Clinico-Physiological Observations and Haematological Profiling of Colicky Equine

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Abstract: The present study was carried out on 17 positive cases (horses) of colic. Two cases died during the treatment. Ten apparently healthy equines (horses) were also included in the study which constituted control group. Clinico-physiological observations included clinical signs, temperature, pulse, respiratory rate per min and colour of mucosa and were noted daily before start of treatment. Laboratory diagnosis of equine colic was done by determining haematological parameters. Blood samples from control group as well as equines suffering from colic were subjected to haematological examinations comprising of haemoglobin, packed cell volume, total erythrocyte count, total leucocyte count, differential leucocyte count and platelet count. There was great variation in duration and severity of clinical manifestations. In all the cases abdominal pain, loss of appetite, depression and change in colour of mucous membrane from light pink to dark pink was present. Pulse rate, respiration rate and capillary refill time were increased highly significantly (p<0.01) than the mean values from control group but temperature was within normal range and non-significantly (p>0.05) changed. In present study haemoglobin, PCV and TEC counts was increased significantly (p<0.05) on 1st day (before treatment) in comparison to healthy control group and after-treatment group. There was non significant (p>0.05) difference in their values between control group and after treatment group. Pattern of variation observed in the present study regarding haematological parameters of colicky cases can contribute in generating data for future studies and in laboratory diagnosis.

Key words: Clinical, colic, equine, haematological parameters, mucous

INTRODUCTION

Equine is an important draught animal species having an edge over oxen, buffaloes and camels with multipurpose utility for draught, transport, sports, patrolling and traditional ceremonies. Equine species plays a very important role in the socio-economic life of the human population. They are the beasts of burden and have also been playing important role in tourism promotion. They require immense scientific attention in terms of prompt and adequate health management. It is important to be able to quickly recognise the disorder as delay in treatment can decrease the likelihood of a successful recovery. Equine colic, a disorder manifested in abdominal pain, is the most frequent cause of emergency treatment and death in horses. Colic is a multi-factorial disorder that appears to be induced by environmental factors and possibly a genetic predisposition (Shirazi-Beechey, 2010). Causes of colic may be alimentary, functional, organic, mechanical or topographic. Scientists are of the opinion that veterinarian should remain aware about the breed differences in horses so that proper handling of colic cases in ponies and larger breeds can be done (Dunkel et al., 2013).

Haematological examination is an important tool to investigate the colic cases. Assessment of parameters

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such as packed cell volume can assist in assessing the cardiovascular status of the horse and can be used to help monitor response to treatment. Various researchers have studied haematological changes in colic. Equine colic is a syndrome that leads to abdominal pain, hydro electrolytic and acid-base disturbances and functional alterations of the vital organs like the lungs and heart (Orsini et al., 2008). Systemic studies in equines colic are lacking in India. Colic is an important cause of mortality and morbidity in domesticated horses yet many questions about this condition remain to be answered. Improvement in the diagnosis of equine colic is becoming an important target of the clinicians. This caused the launching of a study to find out clinico-physiological and haematological observations in the cases of equine colic.

**MATERIALS AND METHODS**

**Animals:** The present study was carried out on 17 positive cases of colic. Two cases died during the treatment. Ten apparently healthy horses (horses) were also included in the study which constituted control group.

**Clinico-physiological observations:** Clinical signs and temperature, pulse, respiratory rate per min and colour of mucosa were noted daily before start of treatment. Pulse rate was recorded by keeping finger tips on sub maxillary artery, respiration rate by keeping the hand in front of nostrils and rectal temperature by clinical thermometer. Oral gingival mucosa color was noted from time to time. Capillary refill time was measured in terms of time (seconds) taken by gingival mucosa to return to its normal colour after being compressed. Abdomen was auscultated on all the five areas (right and left paralumbar fossae, right and left lower flank and anterior ventral mid line) for intestinal sounds. Per rectal examination was also conducted by inserting hand in the rectum.

**Haematological parameters:** Laboratory diagnosis of equine colic was done by determining haematological parameters. Blood samples from control group as well as equines suffering from colic were subjected to haematological examinations comprising of haemoglobin, packed cell volume, total erythrocyte count, total leucocyte count, differential leucocyte count and platelet count. These parameters were analyzed as per the methods described by Jain (1986).

**Sampling procedure:** The techniques applied in relation to sampling procedures and laboratory investigations have been described.

