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Anamnesis and symptomatology: Relevance in diagnosis of canine hepatic dysfunction

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Abstract

The study was aimed to evaluate the relevance of anamnesis and symptomatology in diagnosis of canine hepatic dysfunction. On the basis of preliminary screening, 18 screened dogs suspected for hepatic dysfunction were examined and the observations were made. Complete history of the pet was obtained from the owner. Clinical manifestations recorded in hepatic dysfunction were lethargy to depression, anorexia, vomiting, pale mucosa, diarrhoea, polyuria/polydipsia, pain on abdominal palpation, fever, ascites, icterus, peripheral oedema, emaciation, melena, and petechial haemorrhages. Vomiting was observed in 77.78% dogs and out of these, 44.45% dogs showed yellow vomition while 33.33% showed frothy vomition and 44.44% dogs showed complete anorexia.

Keywords: History, hepatic, anorexia, vomiting

Introduction

Canine hepatic dysfunction is undetected during the early stages and so is a common cause of non-accidental death in dogs. Hepatic dysfunction is visualized in many acute and chronic clinical conditions including infectious, congenital or neoplastic, metabolic diseases, vascular injury, degenerative processes, autoimmune diseases and even blunt trauma. There are numerous causes of liver dysfunction in dogs, including toxin ingestion, drug reactions and infectious agents. A thorough history, including any medications and herbal supplement exposures, should be obtained in order to tailor treatment, including therapy with an antidote when available (Teschke *et al.*, 2012) [17]. The Clinical signs of hepatopathies in canines are highly variable due to the liver's extensive relations with other organs and its unusual regenerative capacity as reported by Dial (1995) [3]. The present work was thus carried out to evaluate the efficacy of drugs in the treatment of canine hepatic dysfunction diagnosed on the basis of anamnesis, symptomatology and clinical alterations. So, this study was conducted to find out the relevance of anamnesis and symptomatology in diagnosis of canine hepatic dysfunction.

Materials and Methods

Canine patients were screened for hepatic dysfunction during the study period at Teaching Veterinary Clinical Complex, C.V.A. Sc, G.B.P.U.A&T, Pantnagar, Udham Singh Nagar, Uttarakhand. On the basis of preliminary screening, 18 screened dogs suspected for hepatic dysfunction were examined and the observations were made.

History taking

Complete history of the pet was obtained from the owner which included age, breed, sex, weight, vaccination and deworming status, history of previous illness (if any) and history of current illness, duration of illness and the treatment given (if any). Clinical signs were noted in chronological order of occurrence especially pyrexia, jaundice, appetite status, vomiting/regurgitation or hematemesis and frequency (if any), epistaxis, defecation status (colour, consistency and frequency), weight loss/ cachexia, water intake, urination status (colour, quantity, frequency and difficulty in urination if any), history of coughing and/or exercise intolerance, abdominal distension, dietary protocol, presence of external parasites, change in attitude, episodes of weakness and seizures or collapse or any other nervous manifestations.

Clinical examination

A thorough physical examination of selected patients was conducted as proposed by Nelson and Couto (2009) [15]. Aspects of the examination included: Rectal temperature, colour of mucus membrane and capillary refill time, heart/arterial pulse rate and hydration status were recorded. Auscultation of the heart (rate, rhythm, adventitious sounds) and lungs was performed and findings were recorded. Patients were also examined for jugular venous pulsation, superficial lymph nodes enlargement and the occurrence of skin bruises, petechiation and/or ecchymoses and hepatocutaneous syndrome. Abdominal palpation and ballottement were performed to check for any form of pain (hepatodynia), fluid accumulation, organomegaly or any abnormal mass. Oral cavity was also examined for ulcerations and abnormal odours. Signs of hepatic encephalopathy (including dementia, seizures, changes in personality and motor disturbances, etc.) also any other indication of reactive hepatopathy were thoroughly observed.

Results and Discussion

The history has an important role to play in the diagnosis of hepatic dysfunction in canines. The clinical signs which are reported in dogs with hepatic dysfunction are non-specific but can include vomition, decreased appetite, diarrhea, dehydration, weight loss and yellowish discoloration in skin, eyes and gums.

