CASE REPORT

Pyloric Stenosis in a Foal

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Summary

A two month old Thoroughbred filly was presented with signs of depression, grinding of the teeth, frothing of the mouth and abdominal pain. These signs had persisted for two weeks despite treatment with mineral oil, dioctyl sodium sulfosuccinate, meperidine and antibiotics. A variety of diagnostic tests were done, the only abnormal finding was a stress leukon. On exploratory laparotomy the stomach was dilated with fluid and gas and the pyloric canal was constricted. Pyloroplasty resulted in correction of the condition.

The etiological possibilities are discussed. This is believed to be the first report of pyloric stenosis in the horse.

Résumé

Sténose pylorique chez une pouliche

Une pouliche Thoroughbred, âgée de deux mois, manifestait les signes cliniques suivants: dépression, grincements de dents, présence de spumosité à la bouche et douleur abdominale. Ces signes duraient depuis deux semaines, en dépit d'un traitement à base d'huile minérale, de dioctylesulfosuccinate de sodium, de mépéridine et d'antibiotiques. On effectua plusieurs épreuves de laboratoire, en vue de poser un diagnostic, mais la seule anormalité consistait en une formule sanguine de stress. Une laparotomie exploratrice révéla une dilatation stomacale, liquide et gazeuse, ainsi qu'une sténose pylorique. Une pyloroplastie permit de corriger l'anomalie. Les auteurs commentent les causes possibles de cette anomalie. Pour eux, il s'agirait du premier rapport d'une sténose pylorique équine.

Introduction

Pyloric stenosis occurs commonly in man and the dog and can be categorized into three etiological groups:

a) congenital hypertrophic pyloric stenosis (CHPS),
b) acquired pyloric stenosis due to gastritis and
c) acquired pyloric stenosis due to neurogenic pylorospasm.

The incidence of CHPS in infants is about 0.25% (17, 26) and the mean age at onset of signs is five and one-half weeks (13, 35). The incidence in domestic animals has not been established. It is seen commonly in puppies (9), rarely in kittens (34, 41) and has not been recorded in any other domestic species. In puppies and kittens the onset of signs occurs at weaning (1, 9).

Acquired pyloric stenosis due to gastritis is common in adult humans (28, 32) and was reported as early as 1828 (36). The usual etiology is the ingestion of corrosive substances which often cause a progressive fibrotic stenosis in three to four weeks (14, 15, 19, 32, 36, 38). Prolonged pylorospasm, secondary to chronic gastritis or gastric ulceration, may result in hypertrophic pyloric stenosis (18). Prepyloric carcinoma and postpyloric ulceration may result in constriction which mimic pyloric stenosis (27). Pyloric stenosis following the ingestion of corrosive agents has not been reported in animals, but hypertrophic pyloric stenosis secondary to chronic gastritis and gastric ulceration, has been reported in dogs (1, 9) and rabbits (25).

Pylorospasm and reticulo-omasal achalasia occur in cattle (4) and sheep (29) due to vagus nerve dysfunction. Pylorospasm leading to hypertrophic pyloric stenosis is reported in the dog (1) and cat (31). Dogs with a nervous disposition, particularly toy and miniature breeds, are most commonly involved. A neurogenic dysfunction, centrally or locally mediated, is suspected (9).

The clinical signs of pyloric stenosis in small monogastric animals and humans are remarkably similar and uniform. Vomition, which becomes progressively more projectile during or after each meal, is the main sign. Abdominal pain after feeding, weight loss or unthriftiness, dehydration and electrolyte imbalance are also common signs. A palpable pyloric "tumor", usually reported in humans, has not been found in animals.

This report describes a case of pyloric stenosis in a foal. There are no previous reports of this condition in the equine species.

