Incidental Intracranial Aneurysm in a Dog Detected by 16-Multidetector Row Computed Tomography Angiography

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This paper describes a small intracranial aneurysm incidentally found in a 24-month-old Nova Scotia Duck Tolling Retriever evaluated for a recent history of lethargy, fever, and cervical pain. The clinicopathological analysis revealed leukocytosis, and increased haptoglobin and C-reactive protein consistent with a severe flogistic process. Nonenhanced computed tomography of the brain and cervical spine showed a diffuse encephalopathy and moderate cervical syringohydromelia. Computed tomography angiography series of the brain showed a small saccular dilation at the joining point of the two rostral cerebral arteries consistent with a small aneurysm. Cerebrospinal fluid examination led to the final diagnosis of aseptic meningitis. The dog was discharged with a long-term corticosteroid therapy for the meningitis. At two-month follow-up evaluation, the cerebrospinal fluid examination was normal and the computed tomography of the brain showed no abnormalities except for the stable aneurysm. To our knowledge, this is the first description of a spontaneous cerebral aneurysm in dogs and serves to broaden the spectrum of cerebrovascular diseases in this species.

1. Introduction

A brain aneurysm is an abnormal, outward pouchning of the artery wall caused by a weakness in the wall of an artery that supplies the brain. In humans, the prevalence of this condition is about 5%. Approximately, 85% of aneurysms develop in the anterior portion of the circulation of the brain and are asymptomatic until they rupture [1–3]. Cerebral aneurysms are classified based on a number of features including etiology, size, shape, the association with the specific intracranial branch, or according to their angioarchitecture features [2, 4–6].

To date, very little is known concerning the type and incidence of variants and anomalies of the cerebrovascular system in dogs and cats [7–9]. Historically, few cases of cerebral hemorrhage in dogs were thought to be correlated to aneurysm, but their existence could not be proved [10]. To the authors knowledge, this is the first description of a spontaneous cerebral aneurysm in dogs.

2. Case Presentation

A 24-month-old 22 kg intact male Nova Scotia Duck Tolling Retriever was evaluated for a recent history of lethargy, fever, and cervical pain. Medical history included another episode of cervical pain that has occurred one year before and rapidly improved with corticosteroid therapy for few days. At the time of presentation, the dog was receiving amoxicillin/clavulanic acid for 2 days (10 mg/kg PO q12).

At physical examination, the dog showed ventroflexion of the neck and was tachypneic (80 breath/min), with 39.3°C rectal temperature and normal heart rate (96 beats/min), increased blood pressure (120–180 mmHg, mean 145 mmHg), and normal mucous membrane appearance and capillary refill time (approximately 1 sec). Heart and thoracic auscultation were normal. Abdominal palpation revealed splenomegaly.

At neurological examination, the dog had cervical rigidity and cervical pain. Cranial nerve function, postural reaction testing, and segmental spinal reflexes were normal. The CBC showed leukocytosis accompanied by a “left shift” in the ratio of immature to mature neutrophils. Serum biochemistry profile showed increased haptoglobin and C-reactive protein. These clinicopathological results indicated a severe flogistic process.

Thoracic and cervical radiographs were normal. Abdominal ultrasound confirmed a diffuse subjective enlargement
of the spleen, but no other anomalies were noted. The dog was then placed under general anesthesia and underwent 16-multidetector-CT (GE Lightspeed 16, GE Medical Systems, Milwaukee, WI) examination of the neurocranium, cervical spine, and abdomen. For the neurocranium, unenhanced scans were obtained followed by an angiographic series. The acquisition, dose, and reconstruction parameters were axial modality, 2 s/rotation, caudal-to-rostral direction, 16 × 0.625 mm detector configuration, 120 kVp, 210 mAs, standard algorithm. For brain CTA, 640 mg I/kg of iodixanol (Visipaque 320, Amersham Health, Princeton, NJ) was injected at 37 °C at a rate of 4 mL/s using a power injector system (Stellant, Medrad, Indianola, IA) through a 20 gauge catheter placed in the left cephalic vein. An injection-to-scan delay of 10 sec. was used. Original data sets of 512 × 512 matrix size were transferred to a freestanding workstation (Advantage Workstation 4.1, GE Medical Systems, Milwaukee, WI) and postprocessed using multiplanar reformation (MPR), maximum intensity projection (MIP), and volume rendering (VR) techniques. Unenhanced CT of the neurocranium revealed a moderate diffuse hypoattenuation of the white matter. In the cervical spine, a slight dilation of the central canal was noted. These CT features were consistent with a diffuse encephalopathy and moderate cervical syringohydromyelia. The CTA series of the brain showed the two rostral cerebral arteries anastomosed with each other rostro-dorsally to the optic chiasm forming a single median artery. At the joining point there was a small saccular dilation, enhancing at the same degree of the arteries, consistent with a small aneurysm (Figures 1, 2, and 3). Measurements of the aneurysm were performed from magnified MPR images with an electronic caliper at the workstation. The aneurysm was 1.3 mm at the neck point, 2.0 mm in width, and 1.8 mm in height (Figure 4).

