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CT findings of an incidental segmental aplasia of the caudal vena cava with azygos continuation in a guinea pig (*Cavia porcellus*)

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Abstract

Background: Segmental aplasia of the caudal vena cava (CVC) with azygos continuation is a congenital malformation macroscopically described in mammals including humans, dogs, and rodents. It is usually detected as an incidental finding and the final diagnosis is reached by computed tomography (CT), fluoroscopy, or post-mortem dissection.

Case Description: A 3-year-old guinea pig (*Cavia porcellus*) presented with subacute dyspnea. A computed tomographic examination was performed for the evaluation of subtle pulmonary changes previously suspected on conventional radiography, and a segmental aplasia of the CVC with azygos continuation was identified as an incidental finding.

Conclusion: According to database negative results, this is the first report describing a segmental aplasia of the CVC and azygos continuation in a guinea pig by CT.

Keywords: Small mammal, Interruption caudal vena cava, Vascular malformation, Congenital malformation.

Introduction

The normal anatomy of the caudal vena cava (CVC), termed the inferior vena cava (IVC) in humans, consists of a single vessel, right-sided to the aorta, formed by the confluence of bilateral common iliac veins at the level of the aortic trifurcation. There are five distinct segments along its pathway to the right atrium: prerenal, renal, prehepatic, hepatic, and posthepatic. The embryogenesis of the CVC is a complex process involving the development, regression, anastomosis, and replacement of paired and symmetric embryonic veins, and during this process, different congenital variants may develop (Chuang *et al.*, 1974; Hunt *et al.*, 1998; Bass *et al.*, 2000; Cornillie and Simoens, 2005; Li *et al.*, 2021).

Segmental aplasia of IVC is infrequently reported in human medicine with a prevalence of 0.6% (Bass *et al.*, 2000; Li *et al.*, 2021). In this condition, there is no communication between the prehepatic and hepatic segments of the IVC. As a result, the renal segment of the IVC continues with the azygos (right-sided) or hemiazygos (left-sided) vein. In dogs, segmental aplasia of the CVC with azygos/hemiazygos continuation has also been described, usually detected as an incidental finding in imaging studies performed for diagnosing other vascular anomalies such as portosystemic shunts, or during laparotomy or anatomic dissection (Barthez

et al., 1996; Hunt *et al.*, 1998; Harder *et al.*, 2002; Fischetti and Kovak, 2008; Schwarz *et al.*, 2009). Although no prevalence has been documented in canine patients, the incidence of this anomaly is thought to be higher than expected and it is now more frequently reported because of the increased use of advanced diagnostic imaging techniques (Schwarz *et al.*, 2009). Congenital CVC anomalies in *Cavia porcellus* are poorly described. The necropsy findings of an absent CVC in a Brazilian guinea pig (*Cavia aperea*), were published (Phisalix, 1898). However, database searches (PubMed, CAB, and Scopus) covering the years 1950–2023 were negative for segmental aplasia of the CVC in *Cavia porcellus*. In this case report, we present the computed tomography (CT) findings of a segmental aplasia of the CVC and azygos continuation in a guinea pig.

Case Details

A 3-year-old, male castrated, 0.980-kg, American guinea pig (*Cavia porcellus*) was referred to the exotic animal service of VetOeiras Veterinary Hospital (Oeiras, Portugal) for progressive respiratory sounds and dyspnea for 1-week duration. Previous medical history was unremarkable.

These were experienced owners and provided excellent husbandry and care. The animal was fed a high-quality timothy hay diet and fresh water *ad libitum*. In addition,

