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A Study on Clinico-Biochemical Evaluation in Equine Colic Patients

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ABSTRACT

The aim of this study was to compare the clinical and biochemical parameters in colic horses (n=20) with that of healthy control animals (n=10). Further, the measured parameters were also compared between survived and non-survived colic patients. Clinical parameters included were rectal temperature, respiration rate (RR), capillary refill time (CRT) and heart rate (HR). Biochemical parameters estimated at the time of admission were sodium, potassium, chloride, calcium and lactate dehydrogenase (LDH). The data were analyzed using Mann-Whitney U test. Except for rectal temperature, the vital parameters viz. RR (mean 28.8 vs. 17.2 breaths/min; $P<0.05$), CRT (mean 2.9 vs. 1.4 sec; $P<0.001$) and HR (mean 64 vs. 30.5 beats per min; $P<0.001$) were significantly elevated in the colic horses as compared to the control animals. No significant difference was observed in the levels of serum sodium, potassium and chloride between control and colic horses. However, mean calcium concentration was significantly lower ($P<0.01$) in colic horses (10.7 mg/dL) as compared to control horses (12 mg/dL). The LDH activity was significantly elevated ($P<0.05$) in colic group (mean 1289 U/L) as compared to control group (mean 649 U/L). A significant association ($P<0.05$) was found between abnormal LDH levels (>700 U/L) and proportion of horses with colic (70%) as compared to healthy ones (30%). Thirty percent (6/20) horses did not survive in this study and none of the measured parameters differ significantly between survived and non-survived colic horses ($P>0.05$). In conclusion, colic in equine patients results in elevation of RR, CRT, HR and LDH and decrease in serum calcium.

Keywords: Equine, colic, vital parameters, electrolytes, LDH, clinical outcome

Colic is relatively most frequently encountered health problem in equine patients that require immediate medical attention. It is one of the major causes of mortality in horses (Egenvall *et al.,* 2006) and, therefore, is a high priority health concern of horse owners (Mellor *et al*., 2001). There are many causes of equine colic, but lesion involving gastrointestinal tract (GIT) is the most important cause of colic (Robertson and Sanchez, 2010). Colic due to GIT problems is more severe and fatal than that originate from other organs (Ihler *et al.,* 2004). Clinical signs vary with severity of pain and include pawing, stamping, kicking, rolling, sweating and sometimes the inability to defecate (Radostits *et al.,* 2007).

Horses with acute colic often develop endotoxaemia (King and Gerring, 1988). Endotoxins, normally present within the bowel, gain access to the blood through damaged intestinal mucosa, or endotoxaemia occurs when gram negative organisms proliferate in body tissues. Endotoxaemia causes acute cardiovascular derangements secondary to intestinal ischemia (Morris, 1991) and amount of cardiovascular derangement is directly related with the observable clinical signs (Robertson and Sanchez, 2010). Physical parameters such as mucous member colour, heart rate (HR) and capillary refill time (CRT) are important for identification and differentiation of medical treatable colic cases from surgical needed cases (Ihler *et al*., 2004; Sutton *et al.,* 2009). These parameters are negatively affected by fatal endotoxaemia (Thoefner *et al.,* 2001; Skyes and Furr, 2005), therefore, plays an important role in evaluating the outcome of the colic patient. Also, secondary to

endotoxaemia, various biochemical imbalances have been reported in equine colic patients (Alsaad and Nori, 2010; Sabev and Dinev, 2011). Some of these parameters could be of importance in predicting prognosis in colic horses. So, for making decision on whether to treat colic medically or surgically or predicting the prognosis, an evaluation of clinico-biochemical parameters deserves investigation. The objective of this study was to investigate clinical and biochemical alterations in horses suffering from colic.

MATERIALS AND METHODS

Animals

This study was performed prospectively at the large animal clinics of the Teaching Veterinary Hospital, Department of Teaching Veterinary Clinical Complex, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab. To be included in the study, horses had to exhibit typical clinical signs of colic (pawing, sweating, and rolling). Other criteria to enter the study included a collection of a blood sample and an initial diagnosis of GIT disease. Each horse was evaluated at the time of admission in the clinics. Twenty equine colic patients with complete data were included in the final study as a colic group. The control group (n=10) consisted of horses stabled at the University and used for riding purpose. An owner's consent was obtained before including every case into the study. The studies performed in the colic and control horses were approved by the Institutional Animal Ethics Committee (GADVASU/2016/IAEC/37/4).

