**Egyptian Journal of Veterinary Sciences**

**https://ejvs.journals.ekb.eg/**

# Variations of Serum Amyloid A, Haptoglobin, Coagulation Profile, and Venous Blood Gases in Arabian Horses in Different Colic Cases

**Mazen H. Elsawaf[1]*, Nagy E. El-Mashad[1], Fayez A. Salib[1], Alaa H. Jaheen[1], and Mohamed A. El-Sherif[1]**

[1]*Department of Medicine and Infectious Diseases, Faculty of Veterinary Medicine, Cairo University, Giza, Egypt.*

**Abstract**

EQUINE colic ranks among the most prevalent field problems in equine practice, and it's believed to be the leading cause of death for horses. This study was performed on sixty-four Arabian horses of both sexes (17 males and 47 females) ranging in age between 6 months to 26 years of which 15 clinically healthy horses joined as the control, and 32 horses are categorized in different colic groups. They were clinically evaluated on the basis of vital signs, intestinal sounds, gastric reflux, and findings of palpation per rectum. Hematology, coagulation profile, serum amyloid A (SAA), haptoglobin, venous blood gas (VBG), and electrolyte analysis were performed on jugular blood samples. The results revealed a significant difference especially for SAA, fibrinogen, ionized calcium ($iCa2^{+}$), anion gap (AG), and acid-base parameters between colic groups and the control group. In conclusion SAA, fibrinogen, and acid-base and electrolyte disturbances could be valuable indicators for the diagnosis and prognosis of colic with no significant value for haptoglobin. Our conclusion shows that SAA, fibrinogen, and acid-base and electrolyte disturbances could be valuable indicators for the diagnosis and prognosis of colic with no significant value for haptoglobin.

**Keywords:** Colic, Arabian horse, SAA, Haptoglobin, Coagulation profile, Venous blood gases.

## Introduction

Even now, colic remains one of the most common reasons for emergency calls to equine practitioner [1]. Colic is the biggest health issue facing horses and the main factor contributing to early deaths and morbidity [2].

Colic etiologies are numerous and differ in its clinical presentation. Horses with gastric impaction have been associated with a variety of presenting symptoms in horses with gastric impaction, ranging from anorexia to severe abdominal pain [3].

Large colon impactions (L.C.I) often appeared with mild signs of colic. During the initial assessment, the most useful parameters to distinguish between simple uncomplicated and serious cases were heart rate and the gut sounds [4].

Large colon displacement takes four main forms: right dorsal displacement, left dorsal displacement, retroflexion of the pelvic flexure, and non-strangulating volvulus of the large colon [5]. While moderate colic symptoms are typical, some horses may exhibit symptoms of severe abdominal pain [6].

Strangulating colic can affect the small intestine (small intestinal strangulation) or the large intestine (colon torsion) the cecum may be involved. Colic begins suddenly; acute abdominal pain, elevated heart rate, delayed capillary refill time, and gastric reflux are all symptoms associated with small-intestinal volvulus and if the volvulus just obstructs the intestinal lumen, the pain may be mild to moderate [7].

Proximal enteritis or duodenitis-proximal jejunitis (DPJ) it is defined by an abrupt start of ileus and

*Corresponding authors: Mazen H. Elsawaf, E-mail: mazenelsawaf97@cu.edu.eg Tel.: 01010620947
(Received 07/05/2024, accepted 30/06/2024)
DOI: 10.21608/EJVS.2024.287886.2067

nasogastric reflux, which is followed by systemic toxemia symptoms [8].

Right dorsal colitis (RDC) is a protein-losing enteropathy that is exclusive to the right dorsal colon in horses. In which non-steroidal anti-inflammatory drugs (NSAIDs) have the potential to be the cause of it. Acute RDC symptoms might include severe colic, excessive diarrhea, dehydration, endotoxic shock, and even death [9].

One of colic etiologies is peritonitis, which is usually associated with colic, fever, and Lethargy was the most often occurring clinical symptoms [10].

Colic is important both clinically and financially, an accurate diagnosis may be obtained by combining clinical and clinicopathological information from the patient [1]. A lack of a visible anatomical problem, and restrictions on the diagnostic methods and procedures available, make it difficult to reach an accurate diagnosis [11].

Inflammatory indicators have been used in several studies to diagnose and prognosticate horse colic early on. Research has shown that horses with colic have unusually high serum levels of Acute phase proteins (APPs) as Serum amyloid A (SAA) [12]. Recent studies on haptoglobin during colic suggest that horses with this condition have a moderate positive APP. However, its role in colic is yet unclear [13].

Several coagulation and fibrinolysis disorders, such as thrombocytopenia, prolonged clotting times: prothrombin time (PT) and partial thromboplastin time (PTT), decreased fibrinogen concentrations, increased d-dimer concentrations, and decreased antithrombin III (AT-III) activities may be a useful prognostic sign in horses suffering from colic [14].

The arterial blood gases, electrolytes, and acid-base balance may give the veterinarian an extra tool for diagnosis and prognosis cases of colic. In particular, the type and severity of the disease seem to have an impact on ionized calcium ($iCa2^{+}$) [15].

This study purpose is to evaluate the changes in hematological profile, and venous blood gases together with the detection of coagulation profiles including PT, PTT, and D-Dimer. It also explores the predictive value of using inflammatory markers including SAA and Haptoglobin in different colic cases compared to healthy control horses.

## Material and Methods

*Ethical approval*

Our study was conducted according to the guidelines of the Institutional Animal Care and Use Committee of the Cairo University CU-IACUC (Ethics approval number: Vet CU 2009 2022502).

*Animals*

Sixty-four Arabian horses of both sexes (17 males and 47 females) ranging in age between 6 months to 26 years of which 15 clinically healthy horses joined as the control. The collection of samples took place between October 2022 and October 2023 from various private schools in Elreagha, Green Belt, Teaching Hospital of Faculty of Veterinary Medicine Cairo University, and Riding Clubs in Madinat Nasr, Cairo Governorate, and 6th October City, Giza Governorate, Egypt.

The clinically affected horses were categorized according to final diagnosis as follow: gastric impaction (n=12), large colon impaction (n=10), Intestinal displacements and strangulations (n=10), proximal enteritis (n=9), colitis (n=4), septic peritonitis (n=4). Records were kept of the physical findings, clinical symptoms, and history. The horses were fed concentrates (barley and commercial pelleted feed) ranging from 0.5 kg to 8 kg (based on the horse's primary use) divided into 2-3 feedings per day in addition to forages (Egyptian clover and Egyptian clover hay) introduced three times a day beside wheat straw which serves as the stall's litter bedding.

The equine acute abdominal pain scale (EAAPS) was used to categorize the severity of abdominal pain, which ranged from 0 to 5 [16].

*Nasogastric intubation*

Nasogastric intubation has been carried out with a nasogastric tube with size 2.7 m x 17 mm and size 30 m x 19 mm (According to horse size) with a funnel-shaped end (H. Hauptner and Richard Herberholz GmbH & Co. KG, Germany) [17].

