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Haemato-biochemical profiles in canine intestinal obstruction: A study of 84 cases

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ABSTRACT

In canine intestinal obstruction, the progressive luminal distension, mucosal congestion and impaired microvascular perfusion, results in severe systemic inflammatory, fluid-electrolyte, and metabolic complications. This clinical study evaluated pre-operative haematological and serum biochemical profiles in eighty-four client owned dogs presenting with confirmed intestinal obstruction due to non-linear foreign bodies ($N = 23$), linear foreign bodies ($N = 32$), intussusception ($N = 25$) and mural affections ($N = 4$). Pronounced neutrophilic leukocytosis accompanied by relative lymphopenia consistently characterized dogs presenting with non-linear foreign bodies, linear foreign bodies and intussusception, reflecting a marked systemic inflammatory response to localized intestinal injury. Dogs affected by intussusception exhibited severe normocytic anemia, hypoproteinemia and hypoalbuminemia, resulting from transmural mucosal damage, intense venous congestion, and exudative protein-losing enteropathy. Markedly elevated systemic serum lactate concentrations were recorded in all dogs, underscoring widespread splanchnic tissue hypoperfusion, cellular hypoxia and anaerobic glycolysis. Moderate elevations in serum glutamic oxaloacetic transaminase and alkaline phosphatase indicated altered cell membrane permeability in ischemic intestinal smooth muscle and secondary reactive hepatopathy induced by circulating inflammatory cytokines and gut-derived toxins. Serum creatinine, blood urea nitrogen, total bilirubin and serum glutamic pyruvic transaminase remained within normal reference limits in all groups. Non-linear intestinal foreign body obstructions produced a classic triad of concurrent hyponatremia, hypokalemia and hypochloremia secondary to intraluminal fluid sequestration and profuse emesis of gastrointestinal secretions.

Keywords: Intestinal obstruction, Haematological, Biochemical, Tissue hypoperfusion, Intussusception, Neutrophilic leukocytosis, Electrolyte imbalances

Introduction

Gastrointestinal obstruction represents one of the most critical and frequently encountered surgical emergencies in small animal veterinary practice. In dogs, intestinal obstruction commonly arises from the ingestion of non-digestible foreign bodies, intussusception, luminal strictures or neoplastic masses (Winkler, 2025) [1]. The clinical transition from initial symptomatic assessment to definitive therapeutic intervention in gastrointestinal (GI) emergencies necessitates a comprehensive diagnostic approach that extends beyond conventional imaging modalities. Although clinical signs such as lethargy, vomiting and abdominal pain are frequently associated with GI emergencies, their non-specific nature limits their diagnostic utility. The mechanical obstruction of the intestinal lumen initiates a rapid cascade of pathological events, beginning with localized luminal distension, mucosal congestion and impaired microvascular perfusion (Coker *et al.*, 2026) [2]. Persistent obstruction leads to progressive pressure necrosis of the intestinal wall, compromised epithelial integrity and the translocation of luminal bacteria and endotoxins into systemic circulation (González *et al.*, 2025) [3]. Understanding the systemic effects of these localized tissue changes is essential for early clinical stabilization and deciding between conservative enterotomy or radical enterectomy. Repeated emesis and sequestration of fluid within obstructed intestinal loops precipitate severe dehydration and hypovolemia, classically manifesting as hemoconcentration with elevated packed cell volume (PCV) and total plasma protein (Mottelieb *et al.*, 1976) [4]. Concurrently, bacterial translocation and localized tissue necrosis evoke a robust systemic inflammatory response characterized by neutrophilic leukocytosis, elevated Systemic Immune-Inflammatory Index (SII) markers, stress leukograms, or degenerative left shifts (González *et al.*, 2025) [3]. Serum biochemical profiles frequently reveal profound electrolyte derangements, most notably hyponatremia, hypokalemia and hypochloremia, alongside tissue hypoperfusion markers such as hyperlactatemia. The present analysis further evaluates the diagnostic and prognostic significance of these haematological and biochemical markers across a range of clinical presentations, with the aim of enhancing clinical assessment and refining surgical decision making.

Materials and Methods

The current study was carried out on 84 canine patients presented with suspected intestinal obstruction. The conditions were diagnosed based on clinical signs, radiographic and ultrasonographic findings as intestinal obstruction due to intestinal FBs ($N = 23$), linear FBs ($N = 32$), intussusception ($N = 25$) and mural obstructions ($N = 4$). The following haemato-biochemical parameters were recorded in this study and correlated with the type of surgical affection.

