Letter to the Editor

Dural arteriovenous fistula presenting as thalamic dementia: a case description with rare imaging findings

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Introduction

Dural arteriovenous fistula (dAVF) refers to an abnormal direct blood connection between an intracranial artery and a dural venous sinus. This fistula accounts for about 10% to 15% of all intracranial vascular malformations (1,2). A dAVF can occur in any part of the brain, with deep brain structures such as the thalamus involved in about 1% to 3% of cases. Patients with dAVF often present with pulsatile tinnitus, orbital congestion, and headache.

Non-hemorrhagic neurological deficits (NHNDs) are rare and consequently underrecognized symptom of dAVF (1). Due to the infrequency of thalamic dementia symptoms in dAVF and the complexity of imaging presentations, dAVF-induced thalamic dementia can easily be misdiagnosed and mistreated. Progressive dementia may be one of the precursors of NHNDs. NHNDs can be induced by venous hypertension in the bilateral thalamus or cerebral cortex, which manifests with basal ganglia edema and bilateral thalamic involvement (3,4). Studies have shown that dAVF-induced retrograde venous hypertension can cause chronic passive hyperemia or venous ischemia, resulting in thalamic dementia and bilateral thalamic lesions (5).

In this case report, we describe a patient with dAVF presenting with thalamic dementia who was completely cured by endovascular embolization.

Case presentation

A 52-year-old previously healthy man was admitted to the local hospital with slow response, inarticulate speech, slow speech, and poor short-term memory lasting for more than 1 week. The patient's initial non-contrast head computed tomography (CT) findings were normal, and there were no background abnormalities or periodic discharges on electroencephalography. A lumbar puncture revealed no white blood cells or red blood cells in tubes 1 and 4. The patient's cerebrospinal fluid (CSF) protein was 1.10 g/L; CSF glucose was 4.20 mmol/L; and no xanthochromia was detected. Cryptococcal antigen, fungal culture, Gram stain, bacterial culture, and India ink stain were negative. However, the patient then deteriorated to the point where he could only give a few words in response to questions.

At 1 week later, magnetic resonance imaging (MRI) examination of the brain showed abnormal changes in the bilateral thalamus, and the patient was transferred to our institution. On arrival, neurological examination revealed hypersomnolence, spatial disorientation, and short-term memory impairment. Relevant cognitive assessments returned the following scores: Mini-Mental State Examination (MMSE): 13 points, Memory and Executive Screening scale (MES): 15 points, Montreal Cognitive Assessment scale (MoCA): 6 points, and Activities of Daily Living scale (ADL): 50 points (the reference values were: ≥27 points, ≥80 points, ≥26 points, and ≤26 points, respectively). There were no obvious abnormalities in laboratory data, including complete blood count (FBC), blood urea nitrogen (BUN), electrolytes, erythrocyte sedimentation rate (ESR), and creatinine. Protein S assay, antithrombin III, anticardiolipin antibody, homocysteine, thyroid stimulating hormone assay, lupus anticoagulant, and protein C assay were also normal. Repeat cranial MRI showed synchronous bilateral thalamic hyper T2 weighted imaging signals. The fluid-attenuated inversion

