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# CLINICAL AND LABORATORY INVESTIGATIONS OF GASTRIC DILATATION IN DRAFT HORSES

## BY

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## ABSTRACT

A total of forty native breed draft horses of both sexes were examined; from which twenty five cases exhibited clinical signs of abdominal pain, in addition to fifteen apparently healthy horses served as a control group. Clinical and laboratory variables were used to investigate gastric dilatation in draft horses. It was concluded that gastric dilatation in draft horses is a life threatening condition because of its difficult diagnosis under field condition; however, it could be tentatively diagnosed when the following variables are detected: severe unrelenting abdominal pain, presence of naso-gastric reflux through naso-gastric tube (only in cases of small intestinal obstruction), metabolic alkalosis, elevated anion gap, hyperlactemia, hyperglycemia and azotemia.

**Key words:** Clinical, Laboratory, Investigations, Gastric, Dilatation, Horses

## INTRODUCTION

Gastric dilatation in horses were a life threatening caused by gastric impaction or secondary to intestinal obstruction. It might be associated with overeating fermentable feedstuffs such as grains, lush grass, and beet pulp **(Khan, 2005).** If untreated, gastric dilatation associated with overeating could rapidly led to gastric rupture. If intestinal

obstruction was the cause, the obstruction most often involved the small intestine **(Khan, 2005 and Radostits et al., 2007).**

The fluid from the obstructed small intestine accumulated in the lumen of the stomach, causing dilatation of the stomach and retrieval of gastric reflux on passage of the nasogastric tube. Impaction of the stomach typically consisted of excessive dry and fibrous ingesta **(Kellam et al., 2000).**

Rupture of the stomach was a fatal complication of gastric dilatation. The stomach generally tear along its greater curvature. About two-thirds of all gastric ruptures occurred secondary to mechanical obstruction, ileus, and trauma; the remaining cases were due to overload or to idiopathic causes **(Owen et al., 1997).** Because the incidence of this condition is low, it was difficult to determine which factors might be most important. The most striking clinical sign associated with gastric impaction was severe abdominal pain. Due to the lack of other characteristic findings, the diagnosis most often was made at surgery, and the decision for surgery was based on unrelenting pain **(Khan, 2005).** Clinical signs associated with gastric dilatation include severe abdominal pain, tachycardia, and retching. The mucous membranes might be pale. Classically, these acute signs were replaced by relief, depression, and toxemia after the stomach had ruptured **(Blikslager, 2005 and Khan, 2005).** The present study is planned to investigate cases of gastric dilatation in draft horses by using some clinical and laboratory variables owing to facilitate their diagnosis.

## MATERIALS AND METHODS

### 1. Animals and Medical Records

A total of forty native breed draft horses of both sexes were examined; from which twenty five cases exhibited clinical signs of abdominal pain (sixteen females and nine males) aged between two to five years. In addition to fifteen apparently healthy horses of both sexes (ten females and five males) were randomly selected and served as a control group. This study was carried out between October, 2004 and october, 2008 at the Veterinary Teaching Hospital, Faculty of Veterinary Medicine, Mansoura University, Mansoura, Egypt. Complete medical record for each animal was recorded depending on the competent history which includes feeding on poor quality roughage as corn silage, rice hulls, rice polish, wheat bran and bean tibn rapidly following hard work with restriction of water intake.

Depending on the results of competent case history, physical examination, clinical findings, laboratory investigations and necropsy findings; the selected horses were categorized into three groups, the first included fifteen apparently healthy horses served as a control group whereas, the second one included ten horses suffered from gastric impaction; however, the third group included fifteen horses had small intestinal obstruction.

**2. Physical Examination:** Heart rate (bpm), respiratory rate (cpm), rectal temperature, appetite, color of visible mucous membranes, capillary refill time, skin fold test, nasogastric reflux/L, severity of abdominal pain, results of rectal examination and peristaltic movement of alimentary tract were recorded according to **Kelly (1984).**

**3. Blood Samples:** Two blood samples were collected from each animal; the first was an arterial type whereas the second was venous type.

