Journal of Equine Veterinary Science 149 (2025) 105422

Contents lists available at ScienceDirect

Journal of Equine Veterinary Science

journal homepage: www.elsevier.com/locate/jevs

Case Report

It is not always what it seems: allantochorion cervical pole necrosis

L. Fischetti [a], F. Perina [a,*], G. Sarli [a], N. Ellero [a], F. Freccero [a], C. Castagnetti [a,b], J. Mariella [a]

[a] Department of Veterinary Medical Sciences (DIMEVET), University of Bologna, Via Tolara di Sopra 50, Ozzano dell'Emilia, Bologna 40064, Italy
[b] Health Sciences and Technologies Interdepartmental Center for Industrial Research (CIRI-SDV), Bologna University, 40100, Italy

ARTICLE INFO

Keywords:
Abortion
Cervical pole necrosis
Placentitis
Pregnant mare
Umbilical cord torsion

ABSTRACT

A 286-day pregnant mare was referred for signs of impending abortion. The ultrasonographic evaluation revealed a high normal combined thickness of the utero-placental unit (CTUP), oligohydroallantois and a living fetus. No vulvar discharge was observed, and the cervical swab culture was negative. After a choke, the mare received NSAIDs and antibiotics and then developed systemic inflammatory response syndrome (SIRS) and aborted. The amniotic portion of the umbilical cord was excessively long and twisted and the chorionic side of the placenta showed a brown discoloration and rough surface area. *E. coli* was isolated from all the fetal membranes, fetal fluids and tissues. Histologically, degeneration and necrosis with dystrophic calcification of the chorial villi at the cervical pole was found. The final diagnosis was abortion due to placental infarction with secondary placentitis. Cervical pole necrosis is a poorly understood condition of the allantochorion, and a histological exam is necessary to obtain a definitive diagnosis.

1. Introduction

Pregnancy loss in mares may occur at any stage of gestation from fertilization to parturition and is a major problem due to its economic and emotional losses [1–3]. Causes of abortion, stillbirth and neonatal death in horses may vary over time and according to the geographical region [4–7]; non-infectious and infectious causes are involved [3].

Non-infectious abortions have been described as accounting for 10.5 %–72 % of the total number of abortions and may occur at any stage of pregnancy in horses; causes include abnormalities of gestation and fetal membranes, i.e. umbilical cord (UC) and placenta, as well as maternal and fetal factors [8]. The pathological conditions of the UC represent the major cause of non-infectious abortion in the mare. In most cases, the cord is too long and twists to such an extent that UC flow of blood and urine is impeded or even completely obstructed, resulting in fetal death. This anomaly is known as UC torsion (UCT) and is responsible for more than half of all non-infectious abortions [9–13]. Cervical pole necrosis, also known as placental necrosis or placental infarction, is a poorly understood disease of the allantochorion and an infrequent cause of non-infectious abortion in horses. Currently, the pathogenesis of this unique lesion is not definitively known and only theories have been proposed to explain the presence of necrosis [14].

Placentitis is a common cause of abortion, stillbirth and neonatal death in horses [1,7,9,10,15,16]. Four morphologic types of placentitis have been described according to the type of lesions and potential pathogenesis: ascending, focal mucoid (nocardioform), diffuse (hematogenous), and multifocal [17]. Ascending placentitis, which is caused by bacteria or fungi ascending through the cervical canal and infecting the placenta, is the most prevalent form of placentitis [18–21]. This condition is clinically characterized by premature udder development, increased uteroplacental thickness at the level of the cervical pole and mucopurulent vaginal discharge. If placentitis is untreated or not early treated, placental function is compromised, placental separation ensues and pathogens can spread to the fetus, resulting in fetal death and expulsion or the birth of a premature and/or septic compromised foal [22]. In chronic placentitis, the fetus may show intrauterine growth retardation. In this report, abortion in a 15-year-old Warmblood mare at 293-day gestation due to UCT, and cervical pole necrosis with secondary placentitis is described.

2. Case presentation

2.1. Ethical Statement

The owner provided informed consent, as part of the hospital consent form, for the use of the horse's data in the study.

* Corresponding author.
E-mail address: francesca.perina2@unibo.it (F. Perina).

https://doi.org/10.1016/j.jevs.2025.105422
Received 4 November 2024; Received in revised form 25 February 2025; Accepted 17 March 2025
Available online 18 March 2025

2.2. Presenting complaint and history

In March 2023, a 15-year- old multiparous Warmblood mare at 286 days of pregnancy was admitted to the Equine Perinatology and Reproduction Unit (University of Bologna, Italy) for signs of impending abortion and abdominal discomfort. The owners reported that she had four previous normal pregnancies without complications. The mare had been housed with other horses, daily fed with hay and concentrates but was not regularly vaccinated nor dewormed. At the breeding soundness examination prior to artificial insemination, the uterine swab cultured positive for *E. coli*. Endometrial cytology was not performed. The mare did not receive any treatment. Due to a poor perineal conformation, a Caslick vulvoplasty has been performed.