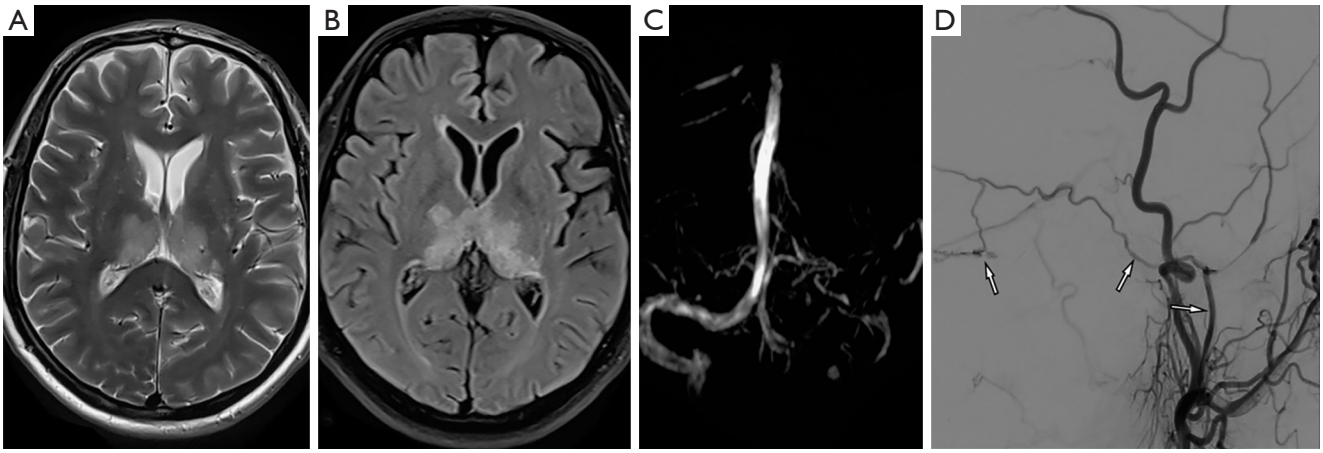

Figure 1 Images obtained at the time of diagnosis. (A) T2WI and (B) FLAIR suggested bilateral thalamus swelling and high signal changes; (C) MRV showed the left transverse sinus was absent; (D) DSA demonstrated a dAVF on the tentorium of the cerebellum fed mainly by the branch of the left middle meningeal artery and drained to the basilar vein (as indicated by the arrows). T2WI, T2-weighted imaging; FLAIR, fluid attenuated inversion recovery; MRV, magnetic resonance venography; DSA, digital subtraction angiography; dAVF, dural arteriovenous fistula.

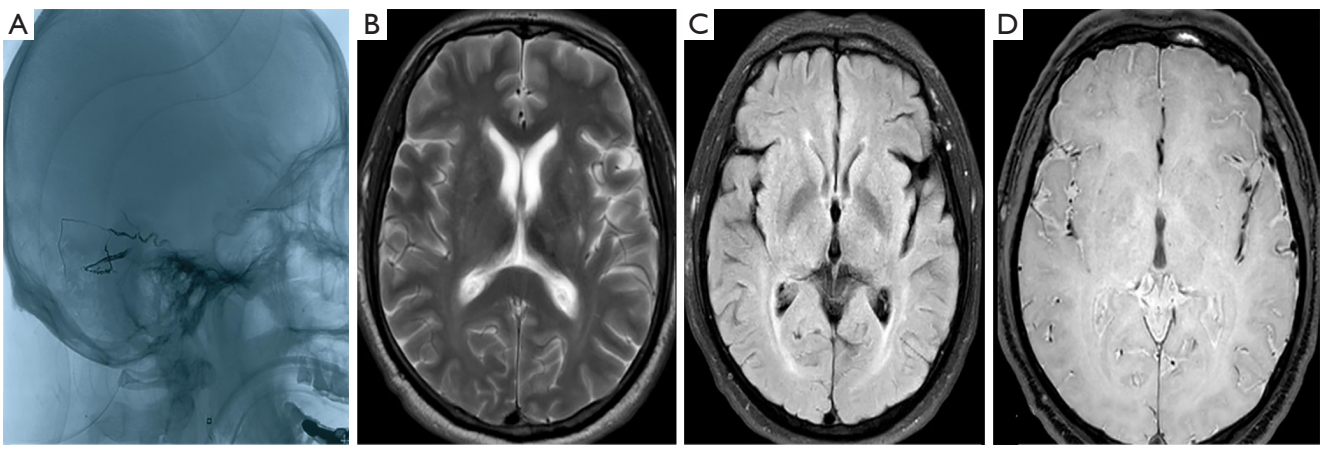

Figure 2 Images obtained after the time of the TAE. The fistula disappeared after the (A) TAE; (B-D) images obtained at the 6-month follow-up after TAE; (B) T2WI and (C) FLAIR showed that there was negligible abnormality in bilateral thalamus; (D) VISTA showed that the disordered venous structure on the left side of the great cerebral vein had negligibly disappeared. TAE, transarterial embolization; T2WI, T2-weighted imaging; FLAIR, fluid attenuated inversion recovery; VISTA, volume isotropic turbo spin echo acquisition.