**3. A. Arterial Blood Samples:** Three ml heparenized syringe were used to obtain arterial blood from the transverse facial artery for estimation of blood pH, partial pressure of carbon dioxide ($PCO_2$), partial pressure of oxygen ($PO_2$), bicarbonate ($HCO_3^-$), and base excess (BE) using blood gas analyzer *(AVL 995-Hb manufactured by AVL List GmbH Medizintechnik and distributed by AVL Medical Instruments UK Ltd)* according to **Reeves et al. (1989)** in addition to plasma electrolytes such as sodium and potassium which were measured using electrolyte analyzer (*AVL 984-S Analyzers manufactured by AVL List GmbH Medizintechnik and distributed by AVL Medical Instruments UK Ltd)* according to **Reeves et al. (1989)**. Chloride was also measured spectrophotometrically using commercial test kits supplied by (ABC diagnostics) according to the method described by **FeldKamp (1974).** The anion gap (AG) was calculated based on the equation $AG = (Na^+ + K^+) - (Cl^- + HCO_3^-)$ adopted by **Feldman and Rosenberg (1981).** Bicarbonate gap was calculated as the difference between delta gap and delta bicarbonate where delta gap = (Calculated anion gap – 12); delta bicarbonate = (24 – measured bicarbonate) according to the method described by **Wrenn (1990) and Oster et al. (1988).** Blood lactate and glucose levels were also determined spectrophotometrically using commercial test kits supplied by (Sigma Diagnostics, St Louis, Mo, USA and Diamond Company) according to the method described by **Noll (1974) and Trinder (1969),** respectively.

## 3. B. Venous Blood Samples

**3. B. i. Whole Blood:** Two ml of blood were collected into a clean, dry vacutainer tubes with 5mg sodium ethylene diamine tetra acetic acid (EDTA) as anticoagulant for hematological evaluation of packed cell volume (PCV %), total and differential leucocytic counts. Packed cell volume (PCV%) was determined using microhaematocrit tubes whereas total leucocytic count was carried out using hemocytometer and turkey's solution as diluents; however, differential leucocytic count was carried out using a stained blood film with Giemsa`s stain according to the method described by **Coles (1986).**

**3. B. ii. Serum Samples:** Ten ml of blood was obtained in a dry, clean and acid washed centrifuge tube without anticoagulant in order to obtain clear blood serum. Only clear, non-hemolysed sera samples were transferred into clean tubes and kept frozen until used for biochemical analysis of total serum protein, albumin, globulin, blood urea nitrogen and creatinine concentrations. The biochemical analysis of blood serum samples were determined spectrophotometrically using the commercial test kits supplied by (Boehringer Mannheim, ABC Diagnostics, Bio diagnostics and ABC Diagnostics) according to the method described by **King and Wootton. (1959), Drupt (1974), March et al. (1965) and Husdan and Rapoport (1968),** respectively.

**4. Post-Mortem examination:** All non-survived horses were subjected to necropsy procedures which were performed according to the method described by **Tayler and Hillyer, (1997)**.

**5. Statistical analysis**: Data were subjected to statistical analysis using statistical software program (SPSS for Windows, version 15, USA) according to **Ihler et al. (2004).**

# RESULTS

**Table (1).** Classification of the selected horses in this study

| Groups | (n = 40) | Age | Gender |
|---|---|---|---|
| **Control** | 15 | 1 – 5 years | Females (n = 10), Males (n = 5) |
| **Gastric Impaction** | 10 | 2 – 5 years | Females (n = 7), Males (n = 3) |
| **Small intestinal obstruction** | 15 | 1 – 3 years | Females (n = 9), Males (n = 6) |

**Table (2) .** Heart rate, respiratory rate and rectal temperature (means ± S D)  in clinically healthy horses and in those with gastric impaction and small intestinal obstruction.

| Groups | Heart rate(bpm) | Respiratory rate(cpm) | Rectal temperature c<sup>o</sup> |
|---|---|---|---|
| Control *(n = 15)* | $33.00 \pm 2.58^a$ | $13.10 \pm 1.59^a$ | $37.65 \pm 0.41^a$ |
| Gastric Impaction *(n = 10)* | $65.66 \pm 23.05^b$ | $28.16 \pm 6.43^b$ | $37.63 \pm 0.49^a$ |
| S. I. Obstruction *(n = 15)* | $91.94 \pm 10.94^c$ | $36.83 \pm 9.10^c$ | $37.83 \pm 0.52^a$ |

<sup>a, b, c</sup> : Variables with different superscript in the same column are significantly different at $p < 0.05$

