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#### **Title**

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#### **Permalink**

<https://escholarship.org/uc/item/6xz10134>

#### **Journal**

Veterinary Radiology & Ultrasound, 45(6)

#### **ISSN**

1058-8183

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#### **Publication Date**

2004-11-01

Peer reviewed

# MYELOPATHY SECONDARY TO AORTOCAVAL FISTULA IN A CAT

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**A 15-month-old neutered male cat was presented for progressive paraparesis of 3 months' duration and suspected cardiomegaly. Neuroanatomical localization was a T3–L3 myelopathy. On abdominal ultrasound, an anomalous vessel with turbulent blood flow was identified arising from the caudal vena cava. Myelography showed a bilateral ventrolateral extradural spinal cord compression from T12 to L4. Nonselective angiography and contrast-enhanced computed tomography clearly indicated a vascular complex and vena caval aneurysm with an engorged internal vertebral venous plexus. Surgical occlusion of the anomalous vessels was unsuccessful. *Veterinary Radiology & Ultrasound, Vol. 45, No. 6, 2004, pp 528–531.***

**Key words:** aortocaval fistula, feline, myelography, vascular myelopathy.

## Introduction

A 15-MONTH-OLD NEUTERED male domestic shorthaired cat was referred to the Veterinary Medical Teaching Hospital, University of California, Davis, for progressive paraparesis of 3 months' duration and suspected cardiomegaly. Abnormalities on physical examination were restricted to a grade 3/6 systolic dynamic, left parasternal, basilar heart murmur, and prominent kidneys with smooth margins. On neurological examination the cat was ambulatory, with moderate paraparesis and ataxia. Postural reactions were reduced in both pelvic limbs. Segmental reflexes were normal. Apparent pain was noted on palpation of the musculature overlying the thoracolumbar junction. Based on these neurological deficits, the neuro-anatomical localization was a T3–L3 myelopathy. Possible causes included congenital vertebral and/or spinal cord anomalies, metabolic disease, vertebral or central nervous system (CNS) neoplasia, infectious diseases (myelitis), intervertebral disc disease, or spinal cord trauma.

A minimum database was done. Results of a complete blood count and serum chemistry panel were unremarkable. Urinalysis findings included a 1+ proteinuria with a specific gravity of 1.016. Serum Feline leukemia virus and Feline immunodeficiency virus antibody titers were negative. Thoracic radiographs were unremarkable. Abdominal ultrasonography demonstrated that the renal cortices were hypochoic bilaterally with diminished corticomedullary

definition. The size of the kidneys (4.8 cm) was at the upper limit of normal. There was a moderate quantity of retroperitoneal fluid bilaterally and a small volume of ascites. A large aneurysmal dilatation of the caudal vena cava was also identified just cranial to the renal vessels (Fig. 1). The caudal vena cava could not be visualized cranial to the dilatation. An anomalous vessel was identified arising from the dilated caudal vena cava that extended dorsolaterally. The anomalous vessel had turbulent blood flow and a systolic component consistent with an arteriovenous fistula or shunt.

An echocardiogram revealed mild septal and papillary muscle hypertrophy. Cardiac chamber sizes and contractility were normal. Color flow Doppler ultrasound confirmed turbulence in the right ventricular outflow tract. However, results of the continuous wave Doppler were normal.

The cat was anesthetized for vertebral column radiographs, lumbar cerebrospinal fluid (CSF) collection and a lumbar myelogram using 0.4 ml/kg of Isovue-200®.\* Vertebral column radiographs were within normal limits. Results of lumbar CSF analysis revealed an albuminocytologic dissociation (protein of 58 mg/dl (normal < 30 mg/dl) and a normal cell count (< 1 cell/μl)) with an abnormal distribution of nucleated cells (19% neutrophils, 11% eosinophils, 51% small mononuclear, and 19% large mononuclear cells (normal 40% small mononuclear cells, 60% large mononuclear cells)). Bilateral ventrolateral extradural spinal cord compression was noted on the myelogram, extending from T12 to L4 (Fig. 2). Nonselective angiography (Fig. 3) and a subsequent postcontrast (1 ml/lb Conray-400®†) computed tomography (CT) (Fig. 4A) revealed an aortocaval fistula and vena caval aneurysm. The CT angiogram demonstrated a ventrolateral bilateral

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Received December 26, 2003; accepted for publication March 25, 2004.  
doi: 10.1111/j.1740-8261.2004.04090.x

\*Iopamidol inj. 41%, Bracco Diagnostics, Princeton, NJ.