2.3. Clinical findings and follow up

On presentation, the mare was in moderate body condition (BCS 5/9), with mild colic signs (constantly kicking and watching the flank), moderate tachypnea (36 breaths/min), normal body temperature (37.7°C), and normal heart rate (40 bpm). She showed laxity of pelvic ligaments, premature udder development, premature lactation and ventral edema. No vaginal discharge was observed, and a poor perineal conformation was confirmed. Since there were signs of impending parturition, the Caslick vulvoplasty was promptly opened. Vaginal examination with a speculum revealed no obvious abnormalities. A cervical swab was taken for bacteriological examination: microorganisms were not cultured. Complete blood cell count (CBC) and blood chemistry parameters were within normal limits (ADVIA 2120, Siemens Healthcare srl, Milan, Italy; Chemistry Analyzer AU400; Olympus Diagnostica GmbH, Lismeehan, Ireland) and serum progesterone (P4) was 5.64 ng/mL (Immulite 2000, Siemens) which was physiological for this gestational age (normal range 3–6 ng/mL at 110-300 days of pregnancy) [13]. Transrectal ultrasound (US) showed a high normal combined thickness of the utero-placental unit (CTUP) of 10.3 mm [23,24]. The fetus was in anterior presentation, and fetal fluids had a normal echogenic appearance. Transabdominal US showed a physiological fetal heart rate at rest of 83 bpm (82–98 bpm at 10 months of pregnancy) [24,25] and moderate fetal activity. The fetal aortic diameter was 18.7 mm which was lower than the normal values (22.25 ± 1.5 at 10 months of pregnancy) [24]. The CTUP in the mid-caudal and right-caudal areas measured 5.4 and 6.1 mm respectively and fluid's depth was subjectively reduced. No placental detachment was observed. One day after, at 288 days of gestation, US examination confirmed the reduced volume of fetal fluids except for the mid-cranial area of the abdomen. The fetal hearth rate remained within normal limits. At 289 days of pregnancy, serum P4 increased to 6.86 ng/mL.

2.4. Treatment and outcome

At 290 days of pregnancy, the mare accidentally injured the left masseter muscle that became edematous and painful by the night and dysphagia with a concurrent transitory episode of esophageal obstruction occurred the following day. On US of the thorax, multiple confluent B-lines associated with initial focal consolidation of lung parenchyma in the left cranio-ventral quadrant were visible. Parameters of CBC were within normal limits except for a slight neutrophilia (9.3×10^6/μL; normal ranges 2.2–8.5 × 10^6/μL) and serum amyloid A (SAA) level increased exponentially to 311 μg/dL (reference range 0–10 μg/dL); therefore, flunixin meglumine (1.1 mg/kg IV BID), broad-spectrum antibiotic treatment with procaine benzylpenicillin and dihydrostreptomycin (15000 UI/kg IM SID) and gastric protection with sucralfate (20 mg/kg PO TID) were started associated with a soft food diet. P4 rapidly peaked at 11.70 ng/mL.

At 292 days of gestation, the mare was depressed with poor appetite, hyperthermia (39.1 °C), tachycardia (56 bpm) and mild tachypnea (24 bpm), and she developed profuse, watery and malodorous diarrhea. Total CBC revealed an increased PCV (40 %), severe leukopenia (2.4×10^3/μL WBC; normal ranges 5.5–12.5 × 10^3/μL) and further increase of SAA (316 μg/mL). Microbiological fecal exam and blood culture tested negative. On transabdominal US, the fetus had a normal heart rate of 90-100 bpm at rest, and subsequently fetal viability was monitored every 4 to 6 h. Considering the clinical signs and collateral exams, the mare was developing systemic inflammatory response syndrome (SIRS) associated with multiple organ dysfunction syndrome (MODS) [26,27]; therefore flunixin meglumine and sucralfate treatments were continued, maintenance fluid therapy (2 mL/kg/h) with 1 % glucose combined with constant rate infusion of lidocaine (0.05 mg/kg/h), paracetamol (20 mg/kg PO BID) and pentoxifylline (10 mg/kg PO BID) were added and procaine benzylpenicillin and dihydrostreptomycin was replaced by ampicillin (20 mg/kg IV TID) and gentamicin (6.6 mg/kg IV SID). During the night, the first signs of laminitis developed, hence cryotherapy was started.

At 293 days of gestation, there had been no clinical improvement, and the US demonstrated the death of the fetus. Transvaginal palpation was performed, and it was possible to feel the head of the fetus due to a dilated cervix, although no evident uterine contractions were found. Dystocia due to fetal malposition developed: the fetus was in anterior presentation, dorso-pubic position, with lateral head deviation and bilateral shoulder flexion. The fetus was extracted via obstetric manipulations *per vaginam*2 h after it was found dead. The fetus was a 30 kg male, in good body condition and moderately autolyzed. The UC was macroscopically abnormal, 145 cm long (125 cm the intra-amniotic portion with 9 coils, 20 cm the intra-allantoic portion with 2 coils), excessively twisted, edematous, congested, inserted between the two horns (Fig. 1a) and the urachus showed segmental fluid-filled dilatations in the amniotic portion and petechiae in its inner walls (Fig. 1b). Placenta passed 2 h after abortion: the allantochorion weighed 5 kg (16 % fetal body weight) with a type III pattern of vascularization and the gravid horn was the right one. Macroscopically, on the chorionic side there was a brownish lesion with firmly adherent calcified materials bordered by congestion, distended vessels and diffuse edema originating from the cervical pole and ascending to the base of the non-gravid horn (Fig. 1c); near the cervical star necrotic areas with transmural perforations were present (Fig. 1d) and on the allantoic side diffuse congestion. The amnion had focal necrotic areas and distended vessels (Fig. 1e). Portions of 1 × 1 cm were collected from fetal membranes (pregnant horn, non-pregnant horn, body, cervical pole and amnion) for microbiological and histopathological examination. The gastric contents of the fetus were collected and submitted for bacteriological analysis. The fetus was sent to the Experimental Zooprophylactic Institute of Lombardia and Emilia Romagna for necropsy and microbiological examination. After stage III parturition, the mare became brighter and alert, the appetite improved, and diarrhea declined. On the same day, the lidocaine infusion was discontinued.

