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Case report

CANINE INTESTINAL LYMPHANGIECTASIA CONCOMITANT WITH RENAL CELL CARCINOMA

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The etiology of dilation of lymphatic vessels, termed as intestinal lymphangiectasia, remains unknown. In most cases, it occurs secondary to other pathologic conditions such as gastrointestinal neoplasms. However, only a few cases of canine intestinal lymphangiectasia concurrent with non-gastrointestinal neoplasms have been reported so far. Moreover, the correlation between intestinal lymphangiectasia and non-gastrointestinal neoplasms has not been discussed in any other literature. In this study, we report a rare case of intestinal lymphangiectasia concomitant with renal cell carcinoma in an 11 year old female mixed Maltese, suggesting that non-gastrointestinal neoplasms could be associated with the development of intestinal lymphangiectasia. On gross observation, the small intestine was irregularly swollen presenting an accordion like shape. Microscopic examination revealed prominent dilatation of the lymphatic vessels, especially, within the submucosa and muscularis layer. The lacteals within the villi were dilated and presented "club-shaped" tips. The carcinoma might trigger intestinal lymphangiectasia by compressing the main lymphatic vessels or the cisterna chyli, subsequently increasing the pressure of the lymphatic vessels in the gastrointestinal tract. Moreover, metastasis of the carcinoma to the gastrointestinal tract could induce intestinal lymphangiectasia. Thus, the occurrence of intestinal lymphangiectasia must be considered when an abdominal neoplasm is located around major lymphatic vessels.

Keywords: Intestinal lymphangiectasia; renal cell carcinoma; lymph drainage; dog

INTRODUCTION

Intestinal lymphangiectasia (IL) refers to the dilation of lymphatic vessels in the gastrointestinal tract. It is often observed in Yorkshire Terriers, Scottish Terriers, Lundehunds, and soft-coated Wheaten Terriers [1-4]. IL leads to the loss of lymphatic fluid, which is enriched with proteins, lipids, lymphocytes, immunoglobulins, and electrolytes and is often associated with protein-losing enteropathy. Thus, IL results in progressive weight loss, malnutrition, lethargy, immunodeficiency, and organ malfunction [4,5].

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IL represents clinicopathological abnormalities such as hypoalbuminemia, panhypoproteinemia, hypocalcemia, hypocholesterolemia, hypocobalaminemia, and lymphopenia. However, these abnormalities are not specific for IL because they are observed in other conditions, such as gastroenteritis, Crohn's disease, liver failure, hepatic cirrhosis, pericarditis, protein-losing nephropathy, and nephrotic syndrome [1,6,7]. Thus, the presence of these biochemical abnormalities indicates the need for further examinations in order to reach a definitive diagnosis. Ultrasonography and endoscopy are suggested as non-invasive diagnostic methods for IL. Dogs with IL present loss of layering, thickening, and edema of the intestinal wall on ultrasound imaging [8]. The endoscopic findings of IL include white spots at the top of the villi, giving it a "snakeskin appearance" [9]. However, it represents only 68 % of the sensitivity and 48 % of the specificity, suggesting that it could be used as a supportive diagnostic method for IL along with laboratory abnormalities [10].

Despite the advantages of the aforementioned diagnostic methods, which include non-invasiveness, low risk of complications after examination, and relatively low costs, histopathological observation of the full-thickness of the intestine has been the golden standard for the diagnosis of IL owing to its high sensitivity and specificity. Microscopically, IL is characterized by a marked dilation of lymphatic vessels in the submucosa, muscularis, and serosa layers of the intestine, and the dilation of lacteals in the mucosa layer. Additionally, marked infiltration of inflammatory cells is observed adjacent to the lymphatic vessels. The inflammatory cells are mainly composed of lymphocytes, plasmacytes, epitheloid and foamy macrophages, and neutrophils; multinucleated giant cells are occasionally observed [4].

Although the presence of IL has been associated with other pathological conditions, its etiology remains unknown. Primary IL is rarely reported in humans and dogs. It occurs during childhood, generally before 3 years of age, and in young adults. Genetic defects are thought to be associated with the development of primary IL, and gene expression levels associated with lymphangiogenesis, such as vascular endothelial growth factor receptor 3 (VEGFR3), VEGF-C, and D, are altered in humans with primary IL [11].