*Samples*

A blood sample was taken from each horse's jugular vein and split into three portions. The first sample was collected on an EDTA tube for hematological analysis using an automated veterinary hematology analyzer (Zoetis Vetscan® HM5, USA), the second portion was taken on a sodium citrate tube for plasma separation and coagulation tests (prothrombin time (PT), partial thromboplastin time (PTT)), and plasma fibrinogen concentration). For serum separation, the third portion was gathered into a standard tube. The serum that was kept at -20°C until it was needed.

Serum samples were used to measure serum amyloid A (SAA), haptoglobin (Hpt), and D-dimer (D2D) concentrations by using equine-specific commercially available ELISA assay kits supplied by (Chongqing Biospes Co., China) according to manufacturer instructions. Serum amyloid A was measured according to [18], Equine haptoglobin was measured as stated in [19], and measurement of D-dimer performed as described in [20].

Using a heparinized syringe and a 23G needle, another blood sample was collected from the jugular vein following manufacturer instructions to analyze the electrolytes and venous blood gases (VBG) using a Sensacore ST-200 CC ABGE Blood Gas Analyzer from India as stated in [21,22].

*Statistical analysis*

The obtained data were analyzed using the Independent-Samples T-test, the SPSS software package for Windows Ver. 20.0 (SPSS Inc., Chicago, IL, USA) and tabulated as mean value ± SE at levels of significance $p \leq 0.001$, $p \leq 0.01$, and $p \leq 0.05$. The p-value of $p \leq 0.001$ was considered highly statistically significant.

## Results

Comprehensive clinical examination and physical findings associated with Arabian horses suffering from gastric impaction, large colon impaction, Intestinal displacements and strangulations, proximal enteritis, colitis, and septic peritonitis were tabulated in Table 1 and Fig 1. Also, colic pain scores were detailed according to the EAAPS (Equine acute abdominal pain scale) as stated by [16] as shown in Table 2 and Figs. 1-5.

*Laboratory findings*

Our hematological results showed a significant decrease in red blood cell (RBC) count, Hemoglobin (Hb) content, and Packed cell volume (PCV%) ($p \leq 0.01$, $p \leq 0.01$, and $p \leq 0.001$ respectively) in the septic peritonitis group compared to the healthy control group. The leukogram results show a significant increase in white blood cell (WBC) count in the gastric impaction group, colitis, and septic peritonitis ($p \leq 0.05$, $p \leq 0.01$, and $p \leq 0.001$ respectively) while neutrophils are showing significant increase ($p \leq 0.001$) in septic peritonitis group with non-significant changes in other groups compared to the healthy control group as tabulated in Table 3.

Regarding coagulation profile and APPs, results of platelet count show significant increases in groups of gastric impaction ($p \leq 0.01$), intestinal displacements and strangulations ($p \leq 0.001$), proximal enteritis ($p \leq 0.01$), colitis ($p \leq 0.001$), septic peritonitis ($p \leq 0.01$). PT results show a significant decrease ($p \leq 0.05$) in the septic peritonitis group, while PTT results show a significant decrease in colitis and septic peritonitis groups ($p \leq 0.01$ and $p \leq 0.05$) respectively. Fibrinogen concentrations showed significant decreases in all groups, gastric impaction ($p \leq 0.001$), L.C.I ($p \leq 0.01$), intestinal displacements and strangulations ($p \leq 0.01$), proximal enteritis ($p \leq 0.01$), colitis ($p \leq 0.05$), and septic peritonitis ($p \leq 0.001$). D-dimer concentrations are showing non-significant increases among all groups. SAA results were significantly increased in L.C.I ($p \leq 0.05$), Intestinal displacements and strangulations ($p \leq 0.05$), colitis ($p \leq 0.01$), and septic peritonitis ($p \leq 0.001$) groups. Hpt showed a significant increase in the septic peritonitis group ($p \leq 0.01$) only with non-significant alterations in other groups compared to the healthy group as represented in Table 4.

Findings of venous blood gas analysis showed a significant decrease in pH in Intestinal displacements and strangulations ($p \leq 0.05$), colitis ($p \leq 0.001$), and septic peritonitis ($p \leq 0.05$), and it is significantly increased in the gastric impaction group ($p \leq 0.05$). Results of $pCO_2$ show a significant decrease in the gastric impaction group ($p \leq 0.01$), and in colitis and septic peritonitis ($p \leq 0.001$) groups.

Results of $HCO^-_3$ and $tCO_2$ show significant decreases ($p \leq 0.001$) in Intestinal displacements and strangulations, colitis, and septic peritonitis with non-significant decreases in other groups. Base excess (BE) results showed a significant decrease in Intestinal displacements and strangulations, colitis, and septic peritonitis ($p \leq 0.001$) groups. Anion gap (AG) results were significantly increased in gastric impaction, intestinal displacements and strangulations, colitis, septic peritonitis, and L.C.I ($p \leq 0.001$, $p \leq 0.01$) groups respectively. $HCO^-_3$ and $tCO_2$ results showed a significant decrease in intestinal displacements and strangulations, colitis, and septic peritonitis ($p \leq 0.001$) groups as represented in Table 5.

Electrolyte concentrations are showing a significant decrease ($p \leq 0.001$) in potassium concentration in the Intestinal displacements and strangulations group. Sodium concentration was significantly decreased ($p \leq 0.001$) in the colitis group. Chloride concentration was significantly decreased and increased ($p \leq 0.05$ and $p \leq 0.01$) in colitis and septic peritonitis groups respectively. While ionized calcium concentrations were significantly decreased ($p \leq 0.001$, $p \leq 0.01$, and $p \leq 0.05$) in intestinal displacements and strangulations, proximal enteritis, and septic peritonitis groups respectively compared to the healthy group as listed in Table 5.

## Discussion

The present study showed that numerous etiologies can cause colic in Arabian horses, with gastric impaction, large colon impaction, intestinal displacements and strangulations, proximal enteritis, colitis, and septic peritonitis as most common encountered, they displayed abdominal pain on EAAPS between the scales of two and five which is reported in previous studies by [16]

As presented in Table 1, in our study the horses showing severe signs of colic are the horses with more deteriorated physical examination parameters and more systemically ill. When intestinal pathology worsens, clinical parameters often deteriorate

simultaneously as stated by [23]. Numerous studies have linked an increased risk of death to both poor cardiovascular status and severe pain [24].

The erythrogram is almost within range compared with the clinically healthy control group except for the group of septic peritonitis (n=4) as it shows a significant reduction in the three red blood cell indices (RBCs count, Hb concentration, and PCV%). This may be caused by post-surgical hemorrhage. Post-surgical internal hemorrhage and peritonitis are common postoperative complications of colic surgeries [25]. The consequences of peritoneal bleeding include abdominal pain and a decrease in hematocrit on regular postoperative blood testing [26]. History of surgical intervention was consistent with the time of the appearance of postoperative complications [27].

