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Original Research

Clinical and Laboratorial Changes in Horses Subjected to a High-Pressure Modified Model of Small Colon Distention

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ABSTRACT

The objectives of the study were to study clinical and laboratorial variables of horses subjected to a modified high-pressure model of equine small colon distention. Eight healthy adult horses were subjected to inhalation anesthesia to undergo celiotomy. To perform intraluminal obstruction, the antimesenteric border was incised, and a sterile ball was introduced into the lumen. After the ball was positioned, it was inflated to 80 mm Hg, and the intestinal segment was relocated to the abdominal cavity. After 4 hours of distention, the ball was deflated and removed through a new incision. Blood samples were obtained immediately before anesthetic induction (T0), at the time when the ball was deflated (T4), and at 12-hour intervals during the postoperative period until 76 hours after surgery (T16, T28, T40, T52, T64, and T76). Peritoneal fluid samples were also collected at T0, T4, T16, T28, T52, and T76. Physical examinations were also performed at T0 and every 12 hours for 76 hours subsequently. In this induction model, the ball was not deformed and did not move due to the action of intestinal peristalsis. Leukocytosis was also detected at T16 and T28 ($P \leq .05$), mainly due to increased segmented and band neutrophils. In the peritoneal liquid, the total leukocyte count was increased at T16 and remained higher than the count recorded at baseline until T76. Clinically, the animals exhibited systemic inflammatory response syndrome because tachycardia, hyperthermia, and leukocytosis were observed at T16.

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1. Introduction

Among the different causes of colic, intestinal obstruction has been identified as the main cause of hospitalization and death in horses around the world [1]. Any condition that interferes with the aboral movement of ingesta in the gastrointestinal tract has the potential to induce intestinal distention and cause severe pain [2]. Simple obstructions are caused by the occlusion of the intestinal lumen, without primary vascular commitment, developing from intraluminal obstruction [3], intramural obstruction, or external compression [4]. Intramural distentions and mural compression of the small colon are typically caused by the accumulation of intestinal contents (compaction) or by concretions or foreign bodies [5].

When colliding with the intestinal wall, concretions initially stimulate spasmodic contractions around the calculus, resulting in acute obstruction and subsequent adynamic ileus. The occlusion of the intestinal lumen prevents the progression of the ingesta, fluids, and gas, resulting in the increased osmolarity of the intestinal contents and changes in the permeability of intramural vessels. Consequently, there is an influx of fluids from

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vessels present in the mucosa to the interior of the lumen. As a result, distention increases, and the resulting increased pressure interferes with venous drainage, promoting congestion and edema in the mucosa. This distention of the wall may cause ischemia as the obstruction continues and is aggravated [2].

The clinical signs exhibited by animals depend on the shape and location of the obstructions and/or concretions. Larger and more regular calculi are known to cause obstructions in thinner portions of the large intestine, such as the pelvic flexure, transverse colon, and small colon, with the two last locations representing the main points of obstruction [6]. Initially, signs of abdominal discomfort are mild and intermittent and may last for several days until the intraluminal obstruction is complete, when the signs of pain become moderate to severe and the manifestation continues [7]. The pressure against the wall decreases the vascular capacity, with a subsequent decrease in the perfusion and accumulation of interstitial fluid, leading to vascular compromise [8], which causes hyperemia and congestion of the affected portion, resulting in ischemia followed by necrosis of the segment. If the obstruction continues, the intestinal wall may be perforated, leading to fatal peritonitis [9].

Due to the severity and importance of obstructive lesions, in the last 25 years, several experimental models of intestinal ischemic obstruction in horses have been developed [10]. However, most of these models have promoted strangulating obstruction [11,12]. Therefore, the objectives of the present work were to study clinical and laboratorial variables of horses subjected to a modified high-pressure model of equine small colon distention, to mimic naturally occurring colic, such as that caused by enteroliths.

