Local cerebral blood flow assessment using transcranial Doppler ultrasonography in a dog with brain infarction in the right middle cerebral artery territory

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ABSTRACT. A 12-year-old neutered male Chihuahua was diagnosed with acute brain infarction in the right middle cerebral artery (MCA) territory. Transcranial Doppler ultrasonography (TCD) was performed to assess the local cerebral blood flow at the time of diagnosis and after 4 and 31 hr. Initially, the right MCA retained blood flow but with a lower cerebral blood flow velocity (CBFV; 14.9 cm/sec) than the left MCA (27.9 cm/sec). The TCD vascular resistance variables were higher in the right than in the left MCA. An increase in the CBFV and a decrease in TCD vascular resistance variables were observed, consistent with improvements in neurological symptoms. TCD can be a non-invasive, and easy-to-use modality for bedside monitoring of cerebral edema and infarction.

KEYWORDS: critical care, ischemic brain disease, neuroimaging, neurology, ultrasonography

Ultrasonography is a noninvasive, low-cost imaging modality that avoids radiation exposure while being an easily repeated point-of-care diagnostic intervention. Owing to these features, ultrasonography is widely used in emergency and critical care in both veterinary and human medicines. For example, focused assessment with sonography for trauma (FAST) is a commonly used point of care ultrasound technique for detection of free abdominal fluid for evaluation of dogs following trauma [6]. In contrast with the abdominal region, the brain is difficult to assess using ultrasonography because most ultrasound waves are reflected by the skull.

Transcranial Doppler ultrasonography (TCD) detects Doppler signals of the cerebral arterial blood flow using low-frequency (1–2 MHz) ultrasound [1]. TCD provides the real-time blood flow waveforms of cerebral arteries and assesses the cerebral blood flow. TCD velocity, especially cerebral blood flow velocity (CBFV), is correlated with directly measured cerebral blood flow [11]. Cerebral blood flow is essential for maintaining the brain function; however, serious neurological diseases, such as intracranial hypertension and cerebral circulatory arrest, can alter cerebral blood flow [4, 10, 21]. TCD can provide information on cerebral blood flow to monitor patients with neurological deficits and allow bedside assessment of the cerebral blood flow in the veterinary medicine. To date, the knowledge of TCD in veterinary medicine is limited.

Cerebrovascular diseases are commonly encountered in dogs and cats [17]. Brain infarction is presumptively diagnosed on the basis of sudden onset of symptoms and findings on advanced imaging (e.g., computed tomography and magnetic resonance imaging) [12, 18]. Infarction results from vascular occlusion or stenosis caused by thrombi or emboli. Blood flow decreases significantly in the cerebral territory supplied by the involved vasculature, and the affected brain tissue is damaged because of ischemia. Cerebral blood flow is an important parameter in cerebral infarction because resumption of cerebral blood flow indicates an improvement in cerebral infarction [2, 9]. Changes in the local cerebral blood flow during brain infarction have not been documented in veterinary medicine. The purpose of this report is to describe the changes in local cerebral blood flow waveform and velocity using TCD in a dog with brain infarction in the right middle cerebral artery territory.
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A 12-year-old neutered male Chihuahua was referred to the Hokkaido University Veterinary Teaching Hospital for the evaluation of acute non-ambulatory left hemiparesis, anorexia, and lethargy for 1 day. The dog had been diagnosed with myxomatous mitral valve disease and pulmonary edema 2 years previously, and pimobendan and angiotensin-converting enzyme inhibitors were prescribed. Although his condition was well maintained, the oral medications were stopped at the owner’s discretion 3 months prior. At presentation, the owner reported of no trauma, medications use, or toxin exposure. The dog presented with a generalized tonic-clonic seizure in the hospital while waiting for a lounge. The seizure stopped spontaneously within a few minutes. He had a body weight of 3.06 kg, heart rate of 160 beats/min with grade III/VI left parasternal systolic murmur, respiratory rate of 56 breaths/min, and rectal temperature of 37.0°C. Neurological examination at presentation revealed an obtunded mental status, right head-turn, non-ambulatory left hemiparesis, decreased postural reactions in the left thoracic and pelvic limbs, lateral strabismus in the right eye, reduced bilateral menace response, and decreased bilateral facial sensations. A right forebrain lesion was suspected; however, further investigation was needed because of the effect of postictal symptoms. Table 1 shows the abnormal findings on complete blood count and biochemical analysis. The hematological data showed inflammatory signs, moderate regenerative anemia, and elevated live enzymes. The coagulation profile was within the reference range.

