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Yetişkin Bir Kedide Kısa Kolon Sendromu ve Sonuçları

Short Colon Syndrome and Its Consequences in an Adult Cat

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ÖZ

Oniki yaşındaki kısırlaştırılmış erkek, kısa tüylü ev kedisi, üç aydır devam eden tenesmus, hematozezi, disşezi ve ishal şikayetleri ile hastanemize başvurdu. Laboratuvar muayenelerinde hiperkalsemi, hipofosfatemi, globulin konsantrasyonunda ve LDH enzim aktivitesinde artış ile yüksek gama-globulin konsantrasyonuna sahip monoklonal gammopati belirlendi. Kontrastlı radyografi bulgularına dayanarak kedide “kısa kolon” ve sekum agenezi tanısı konuldu. Tedavide, yüksek oranda sindirilebilir, düşük kalıntı içeren diyet, psyllium ve sülfasalazin uygulandı. Üç hafta içinde klinik belirtiler düzelterek kısa kolon başarıyla tedavi edildi. Bu olgu sunumunda kısa kolon ve sekum agenezis tanısı konulan kedinin palyatif tedavisi ve kısa dönem sonuçlarının paylaşılması amaçlanmıştır.

ABSTRACT

A 12-year-old castrated male domestic shorthair cat was presented with three months history of tenesmus, haematochezia, dyschezia and diarrhoea. Laboratory examinations revealed hypercalcemia, hypophosphatemia, increased globulin concentration and LDH enzyme activity, and monoclonal gammopathy with high gamma-globulin concentration. Based on contrast radiography findings, the cat was diagnosed with a “short colon” and cecal agenesis. Treatment involved a highly digestible, low-residue diet, psyllium, and sulfasalazine. Clinical signs improved within three weeks, and the short colon was successfully treated. This case report aims to share the palliative treatment and short-term outcomes of a cat diagnosed with a short colon and cecal agenesis.

INTRODUCTION

Short colon syndrome (SCS) in cats is characterized by congenital or acquired shortening of the colon, resulting in a straight configuration with absence of the transverse and ascending segments. Although historically considered rare, recent studies suggest that SCS may be more common than recognized due to wider use of abdominal ultrasonography and advanced imaging techniques.¹⁻³ Congenital gastrointestinal anomalies are uncommon in cats; however, intestinal malformations represent a substantial proportion of defects observed in pre-weaning kittens, accounting for 11-38% of reported congenital anomalies.^{2,3} Colonic abnormalities include atresia^{4,5}, stenosis⁶, duplication^{7,8}, and SCS¹, defined by agenesis of the caecum and proximal colon. Most cases are diagnosed before six months of age.⁹ Nevertheless, emerging evidence indicates that some cats remain asymptomatic for years, with clinical signs developing later secondary to inflammatory or neoplastic processes such as lymphoplasmacytic colitis, chronic enteropathy, or lymphoma.¹

Although SCS is often described as an iatrogenic condition following extensive intestinal resection,¹⁰ congenital agenesis may also underlie the disorder.¹¹ Increasing recognition of both short bowel and SCS suggests these conditions may be more prevalent than previously appreciated.^{1,9,12}

This report describes a 12-year-old cat with chronic diarrhoea, tenesmus, haematochezia, and dyschezia associated with agenesis of the cecum, ascending colon, and transverse colon. The objective is to document late-onset manifestation of congenital SCS and discuss potential mechanisms contributing to delayed clinical presentation.

DESCRIPTION of the CASE

A 12-year-old castrated male domestic shorthair cat was presented with a three-month history of tenesmus, haematochezia, dyschezia, and diarrhoea, which had worsened during the preceding three weeks. Intermittent vomiting of hairballs had

occurred over the previous 20 days. Previous treatments included deworming, dietary modification, and antibacterial therapy. The cat maintained a normal appetite and was fed on commercial dry food supplemented with a homemade diet. No prior gastrointestinal signs had been reported. Weight loss was noted only after onset of clinical signs.

On examination, the cat was underweight (estimated body condition score 4/9), with faecal staining of the perineal region and marked perianal inflammation. Abdominal palpation was largely unremarkable.

Complete blood count, urinalysis, and faecal examinations were within normal limits (Table 1). Serum biochemistry revealed hyperglobulinemia, elevated lactate dehydrogenase (LDH), hypercalcemia, and hypophosphatemia (Table 2). Total thyroxine (T4) concentration was normal (Table 1).¹³

Table 1. Complete blood count results of the case.