The leukogram shows significant leukocytosis in the group of gastric impactions (n=12) which may be physiologic or stress leukocytosis [28]. Horses experiencing advanced stages of acute colitis may exhibit leukocytosis, a sign of a widespread inflammatory reaction [29]. Cases of peritonitis show significant leukocytosis and neutrophilia as reported in some cases [30,31, 32].

Platelets showed significant thrombocytosis in all groups which is suspected to be physiological due to epinephrine-induced splenic contraction in groups of gastric impactions, and colon displacements. Inflammatory response/infection in groups of proximal enteritis, colitis, and septic peritonitis may be implicated [33]. While PT and PTT tests show no alterations from the healthy control group except for the significant reduction in septic peritonitis in the case of PT, and the colitis and the septic peritonitis groups in the case of PTT which may be due to administration of large volumes of lactated ringer solution prior to admission as reported by [34] in an experiment carried out on swine. D-dimer results showed non-significant increases among all groups of colic which may indicate subclinical DIC [14]. Regarding fibrinogen concentration, all groups of colic are showing significant reduction which came in accordance with previous reports [14]. Fibrinogen can be used as an inflammatory and coagulation marker depending on the type, severity, and laboratory method employed, fibrinogen had varying responses in colic. It can be significantly reduced as a result of acute coagulation consumption [35]. Because fibrinogen is consumed during coagulation, responds slowly to inflammation, and has a broad reference range, it is a less sensitive inflammatory marker [36].

Concerning APPs, there was a significant increase in serum amyloid A concentration in groups of L.C.I., intestinal displacements and strangulations, colitis, and septic peritonitis. SAA increased under a variety of conditions in colic [37]. It increased in response to pain. Serum amyloid A (SAA) has been shown in studies to be a valuable prognostic tool because horses with higher SAA levels at admission have lower survival rates [38]. The results of Westerman, et al., 2016 [12] indicate that SAA concentration was the most sensitive variable tested concerning the need for surgical intervention or the appearance of complications in horses exhibiting signs of acute abdominal pain. However, the haptoglobin concentration did not change which is consistent with [13], and with [22] who reported that haptoglobin showed no significant change in a similar study carried out on Arabian horses with simple colic of spasmodic and flatulent nature except for the septic peritonitis group which showed significant increase in haptoglobin concentration that agreed with [12]. It may be a suitable indicator of a persistent inflammatory condition and begins to rise 12 to 24 hours after an inflammatory event [39].

Venous blood gas analysis showed significant differences between the healthy group and the colic case data, with most; of the values being lower in the colic horses showing agreement with [15]. On the other hand, many variables showed no significant difference as reported by [21]. Regarding the acid-base parameters, pH and the metabolic component ($tCO_2$, $HCO^-_3$, and BE) were significantly reduced, pH was significantly decreased in some colic groups in comparison with the healthy control group as reported by [21] which means the presence of degree of metabolic acidosis especially in groups of intestinal displacements and strangulations, colitis, and septic peritonitis which is consistent with [21]. Gastric impactions pH results were elevated this was reported previously [15]. The majority of colic cases did not exhibit metabolic acidosis, which is consistent with the results of another study on colic in horses, where the majority of cases did not show severe metabolic acidosis despite some highly elevated organic acid content [40]. A significant decrease in $pCO_2$ is present in gastric impactions moderately and strongly in colitis and septic peritonitis groups most likely indicating a compensatory reaction to metabolic acidosis brought on by the generation of lactate by anaerobic metabolism, as supported by decreased $HCO^-_3$ and base excess (BE) [15].

The most observed electrolyte disturbance in our study is a significant decrease in ionized calcium ($iCa^{2+}$) especially in groups of Intestinal displacements and strangulations, hypocalcemia may be the most common electrolyte imbalance during colic, especially in horses with strangulation, intestinal lesions, and ileus [41,42,43,44]. According to [41] sepsis, endotoxemia, and diarrhea can all cause a drop in the serum $iCa^{2+}$ level. In horses with some degree of gastrointestinal inflammation or sepsis, or during activity or transportation, acute hypocalcemia can present as ileus [45].

The group of Intestinal displacements and strangulations which are considered severe forms of colic has a significant decrease in potassium which came in accordance with [21,15]. The most prevalent causes of hypokalemia in horses with colic are altered intake and absorption and increased loss from the gastrointestinal tract as a result of diarrhea, [44]. Long-term fluid therapy with lactated Ringer's solution (which may cause sodium-induced diuresis), metabolic alkalosis from high volumes of gastric reflux production, and the use of specific medications may also be suggestive causes of hypokalemia in colic [46]. Other types of colic may have been hypokalemic and then plasma potassium level becomes normal secondary to metabolic acidosis [47].

Regarding sodium ($Na^+$) concentrations they are showing no differences from the healthy control group agree with [21]. With an exception for the group of colitis which shows a significant decrease in sodium ($Na^+$) and chloride ($Cl^-$) as stated by [29] who recorded that metabolic acidemia, azotemia, and electrolyte disturbance such as hyponatremia, hypochloremia, hypocapnia, hypokalemia, and hypocalcemia is common in horses with colitis. That is due to excessive fluid loss along with low chloride and high sodium content in the diarrhea [48]. In the case of chloride ($Cl^-$), it shows no significant changes between colic groups and the control group that agreed with a previous study of [21]. In the septic peritonitis group, showed a significant increase in chloride this is suggested to be secondary to severe metabolic acidosis. As stated by [49] when there is $HCO^-_3$ sequestration in the gut or loss of $HCO^-_3$ from the kidneys or intestinal tract, hyperchloremic metabolic acidosis is observed.

All colic groups showed a significant increase in AG in comparison with the healthy control group which agreed with the results of [21,15]. According to reports [50], these increases may be the result of an increase in unmeasured cations (calcium, and magnesium) or unmeasured anions (lactate, phosphate, sulfate, ketoacids, and albumin). Alternatively, as explained by [51], it could be the result of metabolic acidosis.

## Conclusion

SAA, fibrinogen, and acid-base and electrolyte disturbances could be valuable indicators for diagnosis and prognosis of colic with no significant value for haptoglobin.

*Authors' Contributions*

All authors contributed to the study's conception and design. Data collection, clinical examination, and experimental study were performed by MHE, NEE, and MAE. All laboratory analysis and data analysis were performed by AHG and MHE. FAS, NEE, MHE, and AHG drafted and corrected the manuscript; NEE and FAS revised the manuscript. All authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

*Acknowledgments*

We are grateful to the Department of Internal Medicine and Infectious Diseases, Faculty of Veterinary Medicine, Cairo University, Egypt, where the laboratory work has worked out. Also, to all the private studs in Giza and Cairo Governorates where the study on the horses has been conducted.

*Funding Statements*

None.

*Conflict of interest*

The authors declare no conflict of interest.

