Case Report

Pregnancy hydrops in a recipient mare carrying an in vitro-produced embryo

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Abstract

An 8-year, Thoroughbred mare, was referred on day 225 of pregnancy for progressive abdominal distension and on owner's suspicion of hydrops. Mare was a recipient of a vitrified and warmed embryo that was produced via intracytoplasmic sperm injection. Transabdominal ultrasonography revealed increased allantoic and amniotic fluid depths. Fetal urachus was distended and fetal mouth was open throughout the examination. To salvage the mare, termination of pregnancy was elected on day 230 of pregnancy due to progressive abdominal distension and evidence of fetal compromise. Gross examination of the fetus revealed craniofacial abnormalities that precluded the mouth from closing. This case is another example of fetal craniofacial malformations' association with hydrops; it is also important to consider the potential association of hydrops with advanced reproductive techniques, as observed in cattle following in vitro fertilization and cloning.

Keywords: Mare, fetus, craniofacial malformation, hydrops, pregnancy termination

Background

In mares, hydrops of pregnancy is rare but a potentially life-threatening complication. Hydrops is characterized by excessive fluid accumulations in the amniotic (hydramnios) or allantoic (hydrallantois) cavities, with latter reported more frequently in mares.[1,2] Pathophysiology of hydrops conditions remains poorly understood. Abnormal placentation in association with placentitis or abnormal angiogenesis has been implicated in hydrallantois.[3,4] Furthermore, hereditary factors, umbilical abnormalities, and multiple pregnancies have been suggested.[2,3,5,6] Fetal abnormalities have been implicated in cases of hydramnios.[7–9] Regardless of the etiology, medical intervention is often warranted, as hydrops is a progressive condition with adverse sequelae including body wall injury, uterine rupture, hypovolemic shock, and/or dystocia.[8–11] The most common clinical sign reported is a dramatic increase in abdominal distension;[1] progressive abdominal distension may be accompanied by ventral edema, depression, anorexia, tachycardia, and in some cases, dyspnea.[1,8,9] These clinical signs develop over days to weeks, with hydrops allantois often associated with a more rapid progression of abdominal distension.[7,8] Diagnosis is based on abnormal accumulations of fetal fluids detected via transrectal palpation and transabdominal ultrasonography.[2,8] Differential diagnoses should include twin pregnancies and/or gastrointestinal disease.[11] Historically, treatment has involved early termination of pregnancy and controlled fetal fluid drainage.[7,11,12] Prognosis for survival and future fertility of the mare is good after appropriate medical intervention;[1,12] however, prognosis for fetal survival is believed to be poor even if termination of pregnancy is not elected.[1–3,8,9] Despite the poor prognosis, there are limited number of reports of hydrops resulting in live birth.[13–15] This collection of positive outcomes may warrant reevaluation of management options and research into better understanding the disease process. This report describes the management of an 8-year, recipient mare that developed hydrops following transfer of a vitrified, warmed embryo that was produced via intracytoplasmic sperm injection (ICSI) and documents the congenital craniofacial defects observed in the fetus.

Case presentation

An 8-year, Thoroughbred multiparous mare, was referred on day 225 of pregnancy for progressive abdominal distension and suspicion of hydrops; mare was a recipient of a vitrified, warmed embryo that was produced via ICSI. Serial examinations on days 23, 29, 31, 38, and 45 of pregnancy confirmed a single viable fetus and ruled out the possibility of monozygotic twins. On presentation, mare exhibited subjectively more abdominal distension than expected for a mare on day 225 of pregnancy. On transrectal palpation, the gravid uterus felt domed and slightly taut, extending above the pubic bone.