Thoracic radiography revealed cardiomegaly and a vertebral heart score of 14.0. Echocardiography revealed severe mitral valve regurgitation, with an E wave for 0.8 m/sec and A wave for 0.32 m/sec. Pericardial effusion was observed with a clot in the pericardial space, which could have been caused by left atrial rupture secondary to mitral valve regurgitation. No findings were suggestive of cardiac tamponade.

Because of the seriousness of his condition, brain computed tomography (Aquilion Prime; Canon Medical Systems Corp., Tochigi, Japan) was performed without anesthesia, which revealed a focal low-attenuation area and subtle mass effects in the right telencephalon. The low-attenuation area was in the right temporal lobe, which correlated with the vascular territory of the right MCA (Fig. 1). The dog was diagnosed with brain infarction in the right middle cerebral artery territory based on the acute history and radiological cerebral findings.

Table 1. Abnormalities in the complete blood count and serum biochemistry results at presentation

| Variable                        | Patient values       | Reference range     |
|---------------------------------|----------------------|---------------------|
| White blood cells               | 17,460 cells/µL      | 5,050–16,760 cells/µL |
| Red blood cells                 | 451 × 10⁴ cells/µL   | 565–887 × 10⁴ cells/µL  |
| Hematocrit                      | 26.30%               | 37.3–61.7%          |
| Hemoglobin                      | 9.6 g/dL             | 13.1–20.5 g/dL      |
| Reticulocyte                    | 110,900 cells/µL     | 1,000–110,000 cells/µL  |
| Platelets                       | 14.3 × 10⁴ cells/µL  | 14.8–48.4 × 10⁴ cells/µL  |
| Segmented neutrophils           | 13,850 cells/µL      | 2,950–11,640 cells/µL  |
| Total protein                   | 4.8 g/dL             | 5.8–7.2 g/dL        |
| Glucose                         | 211 mg/dL            | 75–128 mg/dL        |
| Alanine aminotransferase        | 450 IU/L             | 17–78 IU/L          |
| Aspartate aminotransferase      | 59 IU/L              | 17–44 IU/L          |
| Alkaline phosphatase            | 326 IU/L             | 47–254 IU/L         |
| Creatine kinase                 | 500 IU/L             | 49–166 IU/L         |
| C-reactive protein              | 6.2 mg/dL            | 0.0–1.0 mg/dL       |

Fig. 1. Head computed tomography images with (A) transverse and (B) dorsal multiplanar reconstructions. The vascular territory of the right middle cerebral artery shows a low-attenuation lesion (arrowhead), suggesting brain infarction of the right middle cerebral artery territory.
TCD was performed using an ultrasonography machine (Artida; Canon Medical Systems Corp.) with a 3–6-MHz sector probe (PST-50BT, Canon Medical Systems Corp.). This was a conventional ultrasound system using the following settings adjusted for TCD. B-mode examination and color flow Doppler sonography were performed to identify the MCA through the ipsilateral temporal window (Supplementary Movie 1). The ipsilateral MCA was visualized with the blood flow toward the probe (Fig. 2). The Doppler waveforms of the MCA were obtained using 6.0-kHz pulse repetition frequencies, a 94-Hz wall filter, a 2.5-mm sample width, and a 3.0–3.5-cm depth. The Doppler waveforms of the basilar artery (BA) were obtained through the transforaminal window. TCD was first performed for the left MCA, followed by the BA and the right MCA. The obtained Doppler waveforms were manually traced to determine the TCD velocities, including the peak systolic velocity (PSV), end-diastolic velocity (EDV), and time-averaged mean velocity (CBFV). In addition, TCD vascular resistance variables were calculated as follows: resistive index (RI)=(PSV−EDV)/PSV; pulsatility index (PI)=(PSV−EDV)/CBFV; the ratio of systolic to diastolic mean velocity (Sm/Dm)=systolic mean velocity/diastolic mean velocity [15]. The means of three consecutive cardiac cycles was calculated for all TCD variables.