**TABLE 1. Colic signs, severity, and physical examination findings in Arabian horses suffering from different types of colic.**

| Groups / Clinical signs | Gastric Impaction (n=12) | L.C.I (n=10) | Intestinal displacements and strangulations (n=10) | Proximal Enteritis (n=9) | Colitis (n=4) | Septic peritonitis (n=4) |
|---|---|---|---|---|---|---|
| Abdominal pain | | | | | | |
| Restlessness | 9 | 5 | 8 | 8 | 2 | 3 |
| Curling upper lips up | 2 | 4 | 5 | 6 | 2 | 3 |
| Kicking at the belly | 0 | 3 | 3 | 0 | 0 | 1 |
| Looking at the belly | 3 | 9 | 9 | 6 | 4 | 3 |
| Paw at the ground | 5 | 8 | 9 | 8 | 2 | 3 |
| Laying down | 11 | 8 | 10 | 4 | 2 | 3 |
| Rolling | 1 | 3 | 9 | 2 | 2 | 1 |
| Colic Severity | | | | | | |
| Mild | 8 | 3 | 0 | 2 | 2 | 0 |
| Moderate | 3 | 4 | 1 | 4 | 0 | 1 |
| Severe | 1 | 3 | 9 | 3 | 2 | 3 |
| Abdominal distention | | | | | | |
| Absent | 10 | 4 | 1 | 7 | 2 | 2 |
| Present | 2 | 6 | 9 | 2 | 2 | 2 |
| Dehydration | | | | | | |
| Absent | 6 | 1 | 0 | 4 | 0 | 0 |
| mild | 2 | 3 | 2 | 1 | 0 | 0 |
| Moderate | 4 | 3 | 6 | 3 | 2 | 2 |
| Severe | 0 | 3 | 2 | 1 | 2 | 2 |
| Intestinal movement (defecation) | | | | | | |
| Normal | 9 | 0 | 2 | 4 | 0 | 0 |
| Absent | 0 | 2 | 1 | 0 | 0 | 1 |
| Constipation (scanty) | 3 | 8 | 7 | 4 | 0 | 3 |
| Diarrhea (soft contain water) | 0 | 0 | 0 | 1 | 4 | 0 |
| Intestinal sound (borborygmi) | | | | | | |
| Normomotility | 3 | 0 | 0 | 0 | 0 | 0 |
| Hypomotility | 9 | 10 | 7 | 5 | 4 | 0 |
| Hypermotility | 0 | 0 | 0 | 0 | 0 | 0 |
| Ileus | 0 | 0 | 3 | 3 | 0 | 4 |
| Appetite | | | | | | |
| Off food | 9 | 2 | 7 | 5 | 2 | 2 |
| Poor (inappetent) | 3 | 4 | 2 | 4 | 2 | 2 |
| Good | 0 | 4 | 1 | 0 | 0 | 0 |
| Heart rate | | | | | | |
| <44 beat/min | 3 | 1 | 0 | 0 | 0 | 0 |
| <60 beat/min | 8 | 6 | 3 | 3 | 0 | 0 |
| <80 beat/min | 1 | 3 | 2 | 5 | 2 | 3 |
| >80 beat/min | 0 | 0 | 5 | 1 | 2 | 1 |
| Profuse sweating | 3 | 3 | 8 | 1 | 0 | 1 |
| Urination | | | | | | |
| Normal urination | 9 | 6 | 3 | 7 | 2 | 2 |
| Decreased urination | 3 | 4 | 6 | 3 | 2 | 2 |
| Frequent urination | 0 | 0 | 0 | 0 | 0 | 0 |
| Mucous membranes | | | | | | |
| Normal | 9 | 4 | 0 | 2 | 0 | 0 |
| Congested | 3 | 5 | 9 | 4 | 2 | 1 |
| Petechial hg | 0 | 1 | 1 | 3 | 2 | 3 |
| Body temperature | | | | | | |
| Normal | 12 | 9 | 4 | 5 | 0 | 2 |
| Elevated | 0 | 1 | 6 | 4 | 4 | 2 |
| Nasogastric intubation | | | | | | |
| NA | 0 | 0 | 0 | 0 | 0 | 1 |
| No reflux | 0 | 4 | 1 | 0 | 2 | 2 |
| Reflux | 12 | 6 | 9 | 9 | 2 | 1 |
| Rectal examination | | | | | | |
| NA | 6 | 3 | 4 | 0 | 0 | 2 |
| Normal | 3 | 1 | 0 | 0 | 0 | 1 |
| Large intestinal tympany | 3 | 1 | 3 | 0 | 4 | 0 |
| Impacted colon | 0 | 5 | 0 | 0 | 0 | 0 |
| Distended small intestinal loops | 0 | 0 | 0 | 9 | 0 | 1 |
| Anatomical lesion | 0 | 0 | 3 | 0 | 0 | 0 |

**TABLE 2. Colic pain score based on EAAPS* in Arabian horses suffering from different types of colic.**

| Groups / Colic severity | Gastric Impaction (n=12) | L.C.I (n=10) | Intestinal displacements and strangulations (n=10) | Proximal Enteritis (n=9) | Colitis (n=4) | Septic peritonitis (n=4) |
|---|---|---|---|---|---|---|
| Score 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Score 1 | 0 | 0 | 0 | 0 | 2 | 0 |
| Score 2 | 4 | 1 | 0 | 0 | 0 | 0 |
| Score 3 | 3 | 4 | 0 | 4 | 0 | 1 |
| Score 4 | 4 | 2 | 1 | 3 | 0 | 1 |
| Score 5 | 1 | 3 | 9 | 2 | 2 | 2 |

(*) EAAPS = Equine acute abdominal pain scale

**TABLE 3. Hematological profile of different colic groups compared to normal group represented as Mean± SE.**

| Variants / Groups | RBCs ($10^6$/mm$^3$) | Hemoglobin (gm %) | PCV % | WBCs ($10^3$/mm$^3$) | Neutrophils ($10^3$/mm$^3$) |
|---|---|---|---|---|---|
| Control (n = 15) | 7.66± 0.22 | 13.35± 0.36 | 34.73± 0.83 | 9.35± 0.46 | 6.41± 0.33 |
| Gastric impaction (n = 12) | 7.62 ± 0.46 | 12.86 ± 0.71 | 33.36± 1.87 | 11.02± 0.67$^c$ | 7.47±0.69 |
| Large colon impaction (n = 10) | 7.29± 0.51 | 12.94± 0.91 | 33.50± 1.76 | 8.98± 0.75 | 7.10± 0.76 |
| Colon Intestinal displacements and strangulations (n = 10) | 7.61± 0.61 | 13.08± 0.91 | 33.88± 2.41 | 8.95± 0.69 | 6.85± 0.63 |
| Proximal enteritis (n = 9) | 7.52± 0.63 | 13.42± 1.02 | 34.44± 3.06 | 12.56± 3.57 | 8.80± 3.02 |
| Colitis (n = 4) | 8.03± 0.72 | 11.62± 1.32 | 34.25± 2.25 | 11.00± 0.00$^b$ | 6.54± 0.09 |
| Septic peritonitis (n =4) | 5.73± 0.65$^b$ | 9.97± 1.2 $^b$ | 25.50± 3.40 $^a$ | 21.67± 5.88 $^a$ | 16.61± 4.29 $^a$ |

a: $p \leq 0.001$: highly significant; b:$p \leq 0.01$; c: $p \leq 0.05$; NS: Non-significant.