recovery (FLAIR) signal was subtly patchy and there was hyperintense gadolinium enhancement (*Figure 1A*,*1B*). MRI venography revealed that the left transverse sinus was absent (*Figure 1C*). Further digital subtraction angiography (DSA) revealed a Borden-Shucart type III dAVF fed mainly by the limb of the left middle meningeal artery. The fistula was located in the tentorium cerebellum, draining into the basilar vein. During the venous phase, the internal cerebral vein, basal vein, great cerebral vein, and tortuous confluence of veins could be seen, and there was obvious venous regurgitation (*Figure 1D*). The patient subsequently underwent transarterial embolization (TAE) using Onyx, and the fistula was completely embolized (*Figure 2A*).

After treatment, the patient's symptoms were gradually alleviated. Follow-up MRI at 6 months after treatment showed that there was negligible abnormality in bilateral thalamus (*Figure 2B*,*2C*). Volume isotropic turbo spin echo acquisition (VISTA; *Figure 2D*) showed that the disordered

venous structure on the left side of the great cerebral vein had disappeared. Cognitive scale assessment showed MMSE: 27 points, MES: 79 points, MoCA: 21 points, and ADL: 23 points. The patient's cognitive level had significantly improved and returned to normal.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

Through a comprehensive search, 19 (1,6-19) dAVF-induced thalamic dementia cases were identified in the literature (*Table 1*). This form of dementia is seen almost exclusively in men, who make up 95% of all cases, and is most common in those aged between 50 and 70 years (mean age 60±8.7 years; range from 43 to 77 years). All patients with dAVF (100%) experience progressive cognitive dysfunction, including deficits in executive function, memory, attention, and disorientation. Half of patients (50%) have additional neurological deficits, such as aphasia. The highly variable clinical manifestations of dAVF are strongly associated with the location and pattern of venous drainage. Among the 19 cases of dAVF with progressive dementia found in the literature review, there were 9 (47%) and 10 (53%) cases of Borden-Shucart type II and Borden-Shucart type III dAVF, respectively. Feeder vessels mostly include the posterior meningeal artery, middle meningeal artery, and/or occipital artery. While intracerebral hemorrhage and NHNDs rarely appear in Borden-Shucart Type I dAVF, they are a typical observation in Borden-Shucart types II and III. Additionally, almost all patients reported in the literature had a history of rapid deterioration (1,6,7). Treatments include TAE, surgery, or a combination of both. According to our search, 80% of patients initially undergo endovascular embolization, and almost of them (94%) experience subsequent symptomatic improvement or radiographic resolution.

Various symptoms which manifest in dAVF are related to the mode of the venous drainage and the position of the lesion. Pulsatile tinnitus is the most common symptom, due to increased blood flow in the sigmoid and transverse sinus. Symptoms of other neurological deficits, such as seizures, parkinsonism, and thalamic dementia, may vary according to the position of the lesion (20). Patients with dAVF-thalamic dementia often become unable to take care of themself between 3 months and 1 year after onset, and can be easily misdiagnosed clinically (21).

To date, the exact etiology and pathogenesis of thalamic dementia caused by dAVF have not been fully clarified. It has been hypothesized that venous hypertension caused by venous sinus thrombosis (VST) is a key causative factor in the formation of dAVF (22). The mechanisms involved in dAVF resulting from VST are complex. In our patient, MR venography revealed that the left transverse sinus was absent. Initially, he was diagnosed with VST and given systematic anticoagulant therapy. However, this treatment did not relieve his symptoms, and only further aggravated them. Therefore, we hypothesized that venous drainage caused by an established dAVF might result in a turbulent or stagnant flow within the venous sinus, which subsequently leads to VST. After anticoagulant therapy, the VST of the patient may have recovered, meaning no evidence of VST was identified at the time of DSA. Nonetheless, the lack of an efficacious treatment of dAVF and the presence of ongoing venous hypertension led to an exacerbation of the patient's symptoms. Research has shown that venous congestion and ischemia of the thalamus lead to venous hypertension and ultimately result in thalamic dementia (2). Holekamp *et al*. reported 4 cases of thalamic dementia related to dAVF (1), with clinical manifestations similar to those of our patient.