**Table (3) .** Other Clinical findings in horses with gastric impaction and in those with small intestinal obstruction.

| | Gastric impaction *(n = 10)* | Small Intestinal Obstruction  *(n = 15)* |
|---|---|---|
| **Appetite** | **Anorexia** | **Anorexia** |
| **Visible         mucous membranes** | Congested | Congested (13/15), Cyanosed (2/15) |
| **Capillary refill time** | $3.83 \pm 0.05$ | $3.93 \pm 0.01$ |
| **Skin fold test** | $3.50 \pm 0.04$ | $3.83 \pm 1.36$ |
| **Naso-gastric reflux/L** | 0.00 | $5.33 \pm 2.42$ |
| **Severity of pain** | Severe (7/10), Moderate (3/10) | Severe |
| **Rectal examination** | Normal intestinal conformation | Distended loops of small intestine |
| **Abdominal auscultation** | Complete stasis | Complete stasis |

**Table (4).** Arterial blood gas report and acid base parameters  (means ±S D)  in clinically healthy horses and in those with gastric impaction and small intestinal obstruction.

| | | Control (n = 15 ) | Gastric impaction (n = 10) | Small Intestinal Obstruction (n = 15) |
|---|---|---|---|---|
| **PH** | | $7.38 \pm 0.03^a$ | $7.46 \pm 0.3^b$ | $7.48 \pm 0.02^b$ |
| **PCO2** | mmHg | $40.62 \pm 4.73^a$ | $48.1 \pm 3.18^b$ | $49.33 \pm 2.65^b$ |
| **PO2** | mmHg | $94.40 \pm 3.30^a$ | $81.66 \pm 3.93^b$ | $80.66 \pm 3.38^b$ |
| **HCO3** | mmol/L | $24.76 \pm 2.11^a$ | $29.16 \pm 1.47^b$ | $28.50 \pm 1.37^b$ |
| **BE** | mmol/L | $0.69 \pm 1.6^a$ | $7.66 \pm 1.2^b$ | $8.33 \pm 2.58^b$ |
| **Bicarbonate gap** | mmol/L | $0.06 \pm 3.04^a$ | $11.16 \pm 3.92^b$ | $15.78 \pm 2.89^b$ |

[a, b, c] : Variables with different superscript in the same raw are significantly different at $p < 0.05$

**Table (5).** PCV %, Total and differential leucocytic counts (means ± S D) in clinically healthy horses and in those with gastric impaction and small intestinal obstruction.

| | | Control (n = 15 ) | Gastric impaction (n = 10) | Small Intestinal Obstruction (n = 15) |
|---|---|---|---|---|
| **PCV** | % | $34.7 \pm 0.73^a$ | $43.83 \pm 2.88^b$ | $51.83 \pm 3.06^c$ |
| **TLC** | $10^3$ | $8.8 \pm 0.49^a$ | $8.3 \pm 0.29^a$ | $5.48 \pm 1.68^b$ |
| **Neutrophils** | % | $49.9 \pm 9.94^a$ | $51.0 \pm 7.09^a$ | $49.5 \pm 4.3^a$ |
| **Band cells** | % | $0.00^a$ | $0.00^a$ | $2.53 \pm 1.16^b$ |
| **Lymphocytes** | % | $47.5 \pm 9.42^a$ | $47.5 \pm 7.52^a$ | $46.00 \pm 4.0^a$ |
| **Eosinophils** | % | $0.56 \pm 0.43^a$ | $0.5 \pm 0.2^a$ | $0.50 \pm 0.97^a$ |
| **Monocytes** | % | $1.50 \pm 0.26^a$ | $1.00 \pm 0.80^a$ | $1.50^a \pm 0.10$ |

[a, b, c] : Variables with different superscript in the same raw are significantly different at $p < 0.05$

**Table (6).** Biochemical profiles (means ± S D) in clinically healthy horses and in those with gastric impaction and small intestinal obstruction.

