NOTE  Surgery

Computed Tomography Angiography of Situs Inversus, Portosystemic Shunt and Multiple Vena Cava Anomalies in a Dog

Heejin OU1), Jisun KIM1), Yeonho BAE1), Juyeon OH1), Seungjo PARK1), Gahyun LEE1), Sunghoon JEON1) and Jihye CHOI1)*

1)College of Veterinary Medicine, Chonnam National University, Gwangju 500–757, Korea

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ABSTRACT. A 5-year-old Shih Tzu was presented with intermittent vomiting and anorexia. Microhepatica and reversed position of the abdominal organs were observed on radiography. Ultrasonographically, portosystemic shunt (PSS) was tentatively diagnosed. Computed tomography (CT) revealed that the distended portal vein drained into the left hepatic vein. The caudal vena cava (CdVC) split postrenally and converged at the renal level. Cranial to this, the azygos continuation of the CdVC was confirmed. In the thorax, a persistent left cranial vena cava (CrVC) was found along with right CrVC. This is the first report of a dog with persistent left CrVC and multiple abdominal malformations. CT angiography was useful in evaluating the characteristics of each vascular anomaly and determining the required surgical correction in this complex case.

KEY WORDS: azygos continuation of the caudal vena cava, CT, persistent left cranial vena cava, portosystemic shunt, situs inversus.

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Situs inversus is a congenital condition in which the visceral position is reversed as a mirror image. This condition is the main characteristic of Kartagener’s syndrome with rhinosinusitis and bronchiectasis [7]. Situs inversus without dextrocardia has been reported with congenital vascular anomalies including portosystemic shunts (PSS) and azygos continuation of the caudal vena cava (CdVC) [10, 13, 15].

The abdominal CdVC is divided into prerenal, renal, prehepatic, hepatic and posthepatic segments derived from the supracardinal, subcardinal and vitelline veins [10]. The retained middle segment of the supracardinal vein, normally regressed during normal development, plays a role as the connection between the embryonic CdVC and azygos vein, that is, the azygos continuation of the CdVC. The developing CdVC, usually a prehepatic CdVC, can be inappropriately connected with the caudal portion of the vitelline vein developing into a portal branch, and this abnormal connection between the vessels is a PSS [21].

Persistent left cranial vena cava (CrVC) is an uncommon congenital defect [1]. During embryologic development, the caudal portion of the left cranial cardinal vein, normally atrophied with the fusion of the left and right cranial cardinal veins, persists, resulting in a persistent left CrVC. This condition has been described with other congenital defects of the cardiovascular system [1, 2, 6, 11, 12, 19, 22].

To the authors’ knowledge, this is the first report documenting persistent left CrVC with congenital abdominal anomalies including situs inversus, PSS, azygos continuation of the CdVC and split CdVC in a dog. We described the application of CT angiography for distinguishing and comprehending these multiple vascular anomalies and situs inversus in the dog herein.

A 5-year-old, neutered male Shih Tzu was presented with a 2-week history of vomiting and anorexia. On physical examination, there were no abnormal findings including on auscultation. The dog’s complete blood count and serum chemistry were within the reference ranges, except for hepatic enzymes, such as alanine aminotransferase (968 U/l; reference range, 19–100 U/l), gamma-glutamyl transferase (22 U/l; reference range, 0–6 U/l), alkaline phosphatase (280 U/l; reference range, 15–127 U/l) and ammonia (143 µmol; reference range, 0–98 µmol).

On radiography, microhepatica and reversed position of the abdominal organs with normal thoracic visceral position were found (Fig. 1). Ultrasonography revealed decreased vascular size of the intrahepatic portal vein and hepatic vein and normal echotexture of the hepatic parenchyma. A dilated, tortuous portal branch showing an atypical portal flow signal was found in the left cranial abdomen, and PSS was tentatively diagnosed. Apart from the vessel suspected to be a PSS, there were many tortuous vessels, and it was challenging to determine the characteristics of the tortuous vessels, because of concurrent situs inversus.

To identify the suspicious vessels, CT angiography was performed at 30 sec after injection of 800 mg iodine/kg iohexol (Omnihexol 300, Korea United Pharm Co., Seoul, Korea) at a rate of 3 ml/sec using a power injector (Medrad Vistron CT Injector System, Medrad, Inc., Warrendale, PA, U.S.A.). The distended portal vein showed a convoluted course from the mid to left cranioventral abdomen and joined the left hepatic vein (Fig. 2). A single extrahepatic PSS was confirmed. Additionally, two separate CdVC branches converged at the renal level, and then a single CdVC coursed dorsally and connected

*Correspondence to: CHOI, J., College of Veterinary Medicine, Chonnam National University, Yongbong-ro, Buk-gu, Gwangju, 500–757, South Korea.
e-mail: imsono@chonnam.ac.kr
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directly to the azygos vein with the prehepatic CdVC being absent (Fig. 3). The positions of the stomach, duodenum, spleen, colon, cecum and kidneys, which had normal shapes and internal structures, were reversed. In the thorax, a left CrVC driving caudally to the caudodorsal angle of the heart and draining into the right atrium was found along with the right CrVC (Fig. 4). There was no other cardiovascular defect, and this was double checked with echocardiography. On the basis of these findings, the dog was diagnosed as having situs inversus; abdominal vascular anomalies including PSS, azygos continuation of the CdVC and split CdVC; and a thoracic vascular anomaly, persistent left CrVC.

