Case Report

IMPACTION COLIC IN A THOROUGHBRED HORSE AND ITS MANAGEMENT

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ABSTRACT

Nine-year-old Indian Thoroughbred gelding was presented to the Large Animal Medicine Out-patient Unit of Madras Veterinary College Teaching Hospital with a history of restlessness, anorexia, not voided dung and urine, patchy sweating, flank watching, pawing, frequent lying down and getting up. Clinical examination of the animal revealed severe dehydration with tachycardia and absence of borborygmi on both sides of the abdomen. Rectal examination revealed scanty dry faeces in the rectum with distended caecum. Distended intestinal loops with the absence of intestinal peristalsis were observed through ultrasound examination. Haemato-biochemical examination revealed increases creatinine, creatine kinase and lactate dehydrogenase. The animal was treated with Inj. Ringer's lactate and Inj. Flunixin meglumine. The animal was administered with liquid paraffin, magnesium sulphate, and simethicone along with the water through nasogastric intubation. Again the animal showed colic signs. Further the animal was sedated with xylazine and continuous rate infusion of Lignocaine @ 0.05mg/kg/min and fluids were administered through jugular catheter. The animal passed urine and showed a clinical improvement on following treatment. Animal started taking regular feed and voided dung on the third day of treatment.

Keywords: Thoroughbred gelding, Impaction colic and Lignocaine.

Received : 19.0.2024 Revised : 13.12.2024 Accepted : 18.12.2024

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INTRODUCTION

Equine colic is the most common and potentially life-threatening condition that requires immediate attention. Early attention can improve the chance of successful outcome for a horse with colic (Scantlebury *et al.,* 2014). Colic can be caused by a

variety of factors such as gastrointestinal problem, dietary factors, parasites, dental problems and sudden changes in activity level can stress the digestive system. Gastrointestinal involvement includes partial or complete obstruction, impactions, torsions, strangulation or inflammation in the horse's digestive tract (Sanchez, 2018).

Large intestinal impaction is the second most commonly reported cause of colic in horses (Abutarbush *et al.,* 2005) and it is the most frequent type of simple intestinal obstruction (Cribb *et al.,* 2006). It is characterized by the blockage of the intestines due to the accumulation of dehydrated masses of ingested material in the large intestine. Impactions are most frequently diagnosed in the pelvic flexure of the ascending colon, they can also occur throughout the gastrointestinal tract. The common sites for these impactions include the ileum, cecum, pelvic flexure and right dorsal colon (White and Dabareiner, 1997).

CASE HISTORY AND CLINICAL EXAMINATION

Nine-year-old Thoroughbred gelding was presented to the Large Animal Medicine Out-patient Unit of Madras Veterinary College Teaching Hospital with a history of restlessness, not voided dung and urine, flank watching and pawing.

Clinical examination of the animal revealed severe dehydration of increased skin tenting time of more than 8 secs with tachycardia (80 bpm) and absence of borborygmi on both sides of the abdomen. Rectal examination revealed distended caecum withscanty dry firmfaeces in the rectum.Patchy sweating was also noticed in flank and pectoral region. The animal was not responded to initial therapy again showed severe colic signs like frequent lying down and getting up, flank watching, rolling, profuse sweating all over the body and not cooperative (Fig. 1 and 3).

DIAGNOSIS

Blood and serum samples collected for haemato-biochemical examination during mild and severe phase of colic were presented in Table 1. Haematology revealed increased levels of PCV, Hb, total erythrocytic count and total leukocytic count while serum biochemical estimation revealed elevation of blood urea nitrogen (BUN), creatinine, Alkaline phosphatase, Aspartate amino-transferase, Creatine kinase, Lactate dehydrogenase, glucose and blood lactate levels. Transcutaneous abdominal ultrasound examination was performed with a low-frequency (2.5–5 MHz) curvilinear transducer which revealed distended large colon with impacted digesta and the absence of intestinal peristalsis (Fig. 5 and 6). Faecal samples were negative for parasitic eggs and oocysts. Sedimentation technique of faecal sample was negative for sand particles. Urinalysis revealed acidic, proteinuria and absence of crystal, casts and epithelial cells in sediments (Table 2).

