ULTRASONOGRAPHIC AND PATHOLOGIC FEATURES OF INTESTINAL SMOOTH MUSCLE HYPERTROPHY IN FOUR CATS

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The ultrasonographic findings for four cats with intestinal smooth muscle hypertrophy are described. In two cats, intestinal smooth muscle hypertrophy was associated with chronic enteritis. In the remaining two cats, intestinal smooth muscle hypertrophy affected the intestinal tract proximal to stenosis due to alimentary lymphoma and an intestinal foreign body, respectively. Moderate increased thickness of the affected intestinal wall, measuring 7–8 mm, was evident on abdominal ultrasonographic examination of all subjects. In addition, the ultrasonographic five-layered feature of the intestinal wall was maintained, and only the muscular layer appeared thickened. Abdominal ultrasound allowed a presumptive diagnosis of intestinal smooth muscle hypertrophy that was confirmed histologically in all cats. *Veterinary Radiology & Ultrasound, Vol. 44, No. 5, 2003, pp 566–569.*

Key words: abdominal ultrasound, cat, intestinal disorders, smooth muscle hypertrophy.

Introduction

INTESTINAL SMOOTH MUSCLE hypertrophy is a modification of the muscular layer of the small intestine that occurs in a spontaneous form in the horse and other herbivores,[1–5] pigs,[3,6] and humans.[7,8] It has also been reproduced experimentally in rats, guinea pigs, and pigs by creating a surgical stenosis of the small intestine.[9–11] Thus, on the basis of the underlying pathophysiological mechanism, two different types of intestinal smooth muscle hypertrophy are known: one occurring as a compensatory reaction in response to distal stenosis (i.e., secondary hypertrophy); the other occurring in the absence of a detectable stenosis leading to proximal muscular thickening (i.e., idiopathic hypertrophy).

Idiopathic intestinal smooth muscle hypertrophy has seldom been reported in domestic carnivores. The histopathological findings of idiopatic intestinal smooth muscle hypertrophy, which involved the duodenum and cranial portion of the jejunum, were recently described in a dog.[12] An intestinal wall thickening due to hypertrophy of the external muscle layers with little involvement of the mucosal layer has rarely been reported in elderly cats.[13] This condition, usually located in the ileum, was mainly an incidental finding at necropsy. An isolated report of feline idiopathic muscular hyperplasia associated with suspected intestinal carcinoma has also been reported in an article[14] describing the ultrasonographic features of gastrointestinal diseases in small animals. Furthermore, ultrasonographically detectable thickening of the muscular layer has been reported in some cats[15,16] and a dog[17] with chronic inflammatory gastrointestinal disease. Finally, a preliminary report of the pathological findings of feline intestinal smooth muscle hypertrophy was made by some of the authors of the present article.[18]

The purpose of this article is to describe the ultrasonographic and pathological features observed in four cats affected with intestinal smooth muscle hypertrophy.

Materials and Methods

The medical records of cats referred to the Department of Veterinary Clinical Sciences of the University of Bologna from September 1996 to November 1999 were reviewed. Among them, four patients undergoing an ultrasonographic examination of the abdomen in which histological confirmation of intestinal smooth muscle hypertrophy was obtained were selected. All cats had a history of and clinical signs referable to a chronic gastrointestinal disorder. Physical examination, complete blood count, and routine serum biochemical examinations, including serology for feline leukemia virus (FeLV), feline immunodeficiency virus (FIV), and feline coronavirus (FCoV) were conducted in all subjects.

The ultrasonographic evaluation of the abdomen was performed in all cats after 12 hours fasting using a real-time

sector transducer* at a frequency of 7.5 or 10 MHz, with patients placed in lateral and dorsal recumbency. The wall thickness of the small intestine (measured in a transverse axis from the inner hyperechoic interface of the mucosal surface to the outer hyperechoic layer of serosa), the wall layer distribution, the location and extension of any detectable intestinal lesion, the symmetry of the wall at the lesion site, and the gastrointestinal content and motility were evaluated. Furthermore, abdominal lymph nodes were carefully examined.

Exploratory celiotomy was performed in three cats, and specimens of the small intestine were submitted for histologic examination. Two cats underwent enterectomy, with removal of an intestinal foreign body in one cat. Three of the four cats were killed at the owner's request, immediately after ultrasonographic examination (Case 1), after celiotomy (Case 3), or after the recurrence of the symptoms 2 months postenterectomy (Case 2). Necropsy examination was performed in all of these three cats.

