

Title Page**Title:**

Course of apparent diffusion coefficient values in cerebral edema of dural arteriovenous fistula before and after treatment

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Abstract

Apparent diffusion coefficient (ADC) values at magnetic resonance imaging (MRI) are useful to distinguish vasogenic and cytotoxic edema due to cerebrovascular diseases. Dural arteriovenous fistulas (DAVFs) with retrograde leptomeningeal venous drainage may cause cerebral edema by venous congestion. We report herein the course of ADC values of cerebral edema before and after endovascular treatment in DAVFs. A 65-year-old woman with transverse-sigmoid (T-S) sinus DAVFs with retrograde leptomeningeal venous drainage presented with severe edema in cerebellar hemisphere and brainstem. In preoperative MRI, increased ADC values were observed in the edema area. The isolated sinus was obliterated completely by transvenous embolization. On the following day after treatment, the ADC values in cerebral edema area increased slightly without any new neurological deficits and improved at 1 week later. Rapid resolution of venous congestion due to DAVFs may cause a slight, transient progression of vasogenic edema.

Key words: ADC value; cerebral edema; dural arteriovenous fistulas; endovascular treatment; venous congestion

1. Introduction

Dural arteriovenous fistulas (DAVFs) of transverse-sigmoid (T-S) sinus may present with pulsatile tinnitus, bruit, headache, visual impairment, and dementia. Furthermore, DAVFs with retrograde leptomeningeal venous drainage can cause venous congestion resulting in neurological deficits due to brain edema, hemorrhage, and/or ischemia [1, 2, 3]. We describe herein a case with T-S sinus DAVFs that presented with serious edema in the cerebellar hemisphere and brainstem. We also measured apparent diffusion coefficient (ADC) values at magnetic resonance imaging (MRI) to evaluate serious edema before and after endovascular treatment.

2. Case report

A 65-year-old woman initially complained of mild headache and vertigo 1 month before admission in our hospital. The patient was admitted to another hospital for progressive mental and gait disturbance and transferred to our hospital one week later after MRI had revealed a hemorrhagic lesion in the left temporal lobe and edema in the brainstem and left cerebellar hemisphere. Neurological examination showed cerebellar syndrome and mild confusion.

The patient was examined with a 1.5-T MR system (Signa Horizon; GE Medical Systems, Milwaukee, Wis). T1-weighted (repetition time msec/echo time msec, 400/8), T2-weighted (3500/100), and diffusion-weighted images (DWI) were obtained. The pulse-sequence for DWI was a single-shot, spin-echo echo-planar imaging sequence (1600/107; slice thickness 7.5 mm, field of view 24×24 cm, matrix 128×128). Diffusion gradients were applied in each of the x, y, and z directions with five b values (0, 250, 500, 750 and 1000 sec/ mm²). DWI data were transferred to a

computer workstation (SUN Sparc 20; Sun Microsystems, Mountain View, Calif) for determination of the signal intensity and ADC. MR Vision (version 1.5.5; L.A. Systems, Oyama, Japan) was the software program used to generate ADC maps and quantify ADCs of brain edema. Each image used for the creation of the ADC maps was obtained with one signal acquired. The ADC values were measured by manually placing regions of interest (ROIs) in edema regions on the ADC map. The ROIs were placed at the site of hypointense lesion on T1-weighted MR images and hyperintense lesion on T2-weighted MR images only to avoid measurement in areas with hemorrhage. We compared the ADC maps and other MR images carefully and placed the ROIs only in edema.

T1-, T2-weighted MRI and DWI revealed hemorrhagic transformation in the left temporal lobe and edema in the brainstem and left cerebellar hemisphere (Fig. 1A-D). ADC map revealed a hyperintense lesion in the brainstem and left cerebellar hemisphere (Fig. 1E). ADC values in the lesion and contralateral control side were 1.34×10^{-3} and 0.70×10^{-3} mm²/sec, respectively. Cerebral angiogram revealed DAVFs to the left T-S sinus with retrograde leptomeningeal venous drainage (Cognard Type IIb) and occlusion of the T-S sinus at its proximal and distal end (Fig. 2). The feeding arteries were the left occipital artery, the neuromeningeal branch of the left ascending pharyngeal artery, the posterior branch of the left middle meningeal artery, and the left anterior and posterior auricular arteries.