The next day, the mare showed mild hyperthermia (38.7 °C) and stiff gait, she was still leukopenic (3.9×10^3/μL WBC) and SAA was still elevated (249 μg/mL). A transrectal US revealed > 6 cm hypoechoic fluid in the uterine body and right horn; therefore, a uterine lavage was performed with 25 L of sterile isotonic NaCl solution: the recovered fluid was hemorrhagic with abundant fibrinous debris and a 50 mL sample was collected for bacteriological culturing; 10 IU oxytocin IV was administered at the end of the procedure. Together, these signs were indicative of septic puerperal metritis [28–30]. Therapies remained unchanged, and cryotherapy was continued. Uterine lavages were performed every other day until no more fluid was visible on US. Since the submitted samples of fetal membranes, fetal gastric contents and uterine fluid resulted positive for *E. coli* on culture (VELABAC) a Kirby-Bauer susceptibility test was performed, and gentamicin was replaced by amikacin (10 mg/kg IV SID).

Five days after abortion, diarrhea and leukopenia resolved (7.4×10^3/μL), the consolidation was no longer detectable on US of the thorax, but SAA was unchanged. Vaginal examination with a speculum revealed

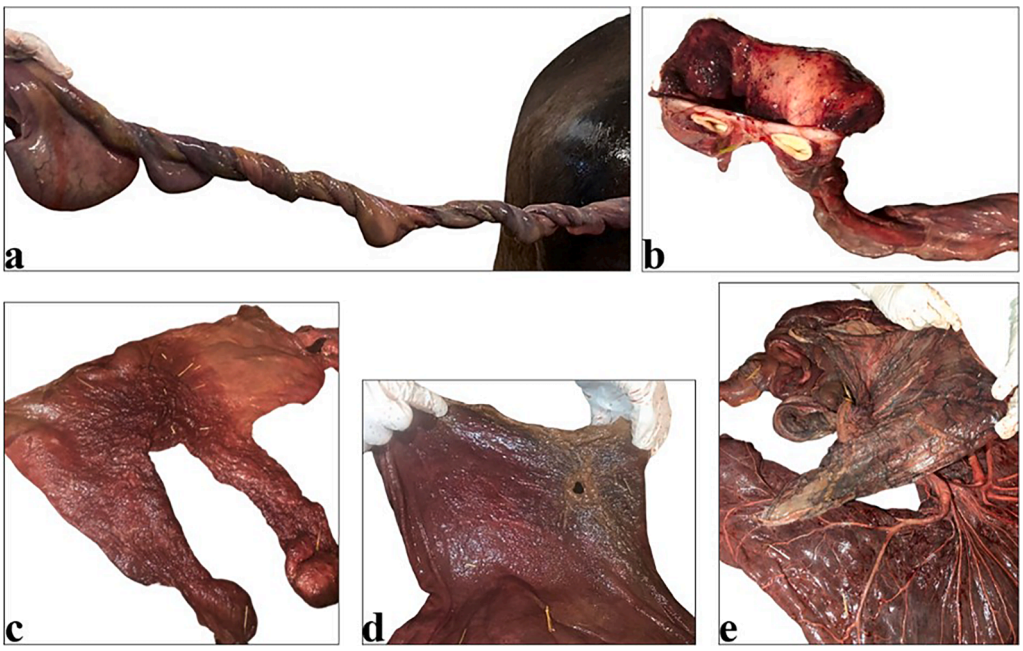

Fig. 1. Umbilical cord (a): 145 cm long (125 cm the intramniotic portion with 9 spires, 20 cm the intrallantoic part with 2 spires), excessively twisted, segmentally edematous, congested and implanted between the two horns. **Urachus (b):** The urachus showed segmental fluid-filled dilatations in the amniotic portion and in its inner surface petechiae were visible. **Allantochorion (c):** Brownish lesion with firmly adherent calcified materials originating from the cervical pole and ascending up to the base of the non-gravid horn. It was surrounded by congestion and diffuse edema. **Cervical star (d):** In proximity of the cervical star, necrotic and devitalized areas were present characterized by fenestrations of the placenta. **Amnion (e):** Diffusely congested, with focal necrotic areas and distended blood vessels.

no obvious abnormalities but on US, an inhomogeneous 3 cm long area with a hyperechoic center was visualized ventral to the cervix suggestive of a uterine wall abscess or hematoma. A low volume uterine lavage was performed, and a 50 mL uterine fluid sample was sent for microbiological examination. Eleven days after abortion, uterine involution was proceeding normally [31] and US demonstrated a 30 mm follicle on the left ovary which ovulated four days later.

Thirteen days after abortion, all therapies were stopped except for antibiotics as the culture of the low volume lavage was still positive for *E. coli* and also cultured positive for *Staphylococcus aureus*. Based on antibiogram results, ampicillin and amikacin were substituted with sulfadiazine/trimethoprim (30 mg/kg PO BID) that was continued for