History

A two month old Thoroughbred filly was presented to the Large Animal Clinic due to persistent grinding of the teeth, frothing at the mouth and chronic abdominal pain. At one month of age, she developed severe diarrhea, following transportation to a breeding farm. The diarrhea disappeared in a few days but at ten days, she began grinding her teeth frequently. Periodically, she would cast herself on her back and lie still for five to ten minutes before assuming lateral recumbency or rising to her feet. Poor response to treatment with mineral oil, dioctyl sodium sulfosuccinate, meperidine and long-acting penicillin accounts for her referral to the Western College of Veterinary Medicine.

Clinical Findings

At presentation, the filly was moderately depressed, but her temperature, pulse and respiratory rate were normal and her physical condition was good. She ground her teeth continuously, working the saliva into a froth which appeared at the lips and nostrils. She displayed signs of abdominal pain by lying on her back. Occasionally small amounts of gas were eructated. She ate insignificant quantities of feed

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but nursed every four to five hours and also drank water. Only very small amounts of normal feces were passed infrequently.

Passage of a stomach tube, endoscopy and contrast radiography of the esophagus, abodominal paracentesis and fecal flotation failed to reveal any abnormality of the digestive tract. Concentrations of blood lead, blood urea nitrogen and serum glutamic oxalacetic transaminase were normal. A complete blood count indicated stress. Radiographic contrast studies of the abdomen were impossible with the available equipment. A partial obstruction of the upper bowel was suspected and the filly was prepared for an exploratory laparotomy.

Surgery

Anaesthesia was induced by the administration of halothane[1] through a mask and maintained by additional administration through an endotracheal tube. When fluid started refluxing from the stomach, 8 L were drained by the placement of a nasogastric tube.

The filly was positioned in dorsal recumbency. The ventral abdomen was clipped from just cranial to the xiphoid cartilage to the mammary glands and scrubbed with a polaxameriodine complex[2] in preparation for an exploratory laparotomy.

The abdomen was entered through an incision of the linea alba extending from the xiphoid cartilage to the umbilicus. Abnormal findings on the exploratory laparotomy were limited to the stomach. The stomach was dilated with fluid and gas but the intestinal tract was almost empty. The antrum pyloricum was distended and demarcated from the rest of the stomach by a constricting ring. The pylorus was hard on palpation, small in circumference and contained a narrow canal whose patency could not be determined externally.

The hepatoduodenal ligament was identified and severed to allow presentation of the pylorus to the abdominal incision. The pylorus was packed off with wet saline towels and a Weinberg modified Heinke-Mikulicz pylorplasty was performed. Two traction sutures of #1 polyglycolic acid[3] were placed through the serosa and muscularis on the ventral aspect of the pyloric sphincter. A full thickness 4 cm longitudinal incision was centered over the pylorus between the traction sutures. It was thought the pylorus was hypertrophied since it was thickened but did not appear or feel fibrotic. Tension on the traction sutures converted to the longitudinal incision into a transverse opening which was closed with a single layer of #0 polyglycolic acid sutures placed closely in a simple interrupted pattern. The pyloroplasty was examined for leakage and lavaged with a crystalline penincillin-physiological saline mixture. The greater omentum was sutured over the incision with a simple continuous pattern of #0 polyglycolic acid sutures.

The abdomen was infused with 20 x 10^6 I.U. crystalline penicillin G in 500 mL of physiological saline. The linea alba was closed with #1 polyester[4] sutures in a simple interrupted pattern. The subcutaneous tissue was closed with #0 polyglycolic acid sutures in a simple continuous pattern while the skin was closed with #00 polyethylene[5] sutures in a continuous horizontal mattress pattern.

The filly was given 360 mg trimethoprim and 1800 mg sulfadoxine solution[6] intravenously daily for six days commencing the day prior to surgery.

Results

Following surgery there were no signs of colic. The grinding of the teeth and the frothing at the mouth continued on the first postoperative day, were markedly decreased on the second day and absent on the third. On day 5 she ate hay and bran mash readily. Six months after surgery, the owner reported that the filly had grown normally and had not shown any signs of illness.

