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Prognostic determinants of survival in dogs with cirrhosis: a clinico-pathologic and sonographic approach#

D. Akilkumar[1], Arun George[2*], V. L. Gleeja[3], V. J. Udayasree[1], N. Madhavan Unny[1] and K. S. Prasanna[4]

[1]Department of Veterinary Clinical Medicine, Ethics and Jurisprudence, [3]Department of Statistics, [4]Department of Veterinary Pathology, College of Veterinary and Animal Sciences, Mannuthy, Thrissur, [2]Department of Veterinary Clinical Medicine, Ethics and Jurisprudence, College of Veterinary and Animal Sciences, Pookode, Wayanad. KVASU, Kerala Veterinary and Animal Sciences University, Pookode, Wayanad, Kerala.

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Abstract

Cirrhosis in dogs is the end stage of chronic liver disease, characterised by fibrosis, regeneration and distortion of normal liver architecture leading to portal hypertension and ascites. The present study was carried out at the University Veterinary Hospital, Mannuthy and Kokkalai, Kerala Veterinary and Animal Sciences University, to evaluate the clinicopathological ultrasonographic and ascitic fluid features of hepatic cirrhosis in dogs. Dogs with clinical signs of abdominal distension, icterus, anorexia, vomiting and melaena were examined using ultrasonography. Haemato-biochemical, coagulation and urine analyses were done. Ascitic fluid was collected for biochemical analysis and bacterial culture. Ten healthy dogs were used as control. Eight dogs were diagnosed with hepatic cirrhosis. Abdominal distension, hyporexia/anorexia and melaena were the predominant clinical signs. Haematological analysis revealed marked anaemia and biochemical findings showed significant elevation of alkaline phosphatase and gamma-glutamyl transferase. Ascitic fluid was predominantly transudate with low protein content (<2.5 g/dL). Ultrasonography consistently revealed a shrunken hyperechoic liver with irregular margins, hypoechoic nodules and free abdominal fluid. Erythrogram values, granulocyte count, albumin, ALP and GGT were significantly correlated with survival. Clinical features including distended abdomen, hyporexia, melaena, haematochezia, dyspnoea and all ultrasonographic criteria also showed significant association with survival time. Cox proportional hazard analysis identified albumin, leucocyte count and glucose as the major prognostic determinants of survival. To conclude, cirrhotic dogs with concurrent leukocytosis, low serum albumin and elevated serum glucose have relatively less chance of survival than cirrhotic dogs with normal leukocyte count, serum albumin and serum glucose.

Keywords: *Liver, cirrhosis, ascites, ultrasonography, prognosis*

Liver is one of the highly active organs in the body performing multiple vital functions (Neagu *et al*., 2024). Injury to liver results from bacteria, virus, drugs, toxins and environmental contaminants (Webster *et al*., 2019). Liver is constantly exposed to toxins from gastrointestinal tract through blood via the portal vein. Dogs are exposed to different toxicants from their immediate environment leading to liver injury. Hepatic injury may be acute or chronic depending upon

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**Corresponding author: arun@kvasu.ac.in, Ph. 9447747992*

the type and number of agents or factors involved. Any damage to liver may or may not result in obvious clinical signs due to its large reserve capacity and regenerative power. Moreover, it also depended on the extent of hepatic damage and whether the lesion is focal, multifocal or diffused. Long-term exposure to toxin may result in chronic hepatopathy. Fibrosis is a mechanism, similar to wound healing response to injury, with alteration of hepatic environment including change in cytokine levels, activation of hepatic stellate cells and synthesis of extracellular matrices. Chronic fibrosis results in end stage liver disease called cirrhosis (Eulenberg and Lidbery, 2018).

Clinicals signs are often nonspecific in liver diseases until the reserve capacity of synthetic function becomes exhausted. Hypoproteinemia and portal hypertension together result in ascites. Icterus could be noticed in final stage of cirrhosis. Melaena, vomiting, cachexia, anorexia and obtundation are the other common clinical signs exhibited by cirrhotic dogs. Acquired portosystemic shunts may develop and result in hepatic encephalopathy (Favier, 2009).

Hematobiochemical analysis is the primary laboratory tool in the diagnosis of liver diseases but lacks specificity. Ultrasonography is a noninvasive imaging technique to establish the diagnosis of cirrhosis (Tantary *et al*., 2014). Biopsy-cum-histopathology is the standard technique for the diagnosis of cirrhosis, however, it carries practical difficulty and risks like bleeding (Rothuizen, 2006). Hepatic diseases could be explored from a diagnostic as well as a prognostic approach. Though the diagnostic approach with determination of likely predisposing and precipitating factors are imperative for the veterinary clinician; prognosis and survivability are the key concerns of pet owners. With this objective in mind, a study was conducted to determine the clinico-pathological and sonographic prognostic determinants of survival in dogs with cirrhosis.

