Veterinary Medical Teaching Hospital
Texas A&M University
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Dear Mrs. Nuttall,

Below is a summary of the evaluation and diagnosis of your foal. Please let us know if you have any further questions or concerns.

Dorothee 18F, a Warmblood colt, was born the morning of 2/25/18. The foal was apparently bright and alert after birth and had 16 oz of colostrum reportedly the morning he was born. The foal had severe tendon laxity and appeared dysmature, so he was brought to Texas A&M for further workup and care.

On presentation, the foal was dull and recumbent. His temperature was 101*F. His heart rate was 150 beats per minute with a physiologic flow murmur present. His respiratory rate was 40 beats per minute and lung fields were clear bilaterally with no crackles or wheezes ausculted. Gastrointestinal sounds were present and normal in all abdominal quadrants. The colt had full thickness abrasions/lacerations on all four fetlocks, and his limbs were hypothermic. The coronary bands and mucous membranes were hyperemic and her sclera were severely injected bilaterally. The cartilage within the ears and the nose appeared underdeveloped. No effusion was noted in any of the joints, although several of the lacerations over the fetlocks were directly over the joint and neurovascular bundle of the limbs. His umbilicus had no heat or swelling.

Radiographs taken of the carpi and tarsi on presentation showed normal ossification of the cuboidal bones. A complete blood count and a serum biochemistry showed minor changes that were not clinically significant.

The foal was treated aggressively with intravenous fluids, hyperimmune plasma, broad spectrum antimicrobials, anti-inflammatory medications, oxygen therapy, nutritional support, wound care and bandaging. In order to keep the foal quiet and not to exacerbate the leg wounds, he was started on continuous sedation. Over the first 24 hours, the foal was still not adequately perfusing blood to his distal limbs so an inotropic drug was started.

Unfortunately, the foal did not respond to aggressive treatment. Due to the extreme fragility of the foal's skin and the concern of sepsis the foal was humanely euthanized. The foal was tested for the genetic defect that causes Warmblood Fragile Foal Syndrome. Post-mortem examination confirmed a diagnosis of Warmblood Fragile Foal Syndrome. Histopathology revealed a spectrum of non-specific changes which in part would seem to reflect recumbency and/or an association with the evident skin damage.

There are changes in the size (reduced) and arrangement (disorganized) of the dermal collagen fibers. These would ideally be assessed in conjunction with a series of sections from an aged-matched control. Nonetheless, they are supportive for a diagnosis of a collagen dysplasia. The lungs were only partially inflated and demonstrate evidence of fetal distress and amnionic aspiration, confirming the clinical suspicion of fetal hypoxia and potential "dummy foal" syndrome. The omphalophlebitis is mild and likely clinically irrelevant with the inflammation limited to the distal portions of the umbilical vessels and the adjacent tissue.

Please let us know if you have any further questions regarding the care and diagnosis of your foal. We are sincerely sorry for the loss of your foal. Thank you for entrusting Texas A&M University.

Kind Regards,
Michelle Coleman, DVM, PhD, DACVIM