Artykuł przeglądowy

Review

Megacolon in cats: Review of the current state of knowledge

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Received 18.04.2025 Accepted 11.06.2025

Kowalczyk P., Dolska M., Galanty M. Megacolon in cats: Review of the current state of knowledge

Summary

A review of the literature on the causes of the disease and the possibilities of conservative and surgical treatment was conducted. The authors set the goal of analyzing the algorithms of the procedure, including qualification for an appropriate treatment method, based on the current state of knowledge. An electronic database review, covering PubMed, CAB Abstracts and Google Scholar collections, was performed in October 2024. Data extracted included study participants, literature reviews, clinical case reports, outcome measurements and results of the above studies. Understanding the cause of disorders in defecation that may lead to the development of large colon syndrome is essential for selection of the correct treatment method. Pharmacological treatment of the initial stage of the disease produces good results, but the progressive nature of the disease may necessitate conversion to surgical treatment. Surgical treatment involves the removal of the obstruction to the passage of food content and, in the case of the idiopathic form, colectomy. Most publications on surgical treatment describe colectomy as the method of choice. This is due to the unsatisfactory effects of conservative treatment of the chronic form of the disease. It is estimated that 90% of cats after colectomy function well, and the risk of postoperative complications is on average 5%. Defecation normalizes within 6 to 12 weeks with a simultaneous increase in the frequency of defecation.

Keywords: constipation, obstipation, megacolon, cat

The term megacolon syndrome is used to describe a condition in which severe treatment-resistant constipation in cats leads to irreversible enlargement of the colon with accumulation of faecal masses and impaired bowel function (3). This problem affects mainly cats and is less common in dogs (12, 19). In dogs, hypertrophic large colon syndrome is due mainly to a bone-rich diet and low physical activity (21). In cats, three main causes of this disease are identified. The idiopathic form, which is the most frequent, accounts for approximately 62% of cases. The form secondary to anal/rectal stricture or pelvic canal narrowing is observed in approximately 23% of cases, while the form secondary to sacral nerve damage, spinal deformities or developmental anomalies of the spine makes up around 5% of all cases (Manx cats) of megacolon syndrome (33, 39, 42-44). Idiopathic megacolon occurs most commonly in obese middle-aged male cats, but can affect cats of any age (3, 31, 39). Dyschezia is a common clinical problem in cats. It initially presents

as constipation, characterized by the passage of small amounts of hard stool. When this process intensifies, and faeces remain in the intestine for a longer period of time, they become dehydrated, turning into faecal stones, which the cat is unable to pass. This condition is referred to as constipation (35). The diagnosis of the disease is based on clinical examination supplemented by imaging tests, including X-rays. It is helpful to determine the ratio of the maximum colon diameter (MCD) to the length of the 5th lumbar vertebra (L5L) (MCD:L5L). In healthy cats, this index (MCD:L5L) should be less than 1.28 (Fig. 1) (34). In most cases, dyschezia in cats manifesting as constipation can be treated conservatively. The most effective treatments include the administration of laxatives, prokinetic agents, stool softeners, enemas and dietary changes. The main aim of treatment is to minimise retention of digesta and to modify the diet to facilitate faecal passage. In the initial phase of the disease, conservative treatment can bring the desired results. In the long term,

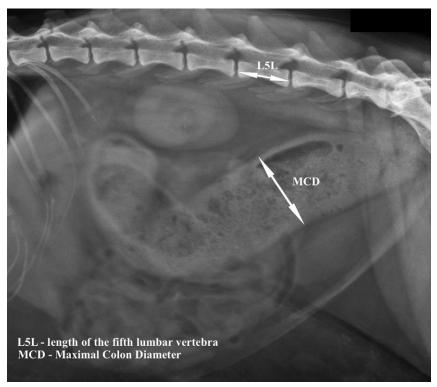


Fig. 1. Example of measurement of the length of the fifth lumbar vertebra and the maximum colon diameter to determine the MCD:L5L index

however, surgical intervention may be unavoidable (26). When pharmacological treatment is ineffective, chronic dyschezia requires surgical intervention. In some cases, this may result in a decision to perform euthanasia. One surgical treatment option for patients with large colon syndrome, including the idiopathic megacolon, is subtotal colectomy (13, 14, 26, 39). In addition to dyschezia, the most commonly observed signs include dehydration, weight loss, abdominal pain, and mesenteric lymphadenopathy (11).