However, in most cases, IL occurs secondary to other pathological conditions. It is mainly associated with an increase in pressure in the lymphatic vessels of the gastrointestinal tract. Most pathological conditions that accompany an increase in lymphatic pressure induce IL. Inflammation in the lymphatic vessels can trigger this disease. Additionally, portal hypertension due to hepatic cirrhosis, pericarditis, and right-sided heart failure increases the pressure in the lymphatic vessels and finally leads to IL. Similarly, gastrointestinal neoplasms obstruct lymphatic drainage in the gastrointestinal tracts thereby resulting in IL.

Neoplasms in the intestine or mesentery are known to cause IL [12-14]. However, only a few cases of IL associated with non-intestinal neoplasms have been reported so far [4]; moreover, to the best of our knowledge, the correlation between IL and

non-intestinal neoplasms has not been discussed in the literature. In this study, we report a rare case of IL with renal cell carcinoma (RCC) in a dog, which indicated that non-intestinal abdominal neoplasms could be associated with the development of the disease.

CASE PRESENTATION

An 11-year-old female mixed Maltese was brought to an animal hospital with symptoms of anorexia, vomiting, and weight loss. She had a history of pyometra and gallbladder myxoma. X-ray examination revealed the presence of a mass in the left kidney, which was surgically removed. During surgery, the small intestines were found to be flaccid and diffusely dilated. Parts of the dilated lesions and the mass collected from the kidney were submitted to the Laboratory of Veterinary Pathology at the Kyungpook National University. The tissues were routinely processed and embedded in paraffin, cut into sections (thickness, 6 μm), and stained with hematoxylin and eosin. In the serum biochemistry analysis, the levels of most proteins, including albumin, calcium, and cholesterol, were within normal ranges. However, an increase in the level of gamma-glutamyl transferase (GGT; 42 U/L; reference range, 0-7 U/L) and decreases in the levels of total bilirubin (<0.1 mg/dl; reference range, 0-0.9 mg/dl) and amylase (413 U/L; reference range, 500-1500 U/L) were observed. The increase in GGT, in this case, might have been caused by the obstruction of the biliary ducts due to the myxoma in the gallbladder.

On gross observation, the small intestine was irregularly swollen and accordion-shaped (Figure 1A). On the cut surface, the wall of the small intestine was irregularly thickened and the lumen was narrow (Figure 1B). Microscopic examination revealed dilatation of the lymphatic vessels within the submucosa and muscularis layer and the lacteals within the villi. Based on these findings, the case was diagnosed as an IL. Among the intestinal layers, the dilation of lymph vessels was particularly remarkable in the muscularis layer. The dilated vessels were filled with eosinophilic protein-rich lymph.

According to the histopathological standards of gastrointestinal disease of the World Small Animal Veterinary Association (WSAVA), IL, so-called lymph dilation, is graded as mild, moderate, and marked lacteal dilation [15]. In mild lacteal dilation, the central lacteals form 50% of the width of villous lamina propria; moderate lacteal dilation indicates central lacteal ballooning which represents 75% of the width of the villous lamina propria; and in marked lacteal dilation, the central lacteals represent 100% of the width of the villous lamina propria [15]. Based on these standards, this dog was diagnosed with moderate lacteal dilation, and some villi presented "club-shaped" tips (Figure 1C-1F). Hipogranulomatous lymphangitis or edema were not observed. Mild infiltration of inflammatory cells was observed, and the most infiltrated cells were lymphocytes and plasmacytes.