†Iothalamate sodium 66.8% inj. U.S.P., Mallinckrodt Inc., St Louis, MO.

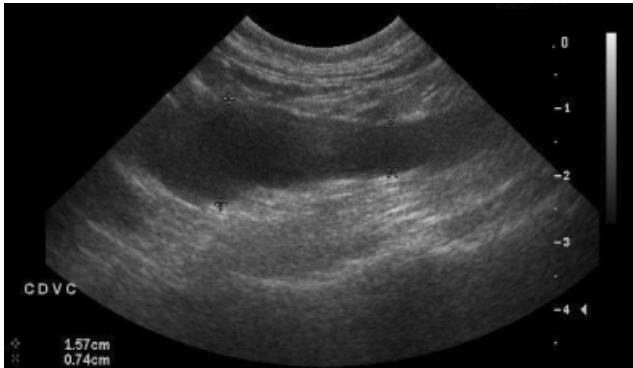


FIG. 1. Ultrasonographic long axis image of the abdomen. Note the marked dilation of the caudal vena cava, outlined by the symbols (+, ×). Cranial is to the left.

compression of the spinal cord by an engorged internal vertebral venous plexus (Fig. 4B).

An exploratory laparotomy was done to attempt surgical closure of the aortocaval fistula. Preoperatively, a blood type determination (Type A) and cross-match were done. There was agglutination of the cat's blood with blood from all of the available donors. The caudal vena cava was en-



FIG. 2. Ventrodorsal myelogram of the thoracolumbar vertebral column. There is mild bilateral, axial compression of the spinal cord, with attenuation of the left and right contrast columns from the level of T12-L4.

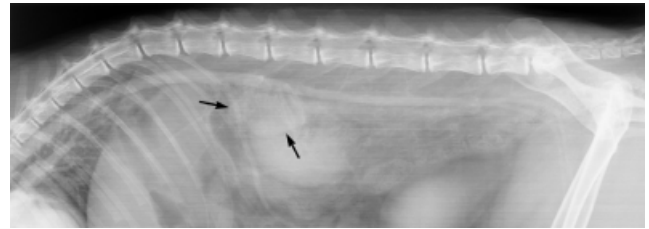


FIG. 3. Nonselective angiogram of the abdomen. Note the dilated vascular complex ventral to the L2 vertebral body (arrows). Right and left kidneys are superimposed. Note the attenuation of the dorsal and ventral aspects of the myelographic contrast columns from T12 to L7.

gorged, pulsing, and had fremitus. An anomalous vessel was identified entering the ventral surface of the caudal vena cava at the level of the renal veins. In addition to the caval distension, there was associated regional venous engorgement including both renal veins. When the anomalous vessel was occluded there was less fremitus in the caudal vena cava. However, engorgement and pulsation of the renal veins persisted. The anomalous vessel was not ligated as it was one of many observed arteriovenous connections. Hemorrhage occurred during the initial dissection