Clinical examination

Colic cases included in this study were subjected to complete clinical examination by the veterinarian on clinical duty in the clinics. Clinical examination of the control horses was performed the authors to ensure that they were clinically healthy. Clinical examination included recording of vital parameters (rectal temperature, HR, RR, colour of mucous membrane and CRT). A complete auscultation of the thorax and abdomen was performed to detect any abnormality of the heart, lungs or intestines. Additionally in colic horses, per-rectal examination, nasogastric intubation and ultrasonography was performed to diagnose the cause of colic.

Biochemical measurement

Blood sample (10 ml) was collected via venipuncture of the jugular vein and immediately transferred into a plain evacuated glass vial. Serum was harvested through centrifugation and stored at -20°C for future analysis of biochemical parameters. Frozen serum samples were thawed to room temperature and concentrations of sodium, potassium, chloride, calcium and LDH were measured *spectrophotometrically* by automated chemistry analyser (Vitros® 350 Chemistry System, Ortho-Clinical Diagnostics Inc.) using diagnostic kits (Johnson & Johnson Private Limited).

Statistical analysis

The statistical analysis was performed with Minitab statistical software (Minitab Inc., Version 14.2, State College, PA, USA). Descriptive statistics were calculated for quantitative and categorical data and the results were presented as mean, standard error (SE), range (minimum and maximum), frequencies and proportions. Non-parametric statistics were used to evaluate difference between the groups because of the small sample size. Relationships between vital parameters (rectal temperature, RR, HR and CRT), electrolyte concentrations, LDH concentrations, treatment groups (healthy and colic group) and clinical outcome (survived and non-survived) were examined by Mann-Whitney U test. Additionally, cases were grouped by whether serum LDH concentration was within the reference range (abnormal LDH concentration, >700 U/L); and Fisher's exact test was used to examine the association between abnormal LDH concentration and categorical data (control and colic; survived and non-survived). A 5% significance level was used.

RESULTS AND DISCUSSION

All control horses (n=10; 9 males) were Thoroughbreds (median 15 years, range 6-25 years). Twenty horses with colic (10 females) were aged 1.5 to 14 years (median 6.25 years) and comprised of 9 Thoroughbreds, 7 Marwaris and 4 non-descript). Majority of the horses (80%) suffering from colic belonged to the age group 2-10 years. Of them, nine horses were diagnosed with large colon impaction, eight with peritonitis (inflammatory colic), and three were diagnosed with spasmodic colic probably due to verminous cause.

Vital parameters recorded in horses belonged to control and colic groups are depicted in Table 1. Rectal temperature did not differ significantly (P>0.05) between the two groups although 55% of the horses had temperature 101ºF or more. Rectal temperature is usually within normal range in colicky horses, but sometimes it may be decreased as in cases of shock (Hillyer *et al*., 2008), or may be elevated due to increased muscular activity in animal while in pain or due to fever caused by inflammatory or infectious conditions in colic patients (Smith, 2000; Alsaad and Nori, 2009).

Table 1: Clinico-biochemical parameters in control and colic groups

Variable	Control group (n=10)	Colic group (n=20)	P value
Rectal temperature (ºF)	100.5±0.1 (100-101.4)	101±0.2 (100-102.8)	NS
Respiration rate (breaths/ minute)	17.2±0.8 (14-22)	28.8±2.6 (12-50)	0.01
Capillary refill time (seconds)	1.4±0.2 (1-2)	2.9±0.2 (2-5)	0.0002
Heart rate (beats/minute)	30.5±1.3 (25-38)	64.2±3.9 (36-100)	0.0001
Sodium (mEq/L)	137±2.5 (123-152)	141.9±1.3 (132-156)	NS
Potassium (mEq/L)	4.1±0.1 (3.6-4.6)	3.8±0.2 (2.8-5.3)	NS
Chloride (mEq/L)	102.6±2.4 (91-120)	106.7±3.9 (90-107)	NS
Calcium (mg/dL)	12.1±0.3 (10.8-13.3)	10.7±0.3 (11.1, 7-12.5)	0.0077
LDH (U/L)	649.3±65.1 (296-974)	1289±157 (536-2300)	0.01