The past history of dogs having hepatic dysfunction reported in the present study showed that 1 (5.56%) dog had no previous history of illness, 2 (11.11%) were presented with unknown history, 3 (16.67%) came with a history of progressive abdominal distention and 6 (33.33%) had history of fever (Table 1). Inappetence (partial anorexia) was seen in 12 (66.67%) dogs whereas history of vomition in 9 (50%) dogs and fits in 2 (11.11%) dogs were observed. History of weakness was a complaint in 8 (44.44%) cases. There was history of canine parvovirus in 2 (11.11%), haematochezia in 2 (11.11%), exercise intolerance in 3 (16.67%), tick infestation in 3 (16.67%), hematemesis in 1 (5.56%) and respiratory distress in 4 (22.22%). Other historical findings

like corneal opacity 1 (5.56%), obesity 8 (44.44%), babesiosis 4 (22.22%), epistaxis 1 (5.56%), gastrointestinal parasitism 2 (11.11%), erythema and alopecia 2 (11.11%), marked weight loss 4 (22.22%), pica 1 (5.56%) and abdominal pain 7 (38.89%) were also reported (Table 1, Fig. 1).

History of vaccination and deworming status showed that out of 18 dogs; 7 had regular and proper vaccination, 4 had irregular vaccination, 2 were not vaccinated and 5 were presented with unknown history of vaccination. Similarly, 7 dogs had regular and proper deworming, 4 were irregularly dewormed, 2 were not dewormed and 5 with unknown history of deworming (Table 2, 3).

Table 1: Previous history of dogs with hepatic dysfunction (n=18)

Previous history	Number (n=18)
Unknown	2 (11.11%)
No previous history of illness	1 (5.56%)
Abdominal distension	3 (16.67%)
Fever	6 (33.33%)
Inappetence	4 (22.22%)
Vomiting	9 (50%)
Fits	1 (5.56%)
Weakness	8 (44.44%)
Anorexia	12 (66.67%)
Haematochezia	2 (11.11%)
Exercise intolerance	3 (16.67%)
Tick infestation	3 (16.67%)
Hematemesis	1 (5.56%)
Gastrointestinal parasitism	2 (11.11%)
Respiratory distress	4 (22.22%)
Corneal opacity	1 (5.56%)
Obesity	8 (44.44%)
Babesiosis	4 (22.22%)
Epistaxis	1 (5.56%)
Erythema and alopecia	2 (11.11%)
Weight loss	4 (22.22%)
Pica	1 (5.56%)
Abdominal pain	7 (38.89%)
Canine parvovirus	2 (11.11%)

Figures in parenthesis indicate percentage. (n) Refers to number of dogs.

