Case Report

Toxicoinfectious metritis with fatal complications following retained fetal membranes in a mare

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Abstract

A 7-year, American Paint maiden mare, was presented 2 days after foaling following manual removal of retained fetal membranes. Fetid vaginal discharge was noticed and subinvoluted uterus was palpated transrectally. Endometrial cytology had Gram-positive cocci, Gram-negative and positive rods, and clostridial organisms with spores. Small fragment of fetal membranes was retrieved during uterine lavage. Mare developed signs of gastrointestinal disease with a large colon impaction and had refractory pain (response to multimodal analgesia). Despite intensive therapy, mare's condition deteriorated. Abdominocentesis revealed turbid fluid with increased protein and lactate consistent with elevated inflammatory cells and peritonitis. Necropsy confirmed necrotizing metritis with vascular thrombosis, localized fibrinous peritonitis, and focal adhesion between uterus and pelvic flexure of the large intestine. This case illustrated the rapid progression from retained fetal membranes to systemic illness and highlighted the diagnostic value of cytology to select antimicrobial agents in advance of culture and sensitivity results.

Keywords: Mare, retained fetal membranes, clostridial metritis, peritonitis, cytology, anaerobic infection

Background

Retained fetal membranes (RFM) is an emergency in mares because of the risk of complications.[1–3] RFM is defined as failure to expel fetal membranes within 3 hours after foaling,[3–5] occurs in up to 10.6 % of mares,[3–6] and may lead to metritis, endotoxemia, laminitis, gastrointestinal dysfunction or in severe cases death.[2–4,7] RFM delays uterine involution and provides an ideal environment for bacterial proliferation, predisposing the mare to toxic/septic metritis.[8–10] Bacterial colonization of RFM and endometrium along with endotoxin translocation across the compromised uterine mucosal barrier result in marked inflammatory response that can progress to metritis, systemic inflammation and associated complications.[10] In horses, the systemic inflammatory response associated with sepsis can result in widespread tissue injury, with the digital lamina being uniquely susceptible in this species, making laminitis a frequent sequela of severe postpartum inflammation.[10] Medical management of RFM includes antimicrobial and oxytocin therapy, uterine lavages and nonsteroidal antiinflammatory drugs (NSAIDs) along with prophylactic treatments (e.g. ice therapy of the lower limbs) to reduce laminitis risk.[5,10,11] Several approaches to facilitate fetal membranes expulsion have been described; manual extraction of the fetal membranes is sometimes used as a proactive intervention.[5–11] However, manual extraction has been associated with complications (e.g. tearing of endometrial and fetal tissue); and may result in retention of fetal membrane fragments, mucosal trauma, hemorrhage, delayed involution and secondary infection.[3,5,10,11]

Most uterine infections are associated with aerobic or facultative organisms (*Streptococcus equi* ssp. *zooepidemicus*, *Escherichia coli*, *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*).[5,10,12,13] Anaerobes are less commonly isolated but may include *Bacteroides* and *Clostridium* spp.[5,10,12–14] Mixed bacterial infections are common and are an important consideration when selecting initial antibiotic therapy for sick mares with RFM. Despite the limited number of reports, evidence suggests that these organisms are recognized as opportunistic pathogens capable of causing toxic/septic metritis when mucosal integrity is compromised.[14–17] Histologically, endometrial epithelial repair is not complete until days 4-7 postpartum.[18–20] Autolysis of RFM provides an ideal hypoxic

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environment that allows anaerobic bacterial proliferation and toxin production. Persistent endometrial inflammation and tissue damage facilitate bacterial invasion beyond the endometrium into deeper uterine layers, resulting in extension of inflammation into myometrium and development of metritis. Once deeper tissue invasion occurs, bacteria and their toxins may enter the systemic circulation, leading to subsequent bacteremia, septicemia, increased proinflammatory cytokine production and in some cases disseminated intravascular coagulation.[4,5,7,14–17] The latter condition arises due to elevated concentrations of proinflammatory cytokines, activation of intravascular clotting mechanisms, consumption of hemostatic factors, inhibition of platelet function and enhanced stimulation of fibrinolytic mechanisms.[21]

Endometrial cytology provides a rapid and inexpensive diagnostic method to confirm inflammation through identification of an abnormally increased number of inflammatory cells (particularly neutrophils) with associated toxic changes expressed per high-power field or as a proportion of total cells and to identify microorganisms while awaiting culture results.[22–24] Postpartum mares' endometrial cytology is influenced by days post foaling, dystocia, RFM, and bacterial type and density. These factors influence neutrophil percentage and persistence in a postpartum mare endometrial cytology. Over 70-80% neutrophils have been reported in the endometrial cytology of eutocic mares in the first 4 days postpartum and combined with no systemic signs represent physiological inflammation. Postpartum endometrial cytology in a mare with systemic signs (e.g. fever, subinvoluted uterus, vaginal discharge), neutrophil percentages of 90-100% with moderate to high density of bacteria (> 2+) is consistent with severe pathological inflammation.[25] Combining Gram stain and modified rapid Wright-Giemsa stains enhances endometrial cytology interpretation; ability to identify both cellular detail, and bacterial morphology, density and Gram type.[26] This evidence-based approach guides initial antimicrobial selection in mares with endometritis/metritis based on bacterial morphology and Gram type prior to culture results with subsequent adjustment if needed once culture and sensitivity results are available.[26]