Among these lesions, PSS was considered the cause of the clinical signs in the dog, and other congenital malformations were determined to be incidental findings. The PSS was surgically corrected using an ameroid constrictor, and then clinical signs and laboratory data returned to normal.

Situs inversus with rhinosinusitis and bronchiectasis are classified as Kartagener’s syndrome [3, 7, 18]. In Kartagener’s syndrome, chronic coughing and nasal discharge are caused by defective cilia motility. Cilia dyskinesia was ruled out in this case because there were no respiratory signs or evidence of bronchiectasis on thoracic radiography, even though electron endoscopy was not performed. Without primary ciliary dyskinesia, situs inversus may not lead to clinical signs. However, this condition can interfere with diagnostic and surgical procedures that require an understanding of the anatomy of the viscera and vascular structures like in this dog.

Ultrasoundography has high sensitivity and specificity as a means of detecting PSS, although it does depend on the examiner’s skill and experience [4]. In this case, PSS was suspected based on ultrasonography; however, the location and number of shunts and the connected vessels could not be clarified due to visceral malposition. Furthermore, anatomic comprehension of other tortuous vascular branches was needed.

CT is an excellent tool for evaluating the morphology of
anomalous vessels, as it provides images of the transverse plane without superimposition of structures and can be used to assess multiple complex anomalies using reconstructed three-dimensional images. CT was useful to not only anatomically discriminate the congenital malformations but also to create a treatment plan based on understanding the hemodynamic state among the 4 vascular anomalies in the dog. Surgical intervention was only necessary for the PSS classified as a single extrahepatic shunt, and other vascular abnormalities, such as azygos continuation of the CdVC, split CdVC and persistent left CrVC, should be avoided unnecessary surgical treatment for hemodynamic circulation.

In azygos continuation of the CdVC, the shunt provides a direct route for venous return to the heart, so that accidental ligation of the shunt can be fatal, as shown in previous case reported in the human literature [5]. One other point claiming our attention in this anomaly is thrombosis, which can occur due to slowing blood flow of the shunt [9, 10]. We could define the functional shunt between the azygos vein and renal CdVC and rule out thrombosis, shown as a filling defect, using CT angiography.

Split CdVC is the duplication of the prerenal and renal CdVC and has no clinical significance [16, 17, 20]. However, this anomaly added complexity to the abdominal vessel anatomy in this case.

During CT scanning, persistent left CrVC was found incidentally in the dog. Persistent left CrVC can be classified as a complete or incomplete type depending on whether distal portion of the left CrVC atrophies [1]. The incomplete type of this vessel can form a ring with the trachea and constrict the esophagus, leading to megaesophagus [14]. Moreover, the clinical significance of this malformation can be determined based on the structure it enters. This has no clinical significance when it enters the right atrium, unlike the case when it enters the left atrium. The present case was a complete type of persistent left CrVC that entered the right atrium without obstructing other structures, such as the esophagus.

Coexistence of situs inversus, PSS and azygos continuation of the CdVC has been reported in 2 other dogs previously [10, 15]. Persistent left CrVC has been reported in association with cardiac defects or other types of thoracic vascular anomalies [1, 2, 6, 8, 11, 12, 19, 22]. A hypothesis concerning genetic factors related to these vascular anomalies has been postulated; however, there has been no case in which persistent left CrVC and abdominal visceral and vascular malformations were in synchrony.

A lot of different types of congenital vascular malformations can simultaneously occur, and the clinical significance
of each type may be different. Moreover, if the visceral position is reversed, it can add difficulty to understanding the characteristics of vascular anomalies. This dog had complicated malformations including situs inversus, PSS, azygos continuation of the CdVC, split CdVC and even persistent left CrVC. Using CT angiography, the anatomy and function of each anomaly could be evaluated in detail, and surgical intervention was successfully performed.

