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# Necrotic omental lipoma in a dog as an unusual cause of sclerosing encapsulating peritonitis - a case report

**Ana Beck[1]*, Marko Stejskal[2], Vladimir Butković[3], Ivan-Conrado Šoštarić-Zuckermann[1], Andrea Gudan Kurilj[1], and Željko Grabarević[1]**

[1]*Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Zagreb, Zagreb, Croatia*

[2]*Clinic for Surgery, Orthopaedics and Ophthalmology, Faculty of Veterinary Medicine, University of Zagreb, Zagreb, Croatia*

[3]*Department of Radiology, Ultrasound Diagnostic and Physical Therapy Faculty of Veterinary Medicine, University of Zagreb, Zagreb, Croatia*

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**ABSTRACT**

Solitary or multiple lipomas in dogs may occur almost anywhere in the subcutaneous tissue of the trunk, gluteal region and proximal limbs, although they rarely originate from abdominal fat deposits. In the present report, we describe a case of a massive rupture of necrotic omental lipoma, and free fat release within the abdominal cavity, causing a severe chronic inflammatory reaction of the peritoneum and resulting in sclerosing encapsulating peritonitis of several abdominal organs.

**Key words:** dog, omental lipoma, blunt trauma, fat necrosis, neutrophils, sclerosing encapsulating peritonitis

## Introduction

Intra-abdominal lipomas in dogs are benign neoplasms with very low incidence, originating mainly from fat tissue deposits. Based on the scarce literature data, intra-adbominal lipomas have been reported in seven middle aged and geriatric dogs (MAYHEW and BROCKMAN, 2002; HAMMOND and REGAN, 2007; CLAPP et al., 2009). As in humans, obesity seems to be one of the major risk factors for the development of this benign neoplasm (CHA et al., 2009). Intra-abdominal lipomas may originate from

*Corresponding author:

Ana Beck, DVM., PhD, Department of Veterinary Pathology, Faculty of Veterinary Medicine, University of Zagreb, 10000 Zagreb, Heinzelova 55, Croatia, Phone: +385 1 2390 310; E-mail: abeck@vef.hr

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the mesentery, omentum, retroperitoneal fat, urachal remnants, or colonic and urinary bladder subserous fat (MAYHEW and BROCKMAN, 2002; HAMMOND and REGAN, 2007; CLAPP et al., 2009), although their origin may also not be identified (MAYHEW and BROCKMAN, 2002). Clinical signs of intra-abdominal lipoma are associated with adjacent organ compression and displacement, such as abdominal distension, intermittent vomiting, faecal tenesmus, inguinal swelling, urinary incontinence, pollakiuria or dysuria (MAYHEW and BROCKMAN, 2002; HAMMOND and REGAN, 2007; CLAPP et al., 2009). Surgical resection provides resolution of clinical signs and post-operative survival time is longer than five years.

Sclerosing encapsulating peritonitis (SEP) represents a chronic form of peritonitis in which the mesothelial lining of the visceral and parietal surfaces of the abdomen is replaced by fibrous tissue (HARDIE et al., 1994). This pathological condition has been reported under different synonyms, such as peritoneal fibrosis and encapsulating peritoneal sclerosis (KAWANISHI et al., 2005). Only ten cases of SEP have been reported in different dog breeds, of different ages and of both sexes (HARDIE et al., 1994; ADAMAMA-MORAITOU et al., 2004; ETCHEPAREBORDE et al., 2010). Human medicine recognizes SEP as a primary idiopathic disorder or, more frequently, as a secondary event to other causative chronic factors, such as ambulatory peritoneal dialysis, abdominal tuberculosis, gastrointestinal malignancy, the presence of fibrogenic foreign material and many others (DEMIR et al., 2007). On the basis of previously reported cases, dogs may develop SEP due to chronic bacterial peritonitis, foreign body irritation and/or intestinal rupture. Idiopathic forms of SEP and cocoon-like fibrotic peritonitis due to leishmaniosis, have been reported in dogs. Regardless of the cause, clinical signs are nonspecific, and include anorexia, abdominal distension, peritoneal effusion, lethargy, vomiting, diarrhoea and abdominal pain (HARDIE et al., 1994; ADAMAMA-MORAITOU et al., 2004). Management of SEP is very complicated and has a poor prognosis. Surgical treatment and resolution of the primary disease is the first step in treatment (HARDIE et al., 1994; ETCHEPAREBORDE et al., 2010).

