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REVIEW ARTICLE OPEN ACCESS

MAIN PARASITIC GASTROENTERITIS IN THE NEONATAL PHASE OF DOGS AND CATS: REVIEW

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ABSTRACT

The neonatal period within veterinary medicine is considered one of the main obstacles in health care. Therefore, pediatric care and support should be involved throughout the neonatal phase, from birth to the first 30 days of age. There are several important differences between neonate puppies and adult dogs and cats, such as anatomy and physiology, those differences play a vital role in the maintenance of animal well-beingand provide theman appropriate therapy with low toxicity. Parasitic diseases are one of the leading disorders that make up the mortality rate of neonates, which is mainly due to immunological immaturity, thereby making them unable to fight pathogens. Hencethis study aims to elucidate the main parasitic gastroenteritis that affects newborns,to reduce the mortality rate caused by protozoa, nematodes, and cestodes. The diagnosis and correct treatment of these worms reduce the risk of contracting worms in puppies and spreading them into the environment. However, there may be situations where the diagnosis of these worms is not always possible, an example of this would be rescued newborns, since these worms can reside from an infected mother to the puppies, thus deworming protocols are recommended.

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INTRODUCTION

During the neonatal phase, the physiology and anatomy of dogs and cats go through a dramatic changeas aresult ofthe adaptation from the intra to the extrauterine life at birth. In addition to this, the newborn's bacterial colonization starts from its passage through the birth canal. Thereby, this conditionassociated with immunological immaturity, makes it susceptible to metabolic and infectious disorders and parasitic diseases. The neonatal stage is equivalent from birth to the first 30 days of life (JERICÓ et al., 2014), and parasitic diseases are one of the major causes of death that affect animals in this period. The mortality rate reaches about 26.0% for dogs and 27.3% for cats (ROOT-KUSTRITZ, 2004). Thus, having the knowledge of the most common parasites in puppies, their dissemination, diagnosis, and how they can be treated, as well as pediatric care are crucial to maintain the health of these animals and reduce the mortality rate. Many physiological, anatomical, and biochemical differences between puppies and adult animals may result in changes in pharmacokinetics and pharmacodynamics, and therefore the bioavailability of drugs. Factors such as absorption, distribution, metabolism, and excretion of drugs must be consideredto avoid drug intoxications.All these aspects are fundamental to determine the right dose for neonates with appropriate knowledge about how they act in the organism. Therefore, this study aims to carry out a literature review about the main parasitic gastroenteritis in the neonatal phase of dogs and cats to contributeto thereduction of mortality rate in these animals.

Neonatology of dogs and cats: Neonatology is a branch of Veterinary Medicinethat studies neonatal care and disordersas well as the physiology of neonates. Pediatric care should be present throughout the neonatal phase since puppies are susceptible to infectious and parasitic agents at birth which are caused by physiological immaturity, mainly of the immune system(SORRIBAS, 2011). The neonatal period varies according to the authors. It can be the interval between birth and when the umbilical stump falls off or even when the puppy acquires immunity (LOURENÇO and FERREIRA, 2015).MCGEADRY et al. (2017) reported that it can be the transition between fetal life and adult life, varying according to

the breed and species of the animal. On the other hand, GRUBB (2018) and MATHEWS (2005) consider the first six weeks of life, while PRATS (2005) differ the neonatal phase of dogs and cats; in dogs he states that it is from birth to the second week of life, and in cats he believes it is from birth to the tenth day.

Aspects of general physiology of neonatal dogs and cats: The knowledge of the physiology of the neonate interferesthe conduct of the veterinarian to the therapeutic choice and the diagnosis of any illness(LOURENÇO and MACHADO, 2013). Neonates are incapable of controlling their body temperature during the first four weeks of life. They have a small amount of subcutaneous fat (CRESPILHO et al., 2007), and the tremor reflexes and vasoconstrictor mechanisms are not fully functional becausehypothalamic control is absent (JOHNSTON et al., 2001). Hence, thermogenesis occurs by the breakdown of brown fat distributed throughout the body, besides that, breastfeeding also works as a heat source (SORRIBAS, 2007), however, the mother must be evaluated to prevent transmission of *Toxocara canis* larvae to the puppies (PETERSON and KUTZLER, 2011). The blood count varies until six months oldfrom then onresembles an adult blood count. At birth, neonates have macrocytosis and high hematocrit. In the third to fourth week, until around two months old, the blood count indicates physiological anemia wherein the pup has a lower red blood cell count, including polychromasia, and the reticulocyte count is high (PRATS, 2005). In the leukogram, there is leukopenia around the seventh to the fourteenth day of life (FRESHMAN, 2005). The neonatal circulatory system is characterized by low blood volume and low blood pressure as well as low systemic vascular resistance. Therefore, the compensatory mechanisms like high cardiac output and heart rate maintain the tissue perfusion (PETERSON and KUTZLER, 2011). The activity of baroreceptors isfunctional at four days old,so a short period of anoxia can induce bradycardia and hypotension, and a loss of volume can lead to a hypovolemic shock (LANDIM-ALVARENGA, et al., 2006).