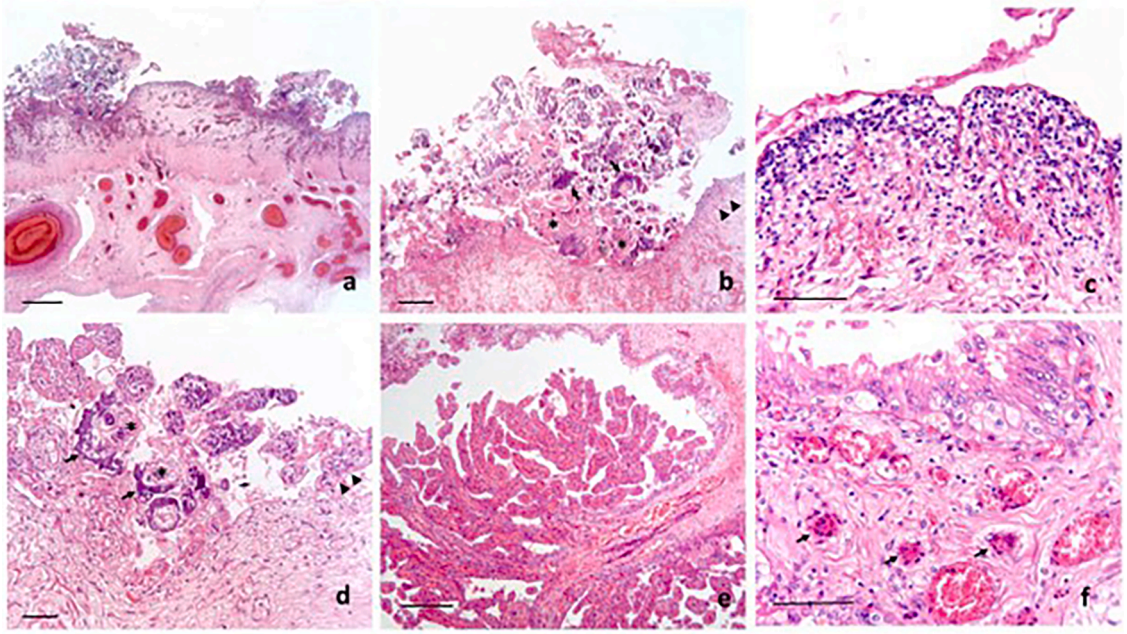

Fig. 2. Placenta at the level of the cervical star (a), (b) and (c). Diffuse congestion of the chorion **(a)** showing necrosis (asterisk) and calcification (arrow) of the chorionic villi and scant inflammatory infiltrate (arrowheads) **(b)**; degenerated neutrophils in the superficial layer of the chorion **(c). Placenta at the level of the uterine body (d)** atrophy and necrosis (asterisk) of chorionic villi and trophoblasts associated with dystrophic calcification (arrow) and mild inflammation (arrowheads). **Placenta at the level of the gravid horn (e), (f).** Placenta of the gravid horn shows normotrophic villi **(e)** but marked dilation and hyperemia of blood vessels and focal microthrombosis (arrow) **(f)**. Scale bar (μm): (a) 800; (b) 500; (c) 200; (d) 100; (e) 300; (f) 200.

10 days; on US, the uterine wall lesion was still present and unchanged. The following day, SAA normalized, and the patient was discharged. Based on the referring veterinarian examination, the mare fully recovered.

2.5. Gross and histopathological diagnosis

At postmortem exam, fetal septicemia was demonstrated by isolation of *E. coli* from the fetal spleen and liver accompanied by subcutaneous icterus, subendocardial petechiae and hepatic necrosis. A real time PCR abortion panel (EHV, EAV, Leptospira; Experimental Zooprophylactic Institute of Lombardia and Emilia Romagna) performed on fetal membranes and tissues was negative. Histological examination of the cervical pole revealed necrosis and calcification of the villi, with sparse degenerated neutrophils in the superficial layer of the chorion (Fig. 2a–c). At the level of the body of the allantochorion severe hyperemia of chorionic vessels and edema were present; moreover, atrophy and necrosis of chorionic villi and trophoblasts associated with dystrophic calcifications and minimal cellular inflammation were the predominant findings (Fig. 2d). Normal villi were scattered but the overlying epithelium and trophoblasts were atrophic and vacuolised. The allantochorion villi of the non-gravid horn were necrotic and focal calcifications of vessel walls and trophoblasts associated with scant neutrophils were further findings. The allantochorion at the level of the gravid horn showed normotrophic and normo-plastic chorionic villi (Fig. 2e) but, in the chorion, marked congestion of blood vessels, focal thrombosis in small vessels (Fig. 2f) and rare perivascular neutrophils were also evident.

3. Discussion

At admission, the mare presented in this report showed typical signs of a high-risk pregnancy and impending abortion, i.e. colic symptoms and premature udder development/lactation [32]; additionally, she had poor perineal conformation and a history of a positive uterine swab before breeding which are predisposing factors for an ascending infection of the reproductive tract due to fecal contamination and/or air aspiration in the vagina and cervix [33,34]. However, no vulvar discharge was present and the cervical swab at admission cultured negative for microorganisms. The transrectal US showed reduced fluids depth and a high normal CTUP [23,24]: decreased allantoic fluid's depth is an indication of fetal distress and is associated with a negative outcome in human beings and horses [35]. In women, it usually develops due to decreased fetal urine production and is a good indicator of fetal chronic hypoxia [36], associated with intrauterine growth retardation and a poor perinatal outcome [37]. In mares, reduced amounts of allantoic fluid have been associated with a poor fetal outcome [35]. Collectively, none of the findings recorded at admission were indicative of an ascending placentitis but the mare was, despite that, closely monitored to detect any potential changes.

Due to the suspected aspiration pneumonia following the mandibular injury and a choking episode, the mare was administered procaine benzylpenicillin. Subsequently, she developed SIRS associated with MODS [26,27,38]; in addition, P4 peaked, indicating fetal stress due to progestogens synthesis from the fetal adrenal gland [24,39,40–42] and on US no more fetal heart activity was detectable. Experimental studies on neonatal leukocytes indicate that killing *E. coli* bacteria with b-lactam antimicrobial agents leads to a greater release of endotoxin and increased cellular synthesis of tumor necrosis factor compared to amikacin alone or the combination of amikacin and ampicillin [43]. In our case, it could be hypothesized that the stress of the choke induced cortisol and prostaglandins release stimulating parturition or that the administration of the antimicrobial drug may have caused a massive release of endotoxins from the fetoplacental unit and/or from the maternal gastro-intestinal tract leading to abortion, or a combination of both. Moreover, the mare had signs of impending delivery at presentation and the timing of abortion may simply reflect progression of the underlying pathology.

