Hyperplasia of the thyroid gland and musculoskeletal deformities in two equine abortuses

Andrew L. Allen

A syndrome of neonatal foals characterized by hyperplasia of the thyroid gland and multiple congenital musculoskeletal deformities, which was described by McLaughlin and Doige in 1981 (1), continues to be an important cause of reproductive loss and foal mortality in western Canada (2–5). The most consistent musculoskeletal lesions include mandibular prognathia, inappropriately ossified (immature or hypoplastic) carpal and tarsal bones, flexural deformities of the forelimbs, and ruptured tendons of the common digital extensor muscles. Between 1980 and 1989, foals with lesions consistent with this syndrome, referred to as either thyroid hyperplasia and musculoskeletal deformities (TH-MSD) (4) or congenital hypothyroidism and dysmaturity (CHD) (5), were found in 2.7% of all perinatal foals submitted to the 8 veterinary diagnostic laboratories in western Canada (4). The TH-MSD syndrome has only been reported in foals at full term, i.e., stillborns and dead neonates, and is often associated with prolonged gestation (4,5). Despite a normal to long gestation, foals with the TH-MSD syndrome have signs of immaturity, which may include a short, soft ("silky") coat; pliable ears; lax tendons and joints; incomplete closure of the abdominal wall; and immature carpal and tarsal bones.

Farm 1 — Case 1

On March 19, 1993, an Arabian horse breeder near Calgary, Alberta, reported that one of his 4 pregnant mares had aborted. She was 1 of 2 mares on the farm that had produced a TH-MSD syndrome foal the previous year. She had been bred naturally under the supervision of the owner on June 26 and 27, 1992, and, therefore, aborted at approximately 265 d gestation. She had been vaccinated against equine influenza, eastern and western equine encephalomyelitis, and tetanus in January 1993. Other than foals born on the farm, no horses had been introduced to the herd for several years. One horse left the farm during the summer of 1992 and returned within a few days. All the mares remained bright and alert, continued to eat, and were considered to be in good health before and after the abortion.

The male fetus (E93-237) had a crown-to-rump length of 87 cm; eyelashes and hair on the mane and tip of the tail; short, fine hair present diffusely over the head and distal portions of the limbs; scant, short, very fine hairs

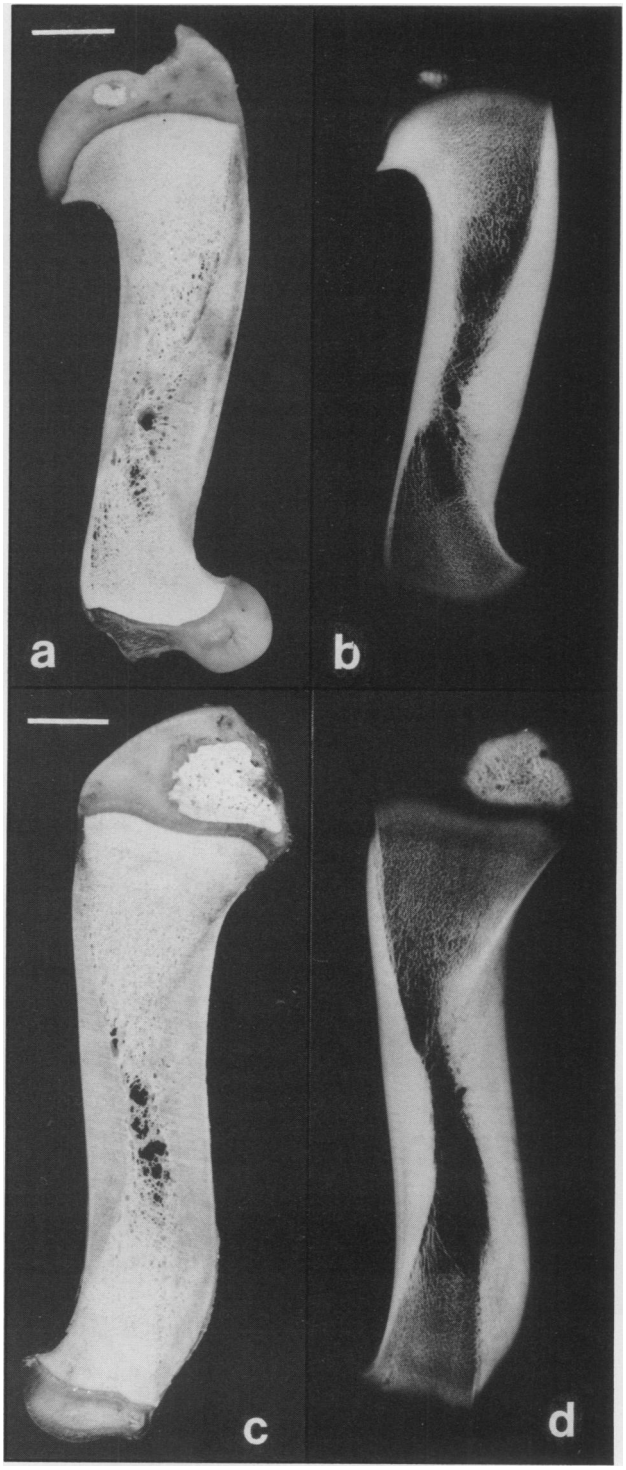

Figure 1. Gross (a and c) and radiographic (b and d) preparations of midsagittal, 3 to 4 mm thick, slab sections of a humerus (a and b) and a femur (c and d) from an equine abortus (N93-2098) with the TH-MSD syndrome. There are markedly thickened cortices, a marked increase of cancellous bone, and a near absence of the marrow spaces, indicative of osteopetrosis. Bar = 2 cm.