Oxygen consumption in neonates is two to three times higher than in adults (CRESPILHO et al., 2007). The high respiratory rate compensates for low volume and ventilation. Factors like a small airway, high metabolic, and lower resistance to muscle fatigue make the pup susceptible to anoxia, and the compensatory respiratory response becomes unviable (LANDIM-ALVARENGA, et al., 2006).
The kidney is undeveloped until three weeks old, both from thephysiological and anatomical point of view.In addition, the neonate has a lower renal blood flow and therefore declinesthe glomerular filtration rate, glucose reabsorption, and urine concentration. Thus, drugs excreted by the kidneydecreases (FEITOSA, 2008). Some liver functions are also not entirely developed in neonates. The biotransformation of drugs in phase 1 reactions occurs more slowly since the hepatic microsomal cytochrome P450 is incomplete, and some liver microsomal enzymes will be fully functional at four to five months old. This condition is a significant contributory factor to the increase of the half-life of drugs metabolized in the liver, and so a dose reduction or longer intervals between drug administration is required (GRUBB, 2018). Moreover, the physiological response to hypoglycemia is insufficient due to the limited hepatic glycogen storage. However, the pup is highly susceptible to hypoglycemia when associated with these factors (CRESPILHO et al., 2007). At birth, the neonate assumes the functions of the digestive system, which were carried out by the placenta. In the first two to three days of life, there is neonatal intestinal colonization by bacteria from the maternal microbiota. Histologically, the intestine villi and digestive enzymes increase as a result of milk ingesting by the pup. Thus,the absorption of drugs from the gastrointestinal tract does not differ from adult animals when administered orally. In addition, peristalsis occurs according to the pressure gradient since the electrical activity of the gastrointestinal tract is present only after 40 days of life (PRATS, 2004).

Immune system: Neonates have an immature immune system, which increases their vulnerability to infectious and parasitic agents. According to SORRIBAS (2011) and SOUZA et al. (2017), the neonatal phase has a mortality rate of 27.3% for catsand 26.0% for dogs in the first weeks of life since the puppies are unable to fight pathogens. The placenta is classified as endotheliochorial once it is composed of four layers of tissue, and the chorionic epithelium involves contact with the maternal endothelium vessels; withal it allows about 5% to 10% of immunoglobulins G (IgG) to cross and reach canine fetuses and 25% to feline fetuses. Colostrum is essential for newborns as it provides them a passive immune transfer thanks to the colostrum intake during the first hours of life. Colostrum is mainly composed by immunoglobulins A (IgA) and IgG, but it also contains immunoglobulins M (IgG) and immunoglobulins E (IgE) (TIZARD, 2017). Theimmunoglobulins are absorbed in the intestine, and then these antibodies become circulating in the body for a period of three to six weeks,which they gradually reduce, being able to stay for up to 14 to 16 weeks(CHASTANT-MAILLARD et al., 2012). The maternal immune status influences the concentration of immunoglobulins in colostrum. Hence, if the mother is not vaccinated, the protection transferred to the pup may not be enough to prevent disease; however, if the mother is vaccinated the antibody titer is higher. In addition, increased litter sizes are related to lower concentration of immunoglobulins distributed to each neonate (GREENE and SCHULTZ, 2006). Colostrum is also highly concentrated in alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT). Therefore, if the puppy does not ingest the colostrum required to cover its needs at birth, it is possible to measure these enzymessince they are absorbed in the gastrointestinal tract.Nevertheless, it is also achievable to diagnosea failure of the passive immunity by measuring IgG in the serum by ELISA.Thus, neonates that have not suckled colostrum or the quantity of colostrum ingested was insufficientmay receive serum from either vaccinated animals or exposed to pathogens (LITTLE, 2013).

Parasitic gastroenteritis

Protozoa

Giardiasis : Giardiasis is a parasitic protozoan disease of dogs and cats caused by *Giardia duodenalis*, also known as*G. intestinalis* and *G. lambia*; the occurrence of giardiasis is of potential significance from a clinical perspective, and it is also a common cause of chronic diarrhea in humans. It belongs to the genus *Giardia*, which are flagellated protozoa from the phylum Sarcomastigophora, class Zoomastigophora and order Diplomonadida. This parasite occurs in cyst and trophozoite forms, in which trophozoite is the activated and mobile form (Figure 1) and the cyst the latent stage of the development (TAYLOR et al., 2017). This agent has a monoxene life cycle. The trophozoites adhere to the intestinal mucosa through a ventral structure denominated adhesive disk (TAYLOR et al, 2017), where they multiply by binary fission. The intestinal pH, immune system, and the stimulation of bile salts promote cyst formation (Figure 2), which are eliminated in the stool for one to two weeks after infection. In addition, under environmental conditions with sufficient humidity and temperate, the cysts can maintain their infectious capacity for more than two months (BARR, 2021).

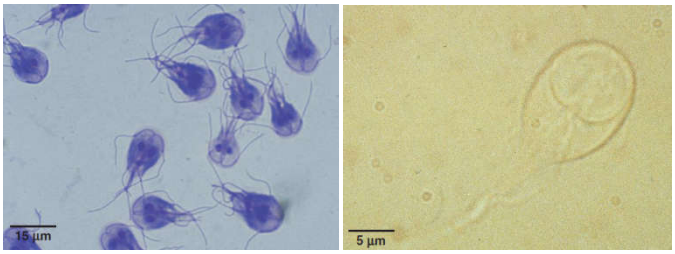

Figure 1. *Giardia* spp. trophozoitein direct smear of stained (left) and unstained (right) feces. (ZAJACand CONBOY, 2021)