Although many equine peritonitis cases originate from gastrointestinal disease, uterine infection may extend through uterine wall, resulting in the contamination of peritoneal cavity.[1,14,16,27] This complication has also been reported after manual removal of RFM and is possibly related to uterine trauma and bacterial translocation.[1,10] Peritoneal fluid analysis, provides valuable diagnostic and prognostic information.[27–29] Peritoneal fluid from healthy horses is typically clear and pale yellow with a lower total protein concentrations and low nucleated cell count. In contrast, peritonitis is commonly associated with increased peritoneal fluid total protein concentrations, elevated nucleated cell count with neutrophil predominance and increased lactate concentrations, reflecting severe inflammatory processes and altered local perfusion and metabolism.[27–29] In healthy mares, median postpartum peritoneal fluid parameters[30] were reported to be: total protein < 2.5 g/dl, nucleated cells/µl < 5,000, neutrophils 42%, monocytes 54% and lymphocytes 1%; and peritoneal lactate concentrations were < 2.5 mmol/l.[31]

We present a case of a postpartum mare that developed necrotizing toxicoinfectious metritis and secondary peritonitis (with a large colon impaction caused by adhesions between uterus and pelvic flexure) following manual RFM removal.

Case presentation

A 7-year, American Paint maiden mare, was presented to the Equine Theriogenology Service 2 days after foaling unobserved on day 308 of pregnancy with her foal. The mare foaled in the early morning. The fetal membranes failed to pass and, as per the referring veterinarian, the entire fetal membranes were removed later that afternoon on the day of parturition. Mare was given intravenous flunixin meglumine (Banamine®, Merck Animal Health, Kirkland, QC, Canada) during the procedure and second oral dose (mg amount not recorded) after 12 hours. Next day the mare became lethargic, hyporexic and febrile (rectal temperature was 39.5°C) and was referred.

On admission, the mare was quiet but alert (heart rate was 48 beats per minute, respiratory rate was 40 breaths per minute, rectal temperature was 38.1°C). Thyroid gland was palpably enlarged. Mucous membrane color was pink and capillary refill time was < 2 seconds. Borborygmi were decreased in all abdominal quadrants and digital pulses were within normal limits. Fetid sanguinopurulent discharge was visible at vulva and tail, accompanied by mild vulvar edema. Mare's body weight was 424 kg (estimated using a weight tape).

Mare exhibited a pain score of 4/12 (assessed using the horse grimace scale; 0: no pain and 12: maximum pain).[32] Reproductive examination was performed after mild sedation with intravenous detomidine (2 mg; Dormosedan®, Zoetis, Kirkland, QC, Canada) and intravenous butorphanol (2 mg; Torbugesic®, Zoetis). Transrectal palpation revealed a large fluid-filled subinvoluted uterus with borders that were not fully palpable. Transrectal ultrasonography identified a 33 mm follicle on the left ovary, no significant structures on the right ovary and 34 mm of echogenic Grade 3 intrauterine fluid. No ultrasonographic evidence of RFM was detected with transrectal ultrasonography. Vaginal examination using a sterile glass speculum revealed a moderately open cervix that was digitally dilated to allow evacuation of a profuse amount of malodorous sanguinopurulent secretions. A sample of this uterine fluid was collected directly from the uterus using a double guarded sterile uterine swab for bacterial culture and cytologic evaluation. Two air-dried and heated-fixed slides were prepared; 1 was stained with a modified rapid Wright-Giemsa stain (Hema 3™, Fisher Scientific, Pittsburgh, PA, USA) and the other with Gram stain (Jorgensen Laboratories, Loveland, CO, USA). Bacterial density was graded on a 1+ to 5+ scale: 1+ was < 1 bacterium per 30 high power field (hpf), 2+ was 1 bacterium per 30 hpf, 3+ was 1 bacterium per 10 hpf, 4+ was 2-10 bacteria per hpf, and 5+ was 11-50 bacteria per hpf. Numerous bacteria (5+) were observed, including Gram-positive cocci, Gram-positive rods of variable size, some with central spores, and Gram-negative rods, accompanied by fields comprised of 100% degenerative neutrophils with toxic changes (Figures 1 and 2).

Diagnosis was mixed bacterial postpartum endometritis/metritis with a presumptive anaerobic infection due to *Clostridium* spp. (Figures 2A and 2B). Large-volume warm water uterine lavages (total of 130 liters of 0.1% povidone-iodine solution [Dovidine®, Laboratoire Atlas, Montréal, QC, Canada]) were performed until the effluent became clearer and a small fragment of fetal membranes was recovered. After lavage, intravenous oxytocin (20 IU; Oxyto-Sure®, Vetoquinol, Lavaltrie, QC, Canada) was given.