PARAMETER	RESULT	REFERENCE INTERVAL ¹³
White Blood Cells ($\times 10^9/L$)	12.30	6.00 – 20.00
Lymphocyte ($\times 10^9/L$)	5.10	1.00 – 7.00
Granulocyte ($\times 10^9/L$)	6.20	2.00 – 15.00
Monocyte ($\times 10^9/L$)	1.00	< 2.00
Lymphocyte (%)	41.50	12.00 – 45.00
Granulocyte (%)	50.10	35.00 – 85.00
Monocyte (%)	8.40	2.00 – 9.00
Red Blood Cell ($\times 10^{12}/L$)	7.49	5.00 – 12.00
Hemoglobin (g/dL)	11.90	9.00 – 15.00
Hematocrit (%)	35.70	28.00 – 49.00
Mean Cell Volume (fL)	47.70	39.00 – 52.00
Mean Corpuscular Hemoglobin (pg)	15.81	13.00 – 21.00
Mean Corpuscular Hemoglobin Concentration (g/dL)	33.30	30.00 – 38.00
Red Cell Distribution Width (%)	18.00	14.00 – 18.00
Platelet ($\times 10^9/L$)	234.00	100.00 – 514.00
Mean Platelet Volume (fL)	9.00	5.00 – 12.00

Given the hyperglobulinemia, serum protein electrophoresis was performed and demonstrated a monoclonal gammopathy with markedly increased gamma-globulin (2.92 g/dL).¹³ Faecal culture was negative for *Salmonella* and *Campylobacter spp.*, and point-of-care testing for *feline leukemia virus*, *feline immunodeficiency virus*, *feline coronavirus*, and *Giardia* was negative.

Survey radiographs were unremarkable. Abdominal ultrasonography revealed a gas-filled stomach and normal-appearing small intestines. The ileocolic junction was not visualized. Marked mural thickening (1.16 cm) with loss of wall layering was observed in the caudal descending colon and rectum, whereas the cranial descending colon was normal (2.9 mm thickness) (Figure 1). Differential diagnoses included

inflammatory bowel disease (IBD) and infiltrative neoplasia. Mechanical obstruction and pseudo-obstruction were excluded. Non-visualization of the ileocecal junction raised suspicion of short colon.

A barium sulphate enema (BaSO₄; 8mL/kg, R-X, Avixa, Istanbul, Türkiye) demonstrated a normally positioned descending colon but complete absence of the caecum, ascending and transverse colon (Figure 2). Contrast progressed cranially into the ileum and jejunum. The colonic mucosa appeared smooth; the small intestine exhibited irregular, fimbriated contours. These findings confirmed congenital agenesis of the caecum and proximal colon, with concurrent small intestinal enteropathy likely secondary to absence of the ileocolic junction.

Table 2. Biochemical and endocrinological laboratory results of the case.

TEST	RESULT	REFERENCE INTERVAL ¹³
Albumin (g/dL)	2.42	2.10 - 3.30
Albumin:Globulin Ratio (g/dL)	0.38	0.45 - 1.19
Alkaline Phosphatase (U/L)	56.34	25.00 - 93.00
Alanine Transaminase (U/L)	46.33	6.00 - 83.00
Amylase (U/L)	1.265.93	532.00 - 2008.00
Total Bilirubin (mg/dL)	<0.012	< 0.20
Blood Urea Nitrogen (mg/dL)	31.23	20.00 - 30.00
Calcium (mg/dL)	13.00	6.20-10.20
Creatine Kinase (U/L)	247.38	73.00 - 388.00
Globulin (g/dL)	6.35	2.60 – 5.10
Phosphorus (P)	4.00	4.50 - 8.10
Gamma Glutamyl Transferase (U/L)	<4.00	1.30 - 5.10
Cholesterol (mg/dL)	108.13	95.00 - 130.00
Creatinine (mg/dL)	1.56	0.80 - 1.80
Lactate Dehydrogenase (U/L)	351.38	63.00 - 273.00
Lipase (U/L)	23.36	0.00 - 83.00
Total Protein (g/dL)	8.77	5.40 - 7.80
Triglycerides (mg/dL)	47.98	25.00 - 133.00
Total Thyroxine (T ₄ ; nmol/L)	27.00	11.00 – 58.00

