



Macroscopic Characteristics of Post Mortem Lesions in Opossums (*Didelphis Virginiana*) in Yucatan

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Abstract

Opossums are synanthropic marsupials that inhabit almost the entire American continent. These animals serve as reservoirs of zoonotic pathogens thanks to their evolutionary adaptation and eating habits. The aim of this study was to determine the etiologies of the diseases in these animals. In the present study, 13 opossums were necropsied in Yucatan, postmortem pathological changes were recorded on a data sheet, and photographs of each individual were captured. Most of the specimens studied presented intestinal pathological changes followed by hepatic and splenic, pulmonary, cardiac, renal and cutaneous alterations. Some pathological changes found are related to interaction in a human environment, while others are of infectious or parasitic origin with zoonotic potential.

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Introduction

The opossums of the genus *Didelphis* are omnivorous marsupial mammals that are characterized by their high degree of adaptation to various environments, which exposes them to different infectious agents. For the same reason, they actively participate as parasitic hosts in natural cycles, a fact supported by numerous publications in the biomedical scientific literature [1,2]. This favors their resistance to many pathogens, which is why they are considered a reservoir of some zoonotic agents, which may represent a risk to public health [3].

On the other hand, the synanthropic habits of American marsupials, mainly *D. virginiana*, make them prone to trauma caused by animals or humans. In addition to enduring fights between members of their own species, they are frequently attacked by domestic dogs. Most humans in Yucatan consider them a pest and seek to get rid of them cruelly and ruthlessly [4]. Undoubtedly one of the most common causes of injury and death are run overs on highways and urban and suburban streets. Likewise, their presence in agricultural and livestock farms exposes them directly to pesticide poisoning [5,6].



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There is little information that allows us to know the health status of *D. virginiana* in both jungle and synanthropic populations. Due to the importance of this species as a reservoir of zoonotic pathogens and its potential as a biomonitor of environmental contamination in Yucatan [7,8], we consider that is relevant to describe and analyze the lesions found in opossums as a diagnostic approximation of the diseases or affectations suffered by this species in its interaction with the human environment.

Material and Methods

Animals studied

The changes recorded in 13 necropsies of opossums (*D. virginiana*) in the period from 2000 to 2019 were studied. The necropsies were carried out at the Campus of Biological and Agricultural Sciences of the Autonomous University of Yucatan. Each animal was sacrificed just before its study with the ap-

proval of the ethics committee of said institution. The data was obtained from an individual record sheet with the most relevant information on each individual such as approximate age, size and date of capture, as well as the result of the necropsy and photographic evidence. At necropsy, external and internal changes and their direct, indirect or probable relationship with some etiologies were recorded.

Study region

All the specimens were found in Yucatan, in the towns of Molas, Komchén and Tetiz.

Results

The external and internal lesions found in the necropsy of each individual are described in Table 1. In the same way, the frequency of affection for each organ is found in Table 2.

Table 1: Main pathological changes present in the studied opossums. NAPC: no apparent pathological changes.