**Fig. 1. A) 8-year-old non-pregnant Arabian mare showing severe abdominal distention secondary to colon volvulus. B) 16-year-old non-pregnant Arabian mare showing abdominal distention and rolling (EAAPS*-5) due to severe L.C.I.** C) 8-year-old non-pregnant Arabian mare pawing at ground (EAAPS-3). D) 10-year-old non-pregnant Arabian mare showed sternal recumbency with attempting to lie down with flank staring (EAAPS 4) due to to colon volvulus.**

**(*) EAAPS = Equine acute abdominal pain scale.**

**(**) L.C.I = Large colon impaction**

**TABLE 4. Coagulation profile and acute phase proteins of colic groups compared to normal group represented as Mean± SE p.**

| Variants / Groups | Platelets ($10^3$/mm$^3$) | PT (sec) | PTT (sec) | Fibrinogen (mg/dL) | D-Dimer (ng/mL) | SAA (mg/dL) | Hpt (mg/dL) |
|---|---|---|---|---|---|---|---|
| Control (n = 15) | 150.93± 0.94 | 10.26± 0.39 | 49.73± 2.57 | 347.07± 1.8 | 606.97± 2.6 | 54.02± 0.47 | 66.20±1.23 |
| Gastric impaction (n = 12) | 198.33±0.20$^b$ | 9.87± 0.34 | 45.91± 1.83 | 195.17± 1.9$^a$ | 646.84±3.1 | 69.88± 7.5 | 69.27±1.94 |
| Large colon impaction (n = 10) | 156.00± 1.6 | 9.52± 0.35 | 49.60± 4.82 | 210± 3.4$^b$ | 625.43± 3.2 | 83.28± 1.3$^c$ | 69.26± 3.70 |
| Colon Intestinal displacements and strangulations (n = 10) | 177.40± 2.8$^a$ | 10.36± 0.60 | 50.30± 3.57 | 254.50± 4.4$^b$ | 626.68± 3.0 | 87.77± 1.7$^c$ | 66.47±0.72 |
| Proximal enteritis (n = 9) | 205.11± 3.6$^b$ | 9.60± 0.18 | 49.33± 2.24 | 222.11± 3.2$^b$ | 702.32±5.5 | 62.81 ± 2.5 | 66.74± 4.46 |
| Colitis (n = 4) | 263.00± 1.5$^a$ | 9.10±0.00 | 41.00±0.00$^b$ | 236.25±2.9$^c$ | 632.32± 6.8 | 83.04± 1.12$^b$ | 67.32± 0.88 |
| Septic peritonitis (n =4) | 234.25± 5.1$^b$ | 8.82± 0.44$^c$ | 41.00± 1.87$^c$ | 192.75± 2.6$^a$ | 661.04± 2.6 | 114.63± 2.6$^a$ | 70.56± 0.87$^b$ |

**TABLE 5. Venous blood gases of colic groups compared to normal group represented as Mean± SE.**

| Variants / Groups | pH | $pCO_2$ (mmHg) | Potassium (mmol/L) | Sodium (mmol/L) | Chloride (mmol/L) | Ionized Calcium (mmol/L) | $HCO^-_3$ (mmol/L) | $tCO_2$ (mmol/L) | Base excess | Anion gab (mmol/L) |
|---|---|---|---|---|---|---|---|---|---|---|
| Control (n = 15) | 7.37 ± 0.005 | 43.94 ± 0.49 | 3.90 ± 0.15 | 138.34 ± 0.57 | 95.87±0.45 | 1.45± 0.017 | 26.05± 0.25 | 27.37± 0.25 | 1.23± 0.28 | 20.30 ± 0.37 |
| Gastric impaction (n = 12) | 7.41 ± 0.17$^c$ | 37.80 ± 2.17$^b$ | 3.61± 0.16 | 140.61±1.51 | 95.71± 0.88 | 1.39± 0.03 | 24.45± 1.04 | 25.58± 1.09 | 0.29± 1.02 | 24.41 ± 0.99$^a$ |
| Large colon impaction (n = 10) | 7.36 ± 0.03 | 42.11 ± 1.11 | 3.29± 0.24 | 139.62±1.90 | 97.77± 3.3 | 1.40± 0.13 | 25.15±0.17 | 26.42± 0.14 | 0.44± 0.36 | 23.88 ± 1.33$^b$ |
| Colon Intestinal displacements and strangulations (n = 10) | 7.32 ± 0.04$^c$ | 38.68 ± 0.01 | 3.08± 0.08$^a$ | 136.87± 1.74 | 95.93± 3.04 | 1.15± 0.05$^a$ | 19.45± 1.95$^a$ | 20.51± 2.02$^a$ | -5.26± 2.16$^a$ | 25.82 ± 1.22$^a$ |
| Proximal enteritis (n = 9) | 7.32 ± 0.04 | 44.5 ± 1.09 | 3.63± 0.49 | 136.37± 1.89 | 95.25± 3.35 | 1.27± 0.08$^b$ | 23.03± 4.06 | 24.37± 4.32 | -2.22± 4.08 | 25.46 ± 1.32$^a$ |
| Colitis (n = 4) | 7.27 ± 0.04$^a$ | 34.04 ± 3.04$^a$ | 4.57± 0.80 | 132.20± 1.73$^a$ | 91.55±2.56$^c$ | 1.69± 0.26 | 15.92± 0.18$^a$ | 16.94± 0.09$^a$ | -9.49± 0.75$^a$ | 29.25 ± 1.83$^a$ |
| Septic peritonitis (n =4) | 7.22 ± 0.12$^c$ | 32.58 ± 0.68$^a$ | 3.12± 0.52 | 140.32± 2.46 | 101.50±2.8$^b$ | 1.31± 0.09$^c$ | 16.14± 2.65$^a$ | 16.37± 2.94$^a$ | -9.85± 3.72$^a$ | 25.78 ± 1.55$^a$ |

a: $p \leq 0.001$: highly significant; b:$p \leq 0.01$; c: $p \leq 0.05$; NS: Non-significant


**Fig. 2. A) Gastric reflux consists mainly of overloading roughage in case of primary gastric impaction, note the incoming feed particles in the nasogastric tube. B) huge reddish-brown reflux which is characteristic for cases of proximal enteritis. C) Huge passive gastric reflux > 20L secondary to ileus and it is a consistent sign with proximal enteritis. D) Conjunctival mucous membrane showing petechial hemorrhage means colic is associated with severe endotoxemia and DIC*. E) Cyanotic conjunctival mucous membrane which is associated with terminal stage of colic. F) Congested (injected) and tacky (dehydrated) oral mucous membrane which is a sign of endotoxemia. G) Oral mucous membrane showing toxic line which indicates severe endotoxemia.**
**(*) DIC = Disseminated intravascular coagulation.**


