

Femoral artery occlusion in Cavalier King Charles Spaniels

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Objective—To investigate development of femoral artery occlusion in Cavalier King Charles Spaniels.

Design—Prospective study.

Animals—954 Cavalier King Charles Spaniels.

Procedure—1,750 cardiovascular examinations consisting of visual inspection of mucous membranes, thoracic auscultation in areas associated with the heart valves, thoracic palpation, and palpation of the femoral arteries were made at 10 dog shows on 954 dogs. Findings of clinically normal, weak, or undetectable femoral pulses were recorded. Pathologic changes in occluded femoral arteries of 2 dogs were examined histologically.

Results—Of the 954 dogs, 22 (2.3%) had an undetectable right or left femoral pulse on 1 or more examinations. Forty (4.2%) additional dogs had weak unilateral or bilateral femoral pulses. Only 1 dog had exercise intolerance, and it had coexistent congestive heart failure. Histologic examination of serial sections of an occluded femoral artery from 1 dog revealed intimal thickening with breaks in the internal elastic lamina proximal to the occluded segment. The occluded segment of the femoral artery was contracted and filled with an organizing, recanalizing thrombus. Similar histopathologic changes were found in sections of a femoral artery from another dog.

Clinical Implications—Femoral artery occlusion is rare in other breeds and is not clinically important in dogs because of adequate collateral circulation; however, its rather common development in Cavalier King Charles Spaniels indicates a genetic predisposition and probable weakness in the femoral artery wall. (*J Am Vet Med Assoc* 1997;211:872–874)

Spontaneous peripheral vascular disease in dogs is uncommon. In most dogs, the disease has been recognized only histologically and includes thrombosis, arteriosclerosis, or atherosclerosis, usually in association with renal or endocrine disorders.^{1-3,a} In a study⁴ of 5,000 dogs, results of cardiovascular examinations did not reveal any dog that lacked a femoral pulse. However, in a similar study⁵ that focused on the high prevalence of mitral valve disease in Cavalier King Charles Spaniels (CKCS), extremely weak or no femoral pulses were occasionally found in examinations of this breed.

In CKCS with vascular disease, genetic factors are indicated by the unusually high prevalence of this disorder in the breed and by the young age and familial relationship of some affected dogs. The purposes of

the study reported here were to investigate development of weak or undetectable femoral pulses in 62 of 954 (6.5%) CKCS and to report histopathologic findings in occluded femoral arteries of 2 dogs.

Materials and Methods

A total of 1,750 physical examinations were performed on 954 CKCS at 10 dog shows from 1990 to 1996. Physical examinations consisted of inspection of mucous membranes, thoracic palpation, palpation of the femoral arteries, and thoracic auscultation of all areas associated with heart valves. Observations were recorded on a standardized form before comparisons were made with results from prior examinations.

In addition, a 7-year-old male CKCS with severe mitral valve disease, refractory congestive heart failure, and an undetectable right femoral pulse was examined in the cardiology service of the University of Pennsylvania veterinary hospital. Lack of a right femoral pulse was noticed on 3 examinations during a 1-year period prior to unsuccessful mitral valve surgery. Direct arterial pressure was recorded via the right dorsal pedal artery at surgery. On necropsy examination, a 1-cm-long segment of the right femoral artery was harvested from a level proximal to the occluded segment to the obviously narrow, firm, occluded area. For histologic evaluations, serial sections at 0.5-mm intervals were stained with Verhoeff's stain. Additional slides were stained with H&E or a trichrome stain. Sections of femoral arteries from another dog were also examined histologically. The dog had weak pulses bilaterally for 3 years before the left femoral pulse became undetectable. One year later, the left femoral pulse was again detectable but weak.

Results

Results of 1,750 examinations of 954 CKCS from 1990 to 1996 revealed 22 (2.3%) dogs without a right or left femoral pulse on 1 or more examinations. There were 12 males and 10 females. Coat colors of dogs included the following: blenheim (n = 16), tricolor (4), black and tan (1), and ruby (1). The sex and coat colors of dogs with a weak or undetectable femoral pulse had the same distribution as the dog show population. Fourteen of the 22 dogs were examined 2 or more times. Five dogs were found to have a weak pulse on 1 or more examinations prior to development of an undetectable femoral pulse. Five dogs had return of weak or clinically normal pulses in subsequent examinations. Femoral arteries in some dogs were smaller than normal and were firm and cord-like. Physical examination of some dogs revealed a firm lump in the femoral artery. Weak pulses were usually detected distal to the occluded segment, and the hind limbs were warm, indicating adequate collateral circulation. Nine of 22 dogs without a femoral pulse were < 2 years old. Most of the dogs > 3 years old without femoral pulses in 1 hind limb had weak pulses in the opposite limb. Lack of femoral pulses in both hind limbs at the same

