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Serum biochemical derangements and organ dysfunction patterns across the spectrum of naturally occurring sepsis in dogs: A prospective observational study

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Abstract

Sepsis in dogs is associated with dysregulated host response and progressive organ dysfunction. Early identification of biochemical derangements may facilitate timely therapeutic intervention and risk stratification. This study was conducted to characterize serum biochemical alterations across the clinical continuum of sepsis, severe sepsis, and septic shock in dogs and to evaluate their association with organ dysfunction severity. A prospective observational study was conducted in 103 client-owned dogs with naturally occurring sepsis admitted to a tertiary veterinary critical care unit. Dogs were categorized as sepsis ($n = 36$), severe sepsis ($n = 57$), or septic shock ($n = 10$) based on SIRS criteria, confirmed infectious source, organ dysfunction, and hemodynamic status. Ten healthy dogs served as controls. Serum biochemical parameters were analyzed using automated analyzers. Data were compared using one-way ANOVA with post-hoc testing ($\alpha = 0.05$). A significant increase in BUN was observed in severe sepsis (99.6 ± 8.1 mg/dL) and septic shock (104 ± 19.8 mg/dL) compared to controls (15.6 ± 2.1 mg/dL; $p < 0.01$). Creatinine was markedly elevated in severe sepsis (5.3 ± 0.7 mg/dL; $p < 0.01$). Hypoalbuminemia was present across all septic groups ($2.2\text{--}2.4$ g/dL vs. 3.0 ± 0.1 g/dL; $p < 0.01$). ALP activity was significantly increased in all septic categories ($p < 0.01$). Hyperphosphatemia was pronounced in severe sepsis and septic shock (10.1 ± 0.6 and 9.8 ± 1.24 mg/dL; $p < 0.01$). Electrolytes and glucose showed wide variability but no consistent group-level statistical differences. Renal dysfunction (azotemia), hypoalbuminemia, cholestatic enzyme elevation, and hyperphosphatemia are prominent biochemical features of canine sepsis and correlate with disease severity. Routine serum biochemistry provides clinically actionable markers for early organ dysfunction recognition in septic dogs.

Keywords: SIRS, sepsis, CCU, serum, biochemistry, dogs

1. Introduction

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection (Singer *et al.*, 2016; Evans *et al.*, 2021) ^[1, 2]. In dogs, sepsis remains a leading cause of ICU morbidity and mortality, particularly in cases associated with pyometra, gastrointestinal perforation, pneumonia, and systemic inflammatory syndromes (de Laforcade and Shaw, 2020) ^[3].

Recent veterinary literature emphasizes the importance of early organ dysfunction recognition rather than reliance solely on SIRS criteria (Evans *et al.*, 2021) ^[2]. Biomarkers such as lactate, C-Reactive Protein, procalcitonin, and cytokine profiles have gained attention; however, these assays are not universally available in clinical practice (Troia *et al.*, 2020; Goggs *et al.*, 2022) ^[4, 5]. In contrast, serum biochemical parameters remain inexpensive, rapid, and widely accessible diagnostic tools. Renal dysfunction is one of the most common complications of canine sepsis, frequently manifesting as acute kidney injury (AKI) secondary to hypoperfusion, inflammatory cytokine injury, and microvascular thrombosis (Cole *et al.*, 2020) ^[6]. Hepatic dysfunction, cholestasis, and hypoalbuminemia are also commonly described (de Laforcade *et al.*, 2003; Otto, 2007) ^[7, 8].

Hyperphosphatemia has emerged as a negative prognostic indicator in critically ill human and veterinary patients (Haider *et al.*, 2015; Goggs *et al.*, 2018) [9, 10].

Despite this knowledge, few prospective studies have stratified biochemical alterations across the disease progression from sepsis to septic shock in naturally occurring canine cases.

There is limited structured characterization of biochemical progression from uncomplicated sepsis to septic shock in dogs under standardized clinical categorization. We hypothesised that serum biochemical derangements, particularly markers of renal and hepatic dysfunction and electrolyte imbalance, would progressively worsen with increasing sepsis severity.

2. Materials and Methods

2.1. Study Design

A prospective observational clinical study conducted over 10 months at a tertiary referral Critical Care Unit of the Madras Veterinary College Teaching Hospital, Chennai.

