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A Study on clinical presentation of hepatobiliary disorders in dogs

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Abstract
A total of 140 dogs were diagnosed with hepatobiliary disorders based on clinico, hemato-biochemical and diagnostic imaging. Common clinical signs observed in hepatobiliary disorders affected dogs were inappetance and anorexia, vomition, anemia, diarrhea, pyrexia, lethargy, icterus, abdominal pain, emaciation, ascites, weight gain, weight loss, respiratory distress, limb edema, nervous signs, polyuria and polydipsia. Vital parameters revealed a slight increase in temperature, pulse and respiratory rates with normal heart rates.

Keywords: Hepatobiliary disorders, dogs, clinical signs

Introduction
Consequent to urbanization, there is an increase in the number of pet owners with growing concern for their beloved pets. In an attempt for better health care, many a times there is an overzealous medication by the owner which adversely affects the functioning of the liver (Johnson, 1994) [4]. Hepatobiliary dysfunctions occur in a number of acute and chronic clinical conditions. Drug-induced hepatotoxicity, infectious diseases, congenital or neoplastic diseases, metabolic disorders, degenerative processes, vascular injury, auto-immune diseases and even blunt trauma may result in hepatobiliary dysfunctions (Kumar *et al*., 2013) [5]. Symptoms, clinical signs and diagnostic results reflect impairments in these functions (Meyer and Rothuizen, 2013) [7]. Common clinical manifestations include anorexia, vomition, ascites, jaundice, constipation or diarrhoea, polyurea and polydipsia, weight loss and coagulation abnormalities (Centre, 2015) [3].

Materials and Methods
The study was conducted on 140 dogs of both genders, aged from 5 months to 14 years, presented to Veterinary Hospital, Bhoiguda, Hyderabad with the history and clinical signs suggestive of hepatobiliary disorders. However, dogs with hepatobiliary disorders due to infectious origin were excluded from the study. Each dog was subjected to detailed clinical examination and thoroughly evaluated for its general condition, inspection of mucous membranes, signs of pain, abdominal distension.

Results and Discussion
All the 140 dogs that were diagnosed for various hepatobiliary disorders revealed manifestations like inappetance and anorexia, vomition, anemia, diarrhea, pyrexia, lethargy, icterus, abdominal pain, emaciation, ascites, weight gain, weight loss, respiratory distress, limb edema, nervous signs, polyuria and polydipsia in 116 (82.86%), 89 (63.57%), 67 (47.86%), 56 (40.00%), 55 (39.28%), 51 (36.42%), 41 (29.29%), 38 (27.14%), 37 (26.43%), 32 (22.86%), 32 (22.86%), 29 (20.71%), 26 (18.57%), 24 (17.14%), 19 (13.57%) and 15 (10.71%) dogs, respectively (Table 1; Fig 1-7.).
The present findings are in agreement with Mircean *et al*. (2008), who reported clinical signs of hepatobiliary disease in dogs as inappetance, vomition, weight loss, abdominal distension, polydipsia, cutaneous lesions and fever. Clinical signs of hepatobiliary disease in dogs can be extremely variable, ranging from anorexia and weight loss to abdominal effusion, jaundice and hepatic coma (Penny and Watson, 2008) [8]. Ascites was the predominant clinical sign noticed, whereas, weight gain, anemia, respiratory distress, anorexia and inappetance, limb edema, lethargic, abdominal pain, and vomition were other manifestations observed.

Inappetance and anorexia was found in most of the cases in the present study was a non specific sign of hepatobiliary disorders and similar observations were made by Mircean *et al*. (2008). Vomiting associated with liver dysfunctions could be attributed to the direct stimulation of the vomiting centre by chemoreceptor trigger zone (CTZ) in the fourth ventricle of the brain by the endotoxins that were not cleared by the diseased liver (Batt and Tweedit, 1994) [1]. Anemia was attributed to chronic nature of this liver disease due to increased transient time of erythrocytes through the spleen due to reduced portal blood flow and or fragility of red cells due to high levels of bile acids. Diarrhoea in hepatobiliary disorders could be due to less bile being pumped into the duodenum and decreased resorption of dietary fat, resulting in hyper osmotic intestinal contents and also might be due to portal hypertension and congestion of intestinal vasculature, which can reduce intestinal water resorption and increase in the volume of intestinal contents (Rothuizen, 2008) [9]. Melena and hemetemesis are a consequence of gastric ulceration that was observed in cirrhosis. Lethargy reported in the present study could be due to increased nutritional requirements of sick animals on one side and on the other side limited by reduced appetite in hepatic diseases and toxins that were not detoxified (Rothuizen, 2008) [9]. Icterus or Jaundice reported in the present study was caused by the accumulation of bilirubin in the tissues, a product of RBC destruction and a major pigment in bile. Abdominal pain noticed in the present study was visceral origin, which is diffuse and afferent fibres terminate in the spinal cord or neurons receiving input from both somatic and visceral receptors. Emaciation in the present study was a result of systemic inflammation from cell injury or activation of the immune system which triggers the release of cytokines into the circulation. Weight gain in the present study could be due to the ascitic fluid accumulation in the abdomen. Ascites and increased weight also found to increase the likelihood of short survival (Selgas *et al.*, 2014) [11]. Respiratory distress, was attributed to the overpressure of the ascetic fluids on the diaphragm and respiratory muscles, whereas subcutaneous edema was a consequence of hypoproteinemia associated liver diseases. A number of nitrogenous waste products such as ammonia, aromatic aminoacids, methionine, *gamma* aminobutyric acid (GABA) etc. pass the blood brain barrier in the animals with significant loss of liver function and cause nervous signs (Watson, 1998). Polyuria and polydipsia have been attributed to the impaired adrenal steroid metabolism, altered portal vein osmoreceptor, loss of renal medullary concentration gradient and encephalopathy (Hess and Bunch, 2000) [2].

