Coronary Arterioventricular Anomaly in a Calf

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2. Current WL: 1988, The biology of *Cryptosporidium.* Am Soc Microbiol News 54:605-611.
3. Garber L: 1993, *Cryptosporidium parvum.* Wise Anim Health Newsl 1993(6):1-4.
4. Garber LP, Salman MD, Hurd HS, et al.: 1994, Potential risk factors for *Cryptosporidium* infection in dairy calves. J Am Vet Med Assoc 205:86-91.
5. Holley HP, Dover *C:* 1986, *Cryptosporidium:* a common cause of parasitic diarrhea in otherwise healthy individuals. J Infect Dis 153:365-368.
6. Janoff EN, Reller LB: 1987, *Cryptosporidium* species, a protean protozoan. J Clin Microbiol 25:967-975.
7. Kappus KK, Juranek DD, Roberts JM: 1991, Results of testing for intestinal parasites by state diagnostic laboratories, United States, 1987. Morbid Mortal Weekly Rep 40:25-45.
8. Levine JF, Levy MG, Walker RL, Crittenden S: 1988, Cryptosporidiosis in veterinary students. J Am Vet Med Assoc 193: 1413-1414.
9. McCluskey BJ: 1992, *Cryptosporidium parvum* oocyst shedding in Florida dairy calves. Anim Health Insight USDA : APHIS : VS Rep N010.1192:1-5.
10. Moon HW, Woodmansee DB: 1986, Cryptosporidiosis. J Am Vet Med Assoc 189:643-646.
11. Moore JA, Blagburn BL, Lindsay DS: 1988, Cryptosporidiosis in animals including humans. Compend Cont Ed Small Anim Pract 10:275-281.
12. Skeels MR, Sokolow R, Hubbard CV, et al.: 1990, *Cryptosporidium* infection in Oregon public health clinic patients 1985-88: the value of statewide laboratory surveillance. Am J Public Health 80:305-308.
13. USDA : APHIS : VS National Animal Health Monitoring System: 1993, *Cryptosporidium* is common in dairy calves. Report No. N119.293. USDA : APHIS : VS, Fort Collins, CO.

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Coronary arterioventricular anomaly in a calf

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A 3-month-old Holstein heifer with a history of chronic respiratory disease was donated to the teaching hospital at the Atlantic Veterinary College. The calf weighed 98 kg and was in poor body condition. The heifer was in severe respiratory distress, and clinical findings were consistent with pneumonia. A grade II-IV/VI holosystolic heart murmur, with point of maximal intensity on the right heart base, was also detected. Because the calf failed to show any clinical improvement following treatment with antibiotics, corticosteroids, and bronchodilators, it was euthanized with intravenous pentobarbital and submitted for necropsy examination.

Necropsy findings included a severe chronic suppurative bronchopneumonia with bronchiectasis. This pneumonic process obliterated 60% of the right lung and 10% of the left lung. The caudodorsal right lung field was rubbery, with mild interlobular emphysema. The heart was of normal shape but appeared slightly enlarged. The total heart weight was calculated to be 0.76% of body weight (normal range, 0.30-0.66%).[9]

A tortuous, thin-walled left circumflex coronary artery was noted on the epicardium of the heart base. This vessel extended posteriorly to the interventricular septum, maintaining an internal diameter of 9 mm. The dilated vessel then descended 4 cm along the epicardial surface of the septum before abruptly entering the myocardium (Fig. 1). The artery then traversed a sinuous path through the interventricular septum, eventually communicating with a multilocular cavitation in the right ventricular aspect of the septum (Fig. 2). Marked dilation of the left main coronary artery and 1 of the proximal branches of the left anterior descending coronary artery was also identified. This latter branch extended into the interventricular septum, communicating with the cavitation noted previously.

Figure 1. Epicardial surface of left ventricle from calf with a coronary artery fistula. Note the markedly dilated, thin-walled left circumflex coronary artery (arrowhead).

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Figure 2. Sagittal section of heart from calf with a coronary artery fistula. Note dilatation of several coronary vessels: left main coronary artery (*), left circumflex coronary artery (LC), branch of left anterior descending coronary artery (arrowhead). An aneurysm located in the inter-ventricular septum distorts the right septal papillary muscle (broad arrow).

This cavitation was 4 x 3 x 2 cm and partially occupied the septal papillary muscle of the tricuspid valve. Several slit-like ostia between the trabeculae carnae permitted passage of blood between the ventral aspect of the cavitation and the lumen of the right ventricle. The largest of these openings was 1 cm in length. No gross abnormalities were noted in the right coronary artery or in the coronary veins and sinus. Samples of heart and lung were fixed in 10% neutral-buffered formalin, embedded in paraffin wax, cut into 6-μm-thick sections, and stained with hematoxylin and eosin and with Verhoff Van Geison's elastin stain.