The thalamus is the subcortical center and transfer station of sensory conduction, but it also has an influence on the activities of the limbic and ascending reticular systems, and the cerebral cortex. Thalamic dementia refers to neurodegenerative diseases that manifest as dementia, including thalamic lesions, neuronal loss, and astrocytic hyperplasia, the symptoms of which include memory decline and cognitive dysfunction. Thalamic dementia is generally caused by damage to the thalamic nucleus and the thalamic tract. Each nucleus in the thalamus has a special function and extensive two-way connections with the cortex and subcortical areas. The symptoms of dementia are mainly related to two complex pathways in the thalamus: the first is interruption of the nipple-thalamic tract/anterior nucleus complex pathway, which leads to an abnormal connection between the hippocampus and the cingulate gyrus; the other is the inner medullary plate-amygdala/dorsal medial nucleus and thalamus. The plate-core complex pathway is abnormal, resulting in the disconnection of the amygdala-orbital/

Table 1 Published cases of dAVF with dementia

Authors	Sex	Age (years)	Borden-Shucart grade	Arterial feeders	Presentation	Treatment	Outcome
Nakada *et al*., 1985 (19)	M	63	III	MMA	RPD, ataxia	Surgery	Death
Ito *et al.*, 1995 (18)	M	49	II	Bilat PMA & OccA	RPD, ataxia, aphasia	Surgery after TAE	Symptomatic/radiographic resolution
Greenough *et al*., 1999 (17)	M	62	Straight sinus	NA	RPD, ataxia	Surgery	Symptomatic/radiographic improvement
Tanaka *et al*., 1999 (16)	M	77	II	Bilat-OccA, rt-APA, bilat-MMA	RPD	Multiple TAEs	Symptomatic/radiographic near resolution
Bernstein *et al*., 2003 (15)	F	69	III	NA	RPD	TAE	Symptomatic/radiographic improvement
Tamamoto *et al*., 2003 (14)	M	67	III	Lt-STA, rt-MMA, lt-PMA	RPD, incontinence	TAE	Symptomatic/radiographic improvement
Tamamoto *et al*., 2003 (14)	M	73	II	Lt-ECA, lt-PMA	RPD, ataxia	Surgery	Symptomatic improvement, radiographic resolution
Gonçalves *et al*., 2008 (13)	M	43	II	Meningeal branches of rt ICA, rt ECA, rt OccA	RPD	TAE	Symptomatic/radiographic resolution
Matsumura *et al*., 2008 (12)	M	73	II	NA	RPD	TAE	Symptomatic improvement, radiographic resolution
Racine *et al*., 2008 (11)			III	Rt-OccA, lt-PMA	RPD	Surgery after TAE	Symptomatic improvement, radiographic resolution
Wilson *et al*., 2008 (10)	M	48	II	NA	RPD, aphasia, ataxia	TAE	Symptomatic/radiographic resolution
Sugrue *et al*., 2009 (9)	M	51	II	Bilat-MMA, PMA, marginal Tent A	RPD	TAE	Symptomatic resolution, radiographic improvement
Yamamoto *et al*., 2010 (8)	M	51	III	Bilat-ICA	RPD	TAE	Symptomatic improvement, radiographic resolution
Santillan *et al*., 2011 (7)	M	50	III	Medial Tent A, MMA	RPD	TAE	Symptomatic/radiographic resolution
Geraldes *et al*., 2011 (6)	M	64	II	Bilat-PMA, rt-MMA	RPD, ataxia	TAE	Symptomatic improvement, radiographic resolution
Holekamp *et al.*, 2016 (1)	M	53	II	Bilat-MMA, inferolateral trunks of ICAs	RPD, aphasia	TAE	Symptomatic improvement, radiographic resolution
Holekamp *et al.*, 2016 (1)	M	59	III	Lt-OccA, bilat-PMA	RPD, aphasia	TVE	Death from unrelated medical condition
Holekamp *et al*., 2016 (1)	M	60	III	Rt-OccA	RPD	Surgery after TAE	Symptomatic/radiographic resolution
Holekamp *et al*., 2016 (1)	M	71	III	Bilat-OccA, bilat-MMA	RPD, hemiparesis	Surgery after TAE	Symptomatic improvement, radiographic resolution
Present study	M	52	III	Lt-MMA	RPD	TAE	Symptomatic improvement, radiographic resolution

dAVF, dural arteriovenous fistula; APA, ascending pharyngeal artery; ICA, internal carotid artery; ECA, external carotid artery; MMA, middle meningeal artery; NA, not available; OccA, occipital artery; PMA, posterior meningeal artery; STA, superficial temporal artery; TentA, tentorial artery; RPD, rapidly progressive dementia; TAE, transarterial embolization; TVE, transvenous embolization; M, male; F, female.