Results

The cats (two males, one neutered male, and one female) ranged from 5 to 10 years of age (mean 7.5 years), and body weight ranged from 2.8 to 3.4 kg (mean 3.1 kg). All subjects had a history of anorexia, vomiting, and diarrhea associated with weight loss that had lasted for at least 2 months. On physical examination, the cats were slightly depressed, clearly underweight, and dehydrated. There was palpable thickening of intestinal loops in all subjects. In addition, a palpable, moveable, round, midabdominal mass was appreciable in one cat. Increased red blood cell count, packed cell volume, and hemoglobin concentration, and hyperprotein-aemia were found in all cats. No other serum biochemical alteration was present. Fecal examination was also performed, and no intestinal parasites were found. All cats were negative for FeLV, FIV, and FCoV on testing.

Ultrasonographically, two cats had symmetric wall thickening with preserved wall layering of several segments of the jejunum. The changes extended up to 5 cm in length. The wall thickness of the above intestinal portions, measured at different sites, ranged from 7 to 7.5 mm. Intestinal wall layering was maintained, but thickening of the muscular layer (5 mm in thickness as a whole) was evident. The other components of the intestinal wall (i.e., mucosa, submucosa, and subserosa/serosa) appeared normal (Fig. 1). The duodenum and the terminal part of the ileum were normal. Gastrointestinal motility was normal, and no ultrasonographic evidence of mesenteric lymphadenopathy was found.

In another cat, two different abnormal ultrasonic patterns of the small intestine were evidenced. One segment of the
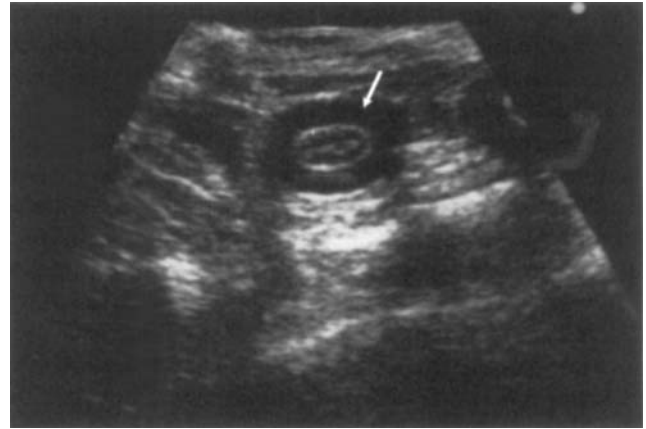
FIG. 1. Transverse ultrasound image of the small intestine. Note the increased thickening of the muscular layer (arrow) due to smooth muscle hypertrophy.

jejunum had symmetric wall thickening, measuring 10 mm, that extended for approximately 15 cm. The disappearance of the normal wall layering and uniformly hypoechoic intestinal wall thickness were evident at this level (Fig. 2). Bright central luminal echoes with distal reverberation and decreased intestinal motility were also found. The intestinal loops proximal to the lesion had a different ultrasonic feature. In particular, a symmetric circumferential thickening (8 mm as a whole) originating from the muscular layer (measuring 5 mm) was found. Neither disruption of wall layering nor changing of intestinal wall ecotexture was present. This intestinal segment was moderately distended with abnormal fluid accumulation. Increased peristaltic activity at this site was also evident. Enlarged, hypoechoic, round, and smoothly margined mesenteric lymph nodes were observed along the thickened intestinal loops.

A moderately distended jejunal segment with increased motility and containing an abnormal amount of ingesta was
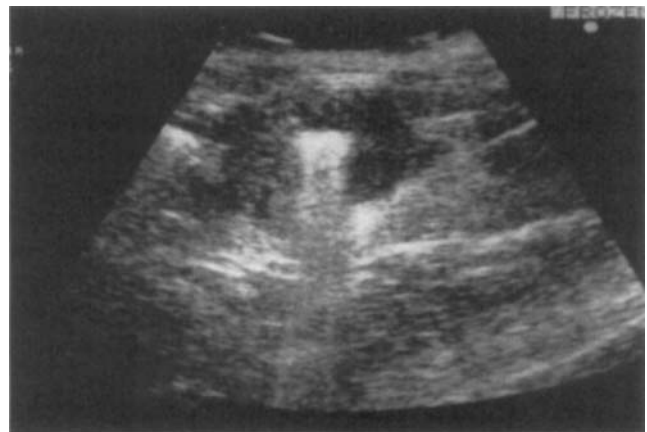

