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Case Report

Duplication of Caudal Vena Cava in a Cat

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Abstract

Caudal vena cava (CVC) may develop abnormally due to its complex embryogenesis. Understanding congenital variants such as duplication of CVC is essential for clinical interventions, especially performed by surgeons and radiologists. In this context, we summarize the imaging and clinical characteristics of CVC duplication and accompanying portosystemic shunt diagnosed in a six-month-old cat using computed tomography angiography. In our patient, the CVC branched into two vessels on either side of the abdominal aorta and merged into a single vessel at the level where the renal veins emerge. The duplication of the CVC, along with portosystemic shunts and ureter anomalies, can increase the risk of thromboembolism, especially in cats with heart disease. Due to the evolving nature of computed tomography technology in animals, the number of diagnoses made using this method is still relatively low. It is anticipated that the rate of CVC duplication in cats and dogs will increase as diagnoses become more frequent.

Keywords: Duplicated caudal vena cava, Venous anomaly, Diagnosis, Computed tomography, Angiography

Introduction

In mammals, the caudal vena cava (CVC) forms as a single vessel on the right side of the aorta as a result of a complex process that includes the development, regression, anastomoses and displacement of three pairs of embryonic vessels: supracardinal, subcardinal and vitelline vessels [1,2]. The presence of the Duplicated Caudal Vena Cava (dCVC) in the abdomen is most commonly due to the persistence of two embryonic supracardinal veins. The dCVC is physiological in whales and dolphins, but has been reported as a rare variant in humans and domestic animals. The prevalence of inferior vena cava duplication in humans has been reported at 0.2-3%. The frequency of dCVC occurrence varies between 3% and 27%, influenced by factors such as the species or breed of the animal and the type of diagnostic equipment employed [3]. It has been reported that awareness of abnormal retroperitoneal vessels is important to avoid diagnostic pitfalls and intraoperative complications during surgical or interventional procedures. The dCVC has rarely been reported in small animals. In reported cases, dCVC was primarily identified as an incidental finding during imaging for other reasons and was associated with other congenital anomalies [2]. When the collateral system ensures sufficient venous return, the majority of congenital venous anomalies, such as dCVC, typically remain undetected and produce no symptoms [2,4,5].

Reflecting on the findings from retrospective veterinary research on domestic animals, it has been observed that numerous variations of the CVC exist. The occurrence of these variants is closely associated with an increased risk of concurrent ureteric anomalies or the development of portosystemic shunts [1]. In this report, we present the symptoms and diagnosis of a rare anatomical variation of the CVC, known as dCVC, using computed tomography. The diagnosis of dCVC is important for recognizing clinical symptoms, preventing diagnostic errors during surgical interventions, and reducing intraoperative complications. Additionally, being aware of congenital anomalies that may accompany this anatomical variation is crucial for a comprehensive patient assessment and treatment planning. The purpose of this case report is to contribute to the enhancement of surgical and diagnostic approaches by addressing the clinical implications of dCVC.

Case History

Ethical Approval

Since this article is categorized as a case report, it is not subject to ethical committee approval. Informed consent was obtained from the animal owner to use the data obtained from the clinical examination.

Clinical Examination of the Cat

A 6-month-old Siamese male cat was admitted to Istanbul University-Cerrahpaşa Veterinary Faculty Animal Hospital with complaints of incoordination and seizures. The cat was reported to undergo daily seizures, particularly occurring after meals. The patient's rectal temperature was 38.5°C and heart rhythm was 160 beats per minute. Neurological examination showed increased tendon reflexes in the front and hind legs. Proprioception and cranial nerve examination were normal. It was reported that the patient had a decreased appetite recently. The patient underwent a blood sampling after a twelve-hour fasting period, during which fasting bile acids and ammonia levels were measured. Subsequently, the patient was fed, and two hours later, another blood sample was taken to analyze postprandial bile acid levels. At presentation, the patient's hemogram values were within normal limits *(Table 1)*, while biochemical parameters showed significant changes. Elevated levels of ammonia, fasting and postprandial bile acids, ALT, and ALP were noted. In contrast, a decrease in BUN levels was observed *(Table 2)*. The patient reported not using any medications.