	Heart	Lungs	Liver	Spleen	Kidneys and adrenal glands	Digestive tube	Skin and mucosa
DV9-Mol	NAPC	Multifocal pleural nodulations.	Increased in size.	Increased in size.	NAPC	Severe diffuse and ulcerative multifocal catarrhal enteritis.	NAPC
DV28-Mol	Apparent area of scarring at the adjacent base of the right atrium.	Pneumonic foci and pleural nodulations.	Increased in size with yellow coloration.	Increased in size with white spotlights.	Renal cortex slightly enlarged.	Ulcerative catarrhal gastroenteritis and presence of nematodes.	NAPC
DV31-Mol	NAPC	Diaphragmatic lobe abscess.	Increased in size.	Increased in size.	NAPC	Diffuse catarrhal and ulcerative enteritis. Serosal nodules.	NAPC
Dch-Dv2	Hydropericardium.	NAPC	Increased in size. Irregular borders and jaundice. Ascites.	Increased in size.	Increased in size. Diffuse severe congestion and yellowing.	Diffuse catarrhal and ulcerative gastroenteritis. Presence of nematodes.	Jaundice.
DV15-Mol	NAPC	NAPC	NAPC	NAPC	Kidneys with apparent scars. Mild ascites.	NAPC	NAPC
DV18-Mol	Enlarged right ventricle with the appearance of an inverted "D".	Slightly edematous.	Congested and enlarged.	Slight increase in size.	There is enlargement of the adrenal glands.	Presence of nematodes.	NAPC
DV20-Mol	NAPC	NAPC	Increased in size, yellowish color and mild ascites.	Increased in size.	NAPC	NAPC	Jaundice
DV56	NAPC	Edema.	Yellow coloring.	NAPC	NAPC	Diffuse severe catarrhal gastroenteritis. Abundant nematodes.	NAPC
DV57	Enlarged, inverted "D" shape, pericarditis.	NAPC	NAPC	Enlarged, broken and scarred.	NAPC	Peritonitis. Ulcerative gastroenteritis. Presence of nematodes.	NAPC
DV61-Mol	No apparent pathological changes.	Edema and diffuse severe congestion.	Diffuse yellow coloration. Mild ascites.	Increased in size.	NAPC	Intestinal intussusception. Rectal prolapse. Presence of nematodes.	NAPC
DV129-Mol	NAPC	NAPC	Increased in size with yellow coloration.	Healing foci.	NAPC	Intestinal intussusception. Multifocal chronic nodular enteritis (parasitic nodules).	NAPC

DV17-Tet	NAPC	Edema.	Friable with rounded edges. Diffuse yellow coloration.	NAPC	Smaller left kidney with slightly irregular borders.	Presence of abundant nematodes and cestodes. Diffuse severe catarrhal enteritis.	Jaundice. Crusts in palmar and plantar regions. Enlarged mammary glands containing milk. Dental abscesses and missing pieces.
DV29-Kom	Focal suppurative endocarditis.	NAPC	NAPC	NAPC	NAPC	Multifocal ulcerative gastroenteritis with the presence of nematodes.	Ulcers on the back and nose. Pale mucous membranes and signs of severe dehydration.

Table 2: Frequency of organic involvement.

Affected organ	Frequency
Heart/pericardium	46.2%
Lungs/pleura	53.8%
Liver	69.2%
Spleen	69.2%
Kidneys	30.8%
Digestive tube	84.6%
Skin/mucosa	30.8%

Discussion

External and internal lesions share certain characteristics in the studied individuals, and that could be related to the etiology of their disease.

External injuries

Skin and mucosa

The main external lesions observed on the body of the animals were alopecic lesions, scars, nodular lesions and scabs on the back and fore and hind limbs.

These injuries can be due to multiple factors. They are free-living animals in rural environments, where they are exposed to wild predators and domestic animals, which can attack and cause injuries. Likewise, in their flight it is common for injuries to be caused by elements of the environment [3,5]. On the other hand, the weather conditions of the Yucatan Peninsula favor the development of ectoparasites such as fleas, ticks and mites, which also parasitize opossums and can cause skin lesions [9].

Some animals presented mucous membranes with signs of severe dehydration, while others presented jaundice, probably secondary to liver disease. Dental and gingival injuries result from trauma or due to biting on foreign objects [3,10].

Internal injuries

Heart

Various cardiac lesions were found in the present study, including cardiomegaly, inverted "D"-shaped enlargement (Figure 1), focal suppurative endocarditis, chronic pericarditis, hydropericardium, and scarred areas.