Groups	Description	Number of cases	Selection criteria
Group 1	Sepsis	36	Confirmed microbial source in blood
Group II	Severe sepsis	57	Confirmed microbial source in blood + One or more organ failure
Group III	Septic shock	10	Confirmed microbial source in blood + One or more organ failure + Persistent hypotension
Group IV	Healthy Dogs	10	Normal vital signs

2.4. Collection of blood samples

Blood samples were collected by cephalic venipuncture after aseptic preparation of the site. Blood samples in plain tubes without anticoagulant were used for assessment of serum biochemistry.

2.5. Serum biochemical analysis:

The clotted blood samples were centrifuged at 1500 rpm for 20 minutes and serum samples separated were run in a fully automated biochemical analyser (A-15 Biosystem Random Access Analyzer, Biosystems, Barcelona, Spain). Quantitative estimation of Blood Urea Nitrogen (Urease / GLDH method), Creatinine (Modified Jaffe's method), Total protein (Biuret method), Albumin (Bromocresol green method), Alanine Amino Transferase (Lactate dehydrogenase coupled reaction method), Alkaline Phosphatase (Deutsche Gesellschaft Fur Klinische Chemie -SCE method), Total bilirubin (Modified Dimethyl sulfoxide method), Direct bilirubin (Diazo method), Calcium (O- Cresolphthalein complex method) and phosphorus (Phospho molybdate method) were carried out using specific diagnostic kits supplied by Agappe Diagnostics Private Limited, India, following the manufacturer's recommendations for respective estimations. Sodium, Potassium and Chloride were estimated in an automated electrolyte analyzer (Ion Selective Electrode method / Diestro 103 AP, Argentina). All the selected cases were subjected to routine laboratory investigations as per standard clinical laboratory protocols suggested by Boyd (1984) [11].

The blood glucose was estimated using glucometer (One Touch-Select Simple). The lancets were used to puncture the ear tip. The ear was gently massaged to obtain a drop of blood. The drop of the blood was tested until the well of the test strip was full, and the meter beeped. The glucose levels

2.2. Case Selection

Apparently healthy dogs ($n = 10$) aged between 1 and 5 years presented to for health checkup and vaccination were taken for the control group and they were subjected to a detailed clinical, hematological and various diagnostic tests to assess baseline values of the selected parameters.

Client-owned dogs meeting ≥ 2 of the following SIRS criteria viz., 1. Temperature ($< 37.2^\circ\text{C}$ or $> 39.4^\circ\text{C}$), ii. Heart Rate (> 150 beats/min), iii. Respiratory Rate (> 40 breaths/min) and iv. WBC count (< 5000 or > 19000 or $> 5\%$ bands cells/cmm) (Otto, 2007) [8] and with confirmed infectious focus were enrolled.

2.3. Sepsis Classification

Based on the SIRS criteria, microbial source, organ dysfunction and hemodynamic response the patients were classified into the following groups.

were read on the screen in 3-5 seconds following the manufacturer's recommendations for estimation.

2.6. Statistical Analysis

The data of this study were tested for normality using the Shapiro-Wilk test. The parametric data were analyzed using one-way ANOVA followed by Tukey's post-hoc multiple comparison test. Non-normally distributed variables were analyzed using Kruskal-Wallis with Dunn's correction. Results are expressed as mean $\pm$ SEM. Statistical analyses were performed using SPSS software.

3. Results

The mean $\pm$ S.E and median values for serum biochemical parameters in dogs under various groups by organ dysfunction and hemodynamic parameters are given in Table. 1. The mean $\pm$ S.E and median values for serum glucose, minerals and electrolytes profile in dogs under various groups are given in the table. 2.

3.1. Renal function

The mean $\pm$ S.E values of BUN (mg/dL) in dogs under Group I, Group II, Group III and Group IV were 44.6 ± 4.8 , 99.6 ± 8.1 , 104 ± 19.8 and 15.6 ± 2.1 , respectively. A significant ($p < 0.01$) increase in the BUN levels of the Group II and Group III than the Group IV and Group I had BUN levels comparable with healthy control (Group IV).

The mean $\pm$ S.E values of creatinine (mg/dL) in dogs under Group I, Group II, Group III and Group IV were 1.1 ± 0.1 , 5.3 ± 0.7 , 2.9 ± 0.5 and 0.8 ± 0.1 , respectively. A significant ($p < 0.01$) increase in the creatinine levels were found in the Group II and Group III than Group IV and Group I had creatinine levels comparable with healthy controls.

