Ultrasonographic and histopathological features in cats with fibrotic small intestinal stricture

Keywords: feline, stenosis, ischaemia, foreign body

Abstract

Benign stricture is an uncommon cause of chronic small intestinal obstruction in the cat. The purpose of this retrospective case series was to describe the ultrasonographic features, histopathological findings and clinical presentation in a group of cats with benign small intestinal stricture. Inclusion criteria were cats presenting during the period 2010-2017, and that had ultrasonography and small intestinal stricture confirmed at surgery. For each cat clinical data and ultrasonographic findings were retrieved from the medical record, and histopathology, where available, was reviewed. Eight cats met the inclusion criteria. The location of strictures was duodenum (1/8), mid- to distal jejunum (4/8) and ileum (3/8). Ultrasonographic findings included gastric distension (8/8) and generalized (3/8) or segmental (5/8) intestinal dilation consistent with high-grade obstruction. Ingesta did not propagate beyond the strictured segment. Wall thickening was mild to moderate (3-6mm). Normal wall layering was disrupted in all cats. Strictures were predominantly hypoechoic (7/8) and associated with hyperechoic peri-intestinal mesentery (6/8). Annular strictures (5/8) were less than 15mm in length whereas long-segment strictures (3/8) were greater than 15mm in length. Histopathology showed transmural disease with fibrosis and inflammation (8/8), often (6/8) extending into the bordering mesentery. The mucosa was the most severely affected layer and epithelial injury accompanied the mucosal fibrosis/inflammation. Clinical presentation reflected delayed diagnosis of chronic bowel obstruction with debilitation (8/8), marked weight loss (8/8) and pre-renal azotaemia (5/8). Benign fibrostenotic stricture should be considered a differential diagnosis in debilitated young cats presenting with chronic bowel disease and ultrasonographic features of intestinal obstruction.

Introduction

Chronic intestinal obstruction may result from intraluminal foreign body, intussusception or intestinal stenosis caused by intra – or extramural disease.[1-4] The clinical signs of chronic obstruction are non-specific including inappetence, lethargy, vomiting, weight loss and diarrhoea and must be distinguished from inflammatory bowel disease (IBD), intestinal small cell lymphoma (ISCL), hyperthyroidism, dietary and infectious disease which can present with similar clinical signs.[1,4-6] Intramural intestinal obstruction in the cat is usually caused by intestinal wall neoplasia.[1,4,7] Non-neoplastic intramural obstruction is uncommon arising secondary to pyogranuloma, duplication cyst and, occasionally, due to benign intestinal stricture.[1,8]

A benign intestinal stricture is a circumscribed narrowing or stenosis of the intestinal lumen caused by inflammation, adhesion, incarceration or cicatricial contracture (fibrostenotic stricture).[9] Strictures are usually solitary lesions and the extent of the intestinal stenosis may be focal and annular or involve long segments of bowel, and result in partial or complete intestinal obstruction.[10-13] Reports of benign stricture in the veterinary literature are rare but have been described in the dog as a post-anastomotic complication, due to strangulation associated with spontaneous mesenteric hernia and secondary to rupture of a congenital intestinal diverticulum.[1,14]

The diagnosis of small bowel obstruction, whether partial or complete, relies on demonstrating segmental small intestinal dilation and hyperperistalsis oral to a transition zone.[3,15,16] The transition zone represents the site at which dilated bowel transitions to normal bowel and localizing the transition zone increases confidence in determining the specific cause of obstruction.[15,16] Abdominal ultrasonography and radiography are the imaging techniques most commonly employed to demonstrate bowel obstruction in the dog and the cat.[1,3]

Description of the ultrasonographic and histological features of benign intestinal stricture as a cause of intestinal obstruction in the cat is lacking. The purpose of this retrospective study was to describe the clinical presentation, ultrasonographic and histopathological features and outcome in a group of cats with chronic small intestinal obstruction secondary to benign small intestinal stricture.

Methods

This was a retrospective, multicentre descriptive study. The ultrasonography databases of two referral hospitals, and a radiology service to three first opinion practices were searched between January 2010 and August 2017 for cats with an ultrasonographic diagnosis of bowel obstruction consistent with a small intestinal stricture. Further inclusion criteria included surgical exploration within 24 hours of the ultrasound diagnosis, confirmation of the stricture at surgery by palpation and an inability to milk intestinal content beyond the stenotic segment, and a histological diagnosis consistent with non-neoplastic stricture characterized by various degrees of fibrosis, inflammation and necrosis. Cats with intestinal stenosis secondary to mural or intraluminal mass lesions or the annular-stenosing form of intestinal adenocarcinoma were excluded as were cats with focal or segmental forms of transmural necrotizing enteritis but without high-grade obstruction at ultrasound examination or at surgery. Two board-certified veterinary radiologists and a board-certified anatomic pathologist determined subject eligibility.