Giardiasis is transmitted by fecal-oral route through the ingestion of cysts incontaminated water or food (BARR, 2021). It is important to note that untreated water may contain Giardia since the cysts are resistant to the chlorination process used in water treatment (ZAJAC and CONBOY, 2021). Moreover, asymptomatic animals are potential

carriers of cysts, whereupon they can eliminate the pathogen in the environment, increasing the incidence of giardiasis (BARR, 2021). Therefore, puppies can contract *Giardia* by living with infected mothers and in environments that are not properly sanitized (JAZAC and CONBOY, 2012). Once the cyst is ingested, it is ruptured by gastric acid and digestive enzymes, and then the parasite divides into two trophozoites that attach to the intestinal microvilli (PAYNE and ARTZER, 2009). *Giardia* leads to villous atrophy and hypertrophy of the crypts triggering the malabsorption syndrome, which manifests symptoms such as diarrhea, pasty stools with mucus, steatorrhea, abdominal pain, weight loss, lethargy, and dehydration (TAYLOR et al., 2017). Immunosuppressed puppies may develop the chronic form of the diseaseand remain withchronic diarrhea for months, producing nutritional deficiencies (PAYNE and ARTZER, 2009).

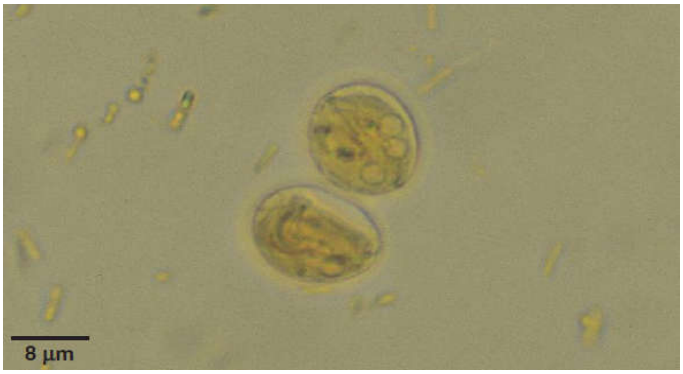

Figure 2. Fecal fluctuation test containing *Giardia* spp. cyst. (ZAJACand CONBOY, 2021)

Isosporosis: Isosporosis is a parasitic disease, commonly called coccidiosis (TAYLOR et al., 2017). It is caused by coccids of the genus *Cystoisospora*, which before the taxonomic revision were called *Isospora*; but today, they are considered synonyms (BARTA et al., 2005). These parasites belong to the phylum Apicomplexa, class Conoidasida, and order Eucoccidioride (TAYLOR et al., 2017). Dogs are most frequently infected by *Cystoisospora canis*, *C. ohioensis*, and *C. burrowsi*. These species differentiate by the oocyststherefore, *C. canis* (Figures 3 and 4) is the largest one (BOWMAN, 2021). Nevertheless, the most common species found in cats are *C. felis* (Figures 3 and 4) and *C. rivolta*, in which *C. rivolta* has the largest oocyst (DUBEY et al., 2009).

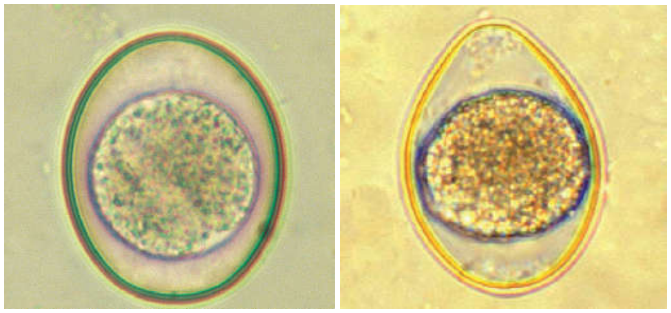

Figure 3. Non-sporulated oocysts of *Cystoisospora canis* (left) and *Cystoisospora felis* (right). (BOWMAN, 2013)

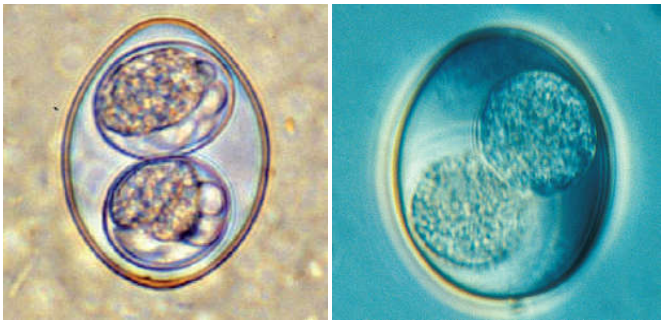

Figure 4. Sporulated oocysts of *Cystoisospora canis* (left) and *Cystoisospora felis* (right). (BOWMAN, 2013)

The non-sporulated oocysts in the animal's feces are released into the environment. Temperatures below 45ºC are favorable for oocysts to sporulate (BOWMAN et al., 2002), commencing a process called sporogony (MENESES, 2016), in which the nucleus of the oocyst forms two sporoblasts, and then develop into a sporocyst, each sporocyst contains four sporozoites (DUBEY et al., 2009). The infection occurs through the ingestion of oocysts from licking anycontaminated surface. Moreover,puppies become infected by living with infected mothers in environments that are not thoroughly cleaned (BOWMAN, 2021).

