Multiple nonlethal congenital anomalies in a llama

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In December, 1986, an approximately 7-month-old sexually intact male llama was presented to the teaching hospital because of chronic hind limb lameness. The llama had bilateral carpus and tarsus valgus. In addition to cow-hocked appearance, the angles of the articulations of the hock joints were more acute than normal, resulting in sickle-hocked conformation. Weakness in the hocks was further evidenced by considerable laxity in the joint associated with placement of the hind foot while walking, as though the llama were unable to fully support the weight of its hindquarters. Further examination detected pronounced fremitus, especially over the left hemithorax. A grade V/VI holosystolic murmur was auscultable.

Radiography indicated that the tibial tarsal (talus) bones in both tarsi were rotated craniad, approximately 90° (Fig 1). Normally, the proximal aspect of the talus forms a trochlear surface for articulation with the distal end of the tibia. In this llama, the cranial rotation of the talus bones made articulation of the distal portion of the tibia with the trochlea of the talus impossible. This caused subluxation of the hock joints as the distal portion of the tibia articulated instead onto the caudal aspect of the talus. The stifle joints were radiographically normal.

Results of 2-dimensional, real-time, linear array echocardiography, using an imager with a 3.5-MHz long-focus transducer,[a] revealed ventricular septal defect (Fig 2). Compensatory cardiac hypertrophy was not apparent.

Numerous congenital anomalies in South American camelids have been reported[1-5]; multiple anomalies are uncommon. One newborn llama had internal hydrocephalus and ventricular septal defect.[6] Ventricular septal defect was also reported in a guanaco.[4] Carpal angular limb deformities in zoo llamas have been associated with irregular growth between the ulnar and radial physes.[7] Another group of zoo llamas had angular limb deformity as well as polydactyly, syndactyly, arthrogryposis, and other congenital anomalies not related to limbs.[8]

Figure 1—Lateral view radiographs, of a normal llama hock joint (left), illustrating correct orientation of the tibial tarsal bone, and the hock of the llama of this report (right), illustrating the 90° cranial rotation of the tibial tarsal bone.

Ventricular septal defects are the most common cardiovascular anomalies reported in cattle,[9] horses,[10] and sheep.[11] Only 1.7% (5/290) of dogs with ventricular septal defects had other obvious musculoskeletal defects,[12] whereas 45% (20/44) of lambs with ventricular septal defect had other gross extracardiac defects.[11]

Slow weight gain in human beings with ventricular septal defect has been reported.[13] The llama of this report, although fed oat hay ad libitum plus a pelleted grain supplement, weighed considerably less than younger llamas being fed a 10% protein ration that was 2% of their body weight (Fig 3).

In spite of these defects, the affected llama was capable of vigorous activity without apparent dyspnea or fatigue. The hock lameness was most perceptible at the walk, being markedly less obvious during running.

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[a]Microimager 1000, Ausonics Pty Ltd, Milwaukee, Wis.

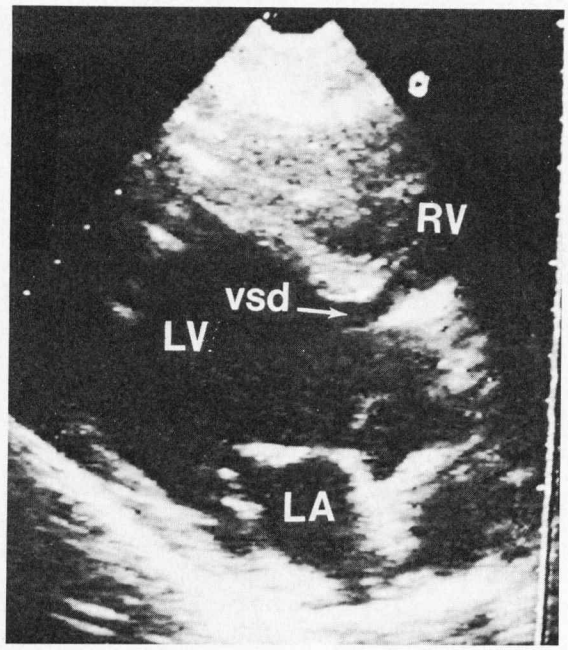

Figure 2—Two-dimensional echocardiogram, long-axis view, right parasternal position, from the affected llama. LA = left atrium; LV = left ventricle; RV = right ventricle; vsd = ventricular septal defect.

