A Filly Born At 280 Days of Gestation: Management, Complications and Final Outcome

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ABSTRACT

A mix breed filly was born after only 280 days of gestation in a spontaneous delivery. The filly was small, had a weak suckle reflex, very thin hair coat, severe tendon laxity and carpi valgus but was bright and alert and without respiratory difficulties. The filly was treated with plasma and prophylactic antibiotics and was fed via an indwelling naso-esophageal tube until she was able to suckle from the mare at the age of 2 weeks. The cause for the premature parturition was not determined, however, placentitis was highly suspected due to the apparent precocious *in utero* maturation of the filly that allowed for her survival without severe complications during the neonatal period. The filly consequently suffered from a traumatic fracture of the third metatarsal bone in the left hindlimb at the age of 3 weeks, which was treated conservatively with external fixation and healed. As a yearling, she developed severe degenerative joint disease in the left shoulder joint which was attributed to osteochondral changes that may have been related to her prematurity. The degenerative changes and chronic severe left forelimb lameness resulted in severe angular limb deformities in both front limbs which eventually led to the euthanasia of the filly at the age of 2.5 years. This case may suggest that severe developmental orthopedic deformities may be a late sequel of prematurity that may eventually hinder the long term survival.

Keywords: Foal, Premature, Placentitis, Developmental Orthopedic Diseases, Angular Limb Deformity.

INTRODUCTION

Prematurity in foals is a well recognized problem encountered by equine practitioners. It is usually marked by incomplete development of different organs and physiological functions. Normal gestation length in mares has a very wide range. Several studies investigated different breeds and different factors affecting this period. The acceptable mean gestational length in mares is 340-345 days with a normal range of 320-370 days (1, 2, 3), although other studies have shown a range as wide as 315-388 days (4). Nevertheless, an acceptable definition of prematurity in foals is having less than 320 day's gestational age (1). Foals born before 320 days of gestation are usually given an unfavorable prognosis for survival (5). The main problems, which limit the survival of premature foals, are lack of full maturity of the respiratory, gastrointestinal and nervous systems and deficient homeostasis function that affects the adjustment to the environment outside the uterus. Other problems include various limb deformities, low birth weight, thin hair coat and weak suckle reflex (1,2). Reports of premature viable foals born before 300 days of gestation are very scarce (6). The case presented hereby describes the spontaneous delivery of a viable filly at 280 days of gestation, the management, treatments, and complications she had endured as well as the final outcome.

CASE REPORT

A 6-year-old mixed-breed mare of unknown parity gave birth to a live filly at 280 days of gestation (August 9th 2005 to May 16th 2006). The presence of the pregnancy was first confirmed by transrectal ultrasound on day 19 post breeding and again at 80 days of gestation. A few days prior to parturition, the mare developed a full size udder and on the day of delivery, milk dripping was observed. The owners did not report any problems with the mare during the pregnancy.