UC length (UCL) in normal gestation can be quite variable and twisting is a physiological phenomenon that affects all its structures, sometimes with pathological implications [44,45]. Coiling is considered pathologic when it is excessive, causing vascular obstruction, edema, hemorrhage, thrombosis of umbilical vessels and/or obstruction of the urine flow [45–48]. UCL is affected by mare parity, age [49,50], fetal sex [49,51] and to date, the only known risk factor associated with UCT in equine fetus is an UCL $>$ 80 cm [45,52–58]. The development of an excessive UCL could be potentially influenced by the site of UC insertion and the type of vascular pattern of the allantoic surface [59] with approximately half of the UCT cases associated with type II or III pattern [56]. More recently, some authors suggested that genetics may explain this condition, but more research is needed to confirm this [60,61]. Another possible explanation may be the association with the amount of fetal movement [62] with the excessive UCL that develops secondary as a response to stretching of the UC because of tension arising from the twisting of the UC [61,63]. The fetal-amniotic unit is highly mobile within the first five months of gestation, hereafter its mobility decreases and ceases by approximately the 7th month [64], mainly because of decreased space within the uterine lumen as the fetus grows [44,64]. Increased length of the amniotic part of the UC has been associated with UCT, and the risk of abortion due to UCT increased by 7.3-fold when the amniotic: allantoic UCL ratio was >1 [58]. These findings combined with the high fetal mobility up to 7th month of gestation and the exponential growth of the amniotic UC in the same time period support the hypothesis that may explain the high prevalence of abortion associated with UCT in the 6th to 8th month of gestation and that UCT is more likely to occur in the amniotic UC part rather than the allantoic one [13,45,52,58,63]. Altogether, these data correspond with those of this study: in fact, the total UCL was longer than normal (145 cm) with a more twisted amniotic portion (125 cm long, 9 coils) compared to the allantoic one (20 cm long, 2 coils), and a vascular type III pattern. However, not all mares with a long UC will abort due to UCT and they may even deliver a clinically normal foal at term [6]. In our case, UCT was macroscopically characterized by pathological twisting with tension and compressive forces on the affected portion of the cord, blanched constricted areas, edema, hemorrhage, aneurysms, tearing of the intima of vessels, thrombosis, and urachal dilatations of varying sizes forming urine-filled sacculations. Moreover, in cases of UCT, as observed in the present study, the fetus is fully developed, with good BCS and slightly to moderately autolyzed consistently with fetal death prior to abortion [45,47,56]. The most consistent histopathological finding related to UCT is the evidence of necrotic changes with secondary deposition of calcified material in the blood vessels of the allantochorion [6,45]. Mineralization due to hypoxia may be caused by the decreased integrity of damaged endothelial cell membranes, allowing extracellular calcium to enter cells and combine with phosphate to produce the vascular endothelial mineralization observed with UC compression [65]. In a recent study by Roach et al (2023), histological assessment of the allantochorion identified three features associated with UCT diagnosis such as autolysis, villous mineralization and villous karyorrhexis but no UCT cases exhibited a significant inflammatory infiltrate in the allantochorion compared to cases without UCT [58]. Overall, these findings are similar with those of the present study since the lesion was mainly characterized by necrosis, mineralization and scarce inflammatory clusters. The presence of karyorrhexis and mineralization likely reflects ischemic injury resulting from poor perfusion of the chorion and, in the case of UCT, as a response to episodes of venous stasis following increased vascular resistance associated with the increase in UCL and/or vascular obstruction. All this suggests a more chronic pathophysiology involving lengthening of the amniotic, not allantoic, UC [58]. Identification of these features enables evidence-based refinement of diagnostic criteria for UCT, which can now undergo consultation to reach a generally accepted consensus [58].

Apart from UCT, cervical pole necrosis was confirmed in the present

case. It has been reported that inadequate perfusion can cause intravascular thrombosis in the peripheral tissues of the allantochorion and possible localized ischemic necrosis in placental portions distant from UC insertion, such as the cervical pole [52,62,66]. In fact, cervical pole necrosis has been theorized to occur either secondarily to placental detachment at the cervical pole or by ischemic insult related to hemodynamic abnormalities associated with long UCs and/or vascular occlusion [14,56]. Grossly, this condition appears as a sharply delineated area of brown discoloration without an increase in thickness and random foci of ischemic necrosis that may produce fenestrations in the allantochorion. Loynachan (2023) reported that 33/57 cases of cervical pole necrosis had no signs of impending abortion while 22 cases presented signs of placentitis and were treated; in 44 cases the outcome was abortion and in one case was also present UCT; UCL was on average 89.4 cm long and fetuses age averaged 272 days of pregnancy [14]. These observations were also noted in the current report. Although the macroscopic lesion on the chorionic side of placenta ascending from the cervical star to the base of non-gravid horn and the isolation of *E. coli* in fetal membranes and tissues could give reason to suspect an ascending placentitis alone as cause of abortion [33,67], a microscopic examination of fetal membranes was needed to reach a final diagnosis. The histological changes described in the present report were suggestive of a chronic lesion of the allantochorion characterized by degeneration, necrosis and dystrophic calcification with concurrent low to moderate inflammatory infiltrate localized mainly at the cervical pole. The differential diagnosis should include placental infarction with subsequent secondary placentitis or primary chronic infectious placentitis. Both diagnostic conclusions are possible but considering the chronicity of the lesion characterized by low grade of inflammation, its main localization at the cervical pole, and the degeneration and necrosis, which are typical consequences of ischemia, the final diagnosis is most likely the former according to previous studies [56,68]. *E. coli* is one of the most common causes of cervical pole placentitis and an abortive agent in the equine species [8,69]. In this report, primary necrosis and devitalization of the cervical pole region, due to chronic ischemia associated with UCT, may have allowed this opportunistic pathogen to access the fetal membranes and consequently/secondarily instigating the placentitis [14,67]. Abortion due to bacterial infections generally is more frequent from the 9th month of gestation [8] as observed in this report.