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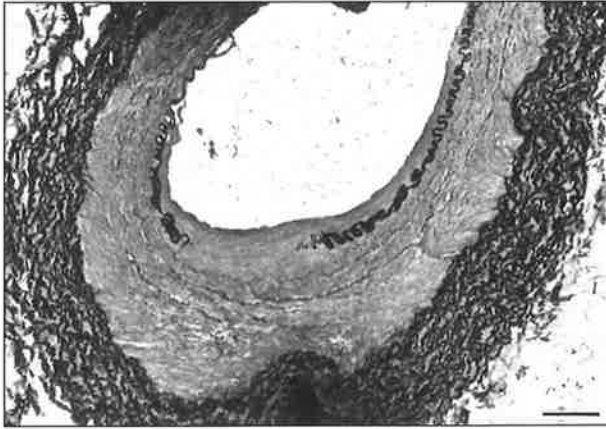


Figure 1—Photomicrograph of a section of an occluded femoral artery just proximal to the thrombosed segment from a 7-year-old dog that lacked a femoral pulse for at least 1 year. Notice the intimal thickening and interruption of the internal elastic lamina. Verhoeff's stain; bar = 150 μ m.

time was not observed in any dog. One dog switched from lack of a right femoral pulse at 6 months of age to lack of a left femoral pulse 2 years later, with return of a weak pulse on the right side. All but 1 of the dogs without a femoral pulse were active and had no obvious signs of hind limb weakness. The 1 dog with exercise limitations also had severe mitral valve disease and congestive heart failure. There were 40 additional dogs with notably weak femoral pulses on 1 or more examinations. In most of these dogs, this was found in only 1 hind limb.

Two 8-month-old littermates, 1 with a weak femoral pulse and 1 that lacked a femoral pulse, had notably small affected femoral arteries that contained firm lumps approximately 2 mm in diameter. A female offspring of the dog that had no right femoral pulse followed by no left femoral pulse had an undetectable right femoral pulse at 13 months of age. The grandfather of another 8-month-old dog without a right femoral pulse and with a lump in the artery had weak femoral pulses at 4 years of age and lack of a left femoral pulse at 5 years of age. The grandmother of an 18-month-old dog without a right femoral pulse lacked a right femoral pulse at 11 years of age and had weak pulses bilaterally at 7, 9, 10, 12, and 13 years of age.

Heart murmurs were detected in 5 of 16 dogs under the age of 7 years that lacked femoral pulses but were only grade I to II in intensity, with the exception of 1 dog that also had confirmed pulmonic stenosis. Five of 6 dogs that were ≥ 7 years old had left-sided, grade-III to -VI systolic murmurs that were associated with the left apex of the heart. The frequency of murmurs was not notably different from the age specific frequency of murmurs in the dog show population.⁵

Direct measurements of blood pressure in the dorsal pedal artery were obtained distal to the occluded femoral artery in a 7-year-old dog during surgery and revealed a low pulse pressure with a mean pressure within the reference range, consistent with adequate collateral circulation. Histologic examination of a se-

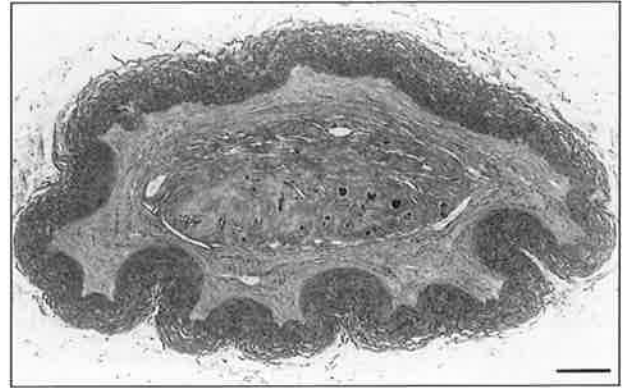


Figure 2—Photomicrograph of a section of an occluded femoral artery through the thrombosed segment, 2 cm distal to the section shown in Figure 1. Notice the contracted state of the artery and thrombus-filled lumen with recanalizing small vessels. Masson's trichrome stain; bar = 150 μ m.

rial section of the affected femoral artery revealed cellular intimal thickening over breaks in the internal elastic lamina just proximal to the occluded segment (Fig 1). In adjacent sections, the intimal thickening extended distally into the well-organized thrombus. The central, occluded region was sclerosed, contracted, and filled with a well-organized, recanalizing thrombus (Fig 2). A fibrosed, recanalizing thrombus was also observed histologically in sections of an affected femoral artery of a second dog. There was no evidence of inflammatory or degenerative changes in the media or adventitia of the femoral arteries from either dog.