Therefore, once the oocysts are ingested,the sporozoites are released and penetrate the epithelial cells of the small intestine (BOWMAN et al., 2002), where they start the process of schizogony, which corresponds to the asexual proliferative phase. Sporozoites mature intoschizonts,which containmerozoites. These organisms may undergo the asexual replicative cycle or may begin the sexual stage, known as gametogenesis, where merozoites differentiate into sexual forms known as macro or microgametocytes. They contain the macro and microgametes, respectively. Thus, the microgametes fertilize the macrogametes forming the oocystsexcreted in the feces (MENESES, 2016). Immunocompromised puppies, as well as early weaned animals, are more susceptible to isosporosis (ZAJAC and CONBOY, 2021). Depending on parasitic load and how immunodeficient the pup is, it may cause severe dehydration and lead to death (LAPPIN, 2010). In milder infections, the growth and development of these animals can become compromised (DAUGSCHIES et al., 2000). The most common symptoms are vomiting, abdominal pain and decreased appetite (LAPPIN, 2010). In severe cases, there may be bloody diarrhea and anemia. In addition, respiratory and neurological signs have been described in some animals (ZAJAC and CONBOY, 2021). *C. canis* infections are generally more severe leading to the formation of petechiae and ulcerations in the intestinal epithelium (BUEHL et al., 2006).

Roundworms

Toxocariasis: Toxocariasis is an enteric disease caused by the parasites of the phylum Nemathelmynthes, class Secernentea, and order Ascaridida. Dogs are infected by *Toxocara canis* (Figure 5), whose males measure around 10cm, andfemales can be as long as 18cm in length. However, *T. cati* (Figure 5) is the parasitic nematode of cats, in which males vary from 3cm to 6cm and females from 4cm to 10cm (TAYLOR et al., 2017). On top of that, *Toxascaris leonina* can also affect dogs and cats, but it occurs less frequently (Figure 5) (EPE, 2009). The adult forms of *Toxocara spp.*lives in the lumen of the small intestine of the host in which they reproduce, and then the embryonated eggs (Figure 6) are shed in the feces. Under ideal conditions of temperature and humidity, the embryo inside the egg undergoes three larval stages, developing from L1 to L3 in a period that can vary from three weeks to months (OVERGAAUW, 1997). These eggs can remain infectious for several years, depending on the environmental conditions (EPE, 2009).

Once the *T. canis* eggs are ingested, they hatch the stomach releasing L3, which enters the intestine and reaches the liver via the hepatic portal system. After that, the third larval stage attains the lungs, where they develop to L4 (CURY and LIMA, 2002)thus, they can go through the hepatic tracheal migration or tracheal migration, in which the larvae penetrate the pulmonary capillaries, migrate through the alveoli into the bronchi, enters the trachea and move to the glottis, where they are swallowed, and then return to the intestine to develop from L4 to L5 and to become adults (OVERGAAUW, 1997). This cycle has a minimum prepatent period of four to five weeks, and this kind of migration usually occurs in dogs of up to two to three months old (TAYLOR et al., 2017). As a result of acquired resistance to parasite in dogs older than three months,somatic migration occurs (OVERGAAUW, 1997) which the larvae return from the lungs to the blood circulationand move to other tissues, such as skeletal muscle, liver, heart, lungs, brain, and gastrointestinal tract (TAYLOR et al., 2017), where they remain latent (EPE, 2009).

Figure 5. Morphology of the anterior region of the adult parasites *Toxocara cati* (left), *Toxascaris leonina* (middle) and *Toxocara canis* (right). (ZAJAC and CONBOY, 2021)

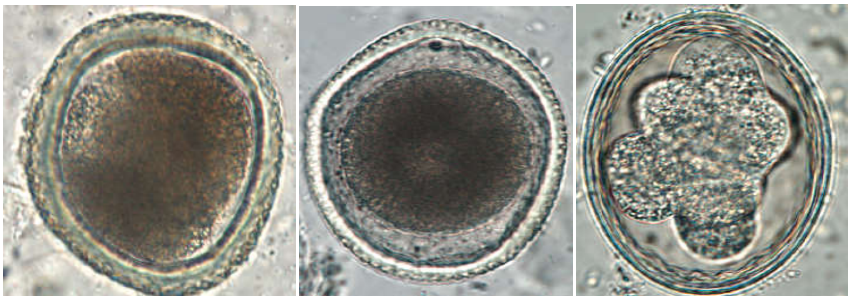

Figure 6. Eggs of*Toxocara canis* (left), *Toxocara cati* (middle) and *Toxascaris leonina* (right). (BOWMAN, 2013)

The *T.cati* cycle is similar. However, the infections of kittens occur through the ingestion of eggs containing L2.These larvae molt to L3 in the stomach through the hepatic tracheal migration, which reaches the intestine and completes development to adult form (TAYLOR et al., 2017). In adult cats, the possibility of tracheal migration is high, despite being even greater in kittens (EPE, 2009; CURY and LIMA, 2002). Due to the influence of hormonal changes in pregnant animals, which arise three weeks before whelping and at the beginning of breastfeeding, the larvae that made the somatic migration leave the latency period and return to the bloodstream (EPE, 2009). Thus, either in cats (BOWMAN et al., 2002) or in dogs, transmammary infection can happen, where the larvae reach the mammary gland through the bloodstream and pass into breast milk (EPE, 2009), in which they can be present for up to 45 days of lactation (TRAVERSA, 2012). It is important to note that in this type of infection, there is no migratory phase since the larva finishes its development in the intestine.Nevertheless, the infection can also occur through transplacental transmission, where the L3 larvaecross the placenta and reach the lungs of the fetus, then they pass through a molting cycle before the pup is born, and after birth, the larvae undergo hepatic tracheal migration to complete the cycle. The prenatal infection has a minimum prepatent period of two to three weeks (TAYLOR et al., 2017). However, this route of infections does not occur in cats (BOWMAN et al., 2002).