FIG. 2. Transverse ultrasound image of the small intestine. Note the disruption of intestinal wall layering and the presence of bright central luminal echoes with distal reverberation (intestinal lymphosaracoma).

found in the final cat. This jejunal portion had increased wall thickening (8 mm) that extended for a length of 3 cm and involved only the muscular layer (measuring 6 mm). Intestinal wall layering was maintained. An endoluminal hyperechoic structure (15 mm in length and 10 mm in width) was seen distal to the intestinal loop and was characterized by muscular thickening. In particular, the above structure had a curved echogenic border with distal shadowing (Fig. 3). No motility was noted at the lesion site, whereas an augmented peristalsis was present in the proximal enlarged part of the bowel. No lymph node abnormalities were detected.

At necropsy, the first two cats had marked cachexia. The bowel was rigid and diffusely thickened. On sectioning, the lumen was narrowed and contained little ingesta, and it was clearly apparent that the major component of the increased thickness involved the muscular layer. In one of these cats, the mucosal surface was markedly reddened. The ileocecal valve was apparently not obstructed or restricted, and no parasites were evident. The mesenteric lymph nodes were mildly enlarged. Histologically, the intestinal wall thickening was the result of a marked hypertrophy of the muscular

layers, which was more apparent in the inner layer (Fig. 4). Smooth muscle fibers were parallel and spindle-shaped, but were increased in size, with long and vesicular nuclei. When cross-sectioned, smooth muscle cells appeared plump, and the cytoplasm varied in staining intensity. The myenteric plexus appeared normal. In sections stained with Gomori and Masson's trichrome, the smooth muscle fascicles were separated by a thin fibrous stroma of reticulin and, at times, dense collagen fibers. The mucosa and the submucosa had severe infiltration of lymphocytes, macrophages, plasma cells, and dense fibrous connective bundles. Villi were blunted, and varied in thickness and length, and there was an abundance of globlet cells. The final diagnosis was severe diffuse intestinal smooth muscle hypertrophy associated with moderate-to-severe chronic lymphocytic-plasmacytic enteritis in both cats.

Cat 3 had a nodular soft swelling of the jejunum, located 10 cm proximal to the ileocecal valve, and a remarkable thickening and rigidity of the bowel cranial to the intestinal mass, extending for 15 cm. On sectioning, the mass was composed of white friable tissue, and the lumen was nearly completely obstructed. The mesenteric lymph nodes were notably enlarged. Histologically, the mass had the infiltrative proliferation of a monotonous population of immunoblast-like lymphocytes that caused the complete disruption of the intestinal architecture. The mucosa, the submucosa, and the muscular layer were severely affected. Marked hypertrophy of the smooth muscle, morphologically similar to that observed in the first two cats, was present proximal to the mass and was associated with mild submucosal lymphoid infiltrate. The mesenteric lymph nodes had a complete loss of their architecture associated with a lymphomatous infiltration of the capsule and perinodal tissues. The diagnosis was diffuse, immunoblastic, high-grade, alimentary lymphoma and secondary intestinal smooth muscle hypertrophy.
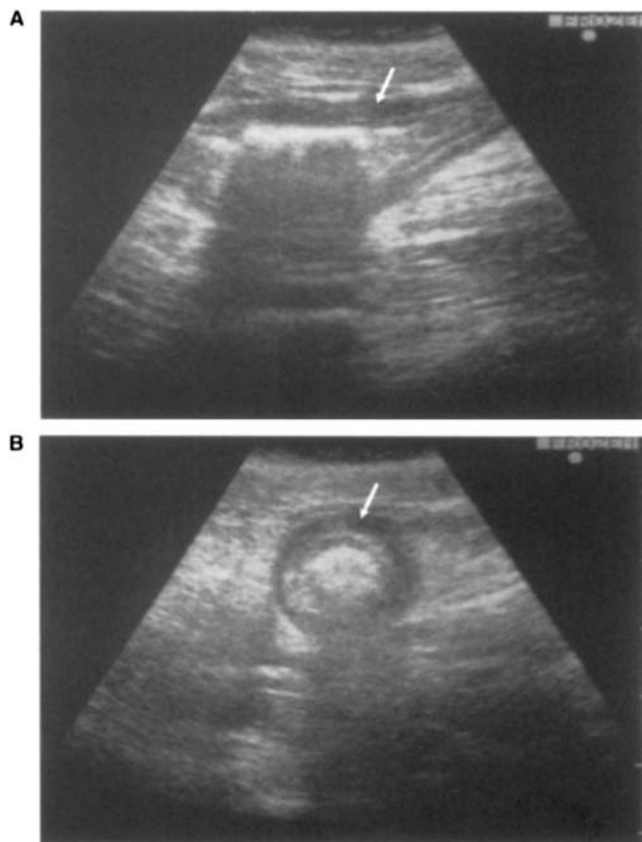

