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LETTER TO EDITOR

Gastric Rupture in A Horse (Bir Atta Mide Rupturu)

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Dear Editor,

Colic is one of the most common problems in equine practice. The cause of the equine colic is divided into two major categories (gastrointestinal and non-gastro-intestinal) [1]. The stomach of the horse may undergo pathological changes of its wall associated with gastric ulcer disease, rupture, chronic impaction or neoplasia [2,3]. Gastric ulceration rarely leads to perforation in adult horses [2]. The risk factors for gastric rupture include feeding grass hay; non-feeding grain [3,4]. Signs of gastric rupture are severe pain, increased heart rate, a distended abdomen and vomiting. Cyanosis and pale membranes may be present due to gastric compression reducing venous return. Although surgical repair of the gastric rupture is mentioned, if gastric rupture is diagnosed, the horse should be euthanized because stomach contents into the abdomen cause septic shock that cannot be adequately reversed with abdominal lavage and repair of the stomach defect [3,5,6]. Most of the gastric ruptures occur along the greater curvature of the stomach [3,5]. In some cases, gastric rupture occurs as a result of an infarction of the stomach wall in the absence of substantial distension or rarely in gastric ulceration leading to perforation [2,5]

Here, the case of gastric rupture due to gastric ulcer in a horse was presented. A 15-year-old, male, English thoroughbred horse was presented to Uludag University, Large Animal Clinics of Faculty of Veterinary Medicine with the signs of mild to moderate abdominal pain of 3 hours' duration. The owner informed minimal defecation and abnormal dietary management in the horse. At presentation, the horse was depressed, nasally regurgitating the gastric ingredient and sweating profusely in condition of standing immobile. Abdomen was enlarged and abdominal palpation was painful. Vital parameters of the horse included 92/min pulsation, 44/min shallow respiration, 4 seconds capillary refilling time and 37.3°C temperature. Mucous membrane color was hyperemic-cyanotic. Bowel motility was not determined in abdominal auscultation, and peripheral cooling was felt in the extremities palpation. Laboratory analysis results are listed in *Table 1*. Nasogastric catheter was indwelled to the stomach, and about 2 liters of content was retrieved from the stomach, which had 5.5 pH. Taking gradually worsen condition of the horse into consideration, medical management was initialed, urgently. Firstly, infusion of saline solution was started and flunixin meglumine (1.1 mg/kg, IV) (Finadyn®, Eczacıbaşı, Turkey), hyoscine-N-butyl bromide (0.14 mg/kg, IV) (Buscopan®, Eczacıbaşı, Turkey), ranitidine HCl (6.6 mg/kg, IV) (Ulcuran®, Abfar, Turkey) , 3 liter oral paraffin liquid (Sokol Likid®, Biofarma, Turkey) were administered, respectively. However, the horse was not stabilized with the medicals, and abdominal cavity was enlarged. At this time, nasogastric catheter was re-indwelled to the stomach but no stomach content was removed. Gastric rupture was diagnosed and emergency laparotomy was decided. But, the horse was suddenly died. At necropsy, abdominal cavity was containing 30 liters of a dark red to brownish, cloudy fluid contained straw and plant material. There was a perforation focus (4 x 4 cm in diameter) along the cutan mucosa of the greater curvature of the stomach *(Fig. 1)*. Hemorrhage and 2 ulcers (2.5 x 3 cm and 4 x 5 cm in diameters) were found in mucosal surface of glandular portion *(Fig. 2)*. Rupture on the greater curvature wall was 20 cm in length. Around the perforation focus in the stomach, there were edematous serosal walls, congestion, severe hemorrhage and moderate purulent inflammation.

It has been suggested with this case that gastric rupture is mortal digestive system pathology for horses and it might be related with the gastric ulcers. The veterinary practitioners should consider the gastric ulcers as causative factor for gastric rupture, and the recognizing signs should be evaluated during clinical examination and medical managements.