TREATMENT AND DISCUSSION

Initially the animal was treated with Inj. Ringers lactate totaling to 15L intravenously and Inj. Flunixin meglumine @ 1.1mg/kg b.wt IV (Fikri *et al.,* 2023).

Enteral fluid therapy of powder MgSO4 @ 1g/kg b.wt. (Lopes *et al.,* 2002) liquid paraffin @ 10ml/kg b.wt., suspension aluminium hydroxide and magnesium hydroxide along with water of about 4L was administered through nasogastric tube (Giusto *et al.,* 2021). In the absence of gastric reflux, enteral fluid therapy through a nasogastric tube could be an effective treatment for distal gastrointestinal tract colic (Bookbinder and Prisk, 2023). But the animal didn't respond to the treatment and recurrence of severe colic occurred after 16 hours of treatment. Hence, for further management the animal was sedated with xylazine @ 0.5mg/kg b.wt. IV (Ruiz-Lopez *et al.,* 2023) and single lumen jugular catheterization was performed and Inj. Lignocaine was administered as a loading dose of 1.3 mg/kg via slow IV bolus over 10 to 15 minutes, followed by a continuous infusion at a rate of 0.05-mg/kg/min 2hours (Brianceau *et al.,* 2002 and Ruiz-Lopez *et al.,* 2023) along with aggressive fluid therapy of Ringer's lactate 25L and normal saline of 10L was administered intravenously. Further the animal was treated with antibiotic amoxicillin and cloxacillin 4.5g and vitamin B complex @ 20 mL intravenously (Fikri *et al.,* 2023). Enema was performed with 2L of water with liquid paraffin using gravity flow (Khan *et al.,* 2019). Animal had micturition after the treatment and became normal without any signs of colic. On 2nd day and 3rd day the animal was treated with fluid therapy, antibiotics and flunixin at anti-endotoxin dose of 0.25 mg/kg b.wt. (Sykes and Furr, 2005). Animal passed dung normally on 3rd day after treatment and started taking regular feed.

Intravenous lignocaine is a reemerging treatment that had been successful in treating impaction colic in horses. Lidocaine is a class 1B sodium channel blocker and used for anti-arrhythmic, analgesic and prokinetic properties (Torfs *et al.,* 2009, Brianceau *et al.,* 2002 and Salem *et al.,* 2016). Besides its prokinetic benefit, it inhibited neutrophil function and decreased inflammation (Cook and Blikslager, 2008 and Cook, *et al.,* 2009). Administration of vitamin B complex as a metabolic stimulant helped in energy metabolism and slightly reduced the lactic acid accumulation (Topliff *et al.,* 1981 and Gitari *et al.,* 2017). Decompression of the distended stomach or intestine through nasogastric intubation and hydration or administration of substances that increased drawal of water into its lumen such as magnesium sulfate helped to soften colonic contents and subsequently resolve the impaction (White and Dabareiner, 1997 and Lopes *et al.,* 2002). Outcome of colic is associated with the type of colic because delay in the identification of complicated cases increased the probability of death (Gitari *et al.,*2017). The treatment of colic includes pain management, decompression of the gastrointestinal tract, correcting biochemical and fluid imbalances, stimulation and maintenance of gastrointestinal motility, reducing gastrointestinal inflammation and elimination of endotoxins (Gitari *et al.,* 2017).