Microscopic examination of the heart revealed generalized myocardial cell hypertrophy. The intraseptal cavity was lined by endothelium. A few intraluminal projections of myxoid stroma covered by endothelium were suggestive of poorly formed venous valves. In other areas of this aneurysm, the wall was thickened by prominent elastic fibers, although a distinct internal elastic lamina could not be discerned. With the exception of the ventrally located ostia, the aneurysm was separated from the right venticular lumen by the endocardium and a layer of myocardium 2-10 cells thick.

The dilated coronary arteries retained an internal elastic lamina and an elastin-rich tunica media and tunica adventitia. Coronary arterioles in the heart apex and in the free right ventricular wall were unremarkable. Numerous sarcocysts were observed throughout the myocardium. Microscopic examination of the caudodorsal lung tissue revealed moderate thickening of alveolar septa by macrophages and foci of type II pneumocyte hyperplasia. The tunica media of pulmonary arterioles were also mildly thickened.

Actinomyces pyogenes was cultured from the purulent pulmonary exudate. Samples of the caudodorsal lung were examined via fluorescent antibody techniques for infectious bovine rhinotracheitis virus, parainfluenza-3 virus, bovine respiratory syncytial virus, and bovine viral diarrhea virus (BVDV). A positive result was obtained only for BVDV, and a noncytopathogenic strain of BVDV was subsequently isolated from lung, thymus, and intestine. A fecal flotation test revealed a heavy infection with *Eimeria* spp. and fewer *Strongyloides.*

Necropsy findings of chronic suppurative bronchopneumonia, BVDV infection, and parasitic infections provided an adequate explanation for the respiratory disease and poor growth observed clinically. The anomalous cardiac vasculature was an incidental finding, but such defects are rare in domestic animals.

Atrial and ventricular septal defects and transpositions of the main vessels are the most commonly recognized cardiac anomalies in cattle.[9] Abnormalities of the bovine coronary vasculature have also been described.[4,10,12] Most of these cases were characterized by an anomalous origin to 1 or both of the coronary arteries, but uncomplicated cases of coronary arterioventricular fistulas with aneurysmal dilatation have also been reported.[12]

The embryogenesis of the cardiac vascular system involves the development of both intertrabecular and coronary vascular spaces.[2] The network of intertrabecular channels communicates with the heart chambers but becomes less prominent as the heart matures.[2] The fully developed cardiac vascular system is extremely complex, with abundant anastomoses between different vascular components and a large capacity for collateral circulation.[1,2,5]

Thebesian vessels are believed to be remnants of the intertrabecular channels.[2] These vessels provide direct connections between the coronary vasculature and the heart chambers, bypassing the coronary sinus. Such connections can be classified into 3 categories: arterioluminal channels, venoluminal channels, and arteriosinusoidal connections.[2,3,13] Some authors have suggested that many arterioluminal connections should be classified as coronary artery fistulas because inappropriate blood flow through these channels can cause myocardial ischemia via a myocardial steal phenomenon.[7,11] Such fistulas are the most common hemodynamically significant coronary artery anomaly in humans.[1]

Venoluminal vessels, also known as Thebesian veins, are most common in the right ventricle, where they provide the majority of postcapillary venous drainage.[2,5] Studies of the dynamics of retrograde cardioplegia in explanted human hearts suggest that blood backup within the coronary sinus venous system is largely shunted to the ventricular chambers via Thebesian veins. Based on the detection of a high density of atrial natriuretic peptide binding sites in the vessel walls, some authors have speculated that Thebesian veins may even be capable of local regulation of blood flow.[5]

Arteriovenous interconnections in the heart allow blood flow between coronary arteries and Thebesian veins, as appeared to be the case in this calf. Coronary artery fistulas connecting with a subendocardial vascular plexus have been

reported in humans.[6,8] The cause of the dilation of the affected coronary arteries is unknown. Most human patients with coronary artery fistulas are asymptomatic,[1] and none of the clinical signs in this calf were attributed to cardiac insufficiency. However, possible sequelae to coronary artery fistulas include myocardial infarction, congestive heart failure, bacterial endocarditis, and fistula rupture.[1]

There are several possible explanations for the heart murmur, myocardial hypertrophy, and hypertensive pulmonary changes noted in this calf. Holosystolic heart murmurs may result from blood flow through coronary artery fistulas.[1] A left-to-right shunt could have increased right ventricular pressure, leading to myocardial hypertrophy and increased pressures in pulmonary arteries. Alternatively, deformation of the septal papillary muscle could have induced minor incompetence of the tricuspid valve, creating a murmur. However, features often linked to tricuspid insufficiency, such as a jugular pulse, hepatic congestion, or atrial "jet" lesions, were not observed. The murmur may have been physiologic, and the thickening of pulmonary arteries may be attributable to the severe chronic pneumonia (cor pulmonale).