The patient was emergently treated with endovascular treatment under local anesthesia. Transfemoral, transvenous embolization through the left jugular vein to the left T-S sinus was performed by a triaxial system (6-French guiding catheter / 4-French catheter / microcatheter) to improve the pushability and handling of a

guidewire and microcatheter [4]. The tips of a 6-French ENVOY XB guiding catheter (Cordis, Miami, FL) and a 4-French Technowood INF catheter with Berenstein type and a shaft length of 125 cm (Tonokura, Tokyo, Japan) were placed on the left jugular bulb using the coaxial method. A 0.035-inch Radifocus guidewire (Terumo, Tokyo, Japan) was inserted into the isolated sinus from the occluded proximal side of the sinus, and a 4-French catheter was navigated into isolated sinus through the thrombosed sinus. A RapidTransit infusion catheter (Cordis) using a 0.016-inch GT-wire (Terumo) was then navigated easily into the isolated T-S sinus. The isolated T-S sinus was completely packed with 427 cm of Soft Detach-18 coils (Cook, Bjaeverskov, Denmark) and Interlocking detachable coils (Boston Scientific, Natick, MA). Cerebral angiogram after embolization showed no DAVFs (Fig. 3).

Neurological deficit with cerebellar syndrome and confusion completely improved a few days postoperatively. Repeat MR studies were performed 1 day and 1 week after endovascular treatment. The ADC values of edema and contralateral control side at 1 day after treatment were 1.42×10^{-3} and 0.68×10^{-3} mm²/sec, respectively. The ADC values of edema and contralateral control side at 1 week after treatment were 1.11×10^{-3} and 0.69×10^{-3} mm²/sec, respectively. Although the ADC values increased once after the procedure with no new clinical symptoms, these values improved 1 week later (Fig. 4). T2-weighted MRI 1 week later revealed an improvement of edema in the brainstem (Fig. 5). The patient was discharged with no neurological deficits 2 weeks after endovascular surgery.

3. Discussion

Intracranial DAVFs account for 10-15% of all intracranial arteriovenous lesions [5]. Venous sinus thrombosis and trauma has been associated with intracranial DAVFs [1]. However, the exact mechanism for this formation has not as yet been described in detail. Intracranial DAVFs Djindjian et al. [6] proposed the first comprehensive classification of intracranial DAVFs based on radiological findings. It was subsequently modified by Cognard et al. [5] and Borden et al. [1]. Cognard et al. [5] classified DAVFs according to patterns of venous drainage into five types as follows: type I, located in the main sinus, with antegrade flow; type II, in the main sinus, with reflux into the sinus (IIa), cortical veins (IIb), or both (IIa + b); type III, with direct cortical venous drainage without venous ectasia; type IV, with direct cortical venous drainage with venous ectasia; and type V, with spinal venous drainage. Borden et al. [1] classified DAVFs into three types as follows: type I, DAVFs drain directly into dural venous sinuses or meningeal veins; type II, DAVFs drain into dural sinuses or meningeal veins but also have retrograde drainage into subarachnoid veins; type III, DAVFs drain into subarachnoid veins and do not have dural sinus or meningeal venous drainage. Furthermore, DAVFs of type III tend to be associated with variceal or aneurysmal venous dilatation, and some patients develop venous hypertension resulting in neurological deficits due to hemorrhage, brain edema, and/or venous stroke [1].