Discussion

Occlusion of femoral arteries in dogs usually has no clinical importance because of collateral circulation. The femoral arteries are routinely ligated at the conclusion of cardiac catheterization procedures if they appear to be damaged. Ligation is possible because dogs have extensive collateral circulation in their hind limbs via lateral circumflex femoral and distal caudal femoral arteries and anastomoses of the latter with the caudal gluteal artery and branches of the deep femoral arteries.⁶ Human beings lack distal caudal femoral arteries; therefore, obstruction of the femoral artery can cause limb ischemia and serious consequences.

Assessment of pulse quality is a subjective clinical finding, and weak pulses may be mistakenly diagnosed in obese or muscular dogs. Weak pulses also may be found in dogs with low blood pressure, congestive heart failure, and various other abnormalities. Almost all of the CKCS examined in our study were in competitive show condition and their similarity in size, conformation, and condition made it possible to more accurately assess undetectable, weak, or normal femoral pulses. In most of the dogs < 7 years old with weak or no femoral pulses, the abnormality was unilateral and the opposite hind limb served as a basis for comparison.

Thrombosis of the femoral arteries in the CKCS of our report is considered a primary form of vascular disease rather than extension of thromboembolism. There

were no signs of embolism in other parts of the body in any of the dogs. In serial sections of the femoral artery examined histologically, the proximal extent of the thrombus was not at a bifurcation but began just distal to the origin of the lateral circumflex femoral artery branch.

Histopathologic changes observed in 2 of these dogs indicated that the femoral artery occlusion may have been initiated by trauma. However, genetic vascular weakness may also have been a factor because of the young age and familial relationship of some dogs.

Development of a vascular disorder in a breed with a high incidence of mitral valve disease supports the hypothesis that an underlying connective tissue disorder exists in CKCS that renders them susceptible to cardiovascular disease. Other signs of connective tissue disease have not been reported in this breed.

Hematologic and coagulation studies were not performed in any of the dogs in our study to determine whether hypercoagulability may play a role in the development of femoral artery thrombosis. Large platelets with low platelet counts but normal clotting ability are known to be characteristic of CKCS.⁷

^aVan Winkle TJ, MacDonald M, Hendricks J. Thrombosis of the portal vein, pulmonary arteries, and aorta in the dog (abstr), in *Proceedings, Annu Meet Am Coll Vet Pathol* 1989;51.

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Book Review:

Clinical Behavioral Medicine for Small Animals. By Karen Overall. 544 pages; illustrated. Mosby Year-Book Inc, 11830 Westline Industrial Dr, St Louis, MO 63146-3318. 1997. Price \$49.99.

Overall, this book is excellent. Being a veterinarian who graduated during an era when behavioral medicine was not part of the curriculum, I found it informative and useful. Although the introductory comments focus on the book's use by practitioners, its structure and style make it seem more useful as a teaching tool. The book would make excellent required reading for veterinary students, because practitioners may not appreciate the in-depth references and supporting data, which could prove cumbersome. However, given the book's secondary purpose of serving as a resource for specialists, the data content is appropriate.

Practitioners and students should heed the introductory comments concerning the veterinarian's role in behavioral medicine and should be stimulated by comments in the introduction and chapter on epidemiology. Special attention to the most common behavioral problems is pertinent and helpful. Even questions clients ask about their healthy pets are addressed.

As a former practitioner, I would like to offer some comments. First, there appears to be some conflicting statements on the concept of ovariohysterectomy and weight gain. For example, in Chapter 3, the author states, "Spayed dogs can experience weight gain because estrogen is a strong appetite stimulant." However, in Chapter 10, the author states, "Estrogen inhibits food intake. . . ."

This criticism aside, the author's extensive use of charts and protocols in appendices is excellent. These are golden nuggets for daily use by practitioners; therefore, more emphasis on ways to access the content of these appendices would be helpful to hurried readers. Chapter 10 covers acral lick dermatitis extremely well; I appreciated reading an unbiased discussion approached from dermatologic and behavioral points of view. The flowcharts are especially useful.

The book is well written, complete, and useful to the veterinary profession. It emphasizes the veterinarian's role in the human-animal bond and provides veterinarians with an opportunity to expand their capabilities into an arena in which they have been greatly lacking. The book will help veterinarians obtain correct information on animal behavior, instead of relying on hearsay, biased information, and uneducated guesses.

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