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150 g of fresh mixed vegetables daily and a tablespoon of guinea pig pellet diet. The cage was cleaned biweekly and the bedding was fleece cage liners. On physical examination, the patient was active, alert, normothermic (39°C), and maintained an ideal body score. Pulmonary auscultation revealed mild tachypnea (134 breaths/minute) with marked respiratory sounds including rhonchi and bilateral crackles, worse on the right side, while the cardiac auscultation was normal with a heart rate of 260 beats/minute. At this time, differentials were made for respiratory distress and included pneumonia, pulmonary edema, upper respiratory tract infection, allergic respiratory disease, or mass-like lesions. Hematology and a biochemistry panel along with thoracic radiographs were proposed to and accepted by the owners. The patient was administered intramuscular butorphanol 0.5 mg/kg (Dolorex 10 mg/ml; Intervet International GmbH, Unterschleissheim, Germany) for peripheral blood collection, in the right *vena saphena lateralis*, and diagnostic imaging of the thorax. Hematology revealed elevated white blood cell count ($14.70 \times 10^3/\mu\text{l}$; reference range $1.9\text{--}11.70 \times 10^3/\mu\text{l}$) with eosinophilia ($1.36 \times 10^3/\mu\text{l}$; reference range $0\text{--}0.71 \times 10^3/\mu\text{l}$), heterophilia ($7.22 \times 10^3/\mu\text{l}$; reference range $0.44\text{--}6.80 \times 10^3/\mu\text{l}$) and lymphocytosis ($6.95 \times 10^3/\mu\text{l}$; reference range $0.48\text{--}4.82 \times 10^3/\mu\text{l}$). The RBC count, hemoglobin, and hematocrit were within normal limits while the biochemistry was unremarkable.

The reference values were taken from the previously published literature (Spittler *et al.*, 2021). Thoracic radiographs were performed and revealed a mixed alveolar-interstitial opacity at the region of the right caudal lung lobe.

Due to the age of the patient, clinical presentation, and radiographic findings, pneumonia was speculated as the main differential. A primary lung neoplasia, such as a pulmonary adenoma, was considered less likely. Antibiotic treatment was administered (enrofloxacin 5 mg/kg PO BID for 2 weeks) with no relevant improvement of the clinical signs nor resolution of the radiographic findings at the 2-week follow-up. A CT evaluation of the thorax was performed to rule out other concurrent conditions such as the presence of lung mass-like lesions.

The patient was pre-anesthetized with a combination of intramuscular 0.5 mg/kg butorphanol and 1 mg/kg midazolam (Midazolam 15 mg/5 ml, Labesfal, Santiago de Besteiros, Portugal), followed by induction with alfaxalone (1 mg/kg diluted injected slowly in the cephalic vein; Alfaxan 10 mg/ml; Jurox Limited; Dublin; Ireland) and maintained with inhaled 2% of isoflurane (Isoflurine 1,000 mg/g; VETPHARMA ANIMAL HEALTH, S.L.; Barcelona, Spain) in a 1.5% flow rate of Oxygen with a facemask. A whole-body CT (Somatom, Siemens) examination was performed with the following technical parameters: 110 Kvp, 70 mA, 0.8 pitch, 1 mm slice thickness, and 0.9 mm slice interval. Pre-contrast images were reconstructed

using soft tissue and lung algorithms and post-contrast images were reconstructed using a soft tissue algorithm. The contrast used was Omnipaque (300 mgI/ml; GE Healthcare, Oslo, Norway) administered at a 300 mgI/kg IV dose through a 26G catheter (Kruuse Venocan Mini IV) placed in the right cephalic vein. The CT revealed multifocal areas of increased attenuation affecting the right cranial, middle, and caudal lung lobes, as well as the left cranial lung lobe, mainly with a ventral distribution except for the right caudal lobe which was affected caudodorsally. In addition, a vascular anomaly affecting the CVC and azygos veins was detected. Multiplanar maximum intensity projections (MIPs) and 3D volume renderings reconstructions were evaluated for a better depiction of the vascular structures (Fig. 1). Immediately cranial to the renal segment, the CVC extended craniodorsal and right-sided, medial to the right kidney and dorsal to the right adrenal gland. It crossed into the *hiatus aorticus* and shared its anatomical intrathoracic pathway abnormally with the azygos vein, which was overtly dilated. A post-hepatic component of the thoracic portion of the CVC was normally detected draining into the right atrium. Communication between the pre-hepatic and hepatic segments of the CVC could not be identified, as reported by CT description in other species. These findings were consistent with a segmental aplasia of the CVC with azygos continuation (Chuang *et al.*, 1974; Bass *et al.*, 2000; Li *et al.*, 2021).