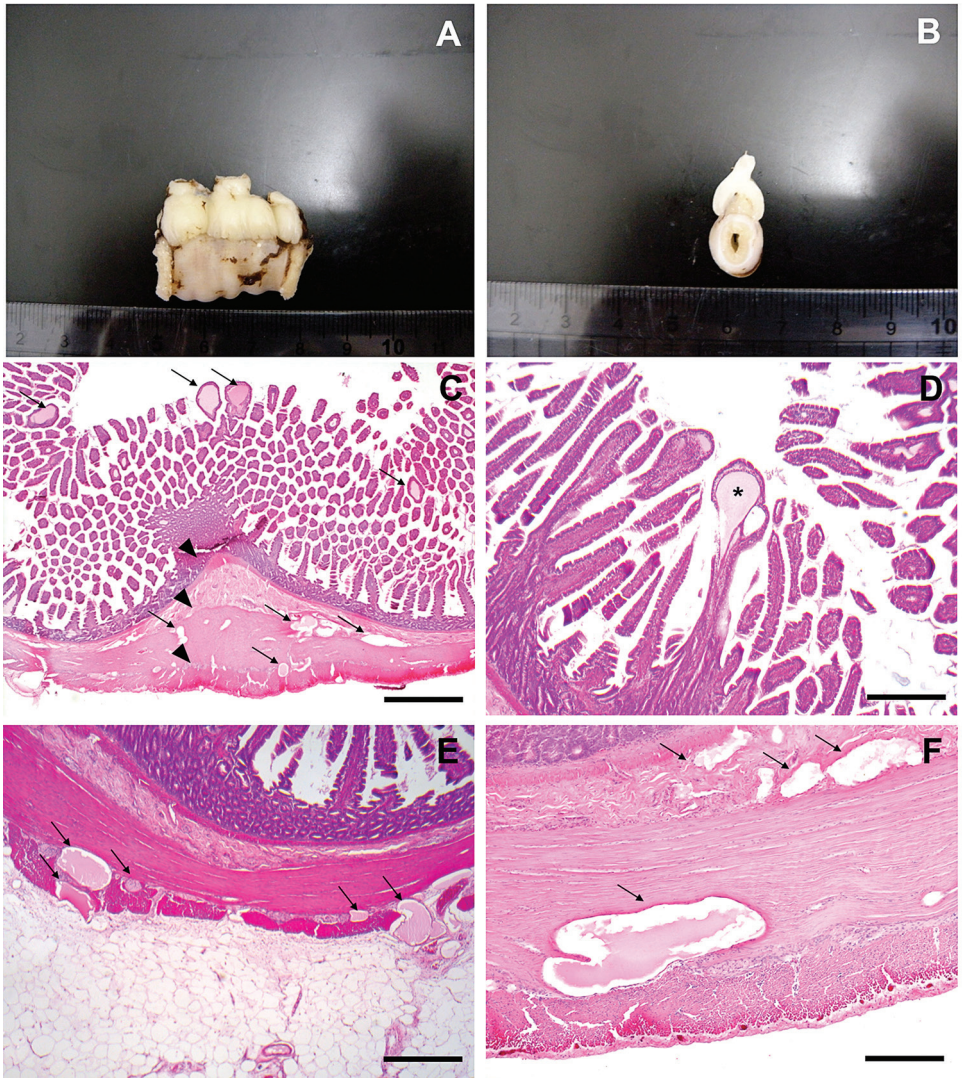

Figure 1. Macroscopic and microscopic findings in intestinal lymphangiectasia (IL). **(A)** Gross findings of lymphangiectasia. The mesentery was swollen, irregular, and accordion-shaped. **(B)** The transverse section of the small intestine with the IL. The intestinal walls were thickened and the lumen was narrowed. **(C)** The lymphatic vessels in the mucosa, submucosa, and muscularis were dilated and filled with eosinophilic lactic fluids (arrows). Some parts of the wall of the small intestine were thickened (arrowheads). Scale bar = 1000 μm **(D)** Distension at the tips of the villi ("club-shaped"; asterisk). Scale bar = 200 μm **(E)** Remarkable dilation of lymphatic vessels in the muscularis layer (arrows). Scale bar = 200 μm **(F)** Mild inflammation. Scale bar = 100 μm. Hematoxylin and eosin (H&E) staining.

The mass of the kidney was large enough to occupy most of the abdomen. It was located in the medulla and cortex of the kidney and protruded out toward the center of the abdomen. The tumor was not encapsulated, but well-circumscribed (Figure

2A). The neoplastic cells were arranged in a tubular pattern with a delicate collagen septum, whereas some areas of the tumor appeared as a solid mass. The mass was composed of epithelial cells with large and round nuclei and abundant eosinophilic cytoplasm. Mitotic figures were often observed (Figure 2B). Based on these findings, this tumor was diagnosed as a RCC. The dog was euthanized immediately after the surgery because of complications and poor prognosis.

