Caecal infarction in a Holstein Friesian calf

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SUMMARY

The present report concerns a case of caecal infarction we observed in a two-week-old female Holstein Friesian calf, which developed diarrhoea with a fatal outcome, despite antibiotic and fluid therapy. At necropsy, multiple and large foci of ischaemic necrosis were observed along the anti-mesenteric side of the caecum. Microscopically, coagulative necrosis of the caecal wall was seen, a thick band of neutrophils infiltrating the submucosa and the inner smooth muscle layer. Pathological findings allowed the final diagnosis of caecal infarction, which is rarely diagnosed in neonatal/young calves (<30 days), its aetiopathogenesis being currently unknown.

KEY WORDS

Caecal infarction; calf; diarrhoea; pathology.

INTRODUCTION

Enteritis is among the major causes of economic loss in livestock production worldwide, and its management is a relevant part of routine veterinary practice (1,2). Calf diarrhoea ("scours») is the most important clinical sign of enteric disease and represents a serious health issue in calves less than 2 weeks old, often resulting in death. It has been estimated that >50% of weaning calf mortality can be attributed to diarrhoea, both in USA (3) and European countries (4).

The aetiology of calf scours is multifactorial (1). Infectious and non-infectious agents usually act together, thus the diagnosis of calf scours is challenging and often requires suitable laboratory tests (5). Depending on the causative agent, lesions may involve several gastrointestinal tracts with different gross and microscopic features. Pathological findings are also valuable in making the correct diagnosis, which is a prerequisite for proper management of any herd health issue (6).

The present case report aims to describe pathological features of caecal infarction, a rather uncommon disease condition observed in a calf.

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CASE DESCRIPTION

In November 2022, a two-week-old female Holstein Friesian calf died spontaneously and was referred to the Veterinary Teaching Hospital of Teramo for both diagnostic and teaching purposes. The calf belonged to a medium-sized dairy farm. Cows were vaccinated against viral respiratory syndromes (Cattle Master 4, Zoetis, 2mL i.m.) twice a year, starting at 6 months of age. In addition, calves aging 20-25 days were administered toltrazuril (Baycox, Bayer, 3 mL/10kg bodyweight *per os*) for the prevention and treatment of coccidiosis.

The calf under study appeared unhealthy since birth, and during the last few days developed yellowish and creamy diarrhoea with a fatal outcome, despite therapy with aminosidine (Aminofarma L 20%, Ceva Vetem, 2.5 mL/10 kg *per os* for 3 days) and fluid *per os*.

At necropsy, the body condition was poor, and the perineum was smeared with yellowish faeces. Pathological findings were mainly observed in the abdominal cavity, wherein fibrin deposits were seen upon the caecum. On closer inspection, multiple and large foci of necrosis affected the caecum, always along the anti-mesenteric side. These foci appeared pale, sharply demarcated, and surrounded by a hyperaemic halo (Figure 1). On section, the underlying mucosal surface was also discoloured, while the surrounding mucosa appeared hyperaemic and oedematous (Figure 2). The mucosa of the abomasum and small intestine were also highly hyperaemic, while mesenteric lymph nodes appeared enlarged and oedematous. Finally, small areas of atelectasis were observed in the cranio-ventral part of both