This report documents an unusual anomaly of the bovine coronary vasculature and serves to emphasize the remarkable compensatory capacity of the cardiac blood supply.

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References

1. Bairn DS, Harrison DC: 1986, Nonatherosclerotic coronary heart disease. *In:* The heart, ed. Hurst JW, 6th ed., pp. 1016-1025. McGraw-Hill, Toronto, ON, Canada.
2. Bardoli G: 1981, The coronary circulation in man. *In:* Structure and function of the circulation, ed. Schwartz CJ, Werthessen NT, Wolf S, vol. 2, pp. 77-190. Plenum, New York, NY.
3. Currie MG, Schemer H, Lanier-Smith KL, Baron DA: 1989, Atrial natriuretic peptide binding sites in the mammalian heart: localization to endomural vessels. Cell Tissue Res 256:233-239.
4. Dennis SM, Gardiner MR: 1972, Congenital coronary artery defect in a cow. Vet Rec 190:281.
5. Gates RN, Laks H, Drinkwater DC, et al.: 1993, Gross and microvascular distribution of retrograde cardioplegia in explanted human hearts. Ann Thorac Surg 56:410-417.
6. Gillebert C, van Hoof R, van de Werf F, et al.: 1986, Coronary artery fistulas in an adult population. Eur Heart J 7:437-443.
7. Kawakami K, Shimada T, Yamada S, et al.: 1991, The detection of myocardial ischemia by thallium-201 myocardial scintigraphy in patients with multiple coronary arterioventricular connections. Clin Cardiol 14:975-980.
8. Martens J, Haseldoncks C, van der Werf F, de Geest H: 1983, Silent left and right coronary artery-left ventricular fistulas: an unusual prominent Thebesian system. Acta Cardiol 38:139-142.
9. Robinson WF, Maxie MG: 1993, The cardiovascular system. *In:* Pathology of domestic animals, ed. Kennedy PC, Jubb KVF, Palmer NC, 4th ed., vol. 3, pp. 7-53. Academic Press, Toronto, ON, Canada.
10. Sandusky GE, Smith CW: 1981, Congenital cardiac anomalies in calves. Vet Rec 108:163-165.
11. Sheikhzadeh A, Stierle U, Langbehn AF, et al.: 1986, Generalized coronary arterio-systemic (left ventricular) fistula; case report and review of literature. Jpn Heart J 27:533-544.
12. van Nie CJ: 1968, Anomalous origin of the coronary arteries in animals. Pathol Vet 5:313-326.
13. Wearn JT, Mettier SR, Klumpp TG, Zschiesche LJ: 1933, The nature of the vascular communications between the coronary arteries and the chambers of the heart. Am Heart J 9: 143-164.

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Multisystem angiotropic lymphoma (malignant angioendotheliomatosis) involving the humerus in a dog

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Malignant angioendotheliomatosis is a rare neoplastic disorder in people originally believed to be a neoplastic disorder of endothelial cells.[8] More recently, utilizing electron microscopy and immunohistochemical techniques, it has been shown to be more than 1 entity, most frequently an angiotropic large-cell lymphoma.[1,2,4,5,9] Angiotropic large-cell lymphoma is characterized by widespread proliferation of large pleomorphic mononuclear cells within the lumina of small blood vessels, with a predilection to sites in the integument, central nervous system, and commonly the eye. Additionally, in many cases examined immunohistochemically, tumor cells have been negative for factor VIII-related antigen and positive for immunoglobulin and other lymphoid cell markers. Ultrastructural features of endothelial cells, such as Weibel-Palade bodies, junctional complexes, pinocytotic vesicles, or basement membrane elaboration, have been shown by electron microscopy in many of these same cases to be absent in the luminal tumor cells.[1,2,4,5,8,9] This disease is extremely rare in animals. It has only been reported in dogs and has a propensity to most severely involve the brain and 1ungs.[3,6,7,10] Of the 8 cases reported in the dog, ultrastructural evaluation in 2[3,10] and immunohistochemical evaluation in 3[3,6,10] have confirmed the lymphoid nature of this entity in dogs.

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