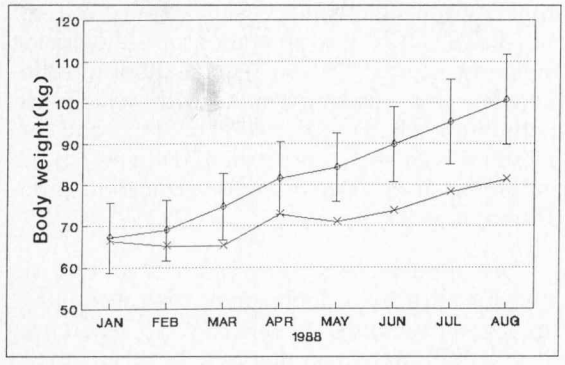

Figure 3—Comparative growth curves: curve 1 (◇—◇) = mean (±SD) body weight of 10 yearling llamas fed a 10% protein ration that was 2% of their body weight; curve 2 (×—×) = the affected llama, fed oat hay ad libitum supplemented with a pelleted concentrate.

Whether the anomalies were genetically transmitted was not known. Genetic transmission of congenital anomalies has been reported in llamas.[7,8] Ventricular septal defects may be inherited in Hereford cattle,[14] and have been reported in familially related goats.[15] Other reports have indicated the probability of a genetic basis for ventricular septal defect in chickens,[16] rats,[17] and Keeshonds.[12] Castration was recommended to prevent the possibility of passing on the undesirable traits; the llama currently remains sexually intact for possible future breeding experiments to ascertain the heritability of these anomalies.

In September 1988, another llama with identical lameness was evaluated at the teaching hospital. Identical lameness was seen recently in a llama in Virginia[b] and in 3 llamas (1 with unilateral involvement) evaluated at the University of California, Davis.[c] There were no accompanying cardiovascular defects in those 4 llamas. These various reports from diverse geographic areas suggest that rotated talus may be a hereditary cause of hind limb lameness unique to llamas.

[b]Solomon S, Piedmont Veterinary Service, Charlottesville, Va: Personal communication, 1988.

[c]Fowler M, University of California, Davis, Calif: Personal communication, 1988.

1. Fenwick BW, Fowler M, Kock M, et al. Complete choanal atresia in a llama. *J Am Vet Med Assoc* 1982;181:1409–1410.

2. Griner LA. *Pathology of zoo animals.* San Diego: Zoological Society of San Diego, 1983;505.

3. Jessup DA, Lance WR. What veterinarians should know about South American camelids. *Calif Vet* 1982; 36(11):12–18.

4. Leipold HW. Congenital defects of zoo and wild mammals: a review. In: Montali RJ, Migaki G, eds. *The comparative pathology of zoo animals.* Washington, DC: Smithsonian Institution Press, 1980;457–470.

5. Wallach JD, Boever WJ. *Diseases of exotic animals: medical and surgical management.* Philadelphia: WB Saunders Co, 1983;327.

6. Brown RJ. Hydrocephalus in a newborn llama. *J Wildl Dis* 1973;9:146–147.

7. Fowler ME. Angular limb deformities in young llamas. *J Am Vet Med Assoc* 1982;181:1338–1342.

8. Cambre RC. Congenital anomalies in a zoo llama herd, in *Proceedings. Am Assoc Zoo Vet Conf* 1984;110–111.

9. Fisher EW, Pirie HM. Malformations of the ventricular septal complex in cattle. *Br Vet J* 1964;120:253–272.

10. Huston R, Saperstein G, Leipold HW. Congenital defects in foals. *J Equine Med Surg* 1977;1:146–161.

11. Dennis SM, Leipold HW. Congenital cardiac defects in lambs. *Am J Vet Res* 1968;29:2337–2340.

12. Patterson DF. Epidemiologic and genetic studies of congenital heart disease in the dog. *Circ Res* 1968;23:171–202.

13. Nugent EW, Plauth WH, Edwards JE, et al. Congenital heart disease. In: Hurst JW, ed. *The heart.* 6th ed. vol 1. New York: McGraw-Hill Book Co, 1986;580–728.

14. Belling TH. Genetic effect of cardiac ventricular septal defects in Hereford cattle. *Vet Med* 1962;57:965–968.

15. Parry BW, Wrigley RH, Reuter RE. Ventricular septal defects in three familially related female Saanen goats. *Aust Vet J* 1982;59:72–76.

16. Siller WG. Ventricular septal defects in the fowl. *J Pathol Bacteriol* 1958;76:431-440.

17. Fox MH. Genetic transmission of congenital membranous ventricular septal defects in selectively inbred strains of rats. *Circ Res* 1967;20:422–433.