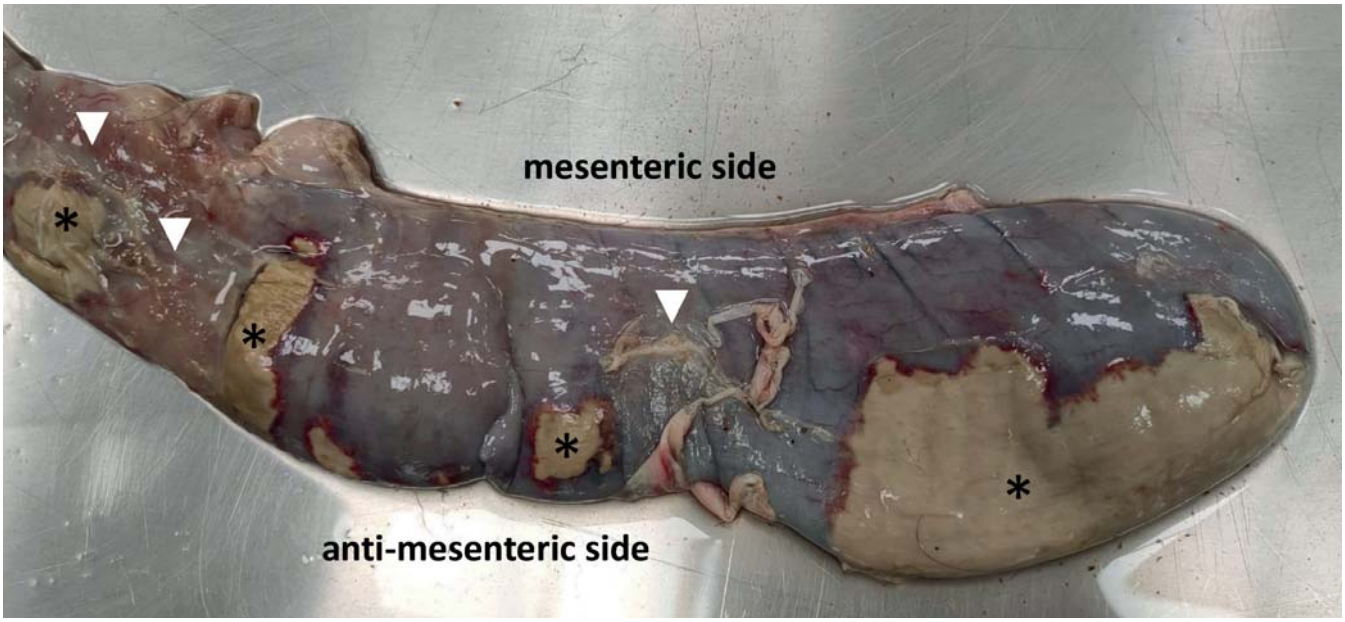

Figure 1 - Calf. Serosal surface of the caecum. Multiple pale, whitish-to-yellowish foci of necrosis (asterisks) are present along the anti-mesenteric side. Such foci have sharp, jagged edges, with hyperemic haloes. Fibrinous peritonitis is also evident (white arrowheads).