The aim of this case report is to present clinical, laboratory, cytology, radiology, necropsy and histology findings caused by omental lipoma necrosis and release of free fat within the abdominal cavity, in order to suggest a novel pathogenesis of SEP in dogs.

**Case history**

An 8-year-old, intact male Labrador cross dog, weighing 35 kg, presented with a history of lethargy, inappetence, abdominal distension, episodes of vomiting and decreased frequency of defecation of one week duration. The dog had no history of recent abdominal surgical treatment or medications with any drugs. The owners indicated that the dog was found wedged under the garden fence a few weeks prior to

the onset of clinical signs. Physical examination revealed no abnormalities except a tense and painful abdomen, which impeded palpation and differentiation of the abdominal organs. Haematological examination revealed increased WBC 29.03 $\times 10^9$/L (reference range 5.50-16.90 $\times 10^9$/L) due to neutrophilia (20.22 $\times 10^9$/L, reference range 2.00-12.00 $\times 10^9$/L) and monocytosis (6.09 $\times 10^9$/L, reference range 0.30-2.00 $\times 10^9$/L). Biochemical parameters (total proteins, albumins, globulins, creatinine, alkaline phosphatase, alanine aminotransferase) were within normal limits, while a slightly increased concentration of blood urea (10.2 mmol/L, reference range 2.5-9.6 mmol/L) was apparent. A red-tinged fluid obtained by abdominocentesis revealed an exudate with total nucleated cell count of 72,000/µL. Cytology of the direct smear stained with Giemsa revealed red blood cells admixed with 60% non-degenerative neutrophils, 30% foamy macrophages exhibiting erythrophagocytosis or fat phagocytosis. Activated mesothelial cells (solitary or in clusters) and variable numbers of inflammatory giant cells and reactive fibroblasts were also noted (Fig. 1).

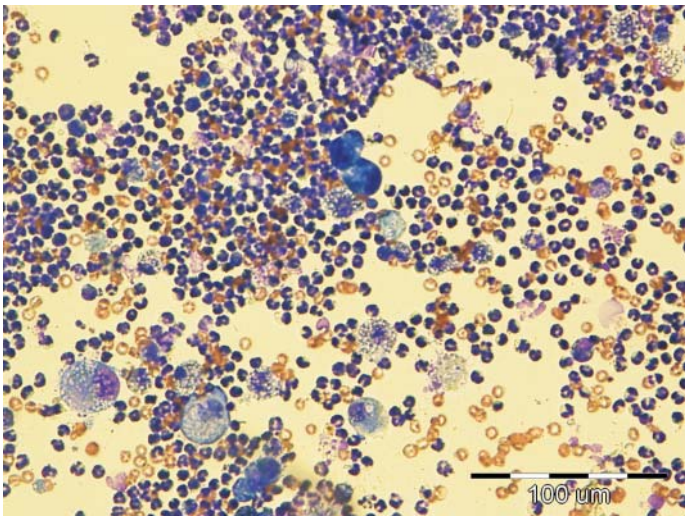


Fig. 1. Direct smear of the abdominal fluid. Numerous neutrophiles admixed with red blood cells, foamy macrophages and a single cluster of activated mesothelial cells. Giemsa stain; ×40.

No microorganisms or evidence of neutrophil degeneration were detected within the specimens, so the diagnosis of sterile peritonitis was made. Peritonitis was confirmed by lateral abdominal radiograph, which demonstrated numerous string-like structures of fibrous tissue density and diffuse soft tissue opacity in the cavity, inhibiting abdominal viscera differentiation. Gas filled intestinal segments were the only distinguishing structures present within the abdominal cavity. A large, opaque soft tissue mass was also

noted in the cranial and ventral aspect of the abdomen under the gastric "bony" density content (Fig. 2). Thoracic radiographs were unremarkable.

