



Case Report

Metastatic Tumor in Pregnancy: Placental Germ Cell Tumor With Metastasis to the Foal



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ABSTRACT

A placental mass was observed in an otherwise-healthy Quarter Horse mare; subsequently, the mare's 52-day-old foal was examined because of hind limb ataxia, urinary incontinence, and raised lesions of the distal limbs. Clinical and biochemical findings were supportive of liver disease and lumbosacral injury. Ultrasonographic evaluation of the abdomen suggested a liver mass, which was confirmed with computed tomography (CT) and determined to be neoplastic via histopathologic evaluation of a liver biopsy sample. Initial histopathology suggested a germ cell tumor. Regions of lysis affecting both femurs and third metacarpal bones and a complete oblique sagittal fracture through the body of the first sacral vertebra were present on CT. Supportive care was provided until CT confirmation of diffuse hepatic neoplasia and vertebral fracture. Necropsy revealed a large multinodular mass within the liver and a pathologic fracture of the first sacral vertebral body. Histopathologic comparison between neoplastic cells examined from the placenta and foal confirmed metastases of a germ cell tumor. Placental tumors are rare in all species; however, when noted in horses, clinicians should be aware of the possibility of metastatic spread to the foal.

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1. Introduction

Neoplasia in the horse is uncommon, and the finding of neoplasia in a young foal is extremely rare. Similarly, although masses in the placenta of mares are seen with a comparatively higher frequency, they are typically benign in nature; only three reports have documented placental neoplasia in the horse [1–3]. This report describes the history, presentation, diagnostics, and outcome of a mixed germ cell tumor in a foal that likely metastasized from the placenta.

2. Case History

A 4-hour-old 54-kg (118 lb) Quarter Horse filly born to a recipient mare was presented to the Lloyd Veterinary Medical Center for diarrhea since birth. Clinical findings included tachycardia (120 bpm), dehydration (approximately 5%), and diarrhea. Omphalophlebitis was diagnosed based on gross and ultrasonographic appearance. After 5 days of supportive care, the filly was discharged as the diarrhea had resolved, and the remainder of the physical examination was unremarkable. During hospitalization, routine postpartum examination of the mare's reproductive tract was unremarkable. However, the placenta was markedly distorted by numerous multifocal, nodular, friable, firm-to-mineralized, red-to-yellow masses ranging in size from 1 to 14 cm (Fig. 1) located on the fetal surface of the chorioallantois and allantoamnion [4]. The largest focal nodular mass (14 cm) was located at the junction of the

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Fig. 1. Gross image of the placenta; note the numerous multifocal, nodular, friable, firm-to-mineralized, red-to-yellow masses ranging in size from 1 to 14 cm located on the fetal surface of chorioallantois and allantoamnion.

umbilical vasculature and chorioallantois, surrounded by numerous similar but smaller nodular masses. On the basis of the histologic appearance and immunohistochemistry results, a diagnosis of a germ cell tumor was made.

2.1. Initial Clinical Findings

The foal was reported to be healthy until 52 days of age at which point she presented for a 5-day history of urine dribbling, intermittent fever, decreased nursing, hind limb weakness, prolonged periods of recumbency, and difficulty rising. The referring veterinarian attributed the urine dribbling to a suspected urinary tract infection and administered one dose of ceftiofur crystalline-free acid (dose unknown) the day before presentation and daily flunixin meglumine (dose unknown) for the 5 days prior. On presentation (day 1), the foal (114 kg; 251 lb) was quiet, but alert and responsive, with a rectal temperature of 38.9°C (102.0°F), heart rate of 120 beats/min, and respiratory rate of 60 breaths/min; the foal was estimated to be 5% dehydrated. The foal was able to stand and walk, but severe bilateral ataxia and weakness (4/5) was present in the hind limbs. Tail tone was absent, and urine was noted dribbling from the vulva. Anal tone was present but depressed. The cranial nerves were intact, panniculus reflex was present bilaterally through all dermatomes, and no obvious neurologic deficits were noted in the thoracic limbs. Neurologic deficits were localized to the lumbosacral region, but no external evidence of trauma was present. Irregular raised lesions on the lateral aspect of the distal third metacarpal bone of the right limb and lateral portion

of the distal third metatarsal bone of the left limb were also present. No heat was associated with these lesions, but pain was elicited with firm digital pressure.

2.2. Initial Diagnostics and Therapy

Complete blood count revealed anemia (hematocrit, 25.1%; reference interval, 31%–44%), mature neutrophilia (10.03×10^9 cells/L; reference interval, $2.7\text{--}9.46 \times 10^9$ cells/L), and monocytosis (0.88×10^9 cells/L; reference interval, $0.05\text{--}0.61 \times 10^9$ cells/L). Serum biochemistry analysis revealed increased activity of creatine kinase (CK, 1,143 IU/L; reference interval, 74–426 IU/L), aspartate aminotransferase (AST, 985 IU/L; reference interval, 282–484 IU/L), and gamma glutamyl transferase (GGT, 95 IU/L; reference interval, 8–38 IU/L). Hyperbilirubinemia (58.14 $\mu\text{mol/L}$; reference interval, 8.55–34.2 $\mu\text{mol/L}$) and hyperlactatemia (4.8 mmol/L; reference interval, <2.5 mmol/L) were also present. Resuscitative IV fluid therapy was initiated with a 2-L bolus of isotonic fluids followed by 1-L boluses of isotonic fluids IV, every 2 hours (105 mL/kg body weight (BW)/d). Gentamicin (6.6 mg/kg BW IV, every 24 hours), flunixin meglumine (1.1 mg/kg BW IV, every 24 hours), and omeprazole (4 mg/kg BW PO, every 24 hours) were also administered.

2.3. Case Progression

The foal remained comfortable and on day 2 was able to occasionally rise on her own to nurse; however, neurologic status was unchanged. Lateromedial and dorsoventral radiographs were performed of the thoracic, lumbar, and sacral spine, revealing no evidence of vertebral fracture or malformation. Cerebrospinal fluid (CSF) was collected under light sedation (butorphanol tartrate, 0.04 mg/kg BW and diazepam, 0.04 mg/kg BW IV) from the lumbosacral space and revealed marked elevation in total protein (214 mg/dL; reference interval, 32–48 mg/dL), pleocytosis (total nucleated cell count, 30 cells/ μL ; reference interval, ≤ 5 cells/ μL), and elevated red blood cells (RBCs) (1,360 cells/ μL ; reference interval, 0 cells/ μL). Cytologic evaluation of the CSF noted 66% neutrophils and 34% large mononuclear cells; large mononuclear cells occasionally contained phagocytized RBCs and cellular debris, consistent with pathologic hemorrhage. Cerebrospinal fluid was submitted for culture but yielded no bacterial growth. Increased activity of CK (4,828 IU/L), AST (1,088 IU/L), GGT (151 IU/L), and sorbitol dehydrogenase (18.1 IU/L; reference interval, 1.1–4.6 IU/L) were present along with hyperbilirubinemia (57.80 $\mu\text{mol/L}$), hypoalbuminemia (23 g/L; reference interval, 33–46 g/L), and hypoproteinemia (49 g/L; reference interval, 52–65 g/L) were measured on day 2.

Because of the persistent elevations in hepatobiliary enzymes, an ultrasonographic examination of the abdomen was performed. Ultrasonographic abnormalities were limited to the liver, revealing numerous round heterogeneous masses of varying size diffusely distributed throughout the liver parenchyma; many of the masses were characterized by a thick hyperechoic rim that produced distal acoustic shadow artifact, consistent with mineralization, whereas the central portions were either

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