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Transrectal ultrasonography revealed normal echogenicity of allantoic and amniotic fluids. Combined thickness of the uterus and placenta (CTUP) measured 0.4 cm (considered normal for this stage of pregnancy).[16] Transabdominal ultrasonography revealed increased allantoic (> 27 cm) and amniotic (> 11 cm) fluid depths. Reported normal allantoic and amniotic fluid depths were 4.7-22.1 and 0.8-14.9 cm, respectively.[17,18] Fetal urachus appeared distended and had luminal diameter (2.0-2.7 cm). Interestingly, the fetal mouth was open with the tongue protruding throughout the examination. Fetal heart rate was 78 beats per minute and biparietal diameter was 6.74 cm (considered normal for this stage of pregnancy).[18,19]

Medical management was attempted on the farm; mare was treated with once daily oral firocoxib (0.11 mg/kg; Boehringer Ingelheim Animal Health, Duluth, GA, USA) and altrenogest (0.088 mg/kg; Merck Animal Health, Summit, NJ, USA). In addition, mare was treated twice daily with oral pentoxifylline (8.5 mg/kg; compounded by Rood and Riddle Veterinary Pharmacy, Lexington, KY, USA), acetylsalicylic acid (50 mg/kg; compounded by Rood and Riddle Veterinary Pharmacy), and doxycycline (10 mg/kg; compounded by Rood and Riddle Veterinary Pharmacy). Antimicrobial therapy was initiated due to the possible association between hydrops and placentitis[3] and mare was monitored for signs of discomfort or impending abortion.

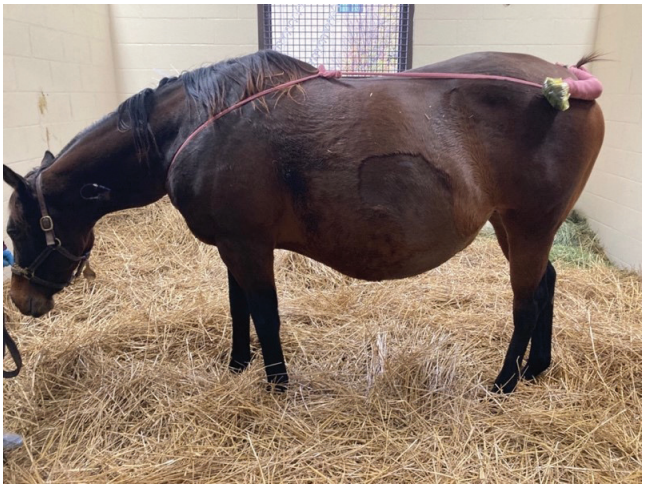

Figure 1. Lateral view of mare on day 230 of pregnancy

Five days after initial evaluation, the mare appeared to have a wider ventral distribution of abdominal distension and was referred due to concern for impending body wall injury. Altrenogest was discontinued and mare was admitted to the referral hospital on day 230 of pregnancy. Physical examination revealed marked abdominal distension and a plaque of ventral abdominal edema (Figure 1). Transrectal palpation and ultrasonography revealed domed gravid uterus extending well above mare's pubic level. The CTUP was 0.6 cm (considered normal for this stage of pregnancy).[16] Transabdominal ultrasonography revealed turgid amniotic membrane, lacking undulations (Figure 2), suggesting increased intra-amniotic fluid pressure. The largest umbilical vessel within the amniotic cavity was 1.13 cm in diameter (Figure 3A); fetal urachus had increased luminal diameter (Figure 3B). Fetal bladder measured 6 x 5 cm and was distended with anechoic fluid. Doppler was used to identify the urachus by lack of blood flow compared to umbilical vessels (Figure 4). Fetal heart rate was 90 beats per minute and pericardial effusion was suspected due to the appearance of hypoechoic fluid around beating heart muscle. Again, the fetus's mouth was open. Additionally, ultrasonography of mare's ventral abdomen revealed marked subcutaneous edema, consistent with impaired lymphatic and venous drainage secondary to gravid uterus weight. A presumptive diagnosis of hydrops was made based on these clinical findings. Termination of pregnancy and induction of parturition was elected to salvage the mare.