The mean rectal temperature (º F) were within normal range in 53.57%, while, 34.29% had pyrexia and 12.14% had subnormal body temperatures. These findings are in agreement with Saxena *et al.* (2016) [10], who documented high rectal temperature (103.2º F), heart rate (>120 beats per minute), pale mucous membranes with increased capillary refill time (> 2 sec) and fluid thrill upon tactile percussion in a Labrador dog affected with ascites of hepatic origin. Pyrexia could be a consequence of hepatocellular damage, infection, sepsis or absorbtion of bacterial toxins. Subnormal body temperatures may be seen oftenly in late stages of diseases. However, a slight increase in the pulse rates in few cases was a consequence of slight rise in the heart rates. The mean respiratory rates (per minute) were within normal range in 70% cases and 30% had increased respiratory rate to compensate for the oxygen demand due to anemia. These findings are in agreement with Kamalakar *et al*. (2016) [6], who recorded increased, temperature (103.4º F) and respiratory rate (52/ minute) in a dog affected with ascites of hepatic origin.

Table 1: Clinical observations of hepatobiliary disorders in dogs

S. No	History and Clinical manifestations	No. of dogs	Percentage
1.	Inappetance and Anorexia	116	82.86
2.	Vomition	89	63.57
3.	Anemia	67	47.86
4.	Diarrhea and melena	56	40.00
5.	Pyrexia	55	39.28
6.	Lethargy	51	36.42
7.	Icterus	41	29.29
8.	Abdominal pain	38	27.14
9.	Emaciation	37	26.43
10.	Ascites	32	22.86
11.	Weight gain	32	22.86
12.	Weight loss	29	20.71
13.	Respiratory distress	26	18.57
14.	Limb edema	24	17.14
15.	Nervous signs	19	13.57
16.	Polyuria and polydipsia	15	10.71

Fig 1: Frothy vomition

Fig 2: Yellow colored vomitus

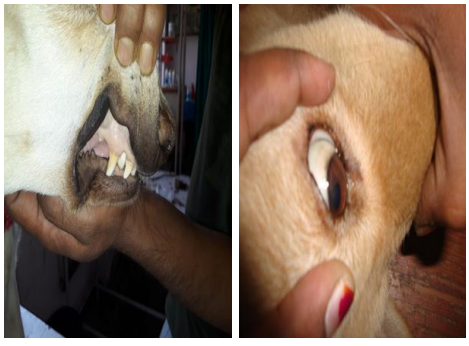

Fig 3: Anemia- Pale buccal and conjunctival mucous membranes

Fig 4: Emaciation

Fig 5: Dark yellow colored urine

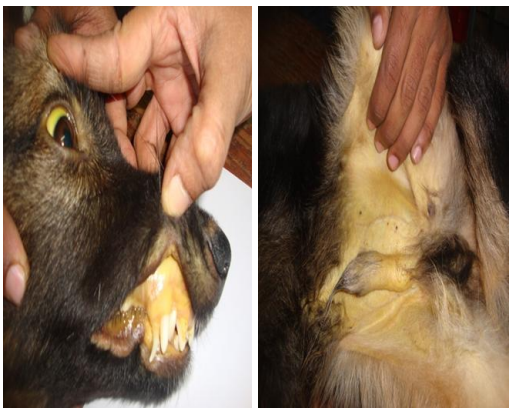

Fig 6: Yellowish discoloration of conjunctival, buccal mucous membranes and ventral abdomen.

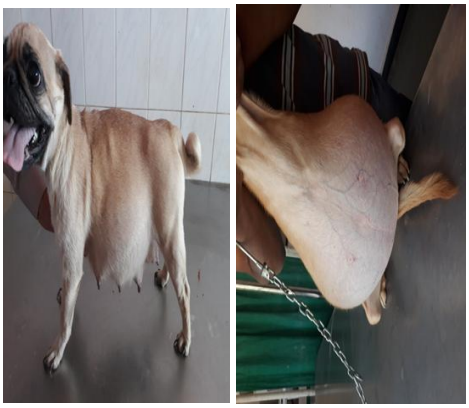

Fig 7: Pear shaped abdomen suggestive of ascites.

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