dorsolateral prefrontal cortex. Abnormalities of the above pathways mainly manifest as amnesia, executive dysfunction, and behavioral abnormal syndrome. Cases of thalamic dementia caused by hypertensive intracerebral hemorrhage are relatively common, whereas dAVF-related cases are rare. In patients with dAVF, changes in the anatomical structure of the lesion area and abnormal hemodynamics form a connection between the thalamic nucleus and the cortex, which leads to the occurrence of thalamic dementia. Terada *et al*.'s research confirmed that venous hypertension is a primary event in the pathogenesis of dAVF (23). Venous hypertension possibly stimulates the expression of vascular endothelial growth factor (VEGF) and is highly implicated in the development of dAVF (24). Elevated venous pressure in dAVF can cause abnormalities such as bleeding, venous dilatation, and venous regurgitation (25), which lead to venous reflux obstruction and subsequently, bilateral thalamic hyperemia or ischemia. Moreover, when the engorged blood exceeds the venous capacity, hemorrhage occurs. Generally speaking, the internal cerebral veins collect the confluence of veins in the superior and medial portions of the thalamus, while the basilar artery recovers blood from veins in the inferior and lateral portions of the thalamus. Our patient presented with hypersomnolence, spatial disorientation, short-term memory impairment, and other symptoms, and speech dysfunction was also observed. Therefore, the symptoms of thalamic dementia caused by dAVF in this patient may be related to the above causes.

Studies have found that dAVF patients with dementia have a subacute onset, and their symptoms can show definite improvement or even disappear entirely after treatment (26-28). Therefore, for dAVF patients with thalamic dementia, early intervention is critical to prevent disease deterioration. However, in addition to the difficulty posed by diagnosis, effective treatment is often challenging for patients with a dAVF in the deep venous system. Endovascular embolization is the prevailing front-line standard of therapy for dAVF. In our literature review, we found that 94% of patients were treated with endovascular embolization and that it led to symptomatic improvement or radiographic resolution. The aim of endovascular embolization is to improve venous outflow of the normal cerebral parenchyma by disrupting the arteriovenous shunt (29). For patients with dAVF-induced dementia, early endovascular embolization may reduce venous hypertension, reverse venous bleeding, and eliminate the risk of hemorrhage. As cerebral venous hemodynamics improve, neurological function is restored over the subsequent few months. In our case and in two previously reported cases (3,29), cognitive amelioration was linked to a reduction in venous hypertension and congestion after occlusion of the dAVF. Postoperative MRI also confirmed this link, showing a reversal of the T2 weighted imaging signal changes and a reduction in the dilated intramedullary veins. Although endovascular embolization is regarded as the front-line criterion for dAVF, in a recent review, Velz *et al*. reported that complete occlusion of the dAVF was achieved in only 33.3% of patients, resulting in extremely high morbidity and mortality (30). Therefore, endovascular treatment is difficult for certain dysplastic dAVFs and in cases where the main blood supply arteries are thin or there is thrombosis or narrowing of the venous sinuses. Microsurgical resection is deemed to be the adequate approach for the treatment of such cases. Nevertheless, compared with endovascular embolization, microsurgical resection carries a higher risk, as exposure of the dAVF is difficult and can potentially lead to bleeding events (31). Moreover, it is difficult to resolve cases of dAVF with complex angioarchitecture and hemodynamics using a single method, and a multiple approach combination including endovascular embolization, microsurgery, and stereotactic radiosurgery often provides better results.