Plan for pregnancy termination included intravenous fluid therapy, controlled drainage of fetal fluids, assisted delivery of the fetus, and subsequent euthanasia of the preterm fetus. Given the risk of hypovolemic shock during fluid drainage and fetal extraction,[1,14] intravenous fluid therapy would be

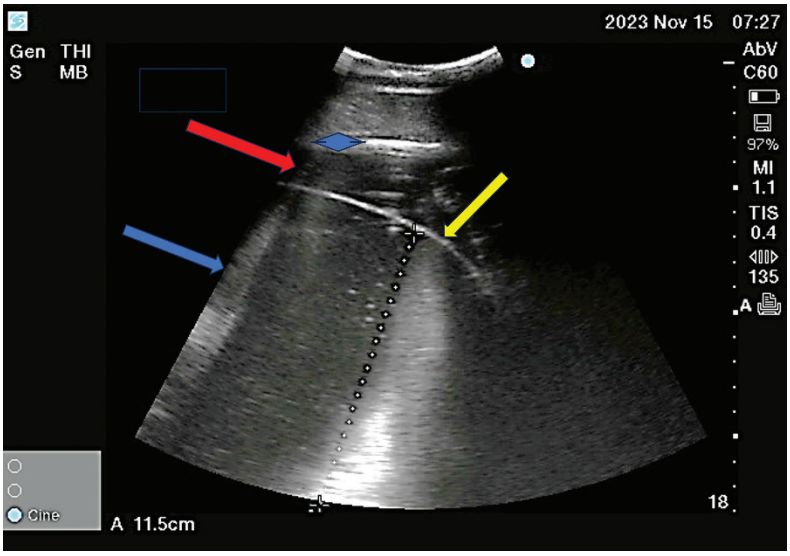

Figure 2. Transabdominal ultrasonographic image of uterus: amniotic membrane (yellow arrow); amniotic cavity (blue arrow); allantoic cavity (red arrow); chorioallantois and endometrium demarcation (blue diamond) where CTUP was measured; note lack of undulations in amniotic membrane.

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initiated prior to drainage, and venous access would be maintained to facilitate rapid fluid replacement as required. Primary objective of this intervention was to preserve mare's life and maintain future reproductive potential.

Treatment

Mare was sedated with 100 mg intravenous xylazine and prepared for the procedure by placement of bilateral jugular catheters. A 14 gauge catheter was placed in the left jugular vein and a 10 gauge double-lumen catheter was placed in the right jugular vein. Two hours prior to transcervical drainage of the fetal fluids, mare received 2 liters bolus of intravenous hypertonic (7.2%) saline followed by lactated ringer solution bolus. Mare's tail was wrapped in brown gauze and the perineum washed with soap and warm water. Initial vaginal examination with a sterile sleeve revealed a moderately toned cervix and on digital palpation 2 fingers could be passed into the cervical canal. Three misoprostol tablets (200 µg each) (Ani Pharmaceuticals, Baudette, MN, USA) were dissolved in 2 ml of sterile saline to form a paste and then manually applied to the cervix with a small amount of sterile lubricant. After ~ 60