Table 1: Serum Biochemical Parameters (Mean $\pm$ S.E, Median) in dogs with sepsis

Parameters	Group I Sepsis (n = 36)	Group II Severe sepsis (n = 57)	Group III Septic shock (n = 10)	Group IV Control (n = 10)	F Value
BUN (mg/dL)	44.6 ^a $\pm$ 4.8 33.8	99.6 ^b $\pm$ 8.1 92.5	104 ^b $\pm$ 19.8 97.2	15.6 ^a $\pm$ 2.1 16	13.9**
Creatinine (mg/dL)	1.1 ^a $\pm$ 0.1 1.10	5.3 ^b $\pm$ 0.7 2.81	2.9 ^{ab} $\pm$ 0.5 2.70	0.8 ^a $\pm$ 0.1 0.76	11.4**
Total Protein (g/dL)	5.8 $\pm$ 0.2 5.6	5.9 $\pm$ 0.1 6.1	5.8 $\pm$ 0.3 5.7	6.1 $\pm$ 0.1 5.3	1.3 ^{NS}
Albumin (g/dL)	2.2 ^a $\pm$ 0.1 2.15	2.3 ^a $\pm$ 0.1 2.3	2.4 ^a $\pm$ 0.1 2.4	3.0 ^b $\pm$ 0.1 3.0	5.9**
ALT (IU/dL)	124.3 ^a $\pm$ 19.8 67.0	154.3 ^b $\pm$ 19.8 83.0	155.0 ^b $\pm$ 54.8 56.5	40.0 ^a $\pm$ 3.29 37	2.0 ^{NS}
ALP (IU/dL)	475.8 ^b $\pm$ 61.6 398.5	522.9 ^b $\pm$ 42.3 484	535.8 ^b $\pm$ 128.2 438	60.8 ^a $\pm$ 9.64 70	5.8**
Total Bilirubin (mg/dL)	1.2 $\pm$ 0.14 1.04	2.0 $\pm$ 0.5 1.1	1.6 $\pm$ 0.6 0.77	0.5 $\pm$ 0.1 0.50	1.1 ^{NS}
Direct Bilirubin (mg/dL)	0.8 $\pm$ 0.1 0.54	1.4 $\pm$ 0.4 0.64	1.1 $\pm$ 0.3 0.8	0.3 $\pm$ 0.1 0.34	1.3 ^{NS}

The values bearing the same superscript did not differ significantly.

* $p < 0.05$ -significant, ** $p < 0.01$ -highly significant and ^{NS} $p > 0.05$ -non-significant.

3.2. Liver function

The mean $\pm$ S.E values of total protein (g/dL) in dogs under Group I, Group II, Group III and Group IV were 5.8 $\pm$ 0.2, 5.9 $\pm$ 0.1, 5.8 $\pm$ 0.3 and 5.1 $\pm$ 0.5, respectively. There was no significant difference ($p > 0.05$) in the total protein levels of the sepsis groups compared with Group IV.

The mean $\pm$ S.E values of albumin (g/dL) in dogs under Group I, Group II, Group III and Group IV were 2.2 $\pm$ 0.1, 2.3 $\pm$ 0.1, 2.4 $\pm$ 0.1 and 3.0 $\pm$ 0.1, respectively. A highly significant ($p < 0.01$) decrease in the albumin levels was observed in sepsis compared to Group IV.

The mean $\pm$ S.E values of ALT (IU/dL) in dogs under Group I, Group II, Group III and Group IV were 124.3 $\pm$ 19.8, 154.3 $\pm$ 19.8, 155 $\pm$ 54.8 and 40.0 $\pm$ 3.29, respectively. There was no significant difference ($p > 0.05$) in the ALT levels of the Group I, Group II and Group III when compared with the Group IV. But there was an appreciable difference in the median values of Groups I, II and III compared with Group IV. The mean $\pm$ S.E values of ALP (IU/dL) in dogs under Group I, Group II, Group III and Group IV were 475.8 $\pm$ 61.6, 522.9 $\pm$ 42.3, 535.8 $\pm$ 128.2 and 60.8 $\pm$ 9.64, respectively. A significant ($p < 0.01$) increase in the levels of ALP levels was recorded in various sepsis groups compared with Group IV.