The parturition was uneventful and was witnessed by the owners. The filly stood up but did not suckle from the udder and thus was given a small amount of milk through a bottle. The attending practitioner (the author) was summoned 8 hours after parturition to treat the filly. The placenta was discarded by the owners and was not available for examination. The filly was very small and lean, with very thin silky hair coat; she had severe tendon laxity of all four limbs, valgus in both carpi, reddish colored tongue and a weak suckling reflex. Nevertheless, she was bright and alert, was able to stand up unassisted and her head was not domed shaped. There did not seem to be any respiratory distress but the owners were informed of the unfavorable prognosis for survival. Due to financial constraints, the filly was not referred to an intensive care facility for further treatment and remained at the care of the owners. The mare had a fully sized udder with abundant milk production; however, the colostrum seemed to be of poor quality or nonexistent on gross examination (white and dilute milk). The filly received 2 liters of equine fresh frozen plasma intravenously, and was treated with broad spectrum antibiotics (procaine penicillin, Norocillin Veterinary, Norbrook Laboratories Ltd., Newry, Ireland. 20,000 U/kg bwt, intramuscularly, twice daily) and gentamicin, Gentaject 50, Eurovet Animal Health BV, Bladel, Netherland, 6.6 mg/kg bwt, intramuscularly, once daily). Furthermore a small diameter indwelling naso-esophageal feeding tube (12 Fr, 108 cm. MILA international Inc., Erlanger, Kentucky, USA) was inserted with endoscopic guidance. The forelimbs were put in full limb thick bandages and plastic splints were applied to the palmar aspect to treat the carpi valgus (suspected incomplete ossification of the small carpal bones). Soft bandages were applied to the hindlimbs in the pastern and fetlock regions to protect the skin against abrasions as a result of the tendon laxity (Figure 1). The filly was fed the mare's milk hourly through the feeding tube in increasing amounts. Because of the very thin hair, the skin was exposed to abrasion and at the end of the first day numerous superficial skin wounds had developed. These were treated topically with silver sulfadiazine cream (Silverol, Teva Pharmaceutical Industries Ltd., Petah Tikva, Israel). The filly seemed to be oversensitive to touch and would not tolerate being covered to protect her skin and body temperature. Meconium and urine were passed without incidence.

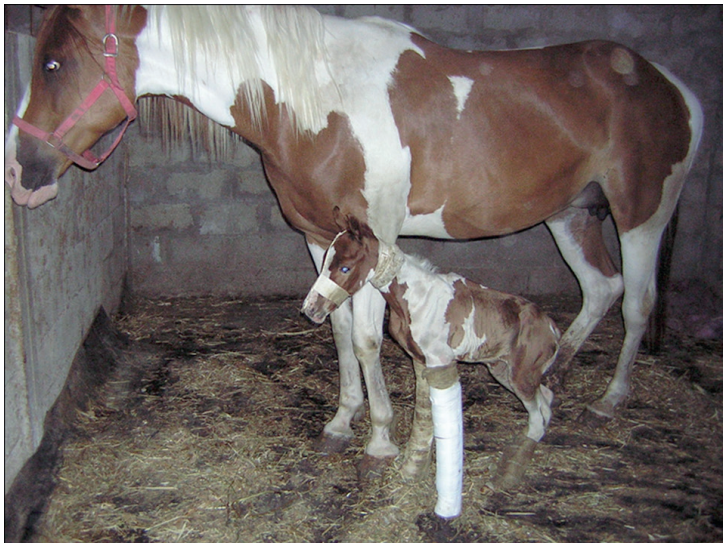

Figure 1: The filly at the age of 9 days. Notice the small body size compared to the dam, the hindlimb laxity and the splinted forelimbs.

After 5 days of steady improvement in strength and suckle reflex, while the filly was being fed by both bottle suckling and through the feeding tube, there was a sudden deterioration in her condition. She was slightly dehydrated and weak and urine was seen dripping from the umbilicus. After inquiry of the owners it appeared that the main cause for the dehydration was mistaken under-feeding. Blood was obtained for complete blood count. The only abnormality was leukocytosis ($22.4x10^3$ Leukocytes/μl, reference range $6\text{-}13x10^3$ Leukocytes/μl) with slight neutrophilic toxicity on the blood smear. With increased feeding her condition quickly improved. Povidone iodine solution (Polydine solution, Ben Shimon Floris Ltd., Misgav, Israel. 1%) was applied daily to the umbilicus and the urine dripping stopped after 5 days. Gentamicin was discontinued after 9 days and procaine penicillin after 14 days when most of the skin wounds had healed.

On day 12 the filly started suckling from the mare and was gradually weaned from the bottle. On day 14 she was completely self reliant for feeding. The bandages on the fore-

limbs were changed every 3-4 days and the splints were gradually removed after 14 days at which time the valgus and laxity had resolved. At this time her body condition improved with noticeable muscle mass gain and she weighed 25 kg.