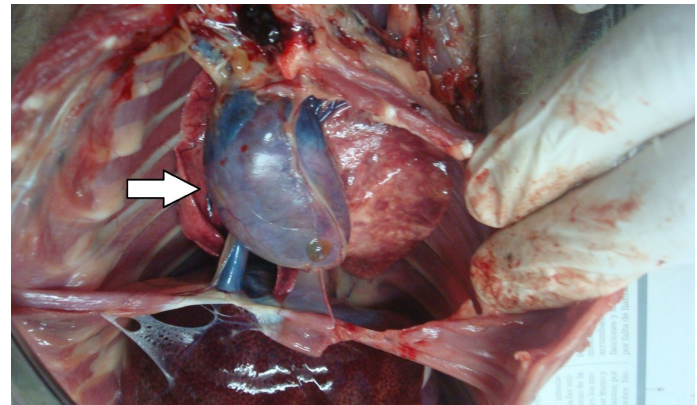


Figure 1: DV18-Mol individual: Thoracic cavity. Enlargement of the right side of the heart was observed (arrow).

Hydropericardium is caused by heart failure or hypoproteinemia, it also occurs in cases of renal failure and pulmonary hypertension. Endocarditis can be the result of a bacterial infection, although it can also occur with fungal infection and parasitic larval migration. Some pyogenic bacteria that affect the endocardium migrate to the myocardium, causing suppurative myocarditis [11]. In one study, Sherwood *et al.* observed myocarditis in 24 of 33 opossums that died in captivity [12]. While some individuals also presented thickened and grayish pericardium, others presented vegetative damage to the aortic or mitral valves. In all animals, acute and chronic focal aggregates of inflammatory cells were observed in the histopathological study.

On the other hand, *Toxoplasma gondii* infection could also be related to cardiac lesions in opossums, as has been shown in domestic animals. In Yucatan, a study by Torres-Castro *et al.* demonstrates the presence of the parasite in the brain of opossums [13]. The authors did not analyze heart samples. However, the cases of heart disease in the present work could be due to this parasite. More specific studies are required to clarify this question.

One opossum presented the heart with the appearance of an inverted "D" in the present investigation. This type of cardiomegaly has been related to diseases of the right ventricle or pulmonary artery in domestic animals such as heartworm disease, chronic obstructive pulmonary disease, or pulmonary thromboembolism. When it becomes chronic, it also affects organs such as the liver, kidneys, and spleen [11,14]. Pathogens such as *Trypanosoma cruzi* have been associated with cardiomegaly in opossums from southeastern Mexico [15,16]. Cardiomegaly has also been observed in opossums with *Mycobacterium* spp. [17].

Lungs

Several specimens presented nodulations in the pleura and lungs (Figure 2). Based on the studies carried out by García-Márquez *et al.* and López-Crespo *et al.* pulmonary lesions are compatible with lesions due to larval migration of parasites [18,19]. Similar lesions were found in opossums from Colima, which presented multifocal nodular granulomas in both lungs and were parasitized by *Paragonimus mexicanus*. On the other hand, Nettles, Prestowood and Davidson reported pleural multifocal nodular lesions in opossums and the presence of *Capillaria aerophila* eggs, although the lung parenchyma appeared to be intact [20]. Other authors have reported different causes for multifocal nodular lesions in the pleura and lungs, such as Rivero-Pérez *et al.*, who performed an isolation and identification of *Mycobacterium avium* from this type of lesions [17]. And Pope and Donell, who isolated *Pasteurella multocida* and *Bordetella bronchiseptica*, and reported lung adenocarcinoma as a spontaneous neoplasm in this species [21].

