Popular Article

Strobilocercus fasciolaris: A common menace in laboratory-maintained rodents

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Abstract

Laboratory animals are often infected with infectious agents that hamper their creditability to be used as experimental models. Strobilocercus fasciolaris, the larval stage of Taenia taeniaeformisis the most common parasitic entity affecting rats and similar rodent laboratory animals. The larval stage affects the liver and induces a chain of immunological alterations in the affected animal. The parasite often causes alterations in the experimental results and a very big nuisance in following scientific protocols. Its pathobiology, impact on host and other associated attributes are described herewith in the present communication.

Introduction

Laboratory animals, particularly rodents such as rabbits, guinea pigs, mice, and rats, are widely used as models to study the pathobiology of various microbial and toxicological agents. However, parasitic infections can substantially influence the results of these experimental studies. These infections change the physiological and immunological characteristics of the hosts, affecting their response to experimental stress and potentially distorting the research outcomes.

Taenia taeniaeformis: Description and Life cycle

Taenia taeniaeformis, a tapeworm of the order Cyclophyllidea, primarily infects wild and domestic carnivores, which act as its definitive hosts. This parasite resides in the small intestines of these carnivores. Rodents, serving as intermediate hosts, ingest gravid proglottids shed in the feces of the definitive hosts. The parasite's oncosphere passes through the rodent's stomach and is released in the small intestine. It then penetrates the intestinal wall and travels via the portal circulation to the liver, lodging in the hepatic capillaries. About six days post-infection, the parasite is found within small vesicles formed by the proliferation of host connective tissue on the liver surface, where it develops into the infective larval stage known as strobilocercus. The larval or metacestode form was previously referred to as Cysticercus fasciolaris. As the cysticercus develops into a strobilocercus in the intermediate host, the parasite is also known as Strobilocercus fasciolaris.

After a maturation period of 6-8 months, the developmental stages become fully grown, infective strobilocerci with caudal vesicles. Felines and other carnivorous definitive hosts become infected by consuming the livers of rats that

harbor the larval stage. This parasite is widespread, with reports from various regions including India, Japan, Egypt, and Mexico.

Pathobiology

The pathology of strobilocercus-induced liver disease in rodents begins with the ingestion of T. taeniaeformis eggs, which develop into larvae that form cysts in the liver parenchyma of rats. This leads to progressive chronic inflammation and vigorous fibroplasia. The strobilocercus lodges in the liver parenchyma, causing physiological and immunological changes, tissue damage, abnormal growth, nutrient competition, and deviations in animal health and research outcomes. Additionally, fibroplastic and inflammatory lesions may progress to fibrosarcoma. There have been reports of spontaneous occurrences of Taenid cyst-associated hepatic mesenchymal tumors with rare systemic metastasis and gastroenteropathy. The parasite also poses a zoonotic risk to animal house workers, as humans can become accidental hosts, with occasional reports of the parasite in the intestines and liver of infected individuals. Experimental studies have revealed a relationship between the implantation of developmental stages and the development of liver tumors.

Rodents infected with T. taeniaeformis exhibited signs of weight loss, lethargy, and mild diarrhea before succumbing. Similar clinical signs of gastroenteropathy have been previously documented. Hepatic tumors induced by T. taeniaeformis led to nonspecific clinical signs such as lethargy, weight loss, anorexia, and sudden death. Infected rats displayed nonspecific clinicopathological changes that could significantly impact the outcomes of experimental studies. These changes may include elevated liver-specific enzyme activity, decreased glucose concentration, and increased counts of neutrophils, lymphocytes, and eosinophils in peripheral blood smears.

Typically, rats up to 4 months of age are predominantly infected. Hanes (1995) observed that rats infected for less than one month usually had few immature cysts without developed scolices, affecting only 1-2 hepatic lobes. Conversely, rats infected for more than one month tended to have a higher number of cysts, affecting the liver parenchyma more severely and involving a greater number of hepatic lobes. Morphologically, the scolex consistently displayed two rows of characteristic penknife-shaped hooks with an equal number of large and small hooks in all cases. However, some researchers have reported 30–40 hooks in the rostellum of the metacestode. Microscopic examination of affected liver sections reveals fatty, inflammatory, metaplastic changes in some areas, along with proliferative fibrous tissue changes in the liver parenchyma. The infiltration of mononuclear cells in the hepatic parenchyma surrounding the cyst observed in this study aligns with previous reports. Histopathological findings indicated fibrosarcomatous changes characterized by spindle-shaped pleomorphic neoplastic cells in the liver parenchyma. Similar lesions have been reported in Sprague-Dawley rats. Chronic infestation of larvae in the liver parenchyma induces chronic irritation and inflammatory changes, which can progress to metaplasia and ultimately neoplasia. The specific pathogenesis of neoplastic changes in the liver is not fully understood, but a causal association of T. taeniaeformis with hepatic tumors is well

established. The causal hypothesis suggests the involvement of the parasite, larval-derived oncogenic substances, chronic inflammation, irritation of liver parenchyma due to the presence of larval cysts, oxidative damage, and immunosuppression caused by the adult parasite and larvae themselves. Histopathological changes in the gastrointestinal tract (GIT) include hyperplasia of mucosal glands and hypertrophy of the mucosa. Infected rats may develop lesions of inhibited gastric acid secretion, hypergastrinemia, and gastric and intestinal mucosal hyperplasia, attributed to larval secretions in the gastric and intestinal lumen. Infection of rats with strobilocercus reduces both male and female fertility, with the decrease in male reproductive parameters attributed to reduced testosterone production.

Diagnosis

Diagnosis is based upon the liver examination and demonstration of cysts of strobilocercus on liver.

Treatment and control

The benzimidazole group of anthelmintics, including albendazole and fenbendazole, has shown significant effectiveness against strobilocercus infections. Additionally, rats that have become ineffective for clinical trials should be culled. Measures should be taken to prevent the spread of infection within the colony of rodents by limiting exposure of carnivores to rat sheds or animal housing facilities. It's important to avoid exposing rodent feed storage units to cats and dogs, as their excreta can contaminate the feed and spread the infection among rodents.