The mother is vulnerable to reinfection while nursing the puppies through the ingestion of shed eggs in the stool of the newborns (CURY and LIMA, 2002). Therefore, due to the mother's low immunity, the parasite can undergo a hepatic tracheal migration, and subsequently, the eggs are eliminated in the feces leading to environmental contamination (TAYLOR et al., 2017). The symptoms of transmammary infection are generally restricted to the gastrointestinal tract, characterized by diarrhea, pendulous abdomen, and it can also cause poor hair quality and flaws in the growth (TAYLOR et al., 2017). In addition, vomiting, obstruction, rupture bowel (RIBEIRO, 2004), intussusception, and abdominal distention may also occur. Severe infections through transplacental migration can lead to clinical signs during larval migrationdue to lung injuries such as coughing, increased respiratory rate, foamy nasal discharge, and pneumonia, and there may also be pulmonary edema (TRAVERSA, 2012). These parasites can cause pancreatic duct obstruction or biliary obstruction, impairing digestion, and absorption of nutrients. However, infections acquired by this route may result in death in the first days of puppies' life (TRAVERSA, 2012).

Ancylostomiasis: It is a hookworm disease associated with high neonatal mortality and morbidity (ROBERTSON and THOMPSON, 2002). Dogs are infected by *Ancylostoma caninum* and cats by *A. tubaeforme* (Figure 7). Dogs and cats can become infected by *A. braziliense*, which is the most zoonotic agent and the one that causes more chronic injuries (MUNDIM et al., 2004). These worms belong to the phylum Nemathelminthes, class Secernentea, and order Strongylida; they measure around 1cm to 2cm long and have a wide buccal capsule containing pairs of teeth used to attach the intestinal mucosa(RIBEIRO, 2004). Additionally, hookworms produce an anticoagulant enzyme localized in the esophageal gland that allows the continuous blood loss from where it is adhered, which persists evenafter the parasite leaves (CURY and LIMA, 2002). The eggs of Ancylostoma sp. (Figure 8) eliminated in the feces of the host hatch into L3 in less than a week under favorable conditions of temperature and humidity (TAYLOR et al., 2017). Hence, puppies can become infected through the penetration of L3 into the skin, which the larvae enter the bloodstream and migrate to the lungs where they develop to L4, and then undertake tracheal or somatic migration (ACHA and SZYFRES, 2003). Once the larvae undergo tracheal migration, they complete the final molting and maturation when they reach the small intestine (CURY and LIMA, 2002).On the other hand, if the larvae migrate through somatic tissues, they return to the circulation and reach the organs and the musculature, where these larvae remain dormant (RIBEIRO, 2004). Another route of infection is the ingestion of L3 larvae (TRAVERSA, 2012; EPE, 2009). Therefore, the larvae can reach the lungs after penetrating the oral mucosa or migrate to the small intestine, where they attainsexual maturity (TAYLOR et al., 2017). The prepatent period in cats infected through oral transmission is 18 to 28 days, while in cats infected through the penetration of L3 into the skin varies from 19 to 25 days. Even so, there is no evidence of transplacental or transmammary transmission of *A. tubaeforme* in these animals (BOWMAN et al., 2002).

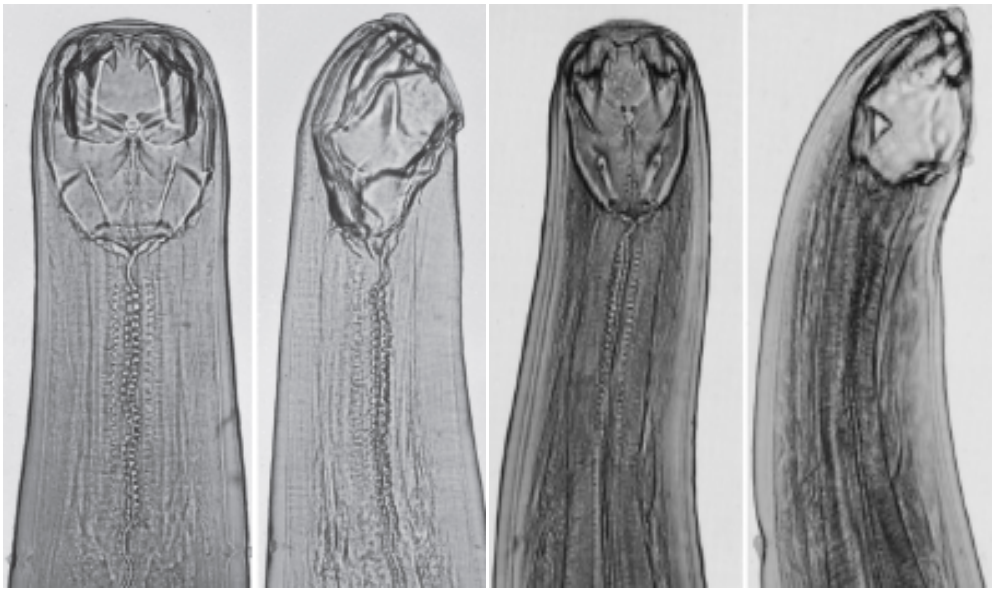

Figure 7. Dorsoventral and lateral view of *Ancylostoma caninum* (left) and *Ancylostoma tubaeforme* (right). (ZAJAC and CONBOY, 2021)