2. Materials and Methods

Eight adult mongrel horses, including four males and four females, aged 11 ± 2 years and weighing 370.5 ± 17.4 kg, were used. Before the experimental period, the animals underwent a clinical and blood evaluation to confirm their healthiness, and only healthy animals were included. The horses presented a good body condition (score 4); this condition was evaluated on a scale of 1 to 9, with 1 being extremely emaciated and 9 being extremely fat [13]. After confirming their healthiness, the animals underwent deworming and ectoparasite control. This study received ethical approval by Comissão de Ética e Bem Estar Animal (CEBEA)—FCAV/Unesp, Jaboticabal, São Paulo (protocol no. 007568-09).

After food and water fasting for 24 and 12 hours, respectively, the anesthetic procedure for celiotomy was initiated. Intravenous 10% detomidine hydrochloride (0.015 mg/kg) was administered as a preanesthetic. After 10 minutes, anesthesia was induced using 10% ketamine (2.0 mg/kg), followed by midazolam maleate (0.1 mg/kg), and the animal was placed in left lateral decubitus. In a continuous motion, orotracheal intubation was performed, and the animal was placed in a supine position on the operating table. Inhalation anesthesia was maintained with isoflurane vaporized in oxygen in a semiclosed circuit under controlled ventilation. The oxygen saturation was measured by the oximeter, remaining throughout the surgical procedure in the range 90 to 110 mm Hg. The mean arterial pressure was monitored through catheter (catheter IV Angiocath 20 G; BD), positioned on the facial artery mandibular branch, connected to the pressure gauge (Medplast Industry Hospital Products, Brazil). To keep the mean arterial pressure within the range of 50 to 90 mm Hg, 0.002 mg/kg/min of dobutamine was administered throughout the surgical procedure. Ringer's lactate solution, equivalent to 5% of body weight per day, was administered (intravenous) to maintain the blood volume.

To perform intraluminal obstruction of the small colon, celiotomy was performed using a preumbilical midline incision. The segment was selected by observing the presence of branches of the mesentery artery that irrigate this region. After identifying the region to be obstructed, the positioning of the oral and aboral portion of the small colon was observed. Subsequently, the antimesenteric border was aborally incised to the segment to be distended. Through this opening, a sterile and deflated ball (13 cm diameter; Water Polo Nabaiji, Decathlon, Villeneuve-d'Ascq, France) was introduced into the lumen of the small colon in the oral direction. After adequate positioning, the ball was inflated with air until reaching a pressure of 80 mm Hg, as measured by a manometer connected by a rubber tube. With the ball inflated, enterorrhaphy was performed using polyglactin 910 no. 2-0 (Fig. 1). In a continuous motion, the intestinal segment was relocated to the abdominal cavity, and the cavity was closed using Allis clamps. During the distention period, the ball pressure was not monitored because in previous tests outside the equine colon, it was observed that the ball did not lose pressure in the range of hours.

The animals were kept under inhalation anesthesia in the supine position for 4 hours, after which the small colon was exposed again to remove the ball. A new incision was made at the antimesenteric border, and the ball was punctured and deflated. After being completely deflated, the ball was removed from the intestinal lumen through the incision. Enterorrhaphy was performed again with polyglactin 910 no. 2-0 wire, and the small colon was repositioned in the abdominal cavity. Throughout the surgical manipulation of the small colon, from the selection of the segment to be obstructed to 45 minutes after the ball was removed (sufficient time to perform enterorrhaphy), the color and the integrity of the serosa were visually evaluated.

Enterorrhaphies were performed in two planes, using a Schmieden suture in the first layer and a Cushing pattern in the second. The linea alba was sutured including the peritoneum using a Sultan pattern with 0.6-mm nylon and a 40 × 1.2–mm needle. The subcutaneous tissue was closed with a continuous zigzag suture using polyglactin 910 no. 0, and the skin was sutured with a Wolff pattern, also using 0.6-mm nylon and a 40 × 1.2–mm needle. Subsequently, a sterile gauze dressing was placed over the skin suture and taped with adhesive cotton–reinforced polyethylene tape. The animals were then removed from the operating room and placed in a postanesthetic recovery room lined with rubber to minimize injuries while the animals recovered from the anesthesia.

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