Materials and methods

The study was conducted in the University Veterinary Hospitals at Mannuthy and Kokkalai, in the Thrissur district of the state of Kerala in India. Dogs presented with clinical signs of distended abdomen, icterus, chronic inappetence, vomiting and melaena were screened for cirrhosis with ultrasonography as per Mattoon and Nyland (2015). A total of eight dogs with cirrhosis were identified and recruited for the study. Whole blood was collected in K3EDTA for haematological analysis in Orphee mythic vet haematology analyser and in citrate vial for coagulation analysis in Erba ECL 412 analyser. Blood in clot activator vial was sent for biochemical analysis in Hospitex screen master T semi-automatic biochemical analyser using kits from Alpha diagnostics. Serum zinc and copper were estimated using biochemical kits from Tulip Diagnostics. Dipstick analysis of urine was done in YD Uriscan optima. Ultrasonography of liver was done using My Lab X8eXP, Esaote machine. Ultrasonographic images of hepatobiliary

Table 1. Criteria for sonographic scoring of hepatobiliary abnormalities

Major criteria	Minor criteria (score)	Score
Liver echogenicity	Diffusely hypoechoic with increased visibility of portal markings	1
	Diffusely hyperechoic with decreased visibility of portal markings	3
	Less than that of kidney cortex	1
	Hyperechoic patches with ill-defined areas of echotexture variation	3
	Hypoechoic patches with ill-defined areas of echotexture variation	3
Liver echotexture	Normal	1
	Coarser but not patchy	2
	Patchy but not nodular	3
	Nodular	3.5
Portal venous clarity	Clear	1
	Unclear	2
Liver borders	Smooth	1
	Irregular	2
Liver edges	Sharp	1
	Rounded	2
Gall bladder wall thickness	Less than 2 mm	1
	2 mm to 3 mm	2
	Above 3 mm	3
Echogenic contents of gall bladder	None	0
	Aggregate sludge (2)	2

Table 2. Demographic parameters

Case No.	Age	Breed	Gender	Body weight (kg)
D1	12	Non-descript	Female	18.2
D2	6	Non-descript	Male	13.2
D3	3	Indian Spitz	Female	7.6
D4	14	Indian Spitz	Female	4.5
D5	6	Non-descript	Male	16
D6	7	Labrador Retriever	Female	22.6
D7	7	Lhasa apso	Female	9.8
D8	13	Indian Spitz	Male	8.9

Table 3. Haematological parameters of control and cirrhotic dogs

Parameters	Diseased dogs		Healthy control		t-value	p-value
	n	Mean ± SE (Range)	n	Mean ± SE (Range)		
Total erythrocyte count (10^6/µL)	8	4.45 ± 0.54 (2.09 – 6.92)	10	6.24 ± 0.20 (5.48 – 7.33)	3.35**	<0.01
Haemoglobin (g/dL)	8	9.06 ± 1.32 (4.3 – 16.2)	9	14.60 ± 0.28 (13.7 – 15.5)	4.33**	<0.01
Volume of packed red cells (%)	8	27.17 ± 3.59 (12.9 – 46.9)	10	40.91 ± 1.27 (32.2 – 45.3)	3.92**	<0.01
Mean Corpuscular Volume (µm^3)	8	61.21 ± 2.06	10	65.74 ± 5.05	1.76	0.09
Mean Corpuscular Haemoglobin (pg)	8	20.20 ± 3.31	10	23.04 ± 2.05	2.23*	0.04
Mean Corpuscular Haemoglobin concentration (g/dL)	8	32.92 ± 2.78	10	34.93 ±1.81	1.84	0.08
Total leucocyte count (10^3/µL)	8	19.66 ± 3.63 (8.8 – 34.8)	10	11.86 ± 1.02 (6.1 – 15.5)	2.06	0.07
Absolute granulocyte count (10^3/µL)	8	16.03 ± 3.03 (6.5 – 29.3)	10	8.64 ± 0.98 (5.0 – 13.3)	2.32*	0.04
Absolute lymphocyte count (10^3/µL)	8	2.53 ± 0.85 (0.54 – 2.2)	9	1.98 ± 0.21 (1.2 – 3.4)	0.63	0.54
Absolute monocyte count (10^3/µL)	8	1.06 ± 0.28 (0.2 – 2.3)	10	0.64 ± 0.06 (0.4 – 1.0)	1.41	0.19
Total platelet count (10^3/µL)	8	377.25 ± 102.49 (46 - 815)	10	295.60 ± 31.08 (166 - 495)	0.76	0.46

**Significant ($p \leq 0.01$), *Significant ($p \leq 0.05$)

abnormalities were scored using the criteria of Feeney *et al*. (2008). Criteria followed for scoring is given in Table 1 as per Feeney *et al*. (2008). Ascitic fluid was collected under ultrasonographic guidance and the biochemical analytes such as total protein and albumin were estimated. Bacterial culture of ascitic fluid was done in brain heart infusion agar. Nucleated cell count was done manually in Neubauer's haemocytometer. Standard therapy was given to all the dogs and survival time was recorded. Ten healthy animals brought for vaccination or general health checkup served as control group. The survival status of all the dogs was monitored.