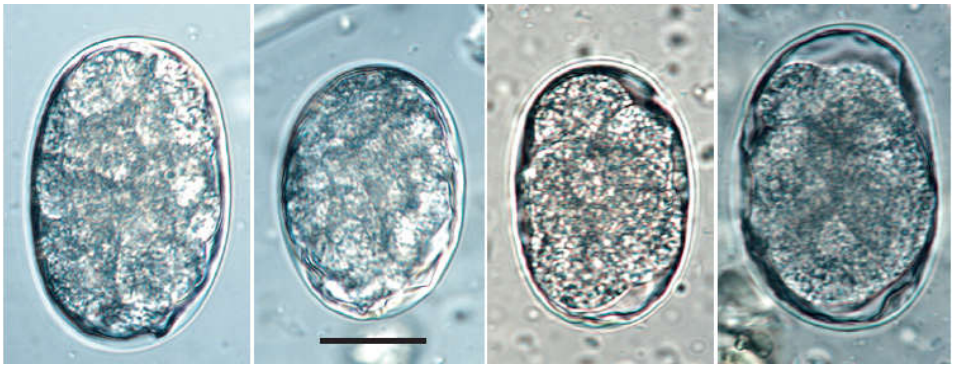

Figure 8. Egg morphology of *Ancylostoma* spp. (BOWMAN, 2013)

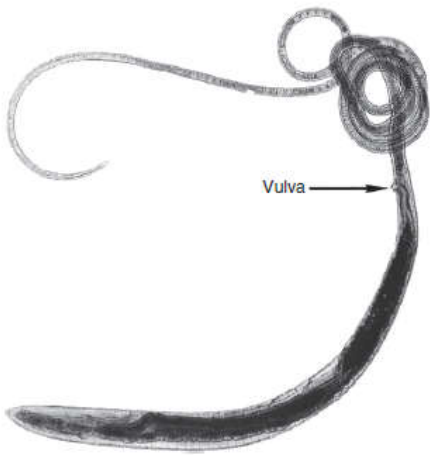

Figure 9. Female adult of *Trichuris vulpis*. (BOWMAN, 2013)

However, *A. caninum* can be transmitted by both pathways in neonatal dogs (BOWMAN, 2021), and the mother can eliminate larvae during breastfeeding for the next three lactations (BOWMAN, 2021; TAYLOR et al., 2017). The clinical signs depend on the virulence of *Ancylostoma sp.* and the host resistance, which*A. caninum* is the most pathogenic species (BOWMAN, 2021).Puppies are vulnerable to hookworm infection due to theiriron deficiency (TAYLOR et al., 2017),and 50 to 100 days larvae A. caninum may be fatal(BOWMAN, 2021). As a consequence of theblood-sucking habit of the parasite (ACHA and SZYFRES, 2003), the daily blood loss causes severe anemia, whichstarts around the eighth day after Infection (TAYLOR et al., 2017). The normocyticnormochromic anemia evolves gradually to hypochromic microcytic due to low iron reserves (RIBEIRO, 2004). Hence, the mucous membranes become pale, and there may also bediarrhea, in whichstool appears blackishsince the blood from the lesions in the small intestinedigested in the final section (BOWMAN, 2021). Nevertheless, respiratory signs may also occur due to the toxic effects of anemia (TAYLOR et al., 2017).

Trichuriasis: *Trichuris vulpis* is the species that causes trichuriasis, which is a large-intestine parasite that affects dogs and less frequently

in cats (EPE, 2009). They belong to phylum Nemathelminthes, class Adenophora, and order Enoplida. These parasites are also known as whipworms (JAZAC and CONBOY, 2012) due to their long and thin anterior end, which attaches to the intestinal mucosa while the posterior part of the body is thick and faces the lumen (Figure 9) (BOWMAN, 2021). Adult wormsin the large intestine release eggs (Figure 10) into the environment via feces. Therefore, under appropriate environmental conditions, the embryo takes upone to two months after excreted to develop inside the eggto the L1 infective stage (CURY and LIMA, 2002). They then remain viable in the environment for yearsdue to their high resistance (BOWMAN et al., 2002).

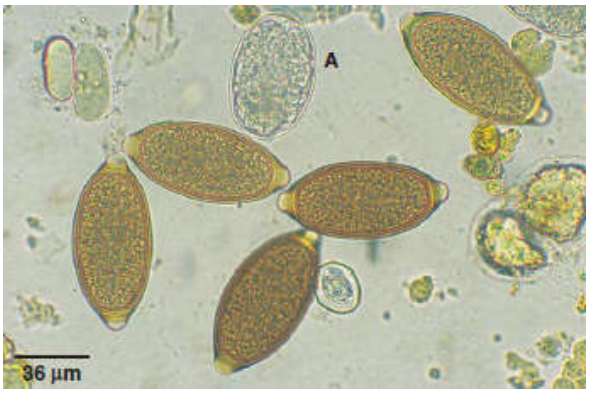

Figure 10. Eggs of *Trichuris* spp. pointed on the arrows, in a sample of faecal fluctuation with eggs of *Ancylostoma* spp. (A). (ZAJAC and CONBOY, 2021)