Values are Mean ± SE (Range); NS: Difference statistically non-significant

Respiration rate was >35 breaths/min in 30% of the colic cases at the time of presentation and it was significantly higher (P=0.01) in colic group (mean 29±2.6 breaths/min) as compared to control group (mean 17±0.8 breaths/min). In a prospective study, Worku *et al.* (2017) also reported higher mean RR (30.9±7.3 breaths/min) in colicky horses. The probable reason for this could be pain, excitation, muscular activity or over-distension of stomach and small intestines that causes compression of the lungs (Smith, 2000; Radostits *et al*., 2007). A horse's mucous membranes are normally pale pink with a CRT of ≤2 sec (Radostits *et al*., 2007). In the present study, the conjunctival mucus membrane was congested in 80% of the colic horses. Mucous membrane may be congested due to toxaemia, septicaemia or development of endotoxaemia, and Ihler *et al.* (2004) concluded that horses with abnormal mucous membranes were having poor prognosis. Mean CRT was significantly prolonged (P<0.001) in colic horses (2.9±0.2 sec) as compared with control animals (1.4±0.2 sec). At the time of admission, CRT was <2 sec in 45% of the horses, 2-3 sec in 35% and >3 sec in 20% of the colic horses. Wormstrand *et al.* (2014) reviewed the colic cases from two equine hospitals in Norway and found the CRT <2 sec in 42.3% horses, 2-3 sec in 48.5% horses and >3 sec in 9.3% horses.

However, Curtis *et al.* (2015) while evaluating horses with clinical signs of abdominal pain, reported CRT of <2.5 sec in 92% of cases and >2.5 sec in 8% of colic horses. Such an increase in CRT in equine colic patients might be due to hypovolemia resulting in poor peripheral perfusion. Congested dark red to purple coloured mucous membranes with prolong CRT are indicative of severe dehydration and poor prognosis (Bryan *et al.,* 2009). The CRT was ≥4 sec in 66.7% of the non-survived colic horses in this study. The mean HR for colic horses in the present study was 64.2 beats per min (bpm), which was significantly higher (P<0.001) than the control horses having a mean HR of 30.5 bpm. More than two fold increase in the mean HR was observed in colic group as compared with control group. In a study on 22 referred colic horses, Hesselkilde *et al.* (2014) also reported significantly higher (P<0.0001) HR in colic patients (51.6±11.8 bpm) as compared to control group (37.4±3.7 bpm). Similarly, higher mean HR (57.5±10.1 bpm) was also reported by Worku *et al.* (2017) in equine colic patients. Persistently elevated HR in colic patients could affect the cardiac functioning.

In a previous study involving postoperative horses, high HR was identified as a risk factor for developing ventricular arrhythmias (Morgan *et al.,* 2011). Heart rate has been consistently associated with abdominal pain severity and

mortality, and identified as a useful prognostic indicator of survival (Jennings *et al*., 2014; Curtis *et al.,* 2015). The elevation in HR in colic cases could be due to violent struggle, severity of pain and concurrent endotoxaemia.

Cardiovascular parameters such as HR, mucous membrane colour and CRT are widely used as indicators of disease severity (Curtis *et al*., 2015). These are the clinical signs associated with endotoxaemia in horses with GIT lesions (Moore, 1994). Results from previous clinical studies indicated that approximately 25-35% of horses presented with colic had concurrent suspected septicemia and endotoxaemia (Senior *et al.,* 2011). Furthermore, the severity of the intestinal lesion has been directly correlated with degree of endotoxaemia which may further lead to development of severe septic shock or multiple organ failure in horses with gastrointestinal disorders that cause colic (Morris, 1991; Danek and Żurek, 2014). The ultimate outcome of endotoxaemia is death due to cardiac failure. So, timely correction of underlying endotoxaemia by evaluating the vital parameters may help reduce mortality in colic patients.