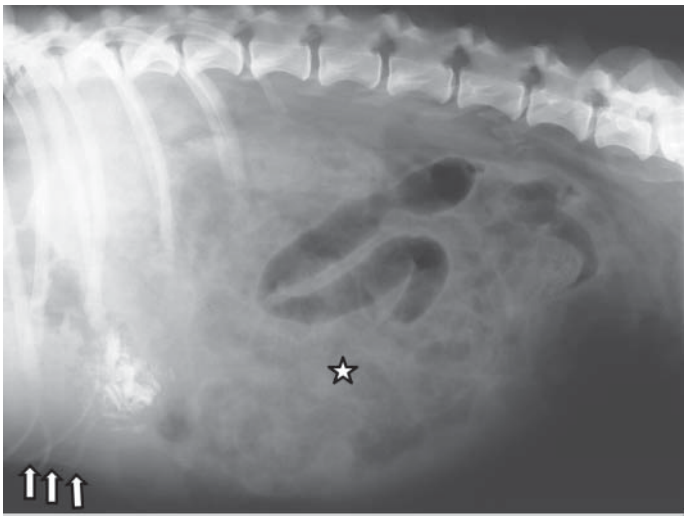

Fig. 2. Lateral radiograph of abdominal cavity showing diffuse indentation of the intestinal wall (asterisk) and the large fat-dense opacity mass in the cranioventral abdomen (arrows).

Based on radiological and cytological findings, abdominal neoplasia of unknown origin was considered as the cause of exudative peritonitis in this case. The dog was euthanized at the owner´s request.

**Necropsy findings**

The distended abdominal cavity contained 2.5 L of dull reddish fluid, rich in fatty droplets. A large abdominal fatty mass (4.4 kg) originating from the omentum was found to occupy the entire cranial and caudoventral abdominal cavity (Fig. 3), displacing the stomach craniodorsally and compressing the duodenum. The mass seemed both friable and firm, and consisted of numerous white fat nodules up to 7 cm in diameter, separated by fibrous tissue ribbons. The cut surface of each nodule was white, homogenous, fatty and tender. A 3.5 cm in diameter aperture of irregular shape and with smooth fibrotic edges was found within the central part of the mass. A solitary yellowish-grey area of necrotic fat tissue was present caudoventrally, near the largest fatty nodule. The left, atrophic pancreatic lobe was enclosed within fatty nodules, while the detached spleen did not show any macroscopic lesion. There were no attachments present between the mass and other abdominal structures or between the abdominal organs. Capillaries of the

parietal, and especially of the visceral peritoneum, were markedly congested. Sporadic petechiation was observed. The visceral peritoneum covering duodenal mesenteric fat, pancreas, parietal aspect of hepatic capsule and urinary bladder was thickened with white to grey, occasionally reddish, fine fibrous membranes. Fibrous tissue deposits caused segmental atrophy of the liver and pancreas, and decreased elasticity of the urinary bladder wall.

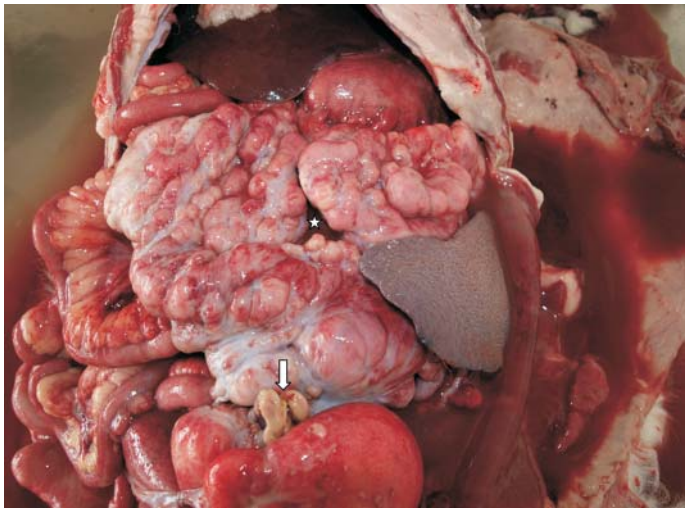

Fig. 3. Ventral aspect of the abdominal cavity. The presence of multinodular omental lipoma with gray-red membrane covering its surface. Irregular aperture, 3.5 cm in diameter located centrally within the tumor (asterisk). Focal area of fat necrosis (arrow).