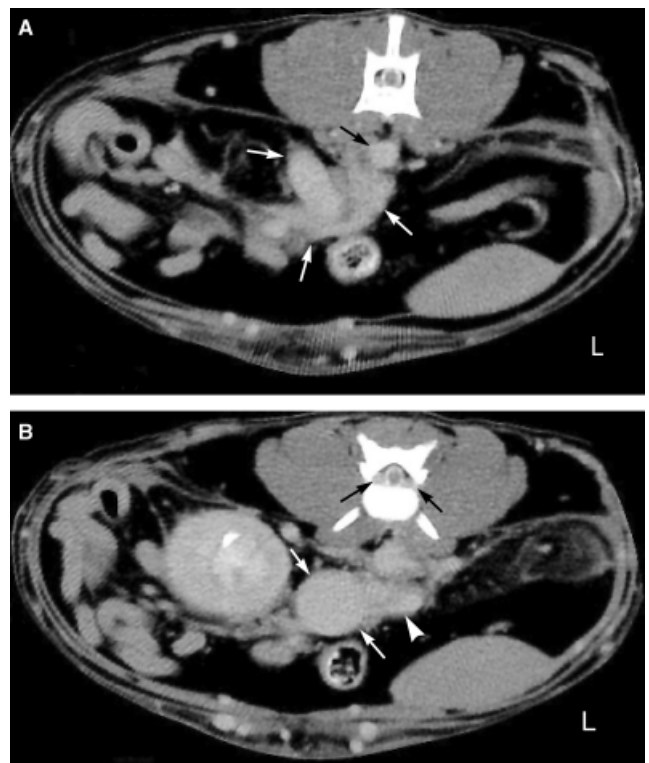


FIG. 4. (A) Postintravenous contrast and postmyelogram transverse computed tomographic (CT) image at the level of the L2 vertebral body. Note the vascular complex ventral (white arrows) to the aorta (black arrow). (B) Postintravenous contrast and postmyelogram transverse CT image 12.0 mm caudal to (A). Note the engorged internal vertebral venous plexus compressing the spinal cord ventrolaterally (black arrows), the dilated blood vessels consistent with the caudal vena cava (white arrows), and the associated vascular complex (arrowhead).

to define the fistula. Therefore, further exploration of the dorsal surface of the caudal vena cava was not done, as a lack of available blood products for transfusion was considered a contraindication. The cat was euthanized because of a poor prognosis and the body was submitted for necropsy.

On gross necropsy examination, the internal vertebral venous plexus was markedly dilated from T12 to L4 (Figs. 5A and 5B). Although the aortocaval fistulae could not be documented in the absence of vascular perfusion, a complex vascular plexus was present in the abdomen extending from the caudate lobe of the liver to the colonic mesentery. The plexus involved both adrenal glands and consisted of numerous small, primarily arterial, vessels. Other necropsy findings included marked periportal congestion within the liver and endocardiosis. Severe Wallerian degeneration was found on microscopic examination of the ventral and lateral portions of the spinal cord from T11 to L1, with moderate Wallerian degeneration in the ventrolateral portions of the spinal cord at T7 and from L3 to L4.

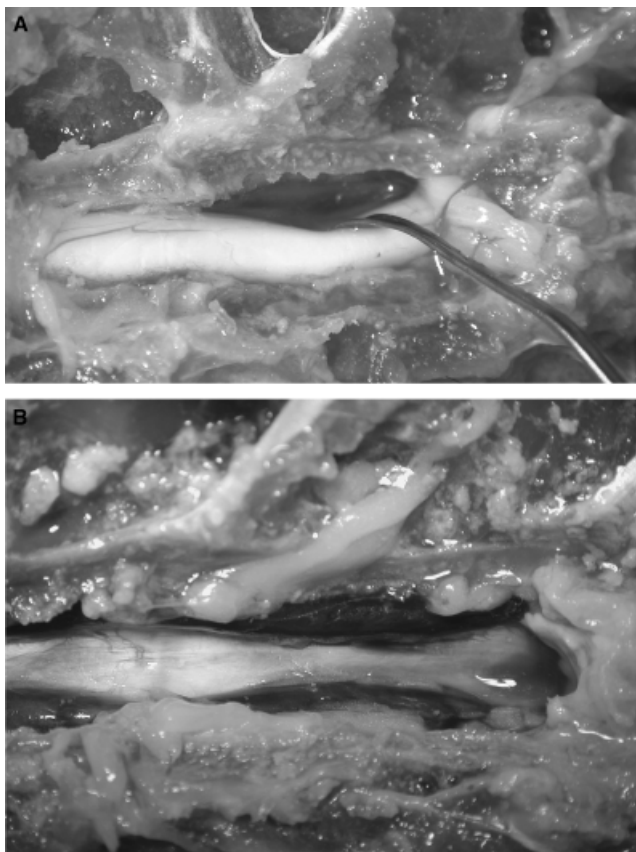


FIG. 5. (A) Postmortem dorsal view of the spinal cord within the lumbar vertebral canal post dorsal laminectomy. The spinal cord has been retracted to illustrate the markedly dilated internal vertebral venous plexus in situ. (B) Postmortem dorsal view of the lumbar vertebral canal post dorsal laminectomy with the spinal cord removed. Note the markedly dilated internal vertebral venous plexus.

## Discussion

Arteriovenous shunts that affect the spinal cord may be either fistulas or niduses, and may be separated into four different groups (paraspinal, epidural, dural, or intradural) according to their location and relationship to the spinal cord dura mater.<sup>1</sup> Paraspinal arteriovenous shunts are located outside the vertebral column, and may result in clinical signs of spinal cord dysfunction.<sup>1</sup> The aortocaval fistula that occurred in the cat in this report may be classified as a paraspinal arteriovenous shunt, and represents an abnormal communication between the abdominal aorta and the caudal vena cava.