Puppies become infected after ingesting eggs containing L1, which hatches in the small intestine and releases the larva. These larvae penetrate the mucous glands of the distal ileum, cecum, and colon, where they become adults (TAYLOR et al., 2017). The prepatent period takes about three months (BOWMAN, 2021). Theclinical signs are related to the parasitic burden. Heavy infections may cause intestinal mucosal, abdominal distension, hematochezia (VASCONCELLOS et al., 2006), diarrhea containing mucus (BOWMAN, 2021), anemia, and weight loss (TAYLOR et al., 2017). Once acute hemorrhagic enteritis may occur, an infected animal can die (BIRCHARD and SHERDING, 2013). However, due to the prepatent period, theclinical signs may not ensue within less than three months (BARUTZKI and SCHAPER, 2011).

Tapeworms

Dipylidiasis: Dipylidiasis is a common intestinal tapeworm of dogs and cats, considered a low pathogenicity parasite (CURY and LIMA, 2002). This parasitic diseasecaused by*Dipylidium caninum*belongs to the phylum Platyhelminthes, order Cyclophyllidea, and class Cestoda (ZAJAC and CONBOY, 2021). They are flattened worms with a segmented bodyformed by proglottids (TAYLOR et al., 2017).

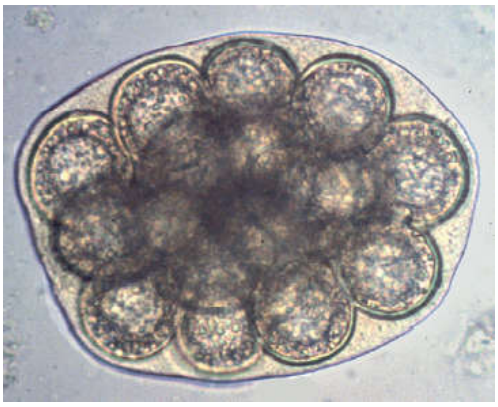

Figure 11. Ovigerous capsule containing oncospheres of *Dipylidium caninum*. (BOWMAN, 2013)

The adult tapeworms are hermaphroditic andattached to the small intestine of dogs and cats. Each proglottid contains a male and female reproductive organ, which releases gravid proglottids into the environment with the feces (BOWMAN et al., 2002).The terminal gravid proglottids contain ovigerous capsules (Figure 11), containing up to 30 embryonated eggs (TAYLOR et al., 2017). Once they are in the environment, the ovigerous capsules release the eggs, which are ingested by flea larvae (*Ctenocephalides* sp.) or, less often, by lice (*Trichodectes* sp., *Felicola* sp.) (TAYLOR et al., 2017). Adult fleas are unable to ingest the eggs due to the oral apparatus, which restricts them to a liquid diet (BOWMAN et al., 2002). When tapeworms reach theintermediate host digestive tract, the eggs hatch and release the oncosphere, which crosses the gastrointestinal wall and achieves the abdominal cavity where it develops intocysticercoid form (BOWMAN et al., 2002). The cysticercoid larvae become mature and infective when the louse or flea reach adulthood (MANI and MAGUIRE, 2009). The development in the louse takes about 30 days, while in the flea may take several months. However, the definitive host becomes infected by ingesting the insect, which upon reaching the stomach, they release the cysticercoid that reaches the adult stage in the small intestine.The neonates can get flea infestations if there are fleas on their mother by keeping proximity or they can ingest them during breastfeeding (TAYLOR et al., 2017). The proglottids are gradually released, which results in discomfort and anal itching. It can also cause gastrointestinal irritation, changes in appetite, and weight loss. In addition to intestinal obstruction, diarrhea or constipation can occur, yet it is rare. In severe cases, cats may have seizures (MANI and MAGUIRE, 2009; BOWMAN et al., 2002).

Diagnosis of parasitic gastroenteritis: The diagnosis of parasitic infections is based on the patient's clinical history and physical examination,such as vaccines, deworming, and hygiene conditions. Thus, the clinical signsmaysuggesta parasitic disease of the gastrointestinal tract and can emerge before eggs eliminated in the feces. However, some eggs can be easily observed in stool since some species are large. In tapeworm infection caused by *D. caninum* is possible to notice proglottids in the perianal region, detect the adult worms instool, and identify the adult nematodes (TAYLOR et al., 2017). The stool examination used to diagnose helminths is routinely utilized since it has a low cost, and the method is easier to execute. It is essential to keep the sample of stool refrigerated to prevent the eggs in the feces from becoming embryonic (TAYLOR et al., 2017). Therefore, among the methods available for the diagnosis of nematodes, cestodes, and protozoa, the direct fecal smear method and flotation techniques are commonly used (BOWMAN, 2021). The direct fecal smear technique requires a small number of feces mixed with water.The mixture is placed on a slide then a coverslip is placed to evaluate using an optical microscope (TAYLOR et al., 2017). Saline solutions can be used instead of water to prevents the trophozoites from degenerating (BOWMAN, 2021).However, this type of method detects infections with a highparasitic load due to the small number of feces used (TAYLOR et al., 2017), thus, negative results are inconclusive, requiring further tests (BOWMAN, 2021). The principle of the flotation technique is to use a solution that has a greater density than the density of helminth eggs and protozoan cysts so they can float. Usually, solutions of sodium chloride (NaCl) or magnesium sulfate (MgSO4) are used. Sucrose solution can also be used (BOWMAN, 2021) since they are efficient in the diagnosis of *Ancylostoma spp*. (TÁPARO et al., 2006; MANDARINOPEREIRA et al., 2010). If *Giardia*is suspected, solutions of zinc sulfate or formalin-ethyl acetate are indicated (TAYLOR et al., 2017).