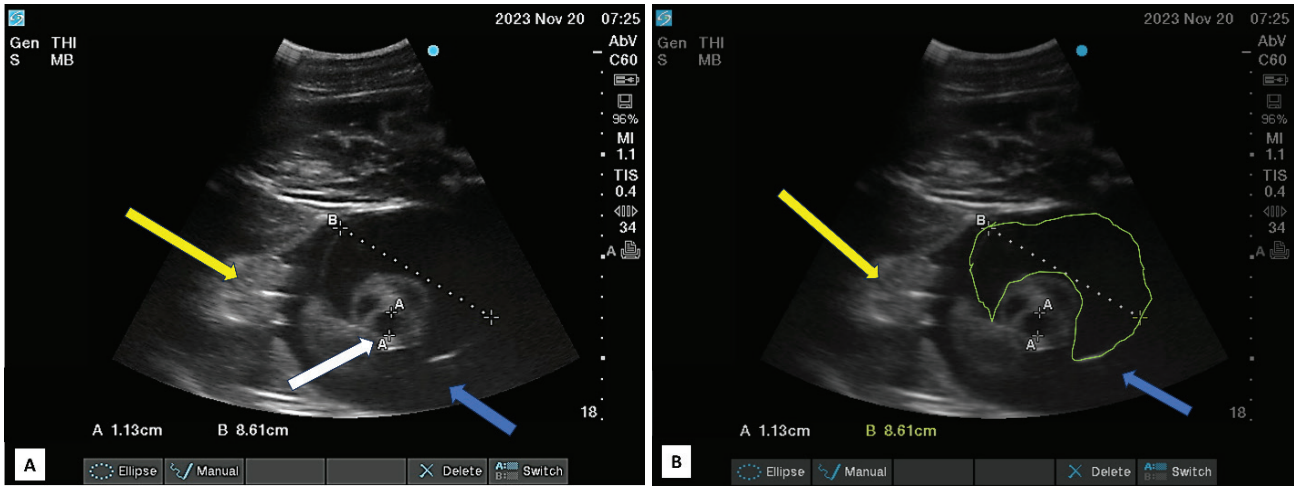

Figure 3. Transabdominal ultrasonographic image of umbilical cord structures within the amniotic cavity: A. diameter of largest umbilical vessel (white arrow); B. diameter of urachus (yellow line); mammary gland of filly fetus (yellow arrow) and amniotic cavity (blue arrow).

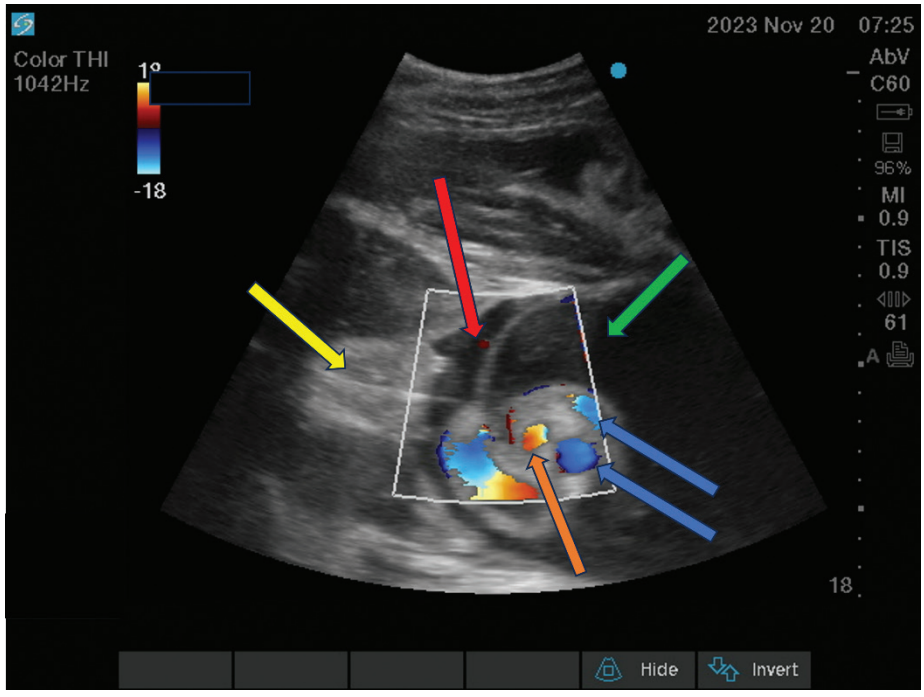

Figure 4. Transabdominal ultrasonographic image of the fetus and umbilical cord structures within the amniotic cavity: distended lumen of the urachus (green arrow); amniotic fluid within the amniotic cavity (red arrow); umbilical vessel presumed to be the umbilical arteries (blue arrows): umbilical vessel presumed to be the umbilical vein (orange arrow); mammary gland of filly fetus (yellow arrow).