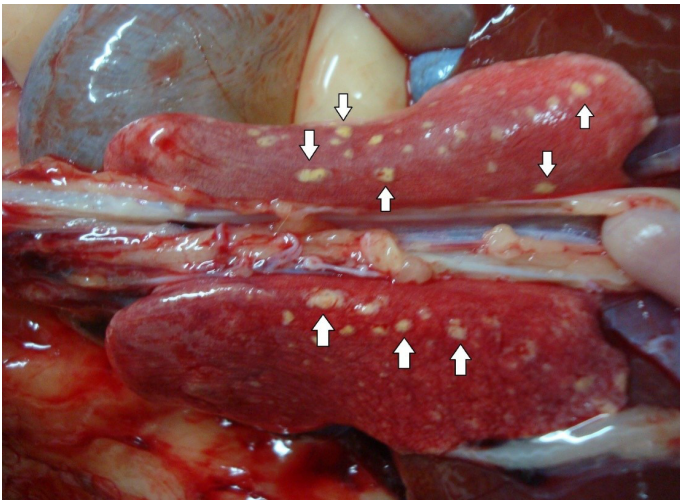


Figure 2: Individual DV9-Mol: Lungs. Light-colored nodulations were observed in the pleura (arrows).

Liver

In most of the animals, the liver was enlarged, in some with yellow coloration, rounded edges and friable consistency, which is characteristic of important conditions such as hepatic lipidosis (Figure 3). This may result from a metabolic state in which body fat stores are mobilized either by long periods of starvation (opossums tend to accumulate fat to survive these periods) or by obesity. In some studies, hepatic lipidosis has been associated with polyparasitism. Lamberski *et al.* studied 11 possums parasitized with *Didelphostrongylus hayesi*, 4 of which had pale and friable livers [22]. Likewise, Nettles, Prestowood and Davidson observed that the liver of animals with polyparasitism was pale yellow and with the presence of fibrotic tissue [20]. In the histopathological analysis, aggregates of histiocytes and neutrophils were observed in the portal areas.

Yellowing of the liver and generalized jaundice could be a consequence of elevated serum bilirubin, as occurs in some mammalian hemoparasite infections [14].

Spleen

Most of the animals in this study had an enlarged spleen, some with whitish foci (Figure 4). According to McGavin and Zachary, splenomegaly can be classified in various ways depending on the consistency [11]. Those that present a firm consistency could be due to macrophage hyperplasia in chronic

infectious processes, which occurs mainly in the red pulp due to the immune response directed towards pathogenic microorganisms or parasitized cells. Another cause of firm splenomegaly is lymphoid hyperplasia, due to an immune-mediated humoral response by B and T lymphocytes in the red and white pulp. Villagran *et al.* found splenomegaly in opossums naturally infected with *T. cruzi* [16]. On the other hand, Jubb, Kennedy, and Palmer mention that splenomegaly is also secondary to congestive heart failure [14].

A ruptured and scarred spleen was observed in one individual (Figure 5), these lesions are not the result of an internal pathological process but rather caused by trauma, such as blows or car accidents, and their severity depends on the magnitude or force of the impact that receive the organ, as well as the affected areas. If the damage is not lethal, the spleen heals [14].



Figure 3: Individual DV17-Tet. Yellow liver with apparent enlargement.

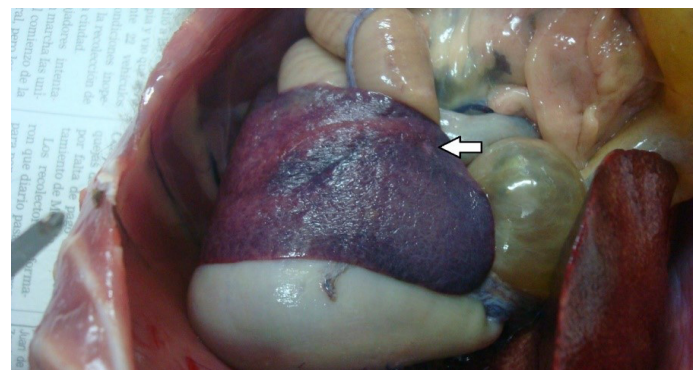


Figure 4: Individual DV28-Mol. Spleen with the presence of some light-colored foci (arrow).