Haemato-biochemical and coagulation parameters of cirrhotic and healthy dogs were compared using students t-test after removing the outliers. All these parameters were subjected to Spearman's rank correlation in order to determine the prognostic factors. Clinical features and ultrasonographic criteria were analysed to find the association with survivability using log rank test. Cox proportional hazard analysis was used to find the parameters that significantly affected survival. All the statistical tests were done using SPSS 20.0.

Results and discussion

The mean age at diagnosis of cirrhosis was 8.5 years with age ranging from three to 14 years (median age – 7 years). Five were female and three were male dogs. Indian spitz type (3/6), non-descript (3/6), Labrador Retriever (1/6) and Lhasa Apso (1/6) were the breeds of dogs diagnosed with cirrhosis. The distribution of age,

Table 4. Comparison of biochemical and coagulation parameters

Parameters	Diseased dogs		Healthy control		t-value	p-value
	n	Mean ± SE (Range)	n	Mean ± SE (Range)		
Alanine aminotransferase (U/L)	7	29.27 ± 6.39 (12 – 54.9)	9	19.44 ± 2.54 (12 - 24)	1.42	0.19
Aspartate aminotransferase (U/L)	8	32.43 ± 9.3 (5 – 77.5)	10	20.17 ± 2.47 (10 - 36)	1.26	0.24
Alkaline Phosphatase (U/L)	8	135.72 ± 34.54 (12 – 261.9)	8	11.25 ± 0.81 (7 - 14)	3.60**	<0.01
Gamma Glutamyl Transferase (U/L)	8	7.87 ± 0.78 (5 - 12)	9	1.57 ± 0.43 (0 - 2)	7.38**	<0.01
Total protein (g/dL)	8	5.08 ± 0.27 (4.2 – 6.02)	10	5.88 ± 0.40 (4.1 – 7.3)	1.57	0.13
Albumin (g/dL)	8	2.19 ± 0.29 (1.0 – 3.2)	10	2.88 ± 0.11 (2.5 – 3.5)	2.17	0.057
Total bilirubin (mg/dL)	8	0.92 ± 0.29 (0.06 – 2.17)	10	0.03 ± 0.30 (0.1 – 0.5)	2.11	0.07
Direct bilirubin (mg/dL)	8	0.26 ± 0.08 (0.02 – 0.52)	10	0.18 ± 0.02 (0.1 – 0.36)	0.96	0.34
Blood urea nitrogen (mg/dL)	8	37.09 ± 15.06 (6.8 – 105.2)	10	8.75 ± 1.18 (5.19 – 16.85)	1.87	0.10
Creatinine (mg/dL)	8	1.4 ± 0.22 (0.8 – 2.4)	10	1.0 ± 0.05 (0.7 – 1.2)	1.73	0.12
Glucose (mg/dL)	8	74.43 ± 6.29 (57 - 101)	10	64.70 ± 4.82 (50 - 108)	1.24	0.23
Serum Copper	8	101.22 ± 21.82 (49.5 – 208.2)	10	75.91 ± 12.12 (31.6 – 119.5)	1.09	0.29
Serum Zinc	8	100.95 ± 19.97 (48 – 177.89)	8	179.04 ± 30.30 (103 – 390.9)	1.91	0.07
Prothrombin time (sec)	7	10.52 ± 1.02 (7.7 – 15.16)	10	8.32 ± 0.42 (5.9 – 10.5)	1.98	0.08
Activated partial thromboplastin time (sec)	7	21.8 ± 3.19 (12.04 – 32.16)	10	20.34 ± 1.01 (14.92 – 24.92)	0.51	0.67

**Significant ($p \leq 0.01$)

breed, gender and body weight is given in Table 2.

The complaints of pet owners on the day of presentation were distended abdomen (8/8), hyporexia/ anorexia (7/8), melaena (4/8), dyspnoea (4/8), vomiting (1/6), recumbency (2/8), haematochezia (2/8) and hypersalivation (1/8). Ascites, dyspnoea, cachexia with muscle atrophy and limb oedema were the other findings recorded in this study. Seizures, often associated with hepatic encephalopathy, have not been reported in dogs in this study. The major clinical signs reported by Favier (2009) in dogs with cirrhosis were vomiting, jaundice, weight loss and signs of encephalopathy occasionally. Ascites (8/8), pale mucous membrane (4/8), cachexia with muscle atrophy (4/8) and limb oedema (1/8) were the other findings recorded during clinical examination.