minutes, digital palpation of the cervix confirmed that the cervix was relaxed enough to allow passage of the trocar and manual exploration of the uterus. A combination of intravenous detomidine hydrochloride (3 mg; Zoetis, Kalamazoo, MI, USA) and acepromazine maleate (5 mg; MWI Animal Health, Boise, ID, USA) was given to provide light sedation. Manual palpation of the cervix was again performed; digital dilation was continued until fetal membranes were palpable, protruding from the external cervical os. A sterilized sharp, 32 French trocar was introduced vaginally, passed through the cervix, and fetal membranes were punctured to initiate drainage of fetal fluids. Trocar was removed, and sterile lavage tubing was placed through the small hole formed by the trocar and into the large fluid pocket. The operator controlled the rate of fluid efflux by positioning their thumb over the end of the tubing as needed; ~ 50 liters of fluid was gradually drained over 1 hour.

Outcome

After sufficient drainage, fetus was grasped by front legs, and delivery was initiated with fetus in anterior-longitudinal presentation, dorsosacral position, with head and both front feet extended. Although the fetus was small, there was some difficulty passing the head and shoulders through the birth canal. Further palpation revealed that fetal mouth was fixed in an open position. Rostral maxilla was catching on the dorsal aspect of mare's pelvis; this was corrected with repulsion of the fetus and realignment assuring that the head and neck remained fully extended to allow passage through the birth canal. Mild traction was applied by a single operator to deliver the fetus. Hairless fetus was euthanized (pentobarbital sodium phenytoin sodium) via cardiac injection and submitted for necropsy.

Postprocedural management

Mare was monitored for signs of shock and maintained on intravenous fluids supplemented with 125 ml of 23% calcium gluconate solution (Durvet, Blue Springs, MO, USA) and 50 units of oxytocin (Bimeda, Le Sueur, MN, USA) per 5 liters lactated ringer solution to aid in passage of fetal membranes. Intravenous antimicrobial therapy (every 6 hours) was initiated with 22,000 IU/kg units buffered penicillin G potassium (WG Critical Care, Paramus, NJ, USA) and once daily 6.6 mg/kg gentamicin sulfate (MWI Animal Health). Antiinflammatory therapy consisted of twice daily intravenous flunixin meglumine (1.1 mg/kg; Merck Animal Health) treatment and oral pentoxifylline (8.5 mg/kg; compounded by Rood and Riddle Veterinary Pharmacy). Due to retained fetal membranes, uterine lavage with 5 liters of sterile isotonic fluid was performed, ~ 4 hours after parturition. Mare's vital parameters remained stable overnight and intravenous fluids were discontinued. Uterine lavage was repeated the following morning and 1,000,000 IU of polymyxin B (Eugia, East Windsor, NJ, USA) diluted in 1 liter lactated ringer solution was infused into the uterus. Fetal membranes passed in several pieces throughout the course of the day. Subsequent uterine lavage and vaginal examination confirmed complete passage of fetal membranes (within 24 hours after parturition). Uterine lavage and infusion of polymyxin B (Eugia) was repeated for 4 days until the efflux was clear. Mare had no signs of systemic illness or discomfort and was discharged 5 days after admission.