Discussion

We suspect pyloric stenosis in this filly was congenital. The clinical signs began at six weeks of age when the foal was starting to eat significant amounts of solid feed. This material would pass through a constricted pylorus with difficulty, precipitating the onset of clinical signs.

The etiology of CHPS is not well understood (21). Genetic (7, 12, 31, 40), neurological (3) and hormonal (10, 11) factors have been considered. There is good evidence that it is inherited in Siamese cats (40). Gastric ulceration and hypertrophic pyloric stenosis can be produced in puppies by the administration of pentagastrin to pregnant bitches or newborn puppies (6, 10, 11). Pentagastrin is a synthetic peptide with a spectrum of biological activity similar to gastrin, a naturally occurring foregut hormone which stimulates the secretion of high acidity digestive juices. However, this model appears to be species specific and it seems unlikely that the transplacental passage of gastrin can be invoked as a cause of CHPS in any other species (23).

Other etiological possibilities could be considered. Chronic gastritis and gastric ulceration predispose to pyloric stenosis in man (18), dog (1, 9) and rabbits (25). Gastric erosion and ulceration of the pars esophagus from mechanical trauma by *Gastrophilus* larvae, stones or other sharp crystalline material, is common in foals two to 12 weeks of age. Ulceration of the pyloric glandular region, secondary to long-term stress or corticosteroid therapy, is less common (37). A teaspoon of undiluted creolin may have been administered to this filly at a month of age as a treatment for diarrhea. This corrosive substance could result in fibrosis of the stomach and pylorus several weeks later. No evidence of ulceration or fibrosis was detected in the esophagus or stomach with endoscopy and surgical exploration.

Pylorospasm, leading to hypertrophic pyloric stenosis is seen in small adult dogs with a nervous disposition. At first, vomition occurs inconsist-

[1]Somnothane, Hoechst Pharmaceuticals, Montreal, Quebec.
[2]Prepodyne Scrub, West Chemical Products Ltd., Montreal, Quebec.
[3]Dexon, Davis & Geck, Cyanamid of Canada Ltd., Montreal, Quebec.
[4]Ticron, Davis & Geck, Cyanamid of Canada Ltd., Montreal, Quebec.
[5]Dermalene, Davis & Geck, Cyanamid of Canada Ltd., Montreal, Quebec.
[6]Trivetrin Injectable, Burroughs Wellcome & Co., London, England.

ently after meals but as muscular hypertrophy develops, it becomes a constant sign (24). This filly was always docile and the clinical signs were constant from their onset.

Teeth grinding in horses has rarely been observed by the authors, and reference to it was not found in the literature. It is commonly seen in cattle with abdominal pain or chronic lead poisoning (4). In this case it was probably a manifestation of abdominal pain due to gastric dilation secondary to pyloric stenosis.

We recommend a midventral abdominal incision extending to the xiphoid cartilage for maximum exposure of the stomach. Presentation of the pylorus to the abdominal incision was difficult and only possible after severing the hepatoduodenal ligament. Care must be taken to avoid the hepatic duct which traverses the hepatoduodenal ligament and mesoduodenum before entering the duodenum at the diverticulum duodeni 12.5-15 cm distal to the pylorus (16, 30). Adequate exposure of the pylorus for surgical treatment may not be possible in larger patients.

Heineke-Mikulicz pyloroplasty is a widely accepted procedure for treatment of pyloric obstruction in man and animals (2, 5, 8, 20, 22, 33, 39, 40). The major objective is to shorten gastric emptying time by increasing the diameter of the pyloric canal. Weinberg's modification of the Heineke-Mikulicz pyloroplasty with a one-layer closure allows for maximum lumen size without unnecessary risk of contamination (2, 5, 20). The pyloric incision was covered with greater omentum to localize reaction if leakage occurred. The pyloric canal appeared considerably larger following the pyloroplasty.

Marked clinical improvement was seen within two days following the surgery. Others have reported retention of varying degrees of clinical signs for various periods of time following pyloroplasty (20, 30, 35, 40).

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