**Histology**

Representative samples of the omental mass, pancreas, spleen, liver, urinary bladder and abdominal wall were fixed in neutral buffered 10% formalin and embedded in paraffin. Tissue samples were cut into 3 to 5 μm thick sections and stained with haematoxylin and eosin (H&E), periodic acid-Schiff (PAS), Grocott's methenamine silver, Gram and Giemsa methods. Microscopically, all samples taken from the omental nodular mass were composed of homogenous mature adipose tissue without cellular atypia, which is compatible with the diagnosis of omental lipoma (Fig. 4). Within the omental lipoma, one superficial area of fat necrosis and pyogranulomatous steatitis was present. Special staining techniques did not reveal microorganisms within the inflammation area or within any of the examined tissues and organ sections. The main microscopic lesions were loose deposits of reactive fibroblasts covering the surface of the omental lipoma, duodenum,

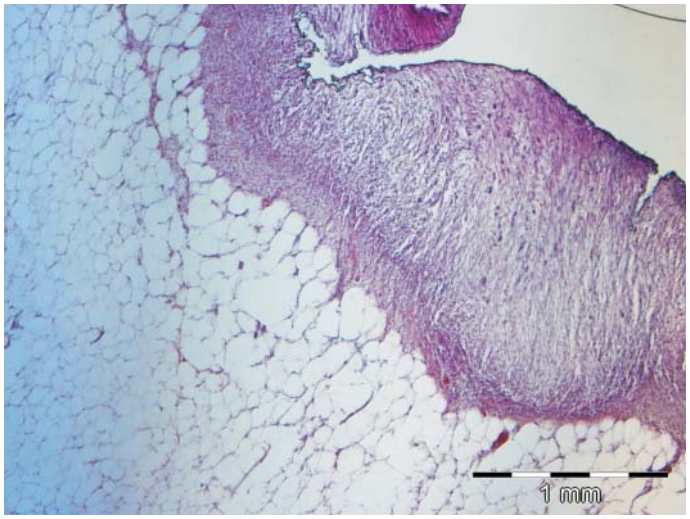


Fig. 4. Photomicrograph of the omental lipoma demonstrating the uniformity and maturity of lipocytes encased within a thick layer of immature connective tissue. H&E; ×4.

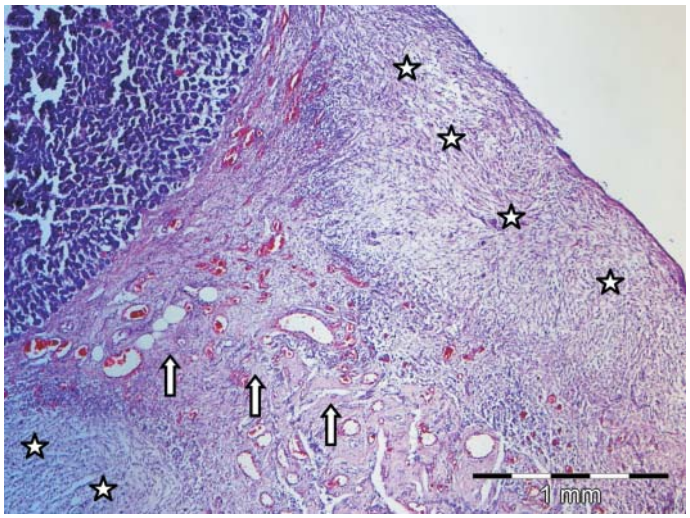


Fig. 5. Photomicrograph of the right pancreatic lobe demonstrating gland tissue atrophy due to marked thickening of the peritoneal membrane. Inner layer of fibrotic tissue expresses evident maturity (arrows) comparing to superficial layers (asterisks). Congestion of blood vessels is marked. H&E; ×4.

pancreas, urinary bladder and the liver (Fig. 5). Fibroblasts exhibiting marked cellular atypia were arranged in irregular layers, bundles and streams composing immature and richly vascularized connective tissue. Mild to moderate infiltrations of neutrophils, macrophages and occasionally multinuclear giant cells were arranged around congested immature blood vessels. Perivascular haemorrhage was also diffusely present within the visceral fibrosis, particularly in the outermost layer of the connective tissue membrane. Mesothelium was not detected on the surface of the organs enclosed within immature connective tissue. Based on these findings, a diagnosis of sclerosing encapsulating peritonitis was made. No evidence of parietal peritoneum involvement was detected.