Filly fetus (12 kg) had craniofacial defects and renal dysplasia. Mandible had an irregular upward bowing and maxilla was enlarged, with medial impingement of the hard palate; both caused maxillary-mandibular malocclusion, oral tongue protrusion and complete inability to close the mouth (Figure 5). There were no gross abnormalities of the fetal kidney; however, histopathology revealed multifocal tubular ectasia, tubular cyst formation, scant intratubular proteinaceous material, tubular hydropic change, and variably sized clusters of small contracted to rudimentary tubules encompassed by bands of fibrous connective tissue. There was moderate distension of the bladder with urine and mild to moderate transmural

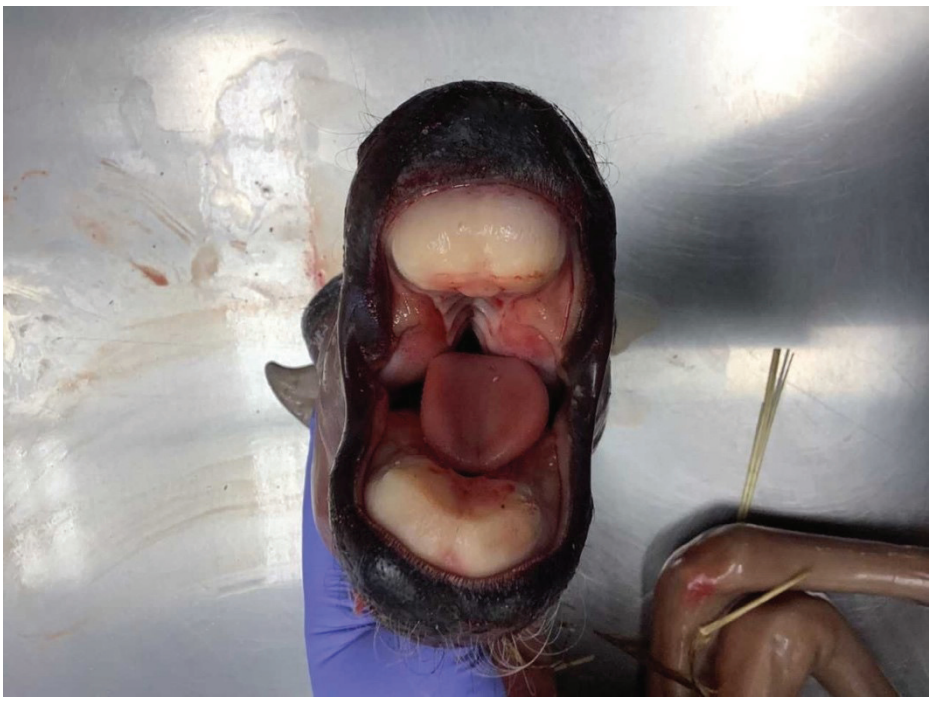

Figure 5. Postmortem image of the fetus; note maxillary-mandibular malocclusion.

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edema of the umbilicus. There was no underlying infectious process diagnosed in the fetus. Gross pathology and histopathology of the fetal membranes, including the amniotic and allantoic portions of the umbilical cord, were not possible because of retained fetal membranes.

Discussion

Our case demonstrated the management of hydrops in an embryo recipient mare and documented fetal abnormalities associated with this condition. Presumptive diagnosis of hydramnios (based on transabdominal ultrasonography findings) was supported by fetal necropsy; however, increased allantoic depths were observed on transabdominal ultrasonography, suggesting that the allantoic compartment was also affected. Definitive diagnosis and differentiation between hydramnios and hydrallantois are challenging as ultrasonography alone lacks specificity.[1] Amniocentesis and subsequent fluid analysis (not performed) could have provided a definitive diagnosis as to which fluid compartment was affected.[15,20] Decision to terminate pregnancy in cases of hydrops depends on the stage of pregnancy, progression of clinical signs, and fetal and/or maternal values. In our case, delivery of a live foal might have been considered of greater value than the life and future reproductive performance of the recipient mare. For this reason, conservative medical management and monitoring was initiated but ultimately, long-term management was not feasible for mare and a positive outcome was unlikely for fetus. Although the delivery of a live foal following hydrops diagnosis has been documented,[13–15] those cases involved mares that were presented with clinical signs further along in pregnancy. In contrast, this mare demonstrated clinical signs on day 225 of pregnancy, well before full term. Additionally, transabdominal ultrasonography evaluation of the fetus suggested progressive fetal compromise and potential abnormalities.