Patient information collected included the following: age, gender, breed, weight, clinical signs and outcome. Ultrasound reports, still images and, when available, video loops, were retrospectively evaluated. Images were reviewed using commercial DICOM software (OsiriX, Pixmeo, Geneva). Ultrasound findings were determined by consensus

by two board-certified veterinary radiologists. An intestinal stricture was considered present if intestinal dilation, consistent with high-grade mechanical obstruction, was demonstrated immediately oral to an abnormal, non-distensible segment of bowel. Strictures were assessed for location within the bowel (duodenum, jejunum, ileum), intestinal wall thickness, and the appearance and length of the stricture. The wall thickness of the stricture was measured from the serosal margin to the mucosa-luminal interface. Stricture length was measured from the transition of dilated to attenuated bowel lumen at the oral aspect, to the transition to bowel with normally defined wall layering at the aboral aspect. Gastric and small intestinal appearance (normal, distended) and motility (hyperperistalsis, ileus) were recorded.

Surgical findings recorded included the location and appearance of the stricture. All available histopathologic samples were retrieved and reviewed by a single board-certified anatomic pathologist. The severity of changes (fibrosis, inflammation and epithelial injury) was scored according to the histopathological criteria published by the World Small Animal Veterinary Association Gastrointestinal Standardization group.[17] Follow-up was determined based on the clinical records or telephonic contact with owners.

Study data, and retrieval of histopathological material for review, was approved, when applicable by the clinical directors of the relevant practices or Ethical Review Board.

Results

Eight cats (five domestic short hair, two Maine Coon, one Ocicat) met the inclusion criteria. Six cats were neutered males and two were neutered females. Ages ranged from two to ten years (median four years). The duration of clinical signs prior to initial presentation to the primary care veterinary surgeon ranged from two days to 21

days(median seven days) and the duration between initial presentation and ultrasonographic diagnosis of a stricture ranged from two days to six weeks(median 20.5 days). Clinical signs were characterized by initial intermittent episodes of vomiting associated with hyporexia and malaise, progressing to recurrent episodic vomiting, anorexia and profound lethargy. Projectile, large volume fluid vomiting or regurgitation was noted in the latter stages in 3/8 cats, diarrhoea was reported in 3/8. Faecal analysis performed in one cat was negative. Abdominal palpation was normal in 2/8 cats and equivocal intestinal thickening was suspected in 6/8 cats. All cats were subjectively debilitated at the stage at which abdominal ultrasonography was performed and pre-renal azotaemia was recorded in 5/8 cats at this stage. Body weight recorded at the time of ultrasonographic assessment ranged from 2.7kg to 5.7kg(median 4.10kg). The percentage loss of body weight at the point of ultrasonography ranged from 9.5% to 32%(median 24.5%).

Ultrasonography was performed with the following machines: CX50 or Epiq 5 (Philips Medical Systems, Eindhoven, The Netherlands) using curved (8-5MHz), and linear (15-7MHz, 18-5MHz) array transducers, RS80a (Samsung, Healthcare, Gangnam-Gu, South Korea) using curved (4-9MHz) and linear (3-16MHz) array transducers and MyLab30 (Esaote, Genoa, Italy) using curved (9-3MHz) and linear (11-3 MHz) array transducers. Ultrasonographic images and video loops were available in 6/8 cats and ultrasound reports only, in 2/8. On ultrasonographic examination moderate to marked fluid gastric distension was evident in all eight cats. Gastric hyperperistalsis was present in 2/8 cats and gastric stasis in 6/8 cats. The location of the stricture was duodenum (1/8), mid- to distal jejunum (4/8) and proximal ileum (3/8). Moderate to marked distension of the small intestine oral to the stenotic segment indicating high-grade bowel obstruction was noted in all cats. This distension was generalized in 3/8 cats with ileal strictures

and segmental in 5/8 cats with duodenal or jejunal strictures. Intestinal ileus was present in 7/8 cats. The stenotic segment was circumferential in all cats. The length of the stricture ranged from 2mm to 50mm (median 15mm). Strictures were divided into two groups based on shape and length. Strictures with a "napkin ring" appearance, were termed annular, whilst those of uniform diameter and cylindrical in shape, were termed long-segment strictures. In all 5/8 cats with annular strictures the stenotic segment was less than 15 mm in length, whereas in all 3/8 cats with long-segment strictures the stenotic segment was greater than 15 mm in length. The transition between dilated oral bowel and non-dilated bowel aboral to annular strictures was abrupt (Figure 1). The long axis of the bowel was kinked at the oral aspect of the stricture. Careful repetitive assessment of the stricture segment and image optimization was usually required to align dilated oral bowel, stricture and non-dilated aboral bowel in the same imaging plane. In long-segment strictures the transition between the dilated oral segment and the stenotic segment was subjectively more gradual. Long-segment strictures subjectively appeared fixed and rigid compared to adjacent bowel loops (Figure 2). In both annular and long-segment strictures the lumen of the focally affected segment was obliterated and intestinal contents did not propagate past the stricture as a result of peristalsis, or in response to transducer pressure, when observed over a period of time. In all cats a population of non-dilated bowel with preserved wall layering and the normal ileocolic junction (ICJ) was identified aboral to the stricture.