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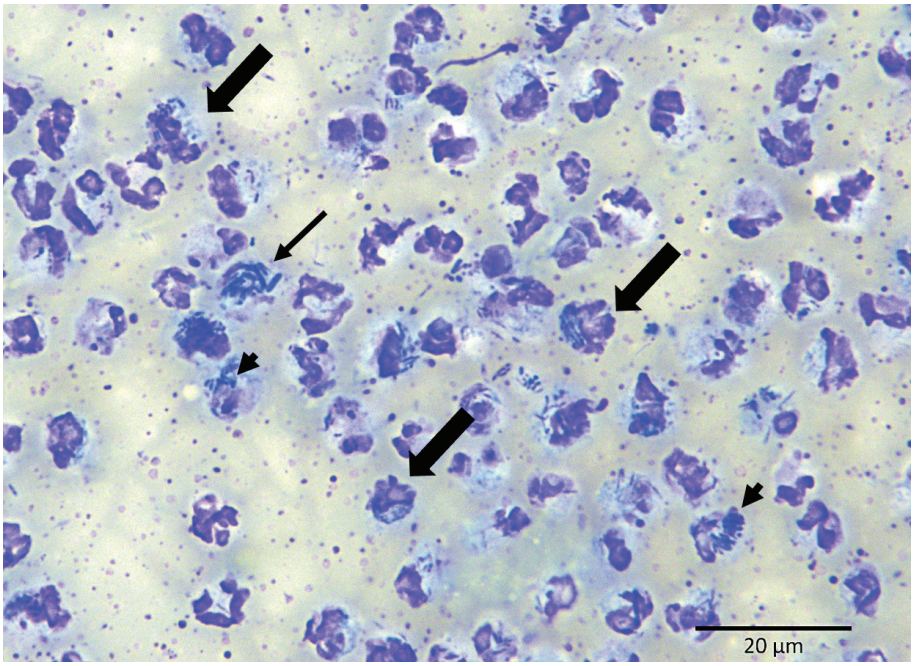

Figure 1. Light microscopic image of a modified rapid Wright-Giemsa stained endometrial cytology at 1,000 x magnification under oil immersion. Degenerate neutrophils with toxic changes (thick black arrows). Intracellular large rods (thin black arrow) and intracellular small rods (small arrowheads) are identified. Background contains abundant proteinaceous material and cellular debris

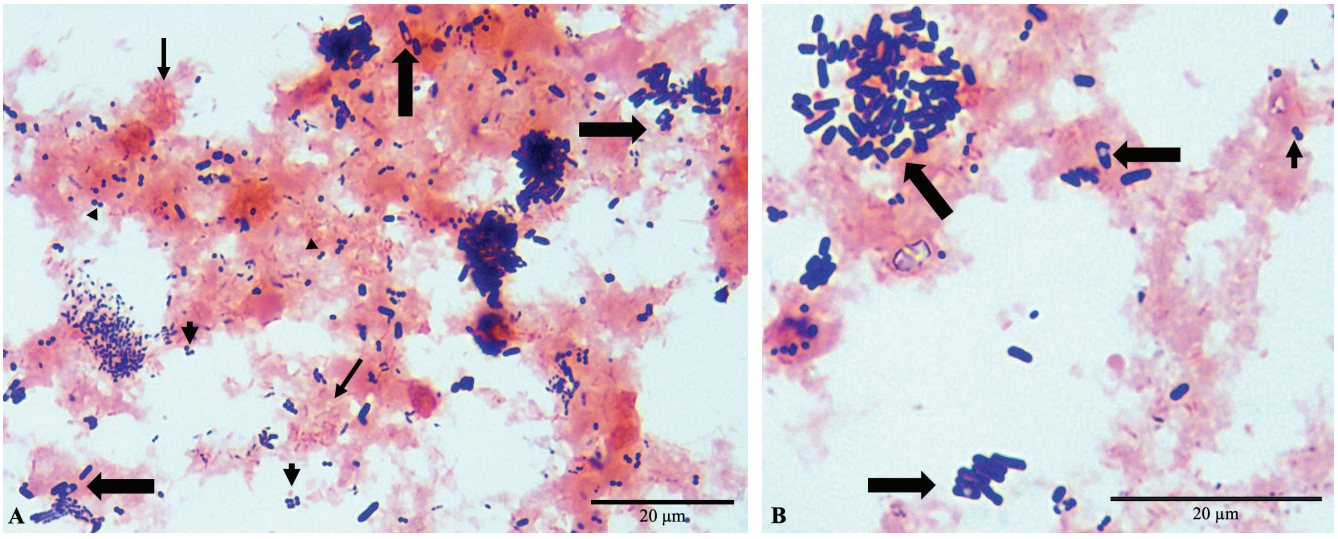

Figure 2. A. Light microscopic image of a Gram stained endometrial cytology at 1,000 x magnification under oil immersion. Large Gram-positive rods with central spores consistent with *Clostridium* spp. (thick black arrows); Gram-positive cocci (small arrowheads) and Gram-negative rods (black arrows) are identified. B. Higher magnification view of large Gram-positive rods with central spores consistent with *Clostridium* spp. (thick black arrows) and Gram-positive cocci (small arrowhead)

