

# Fatal secondary septic peritonitis associated with multiple renal and splenic infarcts in Lhasa Apso dog

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## ABSTRACT

*An eight year old male Lhasa Apso dog was presented for necropsy with the history of persistent vomiting, anorexia, lethargy, dehydration and abdominal distension. Blood picture revealed marked neutrophilic leukocytosis along with left shift. Necropsy examination revealed the presence of secondary septic peritonitis and multiple renal / splenic infarcts. Histologically, perforative/haemorrhagic enteritis, renal infarcts with diffuse glomerular / tubular necrosis of coagulative type and splenic infarction with occluded artery containing bacterial thromboemboli were confirmed. Secondary peritonitis has been observed to be a frequent complication associated with perforation of the intestine. These fatal complications of septic peritonitis present a therapeutic challenge and needs prompt veterinary care and treatment.*

**key words:** Dog, Renal infarcts, splenic infarcts, septic peritonitis

Peritonitis in animals may occur as a primary disorder or may be secondary to other pathophysiological conditions. A variety of agents can cause peritonitis resulting in several clinical symptoms, disease progression and adverse effects. The etiologies range from viral to bacterial to parasitic to physical agents and organ rupture (Zachary, 2012). Gastrointestinal ulcers account for nearly 24% to 35% of gastro-intestinal tract associated peritonitis (Greenfield and Walshaw, 1987). Peritonitis can be classified as acute or chronic depending on the duration; septic or non-septic depending on the infectious nature of

the causative agent; local or diffuse depending on the area involved and adhesive or exudative depending on the type of inflammatory product. The incidence of primary peritonitis is lesser than that of secondary peritonitis and can be infectious or idiopathic. In case of primary peritonitis caused by pathogenic organisms, the organisms spread *via* blood to the peritoneal cavity.

Primary peritonitis is rare in healthy dogs and is often observed in immune compromised animals. In case of secondary peritonitis, the peritoneal cavity becomes contaminated

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with infectious agents *via* translocation or penetration through the wound or leakage from the gastrointestinal tract (most common cause) or through leakage from other infected visceral organs (eg, hepatic/ splenic abscess, inflamed bladder/ uterus, etc.). Pathogenic organisms frequently isolated are *Nocardia*, *Mycobacterium*, *Haemophilus parasuis*, etc. Perforations in the abdomen or wound dehiscence can cause laceration of the visceral organs, followed by translocation of extraneous substances and infectious agents into the peritoneal cavity. Migration of endoparasites can also cause leakage of the ingesta leading to septic peritonitis. The gross and microscopic features of a case of fatal secondary septic peritonitis in a Lhaso Apsog dog along with splenic and renal infarcts as its complication are described in the present communication.

An eight year old male Lhaso Apsog dog was admitted with the history of persistent vomiting, anorexia and lethargy for one week. The dog had injected mucous membranes, sunken eyes with marked dehydration and exhibited abdominal distension and localised pain on cranial abdominal palpation. Blood picture revealed marked leukocytosis with neutrophilia and shift to left. The animal collapsed during clinical examination.

A detailed post-mortem examination was conducted and representative tissue samples from liver, lungs, kidneys, spleen, stomach and intestines were collected in 10% neutral buffered formalin. The tissue samples were processed for histopathological studies employing paraffin embedding technique. Sections of 3-5mm thick were prepared and stained with hematoxylin and eosin (H&E) following standard procedures.

At necropsy, the abdominal cavity contained abundance of sero-sanguinous fluid, fibrin deposits and undigested feed contents (Fig.1). The serosal surface of the intestine and the mesentery showed multifocal, randomly distributed

areas of marked congestion and haemorrhage. Three perforations measuring around 1-2 cm diameter with irregular haemorrhagic borders were present in the duodenal segment (Fig.2). Kidneys were moderately shrunken and had multiple irregular depressed areas with fibrin deposits. The cut surface appeared diffusely congested and had multiple, well demarcated, yellowish white wedge shaped areas of necrosis surrounded by thin zones of congestion (Fig.3). The broader end of spleen appeared markedly enlarged, oedematous, congested and haemorrhagic with discrete red black wedge shaped foci filled with blood and adherent fibrin deposits (Fig.4). Cut surface of spleen showed well demarcated zones of necrosis and haemorrhage.