The wall of the stenotic segment was mildly to moderately thickened, ranging from three to six mm(median 3.9mm). Wall layering within the stricture segment was abnormal in all cats, with complete (1/8) or partial (7/8) loss of layering. In the one cat with complete loss of layering the stenotic segment was a narrow, 2mm length, discrete

circumferential hypoechoic ridge (Figure 1). In the remaining seven cats with partial loss of wall layering the appearance of the stricture segment assessed using medium-frequency transducers was of conspicuously hyperechoic and hypoechoic thickened mucosa and tunica muscularis respectively in one cat (Figure 1B), and predominantly hypoechoic in 6/8 cats. Visualization of indistinct bowel wall layering was improved on high-resolution linear images in these six cats (Figure 3 and Figure 4). The mucosal layer was hypoechoic to isoechoic to the submucosal layer, the submucosal layer was indistinct and mildly thickened and the tunica muscularis presented as the most thickened and prominent wall layer. Extramural changes characterized by focally thickened and hyperechoic mesentery bordered the stenotic segment in 6/8 cats. In all cats with peri-intestinal changes the serosal layer was indistinct. There was no correlation between the presenting signs, location or length of intestinal strictures. Diffuse bowel wall thickening, ranging from 3.4mm to 4.2mm(median 3.5mm), with prominence of the tunica muscularis was noted in the non-distended bowel aboral to the stricture segment in six cats. In one cat with a duodenal stricture a sharply marginated foreign body was located in the distal duodenum aboral to the stricture (Figure 1C). Following ultrasonographic diagnosis of intestinal stricture cats were stabilized, proceeding to surgical exploration within 24 hours. The abnormal intestinal segment (and a duodenal foreign body in one cat) was identified at surgery and enterectomy performed in each case (Figure 5). Vasa recta arising from the terminal jejunal arcade at the stricture site subjectively appeared attenuated compared to adjacent arcades in 2/4 cats for which intra-surgical photographs were available. Post-surgical recovery was uncomplicated in all cats with return of appetite, demeanor and weight gain. One cat presented with aortic thromboembolism two weeks post-surgery and a second cat presented in congestive heart failure two-months after surgery. Echocardiographic

findings consistent with cardiomyopathy were present in both cats and both were euthanazed. Intestinal stricture has not recurred in the remaining six cats (median follow-up time of 18mnths, range of 1 year to 8 years).

Resected intestinal segments were submitted for routine histopathology to four different veterinary diagnostic laboratories. For 6/8 cases, haematoxylin and eosin-stained sections and/or paraffin blocks were then retrieved and reviewed by a single board-certified anatomic pathologist. The same pathologist also reviewed the histopathology reports for the remaining 2/8 cases, for which the original slides and blocks were not available for re-examination. In all eight cases histopathology revealed changes centered mostly on the mucosa and consisting of fibrosis, epithelial injury and inflammation (Figure 6). Fibrosis was the most obvious change. It was always marked and in 3/8 cats the sections available allowed to show that the fibrosis was predominantly responsible for the narrowing of the intestinal lumen. The epithelial injury was a combination of ulceration, necrosis, and loss of villi/crypts. The mucosal inflammation was variable in severity (mild in three cats and moderate in five cats) and always consisting of an admixture of neutrophils, lymphocytes, plasma cells and macrophages. Rare eosinophils were also noticed in two cats. Mucosal hyperemia and small mucosal haemorrhages were also observed in 5/8 cats. In each case the intestinal submucosa and tunica muscularis also displayed fibrotic and inflammatory changes and haemorrhages, but these were much less severe than those affecting the mucosa. The intestinal serosa and adjacent peritoneal adipose tissue also appeared mildly involved by fibrosis, inflammation and hyperemia in 6/8 cats. Occasional small fragments of foreign material were noted in 2/8 cats associated with the lesions. In one case the extraneous material was compatible with plant material and lay on the ulcerated mucosal surface surrounded by few bacteria. In the second case the nature of

the foreign material was uncertain and this material was more deeply located (deep mucosa and submucosa) and surrounded by epithelioid macrophages and multinucleated giant cells.

Discussion

This report demonstrates that the transition zone in fibrotic strictures causing small intestinal obstruction in the cat is variable in length and can present as either an annular or as a long-segment stricture. The length of stricture influenced the ultrasonographic features used to localize and characterize strictures. Annular intestinal strictures in this report were optimally demonstrated when the discrepancy between the dilated oral bowel segment, the stenotic segment and non-dilated aboral bowel were all in the same ultrasonographic imaging plane, along the long axis of the small intestinal loop. This approach is similar to that used to identify a small intestinal intussusception or foreign body.[1,18] The challenge in applying this approach to identify an annular stricture is that the dilated intestinal loop tends to kink or pivot at the oral aspect of the stricture, distorting the normal long axis of the small bowel.[15,16] As a consequence annular strictures may be overlooked as the transition zone is short and the out-of-plane stricture is easily obscured by adjacent distended bowel loops. Intraluminal foreign bodies in contrast are more easily located as they usually increase intestinal diameter, bracing the dilated oral loop and the transition zone tends to remain in the same plane. In comparison long-segment strictures were recognized as longer, fixed, thickened segments of small bowel. These segments shared imaging features with diffuse or segmental bowel thickening due to ISCL, IBD, eosinophilic enteritis and intestinal fibrosis.[5,6,19-21] Distinguishing long-segment strictures from these more common diseases relied on the assessment that suspected strictures were non-distensible bowel