Currently, there is a lack of consensus on the efficacy of subtotal colectomy with regard to complication rates, outcomes, and the need to remove the ileocecocolic junction (ICJ). The rationale for removing the ICJ is to reduce tension at the site of bowel anastomosis and minimize the risk of disease recurrence. It has been shown that the risk of serious postoperative complications, including patient death directly related to the treatment outcome, can occure in 9.9% to 14%, cases. Relapse was observed in approximately 32% of cases. Leaving ICJ has been found to reduce the risk of chronic untreatable diarrhoea secondary to small bowel bacterial overgrowth (13, 26, 27, 31).

Pathophysiology

Currently, there are few well-described cases of canine and feline congenital megacolon. This condition in cats has been described in clinical case reports. Histological examination of colonic scrapings showed a reduced number of ganglia within the Auerbach's plexus and a small number of ganglia within the Meissner's plexus. Immunohistochemical examina-

tion of substance P, neurofilaments and synaptophysin was also performed, with results corresponding to haematoxylin and eosin staining. These findings were consistent with hypoganglionosis in the colon that occurs in the congenital form of congenital aganglionic megacolon, which in humans is called Hirschprung's disease (25, 27, 43). Due to the low incidence of the congenital megacolon, the condition is divided into acquired (secondary) and idiopathic (primary) forms (20, 39). Secondary megacolon may also be referred to as hypertrophic megacolon and may show potential for reversal of the lesions if treated early (21, 40, 43).

Idiopathic megacolon

It is the most common form of feline megacolon, but in most cases, the cause has not been determined. It usually affects cats of middle age, particularly Siamese, domestic short- and long-haired breeds (5, 17, 39). Cases with idiopathic megacolon may not show signs of physical or

functional obstruction. The main role in the development of idiopathic megacolon is believed to be a disruption of innervation, neuromuscular junction activity, or degeneration of smooth muscle nerves in the colon, which consequently lead to peristalsis disorders causing constipation. However, aside from one documented case in cats, no deficiency or disturbances in intestinal innervation have been confirmed (25). Disturbances in the activation of smooth muscle myofilaments appear to be the primary cause of dysfunction in the longitudinal and circular fibres of these muscles. An in vitro comparison of smooth muscle myofilaments from healthy cats and cats with megacolon revealed a decreased sensitivity to neurotransmitters (acetylcholine, substance P and cholecystokinin), the use of potassium chloride for cell membrane depolarisation and the effect of electrical field stimulation on isometric stress in the latter group (41). It has been demonstrated that changes in the thickness of the colonic muscle layer and myocyte viability are dependent on the duration of the disease process. In cats in which it lasted longer than 6 months, there was an increase in the thickness of the colonic muscle layer, an increase in the number of dead myocytes, a decrease in ganglion cells and no response to conservative treatment, compared to animals whose disease lasted less than 6 months (1).

Secondary megacolon

Secondary megacolon can be caused by a number of mechanical or neurological factors that result in the blockage of faecal passage. The most common causes include pelvic fractures leading to the narrowing of the pelvic canal, tumours of the colon or within the pelvic canal, anal or rectal stricture, as well as atresia and innervation trauma. It is believed that pelvic fractures in cats account for 20% of all fractures in this species (30). Although many of these cases can be treated conservatively with a satisfactory clinical outcome, in some patients the pelvic canal becomes narrower, which can lead to chronic dyschezia and a subsequent development of megacolon (9, 22). Other factors that increase the risk of dyschezia are tumours within the pelvic canal, tumours involving the large bowel, as well as diaphragmatic hernia in the pelvic area or rectal diverticulosis. This condition can cause an increase in wall thickness and lead to the formation of hypertrophic megacolon (3, 12, 37). There have been reports of cases in which faecal elimination disturbances were caused by the obstruction of the distal colon segment secondary to surgical interventions. In sterilised cats, post-operative adhesions at the pelvic outlet can lead to a tightening and narrowing of the descending colon which consequently stops the passage of digesta (18, 24).