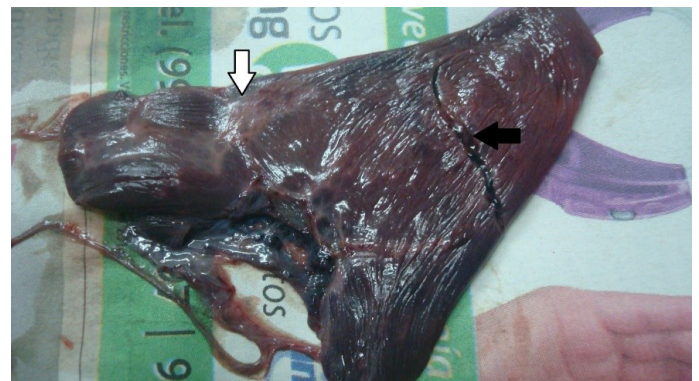


Figure 5: Individual DV57. Spleen with recent injury (black arrow) and scars (white arrow).

Kidneys

Compared with other organs, the kidneys did not show many pathological changes. In most of the individuals studied, a thickening of the cortex was found. In only one individual was the damage severe: both kidneys were found to be enlarged, with apparent medullary thickening and cortical congestion (Figure 6).

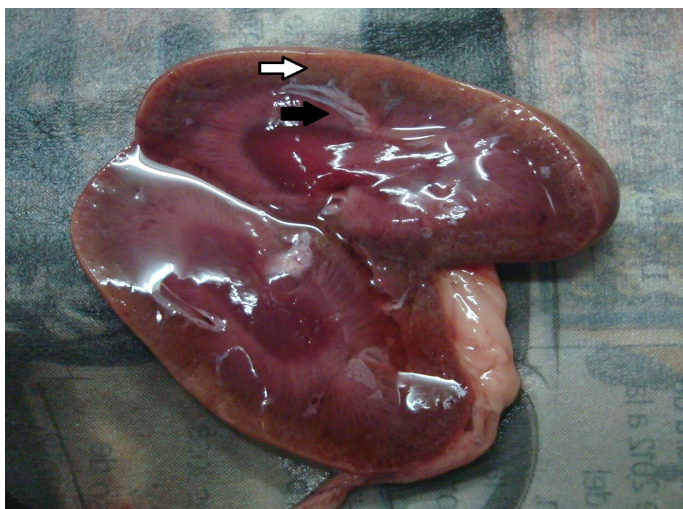


Figure 6: Dch-DV2 individual. Enlarged kidney. The renal cortex (white arrow) presented hemorrhagic lesions with a dotted appearance. The medulla appeared to be enlarged (black arrow).

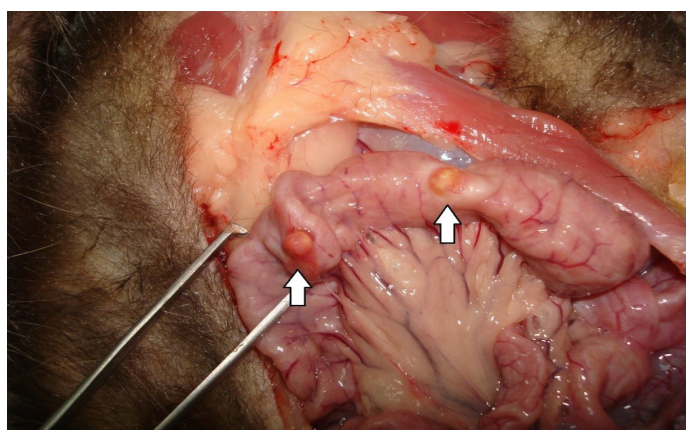


Figure 7: Individual DV31-Mol. Presence of nodules in intestinal serosa (arrows).



Figure 8: Individual DV61-Mol. Intestinal intussusception.

In the present study there is not enough evidence to link the renal pathological changes with a cause, since there were no complementary laboratory tests such as urine and blood tests of the animal to try to determine the type of nephropathy. However, congestive heart failure is related to acute renal failure due to decreased renal perfusion, characterized by increased organ size and red or brown coloration, chronically evolving to ischemia and necrotic foci [11].