segments with inappropriate dilation of the immediate oral small bowel. Persistent luminal narrowing was a key feature and determined by failure to observe ingesta propagate through the stenotic segment due to peristalsis or in response to transducer pressure if peristalsis was absent.[10,22,23] Similar to observations of intestinal strictures in man, affected bowel subjectively appeared rigid without the plasticity of normal bowel segments in response to transducer pressure.[10,22,23] These changes, together with segmental abnormal wall layering and bordering hyperechoic mesentery, were features that helped localize and differentiate long-segment strictures from adjacent, more normal, bowel.

All but one of the strictures in the cats in this report was localized to the mid- to distal jejunum or ileum. In distally located strictures the length of normal non-dilated bowel aboral to the stricture is therefore short and this segment may be overlooked and generalized bowel distension secondary to obstructive disease potentially misinterpreted as functional ileus.[18] Recognizing that there is a discrepancy in bowel size has been conceptualized as the presence of *two populations* of small intestine, one dilated, one normal and is frequently associated with small intestinal obstruction.[15,16,18] Failure to recognize the *two-population* pattern may contribute to a search for a transition zone being abandoned prematurely and the potential for obstructive disease discounted.

Irrespective of the cause, or the imaging modality employed, thickening and abnormal layering of the small intestine wall are considered the most important imaging features used to assess intestinal disease associated with stricture.[23,24] In the cats in this report both these findings were present but as thickening of strictured segments was mild to moderate at most, abnormal layering and a non-distensible lumen were probably more conspicuous features of stricture than wall thickening. Strictures wall thickness was

similar to that reported in IBD but was thinner compared to intestinal smooth muscle hypertrophy or intestinal adenocarcinoma.[4,5,19,25] In man inflammatory mural strictures tend to be thicker than fibrotic mural strictures. As the most prominent histological change in all cats was fibrosis this probably accounts for relatively mild wall thickening documented. Demonstrating abnormal wall layering is best-achieved using high-resolution ultrasound and when present usually reflects more advanced disease with transmural involvement.[23] Histological change in these cats was transmural with the most severe change involving the mucosa whereas ultrasonographically the tunica muscularis was subjectively the most conspicuous and thickened layer in most cats. This comparison suggests that the extent and significance of mucosal change represented in fibrotic strictures may be underestimated using ultrasound. The observation of severe epithelial injury, representing destruction of the mucosa is important as, independent of cause, chronic or recurrent inflammation-induced mucosal damage is a precondition for the initiation of intestinal fibrosis.[26,27] Correlating the extent of mucosal change and luminal stenosis with histological changes, even using high-resolution ultrasound, may be inexact if ultrasonographic images and the location of sampling for histopathological slides are not carefully correlated. This emphasizes that the diagnosis of intestinal stricture relies on the integration of imaging, surgical and histopathological findings. Histopathology also demonstrated that the thickened, hyperechoic mesentery bordering strictures was due to mild inflammation, fibrosis and hyperemia. When extensive this fibrofatty change may displace other bowel loops allowing an abnormal segment to be localized as a transition zone and distinguished from adjacent bowel loops, particularly as in the long-segment strictures in the cats in this series.[23] The ultrasonographic features of wall thickening, abnormal layering and fibrofatty change are however not specific for intestinal stricture. Annular strictures

may share features with the annular-stenosing form of intestinal adenocarcinoma, intestinal ulceration, perforation or infarction.[4,7,28,29] In turn long-segment strictures must be distinguished from diffuse small intestinal disease due to ISCL, IBD, intestinal fibrosis, and eosinophilic enteritis, although bowel obstruction is not usually a feature of these diffuse diseases.[5,19-21]

The aetiology of the intestinal strictures in the cats in this study is uncertain. Multiple factors may be implicated and the aetiology may indeed differ between annular and long-segment strictures. In humans bowel stricture is usually recognized as a complication of chronic inflammatory intestinal diseases such as Crohn's disease but is also associated with adhesions, bowel incarceration, the use of non-steroidal anti-inflammatory drugs, ischaemia and neoplasia.[10,13,24,30,31] The principal underlying factors implicated in the development of bowel stricture are bowel inflammation, bowel ischaemia and direct mucosal damage.[26,30] Although in the presence of intestinal mucosal ulceration and fibrosis veterinary pathologists might consider an intestinal foreign body as the most likely cause of the mucosal damage leading to benign stricture, obstructing foreign material was identified in only one cat in this series. Direct mucosal damage by an intestinal foreign body alone may not be responsible for stricture development. Instead reduced wall perfusion resulting from marked mechanical distension by the foreign body and leading to wall ischaemia is likely to be a significant contributory factor.[18,32] Limited evidence suggests that most non-linear foreign bodies ingested by cats tend to be of a soft or pliable type and less likely to be associated with severe mucosal injury than rigid foreign bodies similar to that removed in one cat in this series.[2] Migrating plant material has also been suggested as a potential cause, however it is recognized that ulceration and mucosal disruption allows extraneous material to

become embedded within the damaged tissue and if not associated with giant-cell reaction such material is likely to be incidental.