4. Conclusions

Cervical pole necrosis is an idiopathic, non-infectious, placental condition that can result in abortion, premature birth, delivery of a weak foal or delivery of a viable foal. A histopathological exam is of paramount importance to reach a final diagnosis of this condition, but future studies are needed to better understand this unique cause of equine abortion.

CRediT authorship contribution statement

L. Fischetti: Writing – review & editing, Writing – original draft, Investigation, Data curation. **F. Perina:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Investigation, Data curation. **G. Sarli:** Writing – review & editing. **N. Ellero:** Writing – review & editing. **F. Freccero:** Writing – review & editing. **C. Castagnetti:** Writing – review & editing. **J. Mariella:** Writing – review & editing, Visualization, Project administration, Methodology, Investigation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgment

Abstract was presented as an Oral Communication at the 1st European Symposium on Animal Reproduction, Nantes, 21-23 September 2023.

References

[1] Giles JMD, Hong CB, Tuttle PA, Petrites-Murphy MB, Poonacha KB, et al. Causes of abortion, stillbirth, and perinatal death in horses: 3,527 cases (1986–1991). J Am Vet Med Assoc 1993;203(8):1170–5. https://doi.org/10.2460/javma.1993.203.08.1170.
[2] Agerholm JS, Klas EM, Damborg P, Borel N, Pedersen HG, Christoffersen MA. Diagnostic survey of aborted equine fetuses and stillborn premature foals in Denmark. Front Vet Sci 2021;8:740621. https://doi.org/10.3389/fvets.2021.740621.
[3] Saey V, van Loo H, Gryspeerdt A, Pronost S, Gasthuys F, et al. Detection of infectious agents in equine pregnancy loss, stillbirth and neonatal death. Vlaams Diergeneeskundig Tijdschr 2023;92(2):58–65. https://doi.org/10.21825/vdt.86337.
[4] Du Plessis JL. Some observations and data in thoroughbred mares. J S Afr Vet Med Ass 1964;35(2):215–22.
[5] Bain AM. Foetal losses during pregnancy in the thoroughbred mare: a record of 2,562 pregnancies. N Z Vet J 1969;17(8):155–8.
[6] Whitwell KE. Investigations into fetal and neonatal losses in the horse. Vet Clin North Am 1980;2(2):313–31. https://doi.org/10.1016/S0196-9846(17)30165-9.
[7] Hong CB, Donahue JM, Giles RC, Petritesmurphy MB, Poonacha KB, Roberts AW, et al. Equine abortion and stillbirth in Central Kentucky during 1988 and 1989 foaling seasons. J Vet Diagn Invest 1993;5(4):560–6. https://doi.org/10.1177/104063879300500410.
[8] Leon A, Pillon C, Tebourski I, Bruyas JF, Lupo C. Overview of the causes of abortion in horses, their follow-up and management. Reprod Domest Anim 2023;58(2):93–101. https://doi.org/10.1111/rda.14406.
[9] Smith KC, Blunden AS, Whitwell KE, Dunn KA, Wales AD. A survey of equine abortion, stillbirth and neonatal death in the UK from 1988 to 1997. Equine Vet J 2003;35(5):496–501. https://doi.org/10.2746/042516403775600578.
[10] Laugier C, Foucher N, Sevin C, Leon A, Tapprest J. A 24-year retrospective study of equine abortion in Normandy (France). J Equine Vet Sci 2011;31(3):116–23. https://doi.org/10.1016/j.jevs.2010.12.012.
[11] Marenzoni ML, Lepri E, Proietti PC, Bietta A, Coletti M, Timoney PJ, Passamonti F. Causes of equine abortion, stillbirth and neonatal death in central Italy. Vet Rec 2012;170:10. https://doi.org/10.1136/vr.100551.
[12] Ricard RM, St-Jean G, Duizer G, Atwal H, Wobeser BK. A 13-year retrospective study of equine abortions in Canada. Can Vet J 2022;63(7):715–21.
[13] Cantòn GJ, Navarro MA, Asin J, Chu P, Henderson EE, Mete A, et al. Equine abortion and stillbirth in California: a review of 1,774 cases received at a diagnostic laboratory, 1990–2022. J Vet Diagn Invest 2023;35(2):153–62. https://doi.org/10.1177/10406387231152788.
[14] Loynachan A. Cervical pole necrosis of Equine Placenta. Equine Disease Quart 2023;32(1):8–9.
[15] Troedsson MHT. Placentitis. In: Robertson NE, editor. Current therapy in equine medicine. Saunders Philadelphia; 2003. p. 297–300.
[16] Lyle SK. Immunology of infective preterm delivery in the mare. Equine Vet J 2014;46(6):661–8. https://doi.org/10.1111/evj.12243.
[17] N.M. Williams, J.M Donahue, D.C. Bolin, et al, Equine placental pathology: kentucky perspective, Proc workshop equine placenta. (2004) 88-92.
[18] Platt H. Infection of the horse fetus. J Repro Fert Suppl 1975;23:605–10.
[19] Whitwell KE. Infective placentitis in the mare. In: Powell DG, editor. Equine Infectious Diseases, Proceedings of the fifth international conference. University Press of; 1988. p. 172–80.
[20] Hong CB, Donahue JM, Giles RCJr, Petrites-Murphy MB, Poonchata KB, Roberts AW, Smith BJ, Tramontin RR, Tuttle PA, Swerczek TW. Etiology and pathology of equine placentitis. J Vet Diagn Invest 1993;5(1):56–63. https://doi.org/10.1177/104063879300500113.
[21] W.W Zent, N.M. Williams, J.M. Donahue, Placentitis in central Kentucky broodmares, Pferdeheilkunde. 15, 6 (1999) 630-632.
[22] I.F. Canisso, B.A. Ball, E. Erol, E.L. Squires, M.H.T. Troedsson, Comprehensive review on equine placentitis. In: Proc 61st annu meet Am assoc equine pract, 61 (2015) 490-509.
[23] Renaudin CD, Troedsson MHT, Gillis CL. Transrectal ultrasonographic evaluation of the normal equine placenta. Equine Vet Ed 2003;11:75–6.
[24] Bucca S, Fogarty U, A.Collins VS. Assessment of feto-placental well-being in the mare from mid-gestation to term: transrectal and transabdominal ultrasonographic features. Theriogenology 2005;64(3):542–57. https://doi.org/10.1016/j.theriogenology.2005.05.011.
[25] Reef VB, Vaala WE, Worth LT, Spencer PA, Hammett B. Ultrasonographic evaluation of the fetus and intrauterine environment in healthy mares during late gestation. Vet Radiol Ultrasound 1995;36(6):533–41. https://doi.org/10.1111/j.1740-8261.1995.tb00308.x.
[26] Roy MF, Kwong GP, Lambert J, Massie S, Lockhart S. Prognostic value and development of a scoring system in horses with systemic inflammatory Response syndrome. J Vet Intern Med 2017;31(2):582–92.
[27] Sheats MK. A comparative review of equine SIRS, sepsis, and neutrophils. Front Vet Sci 2019;12(6):69. https://doi.org/10.3389/fvets.2019.00069.