Mare was transferred to the Large Animal Internal Medicine Service for additional intensive care. Bloodwork revealed a packed cell volume (PCV) of 41% (normal range: 28-44%), a total protein of 85 grams/dl (normal range: 59-73 grams/dl), and a lactate of 2.5 mmol/l (normal range: 0-2 mmol/l) that indicated mild dehydration. Blood gas analysis revealed severe electrolyte imbalances including hyponatremia, hypokalemia, hypochloremia and hypomagnesemia. Serum biochemistry and complete blood count (CBC) results are presented (Table); CBC had a left-shifted leukogram with toxic changes, lymphopenia and hyperfibrinogenemia indicating acute inflammation. Serum biochemical analysis indicated hyperbilirubinemia and increased aspartate aminotransferase (AST) and

Table. Serum biochemistry and complete blood count results of the mare at admission

Serum chemistry	Result	Units	Flag	Normal range	Comment
Sodium	131	mmol/l	Low	132-142	
Potassium	2.8	mmol/l	Low	3.5-5.0	
Chloride	91	mmol/l	Low	92-103	
Bicarbonate	23	mmol/l	Low	26-35	
Anion gap	20	mmol/l		13-21	
Calcium	2.52	mmol/l		2.39-3.80	
Phosphorus	1.3	mmol/l	High	0.53-1.19	
Magnesium	0.32	mmol/l	Low	0.66-1.20	Icterus interference
Urea	3.8	mmol/l	Low	4.1-14.7	
Creatinine	56	µmol/l		52-126	Icterus interference
Glucose	12.9	mmol/l	High	4.1-5.5	
Total bilirubin	97.1	µmol/l	High	2-41	
Direct bilirubin	3.4	µmol/l		1-7	
Indirect bilirubin	93.7	µmol/l	High	3.9-32.8	
GGT	33	units/l		8-33	Icterus interference
GLDH	3	units/l		0-5	
AST	599	units/l	High	6-347	
CK	710	units/l	High	88-439	
Total protein	69	grams/l		60-74	
Albumin	33	grams/l		27-36	
Globulin	36	grams/l		26-41	
Albumin/globulin ratio	0.92			0.8-1.3	

Complete blood count

Leukocytes	Result	Units	Flag	Normal range
Total WBC	7.5	x 10^9/l		5.1-11
Segs	5.33 (71%)	x 10^9/l		1.7-8
Bands	1.05 (14%)	x 10^9/l	High	0-0
Toxic change	1+			
Lymphocytes	0.90 (12%)	x 10^9/l	Low	1.4-4.08
Monocytes	0.225 (3%)	x 10^9/l		0-0.42
Fibrinogen	5	grams/l	High	1-4

creatine kinase (CK), consistent with liver cholestasis and muscle injury.

Treatment

Mare was managed with multimodal therapy aimed to control uterine infection, systemic inflammation, gastrointestinal dysfunction and pain. Broad-spectrum antimicrobial therapy was initiated that consisted of intravenous sodium penicillin (22,000 IU/kg every 6 hours; Fresenius Kabi Canada, Toronto, ON, Canada) diluted in 0.9% sterile saline (Omega Laboratories, Montréal, QC, Canada), intravenous gentamicin (6.6 mg/kg every 24 hours; Gentocin®, Merck Animal Health, Kirkland, QC, Canada) and oral metronidazole (25 mg/kg every 12 hours; Mint-Metronidazole®, Mint Pharmaceuticals, Mississauga, ON, Canada). Antiinflammatory therapy included intravenous flunixin meglumine (1.1 mg/kg every 12 hours; Flunazine®, Vetoquinol, Lavaltrie, QC, Canada) to control inflammation and endotoxemia. Ice boots were applied prophylactically to reduce the risk of laminitis. Oral domperidone (1.1 mg/kg every 24 hours; Sanis Health, Brampton, ON, Canada) was given to support lactation.

Large-volume uterine lavages with dilute iodine 0.1% (Dovidine®, Laboratoire Atlas) were performed twice daily. Intravenous or intramuscular oxytocin (20 IU every 6 hours) Oxyto-Sure®, Vetoquinol) was given to enhance uterine clearance. With every uterine lavage, it was noted that the returning uterine fluid was not improving and the endometrial surface was roughened and sloughing off. Intrauterine sodium penicillin (5 x 10^6 IU; Fresenius Kabi) diluted in 0.9% sterile saline (Omega Laboratories) was given on the day after admission following the third uterine lavage.

Approximately 5 hours after admission, the mare developed signs of abdominal discomfort. A colic workup was performed under sedation using intravenous xylazine (300 mg; Rompun®, Elanco Canada, Mississauga, ON, Canada). Transrectal palpation identified a large colon impaction, and ultrasonography revealed a small volume of peritoneal fluid in the cranioventral abdomen. Nasogastric intubation yielded no net reflux.