When the filly was 23 days old the mare accidentally stepped on her left hindlimb. Physical examination and radiographs revealed a transverse non-comminuted fracture in the diaphesis of the third metatarsal bone with a 2 cm skin laceration located 4 cm proximal to the fracture line. Again, due to financial constrains, surgical repair of the fracture was declined by the owners. The wound was copiously lavaged with saline and routinely sutured. Under short general anesthesia, the fracture was reduced to near normal alignment and a thick bandage with dorsal and plantar plastic splints were placed distal the tarsus. The filly was treated with cephalexin (Cefovit forte, Vitamed Ltd., Binyamina, Israel. 20 mg/kg bwt, per os, three times daily), gentamicin (6.6 mg/kg bwt, intramuscularly, once daily), phenylbutasone (Eliezer Linevitz Ltd., Even-Yehuda, Israel. 2.2 mg/kg bwt, per os, once daily) and tetanus antitoxin 1500 U intramuscularly (Fort Dodge, Overland Park, KS, USA). The filly was able to bear weight comfortably on the splinted leg and was kept in stall rest. The bandages were changed once a week. The first 4 bandage replacements were conducted under heavy sedation to prevent movement and displacement of the fracture. After initial fracture stability was clinically achieved, bandages were replaced without sedation. There was no evidence of wound infection and the sutures were removed after 14 days. Gentamicin was discontinued after 13 days. Cephalexin was discontinued initially after 13 days but was reintroduced after 9 days to treat two pressure sores that developed on the medial and lateral aspects of the fetlock. The cephalexin treatment was discontinued after additional 14 days.

Fifty two days after the injury, the leg seemed to be stable and follow up radiographs showed a large callus around the fracture with some bone filling of the fracture line; however, there did not seem to be enough healing to allow removal of the splints. In the following weeks, the bandages were replaced every 7-10 days, the pressure sores healed and a single plantar splint was applied. After additional 52 days (3.5 months from the injury), follow up radiographs were taken. The callus looked smaller, more than 50% of the fracture line was filled with new bone and there was better alignment of the two fragments. At this stage, severe flexural laxity had developed due to the long term limb fixation, therefore, the bandages and splint were gradually removed over the next 4 weeks at which time there was marked improvement in the laxity and the filly was able to walk and trot without lameness.

OUTCOME

The filly continued to grow and develop at a rate that was compatible with that of another filly residing in the stable, which was of the same age, although she did seem to be somewhat smaller than expected from the size of her parents and her age. Left forelimb lameness at the walk was noticed at the age of 12 months. No improvement was seen with rest and phenylbutazone treatment and she was referred to the Koret School of Veterinary Medicine – Veterinary Teaching Hospital (KSVM-VTH) for further diagnosis. Physical examination, peripheral nerve blocks and intra-articular anesthesia localized the source of the lameness to the left scapulohumeral (shoulder) joint. Radiographs revealed joint dysplasia, flattened gleoid cavity surface, irregular joint surface and osteophytes in the distal scapula. In addition, physitis with new bone formation was observed in the head of the humerus (Figure 2). Diagnosis of severe degenerative changes caused by several developmental osteochondral ab-

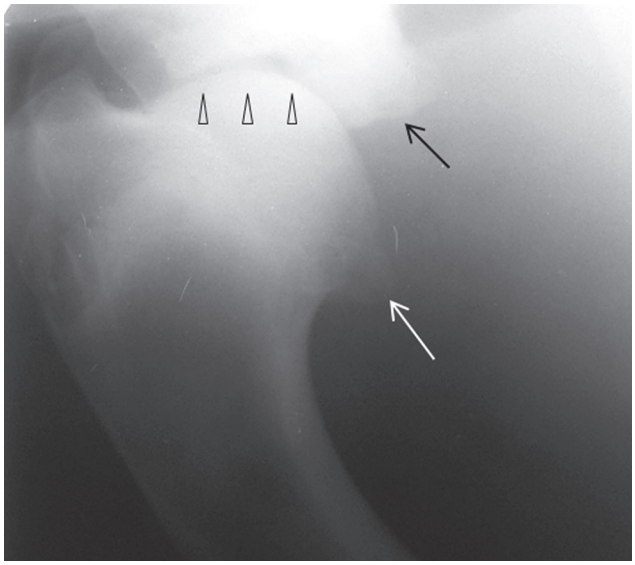

Figure 2: Medial to lateral radiograph of the left shoulder joint taken under general anesthesia at the age of 12 months at the KSVM-VTH. The black arrow indicates the osteophytes in the distal scapula, the white arrow points at the physitis with abnormal bone production in the head of the humerus and the arrow heads indicate the joint incongruity and irregular joint space.