Neurological disorders

Dyschezia in cats can result from trauma or the presence of congenital spinal anomalies, as seen in Manx cats (8). Not all spinal deformities can contribute to colonic dysfunction. Non-traumatic deformities of the lumbosacral spine are observed in 29.74% of cats. The most commonly observed congenital abnormalities of the spine include six lumbar vertebrae, sacralisation and lumbalisation. Acquired abnormalities in the lumbosacral segment may present with bone spurs, narrowing of the intervertebral space, deforming spondylosis and lumbosacral degeneration. It has also been demonstrated that cats with abnormalities in the vertebrae of the lumbosacral region have an increased risk of colorectal problems. Both acquired and congenital abnormalities observed in the lumbosacral spinal region may contribute to the occurrence of feline colorectal disorders (16, 33).

Other causes

Another cause of megacolon is anal atresia (10, 12). Prolonged administration of diuretics, antihistamines, anticholonergics, iron supplements and other substances may also promote constipation that eventually contributes to the development of feline megacolon. An increased risk of constipation is associated with collateral diseases leading to dehydration. These include chronic kidney disease (CKD), diabetes mellitus and hyperthyroidism (2, 3, 12, 32, 36). The sex of the cat has been shown to have no effect on the risk of feline megacolon. In their study, Benjiamin et al. found that constipation is more common around the age of 10 years. Dry food appears to have no effect on the incidence of feline megacolon. Another indicator

considered in assessing the risk of megacolon was the Body Condition Score (BCS). It was noted, however, that both control and experimental groups included cats in all body conditions. After analysing the data, it was concluded that body weight did not influence the occurrence of megacolon. The study showed that cats with a history of constipation are 20 times more likely to experience further episodes of constipation and 3.8 times more likely to develop chronic kidney disease (CKD). Diabetes mellitus, IBD or small-cell lymphoma may or may not promote the development of feline megacolon. Patients with constipation may also have abnormal gasometry, e.g. ionised calcium levels (2). Feline megacolon can also be related to behavioural disorders caused by environmental factors, such as a dirty litter tray, eating foreign bodies, eating bones or excessive licking of the coat. Endocrine disorders, such as hypothyroidism and hypercalcaemia, are directly linked to increased water absorption from digesta, which increases the risk of hardening of faecal masses, resulting in constipation (7, 23, 35).

Treatment methods

Treatment strategies can be divided into conservative and surgical methods, and the selection of the method should be based on the accurate diagnosis of the underlying cause of feline megacolon. Some patients respond well to conservative treatment, but surgery should be considered (13, 16, 18, 20, 21, 26, 27, 30, 31, 40, 42, 43). Both idiopathic and secondary forms caused by the presence of a tumour blocking the pelvic outlet, a non-cancerous proliferative lesion (phytiosis or other granulomatous infections) or another obstacle to faecal transport and defecation, may require surgery regardless of the cause (45). The decision-making process regarding the treatment method should be correlated with clinical examination. Cats with autonomic nervous system failure (dysautonomia) will show other signs in addition to constipation, such as urinary and faecal incontinence, signs associated with giant oesophagus, pupil dilation, prolapsed gland of the nictitating membrane or bradycardia. Clinical examination of a patient with obstipation or constipation, with special emphasis on the per rectum examination, should be performed under sedation or general anaesthesia. The per rectum examination helps identify mechanical causes of constipation in cats, such as improperly healed pelvic fractures, foreign bodies, anal diverticula, narrowing, inflammation, neoplasm or diaphragmatic (perineal) hernia in the pelvic area. The clinical examination should also include a neurological assessment, covering the function of the spinal cord in the tail region. Although there is no direct correlation with relevant laboratory parameters, cats with constipation and megacolon should undergo blood tests (including full blood count, biochemistry, thyroid hormones) and urinalysis. Imaging examinations,