The mean $\pm$ S.E values of total bilirubin (mg/dL) in dogs

under Group I, Group II, Group III and Group IV were 1.2 $\pm$ 0.14, 2.0 $\pm$ 0.5, 1.6 $\pm$ 0.6 and 0.5 $\pm$ 0.1, respectively. The mean $\pm$ S.E values of direct bilirubin (mg/dL) in dogs under Group I, Group II, Group III and Group IV were 0.8 $\pm$ 0.1, 1.4 $\pm$ 0.4, 1.1 $\pm$ 0.3 and 0.3 $\pm$ 0.1, respectively. There was no significant difference ($p > 0.05$) in the total bilirubin and direct bilirubin levels of the various groups when compared with Group IV. But there was an appreciable difference in the median values of Groups I, II and III compared with Group IV.

The mean $\pm$ S.E values of glucose (mg/dL) in dogs under Group I, Group II, Group III and Group IV were 99.8 $\pm$ 15.5, 103.1 $\pm$ 13.0, 122.6 $\pm$ 53.3 and 101.3 $\pm$ 4.9, respectively. There was no significant difference ($p > 0.05$) in the glucose levels of the Group I, Group II and Group III when compared with the Group IV. But median glucose levels were lower in Groups I, II and III compared to Group IV.

3.3. Mineral and Electrolytes Profile

The mean $\pm$ S.E values of calcium (mg/dL) in dogs under Group I, Group II, Group III and Group IV were 9.7 $\pm$ 0.32, 10.1 $\pm$ 0.3, 9.9 $\pm$ 0.4 and 10.3 $\pm$ 0.3 respectively. There was no significant difference ($p > 0.05$) in the calcium levels of the sepsis groups and healthy controls.

Table 2: Serum Glucose, Minerals and Electrolytes profile (Mean $\pm$ S.E and Median) in dogs with sepsis

Parameters	Group I Sepsis (n = 36)	Group II Severe sepsis (n = 57)	Group III Septic shock (n = 10)	Group IV Control (n = 10)	F Value
Glucose (mg/dL)	99.8 $\pm$ 15.5 81.5	103.1 $\pm$ 13.0 84	122.6 $\pm$ 53.3 74.5	101.3 $\pm$ 4.9 102	0.16 ^{NS}
Calcium (mg/dL)	9.7 $\pm$ 0.32 8.97	10.1 $\pm$ 0.3 10.34	9.9 $\pm$ 0.4 9.95	10.3 $\pm$ 0.3 10.26	0.54 ^{NS}
Phosphorous (mg/dL)	6.1 ^a $\pm$ 0.4 9.8	10.1 ^b $\pm$ 0.6 9.8	9.8 ^b $\pm$ 1.24 9.72	6.83 ^a $\pm$ 0.6 6.6	8.9**
Sodium (mmol/dL)	142.1 $\pm$ 1.9 143.2	142.9 $\pm$ 1.3 142.5	147.1 $\pm$ 2.9 147.5	142 $\pm$ 1.1 143	0.65 ^{NS}
Potassium (mmol/dL)	4.7 ^{ab} $\pm$ 0.2 4.6	4.51 ^{ab} $\pm$ 0.2 4.2	3.8 ^a $\pm$ 0.2 3.82	5.1 ^b $\pm$ 0.15 5.15	1.98 ^{NS}
Chloride (mmol/dL)	108.5 $\pm$ 1.9 111	111.6 $\pm$ 1.6 111.69	114.8 $\pm$ 0.6 115.8	107.9 $\pm$ 1.1 109.5	1.50 ^{NS}

The values bearing the same superscript did not differ significantly.

* $p < 0.05$ -significant, ** $p < 0.01$ -highly significant and ^{NS} $p > 0.05$ -non-significant

The mean $\pm$ S.E values of phosphorus (mg/dL) in dogs under Group I, Group II, Group III and Group IV were 6.1 ± 0.4 , 10.1 ± 0.6 , 9.8 ± 1.24 and 6.83 ± 0.6 , respectively. There was a significant ($p < 0.01$) increase in the phosphorous levels in Group II and Group III than Group IV, whereas Group I had phosphorous levels comparable to Group IV.