Ischaemia as model for explaining the evolution of fibrostenotic strictures is compelling as it is an important contributor to intestinal injury caused by systemic and infectious diseases and toxins that compromise intestinal blood flow.[27] The response to ischaemia follows a predictable course of inflammation and repair that is represented by an acute phase with oedema, haemorrhage and necrosis affecting the sensitive mucosa primarily and severely. This is followed by a reparative phase with granulation tissue formation, chronic inflammation and mural fibrosis and a delayed phase associated with stenosis. This course accounts for the protracted waxing and waning clinical history encountered with intestinal stricture, often with temporary improvement, finally presenting with delayed signs of high-grade bowel obstruction.[13,30] The histopathological changes and protracted clinical progression of the strictures in the cats in this report closely correlate this pattern of ischaemic intestinal disease. In these cats, except for mild hyperemia and small haemorrhages, considered more likely to be the result of inflammation rather than the primary cause of the tissue damage/stricture, there was no evidence of other significant vascular change. Ischaemic bowel disease without major vascular occlusion or histological evidence of thrombosis may occur due to vasospasm, cardiogenic shock or low-output states.[27,30] In man is usually associated with cardiovascular diseases such as atrial fibrillation and vasculitis.[30] Therefore that two cats presented with aortic thromboembolism and congestive heart failure associated with cardiomyopathy after successful resection of their strictures warrants consideration. Whether ischaemic disease was primarily responsible for benign stricture in these cats is uncertain but the predominant mucosal distribution of the histopathological changes suggests that ischaemia is likely to be implicated in the

evolution of the strictures irrespective of the primary mechanism involved. The attenuation of the vasa recta of the terminal jejunal vascular arcades at the stricture site in two cats also suggests the role of ischaemia in stricturing disease in these cats should not be discounted. Emerging techniques such as contrast-enhanced ultrasonography may be useful to verify and characterize ischaemia associated with necrotizing and fibrostenotic disease.[23] Pathogenic bacteria, enterotoxins and disturbed intestinal microbiota causing mucosal damage and fibrosis have also been implicated in the evolution on intestinal stricture.[33] Histologically, only in one case were bacteria visible in the resected intestinal sample and these bacteria surrounded the plant material lying on the ulcerated surface, which is more compatible with secondary contaminants rather than primary pathogens. However, in general histology is not a sensitive technique to detect bacteria and even when these are identified, their significance is uncertain, especially in sections of small intestine where bacteria are expected as part of the normal intestinal microflora. A limitation of this report, reflecting the multicentre, retrospective nature of the study is that routine comprehensive faecal analysis for infectious agents was not performed. The hypothesis of an infectious disease as the cause of feline small intestinal stricture remains open and requires further investigation.

The protracted clinical history and late-stage debilitated presentation of the cats in this study is similar to that of gastrointestinal neoplasia, IBD, pancreatitis and hepatobiliary disease.[4-7,34] These diseases are usually recognised in middle-aged to older cats whereas the majority of the cats in this study were considerably younger. Based on ultrasonographic and gross features the annular-stenosing form of intestinal adenocarcinoma is an important differential diagnosis for benign intestinal stricture, but is usually diagnosed in elderly cats.[4,7] Delayed diagnosis in these cats can probably be

attributed to under-estimation of the potential for, and severity of, obstructive intestinal disease based on non-specific clinical findings, predominantly distal location of strictures and treatment factors. This emphasizes that further investigation, including ultrasonography should be prioritized in cats with non-specific gastrointestinal signs, anorexia and weight loss to distinguish chronic intestinal obstruction from other diseases with similar presenting signs. Additionally the ultrasonographic features of gastrointestinal dysmotility in this study were characterized predominantly by gastric stasis and intestinal ileus rather than the hyperperistalsis. Functional ileus secondary to prolonged obstruction reflects bowel exhaustion associated with complex metabolic and electrolyte derangements and the direct effect of inflammatory mediators on intestinal smooth muscle.[32] Such severe gastrointestinal dysmotilty is a factor that may limit localization of the stricture segment and should also be recognised as a risk factor for peri-operative complications, including aspiration pneumonia and oesophagitis, secondary to reflux of large volumes of intragastric fluid.

Limitations of this study include the retrospective nature of the study, the low number of cases and lack of a standardized approach to diagnostic investigation across contributing centers. Future studies should focus on colour Doppler and contrast-enhanced assessment of strictured bowel, a comprehensive evaluation for infectious agents, cardiovascular status, biopsy of other areas of abnormal bowel and a standardized follow-up.