1. Haematological Parameters: Haemoglobin (Hb, g/dL), Total Leukocyte Count (TLC / μ L), Total Erythrocyte Count (TEC $\times 10^6$ (Hughes *et al.*, 1999) [^{5}]/ μ L), Packed Cell Volume (PCV, %), Platelet Count (PLT $\times 10^3$ (González *et al.*, 2025) [^{3}]/ μ L) and Differential Leukocyte Count (DLC, %) were recorded.
2. Biochemical Parameters: Serum Lactate (mmol/L), Total Protein (TP, g/dL), Albumin (Alb, g/dL), Creatinine (mg/dL), Blood Urea Nitrogen (BUN, mg/dL), Total Bilirubin (mg/dL), Serum Glutamic Oxaloacetic Transaminase (SGOT, U/L), Serum Glutamic Pyruvic Transaminase (SGPT, U/L), Alkaline phosphatase (ALP, U/L), Gamma-glutamyl transferase (GGT, U/L) and electrolytes like Sodium (Na, mEq/L), Potassium (K, mEq/L), Chloride (Cl, mEq/L) were recorded.

Results

The quantitative haematological and biochemical parameters recorded at the time of presentation for Group 1 (Intestinal foreign body, $n = 23$), Group 2 (Linear foreign body, $n = 32$), Group 3 (Intussusception, $n = 25$), and Group 4 (Mural affections like intestinal strictures and mass, $n = 4$) are detailed below:

Haematological observations

1. Group 1 (Intestinal Foreign Body): Haemoglobin, packed cell volume, total erythrocyte count and platelet count remained within the normal reference ranges (Allen *et al.*, 2005) [^{6}], while marked leukocytosis ($19,806.00 \pm 2,249.49$ cells/ μ L) accompanied by mature neutrophilia ($84.09 \pm 3.30\%$) was recorded in group 1. Concurrent lymphopenia was present in 13 dogs in this group (Table 1).
2. Group 2 (Linear Foreign Body): Haemoglobin and packed cell volume were normal with mean values of 13.21 ± 0.53 g/dL and $37.21 \pm 1.33\%$, respectively. Marked neutrophilic leukocytosis was observed, with a mean total neutrophil count of $20,282.00 \pm 1,867.03$ cells/ μ L and neutrophils constituting $87.53 \pm 1.99\%$ of the leukocytes, with relative lymphopenia evident in 17 dogs in group 2 (Table 1).
3. Group 3 (Intussusception): Significant normocytic anemia was observed, with mean haemoglobin and packed cell volume values of 9.58 ± 0.61 g/dL and $27.36 \pm 1.71\%$, respectively, falling well below reference limits. The total leukocyte count revealed pronounced neutrophilic leukocytosis, with a mean value of $19,745.00 \pm 2,079.07$ cells/ μ L (Table 1).
4. Group 4 (Mural affections): Slightly reduced haemoglobin (11.62 ± 1.06 g/dL) and packed cell volume PCV ($31.70 \pm 2.54\%$) indicated mild anemia, whereas the total leukocyte count remained within normal limits (Allen *et al.*, 2005) [^{6}], measuring $12,420.00 \pm 2,494.67$ cells/ μ L (Table 1).

Biochemical observations

Serum lactate was markedly elevated above reference limits of 0.3 – 2.5 mmol/L (Hughes *et al.*, 1999) [^{5}] across all groups, measuring 4.72 ± 0.42 mmol/L, 4.18 ± 0.45 mmol/L, 4.02 ± 0.37 mmol/L, and 5.18 ± 0.53 mmol/L in Group 1, Group 2, Group 3 and Group 4, respectively as presented in table 2. Marked hypoproteinemia (4.48 ± 0.18 g/dL) and hypoalbuminemia (2.12 ± 0.14 g/dL) were characteristically present in dogs with intussusception belonging to group 3. Group 4 also exhibited mild hypoproteinemia and hypoalbuminemia, with mean values measuring 5.02 ± 0.36 g/dL and albumin 2.18

± 0.13 g/dL, respectively. Normal protein values were maintained in groups 1 and 2 (Table 2). Serum Glutamic Oxaloacetic Transaminase was moderately elevated across all groups, with values ranging from 37.35 – 47.18 U/L. Alkaline phosphatase was elevated in Group 1, Group 2, and Group 4 with mean values of 145.31 ± 26.39 U/L, 299.11 ± 123.40 U/L, and 164.67 ± 55.89 U/L, respectively. Serum Glutamic Pyruvic Transaminase, Blood Urea Nitrogen, Creatinine and Total Bilirubin remained within normal reference limits in all the dogs and is presented in table 2.