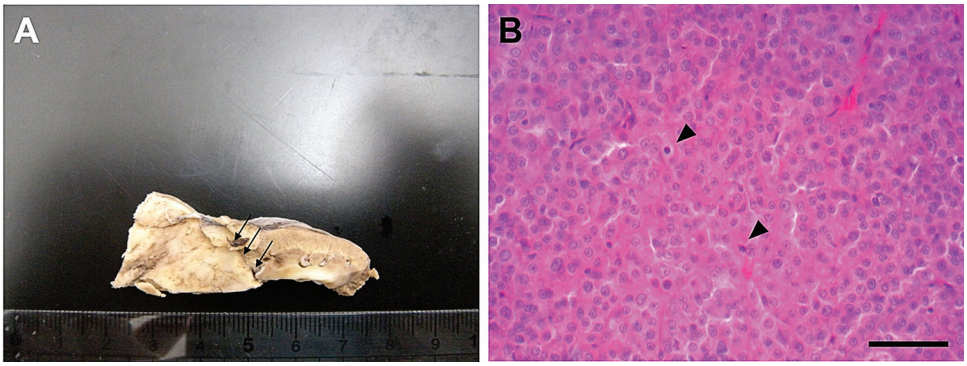

Figure 2. Macroscopic and microscopic findings of renal cell carcinoma (RCC). **(A)** Gross findings. The margin of the mass was distinguishable from the adjacent kidney (arrows). **(B)** Microscopic findings. The neoplastic cells were arranged in a tubular pattern and surrounded by thin collagen fibers. The cells had abundant cytoplasm with round nuclei. Mitotic figures were frequently observed (arrowheads). Scale bar = 50 μm. H&E staining.

DISCUSSION

The etiology of IL is not fully understood; however, it is known that IL is mainly caused by an increase in pressure in the lymphatic vessels, which may be attributed to conditions such as inflammation, portal hypertension, and gastrointestinal neoplasia. In the current case study, IL was observed concurrently with RCC. Unlike gastrointestinal neoplasms, which directly obstruct intestinal lymphatic vessels, the RCC was physically distant from the IL lesion.

The etiology of IL can be complex and related to several pathological conditions at the same time. Therefore, the possible causes of IL were thoroughly considered in the present case study. Gastrointestinal inflammation is one of the well-known causes of IL. The severe infiltration of inflammatory cells in lymphatic vessels disturbs the lymphatic flow. IL can occur secondary to inflammation and be resolved after treating the inflammation with corticosteroids [16]. Most of the IL lesions present with signs of inflammation. In one study, 15 out of 17 dogs with IL presented with varying degrees of had inflammation.

In the current case report, only a mild infiltration of inflammatory cells was observed in the lymphatic vessels and intestinal wall. Thus, the inflammatory cells in the lesions were probably not sufficient to disturb the lymphatic flows. It is difficult to discern the

causative role of inflammation in IL because it could occur as a result of the disease. Lymph fluid could leak from the dilated lymphatic vessels following the development of IL. This could be followed by the infiltration of inflammatory cells around the IL lesion. The infiltrated inflammatory cells, including epithelioid cells and multinucleated giant cells, often form lipogranulomatous lymphangitis around the dilated lymphatic vessels, in response to the chronic leakage of lipid-laden chyle [17]. Therefore, it is difficult to determine the exact role of inflammation in IL.

In one study, 15 out of 17 dogs with IL presented with mild to severe inflammation, while the remaining 2 dogs did not have any inflammation; 4 out of 15 dogs with severe inflammation had mild IL, whereas 1 out of the 2 dogs without inflammation had severe IL [4]. IL induced by inflammation would be expected to be associated with severe inflammation, which is required to disturb or block lymphatic vessels. On the other hand, ILs that are attributed to pathological conditions other than inflammation might be associated with varying degrees of inflammation, depending on the amount and duration of lymph leakage around the lymphatic vessels. Thus, the severity of inflammation is not correlated to or predictive of the severity of IL.