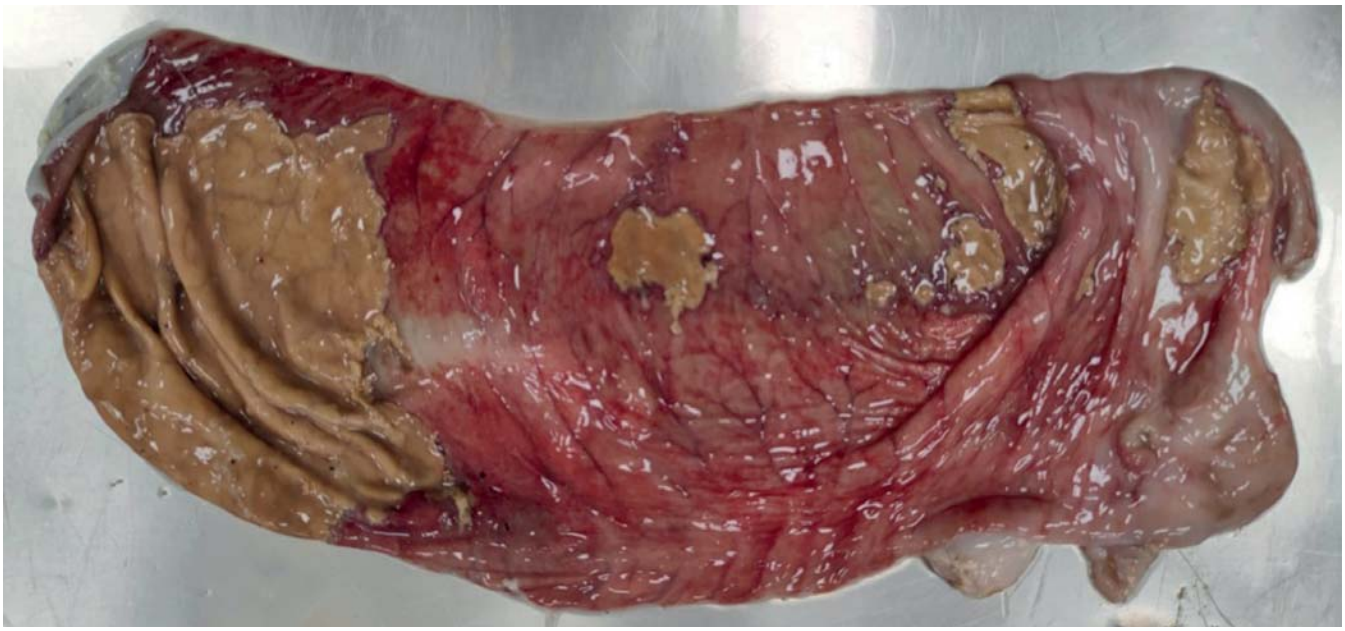

Figure 2 - Calf. Mucosal surface of the caecum. Necrotic areas are also evident on cut section, after opening the caecal lumen. The surrounding mucosa appears hyperaemic.

lungs. As necropsy was carried 72 hours after calf death, bacteriological investigations were not performed.

Caecal tissue samples were fixed in 10% neutral buffered formalin, embedded in paraffin and routinely processed for histopathological examination. Microscopically, postmortem changes were prominent. However, two microscopic features were clearly visible: a) coagulative necrosis of the caecal wall, more pronounced at the level of the outer smooth muscle layer; b) a thick band of neutrophils, mainly involving the submucosa and the inner smooth muscle layer (Figure 3). Caecal necrosis and inflammatory reaction were sharply demarcated, whereas vascular dilation was observed only in the surrounding intestinal wall, which appeared otherwise normal. Overall, pathological findings largely overlapped with those described by Adaska *et al.* (7), allowing the final diagnosis of caecal infarction.

DISCUSSION

Infarction is defined as "*an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage in a particular tissue*" (8). Intestinal infarction is common in companion animals, mostly in horses, and often results from intestinal displacement (strangulation, intussusception, volvulus) or thromboembolism (e.g., parasitic endarteritis). The small