normalities was made which warranted a guarded prognosis. The joint was injected with sodium hyaloronate (51 mg) (HY-50, Bexco Pharma Inc., Mississauga, Canada), triamcenolone (Trigon, Bristol-Myers Squibb, Madrid, Spain. 20 mg) and amikacin (250 mg, Amikacin-Fresenius, Bodene (PTY) Ltd., Port Elizabeth, South Africa). Temporary clinical improvement was observed, however, within 2 weeks the lameness resumed and gradually worsened. In the following months, as the lameness continued, severe angular limb deformities developed in both forelimbs with superficial and deep digital flexure tendons contraction combined with fetlock and carpi varus, which was most severe in the right front leg (Figure 3). The filly was eventually euthanized at the age of 2.5 years due to the severe lameness and limb deformities.

DISCUSSION

To the best of the author's knowledge, this is the first detailed published case report of a viable foal born after only 280 days of gestation. Conventionally, a foal born with less than 320 days of gestation is considered premature with the complex problems associated with this condition (1). Occasionally, foals born between 300 and 320 gestation days can be relatively normal if early maturation occurs *in utero* (2, 7, 8). Rossdale and Silver (6) reported a viable foal born after 281 days of gestation to a mare that suffered from placentitis, however, no data was provided except for the physical and hematological parameters, the response to ACTH stimulation at the age of 12 hours, and the fact that the foal had survived the neonatal period.

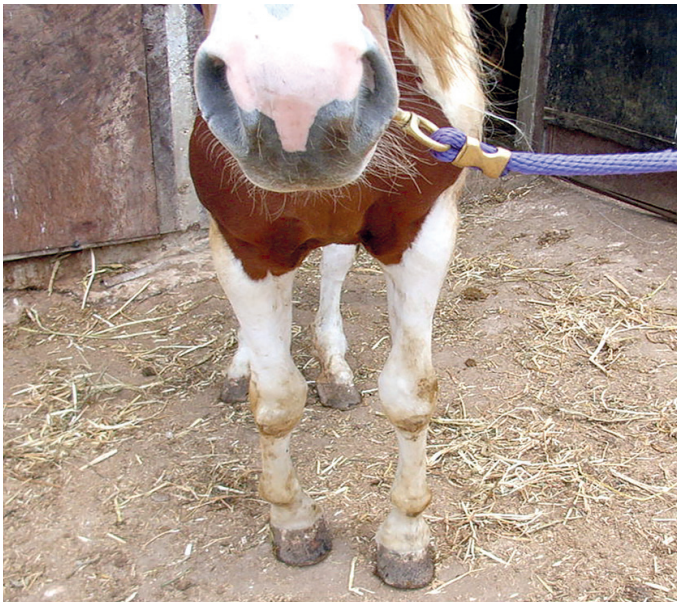

Figure 3: The filly at the age of 23 months. Notice the fetlock joint varus and flexural contracture in both forelimbs, more severe on the right side.