Therefore, the saturated solution is homogenized with the feces, placed in a tube, which is filled to the surface with more saturated solution. Subsequently, a coverslip is placed on the surface of the liquid, which remains for 10 to 15 minutes (TAYLOR et al., 2017). When the sucrose solution is used, it is necessary to wait for 15 to 20 minutes (BOWMAN, 2021). Then the coverslip is removed and placed on a slide for viewing in the optical microscope. The prevalence of *Giardia* is underestimated due to the low sensitivity of the diagnostic methods since there are asymptomatic carriers, and the

elimination of cysts occurs intermittently (MCGLADE et al., 2003). Thereby, the samples are collected on three consecutive days to avoid false negatives (TAYLOR et al., 2017). The enzyme-linked immunosorbent assay (ELISA) is also a powerful method to perform in stool samples (SNAPtest) (TANGTRONSUP et al., 2010).

Treatment, control, and prevention of parasitic gastroenteritis in neonates : The control of parasites by protozoa is crucial to prevent possible reinfections or the infection of healthy animals. The environment cleaning and disinfection using quaternary ammonia, or 1% solution of sodium hypochlorite is essential to control Giardiasis and Isosporiasis (TAYLOR et al., 2017). Giardiasis in neonates is treated using a combination febantel-praziquantel-pyrantel product administered at doses of 37.8 mg/kg for dogs and 7.56 mg/kg for cats, both from four weeks old for five days (SCORZA et al., 2006). Fenbendazole at a dose of 50 mg/kg for three days is recommended for treating pregnant dogs (TAYLOR et al., 2017). The drug of choice to treat isosporiasis belongs to the sulfate group.Sulfadimethoxine is widely used, at a dose of 55 mg/kg on the first day and a half dose, 27.5 mg/kg, for four days or until clinical remission, not exceeding seven days of treatment (ALTREUTHER et al., 2011). Its use should be avoided in puppies younger than 20 weeks however drug dosage ought to be reducedif necessary (CRESPILHO et al., 2007). Good hygiene practices should be done to reduce the risk of roundworm infections. Infected animals must be isolated from the healthy ones and placed in clean places, the feces should be removed, and the floor washed with 1% sodium hypochlorite solution. In some cases, 0.5 kg of sodium borate per m^2 should be used (TAYLOR et al., 2017). Effective drugs used to treatment *Toxocara* spp. and *Ancylostoma* spp. infections are fenbendazole at a dose of 50 mg/kg for three days and a single dose for *Trichuris* sp.; mebendazole is also recommended at a dose of 22 mg/kg for three days for Toxocara spp. and Ancylostoma spp. and five days for Trichuris sp. Pyrantel pamoate is also effective in a single dose of 5 mg/kg although its use does not include puppies younger than 2 weeks old. The second dose of anthelmintic is recommended with an interval of 21 days after the first dose, which corresponds to the ripening time of nematodes (RIBEIRO, 2004). The most used drug on tapeworm infection is praziquantel in a single dose of 5 mg/kg (RIBEIRO, 2004). It is a safe drug for pregnant females, but it should not be used on puppies under four weeks old nor in cats less than six weeks old (BOWMAN, 2021). Therefore, it is important to clean and control the fleas and lice of any area that may be contaminated (RIBEIRO, 2004). Supportive therapies must be performed to treat and avoid the Neonatal Triad, such as enteral nutrition, fluid therapy, and body heating (SANCHES, 2017). Neonatal dogs and cats with a severe infection caused by*Ancylostoma* spp. need a blood transfusion, vitamin B12, and parenteral iron therapy (TAYLOR et al., 2017). In cases where the diagnosis of parasitic diseases is not possible, or the puppies are rescued and the mother is not known, or in kennels and catteries whose parasitic infestations occur more frequently, a deworming protocol is recommended. These animals must be dewormed with fenbendazole at 14, 28, 42, and 56 days old, and therefore, every 30 days until six months old (GRELLET et al., 2018).

CONCLUSION

The neonatal phase is a period of physiological adaptations and anatomical changes in the puppies' life. Errors in pediatric care can lead to death, contributing to an increase in the mortality rate of these animals in Veterinary Medicine. Therefore, knowing the major parasitic diseases that affect puppies, their diagnosis, and treatment promote reducing theneonatal mortality rate. Neonatal animals have greater sensitivity to drugs compared to adult animals. Thereby, they can manifest exacerbated adverse effectswith medication administration. The knowledge of neonatal characteristics is essential to an adequate therapeutic intervention to prevent further complications. In addition, it is crucial to be aware of the hygienic practices and the treatment of the puppy altogether to reduce the risk of reinfections and achieve a satisfactory therapeutic response. The deworming protocols work preventing worm infections, but it is essential to know the reason for its use to reduce the indiscriminate use of the antiparasitic drug. They prevent parasitic infestation by those parasites passed from mother to puppy and can be used in catteries and kennels, where parasitic infestations occur more frequently.

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