**Collection of blood samples:** Using a 20 gauge 2.5 (cm) long sterilized needle and 20 (mL) disposable syringe, 5 mL blood was collected in sterilized test tubes containing disodium salt of ethylene diamine tetra acetic acid (1 mg mL⁻¹ of blood) as an anticoagulant from the jugular vein of colic affected horse before start of treatment and after the treatment (3rd day) for haematological studies. The blood samples from 10 healthy equines (control group) were also collected as described above on first day of presentation only and was subjected to estimation of haematological values. The same values have been considered in this study for comparison of values with colic affected equines after treatment (3rd day).

**Statistical analysis:** The data obtained in research work were statistically analyzed and compared as per the standard statistical procedures suggested by Snedecor and Cochran (1967).

**RESULTS AND DISCUSSION**

**Clinical manifestation:** There was great variation in duration and severity of clinical manifestations. In all the cases abdominal pain, loss of appetite, depression and change in color of mucous membrane from light pink to dark pink was present. Pulse rate, respiration rate and Capillary Refill Time (CRT) were highly significantly (p<0.01) increased than the values from control group but temperature was within normal range and non-significant (Table 1). The clinical variants of equine colic are given in the Table 2.

**Table 1:** Mean±SEM values of clinical parameters

<table>
<thead>
<tr>
<th></th>
<th>Healthy animals (n=10)</th>
<th>Before treatment (n=15)</th>
<th>After treatment (n=15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body temperature (°F)</td>
<td>99.8±0.17</td>
<td>99.5±0.14</td>
<td>99.6±0.14</td>
</tr>
<tr>
<td>Pulse rate (min⁻¹)</td>
<td>38.8±1.27</td>
<td>58.4±2.27</td>
<td>39.8±0.86***</td>
</tr>
<tr>
<td>Respiration rate (min⁻¹)</td>
<td>18.4±0.65*</td>
<td>44.4±2.22***</td>
<td>19.3±0.56***</td>
</tr>
<tr>
<td>Capillary refill time (sec)</td>
<td>1.6±0.12*</td>
<td>2.6±0.11***</td>
<td>1.1±0.09***</td>
</tr>
</tbody>
</table>

**Table 2:** Clinical variants of colicky equine

<table>
<thead>
<tr>
<th>Clinical symptoms</th>
<th>No. of cases (15)</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of appetite and depression, rectal temperature</td>
<td>15</td>
<td>100</td>
</tr>
<tr>
<td>(99.5±0.14°F), pulse rate per minute (58.4±2.27),</td>
<td></td>
<td></td>
</tr>
<tr>
<td>respiration rate per minute (44.4±2.2), capillary</td>
<td></td>
<td></td>
</tr>
<tr>
<td>refill time per second (2.6±0.11), mucous membrane</td>
<td></td>
<td></td>
</tr>
<tr>
<td>color light pink to dark pink, Repeatedly lying down</td>
<td></td>
<td></td>
</tr>
<tr>
<td>and stretching out the body with legs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lip curling, pawing repeatedly with front legs</td>
<td>9</td>
<td>60</td>
</tr>
<tr>
<td>Looking back at flank region, kicking at the abdomen</td>
<td>7</td>
<td>47</td>
</tr>
<tr>
<td>and decreased number of bowel movement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stretching out as if to urinate or difficulty in urination and distension of abdomen, disturbed urination pattern (positioning to urinate but no stream)</td>
<td>6</td>
<td>40</td>
</tr>
<tr>
<td>Rolling from side by side and severe sweating</td>
<td>4</td>
<td>27</td>
</tr>
</tbody>
</table>
Close clinical observations revealed that signs of abdominal pain in order of severity (less severe to most severe) were lying down for excessive periods, inappetence, restlessness, quivering of upper lip, turning of head towards the flank, repeated stretching as if to urinate, kicking with the feet at the abdomen, crouching as if wanting to lie down, sweating, dropping on the ground and rolling, respectively. Pain was either continuous or more commonly, intermittent with bouts of pain lasting as long as 10 min interspersed with similar periods of relaxation. The posture was often abnormal with the animal standing stretched out with the forefeet more cranial and the hind feet more caudal than normal. Dry mucous membranes, sweating and cool extremities were present in more severe cases. These signs are shown in Table 2. The clinical manifestations in present the study corroborated the earlier findings of Alsaaed and Abid (2009), Radositis et al. (2009) and Fielding et al. (2009).