At the time of diagnosis, the right MCA (affected side) retained blood flow but with a lower CBFV (14.9 cm/sec) compared with the left MCA (27.9 cm/sec). In particular, the diastolic blood flow in the right MCA was significantly lower than that in the left MCA (Fig. 3A). The TCD vascular resistance variables for the right MCA (RI, 0.93; PI, 5.56; and Sm/Dm, 3.96) were higher than those for the left MCA (RI, 0.84; PI, 2.34; and Sm/Dm, 2.64). The TCD vascular resistance variables for BA (RI, 0.89; PI, 3.84; and Sm/Dm, 2.74) were notably higher than those for previously reported normal cases (median [range]; RI, 0.72 [0.62–0.78]; PI, 1.57 [1.09–2.12]; and Sm/Dm, 1.63 [1.43–1.75]) [21]. Severe brain edema and secondary intracranial hypertension were suspected based on the obtunded mental status and computed tomography findings; prednisolone (1 mg/kg, subcutaneous [SC]) was administered. Pimobendan (0.15 mg/kg, intravenous [IV]) and IV infusions of dobutamine (3 µg/kg/min) at a continuous rate were administered to support the circulation. TCD was performed 4 hr after treatment initiation. The TCD vascular resistance variables for the right (RI, 0.89; PI, 3.59; and Sm/Dm, 2.98) and left (RI, 0.71; PI, 1.51; and Sm/Dm, 1.82) MCAs were lower than those measured at the time of diagnosis. Nevertheless, the TCD vascular resistance variables for the affected side were higher than those for the contralateral side (Table 2).

Fig. 2. (A) Transcranial Doppler ultrasonography images with color flow using a conventional ultrasound system. The ipsilateral middle cerebral artery is seen with the blood flowing (arrowhead) toward the probe placed on the temporal window. The arterial waveforms are obtained using pulsed Doppler on the visualized lesion. (B) Head computed tomography image of the same position as that of the ultrasonography image. (C, D) A three-dimensional schema of transcranial Doppler ultrasonography to the left middle cerebral artery created from computed tomography data.
Treatments to support the circulation and reduce the brain edema were continued: prednisolone (1 mg/kg, SC, q24 hr), and pimobendan (0.15 mg/kg, IV, q12 hr) were administered. Osmotic diuretics were not administered because of their effects on circulation. Hypertension occurred after starting dobutamine; therefore, dobutamine was gradually tapered and stopped 7 hr after starting treatment. The dog exhibited respiratory distress and decreased oxygen saturation on pulse oximetry 10 hr later. Furosemide (2 mg/kg, IV) was administered every 3 hr. Subsequently, he recovered until he could stand and eat on his own. Pimobendan (0.3 mg/kg, per os [PO]) and benazepril (0.4 mg/kg, PO) were administered 23 hr after starting the treatment. TCD vascular resistance variables for the right MCA (RI, 0.80; PI, 2.36; and Sm/Dm, 2.13) decreased at 31 hr after starting the treatment. Although the TCD vascular resistance variables for the left MCA (RI, 0.80; PI, 2.39; and Sm/Dm, 2.46) slightly increased, they did not differ significantly from those for the right MCA (Fig. 3C). The TCD vascular resistance variables for the BA decreased slightly from the time of diagnosis but were still higher than the values reported previously for normal cases. Table 2 summarizes the changes in the TCD variables.

Despite the treatment, the patient’s respiratory condition and mental status gradually worsened. The dog was euthanized 57 hr after initiating the treatment because of the lack of response to medical management. Postmortem examination could not be performed at the owner’s request.

This is the first clinical report of changes in the local cerebral arterial blood flow in a dog with brain infarction analyzed using TCD. Brain infarction, regardless of whether it is complete or partial, reduces the cerebral blood flow, and the occlusion changes the cerebral blood flow waveform [2]. Cerebral blood flow is dynamically altered depending on the occlusion or recanalization of the blood vessels; however, no such report has been reported in veterinary medicine. In the present case, neurological improvement was observed along with improvement in local TCD variables. Treatment response in cerebrovascular disease depends on the restoration of cerebral blood flow. Cerebral blood flow waveforms were changed in few days in stroke patients during treatment and the patterns of waveform change reflect dynamic collateralization [2, 14]. Similarly, more cases that show the utility of TCD should be documented in veterinary medicine.