Concurrent hyponatremia (137.07 ± 2.43 mEq/L), hypokalemia (3.56 ± 0.23 mEq/L), and hypochloremia (94.50 ± 3.65 mEq/L) was observed in Group 1. Group 4 exhibited hyponatremia (136.50 ± 4.73 mEq/L) along with hypochloremia (93.75 ± 8.06 mEq/L). Isolated hypochloremia was observed in Groups 2 and 3, with mean serum chloride concentrations of 103.06 ± 2.64 mEq/L and 102.00 ± 3.09 mEq/L, respectively (Table 2).

Discussion

The consistently elevated total leukocyte count and neutrophil levels observed across all groups suggest the onset of Systemic Inflammatory Response Syndrome (SIRS) (Mottelil *et al.*, 1976; Singh, 2019) [^{4} , ^{7}]. Marked neutrophilic leukocytosis was a common hematological finding in Groups 1, 2, and 3. The mechanical obstruction caused by a linear or non-linear FB or intussusception leads to the distension of the gastrointestinal tract, pressure induced mucosal necrosis and compromised blood flow (Mohindroo *et al.*, 2006; Kaur, 2014) [^{8} , ^{9}]. Damage to the mucosal barrier promotes the absorption of endotoxins and translocation of bacteria into the systemic circulation, thereby activating a generalized immune response and stimulating the release of neutrophils from the bone marrow. The presence of neutrophilia and relative lymphopenia observed indicates a classic stress leukogram induced by endogenous corticosteroid release secondary to severe abdominal pain and systemic stress (Kaur, 2012; Singh, 2019) [^{10} , ^{7}]. Group 2 exhibited the most pronounced neutrophilic leukocytosis, where linear nature of FBs caused mesenteric plication and intestinal sawing leading to severe mucosal ischemia and microperforations (Kaur, 2014; Hoffmann, 2003) [^{9} , ^{11}]. The persistence of marginally altered total leukocyte counts in Group 4 underscores the chronic, nonstrangulating progression characteristic of mucosal hyperplasia, intestinal masses or strictures (Singh, 2019; Myers *et al.*, 1994) [^{7} , ^{12}].

The severity and mechanism of anemia varied markedly depending on the primary pathology (Singh, 2019) [^{7}]. Most severe anemia was evident in dogs with intussusception (Group 3), wherein the vascular incarceration of the invaginated intestinal segment lead to venous stasis, mucosal sloughing and extensive intraluminal gastrointestinal hemorrhage, compounded by anorexia and nutritional deficits (Kaur, 2014; Joy *et al.*, 1978; Gal *et al.*, 2007) [^{9} , ^{13} , ^{14}]. Dogs with mural affections (Group 4) demonstrated mild anemia. The neoplastic or chronic inflammatory mural lesions such as intestinal mass or chronic strictures, undergo ulceration, leading to persistent, low grade intraluminal blood loss and subsequent microcytic anemia (Peterson *et al.*, 2003) [^{15}]. Dogs with foreign body obstructions (Groups 2 and 3) generally maintained normal erythrocyte counts and PCV levels, which corroborated existing literature (Singh, 2019; Kaur, 2012; Tripathi *et al.*, 2010) [^{7} , ^{10} , ^{16}]. However, the fluid sequestration into dilated bowel loops or emesis can induce hemoconcentration, which may transiently mask underlying anemia in acute obstructive cases (Boag *et al.*, 2005) [^{17}].

Marked hyperlactatemia was present in all dogs. Fluid loss from emesis and intraluminal fluid sequestration during intestinal obstruction results in hypovolemia, systemic hypotension and impaired perfusion (McGlone, 2016) [^{18}]. Consequent cellular hypoxia induces a metabolic shift from aerobic respiration to anaerobic glycolysis, elevating systemic lactate levels (Kaur, 2012; Boag *et al.*, 2005) [^{10} , ^{17}]. Hyperlactatemia thus serves as a critical biomarker of tissue hypoperfusion, splanchnic ischemia and local hypoxia secondary to luminal distension and impaired mesenteric microcirculation (Kaur, 2012;

McGlone, 2016) [10, 18]. Serum lactate concentrations exceeding the 6.0 – 6.4 mmol/L threshold provide a critical indication of gastric necrosis and impending cardiovascular collapse (Papp *et al.*, 1999) [19].