Neoplasms can be found in the lymphatic vessels or intestinal walls of the gastrointestinal tract. Those occurring in the lymphatic vessels of the mesentery are known to induce IL [12]. In addition, a variety of gastrointestinal tumors, including gastric adenocarcinoma, lymphoma, and gastric neuroendocrine carcinoma, are concurrent with IL [13,14]. IL was reported to be resolved after resection of the primary mesenteric angiosarcoma [18]. Furthermore, chemotherapy, used to treat malignant lymphoma, was found to relieve gastrointestinal protein loss in a patient with IL [19]. These findings indicate that IL can occur secondary to the physical obstruction of lymph vessels by gastrointestinal neoplasms and be resolved by getting rid of the primary cause. The lymphatic vessels tend to be focally dilated in cases where gastrointestinal or mesentery tumors are the main cause of lymphatic obstruction. IL is usually located within close range of a concurrent tumor. However, it was difficult to find any neoplasms in the mesentery or gastrointestinal tract during exploratory laparotomy, X-ray imaging, and abdominal sonography in the current case study. To the best of our knowledge, correlations between IL and neoplasms that are physically distant from the impacted lymphatic vessels have not been reported. One study reported that 3 out of 23 dogs with IL presented with non-gastrointestinal neoplasms, including mammary ductal carcinoma, malignant fibrous histiocytoma, and adrenal adenoma; however, the correlation between these neoplasms and IL was not been addressed [4]. Moreover, two out of the three dogs presented with non-abdominal tumors, which might not have been associated with the IL. Though it is uncommon, the RCC is associated with the development of intestinal lymphangiectasia when considering the facts that it was hard to find the general etiology of IL, such as severe inflammation and gastrointestinal tumors. The possibilities that RCC is associated with the development of IL are as follow.

First, physical compression of RCC on the intestinal trunk and cisterna chyli might increase the lymphatic pressure in the intestine. In the present case study, the RCC was located near the cisterna chyli and intestinal trunk. The cisterna chyli receives lymph from the lower part of the body, including the abdomen, pelvis through the intestinal trunk, and lumbar trunks. The size of the RCC was sufficient to obstruct the major lymphatic vessel. Second, the RCC might have metastasized and formed an embolus in the lymphatic vessels. It is known that approximately 69% of canine carcinomas of the kidney metastasize to other organs via the lymphatic or blood vessels [20]. Though RCC does not have a propensity for gastrointestinal metastasis, there are several reports of its metastasis to the gastrointestinal tracts in humans [21,22].Third, apart from physical compression or embolus formation from metastasis, RCCs may attribute to the incidence of IL, which is associated with either the functional alteration of kidney or pathological conditions due to the RCC itself. Albeit uncommon, it has been reported that RCC can be associated to gastrointestinal diseases [23]. RCC is associated with gastrointestinal symptoms such as pain, gastritis, and hiatus hernia [23]. Additionally, other renal diseases such as hemolytic uremic syndrome have been associated with the incidence of IL [24]. Though the mechanism involved remains unclear, renal diseases might be directly or indirectly involved in the occurrence of IL. Large portions of the medulla and cortex in the kidney were replaced by the RCC in the current case study (Figure 2A), which may have led to the malfunctioning of the kidney. Renal dysfunction triggers a decrease in protein reabsorption, which subsequently leads to a decrease in total protein levels in the plasma and lymph.

Sample collection through endoscopy is attempted because it is relatively less invasive than that via exploratory laparotomy. However, the critical limitation is that the endoscopic sample collection is confined to the mucosa layer. As shown in the present case report, the dilation of lymphatic vessels is often remarkable in the muscularis layer, but it is too deep to collect the sample by endoscopy; therefore, the lesion could be missed. In addition, a few samples from endoscopy might be of inadequate quality, which could affect the diagnosis [25]. The present case emphasizes the importance of full-thickness intestine sampling [26].

The limitation of this study is that it was impossible to know whether the resection of RCC resulted in the mitigation of IL, as shown in some other cases of IL with gastrointestinal tumor [18]. The dog was euthanized immediately after the surgery due to complications. If the IL had been relieved after the surgery, we might have been able to confirm that the IL was caused by the non-gastrointestinal tumor.

The treatments of IL are confined to dietary fat restriction, as supportive treatment, apart from the elimination of the primary cause. Therefore, finding out the primary cause of IL is critical. The present case study suggests the possibility that a non-gastrointestinal tumor can cause IL. Additionally, it indicates that IL should be considered when an abdominal mass ismetastatic or located near the major lymphatic vessels in the abdomen.

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Authors' contributions

EJ have made substantial contributions acquisition of data, or analysis and interpretation of data, wrote manuscript. MJ have been involved in drafting the manuscript or revising it critically for important intellectual content. KS have given its study design coordination and final approval of the version to be published and helped to draft the manuscript. All authors read and approved the final manuscript.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Statement of Informed Consent

The owner understood procedure and agrees that results related to investigation or treatment of their companion animals, could be published in Scientific Journal Acta Veterinaria-Beograd.

