Type III atresia coli with underdeveloped colon and hydroperitoneum in a newborn foal

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ABSTRACT: A 48-hour-old mixed breed pony colt was referred to the hospital because of abdominal discomfort, weak suckling reflex and prostration. During clinical investigation, supportive and symptomatic treatments were necessary, and an abdominal radiography was performed revealing a large intestine filled with feces and large amounts of gas, in addition to free fluid in the cavity. After 3 days of treatment, the foal had not yet defecated, thus exploratory abdominal surgery was indicated, but the owners declined. Therefore, the foal was humanely euthanized. Post-mortem examination revealed complete absence of the pelvic flexure. Subsequent portions of the dorsal, transverse and small colons were intensely reduced. In addition, at the abdominal cavity there was 850 ml of yellowish translucent liquid. These findings are compatible with hydroperitoneum and type III atresia coli at the pelvic flexure, associated with underdevelopment of the dorsal, transverse and small colons.

Key words: colic, neonatology, congenital malformation.

RESUMO: Um pônei mestiço de 48 horas de vida foi encaminhado ao hospital para avaliação por desconforto abdominal, diminuição do reflexo de sucção e prostração. Durante a investigação clínica, tratamentos de suporte e sintomáticos foram necessários, e radiografia abdominal foi realizada revelando cólon maior repleto de fezes e grandes quantidades de gás, além de fluido livre na cavidade. Após três dias de tratamento, o potro ainda não havia defecado, sendo a laparotomia exploratória indicada, mas os proprietários recusaram. Portanto, o potro foi humanamente eutanasiado. O exame post-mortem revelou ausência completa da flexura pélvica. Porções subsequentes dos cólons dorsal, cólon transverso e cólon menor se apresentaram intensamente reduzidas. Além disso, na cavidade abdominal havia 850 ml de líquido translúcido amarelado. Esses achados são compatíveis com o hidroperitônio e a atresia coli tipo III na flexura pélvica, associados ao subdesenvolvimento dos cólons dorsal, transverso e menor.

Palavras-chave: cólica, neonatologia, má-formação congênita.

Abdominal pain in foals is a diagnostic challenge; therefore, a good physical examination is critical when assessing a colicky foal (BOHANON, 2005). Colic starting at birth or shortly thereafter can be caused by meconium impaction, uroperitoneum or fecoliths, and also congenital disorders such as atresia coli, aganglionosis and hypoganglionosis (CHAFFIN et al., 1999; BOHANON, 2005).

Since physical examination has limitations to differentiate between surgical and non-surgical lesions, complementary exams are paramount. Peritoneal fluid analysis, nasogastric intubation, radiography and ultrasonography are indicated. Since rectal examination is not possible in young foals, abdominal radiography is very useful to access gas distention and the use of contrast allows better...
Intestinal atresia should be suspected when there is no fecal material on the anus, with presence of clear mucus, absence of meconium staining following repeated enemas, progressive abdominal distention and pain on a neonate (YOUNG et al., 1992; VATISTAS et al., 1996; CHAFFIN et al., 1999; BIASUTTI et al., 2017). It is a rare cause of colic that affects 0.44-3.1% of the equine neonates within the first 24-48h of life, resembling meconium impaction (BARTMANN et al., 2002; BIASUTTI et al., 2017). There are three types of atresia: type I is a membrane atresia, when there is a complete diaphragm obstructing the intestinal lumen, type II is a cord atresia, with blind ends of intestine joined by a fibrous and/or muscular cord, and type III is a blind-end atresia, characterized by a complete absence of an intestinal segment with a gap in the mesentery, accompanied by short bowel. It is believed that intestinal atresia should be suspected when there is no fecal material on the anus, with presence of clear mucus, absence of meconium staining following repeated enemas, progressive abdominal distention and pain on a neonate (YOUNG et al., 1992; VATISTAS et al., 1996; CHAFFIN et al., 1999; BIASUTTI et al., 2017). It is a rare cause of colic that affects 0.44-3.1% of the equine neonates within the first 24-48h of life, resembling meconium impaction (BARTMANN et al., 2002; BIASUTTI et al., 2017).

A 48-h-old mixed breed pony colt foal was referred to the veterinary teaching hospital for assessment of abdominal discomfort, weak suckling reflex and prostration. Owners reported the foal stood and nursed shortly after birth but had a weak suckling reflex. The foal was observed to urinate and pass meconium in the first 24 hours, but a few hours later it started showing abdominal pain. Local veterinarian was called, who decided to refer it to a hospital after it started showing abdominal pain. Local veterinarian was called, who decided to refer it to a hospital after performing the enema unsuccessfully on property.