Clinico-physiological observations: The basic parameters of health, i.e., temperature, pulse rate, respiration rate and capillary refill time was observed in each animal. The mean±SEM values of temperature, pulse rate, respiration rate and capillary refill time in healthy control group and colic group (before and after treatment) are given in Table 1.

Temperature (°F): The mean±SEM values of temperature before and after treatment of colic and of healthy group are presented in Table 1. Result showed that there was slight decrease in the rectal temperature of colic cases as compared to control group but there was no significant change was observed in the cases before and after the treatment when compared to healthy group. There was non significant (p>0.05) difference in values of temperature between control group and affected group (after treatment). These observations corroborated the earlier recordings (Azizunnesa et al., 2008). The decreased value of rectal temperature in colic could also be due to association of shock (Radositis et al., 2009).

Pulse rate (min−1): The mean±SEM values of pulse rate before and after-treatment of equine colic and in healthy group are presented in Table 1. In equines affected with colic on 1st day (before treatment) pulse rate was increased highly significantly (p<0.01) in comparison to healthy control group and after-treatment. There was no statistical difference in value of pulse rate between control group and after treatment group. Similar observations were recorded by earlier researchers (Azizunnesa et al., 2008; Nori and Alsaaed, 2009; Radositis et al., 2009). The immediate increase in pulse rate seems to be due to an impairment of neurotransmission in the Central Nervous System (CNS) and gastrointestinal tract (Roberts and Seawright, 1983). The increase in the pulse rate has been attributed to the distension of colon (Dabareiner and White, 1995). Pulse rate can be used for prediction of survivability in cases of colic and probability of survival decreases with increase in the heart rate (Parry et al., 1983). The increase in the pulse rate is apparently related to the degree of pain, vascular volume and cardiovascular responses and is a good indicator of severity of the condition. The heart rate increases due to pain, hemocoencentration and hypotension therefore, higher heart rates have been associated with more severe intestinal problems (Susan and Asa, 1998). Tachycardia can serve as indicators of abdominal pain, cardiovascular shock and endotoxiemia (Orsini and Divers, 2008).

Respiration rate (min−1): The mean±SEM value of respiration rate before and after treatment and in healthy group are presented in Table 1. In equines affected with colic respiration rate was increased highly significantly (p<0.01) on 1st day (before treatment) in comparison to healthy control group as well as after treatment group. There was no statistical difference in values of respiration rate between control group and after treatment group. The present study was in conformity to findings of Azizunnesa et al. (2008), Nori and Alsaaed (2009) and Radositis et al. (2009). The initial increase in respiration could have been due to impairment of neurotransmission in the Central Nervous System (CNS) and gastrointestinal tract and due to intestinal distension and related pain syndrome (Roberts and Seawright, 1983). Tachypnoea can be attributed to pain and diaphragmatic embarrassment due to abdominal distension and acidosis. Susan and Asa (1998) stated that the respiration rates may be increased due to fever, pain, acidosis or an underlying respiratory problem. Tachypnoea can serve as indicator of abdominal pain, cardiovascular shock and endotoxiemia (Orsini and Divers, 2008).

Capillary refill time (sec): The mean±SEM values of Capillary Refill Time (CRT) before and after treatment of equine colic are presented in Table 1. In the present study, the capillary refill time was increased highly significantly (p<0.01) on 1st day (before treatment) in comparison to both healthy control group and after-treatment group. Non significant (p>0.05) difference in values of CRT between control group and after-treatment group was observed. The present study is in support of studies of Chakrabarti (2007), Nori and Alsaaed (2009), Fielding et al. (2009) and Radositis et al. (2009). Mildly prolonged CRT indicated diminished peripheral perfusion and dehydration. The CRT usually prolonged due to vascular stasis (Susan and Asa, 1998).
Table 3: Mean±SEM value of haematological parameters