Although this vascular anomaly can predispose to thrombus formation and pulmonary thromboembolism (Newman *et al.*, 2020), this was not considered in our case as no aneurysmal dilation of the CVC or filling defects in the pulmonary arteries were identified. Hence, it was deemed an incidental finding unrelated to the clinical signs, whereas the pulmonary changes were probably attributed to an infectious pulmonary process. Tracheo-bronchial lavage was proposed after a CT examination but refused by the owners due to financial restrictions. The patient recovered uneventfully and was discharged the same day. A new antibiotic treatment was started (trimethoprim + sulfamethoxazole 30 mg/kg BID for 30 days), with complete resolution of the clinical signs and radiographic findings at 1-month follow-up.

Discussion

Descriptions of vascular anomalies implying the CVC in guinea pigs (*Cavia porcellus*) are limited to a solitary scientific publication in which the abdominal cavity of euthanized laboratory guinea pigs was observed macroscopically, and duplication of the CVC was frequently recognized, with a 30% prevalence in males and 24% in females (Nakamura *et al.*, 2019). Multiple cardiovascular anomalies were described in another case report of a guinea pig, none of them affecting the CVC (Haris *et al.*, 1976). An absence of the CVC was

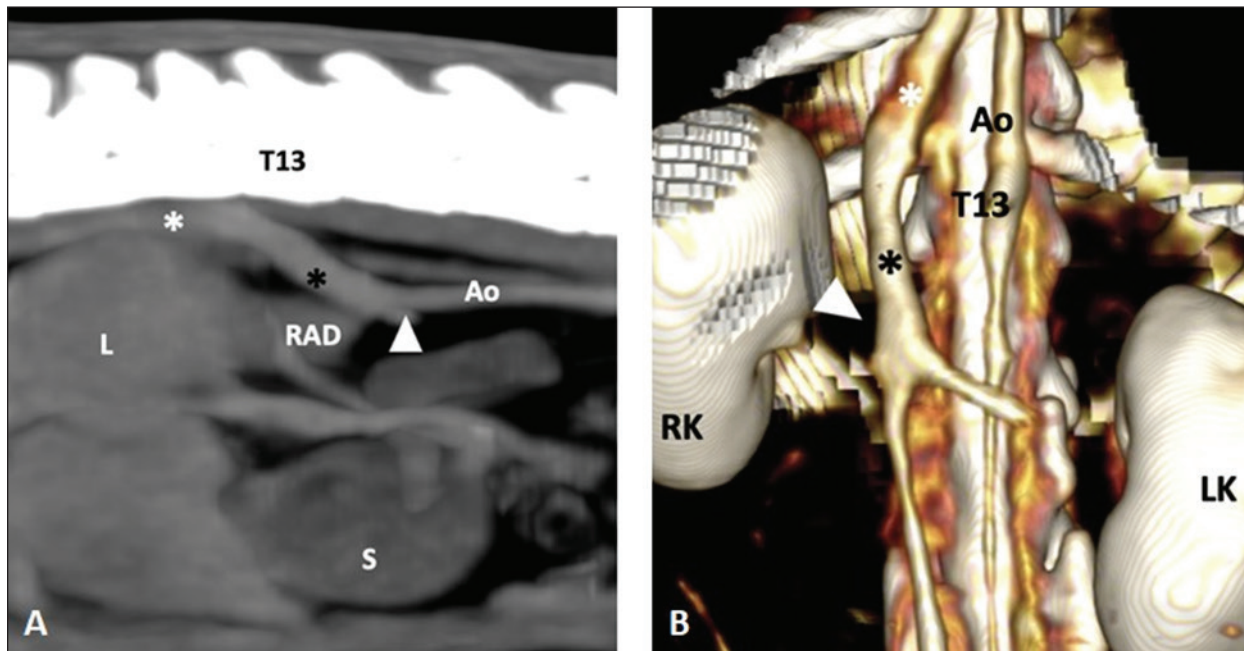


Fig. 1. (A) Parasagittal post-contrast MIP image (WL 40 HU, WW 300 HU) of the abdomen of a 3-year-old, male castrated, 0.980-kg, American guinea pig in which an abnormal communication (black asterisk) is detected between the post-renal segment of the CVC (white arrow) and the right azygous vein (white asterisk). Cranial is to the left of the image. (B) Dorsal 3D view highlighting the anatomy of the CVC and showing the abnormal communication between both vessels. Cranial is at the top of the image. T13: 13th thoracic vertebra, Ao: aorta, RK: right kidney, LK: left kidney, L: liver, S: stomach, RAD: right adrenal gland.

described in a *Cavia aperea*, however no CT findings were reported (Phisalix, 1898).