**Fig. 3. A) hard fecal ball covered with mucous which is a consistent sign with impaction colic. B) small hard fecal ball covered with mucous which is a consistent sign with impaction colic. C) Mucous excreted from the rectum which indicates impaction. D) Tenesmus (straining) a sign of impactions note the tail lifting and bulging anus. E) Rectal prolapse secondary to severe straining due to severe L.C.I.**

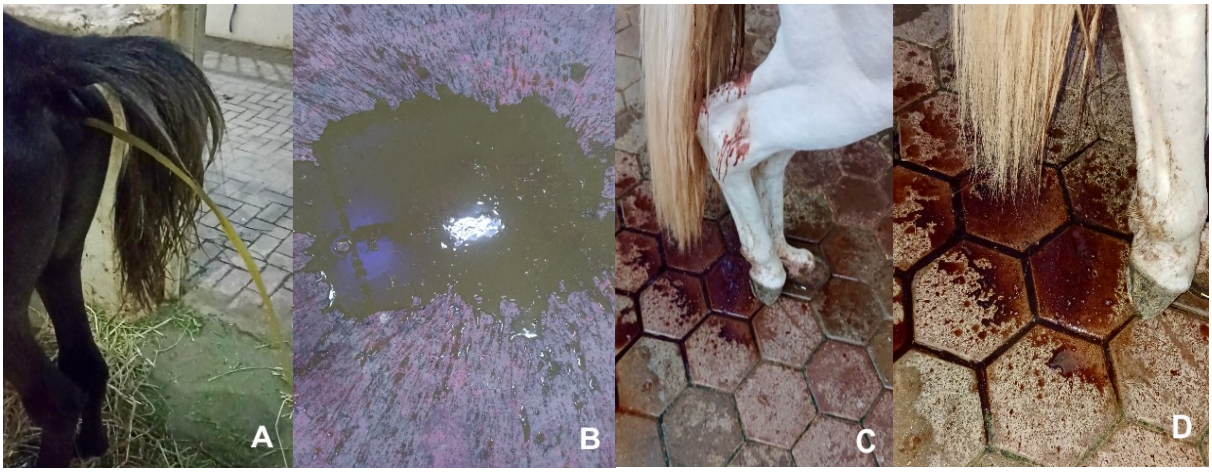


**Fig. 4. A, B) Profuse watery diarrhea of offensive odour in an Arabian colt which is a sign of colitis. C, D) bloody diarrhea of offensive odour in 5-year-old Arabian stallion which is a sign of severe critical colitis.**

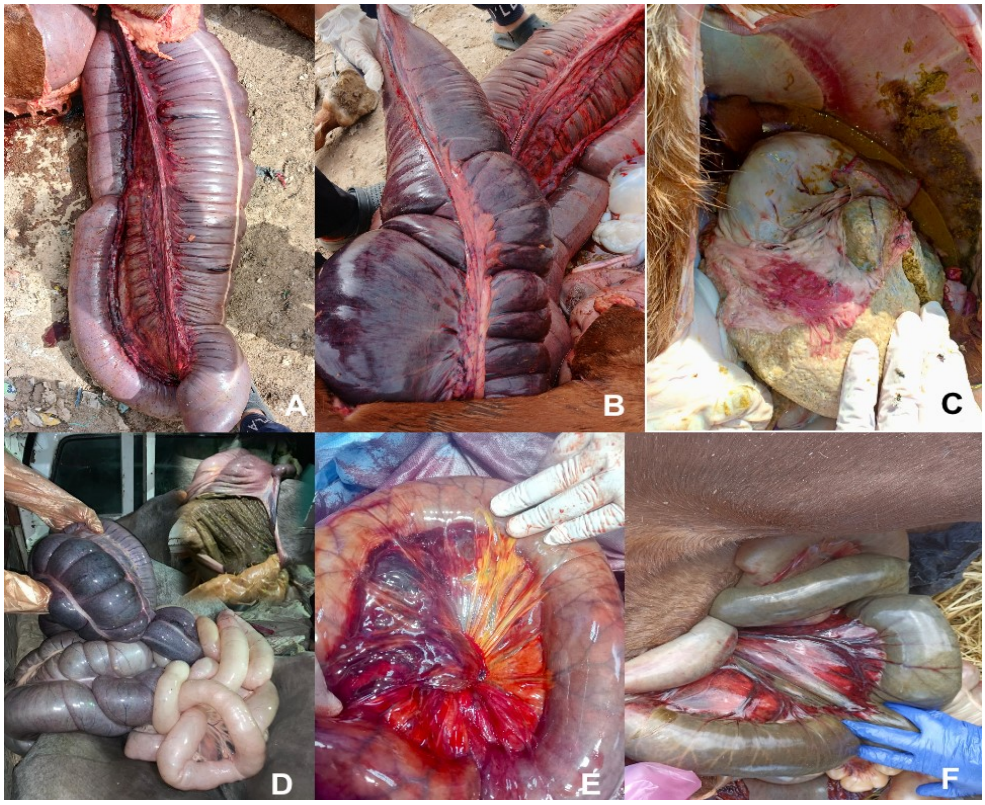


**Fig. 5. A) Large colon torsion note the severe congestion of mesenteric blood vessels between dorsal and ventral colons. B) Large colon torsion with cecal involvement note the dark discoloration of cecum. C) Gastric rupture in 9-month-old Arabian filly secondary to severe gastric impaction note the impacted feed material and ruptured inflamed gastric wall D) Necropsy of Arabian filly showed severe diahrea, dehydration before death due to severe colitis note the clear dark serosa and mucosa (up right corner) of the large colon. E) Strangulated small intestine (jejunum) in 20-year-old Arabian stallion secondary to incarcerated inguinal hernia note the congestion of mesenteric blood vessels. F) Severely inflamed and distended small intestine due to proximal enteritis which is secondary to septic peritonitis note the severely inflamed mesentery.**