Among the finds found by Nettles, Prestwood and Davidson in opossums with polyparasitism, the dark cortical zone is described, which in histopathology was attributed to bile pigment in the renal tubules [20]. On the other hand, in the histopathological analyzes carried out by Sherwood *et al.* in 33 opossums (*D. virginiana*) glomerulonephritis, interstitial nephritis and renal infarction were observed. Most of the animals in that study also had heart disease [12].

Digestive tube

Numerous and important pathological changes were found at this site, such as ulcerative and nodular inflammation in the gastrointestinal mucosa and serosa (Figure 7), intussusception and rectal prolapse (Figure 8) and intestinal polyparasitism (Figure 9).



Figure 9: Individual DV17-Tet. Presence of abundant nematodes in the small intestine.

The causes of mechanical injuries such as intestinal intussusception and rectal prolapse are generally unknown. However, intussusception may be associated with intestinal irritability and hypermotility due to inflammation from foreign objects or intestinal parasites. And rectal prolapse may be due to rectal obstruction or urinary tract infection that cause prolonged periods of tenesmus. The everted part of the organ becomes swollen and congested, followed by signs of ischemia, ulceration, and necrosis [14].

The parasites found in the gastrointestinal tract were not taxonomically identified, but much of the tissue damage is attributed to them, since there are a large number of genera and species of parasites that infect *D. virginiana*, mainly in tropical countries such as Brazil and Colombia. being a potential risk to public health [6]. For their part, Aragón-Pech *et al.* evaluated the frequency, abundance and intensity of eggs and oocysts of gastrointestinal parasites in opossums from Yucatan and found evidence of 9 different genera [23]. However, an exhaustive study for the identification of the species was not carried out; among these are *Eimeria* sp., *Trichuris* sp., *Capillaria* sp., *Ancylostoma* sp., *Cruzia* sp., *Ascaris* sp., *Toxocara* sp., *Turgida* sp., And *Oligacanthorhynchus* sp., Likewise, in a study in Mexico by Acosta *et al.* identify different parasites in opossums that affect the gastrointestinal tract: *Brachylaima didelphis*, *Rhopalias* sp.,

Mathevotaenia sp., *Thaumasioscolex didelphis*, *Oligacanthorhynchus microcephalus*, *Oncicola luehei*, *Porrorchis nickoli*, *Aspidodera railletii*, *Cruzia tentaculata*, *Ganathostoma turgidum*, *Gongylonema* sp., *Turgida turgida*, *Trichuris didelphis*, *Viannaia viannai* and *Travassostrongylus* sp [24].

In Yucatan, there are few studies that characterize lesions in the gastrointestinal tract of *D. virginiana* due to helminths. This is because opossums are hosts to a wide variety of parasites, making it difficult to make a pathological description for each genus that infects this species. However, based on the aforementioned studies it is possible to compare the lesions found in these opossums with lesions in other mammalian species.

Nearly 1000 species of the genus *Eimeria* sp. are known, some being more virulent than others. In pigs it has been observed that the macroscopic lesions characteristic of this parasite result in a diffuse acute catarrhal enteritis, which evolves into a necrotizing fibrinous enteritis with hemorrhagic areas; the most affected intestinal sections are the jejunum and ileum, but the cecum and the colon can also be compromised [25].

Trichuris sp. It is a parasite that adheres to the mucosa of the intestine, mainly the cecum and the colon of the host; In pigs, a hemorrhagic mucofibrinous enteritis can be seen macroscopically, which can be focal or diffuse; the intestinal wall thickens due to the presence of edema; around the site of adhesion of the parasites there is formation of inflammatory nodules, frequently purulent; and in the visceral wall it is possible to notice focal peritonitis. On the other hand, in dogs chronic catarrhal colitis is observed with hyperemia, coagulative necrosis, mucosal congestion and edema. However, these lesions are evident when the degree of infestation is severe [25].