Large colon impaction was managed with intravenous lactated Ringer's solution (2.4 ml/kg/hour; Baxter, Mississauga, ON, Canada) given with enteral fluid therapy supplemented with oral potassium chloride (0.2 mg/kg every 6 hours; Pfizer Canada ULC, Kirkland, QC, Canada). Repeated

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transrectal examinations revealed no improvement and just small quantities of firm feces with white mucus were retrieved manually.

Epidural injection was given for pain therapy, consisting of morphine (0.2 mg/kg; Sandoz Canada, Saint-Hubert, QC, Canada) and detomidine (0.06 mg/kg; Dormosedan®, Zoetis Canada) and the mare was sedated with intravenous detomidine (3 mg; Dormosedan®, Zoetis Canada). Subsequent pain management included intravenous xylazine (200 mg; Rompun®, Elanco Canada), intravenous detomidine (4 mg; Dormosedan®, Zoetis Canada), intramuscular butorphanol (10 mg; Torbugesic®, Zoetis Canada) and intravenous morphine (0.1 mg/kg every 4 hours; Sandoz Canada).

Outcome

During hospitalization the mare exhibited progressive signs of pain including pawing, flank watching, restlessness and decreased fecal output; pain score increased to 7/12 and heart rate increased (ranged 52-80 beats/minute). Borborygmi were decreased to none in all abdominal quadrants. Abdominal ultrasonography revealed 1 cm of free peritoneal fluid. Abdominocentesis revealed turbid fluid with elevated protein (4.8 grams/dl) and lactate (8 mmol/l); findings that were consistent with fibrinopurulent peritonitis. Despite ongoing therapy, mare's systemic condition deteriorated and was characterized by dull mentation, fever (39.2°C), abdominal distension and persistent signs of discomfort. Given the poor prognosis and lack of response to treatment, euthanasia was elected 2 days after admission.

Culture results of the uterine discharge at admission became available 2 days later, after euthanasia; revealed growth of 3+ *Bacteroides fragilis* (*B. fragilis*), 3+ *Clostridium perfringens* (*C. perfringens*), 1+ *Escherichia coli* (*E. coli*), and 2+ *Streptococcus dysgalactiae*. *E. coli* was sensitive to aminoglycosides, and *Streptococcus dysgalactiae* to β-lactam antibiotics. Antimicrobial susceptibility data were available only for these organisms.

Neonatal clinical findings and outcome

The filly was born on day 308 of pregnancy. On presentation the filly was bright, alert, small in stature and had signs of prematurity; weighed 28.5 kg, had a silky haircoat and dropped fetlocks. Thoracic auscultation findings were within normal limits. The filly was mildly hypothermic with a rectal temperature of 37.2°C. According to clinical history, the filly has been doing well since birth and was nursing normally. The filly's PCV was 48% and serum lactate was 1.1 mmol/l. A semiquantitative immunoglobulin test (IDEXX foal SNAP foal IgG Test, Markham, ON, Canada) revealed failure of passive transfer (< 400 mg/dl; normal range > 800 mg/dl). CBC revealed neutrophilic leukocytosis, with a total white blood cell count of 12 x 10^9 cells/l (normal range 5.1-11 x 10^9 cells/l), neutrophils (83%) of 9.96 x 10^9 cells/l (normal range 1.7-8 x 10^9 cells/l) with a left shift with bands at 0.24 x 10^9 cells/l (2%). Serum chemistry revealed low protein (38 g/l; normal range 60-74), elevated glucose (6.9 mmol/l; normal range 4.1-5.5 mmol/l), elevated bilirubin (total, direct and indirect respectively: 55.3 µmol/l; normal range 2-41 µmol/l, 8.3 µmol/l; normal range 1-7 µmol/l, 47 µmol/l; normal range 3.9-32.8 µmol/l) and increased liver enzymes (gamma-glutamyltransferase [GGT] 88 units/l; normal range 8-33 units/l, glutamate dehydrogenase [GLDH] 333 units/l; normal range 0-5 units/l; and AST 615 units/l; normal range 6-347 units/l) along with low globulins (11 grams/l; normal range 26-41grams/l), and a high albumin globulin ratio (2.4; normal range 0.8-1.3). The filly's problem list included: prematurity, failure of passive transfer, systemic inflammation and hepatocellular injury with cholestasis.

Following admission the filly was given: 1 liter of intravenous balanced electrolyte solution (Plasma-Lyte A, Baxter, Mississauga, ON, Canada) as a bolus, a soapy water enema, 950 ml of intravenous hyperimmune plasma (Equiplas Plus®, Templeton, CA), oral sulfamethoxazole and trimethoprim (30 mg/kg every 12 hours; Apo-Sulfatrim®, Apotex, Toronto, ON, Canada), oral sucralfate (500 mg every 6 hours; Teva, Toronto, ON, Canada) and intramuscular selenium (3 mg; Selon-E®, Vetoquinol). Supplemental mare milk replacer (Wet-Nurse™, Prairie Micro-Tech, Regina, SK, Canada) was offered free choice during hospitalization. Lateral and anteroposterior radiographs of the carpus and hock were obtained. Radiographic evaluation revealed a skeletal ossification index score of 2/4, consistent with poor ossification, based on the grading system.[33] In this system, Grade 1 indicates no evidence of ossification in most cuboidal bones of the carpus and tarsus whereas Grade 4 represents complete ossification of all cuboidal bones resembling those of adults in shape, with joint spaces of expected width. Grades 2 and 3 reflect progressive stages of cuboidal bone ossification and physis closure.[33,34]