FIG. 3. Sagittal (A) and transverse (B) ultrasound images of the small intestine. Note the central hyperechoic structure and acoustic shadowing due to an intestinal foreign body (rubber tube). Maintained intestinal layering and increased thickness of the muscular layer (arrow) are also evident.
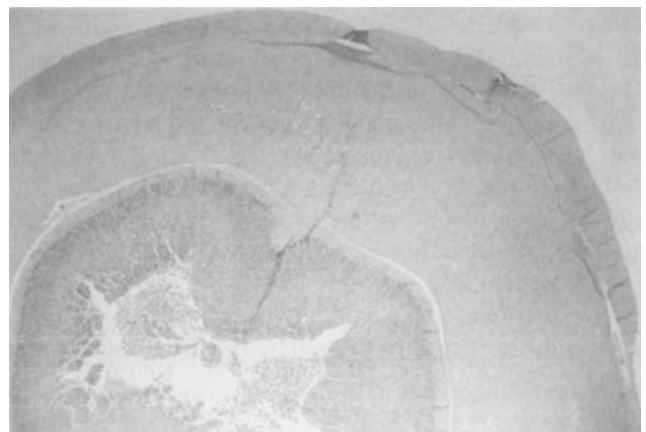

FIG. 4. Photomicrograph of the small intestine. Note the thickening of the intestinal wall due to marked hypertrophy of smooth muscle cells, more apparent in the circular layer. Hematoxylin and eosin, ×10.

Case 4 had a well-delineated, firm swelling of the intestine with proximal thickening and rigidity of the bowel. The intestinal serosal surface was reddened, and there were some fibrin strands. On sectioning, the intestinal wall of the affected area was discolored purple and contained a foreign body (portion of a rubber tube, 10 mm long, and 10 mm in diameter). The mucosal surface was discolored. Histologically, there was necrosis of the mucosa, submucosa, and muscularis corresponding to the area of the foreign body. Peripherally, there was intense infiltration of neutrophils, angiogenesis, fibroplasia, and edema of the submucosa, which were coherent with active granulation tissue demarcating the necrosis. The thickened bowel proximal to the foreign body had marked hypertrophy of smooth muscle. The final diagnosis was transmural, chronic, necrotizing foreign-body enteritis and secondary intestinal smooth muscle hypertrophy.

Discussion

Moderately increased wall thickness of some intestinal tracts (range 7–8 mm) was a consistent ultrasonographic finding in all cats of the present study. Furthermore, the above thickening involved only the muscular layer, whereas the five-layered ultrasonographic appearance of the small bowel was maintained. In the first two cats, no other ultrasonographically appreciable intestinal abnormalities were evident. In the remaining two cats, the above ultrasound feature was associated with stenosis. In Cat 3, transmural

thickening of the intestinal wall, associated with a loss of layering, low-level echogenicity of the wall, and poor motility, was found next to the intestinal tract involved by muscular thickening. This stenotic region was consistent with gastrointestinal lymphosarcoma.[19-21] In Cat 4, a foreign body was present immediately distal to the intestinal tract, which was characterized by muscular thickening.

The ultrasonographic findings led us to suspect intestinal smooth muscle hypertrophy. Most common feline intestinal neoplasms (i.e., adenocarcinoma and lymphosarcoma) are characterized by increased intestinal wall thickness associated with a partial or complete disruption of the normal layered appearance of the intestinal wall.[19,20,22,23] Extensive and symmetric thickening of the intestinal wall associated with retained layering can also be observed in feline chronic inflammatory bowel disease. Mildly thickened bowel (4–5 mm in thickness), mainly involving the mucosa and submucosa, and poor intestinal wall layering definition usually characterize inflammatory bowel disease.[15,16,24] Nevertheless, an ultrasonographic feature of smooth muscular hypertrophy similar to that observed in our patients has also been reported in some cats with inflammatory bowel disease.[16,24]

In conclusion, smooth muscle hypertrophy is an abnormality of the small intestine that has been to date, only briefly described in literature. Ultrasound is useful in diagnosing both the idiopathic and the secondary forms of this disorder.