REFERENCES

1. Littman MP, Dambach DM, Vaden SL, Giger U: Familial protein-losing enteropathy and protein-losing nephropathy in Soft Coated Wheaten Terriers: 222 cases (1983-1997). J Vet Intern Med 2000,14(1):68-80.
2. Bota D, Lecoindre A, Poujade A, Chevalier M, Lecoindre P, Baptista F, Gomes E, Hernandez J: Protein losing enteropathy in Yorkshire Terriers – Retrospective study in 31 dogs. Revue de méd vét 2016, 167(1-2):2-9.
3. Landsverk T, Gamlem H: Intestinal lymphangiectasia in the Lundehund. Scanning electron microscopy of intestinal mucosa. Acta Pathol Microbiol Immunol Scand A 1984, 92(5):353-362.
4. Kull PA, Hess RS, Craig LE, Saunders HM, Washabau RJ: Clinical, clinicopathologic, radiographic, and ultrasonographic characteristics of intestinal lymphangiectasia in dogs: 17 cases (1996-1998). J Am Vet Med Assoc 2001, 219(2):197-202.
5. Huber X, Degen L, Muenst S, Trendelenburg M: Primary intestinal lymphangiectasia in an elderly female patient: A case report on a rare cause of secondary immunodeficiency. Medicine (Baltimore) 2017, 96(31):e7729.
6. Steinfeld JL, Davidson JD, Gordon RS Jr, Greene FE: The mechanism of hypoproteinemia in patients with regional enteritis and ulcerative colitis. Am J Med 1960, 29:405-415.
7. Petersen VP, Hastrup J: Protein-losing enteropathy in constrictive pericarditis. Acta Med Scand 1963, 173:401-410.

8. Louvet A, Denis B: Ultrasonographic diagnosis--small bowel lymphangiectasia in a dog. Vet Radiol Ultrasound 2004, 45(6):565-567.
9. Ohmiya N, Nakamura M, Yamamura T, Yamada K, Nagura A, Yoshimura T, Hirooka Y, Hirata I, Goto H: Classification of intestinal lymphangiectasia with protein-losing enteropathy: white villi type and non-white villi type. Digestion 2014, 90(3):155-166.
10. Larson RN, Ginn JA, Bell CM, Davis MJ, Foy DS: Duodenal endoscopic findings and histopathologic confirmation of intestinal lymphangiectasia in dogs. J Vet Intern Med 2012, 26(5):1087-1092.
11. Hokari R, Kitagawa N, Watanabe C, Komoto S, Kurihara C, Okada Y, Kawaguchi A, Nagao S, Hibi T, Miura S: Changes in regulatory molecules for lymphangiogenesis in intestinal lymphangiectasia with enteric protein loss. J Gastroenterol Hepatol 2008, 23(7 Pt 2):e88-95.
12. Poirier VC, Alfidi RJ: Intestinal lymphangiectasia associated with fatal gastrointestinal bleeding. Am J Dig Dis 1973, 18(1):54-58.
13. Qvigstad G, Kolbjornsen O, Skancke E, Waldum HL: Gastric neuroendocrine carcinoma associated with atrophic gastritis in the norwegian lundehund. J Comp Pathol 2008, 139(4):194-201.
14. Mak KL, Hui PK, Chan WY, Leung KM: Mucosal lymphangiectasia in gastric adenocarcinoma. Arch Pathol Lab Med 1996, 120(1):78-80.
15. Day MJ, Bilzer T, Mansell J, Wilcock B, Hall EJ, Jergens A, Minami T, Willard M, Washabau R; World Small Animal Veterinary Association Gastrointestinal Standardization Group: Histopathological standards for the diagnosis of gastrointestinal inflammation in endoscopic biopsy samples from the dog and cat: a report from the World Small Animal Veterinary Association Gastrointestinal Standardization Group. J Comp Pathol 2008, 138 (Suppl 1):S1-43.
16. Fleisher TA, Strober W, Muchmore AV, Broder S, Krawitt EL, Waldmann TA: Corticosteroid-responsive intestinal lymphangiectasia secondary to an inflammatory process. N Engl J Med 1979, 300(11):605-606.
17. Watson VE, Hobday MM, Durham AC: Focal intestinal lipogranulomatous lymphangitis in 6 dogs (2008-2011). J Vet Intern Med 2014, 28(1):48-51.
18. Castro EC, Galambos C, Shaw PH, Ranganathan S: Primary mesenteric angiosarcoma in a child with associated lymphangiectasia: a case report. Pediatr Dev Pathol 2008, 11(6):482-486.
19. Broder S, Callihan TR, Jaffe ES, DeVita VT, Strober W, Bartter FC, Waldmann TA: Resolution of longstanding protein-losing enteropathy in a patient with intestinal lymphangiectasia after treatment for malignant lymphoma. Gastroenterology 1981, 80(1):166-168.
20. Bryan JN, Henry CJ, Turnquist SE, Tyler JW, Liptak JM, Rizzo SA, Sfiligoi G, Steinberg SJ, Smith AN, Jackson T: Primary renal neoplasia of dogs. J Vet Intern Med 2006, 20(5):1155-1160.
21. Yamamoto D, Hamada Y, Okazaki S, Kawakami K, Kanzaki S, Yamamoto C, Yamamoto M: Metastatic gastric tumor from renal cell carcinoma. Gastric Cancer 2009, 12(3):170-173.
22. Aissa A, Kherifech M, Alouini R, Hajji H, Stita W: Multiple intussusceptions revealing metastases from renal carcinoma to the small intestine. J Visc Surg 2012, 149(3):e223-224.
23. Ojha U, Ojha V: Renal cell carcinoma presenting as nonspecific gastrointestinal symptoms: a case report. Int Med Case Rep J 2018, 11:345-348.