The pathophysiology of venous stroke remains obscure. Obstruction of venous flow is thought to result in raised intracranial pressure and decreased cerebral blood flow, subsequently leading to venous infarction [7]. Increased venous pressure may cause breakdown of the blood-brain barrier and reversible vasogenic edema or may cause reduced cerebral blood flow and irreversible cytotoxic edema

[8]. In venous stroke, perfusion of the affected brain tissue might still be possible at lower flow rates, if the blood is drained through collateral pathways [7]. With such flow conditions, however, swollen cells might be functionally but not irreversibly damaged and therefore have a potential for recovery [7, 9]. Hurst et al. [10] have examined the prevalence and features of DAVFs in patients with progressive dementia or encephalopathy. The potential for recurrent and possibly permanent ischemic damage indicated an aggressive approach to closure of these lesions to relieve excessive venous pressure. They concluded that venous hypertensive encephalopathy resulting from a DAVF should be considered a potentially reversible cause of vascular dementia in patients with progressive cognitive deficits.

ADC values can distinguish the type of edema between cytotoxic and vasogenic edema [11, 12]. Decreased ADC results from restricted diffusion by intracellular water accumulation (i.e., cytotoxic edema). In contrast, increased ADC results from free diffusion by increased extracellular space (i.e., vasogenic edema). ADC values represent only the diffusion coefficient and there could in fact be bio-energetic compromise (i.e. cytotoxic changes) co-existing with the vasogenic edema formation [8, 11, 13]. It has been reported that both vasogenic and/or cytotoxic edema occur in the setting of venous stroke by ADC values [8, 13]. Lo et al. [14] reported DWI and ADC map of spontaneous venous infarction and cortical hemorrhage complicating DAVF. In the case, DWI and ADC map 2hour after transarterial embolization showed cytotoxic edema and ADC decline distributed in a nonterritorial pattern in the unilateral cerebral hemisphere. However, to our knowledge, there has been no report of serial changes of ADC values of the area of edema due to DAVFs before and after endovascular treatment.

In the present case, ADC values at the day after treatment showed slightly increased values without new neurological deficits, suggesting progressive vasogenic edema. Although the increased ADC values after treatment might have been simply related to sampling error, rapid resolution of venous congestion due to change of venous drainage may cause a transient progression of vasogenic edema. Venous congestion due to DAVFs might be caused not only by reversibly vasogenic edema but also irreversibly cytotoxic edema. Therefore, early treatment is necessary to prevent progression of venous stroke due to DAVFs.

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Figure legends

Fig. 1

- A. T1-weighted MRI revealed a hyperintense lesion with bleeding in the left temporal lobe.
- B. T1-weighted MRI revealed a slightly hypointense lesion with edema in the left cerebellar hemisphere.
- C. T2-weighted MRI revealed a hyperintense lesion in the brainstem and cerebellar hemisphere.
- D. Diffusion-weighted MRI revealed a heterogeneous-intense lesion in the left cerebellar hemisphere.
- E. ADC map revealed a hyperintense lesion in the left cerebellar hemisphere

Fig. 2

Pre-embolization left common carotid artery angiogram (lateral view) revealed a left transverse-sigmoid sinus, being occluded at its proximal and distal end, and dural arteriovenous fistulas with a retrograde leptomeningeal venous drainage. The feeding arteries were the left occipital artery, the neuromeningeal branch of the left ascending pharyngeal artery, the posterior branch of the left middle meningeal artery, and the left anterior and posterior auricular arteries.

Fig. 3

Post-embolization left common carotid artery angiogram (lateral view) showed no dural arteriovenous fistulas.

Fig. 4

Time-course of changes of ADC value of ROIs in the edema (circle) and contralateral control side (square) in the cerebellar hemisphere on ADC map. Preoperative ADC values in the edema and contralateral control side were 1.34×10^{-3} and 0.70×10^{-3} mm^2/sec , respectively. The ADC values of ROIs in the edema at 1 day and 1 week after embolization were 1.42×10^{-3} and 1.11×10^{-3} mm^2/sec , respectively. The ADC value of the contralateral control side did not change ($0.68\text{-}0.70 \times 10^{-3}$ mm^2/sec).

Fig. 5

T2-weighted MRI at 1 week after treatment revealed reduction of hyperintense lesion in the brainstem.