Can Vet J 1995; 36: 234–236

Department of Veterinary Pathology, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N 0W0.

The investigation of these cases was part of a larger study funded by the Standardbred and Thoroughbred Divisions of the Alberta Racing Commission Development Fund and the Alberta Agricultural Research Institute. The author was supported by an Interprovincial Graduate Student Fellowship.

Table 1. Features of the history and physical or postmortem examination of 2 abortuses and 2 neonatal foals with hyperplasia of the thyroid gland and congenital musculoskeletal anomalies

	Farm 1		Farm 2	
Feature	E93-237[a]	CE-02	N93-1686	N93-2092[a]
Gestation (days)	265	333	348	318
Sex	male	unknown	female	female
Short, fine ("silky") hair coat	NA[b]	yes	yes	NA[b]
Soft, pliable ears	NA[b]	yes	yes	NA[b]
Degree of mandibular prognathism (mm)	5	12	20	18
Degree of flexural deformity of the front legs	moderate	mild	marked	none
Incomplete closure of abdominal wall	NA[b]	yes	yes	NA[b]
Ruptured CDETs[c]	no	no	yes	no
Immaturity of the carpal and tarsal bones	NA[b]	mild	marked	NA[b]
Degree of osteopetrosis	moderate	unknown[d]	marked	marked
Degree of thyroid gland hyperplasia	moderate	unknown[d]	marked	marked

[a]Abortuses
[b]NA indicates that a given feature was expected in a preterminal fetus and, therefore, evaluation is not appropriate with regard to the TH-MSD syndrome
[c]Tendons of the common digital extensor muscles
[d]The foal remained alive and, therefore, tissues were not available for further examination

present over the body, consistent with a fetus at 240 to 270 d gestation (6). The major pathological findings are presented in Table 1. The diagnosis of flexural deformities of the fore legs was based on the marked resistance to manual extension, which was in contrast to the free range of motion and marked joint laxity typically found in preterminal foals and which was present in other areas of the legs, neck, and head of this fetus.

Radiographic and gross examination of the limb bones demonstrated a pattern of ossification consistent with a fetus that was between 250 and 275 d gestation (7). Midsagittal sections of the humeri and femora revealed cortices that were moderately thickened, had a moderately increased amount of metaphyseal and diaphyseal cancellous bone, and had a corresponding reduction in the marrow space. The thyroid gland was normal in size and shape. However, histologically, there was no colloid and the gland was composed of small, tightly packed follicles with short, plump, columnar epithelial cells, consistent with a diagnosis of hyperplasia (8,9). Histological examination of a variety of other tissues did not reveal any abnormalities.

The placenta was examined, found to be free of obvious lesions, and considered to be normal for one at 265 d gestation (10). No previously described cause of abortion was recognized in this case.

Farm 2 — Case 2

On April 30, 1993, a standardbred horse breeder near Edmonton, Alberta, reported that 1 of his 3 pregnant mares had aborted. The mare was found in lateral recumbency, straining, with the placenta protruding from the vulva. A local veterinary practitioner examined the mare and found a dead fetus engaged in the pelvic canal in a posterior presentation and right-dorsal posture. The veterinarian believed the fetus was freshly dead, as opposed to being dead and autolysed in utero.

The aborting mare had last been bred, naturally, on June 16, 1992, and, therefore, aborted at approximately 318 d gestation. None of the mares had been vaccinated within the previous year. All 3 mares had been moved to other farms and, thus, in contact with other horses during their pregnancies. All the mares remained bright and alert, continued to eat, and were considered to be in good health before and after the abortion.

The female fetus (N93-2092) had a crown-to-rump length of 100 cm; weighed 37.5 kg; had hair typical for a newborn foal over the head and limbs, but short, fine hair over the body; and had soft, pliable ears, consistent with a fetus at 300 to 330 d gestation (6). The pertinent pathological findings are given in Table 1.

Radiographic and gross examination of the limb bones revealed a pattern of ossification consistent with a fetus that was between 290 and 335 d gestation (7). Midsagittal sections of the humeri and femora showed cortices that were markedly thickened, particularly through the diaphyses; a marked increase in the amount of metaphyseal and diaphyseal cancellous bone; and a near absence of the marrow space (Figure 1).