| | | Control (n = 15 ) | Gastric impaction (n = 10) | Small Intestinal Obstruction (n = 15) |
|---|---|---|---|---|
| **Sodium** | mmol/L | $134.99 \pm .51^a$ | $135.33 \pm 0.33^a$ | $135.16 \pm 0.47^a$ |
| **Potassium** | mmol/L | $4.09 \pm 0.11^a$ | $1.98 \pm 0.21^b$ | $1.95 \pm 0.20^b$ |
| **Chloride** | mmol/L | $102.30 \pm 1.01^a$ | $90.00 \pm 2.04^b$ | $85.33 \pm 1.14^c$ |
| **Anion Gap** | mmol/L | $11.52 \pm 0.89^a$ | $18.06 \pm 1.85^b$ | $23.28 \pm 0.90^c$ |
| **Lactate** | mmol/L | $1.37 \pm 0.07^a$ | $4.10 \pm 0.50^b$ | $4.41 \pm 0.61^b$ |
| **Glucose** | mmol/L | $4.84 \pm 0.13^a$ | $6.69 \pm 0.07^{ab}$ | $9.47 \pm 2.11^b$ |
| **Total protein** | g/L | $73.30 \pm 5.69^a$ | $84.66 \pm 2.33^b$ | $85.16 \pm 4.35^b$ |
| **Albumin** | g/L | $36.60 \pm 3.56^a$ | $47.83 \pm 6.33^b$ | $47.33 \pm 2.25^b$ |
| **Globulin** | g/L | $36.70 \pm 4.37^a$ | $36.83 \pm 7.44^a$ | $37.83 \pm 2.78^a$ |
| **BUN** | mmol/L | $4.24 \pm 0.39^a$ | $7.66 \pm 0.46^b$ | $10.10 \pm 0.42^c$ |
| **Creatinine** | µmol/l | $137.0 \pm 15.0^a$ | $145.33 \pm 22.97^a$ | $154.16 \pm 14.28^a$ |

[a, b, c] : Variables with different superscript in the same raw are significantly different at $p < 0.05$
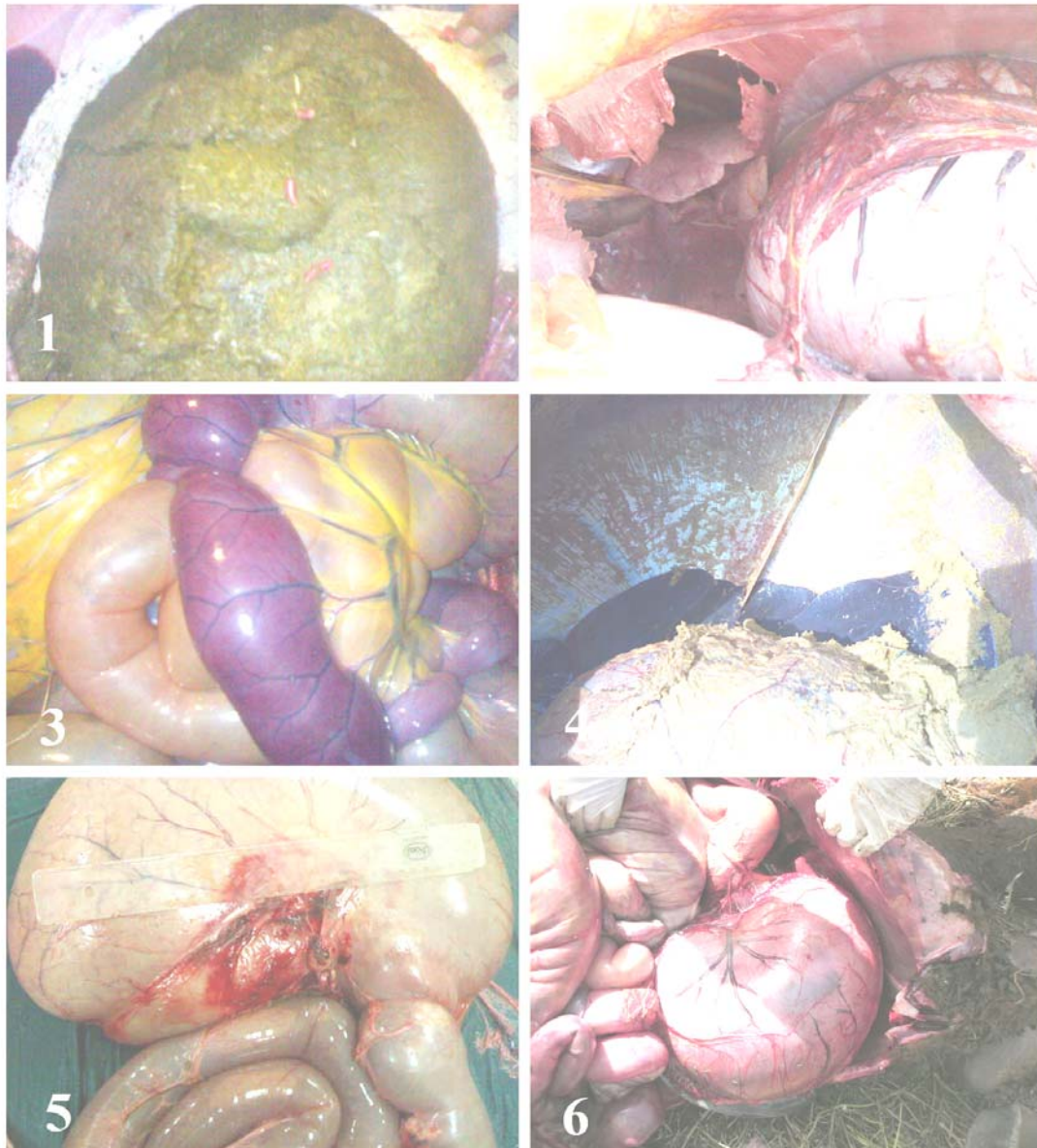
**Necropsy findings:** All cases of gastric impaction and small intestinal obstruction were non-survived and confirmed by P. M examinations. At necropsy, all cases had severe distension of the stomach either by impacted food mass with offensive odour which occurred in cases of gastric impaction (photo:1) or by fluid as in cases of small intestinal obstruction (Photo: 5, 6). Large number of Gastrophilus *equi larvae* were also detected in all cases of gastric impaction. Ruptured stomach was observed in (2/10) of gastric impaction where there was feed particles scattered in the abdominal cavity and on the diaphragm (Photo: 4). Congestion and cyanosis of small intestinal loops were also recorded in cases of small intestinal obstruction (Photo: 3).