On histopathological examination, intestinal segment adjacent to the site of perforation showed marked necrosis and extensive areas of congestion and haemorrhages in the mucosa/sub-mucosa. The renal infarcts were characterized by diffuse glomerular/ tubular necrosis with the preservation of architecture (coagulative necrosis) along with the presence of moderate oedema/ infiltrates in the interstitium and the adjacent cortex (Fig. 5). Splenic artery appeared to be occluded with a bacterial embolic thrombus firmly attached to the lumen of the vessel consisting of concentric layers of fibrin, numerous inflammatory cells and RBC's and had clusters of basophilic bacterial colonies (Fig. 6).

Septic peritonitis observed in the present case was due to the presence of 3 perforations in the duodenal segment. Septic peritonitis has been observed to be a frequent complication associated with injuries that affect the gastrointestinal integrity (Ragety et al., 2011). Perforation of the stomach / intestine leading to leakage of the partially digested feed material as well as rupture of the infected uterus can result in acute secondary septic peritonitis. Agents frequently associated with secondary peritonitis are intestinal foreign bodies

(Penninck and Mitchell, 2003); perforations of the stomach, intestines, rectum, urinary bladder, or uterus; gastrotomy or enterotomy; gastric/intestinal neoplasms (eg, mesothelioma); inflammatory / traumatic injuries of liver, gall bladder, pancreas, dilatation / volvulus of gastrointestinal segments, etc.

The dog had persistent vomiting, was anorectic and lethargic, prior to death. It had injected mucous membranes, sunken eyes with marked dehydration, abdominal distension and localised pain on cranial abdominal palpation. Animals with secondary peritonitis may also show clinical signs associated with the primary disease. The classical observation through radiography is the absence of abdominal details and there is a focal or generalized "ground-glass" appearance. The GI tract can be dilated with air and/or fluid. Free air inside the abdomen can be present when there is rupture of any luminal organ or at times may be due to gas-producing anaerobic organisms in the absence of organ rupture (Fossum *et al.*, 2009).

The chemical mediators of inflammation produced such as histamine, serotonin, proteases and endotoxins as a result of the leaked contents result in enhanced capillary permeability and outpouring of plasma proteins, solutes and water into the peritoneal cavity. The resulting inflammation enhances fibrin deposition, particularly in and around the site of injury and peritoneum, as well as the loss of isotonic fluid into the peritoneal cavity with concurrent haemo-concentration (Culp *et al.*, 2009). The fluid produced is clear and transparent (transudate) during the early stages, but becomes turbid as a result of exuded protein, macrophages and polymorphonuclear leukocytes (Kirby, 2003, Culp *et al.*, 2009). In the present case, the abdominal cavity contained abundance of sero-sanguinous fluid and fibrin deposits. The resulting exudate further inhibits microbial clearance and enhances microbial multiplication (Kirby, 2003).

Infarction of different organs has been frequently reported during peritonitis as a result of ischemic injury caused by the vascular obstruction and thromboembolism. Owing to the greater circulating blood supply (20% to 25%), kidneys and spleen are the most frequent targets of thromboembolism. In the present case also, we observed renal and splenic infarcts. The small blood vessels are frequently occluded, i.e., the interlobular arteries and therefore, infarction occurs mostly in the cortical region (Zachary, 2012). In the present case, renal infarcts were seen in the cortex concurring with the previous reports.

Several agents including gastric mucin, bile salts, haemoglobin, etc., are regarded as adjuvants of the inflammation of peritoneum and they accentuate the localized as well as the systemic inflammation (Kirby, 2003). Opsonins, immunoglobulins and complement components get triggered with increasing severity of the inflammation (Schein and Paladugu 2000, Culp *et al.*, 2009). In addition, the levels of several cytokines including TNF, IL-1 and IL-6, PGE<sub>2</sub> and platelet aggregation factor are also increased (Culp *et al.*, 2009). As a result of the production of these chemical mediators, there is a marked decrease in cardiac output, vasodilation and decreased venous return, which further intensify the systemic hypotension and enhance production of endotoxins. The presence of bacteria, the endotoxins, inflammatory cells and their cytokines lead to endothelial damage and tissue factor expression that result in generalized activation of the coagulation cascade, causing thrombosis and fibrinolysis. This results in disseminated intravascular coagulation with the loss of the anti-thrombin III protein into the abdomen. Micro and macro vascular thromboses may lead to tissue hypoxia and organ damage involving the myocardium, lungs, and gastrointestinal tract (DeLaforcade *et al.*, 2003; Estrinet *et al.*, 2006). These pathological changes that occur during septic