<table>
<thead>
<tr>
<th>Haematological parameters</th>
<th>Healthy animals (n = 10)</th>
<th>Before treatment (n = 15)</th>
<th>After treatment (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g dL⁻¹)</td>
<td>12.7±0.49*</td>
<td>14.86±0.440**</td>
<td>12.96±0.360*</td>
</tr>
<tr>
<td>PCV (%)</td>
<td>36.8±1.59*</td>
<td>43.9±1.340**</td>
<td>37.4±1.160**</td>
</tr>
<tr>
<td>Total erythrocyte count (10⁶ µL⁻¹)</td>
<td>7.62±0.39*</td>
<td>9.06±0.400**</td>
<td>7.97±0.359*</td>
</tr>
<tr>
<td>Total leukocyte count (10⁶ µL⁻¹)</td>
<td>9.22±0.59*</td>
<td>7.84±0.310**</td>
<td>9.13±0.350**</td>
</tr>
<tr>
<td>Total platelet count (10⁹ µL⁻¹)</td>
<td>263.9±8.70*</td>
<td>142.4±6.110**</td>
<td>250.8±23.170*</td>
</tr>
<tr>
<td>Absolute neutrophils (10⁶ µL⁻¹)</td>
<td>6.18±0.34*</td>
<td>4.11±0.110**</td>
<td>6.08±0.230**</td>
</tr>
<tr>
<td>Absolute lymphocytes (10⁶ µL⁻¹)</td>
<td>3.91±1.26*</td>
<td>2.54±0.090**</td>
<td>3.87±0.109**</td>
</tr>
<tr>
<td>Absolute monocytes (10⁶ µL⁻¹)</td>
<td>0.22±0.01</td>
<td>0.19±0.010</td>
<td>0.21±0.010</td>
</tr>
<tr>
<td>Absolute eosinophils (10⁵ µL⁻¹)</td>
<td>0.13±0.01</td>
<td>0.11±0.010</td>
<td>0.12±0.000</td>
</tr>
</tbody>
</table>

*Significant (p<0.05) when compared with the respective healthy mean value; **Highly significant (p<0.01) when compared with the respective healthy mean value

**Haematological parameters:** The mean±SE values of Haemoglobin (Hb), Packed Cell Volume (PCV), Total Erythrocyte Count (TEC), Total Leukocyte Count (TLC), total platelet count and Differential Leukocyte Count (DLC) of healthy control group and colicky equines (before and after treatment) are presented in Table 3.

**Haemoglobin (g dL⁻¹):** In present study, haemoglobin was increased significantly (p<0.05) on 1st day (before treatment) in comparison to healthy control group and after-treatment group. There was no significant (p>0.05) difference in values of Hb between control group and after treatment group. The present study is in conformity with the observations of Darabener and White (1995) and Nori and Alsaad (2009). Sullins (1990) stated that increased Hb might be due to varying degree of dehydration. The distension of intestine increases intraluminal pressure causing outpouring of extra cellular fluid into bowel leading to dehydration (Littlejohn, 1965). Splenic contraction in horses with intestinal colic due to release of catecholamines in response to pain, anxiety and circulating endotoxin (Bayly and Reed, 1980; Moore and White, 1982; Seaborn et al., 1994). Other researchers did not observe significant difference in the values of PCV (Varshney and Yadav, 1995). Many researchers have studied the prognostic significance of PCV and reported that in general, probability of survival decreases as the PCV increases (Parry et al., 1983; Orsini et al., 2008).

**Total erythrocyte count (×10⁶ µL⁻¹):** Total erythrocyte count was increased significantly (p<0.05) in equines affected with colic on 1st day (before treatment) in comparison to healthy control group and after treatment of colicky equines. There was non significant (p>0.05) difference in value of TEC between control group and after treatment of affected group. The present study is in conformity to the observations of Boles and Kohn (1977), Gay et al. (1979), Roberts and Sewright (1983), Auer et al. (1984) and Darabener and White (1995) and Orsini et al. (2008) and seems to be due to varying degree of dehydration (Sullins, 1990). The distension of intestine increases intraluminal pressure causing outpouring of extracellular fluid into bowel leading to dehydration (Littlejohn, 1965).

**Packed cell volume (%):** In present investigation PCV was increased highly significantly (p<0.01) on 1st day (before treatment) in comparison to healthy control and after-treatment groups. There was non significant (p>0.05) difference in value of PCV between control group and after-treatment affected group. The values in present study are in agreement with the observations of Thoefer et al. (2000), Braun et al. (2002) and Radositis et al. (2009). Sullins (1990) observed that increased PCV could be due to varying degree of dehydration. The distension of intestine increases intraluminal pressure causing outpouring of extracellular fluid into bowel leading to dehydration (Littlejohn, 1965).

