Helicobacter mustelae

by Dr. Bruce Williams

Here's an excerpt from my contribution to the lecture notes from the 8th Annual Small Mammal Conference put on by the American Ferret Association in Baltimore, where I had the opportunity to lecture to 120+ vets on GI diseases....

....This bacterium, first described in 1990, has rapidly become the most important disease-causing agent of the pet ferret. H. mustelae has the ability to cause two distinct forms of gastric disease - peptic ulcer disease (presumptive) and chronic atrophic gastritis.

Helicobacter mustelae is extremely widespread in the pet ferret population. Several studies using random source ferrets have shown that almost every ferret carries this bacterium. As the bacterium is passed by a fecal-oral route, kits generally are infected by the mother within the first two weeks of life.

H. mustelae appears to be able to cause two distinct syndromes in the stomach of affected ferrets - chronic atrophic gastritis and peptic ulcer disease. Chronic atrophic gastritis is a common finding in ferrets over four years of age. In these animals, the bacterium causes gastritis via two mechanisms - a) the stimulation of a marked lymphoplasmacytic inflammatory response, resulting in loss of glandular epithelium most prominently in the pylorus, and b) the ability to increase the pH of the stomach. While clinical signs and disease progression may vary markedly between individuals, the progressive damage to the gastric mucosa most commonly results in clinical signs in animals of four years or more.

Additionally, although a definitive cause-and-effect relationship has not yet been conclusively proven between the presence of Helicobacter mustelae and gastric ulcers, evidence connecting the two is beginning to mount. Cytotoxins liberated by several species of Helicobacter have the ability to directly injure gastric mucosal epithelial cells. Additionally, recent evidence has shown that ferrets infected with H. mustelae have elevated levels of plasma gastrin at 30 and 60 minutes following feeding. For these reasons, any treatment of gastric ulcers in ferrets should be combined with concomitant antimicrobial therapy for Helicobacter mustelae.

Treatment for gastric helicobacteriosis should be strongly considered in any ferret with vague gastrointestinal signs including inappetance, loose stools, or intermittent vomiting.

Gross lesions:

Endoscopy or gross necropsy is generally unrewarding in cases of chronic atrophic gastritis. Surgical biopsy of the pyloric region of the stomach is highly recommended for definitive diagnosis of H. mustelae infection. The gross aspects of gastric ulcers have been previously described (above). H. mustelae infection is also the most common cause of mesenteric lymph node enlargement in ferrets, due to the profound inflammatory response which it initiates in the pyloric stomach.

Microscopic lesions:

Silver stains are the stain of choice to demonstrate the presence of the bacteria in the superficial mucus and in extracellular locations within the gastric glands. The pyloric stomach is the preferred biopsy site, although low numbers of bacilli may also be seen in the fundus and duodenum in severely infected animals. In affected animals, varying degrees of lymphoplasmacytic gastritis and loss of gastric glands may be seen, with the most severe damage occurring in the pylorus.

Treatment

There are numerous treatments for H. mustelae in ferrets, most of which have been derived from the treatment of H. pylori, a common bacterium in man, which has been definitively linked to the development of gastric ulcers in man.

The most commonly accepted treatment is a combination of amoxicillin at 10-20 mg/kg twice daily, metronidazole (Flagyl) at 30 mg/kg once daily, and Pepto-Bismol (1/15th of a tablet once daily). This "triple therapy" has been shown to be effective in man, and to a large part effective in ferrets, but must be continued for 4-6 weeks. Unfortunately, ferrets CANT STAND the taste of both Flagyl and Pepto-Bismol, and client compliance with therapy is often a problem.

A recently published protocol for treating ferrets is a combination of Clarithromycin (Biaxin) at 50 mg/kg once daily, and amoxicillin at 35 mg/kg once daily, or 20 mg/kg twice daily. This therapy is only continued for two weeks, and supposedly has great efficacy and causes minimal resistance in the organisms.

Other antibiotics, such as chloramphenicol, Baytril, gentamicin, or sulfa drugs such as DiTrim are of no use in this condition.

Finally, it has been asked whether all ferrets in a household need to be treated. Because Helicobacter is shed in the feces of infected animals, it is very easy for cleared animals to become reinfected. In large facilities, treating all animals, especially with Biaxin is cost prohibitive. In most cases, temporary respite from the ravages of Helicobacter can result in marked clinical improvement in most animals. While you may clear some animals, the majority are often reinfected, or simply maintain the infection at a low level. My recommendations would be to treat only the animals showing clinical signs.

Additional references:

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