peritonitis result in systemic vasodilation, enhanced capillary permeability, reduced cardiac function and multiple organ failure, the characteristic signs of systemic inflammatory response syndrome and septic shock (Schein and Paladugu, 2000). In spite of the advanced treatment strategies available, the prognosis of peritonitis is poor.

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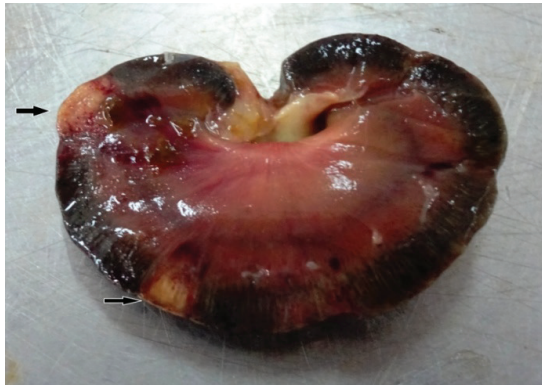
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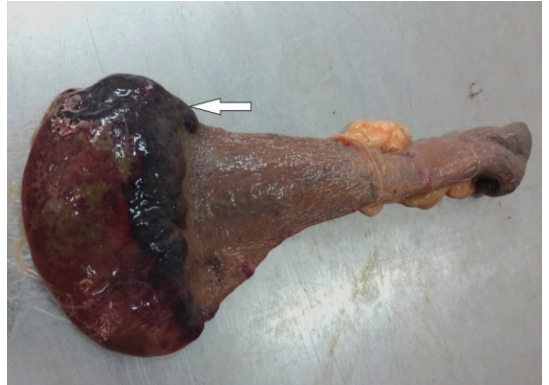
**Figure 1: Gross changes in the abdominal cavity**



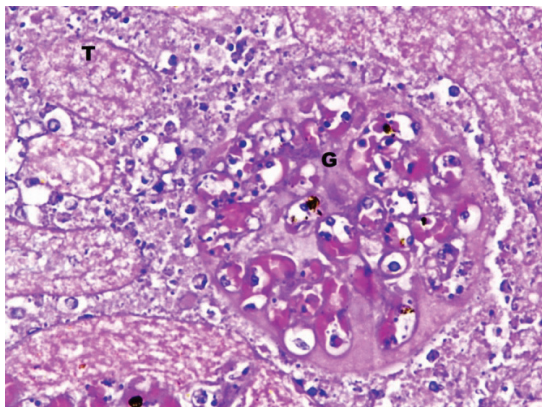
**Figure 2: Intestine showing perforations**



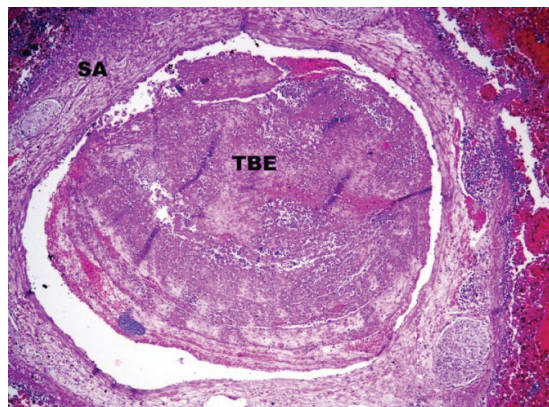
**Figure 3: Cut surface of Kidney showing gross lesions**



**Figure 4: Spleen showing gross lesions**



**Figure 5: Kidney showing infarct and diffuse coagulative necrosis of the glomeruli (G) and the tubules (T), interstitial oedema / congestion H&E, 100 $\times$ .**



**Figure 6: Section of splenic artery occluded with a bacterial embolic thrombus, H & E, 40  $\times$**