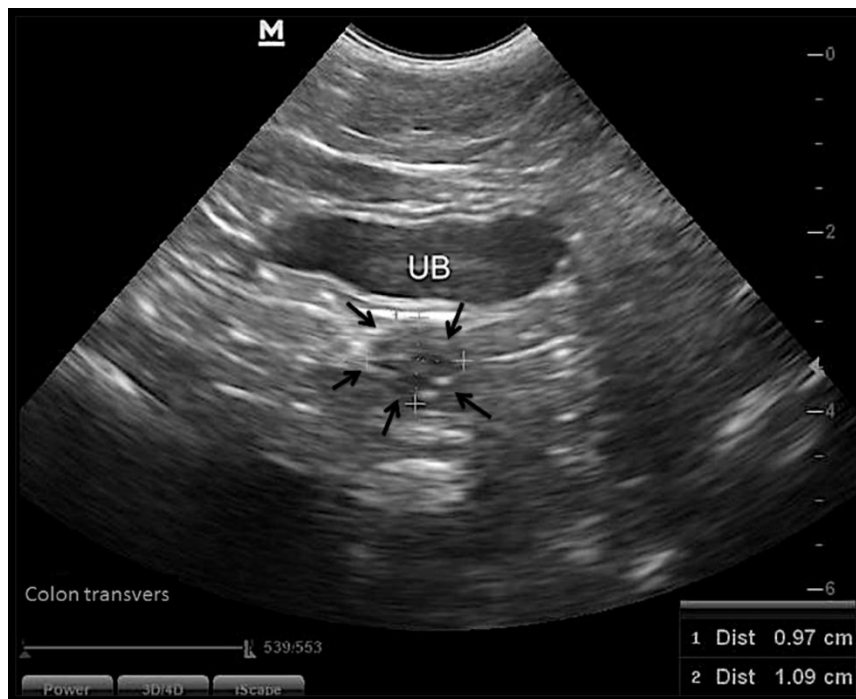


Figure 1. Transverse section of the caudal abdomen imaging the urinary bladder (UB) and the dorsally located caudal portion of the descending colon (surrounded by black arrows). Wall layers were not discernible and mural thickness was increased. More cranially transition to normal intestinal wall layering was visible.

Further diagnostic procedures requiring anaesthesia (biopsy or exploratory laparotomy) were declined due to age-related risk. Conservative management was therefore instituted.

Clinical signs were controlled with a highly digestible diet (Hill's Feline i/d), psyllium supplementation (one teaspoon daily), and sulfasalazine (20 mg/kg PO q12h for five days; adapted from canine colitis protocols).¹⁴

Within three weeks, diarrhoea markedly improved, body weight increased (estimated body condition score 5/9), and perianal inflammation resolved substantially. The owner did not present the cat again for a check-up because the clinical signs improved during treatment, therefore further follow-up was not possible.



Figure 2. A contrast enema was performed with barium sulphate. On the radiographs, the rectum and descending colon are moderately distended with contrast medium along the left abdominal wall followed by small intestinal loops lacking the remaining transverse and ascending colon as well as the cecum. While the colon is sharply delineated, the distal part of the jejunum appears to be spastic and shows a fimbriated inner surface containing small colonies of gas bubbles in the lumen being suggestive of inflammatory bowel disease.

DISCUSSION

SCS in cats, although previously considered rare, is increasingly recognized due to broader use of advanced imaging and improved clinical awareness. This condition is characterized by reduced colonic length, often with absence of the cecum, ascending colon, and transverse colon, resulting in direct communication between the ileum and descending colon. In our case, SCS with caecal agenesis was confirmed by contrast radiography, highlighting the diagnostic value of barium enema in visualizing colonic anatomy.

SCS may develop when a large portion (e.g., more than two-thirds) of the small intestine is absent, either due to surgical resection or congenital anomaly. The syndrome may be transient after intestinal resection because the remaining intestine can undergo adaptive hyperplasia.¹¹ Congenital agenesis of the caecum and proximal colon ("short colon") has been reported in adult cats.^{9,12,15}

To our knowledge, this is the first documented case in Cyprus, with a presentation similar to the cohort described by King et al.¹, in which the median age at diagnosis was also 12 years. In the present case, SCS and caecal agenesis were diagnosed by contrast radiography (Figure 2), which clearly delineated colonic anatomy.

