# **Case Reports**

# Rare intestinal malformation (diverticulum confluens) in a horse

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Keywords: horse; malformation; enteric congenital pathology; equine jejunal pathology

# Introduction

Intestinal congenital diverticula are uncommon in domestic animals (Barker *et al.* 1993). They can arise from the omphalomesenteric (vitelline) duct or from each part of the intestine. Rarely, the vitelline duct may be retained in postnatal life as a patent duct extending from the antimesenteric side of the intestine to the umbilicus (*fistula omphalo-enterica completa*). In some cases, only the portion adjacent to the abdominal wall remains patent forming an umbilical pouch (*fistula omphaloenterica incompleta externa*). More commonly, only the portion immediately adjacent to the intestine remains patent (Meckel's diverticulum or *fistula omphalo-enterica incompleta interna*). The vitelline duct can be obliterated at both its ends and present one or more cysts along the corresponding tract.

In horses, only the fistula completa and Meckel's diverticulum have been described (Godlück 1967). Meckel's diverticulum is present as a short cone-shaped sac of up to 10 cm diameter, which may be attached by mesodiverticular folds to the ventral abdominal wall. Hahn (1877) described a large gastro-like Meckel's diverticulum with capacity of about 7 litres. Other congenital diverticula not arising from the vitelline duct occur rarely in horses as well as in other animal species (Godlück 1967). A classification of the latter is generically based on their shape: sac-like, double, loop, confluence and spindle diverticula. Few cases are described in the veterinary literature. Joest (1910) observed a double diverticulum in the small colon of a horse: from an intestinal loop, 2 sac-like symmetric and opposing dilatations emerged, each of them of a baby's head size, both with normal intestinal wall structure. Schmidt (1940) observed different types of intestinal dilatations in horses, one of them corresponding to a congenital rectal diverticulum. It derived from the ventral flexure of an abnormal colon-rectal loop and was directed (and closed) dorsocaudally.

Confluence diverticula are very rare intestinal dilatations which have been described only in swine. In a case observed by Joest (1905) the diverticulum was formed by 2 neighbour colon loops and 6 small intestinal loops. The case described by Aser (1918) occurred in a 10-week-old swine, where different small intestine loops merged in a fist-sized dilatation. In 2 further cases, Kitt (1923) found dilated, stomach-like sacs, resulting from the confluence of 3 small intestinal loops and consequently endowed with 6 openings.

The present study derives from the observation of a confluence diverticulum in a horse.

# Materials and methods

A male, 11-year-old, Trakhener dressage horse, whose history was known only for the last year, was necropsised at Istituto di Anatomia patologica veterinaria e Patologia aviare of Milan. The animal had shown recurrent colic incidents. During a severe colic episode, severe abdominal pain (unresponsive to analgesics), absence of normal intestinal borborygmi and peristaltic activity and extreme distension of small intestinal loops, at rectal examination, suggested the presence of a mechanical obstruction of small intestine and, therefore, laparotomy was performed. During laparotomy a complicated internal hernia was discovered, the horse was subjected to euthanasia and, after 2 days, submitted for a complete necropsy. Some samples were taken from the intestinal malformation which were formalin fixed and processed routinely for histology. Histological sections were stained with Haematoxilin-Eosin, Van Gieson, Mallory and Gomori's silver stain.

# Results

A sac-like dilatation of the jejunum, about 10 x 10 x 20 cm, 6 m from the pylorus was present. It was provided with a light sagittal groove apparently deriving from the fusion of 2 neighbouring jejunal loops (Fig 1). This dilatation delimited a wide jejunal ring about 1.5 m long, and its mesentery fold presented an opening which *in vivo* had engaged some other intestinal loops. The walls of the ring, as well as the ones of the afferent and efferent jejunal loop, appeared slightly thickened and contracted near the diverticulum. Opening the latter (Fig 2), almost all the jejunum and the opening of the dilatation showed abundant red-brownish fluid content consistent with catharral-haemorrhic exudate. Corresponding to the sagittal groove, a slightly elevated and regular ridge was observed (Fig 3). The intestine showed no evidence of damage due to parasites.