Table 1. *Heamatological values of cat at presentation*

Test	Reference Value	Result
RBC, x10^6/µL	6.61	5.65-8.87
Hct, %	38.9	37.3-61.7
Hb, g/dL	9.9	13.1-20.5
MCV, fL	65.3	61.6-73.5
MCH, pg	15.0	11.8-17.3
MCHC, g/dL	36.0	32.0-37.9
RDW, %	25.6	13.6-21.7
Retic, %	0.6	
Retic, x10^3/µL	30.8	10.0-110.0
WBC, x10^3/µL	12.80	5.05-16.76
Neu, x10^3/µL	6.46	2.95-11.64
Lym, x10^3/µL	5.31	0.92-6.88

RBC: *red blood cell,* ***Hct:*** *haematocrit,* ***Hb:*** *hemoglobin* ***MCV:*** *mean corpuscular volume,* ***MCH:*** *mean corpuscular hemoglobin,* ***MCHC:*** *mean corpuscular hemoglobin concentration,* ***RDW:*** *red cell distribution width,* ***Retic:*** *reticulocyte,* ***WBC:*** *white blood cell,* ***Neu:*** *neutrophil* ***Lym:*** *lymphocyte*

Table 2. *Serum biochemical panel of cat at presentation*

Test	Reference Value	Result
Bile acids (fasting), µg mol/L	0.0-3.0	21.98
Bile acids (postprandial), µg mol/L	0.0-11.0	22.54
Ammonia, µg/dL	23.00-78.00	160
ALT, U/L	12-130	194
ALP, U/L	14-111	158
BUN, mg/dL	7.03-26.98	5.03
Glucose, mg/dL	85	74-143
CK, mg/dL	0,7	0.5-1.8
Calcium, mg/dL	8.6	7.9-11.3
TP, g/dL	5.8	5.2-8.2
Albumin, g/dL	2.5	2.3-4.0
Globulin, g/dL	3.9	2.8-4.8

ALT: *Alanine Aminotransferase,* ***ALP:*** *Alkaline Phosphatase* ***BUN:*** *Blood Urea Nitrogen,* ***CK:*** *Creatin,* ***TP:*** *Total Protein*

Computed Tomography (CT) Examination

CT, Siemens SOMATOM go. Now CT scanner with 32 detectors was used. The patient was given Propofol (Propofol-PF®) at 5 mg/kg for induction and Isoflurane (2%) in oxygen (Isoflurane USP®) for maintenance of anesthesia through the cuffed endotracheal tube. The patient was imaged in the supine position. To determine the scanning area, the topogram image was first taken, and then Opaxol™ contrast agent at a dose of 640 mg/kg was administered in the cephalic vein with a 20 gauge catheter at a speed of 3 mL/s using high-pressure syringe pump. The aortic lumen was manually marked, the predicted threshold contrast enhancement level (100 HU) was set and scanning was initiated. Repeated images taken from a single section in which the aortic lumen was marked were obtained using 'bolus tracking' technique. When the prescribed threshold contrast enhancement level was reached, the scanning device was automatically activated. Sections from the caudal thorax to the pelvis were taken and arterial phase images (0.70 mm collimation, 1 mm section thickness, 1.5 mm table speed per gantry rotation) were obtained. The case was also imaged in portal phase (30 sec after contrast) at the same collimation and section thickness. Then, dorsal, sagittal, maximum intensity projection (MIP), Multiplanar Reformat (MPR) and volume rendering images were created from the transversal images.

Duplicated caudal vena cava was detected incidentally during CT angiography scan. The portal vein diameter was measured as 1.8 mm and was relatively thin in calibration. It was determined that the cranial mesenteric vein was combined with the splenic vein and formed the portal vein. However, in cranial mesenteric vein, there was a varicose dilatation of 16 mm in length and 3.9 mm in width, located in the central abdomen, at the right lateral of the midline, approximately at the level of the caudal pole