Conclusion

This report demonstrated that intramural small intestinal obstruction caused by benign stricture should be considered an uncommon but important differential for chronic progressive gastrointestinal disease, especially in younger, debilitated cats. Strictures

may be variable in length and present as annular or as long-segment strictures. Localization of annular strictures ultrasonographically may be technically challenging due to kinking of the aboral non-distended segment. The sentinel ultrasonographic features of marked gastric distension, intestinal dilation oral to a non-distensible transition zone, with mild to moderate wall thickening, hypoechoic appearance, abnormal wall layering and bordering focal hyperechoic mesenteric reaction should be considered highly suggestive of, but not specific for, intramural fibrostenotic stricture in the cat.

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Figure 1. High-frequency(18MHz) linear ultrasonographic images of (A) an annular mid-jejunal stricture in a cat (A), and (B, C) an annular duodenal stricture and foreign body in a second cat. Oral is to the left of the image, aboral to the right. There is marked dilation of the intestine (asterisk) oral to the stricture (arrows) in both cats (A, B). The ultrasound beam has been orientated to demonstrate the discrepancy in the

diameter of the dilated oral and non-dilated aboral segments of bowel respectively, either side of the stricture. In (A) the stricture (arrows) is narrow and hypoechoic with complete loss of layering. In (B) the mucosa at the level of the stricture (arrows) is the focally hyperechoic and the tunica muscularis is thickened. In (C) a sharply marginated rectangular foreign body in the duodenum aboral to the stricture dilates and distorts the lumen. The bowel aboral to the foreign body is abnormally layered (arrowhead).

Supplementary material: video of Figure 1A demonstrating optimization of dilated oral bowel, stricture and collapsed aboral bowel in the same imaging plane, and video of Figure 1B demonstrating kinking of long axis of the bowel at the oral aspect (to the right of the image) of the stricture.

Figure 2. High-frequency (12MHz) linear ultrasonographic images of a long-segment stricture in a cat. (A) The image is orientated along the long axis of the intestine, oral is to the left, aboral to the right of the image. The transducer width is 38mm. There is moderate concentric wall thickening(3.9mm , calipers), overall the intestinal wall is hypoechoic in appearance and wall layering is abnormal. (B). Transverse image through the stricture. The intestinal lumen is obturated (arrow), the mucosa is mildly hyperechoic, the submucosa indistinct and the tunica muscularis(tm) thickened. Hyperechoic mesentery borders the stricture.

Figure. 3. Medium-frequency (8MHz) microconvex (A) and high-frequency (15MHz) linear (B) ultrasonographic images of an annular intestinal stricture. In both images the non-distended aboral jejunum is on the left of the images, oral is to the right. The intestinal wall changes at the level of the stricture are transmural. In (A) the stricture (arrows) is hypoechoic in appearance and the intestinal lumen cannot be recognized.

(B) Transducer length is 24mm. On the high-frequency image the approximate margins of the stricture are better recognized(arrows). Individual wall layers within the stricture segment are indistinct. In the centre of the stricture all layering is lost and the intestinal lumen is obliterated (dashed line). The submucosa (asterisks) is thickened and indistinct and difficult to distinguish from the marginally hyperechoic mucosa. The tunica muscularis(tm) is thickened. The dilated oral jejunum is not in the image plane.

Figure 4. Surgical specimen (A) and ultrasonographic images of an annular jejunal stricture placed in a water bath (B, C, D). This is the same cat as in Figure 3. Aboral to the left , oral to right in (A) and (B). (A) Note that it is not possible to advance forceps through the stenotic lumen. (B) Longitudinal high-frequency (15MHz) linear ultrasonographic image. The strictured segment measures approximately 10mm. Focal thickening and obliteration of the intestinal lumen is evident centrally. Wall layering is abnormal and is absent in the centre of the stricture (arrowheads). The hyperechoic submucosal(asterisk) layer bordering the stricture is indistinct. (C) Transverse image through the centre of the annular jejunal stricture (C, yellow line). The jejunum at the site of the stricture is reduced in diameter and misshapen. The intestinal lumen is obliterated and wall layering is completely lost. (D) Transverse image at the margin of the annular stricture (D,red line). Note the thickened hypoechoic tunica muscularis (tm) and broadened, ill-defined, submucosal layer (sm) and mucosal ulceration(arrow).

Figure 5. Intra-operative images of mid-jejunal annular strictures (A) and (B) in two cats. Oral to the right, aboral to the left on both images. Note the markedly dilated oral intestinal segment, focal “napkin ring” stricture (black arrow) and non-dilated aboral

bowel. The mesentery along the margin of the stricture in (B) is focally reactive and thickened (asterisk) consistent with fibrofatty change. Note in both cases the attenuation of the vasa recta of the terminal jejunal arcade at the level of the stricture (white arrows, enlarged images) compared to adjacent arcades.

Figure 6. Histological section of an annular stricture. The jejunal mucosa is lost due to ulceration (U) and is replaced by inflammation and fibrosis (F) causing narrowing of the intestinal lumen (L). The inflammation and fibrosis extend into the submucosa and, to a lesser extent, into the tunica muscularis (M). Haematoxylin and eosin stain, 100x magnification.

