SUCCESSFUL MEDICAL MANAGEMENT OF PERITONITIS IN A GREAT DANE DOG

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A 3 years old male Great Dane dog was reported with the history of vomiting, fever and distended abdomen for 3 days. It has been administered with meloxicam for 5 days for the management of fever. Clinical examination showed depression, tachycardia, panting and elevated body temperature. Haematobiochemistry revealed leukocytosis with neutrophilia, elevated blood urea nitrogen and creatinine levels. Radiography revealed ground glass appearance and the abdominal ultrasonography showed hypoechoic free fluid with hyper echoic fibrin shreds. Cytological examination of peritoneal fluid revealed neutrophilia. Animal was administered with dextrose normal saline, Ringer’s lactate, amoxicillin-sulbactum sodium, pantoprazole and furosemide twice daily for 8 days. Feeding habit improved on 3rd day and abdominal distension reduced on 4th day. Animal recovered uneventfully.

Keywords: Dog, Meloxicam, Peritonitis, Ultrasonography.

Peritonitis is inflammation of the peritoneum. Generalised peritonitis is a medical and surgical emergency, which often requires intensive and costly treatment. The NSAIDs in the veterinary market are now being aggressively promoted based on their COX selectivity and the indications for their use are expanding from the treatment of acute, chronic pain and antipyrogen. NSAIDs make the stomach more prone to ulceration by several mechanisms including mucosal irritation, reduction in prostaglandin production, decreasing gastric blood flow and interfering with repair of superficial injury to the mucosa (Trevor et al., 2006). The present case describes the peritonitis associated with the routine use of a COX-2 selective NSAID meloxicam.

Case History and Observations

A 3 year old male Great Dane dog was presented to Teaching Veterinary Clinical Complex, Veterinary College and Research Institute Hospital, Namakkal with the history of anorexia and distended abdomen for 3 days. It was reported to have been administered with meloxicam (@1 mg/Kg) for 5 days for the management of fever. Clinical examination revealed dullness, congested mucous membranes, panting, tachycardia (130/min), elevated body temperature (39.9°C) with ascites. Haemoglobin (12.2 g/dl), packed cell volume (40.17%) and red blood cells (5.93×10⁶/cumm) count were within normal range while leukocytosis (20.07 ×10³/cumm), neutrophilia (90%) elevated blood urea nitrogen (145 mg/dl) and creatinine (4.3 mg/dl) levels were noticed. No blood parasites could be detected on peripheral blood smear examination. Lead II ECG showed sinus tachycardia. Radiography revealed decrease in serosal details with no radio opaque foreign body (Fig. 1). On abdominal ultrasonography hypoechoic free fluid was noticed (Fig. 2). Amber coloured blood tinged viscous peritoneal fluid was obtained through abdominocentesis and subjected to cytology revealed neutrophilia (Fig. 3). Based on the above findings the case was suspected as meloxicam induced peritonitis.

Treatment and Discussion

The animal was intravenously administered with dextrose normal saline and ringer’s lactate based on the dehydration status, amoxicillin+sulbactum sodium (15 mg/kg BID), ondansetron (0.5 mg/kg), pantoprazole (1 mg/kg) and furosemide (2 mg/kg S/C) for 8 days. The animal showed clinical improvement on 2nd day. Haematobiochemical values were within the normal range on 8th day. Animal recovered uneventfully.
Systemically administered corticosteroids and non steroidal anti-inflammatory drugs are the medications most commonly associated with gastroduodenal ulcer disease. NSAIDs inhibit the various isozymes of the COX enzyme and prevent the synthesis of eicosanoids (prostaglandins, prostacyclins, and thromboxane) from arachidonic acid. COX-1 exists at fairly constant concentrations in the cells, such as the gastric mucosa, where prostaglandins serve a physiologic function of cytoprotection as also reported by Livingston (2000). COX-2, the inducible form of the enzyme is synthesized by macrophages and inflammatory cells. It can be upregulated by cytokines, growth factors and tumor promoters and increases in concentration at sites of tissue injury and inflammation as also mentioned by Fitzgerald and Patrono (2001). It is widely accepted that gastric ulcerogenicity is the dose-limiting side-effect common to all established NSAIDs. An abdominal ultrasonographic examination show peritoneal effusion and a hyperechoic liver. The gall bladder had a moderate amount of inspissated, immobile bile sludge as also recorded by Duerr et al. (2004).

References