Fig. 1

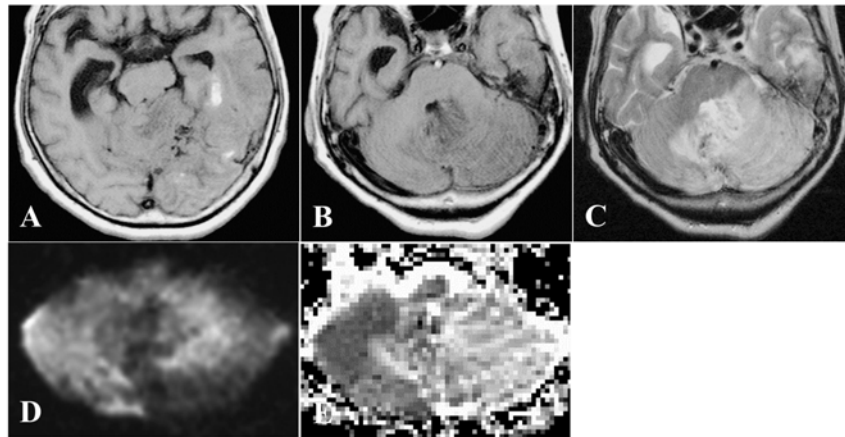


Fig. 2

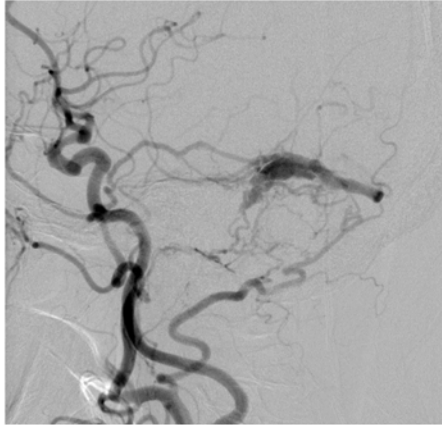


Fig. 3

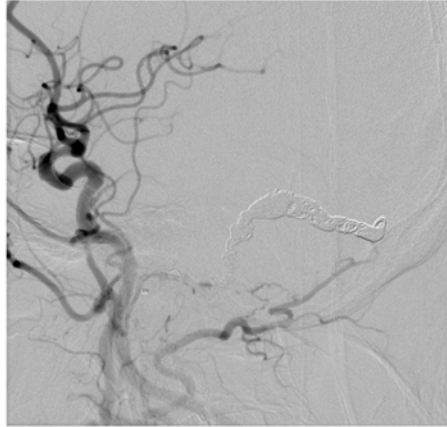


Fig. 4

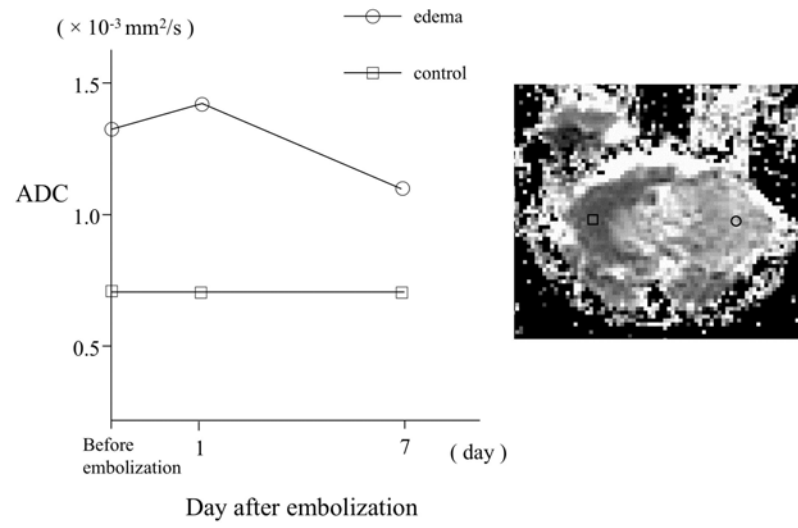


Fig. 5