Haematological parameters of cirrhotic and healthy dogs are given in Table 3. Significantly lower ($p \leq 0.01$) mean total erythrocyte count, haemoglobin level and volume of packed red cells were noticed in cirrhotic dogs than healthy controls. A reduction in red cell mass was observed in dogs affected by hepatobiliary disorders (Tantary *et al*., 2014). Anaemia in liver disorders could be due to altered iron metabolism, blood loss, reduced production and hemolysis (Lawrence and Steiner, 2017; Joseph *et al*., 2023). Non-significant difference in MCV and significantly lower MCH in cirrhotic dogs indicate normocytic hypochromic anaemia. Normocytic hypochromic anaemia has been reported in copper and vitamin B12 deficiency in dogs (Sastry and Rao, 2019). Copper was not identified as a major cause of anaemia in the current study and vitamin B12 could not be estimated. The mean absolute granulocyte count (16.03 x 10^3/µL) was significantly higher in cirrhotic dogs than control group (8.64 x 10^3/µL) at $p<.05$ whereas the mean total leukocyte count showed a non-significant increase. Leucocytosis with granulocytosis was noticed in a study conducted in dogs with cirrhosis by Phom *et al*. (2019). Serum biochemical parameters and coagulation parameters are listed in Table 4 and figure 1. Significantly higher ($p \leq 0.01$) level of mean alkaline phosphatase (ALP) and gamma glutamyl transferase (GGT) was noticed in cirrhotic dogs. ALP and GGT are induction enzymes which increase during cholestasis, steroid exposure and cholangiocellular

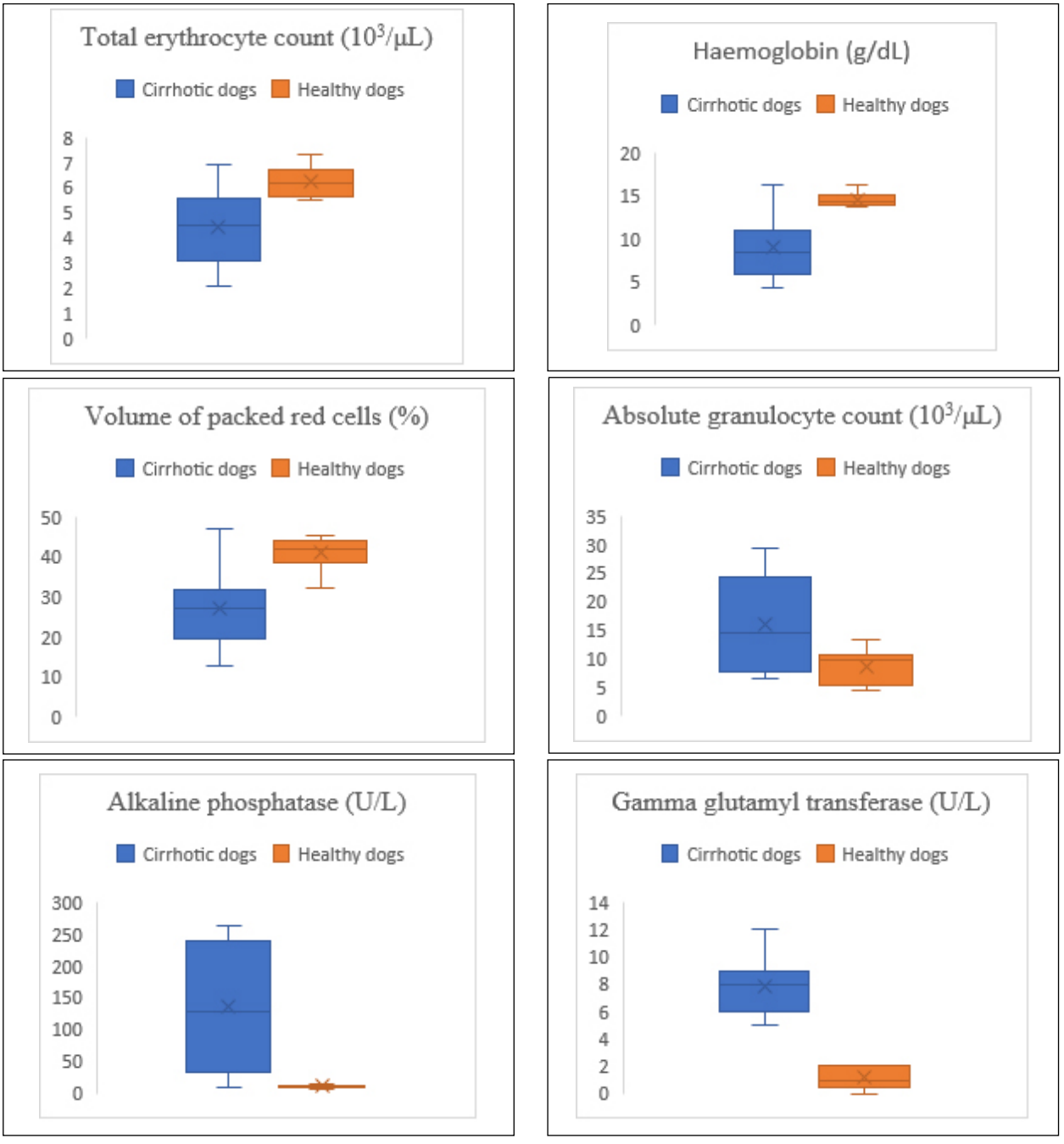

Fig 1. Box and Whisker plot representation of haemato-biochemical parameters that are significantly different from healthy controls

damage (Center, 2007). Increase in ALP and GGT was reported in chronic form of cholangiohepatitis and cirrhosis in dogs (Sevelius, 1995). Brennan *et al*. (2022) reported that increased GGT was a response to increased oxidative stress and reduced glutathione concentration. Relative increase in ALP and GGT were reported in end stage liver diseases when compared to ALT (Webster *et al*., 2019).