Based on the clinical presentation of preterm birth, neonatal hypothermia, and maternal thyroid enlargement subsequent analyses were performed on stored serum and urine. Iodine concentrations were measured using inductively coupled mass spectrometry (Animal Health Laboratory, Guelph, ON, Canada). The filly had serum iodine concentrations of 160 µg/l, urine iodine concentrations of 81 µg/l, and urine iodine/urine creatinine ratio of 500 µg/g.[35] The mare had serum iodine concentrations of 3.5 µg/l (as per laboratory < 10 µg/l means deficiency in adults). Thyroxine concentrations of mare's serum were measured (Prairie Diagnostic Services, Saskatoon, SK Canada) by the Immulite method (Siemens Canada, Oakville, ON Canada) and were < 6.4 mmol/l, below detection concentrations. Results were interpreted to be consistent with maternal and neonatal iodine deficiency as a comorbidity. Iodine deficiency may be the underlying cause of the preterm delivery and likely contributed to filly's hypothermia.[36–39]

The filly was discharged as an orphan foal with a good prognosis and instructions for stall confinement and additional days of treatment with trimethoprim sulfa and sucralfate. Follow-up information provided by the owner 6 months after discharge indicated that the filly was cross-fostered to a mare and had grown normally without any reported health concerns.

Postmortem findings

Mare's postmortem examination confirmed uterus as the primary source of systemic disease. Uterus was enlarged, diffusely flaccid, red in color (Figure 3A) and had moderate amount of red fluid interspersed by a few strands of friable pieces of fetal membranes that were loosely adhered to the endometrium of uterine body and pregnant uterine horn. On cut section, the uterine wall was diffusely thickened and gelatinous with dark red to black discoloration (Figure 3B). Pelvic flexure of the large intestine was focally adhered to the surface of the uterus by organizing fibrinous adhesions. There was a small amount of turbid red free fluid within the peritoneal cavity and there were regions of congestion and hemorrhage throughout the nearby mesentery, mesocolon and small intestinal serosa.