Concentrations of biochemical parameters (sodium, potassium, chloride, calcium and LDH) in the control and colic groups at the time of admission are presented in Table 1. The concentrations of sodium, potassium and chloride did not differ significantly between colic and healthy horses. In agreement with the present study, previous studies also reported non-significant differences in the concentrations of sodium (Baher *et al*., 2014) and potassium (Ayaz *et al.,* 1999) between colic and healthy horses. However, some studies have reported hyponatremia, hyperkalaemia, hypochloraemia and hypocalcaemia in colic horses (Sabev and Dinev, 2011; Hesselkilde *et al.,* 2014). Hyponatremia and hypochloraemia in colic horses might be due to losses with excessive sweating while in pain or due to internal losses into the GIT (Lohmann and Barton, 2004). Hyperkalemia in colic horses might be due to the decreased potassium excretion with urine, induced by hypovolaemia and impaired renal function, as well as trauma and necrosis to muscle cells resulting in intracellular potassium transfer into the extracellular space (Sabev and Dinev, 2011).

Total serum calcium was mildly decreased ($P<0.01$) in colic cases (10.7±0.3 mg/dL) as compared with control animals (12.1±0.3 mg/dL). Similar to this study, Delesalle *et al.* (2005) and Hesselkilde *et al*. (2014) also reported reduced calcium levels in horses suffering from colic as compared to healthy horses. Hypocalcaemia has been observed in humans with gram-negative septicemia (Zaloga and Chernow, 1987) as well as in horses with strangulating and ischemic intestines (Dart *et al.,* 1992). Hypocalcaemia in colic patients could be due to loss of calcium in sweat, lactic acidosis, and functional disturbances of the small intestine (Corley, 2007). Another possible reason for low blood calcium could be endotoxin induced secondary hypoparathyroidism as well as renal damage resulting into inhibition of hydroxylation of 1, 25 vitamin D (Lohmann and Barton, 2004). In the present study, hypocalcaemia was not associated with clinical signs because the decrease in blood calcium levels was very mild.

The LDH activity was significantly higher ($P<0.01$) in colic group (1289±157 U/L) as compared with healthy horses (649.3±65.1 U/L). A significant association ($P<0.05$) was found between abnormal LDH levels (>700 U/L) and proportion of horses with colic (70%) as compared to healthy ones (30%, Table 2).

Table 2: Abnormal lactate dehydrogenase activity in relation to control and colic groups

Lactate dehydrogenase (U/L)	Control group		Colic group	
	No.	Percent	No.	Percent
≤700	7	70	6	30
>700	3	30	14	70

Significant association was found between abnormal LDH levels (>700 U/L) at the time of admission and proportion of colic horses (Fisher's exact test $P<0.05$)

Human tissues have on an average about 500 times the total LDH levels found in the serum, with very high levels reported in the myocardium (25000 IU/g), lung (9500 IU/g), liver (9000 IU/g) and skeletal muscle (9000 IU/g) (Drent *et al.,* 1996). In horses, LDH is found in numerous tissues with the skeletal and cardiac muscle having the highest concentrations (Thornton and Lohni, 1979). Increased levels can serve as indicators of disturbed cellular integrity induced by pathological conditions (Drent *et al.,* 1996). Even a small amount of tissue injury can cause significant elevation in LDH activity in extracellular fluid which can therefore be used to detect cell injury or death

(Panteghini and Bais, 2008). Smuts *et al.* (2016) found significantly higher levels of LDH activity in horses with colic because of sepsis or neoplastic lesion than in those with colic because of non-septic inflammation or mechanical obstructions.

Out of the 20 colic horses in this study, 70% were survived to discharge from the hospital. Of the non-survived cases, two had impaction at the pelvic flexor, two had peritonitis and two had spasmodic colic. According to Christophersen *et al.* (2014), the overall survival rate in colic horses was 68%. Tympanic colic, simple obstruction and displacement of colon had high survival rates (94%, 88% and 89%, respectively), while horses with ruptured viscous had a low survival rate (2.7%).