Coagulation parameters such as prothrombin time and activated partial thromboplastin time were not significantly different from the healthy controls.

On ultrasonography, liver appeared shrunken with lobes floating freely in the anechoic free fluid in the abdomen (Fig. 2). Other hepatic sonographic findings (Table 5) were diffusely hyperechoic hepatic parenchyma with coarser, patchy and nodular echotexture. Portal veins were unclear in all the cases. Liver borders were either smooth or irregular and liver edges were either sharp or rounded. Similar findings were reported by Phom *et al*. (2019) and Elhiblu *et al*. (2015) in dogs with cirrhosis. Mattoon and Nyland (2015) reported anechoic abdominal fluid, hyperechoic liver parenchyma, reduced visibility

Table 5. Ultrasonographic findings in cirrhotic dogs

Major criteria	Minor criteria	Number of animals (n= 8)
Liver echogenicity	Diffusely hyperechoic with decreased visibility of portal markings	8
Liver echotexture	Coarser but not patchy	3
	Patchy but not nodular	3
	Nodular	2
Portal venous clarity	Unclear	8
Liver borders	Smooth	2
	Irregular	6
Liver edges	Sharp	3
	Rounded	5
Gall bladder wall thickness	Less than 2 mm	1
	2 mm to 3 mm	5
	Above 3 mm	2
Echogenic contents of gall bladder	None	0
	Aggregate sludge	3

of portal vessels, reduced size of liver and hypoechoic regenerative nodules as the findings of cirrhosis. Diffusely hyperechoic parenchyma with unclear portal veins and irregular liver borders were the consistent findings in all dogs. Lisciandro (2020) reported that diffusely hyperechogenic liver was seen consistently in dogs with cirrhosis. Webster *et al*. (2019) reported that irregular liver borders were noticed in chronic liver diseases and worsened as condition progressed. Echotexture was truly nodular in two dogs, coarser in two and patchy with near nodular in four cases. Heterogenous altered echotexture had been noted in dogs with cirrhosis (Elhiblu *et al*., 2015; Phom *et al*., 2019).

Sonographic scoring was done as per Feeny *et al*. (2008) and it ranged from 11 to 16 in the eight diseased dogs. Three dogs had sonographic score of 16. The other five dogs were scored 11, 12, 13, 13.5 and 14.5.

Ascites was noticed in all dogs diagnosed with cirrhosis. Results of ascitic fluid analysis are listed in Table 6. Ascites was a predominant clinical finding in dogs with cirrhosis. According to Umesh *et al*. (2025), ascites was usually seen in cases of cirrhosis and heart failure. Mechanism of ascites formation was multifactorial in cirrhosis *viz*, hypoproteinemia with reduced synthetic function of liver, portal hypertension due to structural changes in liver parenchyma, and activation of renin-angiotensin-aldosterone system **(**Eulenberg and Lidbery, 2018). Specific gravity of ascitic fluid was not measured in the present study and hence classification of ascitic fluid was based on total protein level and nucleated cell count as per Dewhurst (2016). Nucleated cell count of all the cases was less than 1500 cells/ µL. Protein rich transudate (> 2.5 mg/dL) and protein poor transudate (< 2.5 mg/dL) were noticed in two and six cases, respectively. Liver disorders and cirrhosis were associated with either type of transudate (Dewhurst, 2016). No bacterial growth was noticed on microbial culture in any of the eight dogs. Saravanan *et al*. (2014) reported similar findings in dogs with cirrhosis. Serum ascites albumin gradient (SAAG) above 1.1 g/dL is an indirect measure of portal hypertension. In the present study, SAAG value above 1.1 g/dL was noticed in six cases.

Urinalysis revealed varying levels of microscopic haematuria, proteinuria, urobilinogenuria, glucosuria and ketonuria in dogs with cirrhosis. Results of urinalysis are listed in Table 7. Hyposthenuria with specific gravity less than 1.008 was noticed in four dogs and similar findings were previously reported (Alleman and Wamsley, 2017). Microscopic haematuria and proteinuria were recorded previously in dogs with liver disorders and were attributed to glomerular damage in chronic inflammatory diseases (Parrah *et al*., 2013; Thelagar, 2017).