During clinical investigation, supportive and symptomatic treatments were necessary and are described as follows. Initially, pain was moderate and controlled with flunixin meglumine (1.1mg/kg IV q.8 h in the first 24h and then when necessary) and short walks. Symptomatic treatment included ranitidine (2 mg/kg IV TID), penicillin (22000UI/kg IM SID), gentamicin (6.6mg/kg IV SID) and ceftiofur (4.4 mg/kg IM BID). Omeprazole were administered once at a dosage of 4mg/kg PO. Frequent enemas with Vaseline and warm water and stomach decompression via nasogastric tube were performed until presumptive diagnosis was reached. It is known that duodenal distention and reflux can cause abdominal distention in foals. Enemas were followed by clear mucus elimination, which was highly suggestive of atresia coli. Therapy with antibiotics was elected based on complete blood count, which was suggestive of sepsis. Central venous catheter was placed in the jugular vein and fluid therapy was instituted based in the Holliday-Seagar formula with Ringer’s lactate solution supplemented with glucose 5% and potassium.

At the end of day 1 of hospitalization, the foal had not yet defecated, and enemas resulted in clear mucus, without feaces in the rectum. Urine was diluted and abundant. Since there was no reflux at this point and the foal manifested interest in nursing, nasogastric tube was removed, allowing the foal to nurse and walk every hour. Blood glucose levels remained around 130-180mg/dL.

On day 2, abdominal distention was intense, and the foal had stopped nursing. Nasogastric tube was placed again, recovering 1L of spontaneous

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Table 1 - Hematological and biochemical profile of a 48-hour-old foal diagnosed with *atresia coli* type III at the Veterinary Hospital of the Universidade Federal de Minas Gerais, Brazil.

| HEMATOLOGY                        | REFERENCE RANGE |
|-----------------------------------|-----------------|
| Fibrinogen                        | 600 mg/dL       |
| Colorless to light yellow plasma  | 100 – 400 mg/dL |
| Red blood cells                   | 9.05 million/mm³|
| 6.6 – 11 million/mm³             |
| Hemoglobin                        | 14 g/dL         |
| 11 – 16 g/dL                      |
| Hematocrit                        | 40 %            |
| 30 – 44 %                         |
| MCV                               | 44.2 fL         |
| 38 – 51 fL                        |
| MCH                               | 35 %            |
| 35 – 39 %                         |
| MCH                               | 15.47 pg        |
| 13 – 19 pg                        |
| Total leucocytes                  | 8.450 / mm³     |
| 5.600 – 11.600 / mm³             |
| Neutrophils                       | 90% (7.605/mm³) |
| 2.600 – 6.700 / mm³              |
| Lymphocytes                       | 7% (591.5/mm³)  |
| 1.100 – 5.700 / mm³              |
| Monocytes                         | 3% (253.5/mm³)  |
| 0 – 700 / mm³                    |
| Platelets                         | 298.000 / mm³   |
| 100.000 – 308.000 / mm³          |

**BLOOD SMEAR EVALUATION:**

It was observed the presence of: Toxic neutrophils, hyper segmented neutrophils, activated monocytes and platelet aggregates.

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**BLOOD BIOCHEMISTRY**

|                       | REFERENCE RANGE |
|-----------------------|-----------------|
| Urea                  | 48.69 mg/dL     |
| 21.4 – 51.5 mg/dL     |
| Creatinine            | 1.12 mg/dL      |
| 0.4 – 2.2 mg/dL       |
| ALT                   | 9.97 U/L        |
| 3 – 23 U/L            |
| AST                   | 190.78 U/L      |
| 226 – 366 U/L         |
| ALP                   | 1107 U/L        |
| 86 – 295 U/L          |
| GGT                   | 26.1 U/L        |
| 6 – 32 U/L            |
| Glucose               | 35 mg/dL        |
| 62 – 134 mg/dL        |
| Total protein         | 3.96 g/dL       |
| 6 – 8 g/dL            |
| Albumin               | 2.51 g/dL       |
| 2.4 – 4.1 g/dL        |
| Globulins             | 1.45 g/dL       |
| 2.6 – 4 g/dL          |

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reflux. Blood gas and electrolytes analysis revealed low oxygen saturation with an increase in bicarbonate and decrease in anion gap (Table 2). Reflux stopped at the end of the day. In order to protect gastric mucosa and to help reduce abdominal distention, a solution containing aluminum hydroxide, magnesium hydroxide and simethicone was administered, and nasogastric tube was removed again. Initially, several attempts to feed the foal were made before complete food withdrawal due to worsening of abdominal distension. Nasogastric tube was placed once again, and all the nursed milk recovered. At this point, blood glucose dropped to 80mg/dL and remained on this level.