REFERENCES

1. Rooney JR, Jeffcott LB. Muscular hypertrophy of the ileum in a horse. Vet Rec 1968;83:217–219.

2. Hancock JL. Muscular hypertrophy of the ileum in the horse. Vet Rec 1968;83:304–305.

3. Cordes DO, Dewes HF. Diverticulosis and muscular hypertrophy of small intestine of horses, pigs and sheep. N Z Vet J 1971;19:108–111.

4. Chaffin MK, Fuenteabla IC, Schumacher J, et al. Idiopathic muscular hypertrophy of the equine small intestine: 11 cases (1980–1991). Equine Vet J 1992;24:372–378.

5. De Lara FCM, Hervas J, Bautista MJ, et al. Intestinal smooth muscle hyperplasia in a goat. J Vet Diagn Invest 1996;8:390–392.

6. Chu RM, Lin YL, Chi CS. Muscular hypertrophy of ileum in swine. J Chinese Soc Vet Sci 1989;15:73–81.

7. Spencer R, Hudson TL. Idiopathic muscular hypertrophy of the gastrointestinal tract in a child. Surgery 1961;50:678.

8. Uhrich GI. Idiopathic muscular hypertrophy of the intestinal tract. Am J Surg 1965;110:973–976.

9. Gabella G. Hypertrophy of intestinal smooth muscle. Cell Tissue Res 1975;163:199–214.

10. Gabella G. Hypertrophic smooth muscle: I. Size and shape of cells, occurrence of mitoses. Cell Tissue Res 1979;201:63–78.

11. Brehmer A, Göbel D, Freiser M, et al. Experimental hypertrophy of myenteric neurones in the pig: a morphometric study. Neurogastroenterol Motil 2000;12:155–162.

12. Santos RL, Coelho MIM. Hipertrofia muscular intestinal idiopatica em cao: relato de caso. Arq Bras Med Vet Zoot 1998;50:525–529.

13. Guilford WG, Strombeck DR. Intestinal obstruction, pseudo-obstruction and foreign bodies. In: Guilford WG, Strombeck DR. Small animal gastroenterology, 3rd ed. Philadelphia: WB Saunders Co, 1996;487–502.

14. Penninck DG, Nyland TG, Kerr LY, Fisher PE. Ultrasonographic evaluation of gastrointestinal diseases in small animals. Vet Radiol Ultrasound 1990;31:134–141.

15. Baez JL, Hendrick MJ, Walker LM, Washabau RJ. Radiographic, ultrasonographic, and endoscopic findings in cats with inflammatory bowel disease of the stomach and small intestine: 33 cases (1990–1997). J Am Vet Med Assoc 1999;215:349–354.

16. Penninck DG. Gastrointestinal tract. In: Nyland TG, Mattoon JS (ed). Small animal diagnostic ultrasound, 2nd ed. Philadelphia: WB Saunders, 2002; 207–230.

17. Spohr A, Koch J, Jensen AL. Ultrasonographic findings in a basenji with immuno-proliferative enteropathy. J Small Anim Pract 1995;36:79–82.

18. Bettini G, Muracchini M, Della Salda L, et al. Hypertrophy of intestinal smooth muscle in cats. Proceedings of the 16th Meeting of the European Society, Veterinary Pathology, Lillehammer, Norway, September 23–26, 1998; 166.

19. Penninck DG. Characterization of gastrointestinal tumors. Vet Clin N Am Small Anim Pract 1998;28:777–797.

20. Penninck DG, Moore AS, Tidwell AS, et al. Ultrasonography of alimentary limphosarcoma in the cat. Vet Radiol Ultrasound 1994;35:299–304.

21. Hittmair K, Krebitz-Gressel E, Kubber-Heiss AK, Mostl K. Feline alimentary lymphosarcoma: radiographical, ultrasonographical, histological and virological findings. Eur J Comp Anim Pract 2001;11:174–183.

22. Rivers BJ, Walter PA, Johnston GR. Ultrasonographic features of intestinal adenocarcinoma in five cats. Vet Radiol Ultrasound 1997;38:300–306.

23. Grooters AM, Biller DS, Ward H, et al. Ultrasonographic appearance of feline alimentary lymphoma. Vet Radiol Ultrasound 1994;35:468–472.

24. Lamb CR. Recent developments in diagnostic imaging of the gastrointestinal tract of the dog and cat. Vet Clin N Am Small Anim Pract 1999;29:307–342.