In conclusion, thalamic dementia is a peculiar but reversible manifestation of dAVF, and its pathogenesis may be associated with venous hypertension. The majority of cases of thalamic dementia caused by dAVF have the characteristics of subacute attack, and its symptoms can be improved or even completely eliminated by treatment. Therefore, for patients with dAVF with rapidly progressive dementia, early and differential diagnosis is critical, and early intervention through surgery or TAE is necessary to prevent further deterioration of cognitive function.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://qims.amegroups.com/article/view/10.21037/qims-21-1054/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related

to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

References

1. Holekamp TF, Mollman ME, Murphy RK, Kolar GR, Kramer NM, Derdeyn CP, Moran CJ, Perrin RJ, Rich KM, Lanzino G, Zipfel GJ. Dural arteriovenous fistula-induced thalamic dementia: report of 4 cases. J Neurosurg 2016;124:1752-65.
2. Iwasawa E, Ishibashi S, Miki K, Yoshino Y, Nemoto S, Mizusawa H. Teaching NeuroImages: reversible cognitive impairment with bithalamic lesions caused by a dural arteriovenous fistula. Neurology 2013;81:e38-9.
3. Brito A, Tsang ACO, Hilditch C, Nicholson P, Krings T, Brinjikji W. Intracranial Dural Arteriovenous Fistula as a Reversible Cause of Dementia: Case Series and Literature Review. World Neurosurg 2019;121:e543-53.
4. Cheng ZJ, Tu JL, He JL, Li J. Rapidly progressive cognitive impairment caused by intracranial dural arteriovenous fistulas (DAVFs): a case report. Neurol Sci 2018;39:1293-6.
5. Hernández-Díaz ZM, Llíbre-Guerra JC, Arteche-Prior M, de la Paz-Bermúdez T, Águila-Ruiz AF, Figueroa-García LM, de Los Ángeles Robinson-Agramonte M. Spontaneous Subdural Hematoma and Behavioral Changes Due to a Dural Arteriovenous Fistula. A Case Report and Literature Review. Behav Sci (Basel) 2019;9:63.
6. Geraldes R, Albuquerque L, Ferro JM, Sousa R, Sequeira P, Campos J. Rapidly progressive cognitive impairment, ataxia, and myoclonus: an unusual presentation of a dural arteriovenous fistula. J Stroke Cerebrovasc Dis 2012;21:619.e3-5.
7. Santillan A, Safdieh JE, Gobin YP, Patsalides A. Neurological picture. Bilateral thalamic venous hypertension caused by a tentorial dural arteriovenous fistula: endovascular treatment. J Neurol Neurosurg Psychiatry 2011;82:749-50.
8. Yamamoto T, Watanabe M, Miura A, Hirahara T, Hirano T, Uchino M. A case of dural arteriovenous fistula associated with bilateral thalamic lesions. Rinsho Shinkeigaku 2010;50:718-24.
9. Sugrue PA, Hurley MC, Bendok BR, Surdell DL, Gottardi-Littell N, Futterer SF, Muro K, Batjer HH. High-grade dural arteriovenous fistula simulating a bilateral thalamic neoplasm. Clin Neurol Neurosurg 2009;111:629-32.
10. Wilson M, Enevoldson P, Menezes B. Intracranial dural arterio-venous fistula. Pract Neurol 2008;8:362-9.
11. Racine CA, Lawton MT, Hetts SW, Josephson SA. Neuropyschological profile of reversible cognitive impairment in a patient with a dural arteriovenous fistula. Neurocase 2008;14:231-8.
12. Matsumura A, Oda M, Hozuki T, Imai T, Shimohama S. Dural arteriovenous fistula in a case of dementia with bithalamic MR lesions. Neurology 2008;71:1553.
13. Gonçalves MB, Maia O Jr, Correa JL, Siqueira SB, Christoph Dde H, Landeiro JA. Dural arteriovenous fistula presenting as thalamic dementia. Arq Neuropsiquiatr 2008;66:264-7.
14. Tamamoto F, Nakanishi A, Takanashi T, Ishizaki H, Nagasawa H, Maehara T, Ohno S, Otsubo Y. Unexpected accumulation of thallium-201 in bilateral thalamic venous infarction induced by arteriovenous fistula in the posterior fossa: report of a case. Ann Nucl Med 2003;17:239-43.
15. Bernstein R, Dowd CF, Gress DR. Rapidly reversible dementia. Lancet 2003;361:392.
16. Tanaka K, Morooka Y, Nakagawa Y, Shimizu S. Dural arteriovenous malformation manifesting as dementia due to ischemia in bilateral thalami. A case report. Surg Neurol 1999;51:489-93; discussion 493-4.
17. Greenough GP, Mamourian A, Harbaugh RE. Venous hypertension associated with a posterior fossa dural arteriovenous fistula: another cause of bithalamic lesions on MR images. AJNR Am J Neuroradiol 1999;20:145-7.
18. Ito M, Sonokawa T, Mishina H, Sato K. Reversible dural arteriovenous malformation-induced venous ischemia as a cause of dementia: treatment by surgical occlusion of draining dural sinus: case report. Neurosurgery