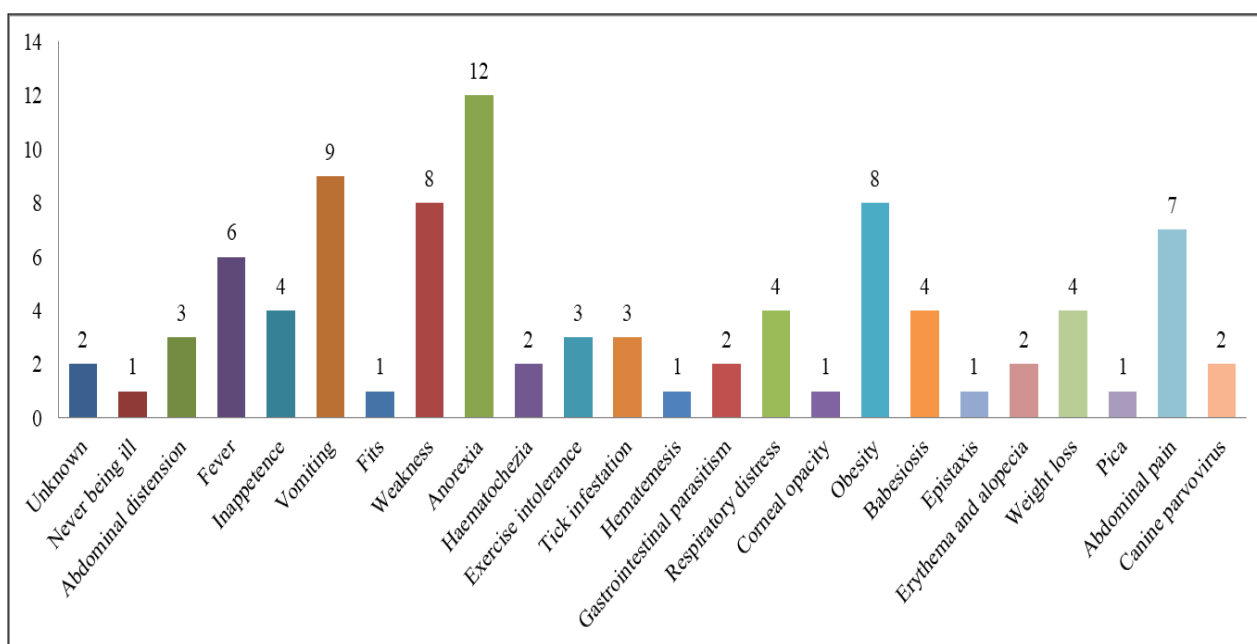


Fig 1: Previous history of dogs with hepatic dysfunction (n=18), where X axis denotes the number of affected animals and Y axis depicts the history of animals.

Table 2: History of vaccination status

Vaccination status	No. of dogs(n=18)
Regular and proper vaccination	7
Irregular vaccination	4
Not vaccinated	2
Unknown	5

Table 3: History of deworming status

Deworming status	No. of dogs
Regular and proper deworming	7
Irregular deworming	4
No deworming	2
Unknown	5

Clinical manifestations

In primary hepatic dysfunction, clinical signs were variable as Holt *et al.* (1995) [11] and Varshney and Hoque (2002) [20] also reported that clinical signs occurring with hepatic dysfunctions were very variable and non-specific compared to neurological and gastrointestinal signs.

Out of 18 dogs with hepatic dysfunction in this study, 8(44.44%) had complete anorexia, 3 (16.67%) dogs had varying degree of inappetence and the remaining 7 (38.89%) dogs had normal appetite status. Bexfield and Watson (2006) [2] and Meyer and Rothuizen (2013) [14] reported anorexia/reduced appetite as a non-specific sign in canine hepatic diseases.

Vomition was observed in 14 (77.78%) dogs and out of these, 8 (44.45%) dogs showed yellow vomition while 6 (33.33%) showed frothy vomition (Table 4). As per Batt and Twedt (1994) [1], vomition occurring with hepatic dysfunction can be due to stimulation of the vomition center by chemoreceptor trigger zone (CRTZ) in the fourth ventricle of the brain by endotoxins that were not metabolized by the damaged liver. This study showed that dogs having hepatic disorders had more signs of inappetence/anorexia, vomition, icterus, depression, abdominal distension, pyrexia, hepatomegaly and hepatodynia which are in concurrence to observations of Kumar and Varshney (2006) [13].

In this study, slight icteric mucus membranes were visible at a minimum plasma bilirubin concentration of 1.9 mg/dL. According to Eddlestone (2000) [5], icterus appears when bilirubin accumulates in the plasma or tissues, but is rarely clinically perceptible until the serum concentration is >1.5 mg/dl. Meyer and Rothuizen (2013) [14] stated that icterus is a frequently observed specific abnormality in dogs with hepatic dysfunction, however approximately 20 percent of dogs having hepatic disease are icteric.

Hepatodynia or pain in the liver is because of stretching of hepatic capsule and is the most important presenting complaint of patients with hepatic disorder as per Dunn *et al.* (1973) [4] and in this study, it was observed in 2 (11.11%) cases. Vomition was observed more frequent compared to diarrhoea in liver affected dogs.

Dogs presented with normal water intake were 3 (16.67%) and it was reduced in 7 (38.89%) dogs and polydipsia was reported in 8 (44.45%) dogs. Polydipsia recorded in the present study can be because of increased ACTH levels which elevate the levels of cortisol thus creating a raised threshold for the release of Arginine Vasopressin which requires a

higher osmolality for stimulating anti diuresis and before reaching that threshold affected dogs become thirsty and start drinking as mentioned by Meyer and Rothuizen (2013) [14].