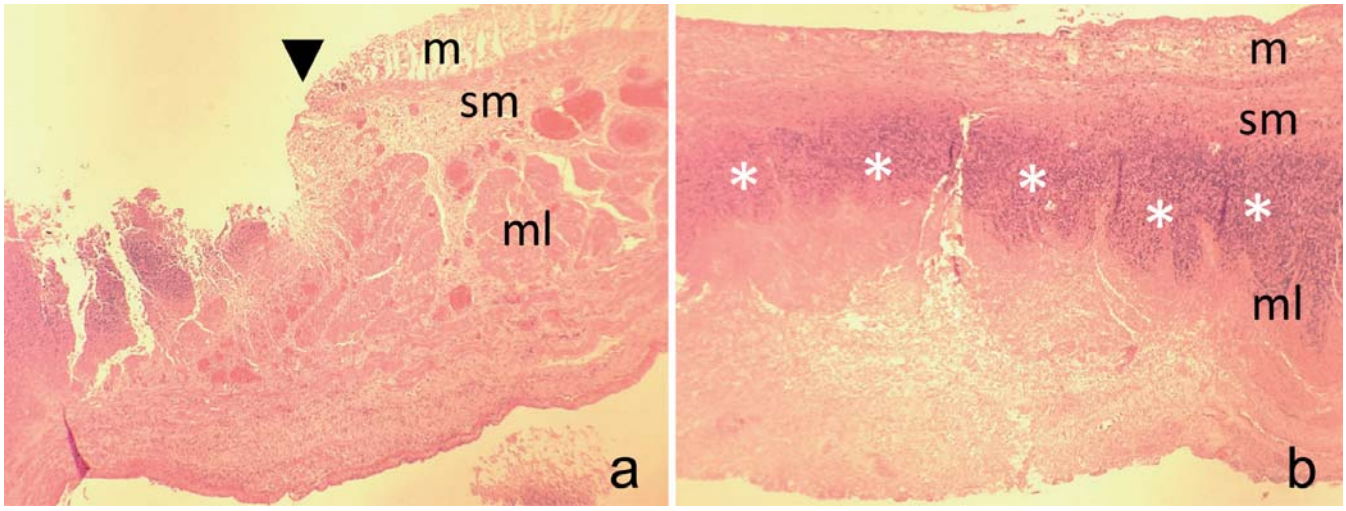

Figure 3 - Caecal infarction. Picture (a) shows the sharp border (black arrowhead) of the lesion. On the right side, the morphology of the caecal wall appears well preserved, despite postmortem changes. On the contrary, such morphology is severely disrupted at the caecal infarction (a, left side). Within the infarct area (b), a thick band of neutrophils is seen (white asterisks), mainly infiltrating the superficial muscular layer. Hematoxylin and eosin stain, final magnification: x200.
Legend: m = mucosa; sm = submucosa; ml = muscular layer.

intestine usually shows prominent lesions after 30 minutes of ischaemia, whereas the colon appears less sensitive. The evolution and outcome of bowel infarction can be very variable. Often, ischaemic necrosis initially affects the entire thickness of the mucosa and triggers a dense and acute inflammatory response involving the submucosal layer. Under favourable conditions, focal lesions may heal by proliferation of granulation tissue and epithelial migration from the surrounding mucosa. Otherwise, prolonged ischaemia may result in necrosis of the entire intestinal wall, which usually leads to fatal septic peritonitis (9).

In humans, infarction usually affects the left colon and shows a segmental pattern, due to peculiar blood supply at the splenic flexure and rectosigmoid junction. On the other hand, caecal infarction is a rare and less well-known entity that affects the elderly and debilitated and is likely to result from a state of low blood flow (e.g., heart failure, cardiac surgery and haemodialysis) (10,11).

Similarly, caecal infarction is rarely diagnosed in calves, a few papers being currently available in the literature. Worthy of note, Adaska *et al.* (7) retrospectively reported 34 cases, the main features of which can be summarised as follows: a) in contrast to humans, all cases were observed in neonatal/young calves aged less than 30 days; b) no correlation was demonstrated among caecal infarction, specific pathogens and/or concurrent disease conditions; c) coagulative necrosis affected almost exclusively the anti-mesenteric side of the caecal wall, involving the mucosa and extending to varying degrees into deeper layers; d) infiltration of degenerate neutrophils was a common finding, while fibrinoid necrosis and thrombosis were occasionally seen. More recently, a single case has been reported in Scotland, which occurred during an outbreak of calf diarrhoea poorly responsive to treatment. In this case, coronavirus infection and clostridial typhlocolitis were also detected (12).

The case described herein largely overlaps with those previously reported (7) and, in our opinion, raises some basic questions. The first concerns the real prevalence of caecal infarction in calves, which may be greatly underestimated. In this respect, the present case report sounds emblematic, as it resulted from an intensive, somewhat unusual necropsy activity for teaching purposes. The second concerns the aetiology and pathogenesis of caecal infarction, which are still largely unknown., From a mere speculative point of view and considering the clinical history of this calf under study a hypovolemic vascular state could lead to intestinal hypoperfusion and necrosis, as suggested in humans (11). Likewise, anatomic features of caecal blood supply might predispose individual calves to infarction (7). Reasonably, in the present case necrosis first affected the caecal mucosa, triggering a dense inflammatory reaction in the submucosa indicates, and later extended to deeper layers.

CONCLUSIONS

The present case report shows the typical features of caecal infarction in calves and could be very useful for veterinarians, who face the diagnostic query of calf scours during their daily practice. Hopefully, our report should stimulate more attention about that disease condition, which is likely overlooked and underestimated, even though easily detectable at necropsy. In this respect, we feel that necropsy is still crucial to estimate the real prevalence of livestock diseases, as well as to evaluate the efficacy of preventive/therapeutic measures implemented.

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