# DISCUSSION

It was noticed that the age of the affected horses were ranged from 1 to 5 years old (Mean, 4.2 ± 4.17) (Table 1). The present findings is nearly similar to the previously obtained by **Radostits et al. (2007)** who reported that horses 2-10 years of age were 2.8 times more likely to develop colic than horses less than 2 years.

The heart rate (bpm) and Respiratory rate (cpm) showed also significant increase in horses with gastric impaction and those with small intestinal obstruction; their levels were (65.66 ±23.05 and 91.94 ± 10.94), respectively for the heart rate and (28.16 ± 6.43 and 36.83 ± 9.10), respectively for the respiratory rate. Such increase in the heart rate could be attributed to excitation and development of endotoxemia and eventually shock. These findings were coincided with those reported by **Moore (2006) and Radostits et al. (2007)** who stated that heart rate was considered as a proxy measure of endotoxemia in horses with colic. However, the increase in the respiratory rate could be attributed to toxemia and compression of the lung due to over-distension of stomach either by food or by fluid resulting from small intestinal obstruction. These findings were in agreement with that reported by **Kobulk et al. (1995), Smith (2000) and Radostits et al. (2007).**

Anorexia was evident in most cases of affected horses **(Table 2)**. The changes in appetite could be attributed to modulation of satiety centers caused by pain. **Kellam et al. (2000) and Radostits et al. (2007)** added that the reduced appetite was likely a consequence of pain which had inhibitory effect on gastrointestinal function causing a feedback loop in which the pain inhibits normal gut motility and function allowing accumulation of ingesta and fluid resulting in distension and further pain.

The color of visible mucus membranes reported in the affected horses was congested in (n = 23) and cyanotic in (n = 2). The congested mucus membranes could be attributed to shock and peripheral circulatory failure; whereas, cyanotic color could be due increase levels of $PCO_2$ **Patrick (1999), Smith (2000) and Radostits et al. (2007).**

Significant increase in both capillary refill time and skin fold test was recorded in affected horses. Such increase could be attributed to development of dehydration. These findings were supported by those reported by **Radostits et al. (2007).**

A nasogastric reflux (5.3 ± 2.42 liter) was obtained by nasogastric tube only in cases of small intestinal obstruction as a result of accumulation and sequestration of fluids in the

stomach and intestinal lumen and failure of their re-absorption in the small intestine leading to gastric dilatation. That finding was similar to those obtained by **Kellam et al. (2000).**