Skin bruises were reported in 4 (38.89%) cases and were attributed to underlying blood vessels rupture, lowered synthesis of blood coagulation proteins and blood coagulation inhibitors as reported by Feldman (1980) [6]. Unkempt hair coat was seen in 5 (27.78%) cases which might be because of prolonged inappetence/anorexia, poor body condition and gastrointestinal disorders.

Dyspnoea was observed in 3 (16.67%) dogs with severe abdominal distension due to peritoneal effusion. Peripheral oedema was detected in 4 (22.22%) dogs. Abdominal distension was observed in 6 (33.33%) cases of ascites (Table 4). According to Silva *et al.* (2007) [19], in a retrospective study on 80 dogs with hepatic cirrhosis, ascites was found to be the most common clinical finding, followed by icterus, anorexia, neurological disturbances, dyspnoea and subcutaneous oedema. Dyspnoea was attributed to the overpressure of ascitic fluids on the diaphragm and respiratory muscles and in some cases to respiratory tract infection, whereas subcutaneous oedema was a consequence of hypoproteinemia associated with liver disease as mentioned by Hall (1985) [10]. Witte *et al.* (1971) [21] reported that in most cases of cirrhosis, both hepatic (high protein) and mesenteric lymph (low protein) were produced at an increased rate and that ascites developed when the return of lymph to systemic venous circulation failed to keep pace. Sevelius (1995) [18] had also observed that ascites was one of the predominant clinical findings in chronic hepatitis dogs.

Corneal opacity (blue eye syndrome) was observed in 1 (5.56%) young unvaccinated dogs suspected for acute infectious canine hepatitis (ICH) and was completely cured after 4 months of treatment. Infectious canine hepatitis is the only virus with primary tropism for the liver and can cause ocular changes as reported by Kearns (2009) [12].

Exercise intolerance was observed in 2 (11.11%) dogs due to anaemia, heart disease (ascites) or other organ systems complications. Anaemic animals have decreased ability of blood to supply tissues with adequate oxygen for proper metabolic functions. As a consequence there will be lethargy, weakness, exercise intolerance, anorexia, heart murmur, dyspnoea and pale mucous membranes as mentioned by Raskin (1994) [16]. Prolonged capillary refill time is because of dehydration and haemoconcentration.

Table 4: Clinical manifestations of dogs with hepatic dysfunction (n=18)

Parameter	Clinical findings	Total
Appetite status	Normal appetite	7 (38.89%)
	Anorexia	8 (44.44%)
	Inappetence	3 (16.67%)
Vomition	No vomition	4 (22.22%)
	Yellow coloured	8 (44.45%)
	Frothy	6 (33.33%)
Water intake	Normal	3 (16.67%)
	Reduced	7 (38.89%)
	Polydipsia	8 (44.45%)
Hepatodynia	Severe abdominal pain (position of relief)	2 (11.11%)
Skin bruises	Skin erosion and ulceration	4 (22.22%)
Exercise intolerance	Fatigue while exercising	2 (11.11%)
Hair coat	Unkempt hair coat	5 (27.78%)
Abdominal distension	Ascites (with or without organomegaly)	6 (33.33%)
	Severe hepatomegaly	14 (17.78%)
Dyspnoea	Difficult breathing	3 (16.67%)
Corneal opacity	Blue eye syndrome	1 (5.56%)
Peripheral oedema	Swelling of hind limbs and subcutaneous tissues	4 (22.22%)
CRT	<2 Sec	1 (5.56%)
	2 Sec	11 (61.11%)
	>3 Sec	6 (33.33%)

Figures in parenthesis indicate percentage. (n) Refers to number of dogs.

Conclusion

Animals showing history of vomition, anorexia, inappetence and abdominal pain is marked in canine hepatic dysfunction cases. Clinical manifestations like anorexia, polydipsia, icterus and distended abdomen due to hepatomegaly with vomition (yellow coloured) inclines diagnosis towards the involvement of hepatic dysfunction. Thus, if history and symptoms are recorded and observed carefully they provide a direction to the practitioner for diagnosing hepatic dysfunction. Hence, anamnesis and symptomatology plays a very important role in diagnosing hepatic dysfunction in canines.

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