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# Persistent left cranial vena cava draining into the left atrium associated with pulmonary stenosis in a French bulldog<sup>☆</sup>

Alessandro Zani, DVM<sup>\*</sup>, Elisa Becchetti, DVM, Paola Leonardi, DVM, Alessandro Sinatra, DVM

*Cardiovet Clinic, Via dei Pelaghi 100-102, 57124 Livorno, Italy*

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## KEYWORDS

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**Abstract** A 5-month-old female French bulldog was evaluated for the presence of a heart murmur. Through clinical and echocardiographic evaluations, a severe Type A pulmonary stenosis was diagnosed. Angiography during right ventricular catheterization for valvuloplasty revealed drainage from a persistent left cranial vena cava (PLCVC) into the left atrium; this was confirmed later by contrast echocardiography. This report is the first to describe this anatomical variant of a PLCVC in a dog. © 2014 Elsevier B.V. All rights reserved.

<sup>\*</sup> A unique aspect of the Journal of Veterinary Cardiology is the emphasis of additional web-based images permitting the detailing of procedures and diagnostics. These images can be viewed (by those readers with subscription access) by going to <http://www.sciencedirect.com/science/journal/17602734>. The issue to be viewed is clicked and the available PDF and image downloading is available via the Summary Plus link. The supplementary material for a given article appears at the end of the page. Downloading the videos may take several minutes. Readers will require at least Quicktime 7 (available free at <http://www.apple.com/quicktime/download/>) to enjoy the content. Another means to view the material is to go to <http://www.doi.org> and enter the doi number unique to this paper which is indicated at the end of the manuscript.

<sup>\*</sup> Corresponding author.

E-mail addresses: [info@cardiovet.eu](mailto:info@cardiovet.eu), [ale.zani@tin.it](mailto:ale.zani@tin.it) (A. Zani).

A 5-month-old female French bulldog, weighing 4 kg, was referred for evaluation of a heart murmur. At the time of the first visit, the dog showed a slight cyanosis inducible after exertion or excitement in the absence of polycythaemia (haematocrit: 36.5%, reference range 37.0–61.7%). At this stage it was not possible to determine the oxygen saturation with plethysmography or blood gases. Serum chemistry panel results were normal, and complete blood count results were in the normal range for puppies (red blood cells: 5.42 M/ $\mu$ l, reference range 5.65–8.87 M/ $\mu$ l; haematocrit: 36.5%, reference range 37.0–61.7%; haemoglobin: 12.9 g/dl, reference range 13.1–20.5 g/dl). Auscultation revealed a grade IV/VI systolic ejection murmur with the

### Abbreviations

PLCVC persistent left cranial vena cava

point of maximum intensity at the left heart base. A wide cleft palate with partial absence of the soft palate was present as well. Thoracic radiographs with right lateral and ventrodorsal projections revealed moderate right atrial and ventricular enlargement and a bulge in the area of the pulmonary artery. The ECG revealed a sinus rhythm with criteria for right ventricular enlargement. Echocardiography demonstrated the presence of moderate right ventricular concentric hypertrophy, slight thickening of the pulmonary valve with doming motion, and the pulmonary annular diameter was slightly reduced compared to the aortic annulus: pulmonary annulus (7.7 mm); aortic annulus (8.2 mm). These changes were compatible with a type A valvular pulmonary stenosis (Video 1). The Doppler examination showed a severe stenotic gradient (maximum velocity of 5.8 m/s; maximum systolic pressure gradient of 134 mmHg). A search for the dilation of the coronary sinus in the right parasternal long-axis view, suggestive of a persistent left cranial vena cava (PLCVC), yielded negative results. On the basis of the echocardiographic findings, the dog was diagnosed with severe pulmonary stenosis. Treatment was begun with atenolol at a dosage of 1 mg/kg PO q 12 h, and percutaneous balloon valvuloplasty was recommended. For the valvuloplasty, vascular access from the left jugular vein was chosen. A cut-down technique was used to insert an 8F vascular introducer.<sup>a</sup> To perform right ventriculography, a pigtail catheter was introduced via the left jugular vein. Instead of progressing into the right atrium as expected, the catheter reached the left atrium after taking an anomalous dorsal path (Fig. 1A and B). Following the injection of iodinated contrast material,<sup>b</sup> left atrial-ventriculography was obtained (Fig. 1A–C; Video 2), confirming the persistence of the left cranial vena cava that drained into the left atrium. No interatrial shunt was highlighted during the injection of contrast into the left atrium, and there was no evidence of a coronary anomaly type R2A associated with pulmonary stenosis (Video 2). Because of the impossibility of accessing the right chambers through the left jugular vein due to the

presence of the PLCVC, right jugular vein access was chosen. Using the right jugular vein access (cut-down technique and insertion of an 8F vascular introducer), it was impossible to advance the guidewire and the pigtail catheter into the right ventricle to perform right ventriculography. Angiography obtained through the pigtail catheter in the right atrium confirmed pulmonary stenosis and post-stenotic dilatation of the pulmonary artery (Video 2). To visualize the path of the right cranial vena cava, angiography was performed with an injection of contrast material through the introducer placed in the right jugular vein, which showed the abnormal course of the cranial vena cava, taking a dorsal and caudal arch and draining caudally into the right atrium; most likely due to right atrial enlargement (Fig. 1D; Video 2). Subsequently, the procedure was discontinued due to a severe reaction to the iodinated contrast consisting of hypotension, bradycardia, and oropharyngeal oedema. Afterwards, two separate contrast echocardiographic examinations were performed to better visualize the course of the venous inflow of the PLCVC, right vena cava, and caudal vena cava for a subsequent attempt at valvuloplasty with a femoral venous approach. Echocardiographic contrast<sup>c</sup> was injected through the right femoral vein and echocardiographic images were obtained from the right parasternal short-axis and left apical four-chamber views demonstrating normal filling from the caudal vena cava and normal sequence of opacification of the cardiac chambers. Agitated saline was then injected (bubble study) into the left cephalic vein with rapid and intense sequential opacification of the left atrium and left ventricle, confirming the presence of a PLCVC with right-to-left shunting (Fig. 2; Video 1). A retrospective analysis of earlier Doppler studies identified the flow from the PLCVC into the left atrium.

Valvuloplasty was performed 7 months later through a left femoral venous approach and immediately resulted in a reduction in the mean peak pulmonary Doppler gradient from 154 mmHg to 37 mmHg. The femoral arterial blood gas revealed a low oxygen pressure (PO<sub>2</sub>: 75 mmHg, reference range 90–100 mmHg) and reduced oxygen saturation of 92% (reference range 93–100%) in the absence of polycythaemia (haemoglobin: 19.6 g/dl, reference range 13.1–20.5 g/dl; haematocrit: 54.2%, reference range 37.0–61.7%) although clinical signs of cyanosis during exercise decreased in the 3 months following valvuloplasty.

<sup>a</sup> Pinnacle, Terumo, Ann Arbor, MI, USA.

<sup>b</sup> Visipaque, 320 mg/ml, GE Healthcare, Oslo, Norway.

<sup>c</sup> SonoVue, Bracco International B.V., Amsterdam, Netherlands.

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