The marked increase in TCD vascular resistance variables for the affected MCA may have been caused by the local cytotoxic edema in the right temporal lobe. Typically, an increase in the blood flow velocity is observed in mild MCA occlusion, and a blood flow disruption is observed in a severe MCA occlusion [9]. However, the blood flow waveform in the present case was characterized by a marked decrease in the diastolic blood flow. From the viewpoint of rheology, high-resistance vascular beds produce a blood-flow
waveform with little or no diastolic flow, whereas low-resistance vascular beds produce a blood-flow waveform with a high diastolic flow [7]. Therefore, cerebral edema in the infarcted area could have increased the vascular resistance, causing a characteristic decrease in the diastolic blood flow.

Monitoring TCD variables seems to be convenient for monitoring cerebral blood flow and cerebral edema. In this case, an increase in CBFV and a decrease in TCD vascular resistance variables were observed, which were consistent with the improvement in clinical symptoms. TCD vascular resistance variables after treatment did not differ significantly between the left and right MCAs, suggesting that the local cerebral edema was improved. In human medicine, changes in TCD vascular resistance variables correspond closely to changes in intracranial pressure on manometry of the cerebrospinal fluid [26]. Intracranial pressure can be monitored using direct measurements in human medicine, but it is invasive, can cause complications, and may not improve outcomes [5, 8]. Evaluation of the optic nerve sheath diameter is used in human medicine for monitoring of treatment response and sometimes, for prognostication [24]. Combination with TCD may improve the accuracy of evaluating intracranial hypertension. Furthermore, subclinical and preclinical evaluations for neurological diseases (e.g., Alzheimer’s disease, Fabry’s disease, sickle cell disease) using TCD have been reported in human medicine [3, 13, 25], and are expected to be applied to veterinary medicine in the future.

In the present case, the dog had atrial rupture and circulatory failure, which may have exerted a systemic effect on the cerebral blood flow and secondarily altered the cerebral blood flow waveform. In particular, the increase in the TCD vascular resistance variables for the right MCA, left MCA, and BA may be associated with intracranial hypertension and a decrease in cerebral perfusion pressure due to circulatory failure. A 30% decrease in cardiac output was reported to cause a 10% decrease in cerebral blood flow [16]. However, from the perspective within a single heartbeat, cardiac output is related to systolic velocity and CBFV in the cerebral blood flow waveform and has little effect on diastolic blood flow [22]. Changes in the cerebral blood flow waveforms have been reported in humans with aortic valve insufficiency and patent ductus arteriosus [19, 20]. Increasing knowledge to interpret canine cerebral blood flow waveforms in various situations is required.

This report has several limitations. First, ultrasonography interpretation highly depends on the observer. Although the repeatability of TCD examination is good [21, 23], it is desirable that the same observer repeats the examination under the same conditions as many times as possible, or training should be provided, similar to other ultrasonography examinations [6]. Second, improvement of cerebral edema has not been confirmed over time by other imaging modalities. In the present case, improvement of neurological symptoms was considered as improvement of cerebral edema. The patient had an unfavorable outcome after TCD evaluation despite improvement in neurological symptoms, which might be attributed to neurological factors such as acute conversion to hemorrhagic stroke or cardiovascular factors such as rapid deterioration of heart failure. However, the cause was unknown because an autopsy could not be performed. Third, this report presents a single case. The time for examination was limited, and the patient had confounding factors such as systemic instability and severe cardiovascular disease with secondary respiratory compromise. Therefore, more such cases should be evaluated, treated, and documented.

Table 2. Transcranial Doppler ultrasonography variables in the right middle cerebral artery (MCA, affected side), left MCA (non-affected side), and basilar artery