[28] Card C, Lopate C. CHAPTER 17—Infectious diseases of the puerperal period (A c. Di). In: Youngquist RS, Threlfall WR, editors. Current therapy in large animal theriogenology. Second ed. W.B. Saunders; 2007. p. 138–44. 2007.
[29] Ferrer MS, Palomares R. Aerobic uterine isolates and antimicrobial susceptibility in mares with post-partum metritis. Eq Vet J 2018;50(2):202–7. https://doi.org/10.1111/evj.12738.
[30] Ferrer MS, Pozor MA. How to manage the post-partum mare with metritis—Lessons learned from a retrospective bacteriological study. In: Proceedings of the 64th annual convention of the american association of equine practitioners, San Francisco, California, USA; 2018. p. 236–9.
[31] Lemes KM, Silva LA, Alonso MA, Celeghini ECC, Pugliesi G, Carvalho HF, Affonso FJ, Silva DF, Leite TG, de Arruda RP. Uterine vascular perfusion and involution during the postpartum period in mares. J Equine Vet Sci 2017;51:61–9. https://doi.org/10.1016/j.jevs.2016.10.010.
[32] Santschi EM, Vaala WE, et al. Identification of the high-risk pregnancy. In: McKinnon AO, et al., editors. Equine reproduction. 2nd ed. Oxford, UK: Wiley-Blackwell; 2011. p. 5–15.
[33] Govaere J, Roels K, Ververs C, Van de Velde M, De Lange V, Gerits I, Hoogewijs M, Van Soom A. Ascending placentitis in the mare. Vlaams Diergeneeskundig Tijdschr 2018;87(3):115–26. https://doi.org/10.21825/vdt.v87i3.16074.
[34] McKinnon AO, et al. Equine reproduction. second ed. USA: Wiley-Blackwell; 2011.
[35] Reef VB, Vaala WE, Worth LT, Sertich PL, Spencer PA. Ultrasonographic assessment of fetal well-being during late gestation: development of an equine biophysical profile. Eq Vet J 1996;28(3):200–8. https://doi.org/10.1111/j.2042-3306.1996.tb03773.x.
[36] Manning FA, Harman CR, Menticoglou S, Morrison I I. Assessment of fetal well-being with ultrasound. Obstet Gynecol Clin North Am 1991;18(4):891–905. https://doi.org/10.1016/S0889-8545(21)00258-8.
[37] Chamberlain PF, Manning FA, Morrison I, Harman CR, Lange IR. Ultrasound evaluation of amniotic fluid volume. I. The relationship of marginal and decreased amniotic fluid volumes to perinatal outcome. Am J Obstet Gynecol 1984;150(3):245–9. https://doi.org/10.1016/s0002-9378(84)90359-4.
[38] Moore JN, Vandenplas ML. Is it the systemic inflammatory response syndrome or endotoxemia in horses with colic? Vet Clin North Am Equine Pract 2014;30(2):337. https://doi.org/10.1016/j.cveq.2014.04.003.
[39] Rossdale P, Ousey J, Cottrill C, Chavatte P, Allen W, McGlanddery A. Effects of placental pathology on maternal plasma progestagen and mammary secretion calcium concentrations and on neonatal adrenocortical function in the horse. J Reprod Fertil Suppl 1991;44:579–90.
[40] Stawicki R, Ruebel H, Hansen P, Sheerin B, O'Donnell L, Lester G, Paccamonti D, LeBlanck M. Endocrinological findings in an experimental model of ascending placentitis in the mare. Theriogenology 2002;58(2):849–52. https://doi.org/10.1016/S0093-691X(02)00864-6.
[41] LeBlanc M. Ascending placentitis in the mare: what we learned from an experimental model. In: Proceeding of the 10 congresso nazionale multisala SIVE perugia. International Veterinary Information Service; 2004.
[42] Ousey JC. Hormone profiles and treatments in the late pregnant mare. Vet Clin North Am Equine Pract 2006;22(3):727–47. https://doi.org/10.1016/j.cveq.2006.08.004.
[43] Bentley AP, Barton MH, Lee MD, Norton NA, Moore JN. Antimicrobial-induced endotoxin and cytokine activity in an *in vitro* model of septicemia in foals. Am J Vet Res 2002;63(5):660–8. https://doi.org/10.2460/ajvr.2002.63.660.
[44] Vanderplassche M, Lauwers H. The twisted umbilical cord: an expression of kinesis of the equine fetus? Anim Reprod Sci 1986;10(3):163–75. https://doi.org/10.1016/0378-4320(86)90027-8.
[45] Williams NM. Umbilical cord torsion. Eq Dis Q 2002;10(3):3–4.
[46] S.W. Ricketts, A. Barrelet, K.E. Whitwell, Equine abortion, Eq Vet Ed, Manual. 15 (2003) 18-21. 10.1111/j.2042-3292.2003.tb01809.x.
[47] Frazer GS. Umbilical cord compromise as a cause of abortion. Eq Vet Ed 2007;19(10):535–7. https://doi.org/10.2746/095777307X246832.
[48] Snider TA. Umbilical cord torsion and coiling as a cause of dystocia and intrauterine fetal loss. Eq Vet Ed 2007;19:532–4. https://doi.org/10.2746/095777307X238534.