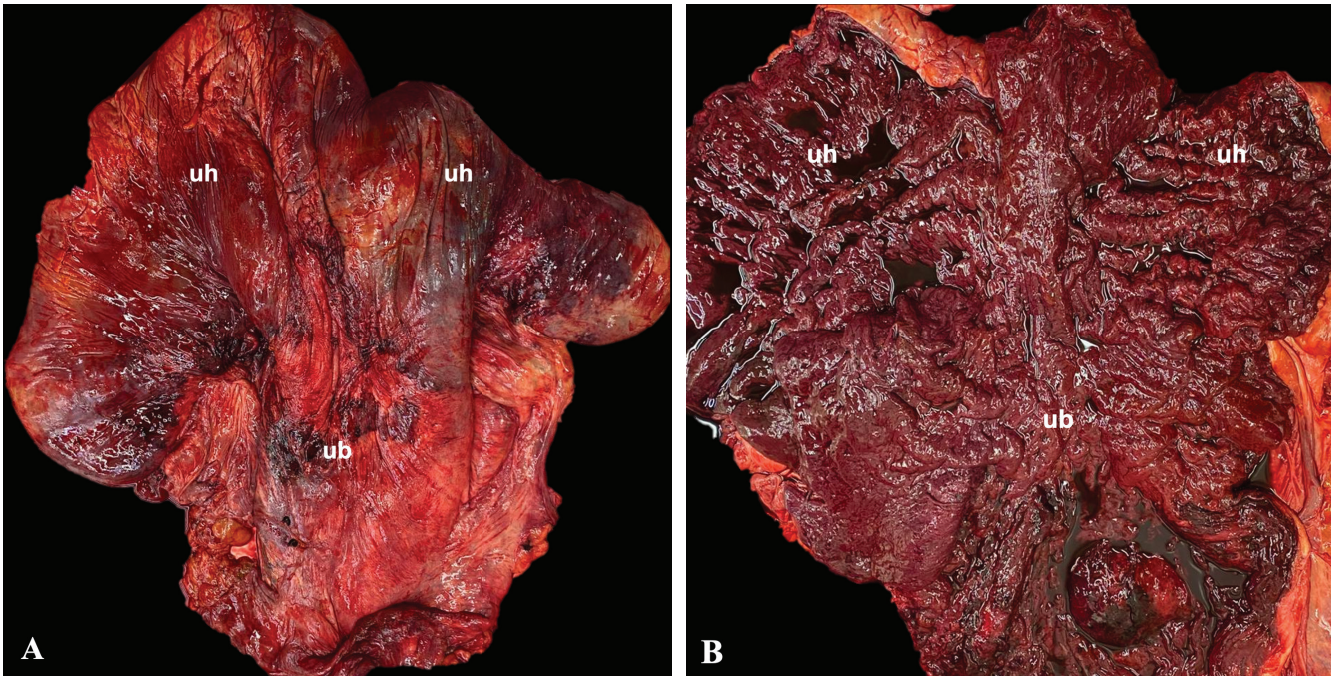

Figure 3. A. Postmortem gross image of the uterus in situ prior to opening. Uterus was enlarged, flaccid and red. Multifocal to coalescing areas of dark red discoloration (hemorrhage) were noticed across the uterine serosa, with coalescing to locally extensive areas of serosal roughening (fibrin exudation and organization). B. Postmortem gross image of the opened uterus demonstrating diffuse endometrial discoloration. Uterine wall is diffusely thickened and gelatinous, with dark red to black discoloration. In both images, the uterine horns (uh) and uterine body (ub) are identified

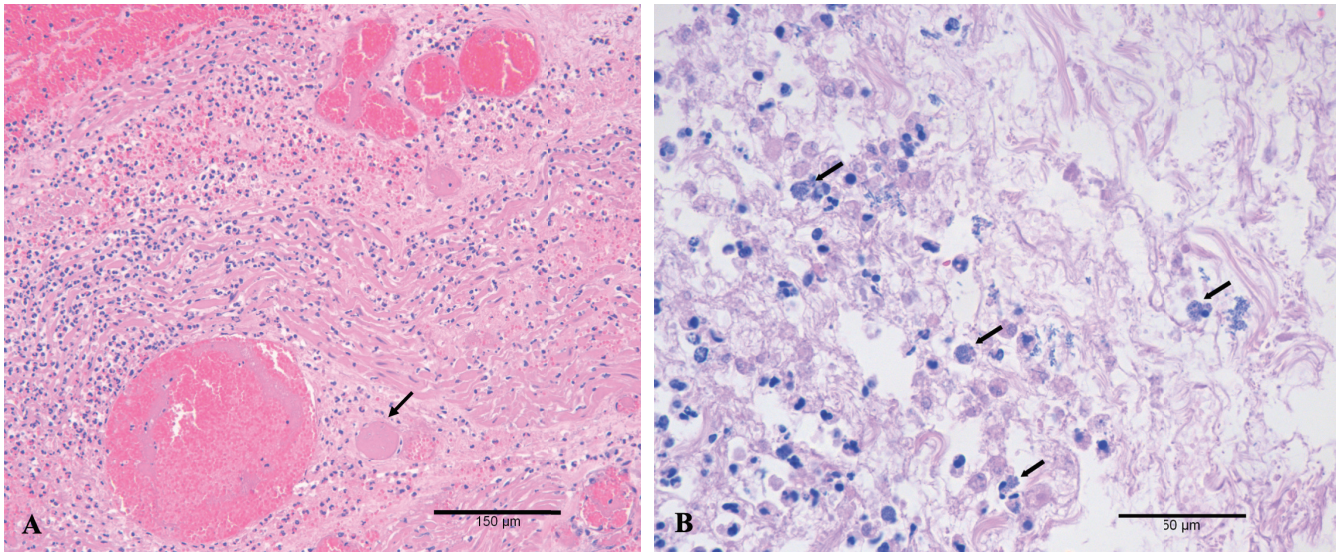

Figure 4. A. Light microscopic image of the uterine myometrium highlighting neutrophilic inflammation, vascular congestion, interstitial edema, hemorrhage and thrombosis (black arrow); 200 x magnification, hematoxylin and eosin histochemical staining. B. Light microscopic image of inflamed uterine tissue highlighting extra and intracellular (black arrows) intralesional bacteria; 600 x magnification, hematoxylin and eosin histochemical staining

Histopathologic examination of the uterus revealed severe neutrophilic and necrotizing metritis with widespread vascular thrombosis, mural edema and hemorrhage (Figure 4A and 4B). Multifocal regions of necrosis and/or inflammation inundated with myriad mixed bacterial populations including Gram-positive rods, Gram-negative cocci and Gram-negative coccobacilli.

Discussion

This case demonstrated the rapid progression of RFM to necrotizing toxicoinfectious metritis, fibrinopurulent peritonitis and gastrointestinal complications in a postpartum mare. Although manual extraction of the fetal membranes has been described as an iatrogenic risk in mares due to potential

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uterine trauma and bacterial translocation, prolonged retention of devitalized fetal membranes alone can predispose mares to severe uterine infection, even without manual intervention.[3,5,10,11] Retention of even a small portion of fetal membranes may be as clinically significant as retention of a larger tissue piece.[10] In this mare, lack of early uterine management and delay in initiation of systemic antimicrobial therapy likely provided favorable conditions for anaerobic bacteria, including *C. perfringens* and *B. fragilis* to proliferate within devitalized uterine tissue and progressed to systemic disease.