The hypoalbuminemia observed in groups 3 and 4 is a clinical hallmark of protein losing enteropathy or chronic neoplastic infiltration. In dogs with intussusception, severe transmural mucosal damage paired with venous congestion caused direct leakage of plasma proteins into the intestinal lumen, a loss further exacerbated by malabsorption and malnutrition (Joy *et al.*, 1978; Kumar *et al.*, 2015) [13, 20]. In dogs with mural affections, chronic wall inflammation, mucosal ulceration and disrupted lymphatic drainage impair protein absorption and promote exudative luminal loss (Kaur, 2014; Peterson *et al.*, 2003) [9, 15]. Hypoproteinemia impairs wound healing and increases the risk of postoperative dehiscence. Serum albumin concentration of ≤ 2.5 g/dL is a significant predictor of anastomotic leakage (Ralphs *et al.*, 2003) [21].

The moderate elevations in Serum Glutamic Oxaloacetic Transaminase (SGOT) observed across all groups indicated compromised cell membrane permeability, leading to enzyme leakage from ischaemic intestinal smooth muscles, skeletal muscle stress or secondary tissue injury (Singh, 2019; Dial, 1995) [7, 22]. Elevated alkaline phosphatase levels in Groups 1, 2, and 4 reflect secondary reactive hepatopathy or mild hepatobiliary cholestasis, caused by circulating inflammatory cytokines, gut derived toxins and altered intestinal motility (Kaur, 2014; Hoffmann, 2003; Jawre *et al.*, 2008) [9, 11, 23].

The clinical presentation of electrolyte derangements depended significantly on both the site and chronicity of the gastrointestinal obstruction (Kaur, 2014; Boag *et al.*, 2005; Strombeck *et al.*, 1996) [9,

17, 24]. Specifically, dogs presenting with intestinal foreign bodies (Group 1) exhibited the classic triad of concurrent hyponatremia, hypokalemia, and hypochloremia (Kaur, 2014; Gal *et al.*, 2007) [9, 14]. Dogs with mural affections (Group 4) exhibited hyponatremia and hypochloremia. The maintenance of normal potassium concentrations in this group suggested a temporary compensatory renal preservation mechanism. Profuse vomiting and fluid sequestration within the intestinal lumen led to substantial depletion of gastrointestinal secretions rich in sodium, potassium and chloride (Boag *et al.*, 2005) [17]. Dogs belonging to Group 2 and Group 3, showed mild isolated hypochloremia, likely attributable to moderate loss of chloride rich gastric secretions via emesis (Kaur, 2014; Hoffmann, 2003) [9, 11].

Conclusion

Comprehensive haemato-biochemical profiling serves as an indispensable adjunctive diagnostic tool in canine intestinal obstruction, providing critical insights into the severity of tissue ischemia, systemic inflammation and metabolic derangement beyond non-specific clinical signs. Baseline evaluation is essential to establish disease severity, guiding fluid resuscitation and predicting surgical outcomes in dogs with intestinal obstruction. Neutrophilic leukocytosis represents a nearly universal systemic inflammatory response to localized gastrointestinal injury and alerts to potential SIRS associated sequelae. Hyperlactatemia serves as the definitive biomarker for tissue hypoperfusion and anaerobic metabolism, with values exceeding 6.4 mmol/L suggesting a guarded prognosis. Preoperative hypoalbuminemia and electrolyte imbalances are the most significant predictors of surgical complications, particularly anastomotic leakage.