Ancylostoma spp. They not only parasitize the digestive tract, they are also characterized by their ability to migrate to other organs; Ulcerative hemorrhagic enteritis can be observed in the gastrointestinal tract of dogs, due to the adhesion of the nematode to the mucosa. In addition, ascites can be seen in the abdominal cavity [25].

Cruzia spp. They parasitize a wide variety of marsupials, reptiles, and amphibians. Information on histopathological characteristics of the host is scarce. Mollericon and Nallar identified the parasite in the marsupial *Didelphis pernigra* without finding significant pathological lesions, although they mention that it can cause typhlitis [26].

On the other hand, ascariidosis (Order Ascaridida) are of great importance in domestic animals. The nematode is characterized by migrating to the liver, although it can also be found in the lungs and heart. According to Cordero del Campillo *et al.*, the lesions in the intestine are minor compared to those of other organs, which is why petechiae, cellular infiltration, submucosal edema, mild hyperemia, enteritis, erosions can be found, and calcified granulomas can form encapsulating erratic larvae [25]. The lesions caused by this parasite in dogs and cats depend on the parasite load, but moderate to severe catarrhal enteritis, obstruction, and intestinal perforations have been observed. Parasites belonging to the Ascaridiidae and Toxocaridae families are present in *D. virginiana* [23].

Turgida sp. in opossums around the world, mainly from *T. turgida*. Gray and Anderson studied these parasites in opossums in Florida, and determined that the degree of lesions depended on the time of infection [27]. Parasitosis less than 80 days old had localized ulcers less than 1 mm in diameter in the

anchorage areas of the gastric fundus, in infections older than 100 days there were 2-10 mm ulcers associated with groups of up to 12 parasites. In individuals with an infection of more than 100 days, the mucosa was destroyed and the submucosa was infiltrated with granulation tissue, the ulcers extended to the submucosa and in some animals the tissue was necrotic with the presence of fibrinous exudate on the surface of the serosa [27].

Another parasite found in opossums is *Oligacanthorhynchus* spp., an acanthocephalan of which a large number of species have been described. In one study, two opossums were experimentally infected with *O. tortuosa*. At 42 and 48 days post infection, the parasites occupied 60% of the small intestine and caused granulomatous lesions at the anchorage points [28]. For their part, Gallas and Silveira found another species of *Oligacanthorhynchus* spp. in a wild feline in Brazil, it presented macroscopic lesions in the adhesion sites in the intestinal lumen described as ulcerative nodular enteritis, in which the ulcers were smaller than 1 mm and the nodules present in the serous layer measured 3.5 to 6 mm in diameter [29].

Many individuals presented some degree of parasitism or polyparasitism in the present work, for which reason the lesions found could be associated with one of the parasites already described, or with other infectious pathologies. Nettles, Prestwood and Davidson studied opossums infected with multiple species of parasites, finding different stages of helminths in the stomach that caused hemorrhagic ulcerative gastritis with necrotic foci in the mucosa and submucosa. In addition, there were different parasites in the intestine and darkened contents, indicative of hemorrhagic areas [20].

Finally, in several animals ascites was found. This is most likely the result of two important conditions: the first as a consequence of chronic congestive heart failure. And the second due to hypoproteinemia, possibly caused by the presence of parasites in the gastrointestinal system [11]. This is consistent with the findings found by Nettles, Prestwood and Davidson in the abdominal cavity of opossums with polyparasitism [20].

Conclusion

The species *Didelphis virginiana* is an animal that is found in free life, which is why it is a carrier of various agents: viruses, bacteria, fungi and parasites; many of which have zoonotic potential and pose a risk to public health. Most of the injuries found in this study can be attributed to these pathogens or to events related to the environment, such as polytrauma from being run over. However, generalized descriptions were made, so it is necessary to carry out similar studies including complementary clinical laboratory and histopathology tests to narrow down the description of the lesions at the tissue level, as well as the taxonomic classification, morphological characterization and genomic sequencing of the agents found.

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