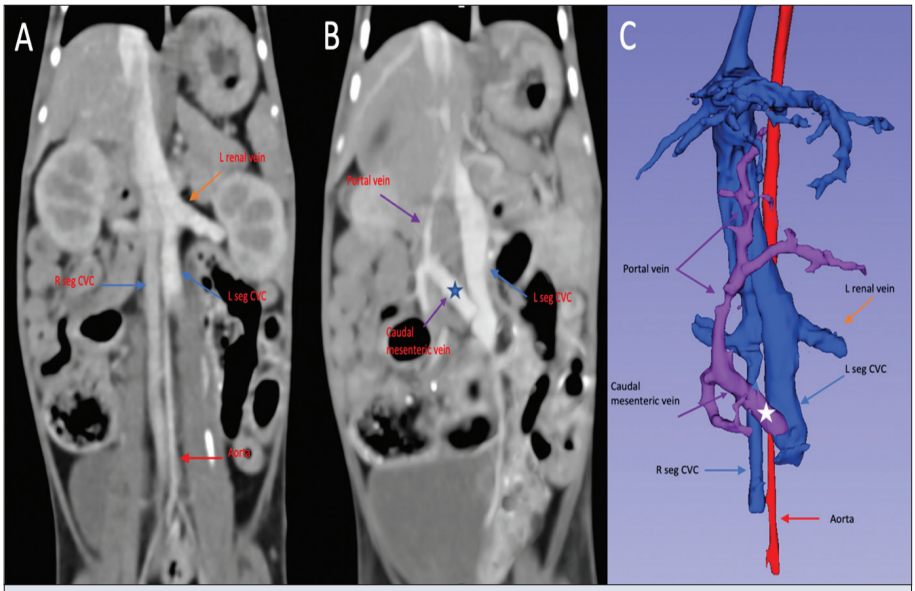

Fig 1. Duplication of the caudal vena cava and demonstration of a portosystemic shunt, **A, B:** Abdominal contrast-enhanced computed tomography image, ventro-dorsal section projection was used, **B:** Caudal mesenteric vein, in communication with the left segment of CVC (left subracardinal vein). CVC, vena cava. L, left. L seg CVC, left segment of the vena cava. R seg CVC, right segment of the vena cava. (star) Shows the direct communication between the caudal mesenteric vein and the CVC in CT image in portal phase, **C:** Image of dorsoventral 3D reconstruction showing the shunt vessel between the left segment of the CVC and the mesenteric vein

of the right kidney. A joint appearance with a diameter of 1.5 mm between the described vein and the left branch of the dCVC was found to be compatible with shunt. It was determined that the dCVC merged approximately at the level of the left renal vein and entered the hepatic hilus as a single caudal vena cava *(Fig. 1)*.

Discussion

Caudal Vena Cava duplication is among several congenital anomalies that may have potential clinical implications. However, this condition is usually asymptomatic and often discovered incidentally during the routine imaging techniques.

The development of the inferior/caudal vena cava in humans and domestic animals has not yet been fully elucidated, and various developmental theories have been suggested. Our case exhibited a rare instance of complete duplication among the variations of the caudal vena cava. In complete duplication, CVC takes a double form by dividing into two branches in the renal and pre-renal parts. Renal veins arise separately from both branches [1].

The presence of CVC variations significantly elevates the likelihood of concurrent development of ureter anomalies and portosystemic shunts [2,4,5]. In our patient, the cranial mesenteric vein was connected to the left branch of the dCVC through a shunt (cranial mesenteric-caval shunt). White et al.[6] found a shunt between the cranial mesenteric vein and CVC in three cats included, two of which having duplicated CVC. Especially in cats, the presence of dCVC may increase the likelihood of observing a cranial mesenteric-caval shunt.

Clinical symptoms that may result from this anomaly include deep vein thrombosis and pulmonary embolism in the lower extremities in humans [7]. In particular, Vena Cava anomalies have become a recognized risk factor for deep vein thrombosis of the lower extremities in young people [8]. One study found potential Vena Cava abnormalities in spontaneous, unprovoked deep vein thrombosis in 5% of young patients. In humans, the rate of Inferior Vena Cava (IVC) thrombosis in patients with Congenital Inferior Vena Cava (CIVC) anomaly is 60% to 80% [1]. IVC duplication may create a tendency to venous stasis and therefore venous thromboembolism due to insufficient blood circulation [9,10]. Myocardial heart diseases are common in cats. Thromboembolism is very common especially in these animals [11]. dCVC may be an additional factor to increase the risk of thromboembolism in these patients, as in humans.

dCVC is usually detected during surgery, necropsy or CT angiography [12]. This is thought to be due to the newly developing imaging methods in veterinary medicine. We predict that as the number of CT scans increases, the number of dCVC diagnosis will increase. The symptoms in our case were compatible with portosystemic shunt, however the effects of dCVC on the patient's clinical condition could not be evaluated.

In conclusion, it is important to evaluate patients for dCVC, especially those diagnosed with development of

ureter anomalies, congenital portosystemic shunts and thromboembolism. This case report provides significant insights into the diagnosis of dCVC. It offers valuable understanding in defining and comprehending this rare condition, thereby enriching the existing information in the literature. However, the scarcity of similar cases in the current literature limits the broader contextual evaluation of our findings.

Availability of Data and Materials

The data that support the findings of this case report are available from the corresponding author (Y. Kocak) upon reasonable request.

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We would like to thank VETMR for conducting the computed tomography scan.

Competing Interests

The authors declared that there is no conflict of interest.

Author Contributions

Clinical examinations were conducted by YK. The interpretation of the computed tomography scans was done by YK and ZM. YK wrote the article.

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