**Discussion**

In dogs, lipomas commonly occur in the subcutaneous tissues of the ventral thorax and abdomen of middle-aged and geriatric patients (VOLTA et al., 2006). Contrary to subcutaneous lipomas, intra-adbominal lipomas are rare and require a complex diagnostic approach. Intra-abdominal lipomas consist of well-differentiated adipose tissue surrounded by fibrous capsule of variable thickness. Frequently they become very large, ranging from 3.8 kg to 5.5 kg due to the large volume of the abdominal cavity, which allows them to develop undisturbed until they reach a size that interferes with the surrounding structures (MAYHEW and BROCKMAN, 2002). In the present case, the dog did not show intestinal disturbance until the omental lipoma started to compress the duodenum. At that time the tumour was very large, comprising about 12.5% of the dog's total body mass. Lipomas are generally very fragile and prone to rupture due to the high fat content within the cells and variably thick fibrous layer of the surrounding capsule. Based on Doppler ultrasonography, they are also poorly vascularized, with the majority of blood flow distributed at the periphery, making the central parts of intra-abdominal lipomas ischemic (HAMMOND and REGAN, 2007). This fact is in correlation with the high occurrence of central tumour necrosis in previously described cases (MAYHEW and BROCKMAN, 2002; HAMMOND and REGAN, 2007; CLAPP et al., 2009). Likewise LEE GROSS et al. (2005) claimed that subcutaneous fat tissue necrosis is also associated with decreased blood supply caused by chronic pressure or blunt trauma. Abdominal cavity organs and structures are typically poorly protected from blunt trauma compared to thoracic cavity organs. Therefore, we hypothesize that the intra-abdominal lipoma must be particularly sensitive to blunt trauma impacts. In this case we assumed that the primary omental lipoma with a central area of necrosis ruptured due to blunt trauma (abdominal compression) which caused aperture formation and segmental tissue abatement. Numerous peripheral lipoma remnants "survived" attached to the omentum and continued to proliferate individually, creating the unique multinodular appearance. Free lipids released from the necrotic tumour centre and discharged from damaged lipocytes into the abdominal cavity became