Histology of the involved portions of jejunum showed, as

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Fig 1: Diverticulum confluens (DIV) not opened: proximal or afferent jejunal segment (AF), jejunal ring (JR) caused by the diverticulum and distal or efferent jejunal segment (EF).



Fig 2: Diverticulum confluens: white dash lines correspond to the cuts performed to open the diverticulum.



Fig 3: Diverticulum confluens opened: afferent jejunal segment (AF), proximal segment of the jejunal ring (JRP), distal segment of the ring (JRD), efferent jejunal segment (EF). Note the severe redness of the mucosa together with the sagittal prominent line (arrowheads).



Fig 4a: Diverticulum confluens: section of sagittal ridge showing a regular structure (compared with 4b) of the intestinal layers with interruption of the muscular layer which includes isolated bundles of smooth muscular cells. Mucosal autolysis, due to postmortem artefacts, is also evident.

well as *postmortem* artefacts, congestion and haemorrhages in the mucosa and a loose infiltration of inflammatory cells (eosinophils and macrophages) in *lamina propria* and submucosa. The above features were also present in the jejunal loop, that was also characterised by severe submucosal oedema in its distal portion. The walls of the confluent diverticulum revealed a regular structure of the intestinal layers which showed an inflammatory reaction of the mucosa, *lamina propria* and submucosa, similar to that of the afferent and efferent jejunum. The sagittal ridge sections showed the same structure as above, with only a thin interruption of the muscular layer and without any evidence of connective proliferation or scarring. In different points of the ridge, the interposed connective tissue sometimes included isolated bundles of smooth muscular cells (Fig 4). The loose nature of connective tissue was confirmed by means of Van Gieson, Mallory and Gomori stains.

## Discussion

Pathological findings observed in this case corresponded to the description of confluent diverticula described in swine, even if the reports of Joest (1905), Aser (1918) and Kitt (1923), which were later discussed by Godlück (1967), presented more complex situations deriving from the confluence of at least 3 loops of the small intestine. In our case, the fusion of 2 jejunal tracts creates a complete patent jejunal ring and the formation of a wide diverticulum with 4 gaps. The existence of a thin sagittal raphe shows how the diverticulum has originated from the fusion of the 2 neighbouring intestinal loops. The congenital origin of this diverticulum had been further proved by the following observations:

- a) The diverticulum had a slight sagittal ridge and a single large lumen with no fistulous connections, very similar to the one described by Kitt (1923) in swine.
- b) Histologically, there were no significant differences in the structure and thickness of both the diverticulum and the intestinal walls.
- c) The muscular layers, derived from the confluent intestinal loops, showed a thin linear interruption along the sagittal raphe, replaced by loose connective tissue, sometimes including isolated bundles of smooth muscular cells.
- d) The mucosa of the confluent loops showed no structural gap.

In the present case, the presence of the diverticulum allowed an apparently regular growth and activity (despite the fact that food had to pass through a complicated intestinal course). The only exceptions were episodic colic accidents, at least during the animals last months. However, these observations do not contradict Godlück's who claimed that swine affected by *confluence diverticula* had no evident functional disturbances. Our opinion is that the malformation could be the cause of recurrent mild colics, because of both abnormal bacterial proliferation, due to irregular food intestinal course and/or partial intestinal obstruction due to the passage of some intestine loops through the tear of jejunal ring mesentery, usually with spontaneous remission.

According to Kitt (1923) and Godlück (1967), acquired intestinal confluence may sometimes occur due to foreign bodies which pierce neighbour intestinal walls determining adherence at first and communication subsequently, but the result, in these cases, is an *enteroanastomosis fistulosa*.

Other possible origins of these intestinal anomalies may be traced back to fetal intestinal dislocations (Godlück 1967) or wrong embryonic growth either during the embryonic formation of the intestinal loops or during the differentiation of the small and large intestine.

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Received for publication: 21.6.99 Accepted: 20.10.99