Distension of fetal urachus and bladder suggested partial occlusion or compression of the amniotic umbilical cord impeding urinary drainage; renal dysplasia may have resulted from this obstructive uropathy, as demonstrated in fetal lambs.[21] No prior reports linked these abnormalities with equine hydrops. A hydrallantois case resulted in a live foal that developed uroabdomen associated with a necrotic urachus and torn, necrotic bladder, although it was suggested that these lesions could have occurred postnatally.[15] A case of hydramnios described a large, twisted umbilicus in addition to fetal craniofacial abnormalities.[8] Excess amniotic fluid may predispose the umbilical cord to twisting.[22] Twisting of the amniotic portion of the umbilical cord may result in urachal distension and compromise blood flow to the fetus.[23] In our case, umbilical cord was not examined to confirm ultrasonographic findings and to further characterize the suspected pathology.

Fetal pericardial effusion was suspected and has not been previously reported in association with equine hydrops. In humans, fetal pericardial effusion occurs as a manifestation of fetal hydrops.[24] Fluid accumulates within the fetus due to an imbalance in interstitial fluid production and lymphatic drainage brought on by cardiovascular dysfunction, hypoproteinemia, or lymphatic obstruction.[25] Fetal hydrops has been associated with polyhydramnios (excess amniotic fluid) in humans[24]; although the exact mechanism in this case remains unclear, cardiovascular compromise and impaired venous or lymphatic drainage were hypothesized.

Fetal mouth was open with the tongue protruding in each transabdominal ultrasonography session. Although the clinical implication was unclear at the time, necropsy revealed craniofacial defects consistent with the ultrasonographic findings. This case is an additional report of the association between hydrops and fetal abnormalities. A case of hydrops amnion described in a Welsh pony was attributed to fetal brachygnathia impeding normal fetal swallowing.[8] Additional cases described a fetus with a congenital diaphragmatic hernia and delivery of a fetal monster.[9,26] These malformations are hypothesized to preclude swallowing or intestinal transport of amniotic fluid, as fetal swallowing is an important pathway for amniotic fluid resorption.[8,27] However, experimental studies in sheep have refuted this mechanism as a primary cause of hydramnios.[28]

We also examined the potential association between advanced reproductive techniques (e.g. in vitro embryo production) and the occurrence of hydrops, as these techniques are growing in popularity in commercial equine reproduction. In cattle, an increased incidence of hydrallantois has been documented in in vitro-produced (IVP) calves.[29,30] Furthermore, increased percentage of congenital malformations were observed in calves derived from IVP embryos (3.2%) compared to calves from in vivo-derived embryos following artificial insemination (0.7%).[29] To the authors' knowledge, this is the third case of hydrops in a recipient mare carrying an embryo produced via ICSI.[13,31] One case did not involve apparent fetal abnormalities, but similarly suspected that both allantoic and amniotic cavities were affected.[13] The other case reported hydrops in conjunction with monozygotic twins following a single IVP equine embryo transfer.[31] Given the small number of cases, it is difficult to even speculate on the relationship between hydrops and IVP embryos. However, as the number of IVP embryos increase in mares, the prevalence of pregnancy complications and fetal abnormalities should be monitored.

Mare was retired and adopted as a riding horse following discharge from the hospital; decision to retire was based on the possibility of this mare developing hydrops or other complications in future pregnancies. Literature does not support this notion and on the contrary has reported normal pregnancies following hydrops:[1,12] 95% (20/21) of mares produced future foals and 75% delivered a live foal the year after hydrops management.[1] However, given the delicate nature of embryo transfer, mares with a history of reproductive pathology are generally removed from the recipient population.

Conflict of interest

None to report.

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