Contrast-enhanced abdominal CT revealed a saccular dilation of the prehepatic segment of the portal vein and a small dilation of the umbilical part of the left portal branch within the liver. These features were consistent with extrahepatic and intrahepatic portal vein aneurysms.

Cisternal cerebrospinal fluid (CSF) was collected at the end of the CT scans. CSF had 324 white blood cells/μL (reference range, ≤5 cells/μL), 142 red blood cells/μL, and 21 mg/dL protein (reference range, ≤25). Glucose concentration was 76 mg/dL (reference range, 53–104 mg/dL). The differential cell count indicated a neutrophilic and macrophagic pleocytosis (80% neutrophils and 20% macrophages).

Based on these findings, the final diagnosis was of aseptic meningitis. The dog was discharged with a long-term corticosteroid therapy. The owner reported that the dog had a rapid improvement, and at two-month follow-up evaluation, the cerebrospinal fluid examination was normal. CT of the neurocranium showed the rostral cerebral small aneurysm stable. The enhanced abdominal scan showed a large filling defect in the aneurysmal portal vein, consistent with thrombosed portal vein aneurysm. The small intrahepatic aneurysm was stable. Followup by CT scan and/or US of the abdomen was suggested.

3. Discussion

The overall schema of the blood supply to the cranial region and the brain, the histological structure of the walls of the arteries, and the basic pattern of the embryological development are essentially the same in dog as they are in man (Figure 5) [7, 11]. Many variations of the Circle of Willis have
been described in humans, and they play a significant role in the cerebral aneurysms formation, in terms of hemodynamic stress caused by variations [12, 13]. In contrast, variants of the Circle of Willis have been rarely described in dog so far [7, 14, 15]. In most dogs, the two rostral arteries form a common median trunk, as described in the present case.

In humans, various shear stresses (flow, turbulences, jet effects, and others) are known to produce aneurysms. These represent "luminal" aneurysmal vasculopathies in which it is postulated that these stresses induce pathogenetic changes in normal vessel walls. In contrast, structural vessel-wall diseases (inflammatory, infectious, collagen diseases, and others) are "abluminal" aneurysmal vasculopathies. In these, there is a primary abnormality of the vessel wall which is potentially aggravated by shear stresses [1, 3–6].

From the clinical point of view, most human patients have no symptoms or complaints until the aneurysm ruptures. Neurological signs in our patient were attributed to the meningitis. The meningitis of our Nova Scotia Duck
Tolling Retriever was diagnosed as aseptic meningitis, a noninfectious inflammatory disorder frequently diagnosed in this breed of dog [16]. Aetiological hypotheses of the cerebral aneurysm in our dog include congenital weakness or degenerative changes in vessel walls due to a connective tissue disorder or a local inflammatory process. This dog had simultaneous extrathoracic and intrathoracic portal vein aneurysms. Portal vein aneurysm is a rare condition we recently described in a series of dogs including the dog of this paper [17]. Aneurysm of the portal vein can be either congenital or acquired and thrombosis of the aneurysmal portal vein with its consequences is a possible complication. The development of more than one aneurysm at different sites is an uncommon event in human patients [18, 19] and it has been reported once in a dog [20].

This incidentally discovered cerebral aneurysm broads the spectrum of cerebrovascular diseases in dogs. Intracranial vessels can now be routinely assessed in living animals using CT and magnetic resonance (MR) imaging [21–24]. With the widespread use of these advanced imaging technologies, the potential to identify variants of the Circle of Willis or anomalies of the cerebrovascular system in dogs can substantially increase. Moreover, the non- or minimally invasive nature of these techniques, combined with the ability to obtain information about the brain parenchyma within the same examination, represents a clear advantage of CT-angiography (CTA) and MR angiography (MRA) over all other imaging techniques [21–23].

References