1995;37:1187-91; discussion 1191-2.
19. Nakada T, Kwee IL, Ellis WG, St John JN. Subacute diencephalic necrosis and dural arteriovenous malformation. Neurosurgery 1985;17:653-6.
20. Hwang H, La YK, Baek MS, Baik K, Suh SH, Kim WJ. Dural Arteriovenous Fistula Manifested as Rapid Progressive Dementia Successfully Treated by Endovascular Embolization Only. Neurointervention 2017;12:50-3.
21. Morparia N, Miller G, Rabinstein A, Lanzino G, Kumar N. Cognitive decline and hypersomnolence: thalamic manifestations of a tentorial dural arteriovenous fistula (dAVF). Neurocrit Care 2012;17:429-33.
22. Hoang TT, Do HD, Do HT, Nguyen CN, Alward WLM. Bilateral Blood Reflux Into Schlemm Canals Caused by a Unilateral Dural Sinus Arteriovenous Fistula in a Patient With Cerebral Venous Thrombosis. J Neuroophthalmol 2021. [Epub ahead of print]. doi:10.1097/WNO.0000000000001419.
23. Terada T, Higashida RT, Halbach VV, Dowd CF, Tsuura M, Komai N, Wilson CB, Hieshima GB. Development of acquired arteriovenous fistulas in rats due to venous hypertension. J Neurosurg 1994;80:884-9.
24. Bhogal P, Yeo LL, Henkes H, Krings T, Söderman M. The role of angiogenesis in dural arteriovenous fistulae: the story so far. Interv Neuroradiol 2018;24:450-4.
25. Jain NK, Kannath SK, Kapilamoorthy TR, Thomas B. The application of susceptibility-weighted MRI in pre-interventional evaluation of intracranial dural arteriovenous fistulas. J Neurointerv Surg 2017;9:502-7.
26. Wen HY, Yang ST, Lo YL. Hydrocephalus with Ventricular Papillary Structure Induced by Aggressive Intracranial Dural Arteriovenous Fistulas. World Neurosurg 2019;132:208-10.
27. Sadeh-Gonike U, Magand N, Armoiry X, Riva R, Labeyrie PE, Lamy B, Lukaszewicz AC, Lehot JJ, Turjman F, Gory B. Transarterial Onyx Embolization of Intracranial Dural Fistulas: A Prospective Cohort, Systematic Review, and Meta-Analysis. Neurosurgery 2018;82:854-63.
28. Carrasco R, Pascual JM. Letter to the Editor: Dural arteriovenous fistula: a clinical model of thalamic dementia? J Neurosurg 2017;126:1020-2.
29. Zeidman SM, Monsein LH, Arosarena O, Aletich V, Biafore JA, Dawson RC, Debrun GM, Hurko O. Reversibility of white matter changes and dementia after treatment of dural fistulas. AJNR Am J Neuroradiol 1995;16:1080-3.
30. Velz J, Kulcsar Z, Büchele F, Richter H, Regli L. The Challenging Clinical Management of Patients with Cranial Dural Arteriovenous Fistula and Secondary Parkinson's Syndrome: Pathophysiology and Treatment Options. Cerebrovasc Dis Extra 2020;10:124-38.
31. Lin N, Brouillard AM, Mokin M, Natarajan SK, Snyder KV, Levy EI, Siddiqui AH. Direct access to the middle meningeal artery for embolization of complex dural arteriovenous fistula: a hybrid treatment approach. J Neurointerv Surg 2015;7:e24.

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