Haemato-biochemical parameters were subjected to Spearman's rank correlation analysis (Table 8) and the following parameters correlated negatively to the days of survival *viz*. total erythrocyte count, haemoglobin, volume of packed red cells, absolute granulocyte count, ALP and GGT. Serum albumin correlated positively with the number of days of survival. Neutrophilia was previously reported as negative prognostic indicator in dogs with liver diseases (Poldervaart *et al*., 2009; Breheny *et al*., 2020). Increased GGT levels were associated with poor survival in dogs with chronic liver diseases in a study conducted by Assawarachan *et al.* (2023). Anaemia was a feature of cirrhosis (Lawrence and Steiner, 2017) which had

Table 6. Results of ascitic fluid analysis

Sl.No.	Colour	Nucleated cell count (cells/ µL)	Total protein (g/dL)	Albumin (g/dL)	SAAG (g/dL)
D1	Clear, transparent	63	2.3	0.4	1.2
D2	Clear, transparent	13	2.4	0.4	0.6
D3	Clear, transparent	125	1.1	0.7	1.2
D4	Clear, yellow	50	0.1	0.1	3
D5	Clear, transparent	500	2.8	0.5	2.7
D6	Clear, transparent	750	1.4	0.2	2.9
D7	Clear, transparent	75	5.3	1.2	0.5
DS8	Clear, transparent	30	1.1	0.5	1.3

Table 7. Observations of urine dipstick analysis

Parameters		Nil (-)	Trace (+/-)	Mild (+)	Moderate (++)	Severe (+++)
Number of animals	Blood	2	1	2	1	2
	Bilirubin	7	1	-	-	-
	Urobilinogen	1	1	6	-	-
	Ketone bodies	6	2	-	-	-
	Protein	1	1	2	3	1
	Glucose	6	-	1	1	-

negative effect in survival of dogs with cirrhosis. Tantary *et al*. (2014) recorded association between jaundice/ hyperbilirubinemia with survival whereas Assawarachan *et al.* (2023) had found no association between them. This is in harmony with the present study and icterus has no association with days of survival in cirrhotic dogs. Shih *et al*. (2007) concluded that albumin had no role in predicting the prognosis of dogs with chronic hepatitis. But in the present study, five dogs had hypoalbuminemia <2.0 g/dL and albumin correlated positively with the number of days of survival in the present study.

The clinical and sonographic features of prognostic importance were explored using log rank test. A comparison of the number of days of survival of cirrhotic dogs based on clinical as well as sonographic features was done using log rank test and the details are given in Table 9. Among the clinical features ascites, hyporexia, melaena, dyspnoea, hematochezia, pale mucous membrane and cachexia had statistically strong association with poor survival of cirrhotic dogs. Gastrointestinal ulcers associated with liver diseases could be responsible for melaena, haematochezia and one of the possible causes of anemia (Webster *et al*., 2019). Though anaemia was not previously reported to have prognostic significance in liver diseases, it was a consistent finding in dogs with liver diseases (Loan *et al*., 2025). Ascites was considered a negative prognostic indicator in chronic liver diseases (Raffan *et al*., 2009). Calleja *et al*. (2023) found no significant association between the presence of ascites and survival in dogs with intrahepatic portal hypertension. Ascites was invariably seen in all dogs of this study because ascites is a complication of end stage cirrhosis.

The analysis of sonographic scores using log rank test revealed significant ($p \leq 0.01$) association with the survival. As expected, animals with high score tend to have earlier death than dogs with lower scores. Similarly, increased echogenicity, variations in echotexture, reduced portal venous clarity, irregular borders as well as thickened liver edges, and thickened wall of gall bladder have been associated with poor survival and the details are given in Table 8. Assawarachan *et al*. (2019) attempted hepatobiliary scoring in dogs with liver diseases, however prognosis had not been determined in their study. The current study involving ultrasound scores targeted successfully in predicting both severity and prognosis.

Cox proportional hazards analysis is a statistical technique in survival analysis that investigates the relationship between a set of predictor variables and the time until a specific event occurs, such as death. This model helps to determine the factors that influence survival and to what extend it contributes to the outcome of death. Clinico-haemato-biochemical parameters as well as sonographic findings were analysed using the Cox proportional hazards model with the forward likelihood ratio method. The omnibus test for model coefficients showed overall significance ($p = 0.016$, $p < 0.05$). Variables in the equation are given in Table 10.

The final model retained total leucocyte count, albumin and glucose as significant predictors of survival. Albumin had a negative regression coefficient with a hazard ratio of 0.11, indicating a protective effect, where normal albumin levels reduced the risk of death. In contrast, cirrhotic dogs with higher total leucocyte counts were 1.27 times more likely to have reduced survival and those with higher glucose levels were 1.08 times more likely to have reduced survival. In short, total leukocyte count, serum albumin and glucose were the important prognostic determinants of survival in dogs with cirrhosis. In other words, cirrhotic dogs with concurrent leukocytosis, low serum albumin and elevated serum glucose have poor chance of survival than cirrhotic dogs with normal leukocyte count, serum albumin and serum glucose.