Table 3: Clinico-biochemical parameters according to clinical outcome

Variable	Clinical outcome		P Value
	Survived (n=14)	Non-survived (n=6)	
Rectal temp. (ºF)	100.8±0.2 (100-102.2)	101.5±0.5 (100-102.8)	NS
Respiration rate (breaths/min)	26.4±3.3 (12-50)	34.2±3.5 (20-45)	NS
Capillary refill time (seconds)	2.9±0.3 (2-5)	2.8±0.3 (2-4)	NS
Heart rate (beats/minute)	62.4±5.3 (36-100)	68.3±4.3 (56-86)	NS
Sodium (mEq/L)	141.64±1.24 (135-153)	142.5±3.36 (132-156)	NS
Potassium (mEq/L)	3.82±0.18 (2.8-5.3)	3.85±0.39 (3.0-5.2)	NS
Chloride (mEq/L)	107.9±5.62 (90-107)	103.67±1.43 (99-108)	NS
Calcium (mg/dL)	10.73±0.29 (8.6-12.5)	10.75±0.79 (7.0-12.5)	NS
LDH (U/L)	1271±189 (536-2250)	1332±312 (45-2300)	NS

Values are Mean ± SE (Range); NS: Difference statistically non-significant.

Although the mean rectal temperature, RR and HR were higher in non-survived cases as compared to survived cases, but the statistical analysis did not revealed significant differences (P>0.05) between them (Table 3). The role of vital parameters in predicting the outcome in horses with colic has been demonstrated in previous studies. Lee *et al.* (2015) reported significant increase in the mean rectal temperature in the non-survived colic horses (101.6 ºF) as compared to the survived horses (100.6 ºF); whereas, Ihler *et al.* (2004) reported no prognostic significance of the body temperature in horses with colic. Youssef *et al.* (2009) reported a significant increase in RR in the non-survived horses (30±1.9 breaths/min) as compared to the survived horses (17.8±0.6 breaths/min); however, Lee *et al.* (2015) could not able to find significant difference in RR between survived and non-survived horses. According to Southwood *et al.* (2010), CRT is of great value in assessment of prognosis of colicky horse, as more it increases, the most the case gets worse. Parry *et al.* (1983) reported significantly increased CRT in non-survived horses as compared with survived colic cases. Youssef *et al.* (2009) and Lee *et al.* (2015) reported significantly high mean HR in the non-survived colic horses as compared to the survived horses.

Similarly, electrolyte concentrations also did not differ significantly between survived and non-survived colic horses in this study (Table 3). In corroboration with this study, Lee *et al*. (2015) also found non-significant differences in the sodium, potassium, chloride and calcium concentrations in survived and non-survived colic horses. However, study conducted by Youssef *et al.* (2009) showed significant associations between non-survived horses with colic and increased plasma sodium (P<0.001; OR: 2.897; CI: 1.754-4.784) and decreased levels of potassium (P<0.001; OR: 0.074; CI: 0.023-0.243) and chloride (P<0.001; OR: 0.535; CI: 0.379-0.753).

Further, the mean LDH concentration did not differ significantly between survived and non-survived colic horses (Table 3) and the proportion of horses with abnormal LDH levels (>700 U/L) was not associated with negative outcome (P>0.05) (Table 4). However, Lee *et al.* (2015) in a retrospective study evaluated the records of 119 horses admitted to the equine hospital and found significantly higher LDH (U/L) levels in non-surviving colic horses (601.5±249.7 U/L) as compared with surviving horses (374.9±194.7 U/L). The non-significant results found in

present study could be ascribed to comparatively small number of non-survived cases.

Table 4: Abnormal lactate dehydrogenase levels in relation to clinical outcome

Lactate dehydrogenase (U/L)	Clinical outcome			
	Survival		Non-survival	
	No.	Percent	No.	Percent
≤700	4	28.6	2	33.3
>700	10	71.4	4	66.7

No significant association was found between survivability and LDH levels >700 U/L at the time of admission (Fisher's exact test P>0.05).

In conclusion, clinical signs, such as RR, HR and CRT and biochemical parameters, such as calcium and LDH are negatively affected by colic in this study. Although, these parameters are significantly influenced by the severity and duration of the colic, as well as owner's decision to bring colic patient to the veterinary clinics, but their timely evaluation could help in better management of equine colic cases.

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