Vascular malformations of human beings have been classified on the basis of the caliber and configuration of the constituent vascular channels, their continuity with normal vasculature, their location, and the quantitative relationship between the blood vessels and the CNS parenchyma.<sup>2,3</sup> Such classifications include discrete arteriovenous, cavernous, venous, capillary, and mixed types.<sup>2</sup> These malformations generally have been regarded as congenital lesions that arise as a result of disordered mesodermal differentiation during gestation, with associated lack of development of the local capillary bed.<sup>2,4,5</sup> Multiple communications are present between arteries and veins as they begin to differentiate, and congenital arteriovenous fistulas arise from persistence of these communicating branches.<sup>4</sup> However, some vascular malformations of the CNS may be acquired.<sup>2</sup> Acquired arteriovenous fistulas may occur secondary to trauma (penetrating wound or surgery), erosion or rupture of an arterial aneurysm into a vein, or surgical ligation of blood vessels.<sup>4,5</sup>

Fistulas between the abdominal aorta and the inferior vena cava are rare in human beings and almost always are acquired.<sup>4</sup> In human beings, aortocaval shunts most often are caused by erosion of an aortic aneurysm into the vena cava.<sup>6</sup> Previous reports exist of peripheral arteriovenous fistulas and an aortocaval fistula in cats.<sup>7-9</sup> The aortocaval fistula described in the cat in the present report most likely is a developmental anomaly, considering the age of the cat, the slow progression of clinical signs, and the extent of the vascular anomaly. However, an acquired fistula must be considered possible.

The progressive myelopathy observed in the cat in this report may be attributed to alterations in the dynamics of vertebral and spinal cord blood flow caused by the aortocaval fistula. It is most likely that an increased blood volume, increased vascular resistance, and increased systemic blood pressure because of the arteriovenous fistula caused the blood in the caudal vena cava to be diverted via the azygous vein, basivertebral veins, and internal vertebral venous plexus. Dilatation of the internal vertebral venous plexus in the ventral epidural space resulted in compression of the spinal cord and clinical signs of a progressive mye-

lopathy. The high venous pressure in the epidural venous system also may have resulted in intradural venous hypertension by increasing the resistance to outflow.<sup>3</sup> The shunting of large quantities of blood into the venous system also may have diverted blood flow from the spinal cord (so called "arterial steal").<sup>3</sup> In human beings, a direct communication between an extradural artery and vein may lead to engorgement of the epidural venous system, compression of the spinal cord, and resultant progressive myelopathy.<sup>1,3</sup> Secondary changes reported to result from the hemodynamic alterations created by an arteriovenous fistula in human beings include dyspnea, cardiomegaly, arrhythmias, hypertension, cerebral vascular insufficiency, oliguria, and azotemia.<sup>4</sup>

In human beings, angiography of paraspinal shunts remains the gold standard for analysis of the anatomical, morphological, and architectural features necessary for therapeutic decisions.<sup>1</sup> Abdominal ultrasound examination, myelography, CT imaging, and nonselective angiography were used in the diagnosis of the aortocaval fistula in the cat in this report. The abdominal vascular abnormality initially was visualized by means of abdominal ultrasound, illustrating the importance of this technique in the workup

of a cat presenting with clinical signs of a progressive myelopathy. Postcontrast CT imaging and nonselective angiography provided specific information regarding the location, the extent, and the role of the vascular abnormality in causing a progressive myelopathy. Myelography demonstrated the precise location and the extent of spinal cord compression.

The primary goal of treatment of paraspinal arteriovenous shunts in human beings is complete closure of the shunt.<sup>4</sup> Following closure of the fistula, reduction of heart size and return to hemodynamic normalcy are anticipated.<sup>4</sup> In human beings, paraspinal arteriovenous malformations rarely require open surgery because they can be treated very effectively by means of endovascular procedures.<sup>3</sup> Embolization may be achieved by means of acrylic glue (or coils and balloons).<sup>1</sup> The technique of treatment and the choice of embolic agents should be adapted to the angio-architecture of the shunt.<sup>1</sup> In the cat in this report, an attempt to occlude the aortocaval fistula by means of open surgery was unsuccessful. It is anticipated that endovascular approaches to shunt occlusion will become available for use in dogs and cats in the future.

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