| Variable                             | At diagnosis | 4 hr after | 31 hr after |
|--------------------------------------|--------------|------------|-------------|
| Peak systolic velocity (PSV, cm/sec) |              |            |             |
| PSV-right MCA                        | 86.9         | 92.9       | 68.8        |
| PSV-left MCA                         | 76.0         | 79.8       | 75.5        |
| PSV-basilar artery                   | 152.0        | 114.9      | 117.9       |
| End diastolic velocity (EDV, cm/sec) |              |            |             |
| EDV-right MCA                        | 6.3          | 10.1       | 13.6        |
| EDV-left MCA                         | 12.3         | 22.9       | 15.2        |
| EDV-basilar artery                   | 16.3         | 15.6       | 20.0        |
| Time-averaged mean velocity (CBFV, cm/sec) |          |            |             |
| CBFV-right MCA                       | 14.9         | 23.5       | 23.5        |
| CBFV-left MCA                        | 27.9         | 37.7       | 25.2        |
| CBFV-basilar artery                  | 35.9         | 32.1       | 40.7        |
| Resistive index (RI)                 |              |            |             |
| RI-right MCA                         | 0.93         | 0.89       | 0.80        |
| RI-left MCA                          | 0.84         | 0.71       | 0.80        |
| RI-basilar artery                    | 0.89         | 0.86       | 0.83        |
| Pulsatility index (PI)               |              |            |             |
| PI-right MCA                         | 5.56         | 3.59       | 2.36        |
| PI-left MCA                          | 2.34         | 1.51       | 2.39        |
| PI-basilar artery                    | 3.84         | 3.12       | 2.54        |
| Ratio of systolic to diastolic mean velocity (Sm/Dm) | | | |
| Sm/Dm-right MCA                      | 3.96         | 2.98       | 2.13        |
| Sm/Dm-left MCA                       | 2.64         | 1.82       | 1.88        |
| Sm/Dm-basilar artery                 | 2.74         | 2.81       | 2.46        |
To our best knowledge, this is the first clinical report of local cerebral arterial blood flow changes occurring in a dog with brain infarction evaluated using TCD. Neurological improvement was observed along with an improvement in local TCD variables. TCD is a non-invasive and easy-to-use modality for bedside monitoring of cerebral blood flow that can show changes in real time. Future studies elucidating the association between the disease and prognosis through the accumulation of more cases are required.

CONFLICT OF INTEREST. The authors declare no conflict of interest for this research.