[49] Whitwell K, Wood J. The length of the umbilical cord: association with abortion in Thoroughbreds and investigations into factors influencing length. In: Proceedings of the 31st British equine veterinary association congress, Newmarket; 1992. p. 53–6. Equine Vet J.
[50] Oulten R, Fallon L, Meyer H, Zent W. Observations of the foaling data from two central Kentucky thoroughbred operations during the spring of 2003. In: Powell D, Furry D, Hale G, editors. Proceedings of the workshop on the equine placenta. University of Kentucky; 2003. p. 68–71.
[51] Mariella J, Iacono E, Lanci A, Merlo B, Palermo C, Morris L, et al. Macroscopic characteristics of the umbilical cord in Standardbred, Thoroughbred and Warmblood horses. Theriogenology 2018;113:166–70. https://doi.org/10.1016/j.theriogenology.2018.03.004.
[52] Whitwell KE. Morphology and pathology of the equine umbilical cord. J Reprod Fert Suppl 1975;23:599–603.
[53] Ricketts SW, Barrelet A, Whitwell KE. A review of the causes of abortion in UK mares and means of diagnosis used in an equine studfarm practice in Newmarket. Pferdeheilkunde Eq Med 2001;17(6):589–92.
[54] Szeredi L, Tenk M, Jánosi S, Pálfi V, Hotzel H, Sachse K, Pospischil A, Bozsó M, Glávits R, Molnár T. A survey of equine abortion and perinatal foal losses in Hungary during a three-year period (1998-2000). Acta Vet Hung 2008;56(3):353–67. https://doi.org/10.1556/AVet.56.2008.3.9.
[55] Laugier C, Foucher N, Sevin C, Leon A, Tapprest J. A 24-year retrospective study of equine abortion in Normandy (France). J Equine Vet Sci 2011;31(3):116–23. https://doi.org/10.1016/j.jevs.2010.12.012.
[56] Begg A, Carrick J, Shearer P, Stewart M, Brookes V. Visual atlas of the gross pathology of equine reproductive loss. AgriFutures Australia; 2002.
[57] Juffo GD, Antoniassi NAB, Bassuino DM, Gomes DC, Snel GGM, Pavarini SP, Driemeier D. Causes of abortion, stillbirth, and perinatal mortality in horses /causas de aborto, natimortalidade e morte perinatal em equinos. Pesqui Vet Bras 2022;42:e06808. https://doi.org/10.1590/1678-5150-pvb-6808.
[58] Roach JM, Foote AK, Verheyen KL, Bryan J, Smith KC, de Mestre AM. Gross and histological features associated with umbilical cord torsion in equine abortions. J Equine Vet Sci 2023;125:104815. https://doi.org/10.1016/j.jevs.2023.104815.
[59] A.E. Whitehead, T.S. Chenier, R.A Foster, Placental characteristics of standardbred mares, 51st Proc Annu Conv Am Assoc. (2005) 215–20.
[60] Roach J, Arango SJC, Smith K, Foote A, Verheyen K, de Mestre AM. Multivariable analysis to determine risk factors associated with abortion in mares. Reprod Fertil 2022;3(4):301–12. https://doi.org/10.1530/RAF-22-0087.
[61] Lawson JM, Verheyen K, Smith KC, Bryan JS, Foote AK, de Mestre AM. The equine umbilical cord in clinically healthy pregnancies. Equine Vet J 2024;56(4):742–50. https://doi.org/10.1111/evj.14055.
[62] Schlafer DH. The umbilical cord – lifeline to the outside world: structure, function, and pathology of the equine umbilical cord. In: Proceedings of the a Workshop on the equine placenta. University of Kentucky; 2003. p. 92–9.
[63] Christoffersen M, Nielsen SB, Madvig CB, Agerholm JS. Potential risk factors for fetal loss due to umbilical cord torsion in the mare. Theriogenology 2024;214:182–6. https://doi.org/10.1016/j.theriogenology.2023.10.026.
[64] Ginther OJ, Griffin PG. Equine fetal kinetics: presentation and location. Theriogenology 1993;40(1):1–11. https://doi.org/10.1016/0093-691x(93)90336-4.
[65] Poggi SH, Bostrom KI, Demer LL, Skinner HC, Koos BJ. Placental calcification: A metastatic process? Placenta 2001;22(6):591–6. https://doi.org/10.1053/plac.2001.0688.
[66] Whitwell KE. Equine placental pathology: the Newmarket perspective. In: Proceedings of the a workshop on the equine placenta. University of Kentucky; 2003. p. 84–7.
[67] Todhunter KH. Pathology of equine amnionitis and fetal loss: clinical and experimental studies. Australia: University of Queensland; 2014. PhD thesis.
[68] Begg CJ, Stewart M, Carrick J, Shearer P, Brookes V. Visual atlas of the histopathology of equine reproductive loss. AgriFutures Australia 2022.
[69] Macpherson ML. Diagnosis and treatment of equine placentitis. In: Turner SA, editor. Veterinary clinics of North America: equine practice. Veterinary clinics of North America: equine practice, 22. London, UK: Elsevier; 2006. p. 763–76.