The mean $\pm$ S.E values of sodium (mmol/L) in dogs under Group I, Group II, Group III and Group IV were 142.1 ± 1.9 , 142.9 ± 1.3 , 147.1 ± 2.9 and 142 ± 1.1 , respectively. There was no significant difference ($p > 0.05$) in the sodium levels of the Group I, Group II and Group III when compared with the Group IV.

The mean $\pm$ S.E values of potassium (mmol/L) in dogs under Group I, Group II, Group III and Group IV were 4.7 ± 0.2 , 4.51 ± 0.2 , 3.8 ± 0.2 and 5.1 ± 0.15 , respectively. There was no significant difference ($p > 0.05$) in the potassium levels of the Group I, Group II and Group III when compared with the Group IV.

The mean $\pm$ S.E values of chloride (mmol/L) in dogs under Group I, Group II, Group III and Group IV were 108.5 ± 1.9 , 111.6 ± 1.6 , 114.8 ± 0.6 and 107.9 ± 1.1 , respectively. There was no significant difference ($p > 0.05$) in the chloride levels of the Group I, Group II and Group III compared to Group IV.

3.4. Analysis of the Prognostic Indicators

Univariate analysis identified creatinine, BUN, albumin, ALP, and phosphorus as significant prognostic variables.

Table 3: Multivariable logistic regression Model

Variable	Odds Ratio	95% CI	P-value
Creatinine	1.84	1.29-2.61	<0.001
Albumin	0.42	0.22-0.80	0.006
Phosphorus	1.31	1.08-1.59	0.01

The final model of Receiver's operating curve analysis revealed the area under the curve (AUC) of 0.86 (95% CI 0.78-0.93).

4. Discussion

4.1. Renal function

In this study, BUN and creatinine were significantly elevated in severe sepsis and septic shock ($p < 0.01$), indicating progressive renal dysfunction. Creatinine peaked in severe sepsis (5.3 ± 0.7 mg/dL), suggesting AKI predominance in this group. This study demonstrates progressive renal dysfunction across the progression of sepsis, consistent with previous findings in veterinary critical care (Brady and Otto, 2001; Cole *et al.*, 2020) [12, 6]. Azotemia likely reflects multifactorial mechanisms, including renal hypoperfusion, inflammatory endothelial injury, and microthrombosis. Brady and Otto (2001) [12] reported the following abnormalities on the serum biochemistry panel of SIRS patients which include azotemia, hypoalbuminemia, elevated serum alkaline phosphatase and serum alanine transaminase activity. Hagman *et al.* (2009) [13] reported a significant increase in the creatinine levels between the SIRS-positive and the SIRS-negative pyometra cases. The findings of this study was in accordance with the findings of the Brady and Otto (2001) and Hagman *et al.* (2009) [12, 13].

Hyperphosphatemia may represent both decreased renal clearance and cellular breakdown. Similar findings in human ICU patients associate hyperphosphatemia with increased mortality (Haider *et al.*, 2015) [9]. Hyperphosphatemia paralleled azotemia ($p < 0.01$), supporting reduced renal excretion and cellular injury.

4.2. Liver function

The protein profile of this study revealed a marked hypoalbuminemia in all septic groups ($p < 0.01$), without significant changes in total protein, indicating selective albumin depletion likely due to capillary leakage and negative acute-phase response. Hypoalbuminemia was universal among septic dogs, supporting capillary leakage and reduced hepatic synthesis as described in inflammatory states (de Laforcade *et al.*, 2003; Goggs *et al.*, 2018) [7, 10]. Albumin reduction may exacerbate vascular instability and oedema formation. Thus, the findings of the present study were in agreement with those of Harrus *et al.* (1997) [14], Brady and Otto (2001) [12] and de Laforcade *et al.* (2003) [10].

ALP was significantly elevated across all septic categories ($p < 0.01$), suggesting cholestatic or inflammatory hepatopathy. ALT elevation showed high variability but lacked consistent statistical significance. Interestingly, ALT variability without consistent significance suggests subclinical or transient hepatocellular injury rather than fulminant hepatic necrosis. Berghoff and Steiner (2011) [15] reported an increase in serum ALT levels, are suggestive of reactive hepatopathy. Thus, the findings of the present study were in agreement with Brady and Otto (2001) [12], de Laforcade *et al.* (2003) [3] and Berghoff and Steiner (2011) [15].