The embryogenesis of the CVC (IVC in humans) is widely illustrated in the literature (Chuang *et al.*, 1974; Barthez *et al.*, 1996; Bass *et al.*, 2000; Cornillie and Simoens, 2005; Li *et al.*, 2021) and related to the paired posterior cardinal, subcardinal, and supracardinal embryonic veins. If failure occurs at the time of fusion between the right subcardinal and hepatic (formerly vitelline) veins, then the communication between the postrenal CVC and the cranial portion of the supracardinal vein (precursor of the right azygos or left hemiazygos vein) will be patent (Hunt *et al.*, 1998; Bass *et al.*, 2000; Fischetti and Kovak, 2008; Li *et al.*, 2021). Other explanations have been proposed according to different embryologic models of the CVC genesis (Barthez *et al.*, 1996). It results in the venous blood flow from the renal and post-renal segments being shunted into the azygous or hemiazygos (less frequently) vein with no communication between the pre-hepatic and hepatic segments of the CVC. Although right and left azygos veins are present in the embryo, the pattern is later commonly simplified: in the horse, dog, and cat the right vein persists while in the pig the left or occasionally both persist; in ruminants, both veins are usually present (König and Liebich, 2020). This could lead to variants in this vascular anomaly depending on the species-specific azygos system. In guinea pigs, it is derived from the right postcardinal vein (Potter *et al.*,

1958) which is consistent with the right-sided azygos identified in our case.

In human and canine patients, most of the cases with this condition are detected incidentally (Harder *et al.*, 2002). Nevertheless, it has also been reported in symptomatic patients associated with thrombosis of the CVC and/or renal veins, and pulmonary thromboembolism (Harder *et al.*, 2002; Lambert *et al.*, 2010; Lockwood *et al.*, 2018; Newman *et al.*, 2020), as well as with other congenital malformations such as polysplenia, situs anomalies or portosystemic shunt (Bass *et al.*, 2000; Fischetti and Kovak, 2008; Li *et al.*, 2021). In the case described herein, the patient had a CT examination as a part of a diagnostic investigation of clinical signs related to the respiratory system, and the abdomen was included in the protocol to rule out clinically relevant comorbidities.

The clinical signs and thoracic CT findings did not appear to be related to the vascular anomaly, which was considered an incidental finding. Consequences of this type of congenital defect, other than the previously mentioned thromboembolic events, include implications for renal surgery and cardiac catheterization in humans, leading to potential hemorrhagic events or vascular injury when not detected in the presurgical planning (González *et al.*, 2017). Another consideration is described in dogs with right adrenal tumors in which a CVC venectomy was required, even with no vascular

invasion, when the adrenal gland was displaced ventrally relative to the CVC (Takagi *et al.*, 2021). While CT imaging is not as commonly employed in small mammals like guinea pigs compared to dogs and cats, its utility extends to various clinical scenarios due to its inherent cross-sectional nature, which mitigates the superimposition of anatomical structures. Indications include evaluation of dental disease and other bony structures of the head (Krautwald-Junghanns *et al.*, 2011) or assessment of the thoracic and abdominal cavities for anatomical and medical purposes (Parkinson *et al.*, 2017; Zehtabvar *et al.*, 2023). Furthermore, the injection of intravenous contrast medium enhances the vascular structures of the body and allows the evaluation of potential vascular malformations.

Conclusion

In conclusion, this is the first case report describing the tomographic features of a congenital segmental aplasia of the CVC with azygos continuation in a *C. porcellus*. This finding is usually asymptomatic, but potential clinical or surgical implications emphasize the importance of its identification.

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Conflict of interest

The authors have no conflict of interest to declare.

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Author contributions

Antón Costas: Main author of the manuscript and literature review. Carolina Magro and Pierantonio Battiato: Co-authors of the paper. Vicente Cervera: review of the process and manuscript.

Data availability

The data supporting the findings of this study are available within the article.

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