such as X-rays or CT scans, should also be performed to exclude spinal abnormalities, pelvic fractures or radiopaque foreign bodies. Additional examinations, such as ultrasound, endoscopy and contrast imaging, as well as cerebrospinal fluid examination, can also be considered in the diagnosis of constipation in cats. In the case of an aganglionic megacolon, anorectal manometry and colon biopsy may be necessary (37). Some of the diagnostic procedures mentioned above require sedation or general anaesthesia.

Conservative treatment

An attempt at conservative treatment should always be considered. Initial dyschezia in the form of constipation may not require medical treatment, but only hydration of the patient. This is achieved through systemic rehydration with parenteral fluid therapy. Subcutaneous fluid administration performed at home, feeding canned food, offering meat broths or adding water or broth to food are also useful in patient rehydration. Conservative treatment also includes administration of osmotic laxatives, stool softeners, enemas and manual stool evacuation. The removal of lingering faecal masses reduces the toxic and mechanical impact of these masses on the intestinal wall. Manual removal of faecal masses is carried out under sedation so as to evacuate stool completely and reduce the risk of iatrogenic injury. In some cases, hospitalisation is recommended during the initial treatment period.

Auxiliary substances for the removal of faecal masses used in the treatment of constipation include laxatives and stool softeners, e.g. dioctyl sodium sulfosuccinate (DSS) or Bisacodyl (whose laxative effect consists in the stimulation of epithelial secretion). In the case of Bisacodyl, prolonged use can lead to mucosal neuronal damage. Glycerine suppositories for children can also be helpful in treating constipation. The enema solution consists of warm tap water mixed, for example, with DSS, mineral oil or lactulose. It can be administered with a soft rubber catheter and a lubricant. DSS increases the absorption of mineral oil through the mucosa, and therefore the two substances should not be combined in an enema solution. Sodium phosphate enemas should be avoided in cats because of the risk of electrolyte imbalances (hyponatraemia, hyperphosphatemia and hypocalcaemia). Given the potential neurotoxicity of hexachlorophene, attention should be paid to the composition of soaps used to prepare enema solutions. Enemas should be performed slowly, which reduces the risk of vomiting, bowel perforation and insufficient stool softening. Due to the risk of vomiting, the patient should be intubated before the enema (29). A therapeutic diet for the treatment of constipation should include foods high in fibre. Insoluble (poorly fermentable) fibre, e.g. from wheat bran or cereal grains, normalises colonic motility by expanding its lumen, increasing water content and thus diluting toxins, such as bile acids, ammonia and ingested toxins. It also speeds up the gastrointestinal passage, while increasing the frequency of defecation. This facilitates the stimulation of smooth muscle contractions and defecation.

Prokinetic agents include cisapride (a substituted piperidinyl) (41). Studies have shown that other prokinetic agents, such as metoclopramide, have impact on the proximal gastrointestinal tract. Therefore, metoclopramide should not be used in the treatment of megacolon. Other prokinetic agents with intestinal motility-stimulating effects include prucalopride and tegaserod (28, 29, 38).

Surgical treatment

The scope of the surgical procedure for idiopathic megacolon varies according to the cause of the disease, either addressing the colon itself or removing the underlying cause of the hypertrophic colon, depending on the location of the lesion. Defecation may be obstructed by conditions that involve the pelvic canal, such as neoplasms, abnormal healing of pelvic fractures or postoperative adhesions causing pressure on the colon (3, 9, 12, 18, 22, 24, 30, 37). Patients with hypertrophic megacolon should be referred for a surgical intervention to remove the obstruction preventing the proper faeces passage. Surgical treatment may consist of removal of the proliferative lesion, widening of the pelvic canal (in the case of abnormal pelvic fracture fusion), release of adhesions that compress the colon, surgical treatment of anal atresia or other procedures, including removal of pseudomycetoma lesions (10, 18, 22, 24, 30, 45).