Abdominal pain was severe in (n = 22) and moderate in (n = 3) **(Table 2).** Similar observation was reported by **Radostits et al. (2007)** who stated that the intensity of pain reaction is often, but not always, related to the severity of the inciting disease. Rectal examination revealed that horses with small intestinal obstruction showed distention of small intestinal loops. However, horses with gastric impaction showed normal intestinal conformation. These findings were similar to that obtained by **Snyder and Spier, (1996).**

On abdominal auscultation, complete absence of peristaltic movement was observed in all cases of the affected horses. Such reduction of the borborygomi could be attributed to the gaseous distension of intestine or over-distension with food material which consequently caused prolongation of the so-journ of food in the intestine. The ileus associated with fluid distension of the small intestine and stomach causes severe pain leading to gastric rupture. These findings were in agreement with those reported by **Patrick (1999) and Radostits et al. (2007).**

PCV % showed significant increase in its levels in horses with small intestinal obstruction (51.83 ± 3.06 %) and those with gastric impaction (43.83 ± 2.88 %). Such increase in the levels of PCV % could be attributed to dehydration which resulted from excessive fluid loss. These findings were in harmony with those reported by **Snyder and Spier (1996), Snyder et al. (1996), El-Ghareib (1997), Kaneko et al. (1997), Susan and Denise (1997), O`Gilvie (1998), Smith (2000) and Radostits et al. (2007).**

The results of arterial blood gases and acid base parameters in horses with gastric impaction and small intestinal obstruction showed significant increase in blood pH values (7.46 ± 0.3 and 7.48 ± 0.02), respectively when compared with control group indicating presence of alkalosis, significant increase in $PCO_2$ (48.1 ± 3.18 and 49.33 ± 2.65), respectively indicates normal compensatory mechanism due to hypoventilation. Significant increase in bicarbonate levels (29.16 ± 1.47 and 28.50 ± 1.37 mmol/L), respectively base excess (7.66 ± 1.2 and 8.33 ± 2.58 mmol/L), respectively and bicarbonate gap (11.16 ± 3.92 and 15.78 ± 2.89 mmol/L), respectively were recorded with significant decrease in $PO_2$ levels (81.66 ± 3.93 and 80.66 ± 3.38 mmHg), respectively. The changes which recorded could be attributed to alkalosis associated with sequestration and loss of hydrochloric acid rich gastric secretions into gastric lumen. These findings were in harmony with those obtained by **Oster**

**et al. (1988), Adams and Polzin (1989), Malley (1990), Wrenn (1990), Kobulck et al. (1995), Kaneko et al. (1997), Ramamoorthy and Vora (1998), Kellam et al. (2000), Latimer et al. (2003) and Radostits et al. (2007)**. It is suggested that acute alkalosis can interfere with the body's ability to offload oxygen from the hemoglobin into tissues leading to cellular hypoxia (**Kahn et al., 2005).**

Total leucocytic counts (x $10^3$ ) showed significant decrease (5.48 ± 1.68)  with shift to left indicated by significant increase in band cell % (2.53 ± 1.16) in cases of small intestinal obstruction (**Table 5**). These changes could be attributed to endotoxemia and development of shock. These findings were coincided with those reported by **Giles et al. (1985), Chruch et al. (1986), Susan and Denise, (1997), O`Gilvie (1998),  Smith (2000) and Radostits et al. ( 2007).**

Significant decrease in plasma potassium and chloride concentrations was observed in horses with gastric impaction (1.98 ± 0.21 and 90 ± 2.04 mmol/L), respectively and those having small intestinal obstruction (1.95 ± 0.2 and 85.33 ± 1.14 mmol/L), respectively. These findings could be attributed to the state of anorexia and sequestration of these electrolytes into gastric lumen and failure of re-absorption of these electrolytes again from small intestine due to failure of re-absorption of $H^+$. Loss of the electrolytes into gastro-intestinal tract by hyper-osmolality of intestine that caused by long standing of food and feces in the GIT as in cases of impaction colic could be considered. These results were in agreement with those reported by **El-Ghareib (1997), Susan and Denise (1997), Tayler and Hillyer (1997), Ayaz et al. (1999), and Radostits et al. (2007).**