24. Bogdanović R, Stanković I, Jojić N, Ognjanović M, Zlatković M, Popović O, Nikolić V: Recurrent hemolytic uremic syndrome with hypocomplementemia and intestinal lymphangiectasia. Nephron 1997, 76(4):481-484.
25. Willard MD, Mansell J, Fosgate GT, Gualtieri M, Olivero D, Lecoindre P, Twedt DC, Collett MG, Day MJ, Hall EJ, Jergens AE, Simpson JW, Else RW, Washabau RJ: Effect of sample quality on the sensitivity of endoscopic biopsy for detecting gastric and duodenal lesions in dogs and cats. J Vet Intern Med 2008, 22(5):1084-1089.
26. Fogle JE, Bissett SA: Mucosal immunity and chronic idiopathic enteropathies in dogs. Compend Contin Educ Vet 2007, 29(5):290-302.

INTESTINANA LIMFANGIEKTAZIJA U VEZI SA KARCINOMOM BUBREGA KOD PSA

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Etiologija dilatacije limfnih sudova, poremećaj koji je poznat kao intestinalna limfangiektazija, još uvek nije razjašnjena. U većini slučajeva, nalazi se kao sekundarni poremećaj ostalih patoloških stanja kao što su to gastrointestinalne neoplazme. Međutim, do sada je kod pasa opisano samo nekoliko slučajeva intestinalne limfangiektazija koja je prisutana zajedno sa ne-gastrointestinalnim neoplazijama. Do sada u literaturi nisu objavljene diskusije korelacije između intestinalne limfangiektazije i negastrointestinalnih neoplazija. U studiji, opisan je redak slučaj intestinalne limfangiektazije koja je bila istovremeno prisutna sa renalnim ćelijskim karcinomom, kod ženke Malteškog mešanca, stare 11 godina što ukazuje da negastrointestinalne neoplazije mogu da budu povezane sa razvojem intestinalnih limfangiektazija. Prilikom makroskopskog pregleda, tanka creva su bila neravnomerno edematozna. Mikroskopskim pregledom, uočena je naglašena dilatacija limfnih sudova naročito u okviru submukoze i mišićnog sloja. Segment crevnih resica bio je dilatiran, a vrhovi štapićasti. Karcinom je mogao da dovede do nastanka intestinalne limfangiektazije pritiskajući glavne limfne sudova ili hilusne cisterne, istovremeno povećavajući pritisak u limfnim sudovima gastrointestinalnog trakta. Metastaze karcinoma gastrointestinalnog trakta mogu da indukuju limfangiektazije creva. Otuda bi pojava intestinalne limfangiektazije morala da se uzme u obzir u slučaju pojave neoplazija abdomena koje su locirane u blizini glavnih limfnih sudova.