However, a large number of publications on the treatment of idiopathic megacolon focus on subtotal colectomy procedures. Due to the low efficacy of the pharmacological treatment of the chronic forms (e.g. with cisapride), surgical treatment is regarded as the preferred approach.

When both perineal hernia and megacolon are present, colectomy should be performed before herniorrhaphy, as it has been noted that colectomy alone often results in resolution of clinical signs (40). Performing enemas prior to colectomy is not advisable, because of the increased risk of intraoperative contamination of the peritoneal cavity by liquid digesta. Faecal masses should be removed from the anal area and anal canal with a finger. This procedure makes it easier to pass faeces in the post-operative period. Antibiotic prophylaxis includes the administration of cefazolin at a dose of 20 mg/kg body weight at the induction of anaesthesia and continuing for 24 hours. During surgery, the entire length of the dilated bowel is removed. The resection site ends distally in the ileum for subtotal colectomy or 2 to 4 cm distal to the caecum for subtotal colectomy, and as far distally as possible to enable the formation of an anastomosis, i.e. 2 cm upward from the edge of



Fig. 2. Colectomy surgery. Intraoperative view. Colon emptied of faeces. Ligation of mesenteric artery branches (arrows)

the pubic symphysis. Some authors believe that a total colectomy is easier to perform (3). Patients with a history of pelvic fracture may develop adhesions in the pelvic inlet region, making surgical access difficult (26). To facilitate intestinal anastomosis, the bowels should be emptied of hard faecal masses. When performing the procedure, it is important to carefully ligate blood vessels without damaging the mesenteric artery (Fig. 2). In total colectomy, there are difficulties with bowel anastomosis due to the large difference in diameter. In this scenario, an oblique incision of the ileum on the antimesenteric side helps align the ends of the anastomosis. It is believed that total or subtotal colectomy in patients unresponsive to conservative treatment has a satisfactory therapeutic outcome (4, 5). The small intestine is capable of adapting to absorb water and increase its diameter (5). Complications following this procedure are rare. They may include the narrowing of the enterocolostomy site (1 out of 150 cases). Treatment consists of dilating the stenosis site with a balloon catheter (35). Other complications include abscess at the incision site, death due to peritonitis or death from unknown causes (1 out of 150 cases). Persistent diarrhoea is observed in 1 out of 75 cases and is thought to be related to an overgrowth of the small intestinal bacterial flora. Some patients can be successfully treated with metronidazole. To prevent bacterial overgrowth, some authors also recommend preservation of the ileocecal valve (3, 5). Constipation recurrence was observed in 3 out of 38 cases. It is assumed that 90% of cats function normally after this procedure without long-term complications. The overall complication rate is less than 5% (26). In most patients, bowel movements normalize within 6 to 12 weeks after surgery, and soft, formed stools are observed, but an increased frequency of defecation has been noted (2-6) times a day) (3). Occasional constipation can usually be successfully treated conservatively (5).

Conclusion

Megacolon syndrome, frequently observed in cats, is often idiopathic and seems to arise from improper development of the colon. However, it should be remembered that cats, as outdoor animals, may suffer traumas often leading to pelvic fractures, and that abnormally healed pelvises lead to a hypertrophic form of this condition. Conservative treatment of the initial stage of the disease produces satisfactory results. but veterinarians should inform pet owners about the early signs indicating an abnormal passage of faeces and defecation. Proper management of the patient, including a balanced diet and adequate hydration, may help reduce the incidence of feline megacolon. In the advanced stage of the disease, surgical treatment involving the elimination of the obstructing factor and colectomy is crucial for achieving therapeutic success. The long-term results of patients treated surgically confirm a satisfactory clinical outcome.

The ultimate success in the treatment of megacolon in cats depends on an accurate diagnosis that determines the direct cause of the condition.

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