animals [26]. The parasite colonises the uterus, cervix, and vagina but survives less in the vulva; it does not prevent fertilisation but causes early embryonic death, resulting in irregular extended return to oestrus [4].

5.2. Clinical Signs. Neither the cow nor the bull appears ill at any time, when they are infected with this organism [56]. However, there is marked subfertility, extended return to oestrus, and long calving intervals. There is presence of pyometra and early gestation abortions (1–6 weeks). In some cases, despite infection, pregnancy is not terminated by abortion and a normal, full-term calf is born [55]. Infected cows or heifers may have a uterine discharge for several months [26]. The aborted foetus is usually fresh and parasites disappear quickly from vaginal discharges after abortion (usually within 7 days) [4].

5.3. Diagnosis. Diagnosis is confirmed by demonstrating *T. foetus* organisms from specimens obtained from genital tract of female cattle, prepucial material of bulls or aborted foetal, and placental tissues [57]. Other diagnostic procedures include culture of the organism, polymerase chain reaction (PCR), and immunological tests [58–60].

5.4. Prevention/Control. Trichomoniasis is a "self-limiting" disease in the nonpregnant cow with an involuted uterus. After being sexually rested for 3 or 5 cycles, many cows develop some immunity and their fertility improves. Once infected, however, males become permanent carriers of the pathogen; therefore, only clean bulls or semen should be used for breeding and cows with abnormal genital tracts should be culled [52]. "Carrier" bulls can reinfect treated, recovered, and susceptible females and should, therefore, be culled.

5.5. Treatment. There is no approved chemotherapeutic agent for treatment of trichomoniasis in cattle [60].

6. Concluding Remark

The most serious deterrent to efficient animal production and herd profitability is pregnancy loss, of which parasitic infections can be a possible cause. These infections are, however, often poorly considered during investigations for a diagnosis. This may lead to inaccurate diagnosis of the cause of infertility. Maximizing reproductive efficiency, however, is a major production management goal and cannot be compromised, for the success and financial profitability of enterprises. Therefore, parasitic infections should be borne in mind during such investigations.

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