Splenic contraction in horses with intestinal colic, due to release of catecholamines in response to pain, anxiety and circulating endotoxin has also been reported to cause an increase in PCV values (Bayly and Reed, 1980; Moore and White, 1982; Seaborn et al., 1994). Other researchers did not observe significant difference in the values of PCV (Varshney and Yadav, 1995). Many researchers have studied the prognostic significance of PCV and reported that in general, probability of survival decreases as the PCV increases (Parry et al., 1983; Orsini et al., 2008).

**Total leukocyte count (×10⁶ µL⁻¹):** In equines affected with colic on 1st day (before treatment) TLC was decreased significantly (p<0.05) in comparison to healthy control group and after treatment of colicky equines. There was non significant (p>0.05) difference in value of TLC between control group and after treatment of affected group. The decrease in total leukocyte count agrees with the observations of McClure et al. (1992), Darabener and White (1995), Cohen et al. (1995), Weiss and Evanson (2003), Orsini et al. (2008), Orsini and Divers (2008) and Radositis et al. (2009). On the contrary, Singh et al. (1975) and Boles and Kohn (1977) observed leukocytosis in fibrous foreign body impaction colic.

Bayly and Reed (1980) were of the opinion that leucopenia characterized primarily by neutropenia and lymphopenia in acute abdominal crisis suggests...
endotoxaemia and could be ascribed to neutrophil migration, sequestration and reticulo-endothelial removal in endotoxaemias (Arden, 1989). Enhanced permeability of the capillaries with chemotactic attraction of leukocytes may also be a possible factor. Dabareiner and White (1995) observed low number of leukocytes in non-survivors than in survivors in large colon impaction due to impacted mass causing pressure necrosis and mucosal damage. The decrease in total leucocyte count was due to relative decrease in absolute neutrophil and lymphocyte counts.

**Total platelet count (×10^9 µL^-1):** In equines affected with colic on 1st day (before treatment) TPC was decreased highly significantly (p<0.01) in comparison to healthy group and after treatment of colicky equines. There was non significant (p>0.05) difference in value of TPC between control group and after treatment of affected group. The values in present study corroborated the earlier observations (Nori and Alsaad, 2009; Radositis et al., 2009). This might be attributed to the release of endogenous mediators such as platelet activating factor in inflammatory disorders. The possible explanation may be related to endotoxin absorption from intestines in this study as in agreement with various other scientists (Sandholm et al., 1995; Weiss and Rashid, 1998).

**Differential leucocyte count (×10^9 µL^-1):** In equines affected with colic on 1st day (before treatment) absolute neutrophil count was decreased significantly (p<0.05) in comparison to healthy control group and after treatment of colicky equines. There was non significant (p>0.05) difference in value of absolute neutrophil count between control group and after treatment of affected group. Similar findings were observed by earlier researchers (Orsini et al., 2008; Radositis et al., 2009). Bayly and Reed (1980) were of the opinion that leucopenia characterized primarily by neutropenia in acute abdominal crisis suggests endotoxaemia and could be ascribed to neutrophil migration, sequestration and reticulo endothelial removal in endotoxaemias (Arden, 1989).

**Absolute lymphocyte count (×10^9 µL^-1):** The absolute lymphocyte count in equines affected with colic on 1st day (before treatment) was decreased significantly (p<0.05) in comparison to healthy control group and after treatment of colicky equines. There was non significant (p>0.05) difference in value of absolute lymphocyte count between control group and after treatment of affected group. The mean values are in agreement to those obtained by Wegmann et al. (1986) and Cohen et al. (1995). Bayly and Reed (1980) were of the opinion that leucopenia characterized primarily by lymphopenia in acute abdominal crisis suggests endotoxaemia. Lymphopenia should suggest sepsis, bacterial enteritis and severe viral enteritis (Cohen et al., 1995).

**Absolute monocyte count (×10^9 µL^-1):** The absolute monocyte count in equines affected with colic on 1st day (before treatment) was non significantly (p>0.05) decreased in comparison to healthy and after treatment mean value of colicky equines. There was also non significant (p>0.05) difference in value of absolute monocyte count between control group and after treatment of affected group.

**Absolute eosinophil count (×10^9 µL^-1):** The mean±SEM values of absolute eosinophil count are presented in Table 3. In equines affected with colic on 1st day (before treatment) the absolute eosinophil count was non significantly (p>0.05) decreased in comparison to healthy and mean value obtained after treatment of colicky equines. There was also non significant (p>0.05) difference in value of absolute eosinophil count between control group and after treatment of affected group.

**REFERENCES**