**References**


1. Rhodes, D. M. and Madrigal, R. Management of Colic in the Field. Veterinary Clinics: *Equine Practice,* **37**(2), 421–439 (2021).
2. Bland, S. D. Equine colic: a review of the equine hindgut and colic. *Veterinary Science Development,* **6**(1),6223 (2016).
3. Bird, A. R., Knowles, E. J., Sherlock, C. E., Pearson, G. R. and Mair, T. S. The clinical and pathological features of gastric impaction in twelve horses. *Equine Veterinary Journal*, **44**, 105-110 (2012).
4. Jennings, K. M., Curtis, L., Burford, J. H. and Freeman, S. L. Prospective survey of veterinary practitioners' primary assessment of equine colic: clinical features, diagnoses, and treatment of 120 cases of large colon impaction. *BMC veterinary Research,* **10**, 1-10 (2014).
5. Whyard, J. M. and Brounts, S. H. Complications and survival in horses with surgically confirmed right dorsal displacement of the large colon. *The Canadian Veterinary Journal,* **60**(4), 381 (2019).
6. McGovern, K. F., Bladon, B. M., Fraser, B. S. and Boston, R. C. Attempted medical management of suspected ascending colon displacement in horses. *Veterinary Surgery,* **41**(3), 399-403 (2012).
7. Moore, J. N. Diseases Associated with Colic in Horses by Anatomic Location https://www.msdvetmanual.com/digestive-system/colic-in-horses/overview-of-colic-in-horses (2021). Accessed on 28th Mar. 2024.
8. Arroyo, L. G., Gomez, D. E. and Martins, C. Equine duodenitis-proximal jejunitis: a review. *The Canadian Veterinary Journal*, **59**(5), 510 (2018).
9. Galvin, N., Dillon, H. and McGovern, F. Right dorsal colitis in the horse: minireview and reports on three cases in Ireland. *Irish Veterinary Journal,* **57**, 1-7 (2004).
10. Arndt, S., Kilcoyne, I., Vaughan, B. and Dechant, J. E. Clinical and diagnostic findings, treatment, and short-and long-term survival in horses with peritonitis: 72 cases (2007-2017). *Veterinary Surgery,* **50**(2), 323-335 (2021).
11. Bowden, A., England, G. C., Brennan, M. L., Mair, T. S., Furness, W. A., Freeman, S. L. and Burford, J. H. Indicators of 'critical' outcomes in 941 horses seen 'out-of-hours' for colic. *Veterinary Record,* **187**(12), 492-492 (2020).
12. Westerman, T. L., Foster, C. M., Tornquist, S. J. and Poulsen, K. P. Evaluation of serum amyloid A and haptoglobin concentrations as prognostic indicators for horses with colic. *Journal of the American Veterinary Medical Association,* **248** (8), 935–940 (2016).
13. Dondi, F., Lukacs, R. M., Gentilini, F., Rinnovati, R., Spadari, A. and Romagnoli, N. Serum amyloid A, haptoglobin, and ferritin in horses with colic: association with common clinicopathological variables and short-term outcome. *The Veterinary Journal,* **205**(1), 50–55 (2015).
14. Nikvand, A. A., Jalali, S. M., Ghadrdan Mashhadi, A., Razi Jalali, M. and Hassanpour Amirabadi, S. Clinical, hematologic, hemostatic, and serum biochemical findings related to survival in Arabian horses with colic. *Veterinary Clinical Pathology,* **48**(3), 441–448 (2019).
15. Viterbo, L., Hughes, J., Milner, P. I. and Bardell, D. Arterial Blood Gas, Electrolyte and Acid-Base Values as Diagnostic and Prognostic Indicators in Equine Colic. *Animals,* **13**(20), 3241(2023).
16. Maskato, Y., Dugdale, A. H., Singer, E. R., Kelmer, G. and Sutton, G. A. Prospective feasibility and revalidation of the equine acute abdominal pain scale (EAAPS) in clinical cases of colic in horses. *Animals,* **10**(12), 2242 (2020).
17. Londoño, A. S. Nasogastric Intubation. Manual of Clinical Procedures in the Horse, 172–176 (2017).
18. Satoh, M. Sandwich enzyme-linked immunosorbent assay for serum amyloid A protein in horses. *Japanese Journal of Veterinary Research,* **42**(1), 31–31 (1994).
19. Gy, C., Leclere, M., Vargas, A., Grimes, C. and Lavoie, J. Investigation of blood biomarkers for the diagnosis of mild to moderate asthma in horses. *Journal of Veterinary Internal Medicine,* **33**(4), 1789–1795 (2019).
20. Monreal, L., ANGLES, A. M., Monreal, M., ESPADA, Y. and Monasterio, J. Changes in haemostasis in endurance horses: detection by highly sensitive ELISA-tests. *Equine Veterinary Journal*, **27**(S18), 120–123 (1995).
21. Navarro, M., Monreal, L., Segura, D., Armengou, L. and Añor, S. A comparison of traditional and quantitative analysis of acid-base and electrolyte imbalances in horses with gastrointestinal disorders. *Journal of Veterinary Internal Medicine,* **19**(6), 871–877 (2005).
22. Elsawaf, M. H., El-Mashad, N. E., Salib, F. A., Jaheen, A. H. and El-Sherif, M. A. Altered of Serum Amyloid A, Haptoglobin, Coagulation Profile, and Venous Blood Gases of Arabian Horses Suffering from Spasmodic and Flatulent Colic in Egypt. *Egyptian Journal of Veterinary Sciences,* **55**(7), 2079-2088 (2024).
23. Singer, E. R. and Smith, M. A. Examination of the horse with colic: is it medical or surgical?. *Equine Veterinary Education*, **14**(2), 87-96 (2002).
24. Burrell, K., Sutton-Walker, G., England, G. C., Burford, J. H. and Freeman, S. L. Prospective case study of critical decision making for horses referred for treatment of colic. *Veterinary Record,* e3615 (2023).
25. Burke, M. and Blikslager, A. Advances in diagnostics and treatments in horses with acute colic and postoperative ileus. *Veterinary Clinics: Equine Practice,* **34(**1), 81-96 (2018).
26. Gray, S. N., Dechant, J. E., LeJeune, S. S. and Nieto, J. E. Identification, management and outcome of postoperative hemoperitoneum in 23 horses after

emergency exploratory celiotomy for gastrointestinal disease. *Veterinary Surgery,* **44** (3), 379-385 (2015).