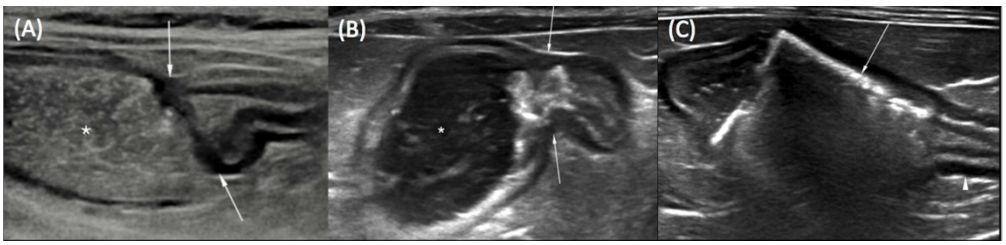
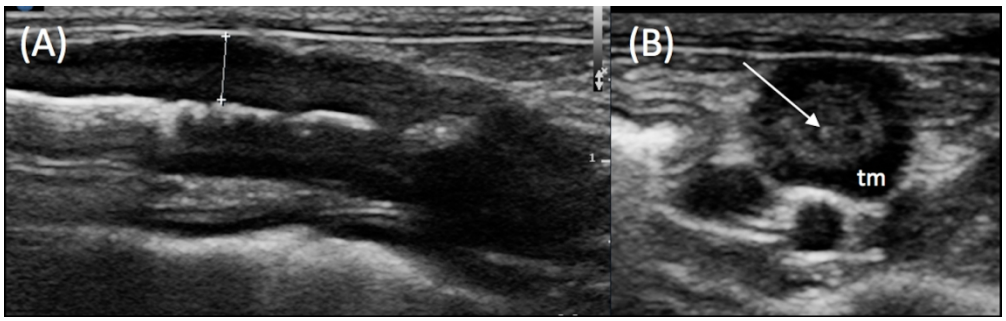


Figure 1. High-frequency(18MHz) linear ultrasonographic images of (A) an annular mid-jejunal stricture in a cat (A), and (B, C) an annular duodenal stricture and foreign body in a second cat. Oral is to the left of the image, aboral to the right. There is marked dilation of the intestine (asterisk) oral to the stricture (arrows) in both cats (A, B). The ultrasound beam has been orientated to demonstrate the discrepancy in the diameter of the dilated oral and non-dilated aboral segments of bowel respectively, either side of the stricture. In (A) the stricture (arrows) is narrow and hypoechoic with complete loss of layering. In (B) the mucosa at the level of the stricture (arrows) is the focally hyperechoic and the tunica muscularis is thickened. In (C) a sharply marginated rectangular foreign body in the duodenum aboral to the stricture dilates and distorts the lumen. The bowel aboral to the foreign body is abnormally layered (arrowhead). Supplementary material: video of Figure 1A demonstrating optimization of dilated oral bowel, stricture and collapsed aboral bowel in the same imaging plane, and video of Figure 1B demonstrating kinking of long axis of the bowel at the oral aspect (to the right of the image) of the stricture.



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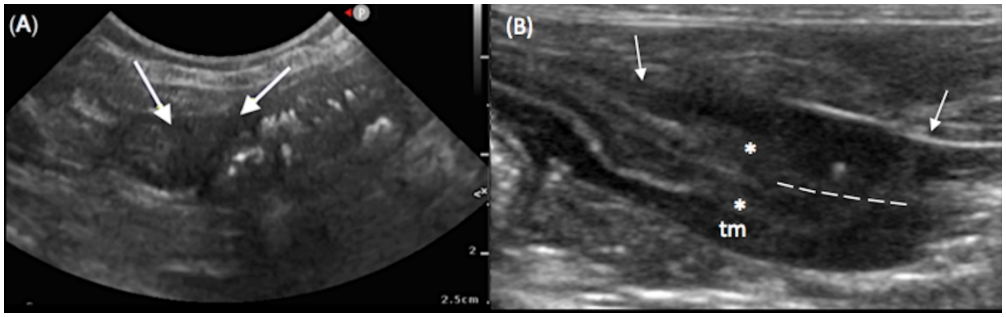