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Table 1. *Hematologic and serobiochemistry values of the present case*
Tablo 1. *Sunulan olgunun hematolojik ve serobiyokimyasal değerleri*

Parameters	Present Case	Reference Range
White blood cell (µL)	1.31 x10^3	5.4-14.3
Red blood cell (µL)	14.4 x10^9	6.8-12.9
Hemoglobin (g/dl)	22.7	11-19
Hematocrit (%)	68	32-53
Neutrophil (%)	31.4	32-91
Urea (mg/dl)	56	10-24
Creatinin (mg/dl)	2.67	0.9-1.9
Glucose (mg/dl)	93.3	75-115
Creatine kinase (IU/L)	309	2.4-23.4
Gamma-glutamyl transferase (IU/L)	21.8	4-44
Aspartate aminotransferase (IU/L)	650	226-336
Potassium (mEq/L)	>12	2.4-4.7

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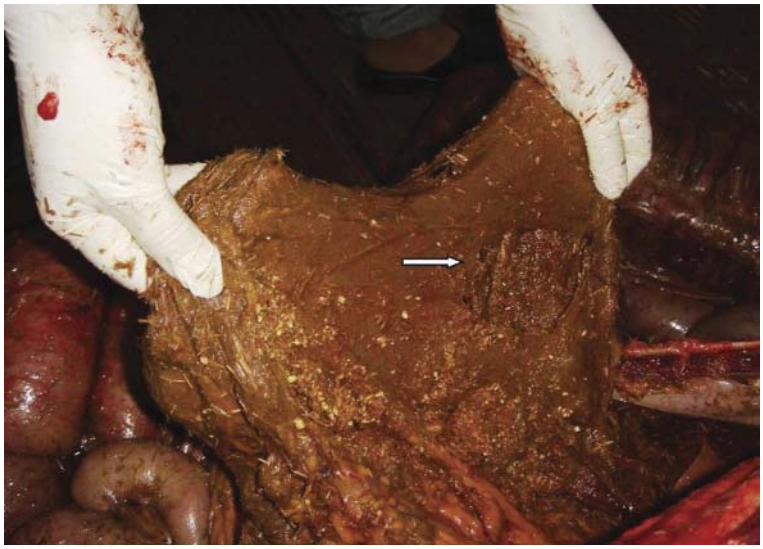

Fig 1. This view shows the perforation focus (4x4 cm in diameter) along the cutan mucosa of the stomach *(white arrow)*

Şekil 1. Bu görünüm midenin kutan mukozasındaki *(beyaz ok) perforasyon odağını (4x4 cm çapında) g*österir

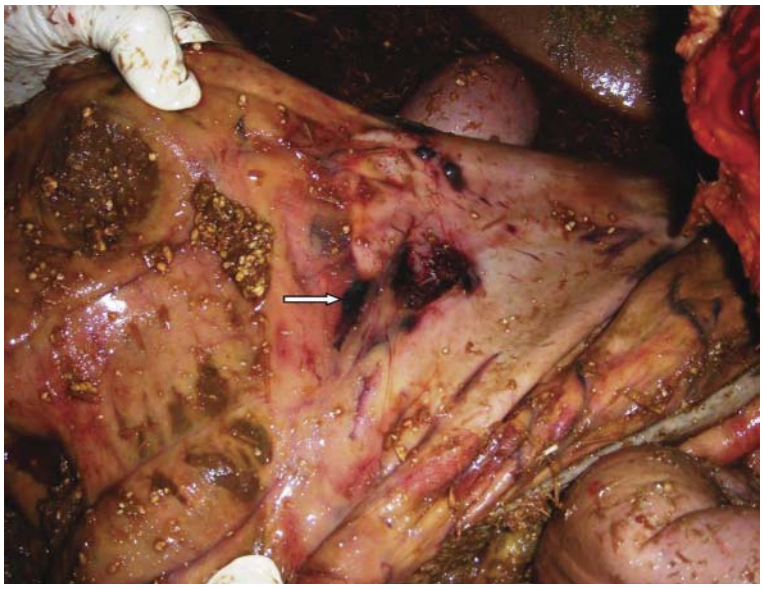

Fig 2. Hemorrhage and ulcers (2.5x3 cm and 4x5 cm in diameters) on mucosal surface of glandular portion of the stomach *(white arrow)*

Şekil 2. Midenin glandular kısmının mukozal yüzeyi üzerinde *(beyaz ok)* hemorraji ve ülserler (2.5x3 cm and 4x5 cm çapında)