The filly described here had many of the physical characteristics of prematurity such as low body weight, silky hair, severe laxity of limbs and ears, angular limb deformities, red colored tongue and a poor suckle reflex. Other, more debilitating characteristic features, were not observed. There was no respiratory distress at any point, no gastrointestinal abnormalities and there did not seem to be any severe endocrine or metabolic abnormalities (although specific diagnostic blood tests were not conducted). Moreover, although she did not receive colostrum in the first few hours and plasma treatment was delayed for more than 8 hours, she did not show any signs of severe infection or sepsis. The development of umbilical urine dripping, evidence of a patent urachus, on day 5 was probably caused by localized infection and not a sign of generalized infection or sepsis since the urine dripping resolved within 5 days and no other organs showed signs of infection (9). It may be concluded that the main reason for the filly's survival, besides the supportive treatment and the owners' devoted commitment, was that there was sufficient accelerated and precocious *in utero* maturation preceding the early parturition.

Causes of spontaneous premature parturition in mares include placental infection (placentitis), placental insufficiency, placental edema or premature separation, twinning, systemic illness of the mare and other causes of stress such as general anesthesia (2, 10). Premature udder development and lactation, with consequent early parturition are characteristics of placentitis. Other clinical signs of placentitis include vaginal discharge and rarely abdominal discomfort and fever (8, 11). Although the owners did not observe any abnormalities during the pregnancy and ultrasonographic evaluation of the placenta was not conducted during the later stages of the pregnancy, placentits was the most likely cause for the premature parturition in this case. Live foals born to mares affected by placentitis are commonly born weak and unthrifty and may develop sepsis or hypoxic ischemic encephalopathy ("dummy foal") (11). Contrary to these compromised foals, the unfavorable intra-uterine conditions caused by the infection and inflammation due to placentitis have been shown to prompt early maturation of the hypothalamus-pituitary-adrenal axis and

of lung function in the fetus and this accelerated development may have accounted for the described filly's post parturient thrive and survival (2, 6, 7, 11, 12). Unfortunately, the placenta was discarded after parturition and was unavailable for examination, thus, definitive diagnosis of placentitis was not achieved.

The left forelimb lameness that the filly developed as a yearling was attributed to the complex of developmental orthopedic diseases (DOD) (13) or the newly defined Juvenile osteochondral conditions (JOCC) (14). The radiological abnormalities could be attributed to osteochondritis dissecans (the flattened gleoid cavity surface, irregular joint surface and the osteophytes in the distal scapula). The new bone production in the humeral head was most likely due to severe and chronic physitis. Developmental disorders (DOD or JOCC) of the shoulder joint are well recognized in weanling and yearling foals (13, 14, 15). The abnormal cartilage and subchondral bone development results in an abnormally shaped humeral head and glenoid cavity contour which cause joint instability and lead to secondary degenerative changes. Suspected causes for DOD/JOCC are rapid growth, dietary imbalances, trauma, under or over exercise at a young age, anatomical abnormalities and genetic predisposition (15, 16, 17). Excessive loading of the forelimbs due to the hindlimb fracture in the described filly is a possible but not very likely cause for the forelimb lameness since the hindlimb injury occurred several months prior to the appearance of the forelimb lameness. In addition, the filly was not severely lame throughout the recuperation period with the aid of the hindlimb external fixation. One of the above factors could have been the cause of the DOD in the described filly, however, a causal relationship to her prematurity may be suspected as well. Prematurity has not been previously regarded as a predisposing factor for abnormal cartilage and subchondral bone development leading to osteochondritis dissecans or other DODs. Nevertheless, this case may suggest that the abnormal pressure and stress applied to the incompletely developed joints as a result of the "unscheduled" and premature weight bearing could be a contributing factor. The severe laxity and the carpi valgus that were observed at birth and were related to the prematurity (18), although treated and resolved, may have been contributing factors as well.

The acquired angular limb deformities that developed at a later stage were most likely as a result of the chronic left forelimb lameness that caused overloading of the contra-lateral limb and abnormal weight-bearing in the affected limb (19). The combination of the refractory left forelimb lameness and the severity of the acquired limb deformities indicated a poor prognosis and the filly was eventually euthanized. Although the filly survived the neonatal period without severe complications related to her prematurity, the possibility of association between the prematurity and the development of the severe later skeletal abnormalities indicate that owners should be advised of potential long term complications for premature foals that are not noticed at birth or in the neonatal period.

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