27. Norman, T.E. Abdominal ultrasound and palpation per rectum as complementary modalities in diagnosing equine abdominal pain. *AAEP Proceedings*, **60**,205-208 (2014).
28. Gavazza, A., Delgadillo, A. J., Gugliucci, B., Pasquini, A. and Lubas, G. Haematological alterations observed in equine routine complete blood counts. A retrospective investigation. *Comparative Clinical Pathology,* **11**, 131-139 (2002).
29. McConnico, R. S. Acute colitis in horses. Robinson's current therapy in equine medicine, 297 (2015).
30. Southwood, L. L. and Russell, G. The use of clinical findings in the identification of equine peritonitis cases that respond favorably to medical therapy. *Journal of Veterinary Emergency and Critical Care,* **17**(4), 382-390 (2007).
31. Nógrádi, N., Tóth, B. and Macgillivray, K. Peritonitis in horses: 55 cases (2004–2007). *Acta Veterinaria Hungarica,* **59**(2), 181-193 (2011).
32. Collins, N. M. and Pirie, R. S. Case series: diagnostic investigation and treatment of peritonitis in six horses. *Aust. Equine Vet.,* **30**(1), 47-52 (2012).
33. Walton, R. M. and Lawson, C. A. Equine hematology. *Equine hematology, cytology, and Clinical Chemistry,* 9-26 (2021).
34. Kiraly, L. N., Differding, J. A., Enomoto, T. M., Sawai, R. S., Muller, P. J., Diggs, B. and Schreiber, M. A. Resuscitation with normal saline (NS) vs. lactated ringers (LR) modulates hypercoagulability and leads to increased blood loss in an uncontrolled hemorrhagic shock swine model. *Journal of Trauma and Acute Care Surgery,* **61**(1), 57-65 (2006).
35. Monreal, L., Anglés, A., Espada, Y., Monasterio, J. and Monreal, M. Hypercoagulation and hypofibrinolysis in horses with colic and DIC. *Equine Veterinary Journal,* **32**(S32), 19-25 (2000).
36. Ludwig, E. K., Hobbs, K. J., McKinney-Aguirre, C. A. and Gonzalez, L. M. Biomarkers of intestinal injury in colic. *Animals,* **13**(2), 227 (2023).
37. Jacobsen, S. Use of serum amyloid A in equine medicine and surgery. *Veterinary Clinical Pathology,* **52**, 8–18 (2023).
38. Runge, K. E., Bak, M., Vestergaard, A., Stærk-Østergaard, J., Jacobsen, S. and Pihl, T. H. Serum amyloid A does not predict non-survival in hospitalised adult horses with acute colitis. *Veterinary Record*, **192**(7), 2644(2023)
39. Cray, C. and Belgrave, R. L. Haptoglobin quantitation in serum samples from clinically normal and clinically abnormal horses. *Journal of Equine Veterinary Science,* **34**(2), 337–340 (2014).
40. Nappert, G. and Johnson, P. J. Determination of the acid-base status in 50 horses admitted with colic between December 1998 and May 1999. *The Canadian Veterinary Journal*, **42**(9), 703 (2001).
41. Dart, A., Snyder, J., Spier, S. and Sullivan, K. Ionized calcium concentration in horses with surgically managed gastrointestinal disease: 147 cases (1988-1990). *Journal of the American Veterinary Medical Association,* **201**(8), 1244–1248 (1992).
42. Garcia-Lopez, J. M., Provost, P. J., Rush, J. E., Zicker, S. C., Burmaster, H. and Freeman, L. M. Prevalence and prognostic importance of hypomagnesemia and hypocalcemia in horses that have colic surgery. *American Journal of Veterinary Research,* **62**(1), 7–12 (2001).
43. Van Der Kolk, J., Nachreiner, R., Refsal, K., Brouillet, D. and Wensing, T. Heparinised blood ionised calcium concentrations in horses with colic or diarrhoea compared to normal subjects. *Equine Veterinary Journal*, 34(5), 528–531 (2002).
44. Adami, C., Westwood-Hearn, H., Bolt, D. M. and Monticelli, P. Prevalence of electrolyte disturbances and perianesthetic death risk factors in 120 horses undergoing colic surgery. *Journal of Equine Veterinary Science,* **84**, 102843 (2020).
45. McAuliffe, S. B. Disorders of Metabolism, Nutrition and Endocrine Diseases. Knottenbelt and Pascoe's color atlas of diseases and disorders of the horse. *Elsevier Health Sciences*, 218-229 (2014).
46. Borer, K. E. and Corley, K. T. T. Electrolyte disorders in horses with colic. Part 1: potassium and magnesium. *Equine Veterinary Education,* **18**(5), 266-271 (2006).
47. Johnson, P. J., Goetz, T. E., Foreman, J. H., Vogel, R. S., Hoffmann, W. E. and Baker, G. J. Effect of whole-body potassium depletion on plasma, erythrocyte, and middle gluteal muscle potassium concentration of healthy, adult horses. *American Journal of Veterinary Research*, **52**(10), 1676-1683 (1991).
48. Gomez, D. E., Arroyo, L. G., Stämpfli, H. R., Cruz, L. E. and Oliver, O. J. Physicochemical interpretation of acid-base abnormalities in 54 adult horses with acute severe colitis and diarrhea. *Journal of Veterinary Internal Medicine,* **27**(3), 548-553 (2013).
49. Bohn, A. A. Acid-Base and Electrolytes. *Equine Clinical Pathology,* 103-118 (2013).
50. Seahorn, T. L., Cornick, J. L. and Cohen, N. D. Prognostic indicators for horses with duodenitis-proximal jejunitis 75 horses (1985–1989). *Journal of Veterinary Internal Medicine*, **6**(6), 307–311 (1992).
51. Ranninger, E., & Bettschart-Wolfensberger, R. Polymorphic tachycardia in an anaesthetised horse with an undiagnosed pheochromocytoma undergoing emergency coeliotomy. *Veterinary Record Case Reports,* **8**(1), e001000 (2020).

# الاختلافات في مصل الأميلويد A والهابتوغلوبين وملف التخثر وغازات الدم الوريدية في الخيول العربية في حالات المغص المختلفة

**مازن حازم الصواف[1]، ناجي السيد المشد[1]، فايز عوض الله صليب[1]، الاء هلال جاهين[1] ومحمد أحمد الشريف[1]**

قسم الباطنة و المعدية - كلية الطب البيطرى - جامعة القاهرة - القاهرة - مصر.

**الملخص**

يعد مغص الخيول من أكثر المشاكل الميدانية انتشارًا في الخيول، ويعتبر السبب الأكثر شيوعًا للوفاة في الخيول. يختلف المغص في طبيعته فقد يكون انحشارًا أو انزياحًا أو خنقًا أو نوعًا من المغص الالتهابي. أجريت دراستنا على أربعة وستين حصاناً عربياً من كلا الجنسين (17 ذكراً و47 أنثى) تتراوح أعمارهم بين 6 أشهر إلى 26 سنة، منهم 15 حصاناً يتمتع بصحة جيدة سريرياً كمجموعة سيطرة، وتم تصنيف 32 حصاناً إلى مجموعات مغص مختلفة. استخدام أدوات تشخيصية مختلفة مثل التحليل المعملي حيث يمكن أن تكون تطبيقات APP وملف التخثر وغازات الدم الوريدية والكهارل بمثابة أداة قيمة لتشخيص حالات المغص المختلفة والتشخيص لها. وتم تقييمهم سريريًا على أساس العلامات الحيوية، وأصوات الأمعاء، وارتجاع المعدة، وفحص المستقيم. تم جمع عينات من الدم الوداجي وتحليلها لفحص أمراض الدم، وملف التخثر، و) SAAمصل الأميلويد (A ، والهابتوغلوبين، وغازات الدم الوريدي (VBG) وتحليل الشوارد. أظهرت النتائج وجود فرق معنوي في بعض المعايير المختبرة وخاصة بالنسبة لـ SAA والفيبرينوجين والكالسيوم المتأين($iCa^{2+}$) والفجوة الأنيونية (AG) والمعايير الحمضية القاعدية بين مجموعتي المغص والسيطرة. يوضح استنتاجنا أن اضطراباتSAA والفيبرينوجين والحمض القاعدي والكهارل يمكن أن تكون مؤشرات قيمة للتشخيص والتشخيص للمغص مع عدم وجود قيمة كبيرة للهابتوغلوبين.

**الكلمات المفتاحية:** المغص، الحصان العربي، SAA، الهابتوغلوبين، ملف التخثر، غازات الدم الوريدية.