Abdominal radiography was performed on day 3, revealing a large intestine filled with feces and large amounts of gas, in addition to free fluid in the cavity (Figures 1 and 2). Peritoneal fluid sample was referred to the laboratory with a paired serum sample. Both peritoneal fluid and serum samples were with normal range for equine species (GRINDEM et al., 1990). Based on the results, uroperitoneum was excluded from the differential diagnosis and presumptive diagnosis was complete intestinal obstruction, probably due to meconium impaction. Due to the presence of nasogastric reflux and the possibility of meconium impaction with distention, contrast radiography was not performed nor orally neither via rectum. Also, due to worsening of the clinical status and financial restraints, no further diagnostic procedures were made. Although
bowel sounds and mucous membranes have shown improvement with medical treatment, the foal had not yet defecated until that moment. Exploratory abdominal surgery was also declined by the owners. Thus, after 60h of medical management, the foal was humanely euthanized.

Despite the fact that uroperitonium does not lead to obstruction, it was considered a possibility, once the patient showed strangury with ventroflexed posture and limbs stretched out. However, the foal urinated large volumes, while abdominal ultrasound did not show enough free fluid in the abdominal cavity and bladder was intact. Later, after laboratory analysis, free fluid in the abdomen seen in radiographs was not considered urine. Fecaliths were considered another possible cause of gastrointestinal obstruction without vascular impairment. Based on this suspicion, surgical intervention would have both

### Table 2 - Blood gas and electrolyte profile of a 48-hour-old foal diagnosed with atresia coli type III at the Veterinary Hospital of the Universidade Federal de Minas Gerais, Brazil.

| Parameter | Foal | Healthy foal |
|-----------|------|--------------|
| pH        | 7.412| 7.32 – 7.44  |
| pCO2      | 46.3 mmHg | 38 – 46 mmHg |
| pO2       | 61.3 mmHg | 63 – 77 mmHg |
| sO2       | 87.5 % | 92 – 96 %    |
| cK+       | 3.2 mM | 2.4 – 4.7 mM |
| cNa+      | 142 mM | 132 – 146 mM |
| cCl-      | 109 mM | 99 – 109 mM  |
| cHCO3-(P)c| 30.3 | 20 – 28      |
| cBase(B)c | 5.5 mM | 3.51 – 9.39 mM |
| AnionGap K+| 5.9 mM | 12.08 – 16.62 mM |
| cLac      | 1.6 | 1.1 – 2.3 |

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Although rare, \textit{atresia coli} was another probable cause of obstruction; yet the presence of nasogastric reflux did not allow oral administration of barium solutions to perform contrast radiography. It should be acknowledged that the use of contrast rectally could have confirmed diagnosis \textit{ante mortem}. However, the possibility of meconium impaction and further distention of the rectum was our main concern. Also, the worsening of clinical signs and financial restraint limited additional diagnostic procedures. \textit{Atresia coli} would have been diagnosed anyway should the owners have decided for exploratory laparotomy. Even though, medical treatment was still attempted until other probable causes were excluded or exploratory surgery authorized.

Complete blood count in this case was suggestive of sepsis and hypoproteinemia indicated that a failure of passive immunity might have occurred. Nevertheless, our foal did not manifest clinical signs consistent with sepsis. Abdominal ultrasound and radiographs were highly suggestive of a gastrointestinal obstruction, and we hypothesized this obstruction was the cause of both the inflammatory status seen in the laboratory analyses and hydroperitonium.

Since the patient showed no improvement with medical therapy and the owners declined surgery, euthanasia was recommended. Yellowish mucous membranes were attributed to the inability to feed the...
foal, since every attempt was followed by worsening of abdominal distention and need of nasogastric intubation. Hydroperitoneum was likely due to hypoproteinemias and abdominal inflammation. It is important to observe that this foal had two congenital abnormalities in the colon: type III atresia coli at the pelvic flexure and hypoplasia of the large colon caudal to the atresia. Considering this situation, the odds of a successful surgical intervention were minimal.

Although atresia coli is a rare condition in foals, it must be considered when dealing with a newborn foal with abdominal pain and distension. Clinicians should pay special attention when colic resembling meconium impaction responds poorly to medical management, especially if there is no brown staining after repeated enemas and no clinical signs that suggest strangulating intestinal lesions.

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DECLARATION OF CONFLICT OF INTERESTS

The authors declare no conflict of interest. The founding sponsors had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript, and in the decision to publish the results.

AUTHORS’ CONTRIBUTIONS

All authors contributed equally for the conception and writing of the manuscript. All authors critically revised the manuscript and approved of the final version.

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