Anion gap and blood lactate levels (mmol/L) showed significant increase in horses with small intestinal obstruction and those with gastric impaction. Their levels were (23.28 ± 0.90 and 4.41 ± 0.61mmol/L) and (18.06 ± 1.85 mmol/L and 4.10 ± 0.50 mmol/L), respectively **(Table 6)**. The increased anion gap and blood lactate levels in such cases could be attributed to development of dehydration with subsequently peripheral lactic acidemia at the onset of shock. The excessive increase in blood lactate levels in cases of small intestinal obstruction could be due to increased absorption of lactate from small intestine which added to the lactate produced by anaerobic metabolism under the hypoxic state of shock. These results were in agreement with those reported by **Donawick et al. (1975), Moore et al. (1976), Bristol (1982), Gossett et al. (1987) and Van Hoogmoed (2005).**

Plasma glucose levels (mmol/L) showed significant increase in horses with small intestinal obstruction **(Table 6)**. Its level was (9.47 ± 2.1 mmol/L). Such increase in the glucose levels could be attributed to stress condition associated with catecholamine and glucocorticoid release. These results were coincided with those reported by **Coffman (1975) and Susan and Denise (1997) and Smith (2000).**

Total serum protein and albumin levels showed significant increase in horses with gastric impaction and those with small intestinal obstruction. Their levels were (85.16 ± 4.35 and 47.33 ± 2.25 g/L) and (84.66 ± 2.33 and 47.83 ± 6.33 g/L), respectively **(Table 6).** These changes could be attributed to dehydration and development of hypovolemia. These findings were in agreement with those reported by **Coles (1986), EL-Ghareib (1997)** and **Radostits et al. (2007).**

Blood urea nitrogen (mmol/L) showed significant increase in horses with gastric impaction and those with small intestinal obstruction. Their levels were (7.66 ± 0.46 and 10.10 ± 0.42 mmol/L), respectively. However, creatinine levels showed non significant changes in affected horses. The increased values of BUN indicates pre-renal azotemia. This could be attributed to reduction in the glomerular filteration as a result of dehydration and shock (**Table 6).** These results were in agreement with those reported by **Snyder et al. (1996), Craig (1998) and Susan and Denise (1997).**

In this study, all cases of gastric dilatation were detected at necropsy procedure because of the absence of satisfactory diagnostic tests and increased the need for exploratory laparotomy. Similar results were also reported by **Radostits et al. (2007).**

From the aforementioned results, it could be concluded that gastric dilatation in draft horses is a life threatening condition because of its difficult diagnosis under field condition; however, it could be tentatively diagnosed when the following variables are detected: severe unrelenting abdominal pain, presence of naso-gastric reflux through naso-gastric tube (only in cases of small intestinal obstruction), metabolic alkalosis, elevated anion gap, hyperlactemia, hyperglycemia and azotemia.

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الملخص العربي

فحوصات إكلينيكية ومعملية عن تمدد المعدة في خيول الجر

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تمت هذه الدراسة على عدد ٤٠ من خيول الجر المحلية تراوحت أعمارها بين سنتين إلى خمس سنوات منهم ١٦ فرسا و٩ أحصنة ظهرت عليهم جميعا علامات المغص هذا بالإضافة إلى عدد ١٥ من الخيول الطبيعية من الناحية الظاهرية و المعملية اختيرت كمجموعة ضابطة. تم تقسيم هذه الحيوانات علي أساس تاريخ الحالة المرضي و الأعراض الإكلينيكية والتغيرات في الدم ومصله والصفة التشريحية بعد النفوق إلى ثلاث مجموعات الأولى ضمت ١٥ من الخيول الطبيعية من الناحية الظاهرية و المعملية و ضمت الثانية عدد ١٠ خيول يعانون من لكمة المعدة أما المجموعة الثالثة فقد ضمت عدد من ١٥ الخيول يعانون من التفاف الأمعاء الدقيقة. خلصت هذه الدراسة إلى أن تمدد المعدة في خيول الجر يمثل تهديدا لحياتـــة وذلك لصعوبة تشخيصه على أرض الواقع ولكن بمساعدة الفحوصات الإكلينيكية والمعملية مثل وجود الألم المستمر الذي لا يزول بالمسكنات, الارتجاع المعدي وارتفاع معدل كلا من قلوية الدم و الفارق الانيونى ، وزيادة نسبة كلا من لاكتات و جلوكوز ويوريا لدم يمكن التنبؤ وتشخيص مثل هذه الحالات.