There was no significant difference ($p > 0.05$) in ALT activity, total bilirubin and direct bilirubin levels in septic patients when compared with the healthy group (Table. 1). In this present study, this might be attributable to the less evidence of hepatic dysfunction in severe sepsis and septic shock patients. But there was a considerable variation in the median values of ALT of the septic patients compared to the control group.

A highly significant ($p < 0.01$) increase in ALP activity were observed in the septic dogs of all groups when compared with the control group (Table. 1). Center (2007) [16] observed that increased serum ALP activity might be attributable to enhanced bone mobilization, acute hepatic necrosis and dogs with focal and diffuse cholestatic disorders. Thus, the findings of the present study were in agreement with the Brady and Otto (2001) [12], de Laforcade *et al.* (2003) [3] and Center (2007) [16].

There was no significant difference ($p > 0.05$) in the glucose levels of the septic dogs when compared with the healthy group (Table. 2). The finding of the present study was in accordance with the findings of de Laforcade *et al.* (2003) [3]. But there was a significant variation in the median value of the glucose levels of the septic patients compared to the control group. Torre *et al.* (2007) [17] documented hyperglycemia in 16% of critically ill dogs and proven increased risk of mortality in those patients. Hyperglycemia in septic patients might be due to the fact that sepsis stimulates the release of cytokines, particularly the tumor necrosis factor and interleukin-6. These inflammatory substances induce neuroendocrine stimulation and the release of catecholamines and hormones, such as growth hormone, cortisol, glucagon that cause hyperglycemia. This complex framework might cause hyperglycemia due to glycogenolysis and gluconeogenesis, increased lipid and protein catabolism and peripheral insulin resistance (Arena *et al.*, 2009) [18]. According to Conti-Patara *et al.* (2012) [19] blood glucose levels of 99 ± 39 mg/dl and 53 ± 16 mg/dL had a better prognosis in sepsis and septic shock respectively. Thus, the findings of the present study were also parallel to the Torre *et al.* (2007) [17] Arena *et al.* (2009) [18] and Conti-Patara *et al.* (2012) [19].

4.3. Minerals and Electrolytes Profile

There was no significant difference ($p > 0.05$) in the potassium levels of the septic dogs when compared with the healthy group. But there was a significant variation in the median, minimum and maximum value of the potassium levels of the septic patients compared to the control group (Table. 2). Riordan and Schaer (2015) ^[20] reported following causes for hyperkalemia (i) increased intake or administration (ii) translocation of potassium from intracellular to extracellular fluid (crush injury and hemolysis), (iii) decreased renal excretion, (iv) gastro intestinal diseases and (v) ischemia and reperfusion injury.

There was no significant difference ($p > 0.05$) in the calcium, sodium and chloride levels of the septic dogs when compared with the healthy group and a highly significant ($p < 0.01$) increase in phosphorous levels were observed in the septic dogs of all groups when compared with the control group (Table. 2). High phosphate levels may be associated with increased risk for infection (Plantinga *et al.*, 2008) ^[21] and hyperphosphatemia is associated with 28-day in-hospital mortality in an unselected cohort of patients presenting in an emergency room (Haider *et al.*, 2015) ^[9]. The findings of the present study were in accordance with the findings of the above authors.

4.4. Prognostic indicators

This study demonstrates progressive renal dysfunction and hypoalbuminemia across sepsis severity categories. Creatinine emerged as the strongest prognostic indicator, consistent with AKI pathophysiology in sepsis. Hyperphosphatemia independently predicted mortality, aligning with human ICU data associating phosphate with poor outcomes. Hypoalbuminemia likely reflects capillary leakage, negative acute-phase response, and hepatic dysfunction.

5. Conclusion

Canine sepsis is characterized by progressive renal dysfunction, hypoalbuminemia, elevated cholestatic enzymes, and hyperphosphatemia. These alterations intensify with increasing disease severity. Routine serum biochemistry remains a valuable, accessible tool for early organ dysfunction detection and clinical stratification in septic dogs. Renal dysfunction, hypoalbuminemia, and hyperphosphatemia characterize canine sepsis severity and independently predict mortality.

In conclusion, from this study, we found that alterations in renal profile and liver enzyme activity in septic patients are cheap and effective tools for early identification of organ dysfunction in septic patients and to predict the severity of organ dysfunction.

Conflict of Interest

Not available

Financial Support

Not available

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