Fig.1. Flank watching

Fig. 2. Nasogastric intubation for gastric reflux and enteral fluid therapy

Fig.3. Profuse sweating noticed all over the body

Fig. 4. Enema

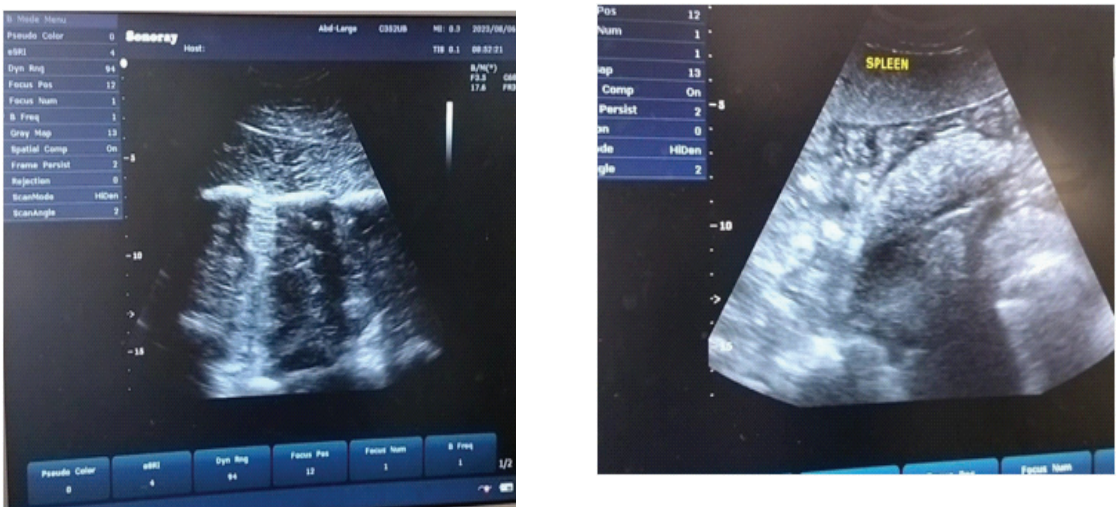

Fig. 5. Left-sided fast, localized abdominal sonography of the horse (FLASH) showed normal spleen and distended colon with impacted digesta

Fig. 6. Right-sided fast localized abdominal sonography of the horse showed normal liver and right dorsal colon with impacted digesta and absence of intestinal peristalsis

Table 1- Haemato-biochemical changes during mild, severe colic signs and after recovery

Parameters	Mild colic signs	Severe colic signs	3rd day after recovery	Reference value
PCV (%)	40.2	50.1	35.7	32-53
Hb (g/dL)	15.1	18.7	12.4	11.0-19.0
RBC (x 106/µL)	6.26	11.41	6.90	6.8-12.9
WBC (/µL)	7600	10,000	10800	5400-14300
Platelets (/µL)	1,88,000	2,32,000	1,96,000	1,00,000-6,00,000
Glucose (mg/dL)	98	135	78	75-115
BUN (mg/dL)	15.30	36.13	23.31	10-24
Creatinine (mg/dL)	1.95	2.73	0.91	1.2-1.9
Creatinekinase (IU/L)	158	489	89	2.4-23.4
LDH (U/L)	832	1326	192	162-412
ALP (U/L)	161	454	123	143-395
AST (U/L)	190	448	269	226-366
Lactate (mmol/L)	2.3	4.47	1.61	1-1.5
Albumin (g/dL)	3.6	2.8	3.4	2.6-3.7
Sodium (mmol/L)	144.5	147.6	139.6	132-146
Potassium (mmol/L)	5.61	4.23	3.92	2.4-4.7
Chloride (mmol/L)	108.9	109.9	103.4	99-109

Table 2- Urinalysis during severe colic signs

Parameters	Observed	Reference
Colour	Yellow	Yellow – colourless (creamy)
Specific gravity	1.020	1.008-1.040
pH	5.0 (acidic)	Alkaline
Protein	10 mg/dL Present +	7.5-8.5
Glucose	Negative	Negative
Ketone bodies	Negative	Negative
Blood	Absent	< 5 RBC
Bile salts	Absent	Negative

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