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REFERENCES

1. Buchanan, J. W. 1963. Persistent left cranial vena cava in dogs: Angiocardiography, significance, and coexisting anomaliesI. Vet. Radiol. 4: 1–8. [CrossRef]
2. Buchanan, J. W. 2004. Tracheal signs and associated vascular anomalies in dogs with persistent right aortic arch. J. Vet. Intern. Med. 18: 510–514. [Medline] [CrossRef]
3. Cavrenne, R., De Busscher, V., Bolen, G., Billen, F., Clercx, C. and Snaps, F. 2008. Primary ciliary dyskinesia and situs inversus in a young dog. Vet. Rec. 163: 54–55. [Medline] [CrossRef]
4. d’Anjou, M. A., Penninck, D., Cornejo, L. and Pibarot, P. 2004. Ultrasonographic diagnosis of portosystemic shunting in dogs and cats. Vet. Radiol. Ultrasound. 45: 424–437. [Medline] [CrossRef]
5. Effler, D. B., Greer, A. E. and Sifers, E. C. 1951. Anomaly of the vena cava inferior; report of fatality after ligation. J. Am. Med. Assoc. 146: 1321–1322. [Medline] [CrossRef]
6. Fernandez del Palacio, M. J., Bernal, L., Bayon, A. and Luis Fuentes, V. 1997. Persistent left cranial vena cava associated with multiple congenital anomalies in a six-week-old puppy. J. Small Anim. Pract. 38: 526–530. [Medline] [CrossRef]
7. Foodman, M. S., Giger, U., Stebbins, K. and Knight, D. 1989. Kartagener’s syndrome in an old miniature poodle. J. Small Anim. Pract. 30: 96–100. [CrossRef]
8. Garcia-Rodriguez, M. B., Granja, M. A., Garcia, C. C., Gonzalo Orden, J. M., Cano Rabano, M. J. and Prieto, I. D. 2009. Complex cardiac congenital defects in an adult dog: an ultrasonographic and magnetic resonance imaging study. Can. Vet. J. 50: 933–935. [Medline]
9. Harder, M. A., Fowler, D., Pharr, J. W., Tryon, K. A. and Shmon, C. 2002. Segmental aplasia of the caudal vena cava in a dog. Can. Vet. J. 43: 365–368. [Medline]
10. Hunt, G. B., Bellenger, C. R., Borg, R., Youmans, K. R., Tisdall, P. L. C. and Malik, R. 1998. Congenital interruption of the portal vein and caudal vena cava in dogs: six case reports and a review of the literature. Vet. Surg. 27: 203–215. [Medline] [CrossRef]
11. Jacobs, G., Bolton, G. R. and Watrous, B. J. 1983. Echocardiographic features of dilated coronary sinus in a dog with persistent left cranial vena cava. J. Am. Vet. Med. Assoc. 182: 407–408. [Medline]
12. Jenni, S. D., Makara, M. A., Jenni, R., Ohlert, S. and Glaus, T. M. 2009. Diagnosis of pulmonary arterio-venous fistula and persistent left cranial vena cava by 3-dimensional computed tomographic reconstruction. J. Vet. Intern. Med. 23: 190–195. [Medline] [CrossRef]
13. Kayanuma, H., Suganuma, T., Shida, T. and Sato, S. 2000. A canine case of partial heterotaxia detected by radiography and ultrasound. J. Vet. Med. Sci. 62: 897–899. [Medline] [CrossRef]
14. Larcher, T., Abadie, J., Roux, F. A., Deschamps, J. Y. and Wyers, O. 2004. Angiocardiography, significance, and coexisting anomalies in dogs with persistent right aortic arch. J. Comp. Pathol. 135: 150–152. [Medline] [CrossRef]
15. Lohse, C. L., Selcer, R. R. and Suter, P. F. 1976. Hepatopancreatico-enteric association with situs inversus of abdominal organs and vascular anomalies in a dog. J. Am. Vet. Med. Assoc. 168: 681–688. [Medline]
16. May, N. D. S. 1960. Absence of the prerenal segment of the posterior vena cava of the dog. Aust. Vet. J. 36: 67–68. [CrossRef]
17. McClure, R. C. and Constantinescu, G. M. 1987. An uncommon right azygos vein and caudal vena cava in a dog. Anat. Histol. Embryol. 16: 84.
18. Neil, J. A., Canapp, S. O. Jr., Cook, C. R. and Lattimer, J. C. 2002. Kartagener’s syndrome in a Dachshund dog. J. Am. Anim. Hosp. Assoc. 38: 45–49. [Medline]
19. Patterson, D. F. 1968. Epidemiologic and genetic studies of congenital heart disease in the dog. Circ. Res. 23: 171–202. [Medline] [CrossRef]
20. Schwarz, T., Rossi, F., Wray, J. D., Å Blad, B., Beal, M. W., Kinns, J., Seiler, G. S., Dennis, R., McConnell, J. F. and Costello, M. 2009. Computed tomographic and magnetic resonance imaging features of canine segmental cranial vena cava aplasia. J. Small Anim. Pract. 50: 341–349. [Medline] [CrossRef]
21. Tisdall, P. L., Hunt, G. B., Bellenger, C. R. and Malik, R. 1994. Congenital portosystemic shunts in Maltese and Australian cattle dogs. Aust. Vet. J. 71: 174–178. [Medline] [CrossRef]
22. Tobias, A. H., Thomas, W. P., Kittleson, M. D. and Komtebedde, J. 1993. Cor triatriatum dexter in two dogs. J. Am. Vet. Med. Assoc. 202: 285–290. [Medline]