foreign bodies within the abdomen, likely enhancing the strong peritoneal inflammatory reaction. It is well known that lipids released from damaged lipocytes are a vigorous stimulator of inflammation in the subcutis. Once lipids are liberated, hydrolysis and saponification occurs, releasing proinflammatory fatty acids and glycerine, which in turn cause necrosis and enhanced inflammation of the surrounding tissue (LEE GROSS et al., 2005). Namely, these compounds attract numerous neutrophils and monocytes into the necrotic area. Severe left shift neutrophilia and monocytosis found in the blood and numerous neutrophils and macrophages detected within the abdominal cavity exudate indicate that the inflammatory compounds within the abdominal cavity emerged in this case in a manner similar to that described for subcutaneous tissue. However, segmental omental lipoma necrosis could be a single self-limited, benign condition, resolving spontaneously in our case. Unfortunately, sustained fat leakage into the abdominal cavity due to perpetual ischemic fat necrosis and long lasting neutrophil accumulation and release of their cytoplasmic enzymes likely initiated mesothelial lysis. Neutrophils can also provide angiogenic and fibroblastic cytokines, as well as damaged mesothelium and fibroblasts, which may be an alternative source of cytokines. Interleukin-1 released by activated peritoneal macrophages also stimulates submesothelial mesenchymal cells to proliferate and differentiate into fibroblasts, thus also amplifying peritoneal fibrosis (HARDIE et al., 1994). The explanation of adhesion absence in this case, and in most dog SEP cases previously described, is based on the active fibrinolytic system characteristics of this species (HARDIE et al., 1994). In our case, necropsy and histopathology confirmed replacement of the mesothelium layer with a thick fibrous tissue layer over the urinary bladder, visceral hepatic capsule, the duodenal segment including the pancreas and the omental lipoma. Fibrous tissue deposits and omental lipoma mass compression caused segmental pancreatic and hepatic atrophy but this finding was not reflected in the blood biochemical profile. Fibrous tissue deposits decreased elasticity of the urinary bladder wall also without clinical consequences. Severe superficial duodenal fibrosis probably improved intestinal hypomotility and vomiting exacerbation in this case. ADAMAMA-MORAITOU et al. (2007) pointed out that immaturity of the fibrous tissue in canine SEP plays the main role in ascites pathogenesis, due to numerous immature blood vessels that are unable to retain fluid within their lumen. Long lasting loss of fluid, proteins and red blood cells within the abdominal cavity represents the main complication of SEP in dogs. These complications lead to dehydration, haemoconcentracion and hypoproteinemia, which are the main reasons for clinical impairment in canine SEP cases (HARDIE et al., 1994). Necropsy confirmed severe intra-abdominal fluid and erythrocytes loss, although it was not evident from the haematology and biochemistry blood values in this case. Necropsy and histopathology findings ruled out other possible causes of abdominal fibrosis in this case such as gastric, intestinal and urinary bladder rupture or foreign body irritation, as well as primary or metastatic malignant alteration. Bacterial, fungal and

protozoan infections were excluded based on the negative histology findings. Since the dog had had trauma prior to the onset of clinical signs, idiopathic SEP was excluded as a potential differential diagnosis. The random affection of the visceral peritoneum is unclear, though it could be connected to the random free lipid leaking and irritation within the abdominal cavity.

In conclusion, as in humans, different causes may initiate different types of canine SEP, and this is the first description of free fat causing peritoneal fibrosis. In our opinion, surgical elimination of the intra-abdominal lipoma as the source of free lipids could likely have stopped the peritoneal irritation. Surgical planning in cases of suspected necrotic intra-abdominal lipoma has to be based on precise radiological and cytological diagnoses. A plain radiograph is insufficient and does not provide accurate characterization of the benign characteristics of intra-abdominal lipomas. A precise diagnosis has to be provided using sonography or computed tomography combined with cytology (MAYHEW and BROCKMAN, 2002; HAMMOND and REGAN, 2007; CLAPP et al., 2009). In order to confirm the diagnosis of SEP in dogs, the same complex diagnostic algorithm is needed. As in humans, laparotomy offers the most accurate diagnosis (KAWANISHI et al., 2005).

It is important to stress that obese patients with a history of abdominal trauma may develop traumatic fat tissue necrosis.

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**BECK, A., M. STEJSKAL, V. BUTKOVIĆ, I.-C. ŠOŠTARIĆ-ZUCKERMANN, A. GUDAN KURILJ, Ž. GRABAREVIĆ: Nekrotični lipom omentuma kao neuobičajeni uzrok sklerozirajućega inkapsulirajućega peritonitisa u psa - prikaz slučaja. Vet. arhiv 82, 391-399, 2012.**

**SAŽETAK**

Pojedinačni ili višestruki lipomi u pasa mogu se pojaviti gotovo bilo gdje u potkožnom tkivu trupa, glutealne regije te proksimalnih dijelova udova. Lipomi podrijeklom od trbušnoga masnog tkiva izrazito su rijetki. U ovom radu prikazan je slučaj masivne rupture nekrotičnog lipoma omentuma uslijed koje je došlo do curenja slobodne masti u trbušnu šupljinu i pokretanja opsežne kronične upalne reakcije potrbušnice koja je u konačnici rezultirala sklerozirajućim inkapsulirajućim peritonitisom nekoliko trbušnih organa.

**Ključne riječi:** pas, lipom omentuma, tupa trauma, nekroza masti, neutrofili, sklerozirajući inkapsulirajući peritonitis