It is worth noting that five dogs out of eight were less than seven years of age and in these dogs, hepatic

Table 8. Spearman's rank correlation analysis of haemato-biochemical and coagulation parameters

Parameters	Survival (days)	
	Correlation coefficient	p-value
Total erythrocyte count	- 0.580	0.012
Haemoglobin count	- 0.617	0.006
Volume of packed red cells	- 0.576	0.012
Absolute granulocyte count	- 0.518	0.028
ALP	- 0.637	0.004
GGT	- 0.852	0.000
Albumin	+ 0.502	0.034

dysfunction cannot be attributed to age factor. This warrants investigation for other factors associated with hepatic injury and with this objective the serum levels of copper and zinc were determined. However, in this study the mean levels of serum copper and zinc were not different from that of the healthy controls. It must be noted that estimation of the level of copper and zinc in liver biopsy samples is the best method to rule out hepatic toxicity associated with these metals. Other possible aetiological factors are environmental contaminants or pollutants or toxins. In the present study two dogs were found to have chronic exposure to paints and related chemicals and two dogs had chronic exposure to chocolates. Brauner *et al*. (2020) reported that exposure to organic solvents in paints, thinners, resins, varnish, dyes, plastics etc. could cause hepatopathy leading to hepatic steatitis and cirrhosis.

Table 9. Log rank test of clinical features and sonographic score

Parameters	p-value
Clinical signs	
Distended abdomen	0.000
Hyporexia	0.002
Melaena	0.011
Dyspnoea	0.005
Haematochezia	0.016
Fluid thrill	0.000
Pale mucous membrane	0.000
Cachexia	0.002
Hepatic sonogram	
Liver echogenicity	0.000
Liver echotexture	0.000
Portal venous clarity	0.000
Liver borders	0.000
Liver edges	0.003
Gall bladder wall thickness	0.000
Sonographic score	0.000

One of the limitations of this study is that a confirmative diagnosis of cirrhosis by biopsy and histopathology was not attempted. Large scale studies on prognostic significance in dogs with liver diseases would further validate this study. A study on the association of environmental contaminants and hepatopathy in dogs seems to be the need of the hour.

Conclusion

Canine cirrhosis is a progressive liver disorder with distinct clinical, hematological, and ultrasonographic changes. The study identifies abdominal distension, anorexia, melaena, dyspnoea and cachexia as clinical features associated with poor survival. However, seizure which is an indicator of poor survival has not been reported in this study. Low red cell mass, high granulocyte counts,

Table 10. Cox proportional hazards model in dogs with cirrhosis

Parameters	B	SE	Wald	df	Sig.	Hazard ratio	95% CI for HR
Total leucocyte count	0.244	0.155	2.485	1	0.115	1.277	0.942- 1.730
Serum Albumin	-2.163	1.361	2.525	1	0.112	0.115	0.008- 1.657
Serum Glucose	0.081	0.044	3.437	1	0.064	1.085	0.995- 1.182

Table 11. Haemato-biochemical and coagulation analysis in cirrhotic dogs

Case No.	RBC	HGB	VPRC	TLC	AGC	ALC	AMC	PLT	ALT	AST	ALP	GGT	TP	ALB	TB	DB	BUN	CRE	GLU	PT	APTT	Cu	Zn	Survival
D1	4.58	7.8	24.2	11.6	8.6	2.2	0.7	679	21	17	106.9	9	5.93	1.75	1.79	0.39	6.8	0.8	73	8.06	12.04	208.3	761.4	77
D2	4.94	8.8	27.2	8.8	6.5	2	0.3	67	135.7	5	261.9	9	6.02	1.65	2.17	0.52	105.2	2.4	151	12.02	28.52	74.38	82.33	12
D3	2.77	5.2	17.9	34.8	25	7.8	2.1	266	23	67	252	8	4.5	1	1.57	0.13	8.8	0.92	101	12.2	30.2	-	-	7
D4	6.92	16.2	46.9	24.1	21.9	1.7	0.5	613	12	13	95	12	4.7	1.9	0.26	0.02	103.4	1.1	86	7.71	18.12	120.9	142.9	20
D5	4.49	8.3	26.9	8.8	7.6	1	0.2	46	54.9	77.5	196.5	8	4.2	3.1	0.95	0.7	35	2.4	58	15.16	19.48	49.5	80.39	106
D6	5.75	10.9	32.9	21.2	15.9	4.2	1.1	325	27	29	10.5	6	5.12	3.2	0.06	0.03	7.75	1.11	76	9.03	12.28	67.26	74.03	63
D7	4.1	11	28.5	15.3	13.5	0.54	1.2	815	16	15	12	6	4.27	1.89	0.32	0.22	7.78	1.14	51	-	-	136.1	177.9	49
D8	2.09	4.3	12.9	32.7	29.3	0.87	2.38	207	51	36	151	5	5.9	3.1	0.25	0.12	22	1.4	76	9.46	32.16	52.2	48.09	33

TEC- Total erythrocyte count (10^6/µL), HGB- Haemoglobin (g/dL), VPRC- Volume of racked red cells (%), TLC- Total leucocyte count (10^3/µL), AGC- Absolute granulocyte count (10^3/µL), ALC- Absolute lymphocyte count (10^3/µL), AMC- Absolute monocyte count (10^3/µL), PLT- Platelet count (10^3/µL), ALT- Alanine aminotransferase (U/L), AST- Aspartate aminotransferase (U/L), ALP- Alkaline Phosphatase (U/L), GGT- Gamma glutamyl transferase (U/L), TP- Total protein (g/dL), ALB- Albumin (g/dL), TB- Total bilirubin (mg/dL), DB- Direct bilirubin (mg/dL), BUN- Blood urea nitrogen (mg/dL), CRE- Creatinine (mg/dL), PT- Prothrombin time (sec), APTT- Activated partial thromboplastin time (sec), Cu- Copper (µg/dL), Zn- Zinc (µg/dL). Survival – Number of days survived.