Embryologically, the digestive tract originates as a simple tube that differentiates into foregut, midgut, and hindgut. The caudal midgut forms the cecum and ascending and transverse colon, whereas the hindgut develops into the descending colon and rectum.¹⁶ In this cat, failure of caudal midgut development likely resulted in direct ileocolic communication. Barium enema was instrumental in confirming this abnormality.

Schlesinger et al.⁹, reported absence of the caecum and proximal colon in a 12-year-old cat identified by exploratory laparotomy, with direct ileocolic communication on the left side of the abdomen. Lawler et al.², described two cats with SCS diagnosed radiographically, showing an abnormally short, straight colon and left-sided caecum. These cats developed diarrhoea at two and eight years of age. It is unlikely that diarrhoea was unnoticed in all cases; a more plausible explanation is that signs developed only after mild inflammatory bowel disease (IBD) occurred. Combined with reduced absorptive capacity of the shortened colon, this may have resulted in chronic diarrhoea.¹

In our patient, absence of clinical signs for 11.5 years suggests that diarrhoea, tenesmus, haematochezia, and dyschezia are not necessarily early manifestations of congenital colonic agenesis. IBD was included among differential diagnoses. Although clinical, radiographic, and ultrasonographic findings (Figure 1) were suggestive, definitive diagnosis requires intestinal biopsy and exclusion of other causes. Haematological findings are often unremarkable in IBD, as observed here. While hypoalbuminemia and hypoglobulinemia characterize protein-losing enteropathy and hypocholesterolaemia may indicate malabsorption, our patient exhibited hyperglobulinemia with normal cholesterol.

Monoclonal gammopathy, hyperglobulinemia, and hypercalcemia raised suspicion for an underlying lymphoproliferative disorder such as B-cell lymphoma or multiple myeloma.¹⁷ Although only one cat in King's study¹ had small cell lymphoma affecting both colon and ileum, chronic inflammation is a recognized contributor to oncogenesis. The concurrent monoclonal gammopathy and elevated LDH therefore strengthen the hypothesis that late-onset clinical signs were associated with a neoplastic process superimposed on a previously compensated congenital abnormality.

Serum protein electrophoresis confirmed a monoclonal gammopathy with increased gamma-globulin. Such findings are commonly associated with lymphoproliferative disorders including B-cell lymphoma or plasma cell neoplasia.¹⁸ However, no clinical evidence of malignancy-such as lymphadenopathy, organomegaly, or leukocyte

abnormalities-was detected. Elevated total calcium and decreased phosphorus raised concern for hypercalcemia of malignancy, often linked to lymphoma in cats, but ionised calcium was not measured, and no additional findings supported this diagnosis. Although these abnormalities suggested possible systemic pathology, the absence of hypoalbuminemia-related changes and the favourable response to palliative therapy were more consistent with a reactive or inflammatory process. Lack of follow-up testing, including repeat electrophoresis, remains a limitation in fully characterizing the underlying condition.^{18,19}

Elevated lactate dehydrogenase (LDH) activity (351 U/L) was also observed. LDH, present in most cell types, reflects cellular damage or increased turnover when elevated.²⁰ Although non-specific, in this context it may indicate neoplastic proliferation or chronic inflammation. Together with monoclonal gammopathy and hypercalcemia-both suggestive of lymphoproliferative disease-elevated LDH supports the possibility of occult malignancy. King et al.¹, reported small cell lymphoma in a subset of cats with SCS, emphasizing chronic gastrointestinal inflammation as a potential substrate for neoplastic transformation. It is therefore plausible that our patient's late-onset clinical signs, manifesting after 11.5 years of life, were triggered by development of a lymphoproliferative disorder in the context of a previously compensated congenital colonic anomaly. This case highlights the importance of considering secondary inflammatory or neoplastic processes when congenital anomalies present later in life. It also underscores the diagnostic value of imaging and the potential effectiveness of conservative management when invasive procedures are declined or contraindicated.

In conclusion, palliative treatment resulted in significant clinical improvement. A highly digestible, low-residue diet likely reduced colonic workload, psyllium improved stool consistency and motility, and sulfasalazine provided anti-inflammatory benefit. Although advanced imaging established the diagnosis, further invasive procedures were not pursued due to anaesthetic risk. This case illustrates that selected geriatric cats with congenital gastrointestinal abnormalities such as SCS may respond well to conservative management. Careful dietary control and ongoing monitoring remain essential for maintaining long-term quality of life.

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