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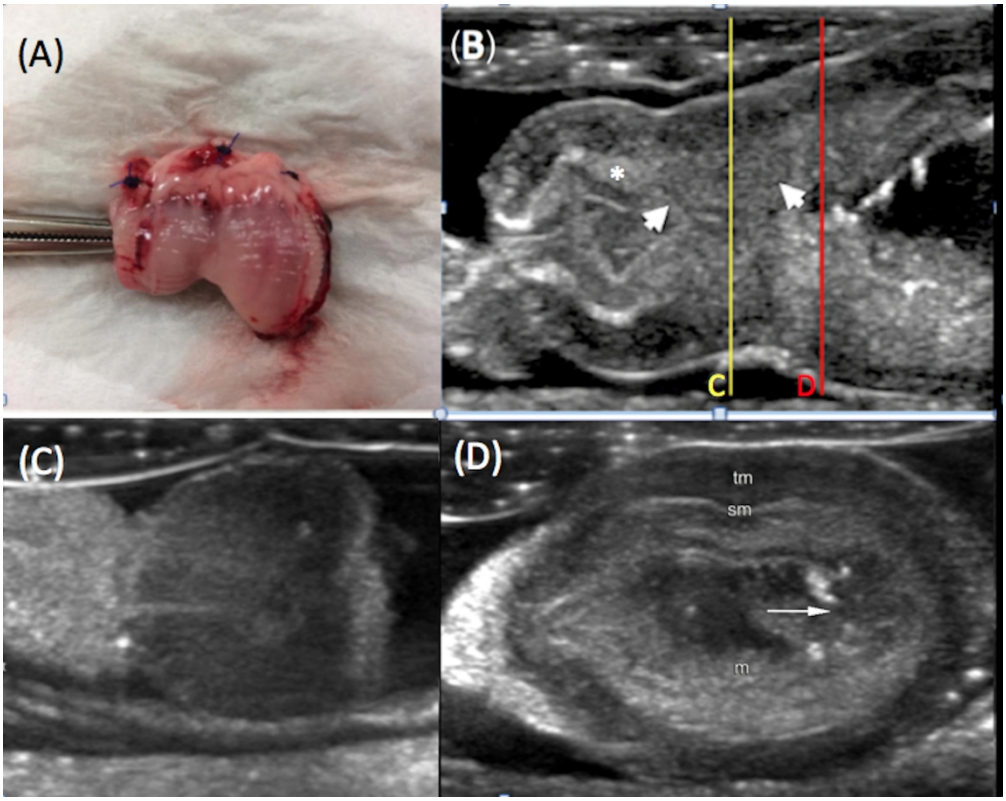

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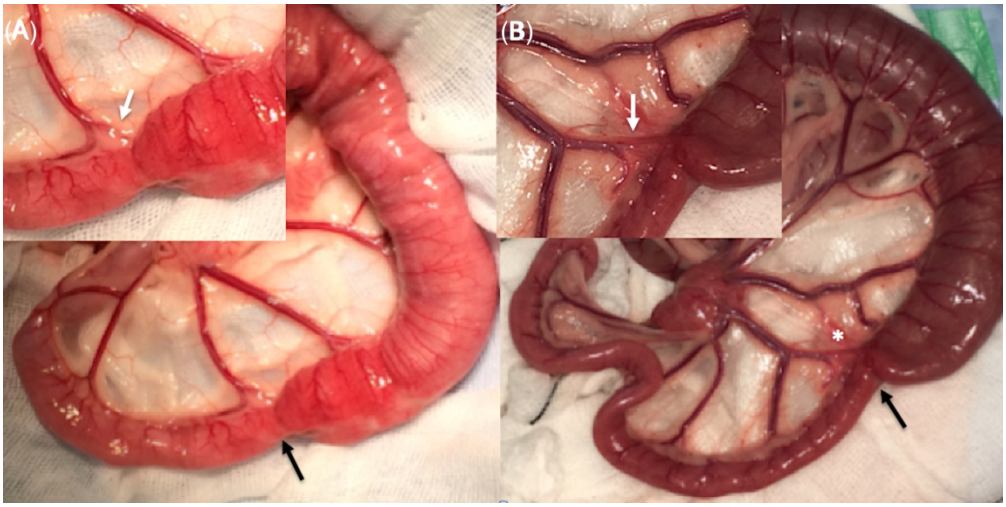

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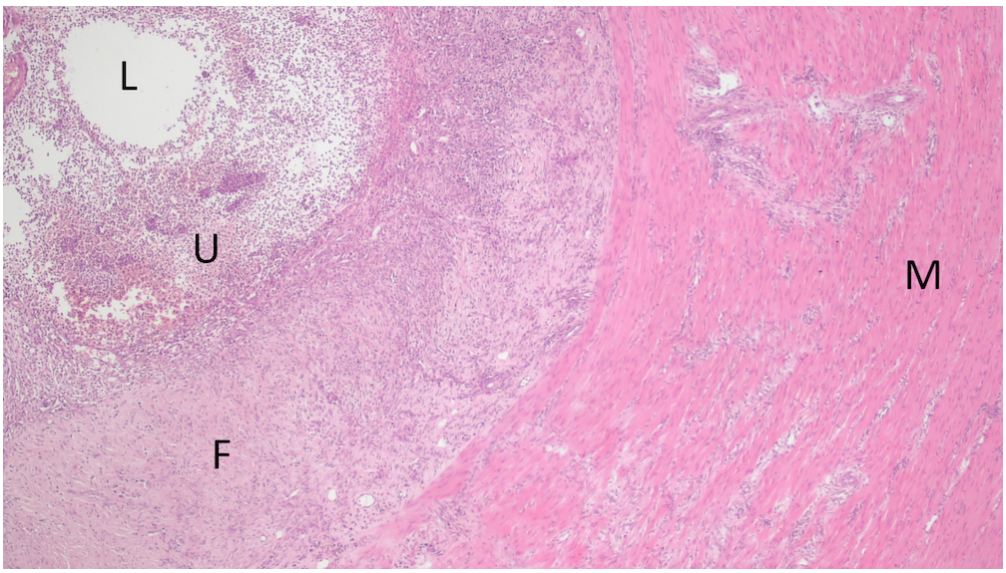

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84x47mm (300 x 300 DPI)