* Outliers (not included for statistical analysis)

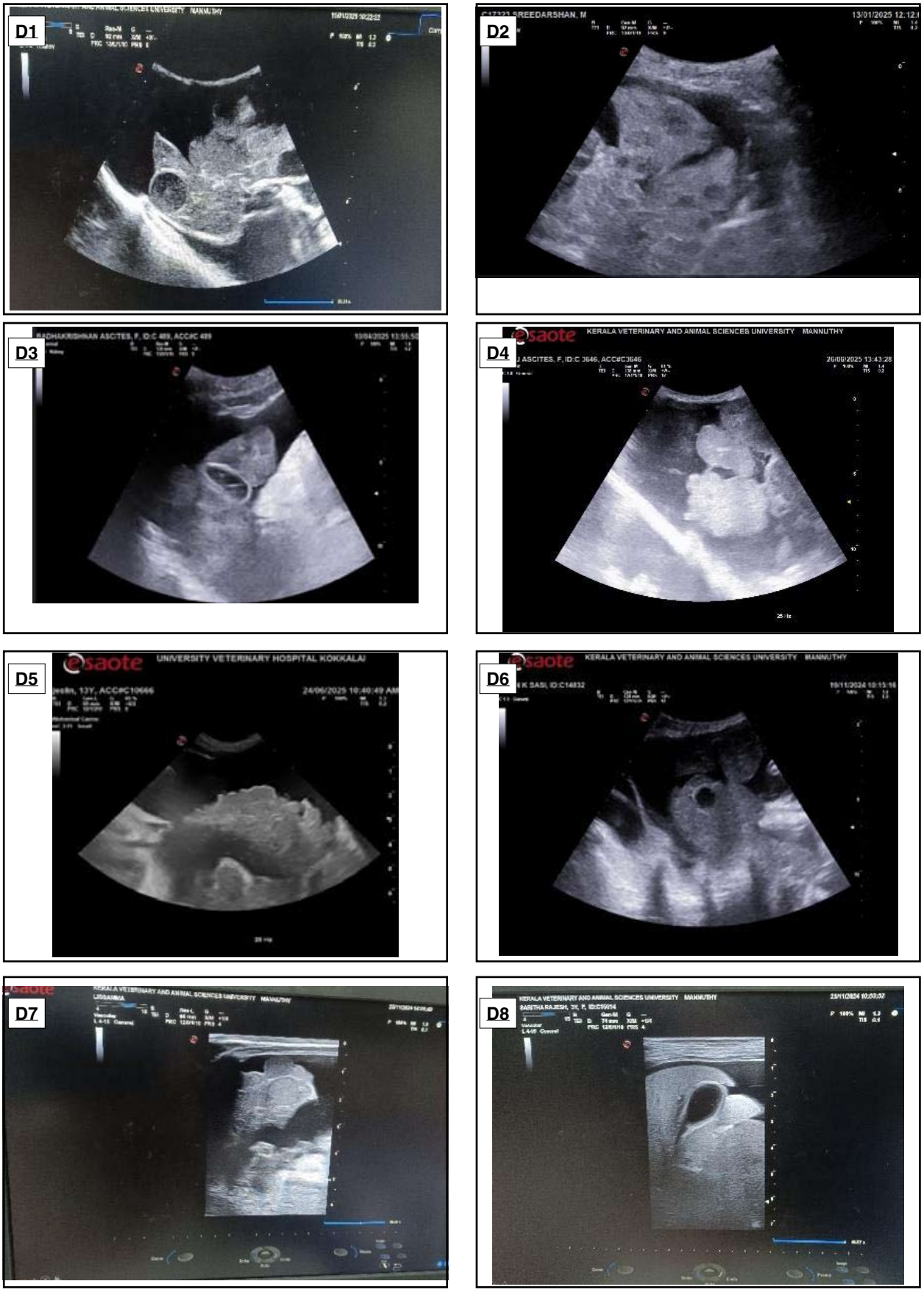

Fig.2. Ultrasonographic findings of cirrhosis

elevated ALP as well as GGT and reduced serum albumin levels were the haemato-biochemical parameters that strongly influenced prognosis. Hepatic hyper echogenicity, irregular borders and heterogeneous echotexture were the features found consistent on diagnostic ultrasonogram in dogs with cirrhosis. Overall, integration of clinical signs, laboratory findings, and ultrasound scoring provides reliable prognostic information in canine cirrhosis. To conclude, cirrhotic dogs with concurrent leukocytosis, low serum albumin and elevated serum glucose have relatively less chance of survival than cirrhotic dogs with normal leukocyte count, serum albumin and serum glucose.

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Conflict of Interest

The authors declare no conflicts of interest.

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