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REFERENCES

1. Aaslid R, Markwalder TM, Nornes H. 1982. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 57: 769–774. [Medline] [CrossRef]
2. Akopov S, Whitman GT. 2002. Hemodynamic studies in early ischemic stroke: serial transcranial Doppler and magnetic resonance angiography evaluation. Stroke 33: 1274–1279. [Medline] [CrossRef]
3. Alswathan M, Murman DL, Bashford G. 2019. Cerebrovascular reactivity impairment in preclinical Alzheimer’s disease. J Neuroimaging 29: 493–498. [Medline] [CrossRef]
4. Bellner J, Rommer B, Reinsrump P, Kristiansson KA, Ryding E, Brandt L. 2004. Transcranial Doppler sonography pulsatility index (PI) reflects intracranial pressure (ICP). Surg Neurol 62: 45–51, discussion 51. [Medline] [CrossRef]
5. Bhatia A, Gupta AK. 2007. Neuromonitoring in the intensive care unit. I. Intracranial pressure and cerebral blood flow monitoring. Intensive Care Med 33: 1263–1271. [Medline] [CrossRef]
6. Boysen SR, Rozanski EA, Tidwell AS, Holm JL, Shaw SP, Rush JE. 2004. Evaluation of a focused assessment with sonography for trauma protocol to detect free abdominal fluid in dogs involved in motor vehicle accidents. J Am Vet Med Assoc 225: 1198–1204. [Medline] [CrossRef]
7. Chavhan GB, Parra DA, Mann A, Navarro OM. 2008. Normal Doppler spectral waveforms of major pediatric vessels: specific patterns. Radiographics 28: 691–706. [Medline] [CrossRef]
8. Chesnut RM, Temkin N, Carney N, Dikmen S, Rondina C, Petroni G, Videtta W, Petroni L, Lujan S, Pridgeon J, Barber J, Machamer J, Chaddock K, Celix JM, Cherner M, Hendrix T, Global Neurotrauma Research Group. 2012. A trial of intracranial-pressure monitoring in traumatic brain injury. N Engl J Med 367: 2471–2481. [Medline] [CrossRef]
9. Demchuk AM, Burgin WS, Christou I, Felberg RA, Barber PA, Hill MD, Alexandrov AV. 2001. Thrombolysis in brain ischemia (TIBI) transcranial Doppler flow grades predict clinical severity, early recovery, and mortality in patients treated with intravenous tissue plasminogen activator. Stroke 32: 89–93. [Medline] [CrossRef]
10. Fujikushima U, Miyashita K, Okano S, Higuchi S, Takase K, Hagi M. 2000. Evaluation of intracranial pressure by transcranial Doppler ultrasonography in dogs with intracranial hypertension. J Vet Med Sci 62: 353–355. [Medline] [CrossRef]
11. Fujikushima U, Sasaki S, Okano S, Takase K, Hagi M. 1999. The comparison between the cerebral blood flow directly measures and cerebral blood flow velocity in the middle and basilar cerebral arteries measured by transcranial Doppler ultrasonography. J Vet Med Sci 61: 1293–1297. [Medline] [CrossRef]
12. Garosi L, McConnell JF, Platt SR, Barone G, Baron JC, de Lahunta A, Schatzberg SJ. 2006. Clinical and topographic magnetic resonance characteristics of suspected brain infarction in 40 dogs. J Vet Intern Med 20: 311–321. [Medline] [CrossRef]
13. Hirtz D, Kirkham FJ. 2019. Sickle cell disease and stroke. [Medline] [CrossRef]
14. Labiche LA, Malkoff M, Alexandrov AV. 2003. Residual flow signals predict complete recanalization in stroke patients treated with TPA. J Neuroimaging 13: 28–33. [Medline] [CrossRef]
15. Maruyoshi H, Kojima S, Kojima S, Nagayoshi Y, Horibata Y, Kaikita K, Sugiyama S, Ogawa H. 2010. Waveform of ophthalmic artery Doppler flow velocity in the middle and basilar cerebral arteries measured by transcranial Doppler ultrasonography. J Vet Med Sci 63: 1198–1208. [Medline] [CrossRef]
16. Meng L, Hou W, Chui J, Han R, Gelb AW. 2015. Cardiac output and cerebral blood flow: The integrated regulation of brain perfusion in adult humans. Anesthesiology 123: 1198–1208. [Medline] [CrossRef]
17. Ozawa T, Miura N, Hasegawa H, Uemura T, Nakamoto Y, Tsuji M, Takeuchi T, Shiraishi M. 2002. Characteristics and outcome of suspected cerebrovascular disease in dogs: 66 cases (2009–2016). J Small Anim Pract 63: 45–51. [Medline] [CrossRef]
18. Paul AE, Lenard Z, Mansfield CS. 2010. Computed tomography diagnosis of eight dogs with brain infarction. Aust Vet J 88: 374–380. [Medline] [CrossRef]
19. Perlman JM, Hill A, Volpe JJ. 1981. The effect of patent ductus arteriosus on flow velocity in the anterior cerebral arteries: ductal steal in the premature newborn infant. J Pediatr 99: 767–771. [Medline] [CrossRef]
20. Rodriguez RA, Cornel G, Weerasena N, Hosking MC, Murto K, Helou J. 1999. Aortic valve insufficiency and cerebral “steal” during pediatric cardiopulmonary bypass. J Thorac Cardiovasc Surg 117: 1019–1021. [Medline] [CrossRef]
21. Sasaoka K, Nakamura K, Osuga T, Morita T, Yokoyama N, Morishita K, Sasaki N, Ohta H, Takiguchi M. 2018. Transcranial Doppler ultrasound examination in dogs with suspected intracranial hypertension caused by neurologic diseases. J Vet Intern Med 32: 314–323. [Medline] [CrossRef]
22. Sasaoka K, Ohta H, Ishizuka T, Kojima S, Kojima S, Takiguchi M. 2022. Transcranial Doppler ultrasound detects the elevation of cerebral blood flow during ictal-phase of pentetrazol-induced seizures in dogs. Am J Vet Res 83: 331–338. [Medline] [CrossRef]
23. Seo M, Choi H, Lee K, Choi M, Yoon J. 2005. Transcranial Doppler ultrasound measurement of the optic nerve sheath diameter in healthy dogs. J Vet Emerg Crit Care (San Antonio) 28: 31–38. [CrossRef]
24. Smith JJ, Fletcher DJ, Cooley SD, Thompson MS. 2018. Transpalpebral ultrasonographic measurement of the optic nerve sheath diameter in healthy dogs. J Vet Emerg Crit Care (San Antonio) 28: 31–38. [CrossRef]
25. Vagli C, Fisicaro F, Vinciguerra L, Puglisi V, Rodolico MS, Giordano A, Ferri R, Lanza G, Bella R. 2020. Cerebral hemodynamic changes to transcranial Doppler in asymptomatic patients with Fabry’s disease. Brain Sci 10: 546. [Medline] [CrossRef]
26. Wakerley BR, Kusuma Y, Yeo LL, Liang S, Kumar K, Sharma AK, Sharma VK. 2015. Usefulness of transcranial Doppler-derived cerebral hemodynamic parameters in the noninvasive assessment of intracranial pressure. J Neuroimaging 25: 111–116. [Medline] [CrossRef]