Anaerobic bacteria (e.g. *C. perfringens* and *B. fragilis*) are opportunistic pathogens that proliferate in hypoxic postpartum environments, particularly when endometrial integrity is compromised. Uterine manipulation can cause microtrauma that promotes bacterial colonization, toxin release and vascular thrombosis.[10,15] Once infection is established, clostridial toxins induce rapid tissue necrosis and systemic toxemia. These changes explain the mare's rapid systemic deterioration and gastrointestinal hypomotility despite aggressive therapy. Clinical deterioration within 48 hours after admission was consistent with reports of toxicoinfectious metritis secondary to RFM.[5,10] This highlighted the fulminant nature of anaerobic uterine infections, particularly those involving clostridial organisms and how limited the opportunity for effective treatment becomes once infection is established.

Reports of clostridial metritis in equids are limited[14–16] but similar infections have been described in other species. In women, clostridial endometritis is uncommon but can be potentially fatal if recognition and treatment are delayed.[40–42] Postpartum gangrenous metritis due to *Clostridium* spp. has also been reported in dairy goats and cattle, and is frequently associated with fatal outcomes.[43–45] These findings confirmed that clostridial species can cause life threatening uterine disease across species in anaerobic conditions, tissue trauma and/or contamination.

In this case, transmural uterine necrosis secondary to bacterial infection led to the development of localized fibrinopurulent peritonitis. Organizing fibrinous adhesions between devitalized uterine surface and pelvic flexure of the large colon likely compromised gastrointestinal motility. Although equine peritonitis most often results from gastrointestinal sources, uterine origin should be considered in postpartum mares with fever, colic and abdominal pain following intrauterine manipulation.[1,5,10]

The mare's pain remained refractory despite multimodal therapy. Poor therapeutic response emphasized the grave prognosis associated with anaerobic metritis once necrosis and vascular thrombosis have developed. Although antimicrobial therapy targeting anaerobic organism was given, antimicrobial susceptibility testing for *C. perfringens* was not available in this case. Regardless of antimicrobial selection, clostridial metritis is associated with rapid toxin-mediated tissue destruction, systemic inflammatory response and high mortality once advanced disease is established.[10,15] Although postpartum metritis commonly affects future fertility,[5,8,9] clostridial involvement represents an immediate risk to survival. Prophylactic antimicrobial treatment should be considered in RFM, to minimize the risk of this potentially fatal complication. It is uncertain how iodine deficiency affected mare's clinical progression but it would have been an additional source of physiologic stress and may have contributed to the lack of response to therapy.

Endometrial cytology was a helpful diagnostic tool in this case. Identification of large Gram-positive rods with the characteristic central spores provided early presumptive evidence of clostridial infection to include metronidazole in the initial antibiotic therapy. This is an advantage of combining rapid Wright-Giemsa stain (provides good cellular detail) with Gram stain (enables Gram bacteria type identification) in mares with uterine discharge.[22–24,26] *E. coli* and streptococcal bacteria were sensitive to antibiotics used; however, the continued uterine discharge was an indicator that the mare was not responding to treatment. We speculated that hysterectomy may have provided the best chance for the mare's survival if it had been performed closer to admission.

This case illustrated the diagnostic challenges associated with RFM when fetal membrane examination at parturition is incomplete or undocumented and ultrasonographic findings are inconclusive. Fetal membranes provide valuable information regarding pregnancy and neonatal health.[8–10] In this mare, no definitive evidence of RFM was identified on ultrasonographic examination at admission, likely due to marked uterine distension and large volume of intrauterine fluid, and retention was only confirmed during uterine lavage. These findings emphasize the importance of careful fetal membrane evaluation following foaling, as retention of even small fetal membrane fragments may result in severe clinical consequences despite externally visible membranes.

In summary, this case demonstrated the rapid and potentially fatal progression of RFM when not managed promptly and highlighted the importance of timely diagnostic assessment in postpartum mares. Cytologic evaluation of uterine discharge provides a rapid, point-of-care diagnostic tool that enables early identification of anaerobic pathogens, including *Clostridium* spp. that are not routinely cultured from the equine uterus. Although recognition may not alter the outcome in advanced cases such as this one, prompt initiation of systemic antimicrobial and NSAID therapy guided by cytologic findings are key to prevent the development of clostridial metritis and secondary peritonitis, thereby improving clinical outcomes.

Learning points

- Postpartum uterine infections following RFM may progress rapidly and cause systemic toxemia and peritonitis
- Endometrial cytology should be used to identify bacterial organism(s) by morphology and their Gram type as a point-of-care approach to guide initial antibiotic therapy before culture and sensitivity results are available
- Prompt antimicrobial and NSAID therapy are critical to prevent fatal systemic complications of RFM
- Anaerobic uterine pathogens in cases of RFM may be associated with poorer outcomes
- Routine fetal membranes examination following parturition is essential, as retention of even small fetal membrane fragments may lead to severe postpartum uterine infection and systemic complications

Conflict of interest

None to declare.

Author's contribution

CE prepared the original draft; JG, MA, FU, MZ and CC were involved in clinical case management and manuscript content; MZ conducted necropsy and histopathologic evaluation. All authors reviewed and approved the final version of the manuscript.

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