The thyroid gland was normal in size and shape, but histologically, it was extremely cellular and devoid of colloid. The follicles were small, or collapsed and irregular with barely discernable lumina, and were composed of large cuboidal to short columnar epithelial cells. This appearance was consistent with a diagnosis of hyperplasia. Histological examination of a variety of other tissues did not reveal any abnormalities.

The placenta was examined, found to be free of obvious lesions, and considered normal for one at 318 d gestation. No previously described cause of abortion was recognized in this case.

The 3 other pregnant mares on Farm 1 produced live foals during May and June 1993. One of the foals (CE-02) was diagnosed as having the TH-MSD syndrome, based on a full gestational period and the presence of multiple anomalies consistent with the disease, as outlined in Table 1. The 2 remaining pregnant mares produced normal foals.

The other 2 pregnant mares on Farm 2 produced live foals that died shortly after birth. The 1st of these mares

produced a TH-MSD syndrome foal (N93-1686). The mare exhibited mammary development and filling of the udder, waxing at the end of the teats, and leaking of milk on March 6, at 323 d gestation. However, the foal was not delivered until March 31, at 348 d gestation. The foal never stood, did not suckle, and died the night of April 1, 1993. The postmortem findings were consistent with the TH-MSD syndrome and are summarized in Table 1. The 3rd mare foaled on May 3, 1993, after a 343 d gestation. This 3rd foal was weak and lethargic from birth, had excessive tendon and joint laxity in the limbs, and died prior to 2 d of age. However, it did not have other lesions consistent with the TH-MSD syndrome.

Delayed development of externally visible adnexal structures and the skeletal system could not be detected in the 2 abortuses presented here. This may be because there was no delay in their development or, possibly, because the criteria for assessing equine fetal maturation prior to a spontaneous delivery after 320 d gestation are very imprecise. However, the history of other foals on the same farms suffering with the TH-MSD syndrome and the postmortem findings of hyperplasia of the thyroid gland, mandibular prognathia, osteopetrosis, and, in 1 of the abortuses, flexural deformities of the forelimbs support a diagnosis of TH-MSD. No other cause for the abortions was detected and no other abortions occurred in either herd.

I believe that these abortions represent the first described in association with the TH-MSD syndrome. A recently published retrospective study (4) found that 2.7% (79/2946) of all perinatal equine submissions to veterinary diagnostic laboratories in western Canada had lesions consistent with the TH-MSD syndrome. However, no abortuses were present in this group, so one could conclude that over 4.5% (79/1740) of full term foals submitted for necropsy had lesions consistent with the TH-MSD syndrome. The incidence of perinatal loss associated with the TH-MSD syndrome could have been higher than 4.5% if the syndrome was associated with abortion, but not recognized. The thyroid glands of these 2 fetuses were grossly normal, but had moderate to marked microscopic lesions of hyperplasia. This emphasizes again the importance of microscopic examination of the thyroid gland of any perinatal horse examined postmortem in which impaired in utero thyroid gland function is suspected (4,9). Veterinarians and pathologists should be cognizant of the TH-MSD syndrome when investigating morbidity and mortality in neonatal foals, stillbirths, and abortions. CVJ

References

1. McLaughlin BG, Doige CE. Congenital musculoskeletal lesions and hyperplastic goitre in foals. Can Vet J 1981; 22: 130–133.
2. McLaughlin BG, Doige CE, McLaughlin PS. Thyroid hormone levels in foals with congenital musculoskeletal lesions. Can Vet J 1986; 27: 264–267.
3. Kreplin C, Allen A. Congenital hypothyroidism in foals in Alberta. Can Vet J 1991; 32: 751.
4. Allen AL, Doige CE, Fretz PB, Townsend HGG. Hyperplasia of the thyroid gland and concurrent musculoskeletal deformities in western Canadian foals: Reexamination of a previously described syndrome. Can Vet J 1994; 35: 31–38.
5. Allen AL, Doige CE, Fretz PB, Townsend HGG, Card CE. Congenital hypothyroidism, dysmaturity and musculoskeletal lesions in western Canadian foals. Proc 39th Annu Conv Am Assoc Equine Pract 1993: 207–208.
6. Roberts SJ. Veterinary Obstetrics and Genital Diseases. 3rd ed. Woodstock, Vermont: Roberts, 1986: 27.
7. Getty R. Osteology. In: Getty R, Rosenbaum CE, Ghoshal NG, Hillman D, eds. Sisson and Grossman's The Anatomy of the Domestic Animals. 5th ed. Vol 1. Toronto: WB Saunders, 1975: 255–348.
8. Capen CC. The endocrine glands. In: Jubb KVF, Kennedy PC, Palmer N, eds. Pathology of Domestic Animals. 4th ed. Vol 3. Toronto: Academic Press, 1993: 267–337.
9. Doige CE, McLaughlin BG. Hyperplastic goitre in newborn foals in western Canada. Can Vet J 1981; 22: 42–45.
10. Whitwell KE, Jeffcott LB. Morphological studies on the fetal membranes of the normal singleton foal at term. Res Vet Sci 1975; 19: 44–55.