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Inducing Diabetic Remission in Cats

This clinical review compiled information from human and veterinary studies regarding newer long-acting insulin in cats with diabetes mellitus. Diabetes mellitus affects up to 1.24% of cats; Maine coon, Russian blue, and Siamese breeds are over-represented in the U.S. Most diabetic cats suffer from type-2 diabetes, which includes insulin resistance and a progressive reduction in insulin production resulting from β -cell dysfunction. Resolution of glucose toxicity because of tight glycemic control in newly diagnosed diabetic cats is thought to enhance probability of remission. Remission is likely with the use of newer, long-acting insulin such as glargine and detemir, low-carbohydrate diet, rapid initiation of therapy, intensive in-hospital or at-home glucose monitoring, and adjustment of insulin dose to achieve normal or near-normal blood glucose concentration. Clinical studies suggest twice-daily glargine administration is more likely to increase the probability of remission than once-daily glargine,

twice-daily PZI, and twice-daily porcine lente insulin, especially when used in newly diagnosed diabetic cats. Detemir remission rates were also promising in newly diagnosed diabetic cats fed a low-carbohydrate diet. When using long-acting insulin, the goal is to achieve normal or near-normal blood glucose with a nadir of 65–117 mg/dL at home or 72–160 mg/dL in the hospital. Although data are limited, studies suggest that remission is achievable in 80%–90% of diabetic cats when rapid and intensive blood glucose control is achieved using detemir or glargine.

■ Global Commentary

Cats are not small dogs. Never has this statement been more accurate than when addressing diabetes. Most cats suffer from type-2 diabetes in contrast to dogs, which suffer from type-1 or type-3 diabetes.

Unlike dogs, many cats go into remission.

Time to remission is variable and likely depends on how long the hyperglycemia

and glucose toxicity have been present and whether there are remaining functional β -cells in the pancreatic islets. In most cats, remission occurs in the first few months after the start of treatment, but in some cases it can take longer; for example, one of my cases involved a cat that had been diabetic for 18 months before suddenly and unexpectedly going into remission.

Although many diabetic cats go into clinical remission and seem to remain in remission, it is important to remember that remission does not necessarily mean cure. Care with diet, exercise, and avoidance of aggravating factors (eg, progesterone, progestogens, glucocorticoids, obesity) are important in retaining disease-free status.—*Michael E. Herrtage, MA, DVSc, DVR, DVD, DSAM, DECVIM, DECVDI, MRCVS*

■ ■ Source

Feline diabetes mellitus: Clinical use of long-acting glargine and detemir. Bloom CA, Rand J. *J FELINE MED SURG* 16:205-215, 2014.

Redefining Hemorrhagic Gastroenteritis

Hemorrhagic gastroenteritis (HGE) syndrome in dogs is characterized by the acute onset of bloody diarrhea and vomiting. Histopathology performed at necropsy has shown superficial mucosal hemorrhagic necrosis in the intestines of dogs with HGE. While the syndrome's exact cause is unknown, *Clostridium perfringens* has been a previously associated pathogen. Previous histologic and microbiologic studies of dogs with HGE have only evaluated postmortem GI tissues. This prospective study was the first to perform intra vitam histologic and microbiologic evaluations in dogs with HGE.

Ten dogs with HGE and 11 dogs with non-HGE intestinal disease were included. Endoscopic biopsies were taken from the stomach, duodenum, ileum, and colon and examined for endoscopic and histologic changes, and the first duodenal biopsy sample obtained with sterile forceps was submitted for bacterial culture. Lesions of the mucosa were only identified in the intestines and not in the stomach of dogs with HGE. In addition, all of the dogs with HGE had *Clostridium* spp identified by culture or histopathology in

the small intestine. There is an association between *C perfringens* and acute hemorrhagic diarrhea, and it was recommended that HGE be renamed to *acute hemorrhagic diarrhea syndrome*, as the study found no evidence of gastritis in dogs with HGE.

■ Commentary

This study's results were somewhat surprising in that the stomach does not appear to be primarily involved despite the common clinical sign of vomiting in many of these patients. Also, the major histologic finding within the small and large intestines involves necrosis rather than inflammation, thereby invalidating the nomenclature of the disease. Finally, the suspicion of *Clostridium* spp factoring into the pathogenesis supports the need for appropriate therapeutic antibiotic coverage in these cases.—*Dara Zerrenner, MS, VMD, DACVIM*

■ ■ Source

Endoscopically visualized lesions, histologic findings, and bacterial invasion in the gastrointestinal mucosa of dogs with acute hemorrhagic diarrhea syndrome. Unterer S, Busch K, Leipig M, et al. *JVIM* 28:52-58, 2014.