Table 1. Mean $\pm$ SE values of preoperative haematological observations in dogs affected with intestinal obstruction

Haematological Parameters	Group 1: Intestinal FB (n = 23)	Group 2: Linear FB (n = 32)	Group 3: Intussusception (n = 25)	Group 4: Mural/Intramural (n = 5)
Haemoglobin (Hb, g/dL)	13.47 $\pm$ 0.63	13.21 $\pm$ 0.53	9.58 $\pm$ 0.61	11.62 $\pm$ 1.06
Total Leukocyte Count (TLC, μ L)	19,806.00 $\pm$ 2,249.49	20,282.00 $\pm$ 1,867.03	19,745.00 $\pm$ 2,079.07	12,420.00 $\pm$ 2,494.67
Total Erythrocyte Count (TEC, $\times 10^6/\mu$ L)	5.83 $\pm$ 0.28	5.62 $\pm$ 0.24	4.37 $\pm$ 0.24	5.39 $\pm$ 0.55
Packed Cell Volume (PCV, %)	36.14 $\pm$ 1.69	37.21 $\pm$ 1.33	27.36 $\pm$ 1.71	31.70 $\pm$ 2.54
Platelet Count (PLT, $\times 10^3/\mu$ L)	317.13 $\pm$ 28.49	335.34 $\pm$ 25.74	337.04 $\pm$ 37.17	395.60 $\pm$ 67.76
Neutrophils (N, %)	84.09 $\pm$ 3.30	87.53 $\pm$ 1.99	85.44 $\pm$ 1.94	74.20 $\pm$ 2.33
Lymphocytes (L, %)	13.83 $\pm$ 3.11	11.72 $\pm$ 1.95	13.76 $\pm$ 1.94	24.00 $\pm$ 0.63
Eosinophils (E, %)	4.75 $\pm$ 0.92	2.75 $\pm$ 0.37	2.50 $\pm$ 0.33	9.00 $\pm$ 0.00

Table 2. Mean $\pm$ SE values of preoperative serum biochemical observations in dogs affected with intestinal obstruction

Biochemical Parameters	Group 1: Intestinal FB (n = 23)	Group 2: Linear FB (n = 32)	Group 3: Intussusception (n = 25)	Group 4: Mural affections (n = 5)
Serum Lactate (mmol/L)	4.72 $\pm$ 0.42	4.18 $\pm$ 0.45	4.02 $\pm$ 0.37	5.18 $\pm$ 0.53
Total Protein (TP, g/dL)	5.73 $\pm$ 0.29	5.66 $\pm$ 0.26	4.48 $\pm$ 0.18	5.02 $\pm$ 0.36
Albumin (Alb, g/dL)	2.78 $\pm$ 0.16	2.85 $\pm$ 0.20	2.12 $\pm$ 0.14	2.18 $\pm$ 0.13
Creatinine (Cr, mg/dL)	0.99 $\pm$ 0.16	0.98 $\pm$ 0.11	1.05 $\pm$ 0.21	0.92 $\pm$ 0.18
Blood Urea Nitrogen (BUN, mg/dL)	21.65 $\pm$ 4.99	22.84 $\pm$ 2.94	24.56 $\pm$ 7.22	16.00 $\pm$ 4.69
Total Bilirubin (TB, mg/dL)	0.20 $\pm$ 0.06	0.57 $\pm$ 0.28	0.12 $\pm$ 0.02	0.10 $\pm$ 0.00
SGOT / AST (U/L)	37.35 $\pm$ 3.85	46.67 $\pm$ 7.49	47.18 $\pm$ 6.62	42.25 $\pm$ 2.93
SGPT / ALT (U/L)	39.67 $\pm$ 4.58	46.41 $\pm$ 9.74	33.08 $\pm$ 3.43	46.67 $\pm$ 11.46
Alkaline Phosphatase (ALKP, U/L)	145.31 $\pm$ 26.39	299.11 $\pm$ 123.40	89.75 $\pm$ 19.66	164.67 $\pm$ 55.89

(continued on next page)

Biochemical Parameters	Group 1: Intestinal FB (n = 23)	Group 2: Linear FB (n = 32)	Group 3: Intussusception (n = 25)	Group 4: Mural affections (n = 5)
Gamma-Glutamyl Transferase (GGT, U/L)	10.43 ± 0.82	12.74 ± 2.51	7.88 ± 1.55	16.00 ± 4.93
Sodium (Na, mEq/L)	137.07 ± 2.43	142.69 ± 1.48	143.30 ± 1.22	136.50 ± 4.73
Potassium (K, mEq/L)	3.56 ± 0.23	4.08 ± 0.16	4.12 ± 0.20	4.33 ± 0.51
Chloride (Cl, mEq/L)	94.50 ± 3.65	103.06 ± 2.64	102.00 ± 3.09	93.75 ± 8.06

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