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Case Report

Dural Arteriovenous Fistula of the Transverse and Sigmoid Sinus Manifesting Ascending Dysesthesia: Case Report and Literature Review

Yoshinobu Kamio,[1] Hisaya Hiramatsu,[1] Shuhei Yamashita,[2] Mika Kamiya,[2] Yasushi Sugiura,[3] and Hiroki Namba[1]

Cases involving intracranial dural arteriovenous fistulas (AVFs) with spinal perimedullary venous drainage exhibit variable presentations, which results in delayed diagnoses. We describe a case of a 66-year-old female with a transverse-sigmoid sinus dural AVF with spinal perimedullary venous drainage who developed dysesthesia and hypalgesia that ascended from the peripheral lower extremities. Sixty cases of intracranial dural AVFs resulting in myelopathy have been reported, and an absence of brainstem signs significantly correlated with a delay in diagnosis (positive group: 3.4 months vs. negative group: 9.6 months, $P < 0.05$). Intracranial dural AVFs with brainstem signs should be diagnosed without delay because the myelopathy and bulbar symptoms could progress aggressively without alternative drainage routes besides the perimedullary veins. We emphasize that intracranial dural AVFs should be considered as a differential diagnosis in case presenting with symptoms, such as atypical dysesthesia and hypalgesia ascending from the toes, without brainstem signs. Moreover, we should perform cerebral angiography as early as possible because dural AVFs with slow-flow venous drainage can produce false negatives on magnetic resonance angiography.

Keywords: dysesthesia, intracranial dural AVF, myelopathy, cerebral angiography

Introduction

A spinal dural arteriovenous fistula (AVF) occasionally produces motor and sensory disturbances of the lower extremities. Generally, intracranial dural AVFs result in headache, pulsatile tinnitus, ocular symptoms (chemosis, exophthalmos, and diplopia) and focal signs, including hemiparesis and hemisensory disturbances. In the present case, we experienced a patient with an intracranial dural AVF that was identified by ascending dysesthesia and hypalgesia from both toes to the thighs on both sides, no motor paresis of the lower extremities, and no symptoms of the upper extremities. Therefore, we describe this case and review the past relevant literatures.

Departments of [1]Neurosurgery and [2]Radiology, Hamamatsu University School of Medicine, Hamamatsu, Shizuoka
[3]Department of Endovascular Neurosurgery, Seirei Mikatahara General Hospital, Hamamatsu, Shizuoka

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Case Report

A 66-year-old female presented with progressive dysesthesia and hypalgesia ascending from both toes to the thighs on both sides. A neurological examination revealed that the cranial nerve, deep tendon reflex, strength, and coordination of both upper and lower extremities were almost normal and that the dysesthesia and hypalgesia occurred at less than the L3 level. Her symptoms were suspected to be due to a spinal dural AVF, inflammatory demyelinating disease, or a spinal tumor at the lumbar level. Thoracic and lumbar magnetic resonance imaging (MRI) with a T_2-weighted image revealed no lesion. Her symptoms were not due to a pathological reason. She underwent a medical examination by several orthopedists and a neurologist. Cervical MRI with a T_2WI revealed a high intensity lesion on the dorsal surface of the cervical cord between the C2 and C4 levels (Fig. 1A, B). Cervical enhanced MRI revealed serpentine vessels around the cervical spinal cord and medulla oblongata. She underwent angiography, and an intracranial dural AVF was suspected. Eight months after onset, she was referred to our institution. A neurological examination revealed normal cranial nerves, normal motor function of both upper and lower extremities, both dysesthesia and hypalgesia at less than the L3 level, impaired vibration sense at the knee and foot joint, and hyperreflexia of the deep tendon reflex of both lower extremities. An angiogram revealed that a left transverse-sigmoid sinus dural AVF (Cognard type V) was supplied by the left occipital artery and middle meningeal artery, with a shunting site isolated at the left transverse-sigmoid sinus (occlusion of the proximal side of the left sigmoid sinus and severe stenosis of the junction of left transverse-sigmoid sinus), and drainage of the anterior spinal perimedullary vein (ASPV) by the superior petrosal sinus and petrosal vein, and anterior pontomesencephalic vein. Another drainage route other than the ASPV at the C-2 level was the internal vertebral venous plexus through the lateral medullary vein and bridging vein. This dural AVF drainage occurred through the right jugular vein through the left transverse sinus, confluence, right transverse sinus, and right sigmoid sinus (Fig. 1C, D). In this case, venous hypertension of the spinal cord was suggested to be the cause of the cervical myelopathy.

In order to reduce the venous hypertension and obliterate the arteriovenous shunt, transvenous embolization of the left sigmoid sinus and transverse sinus was performed. After

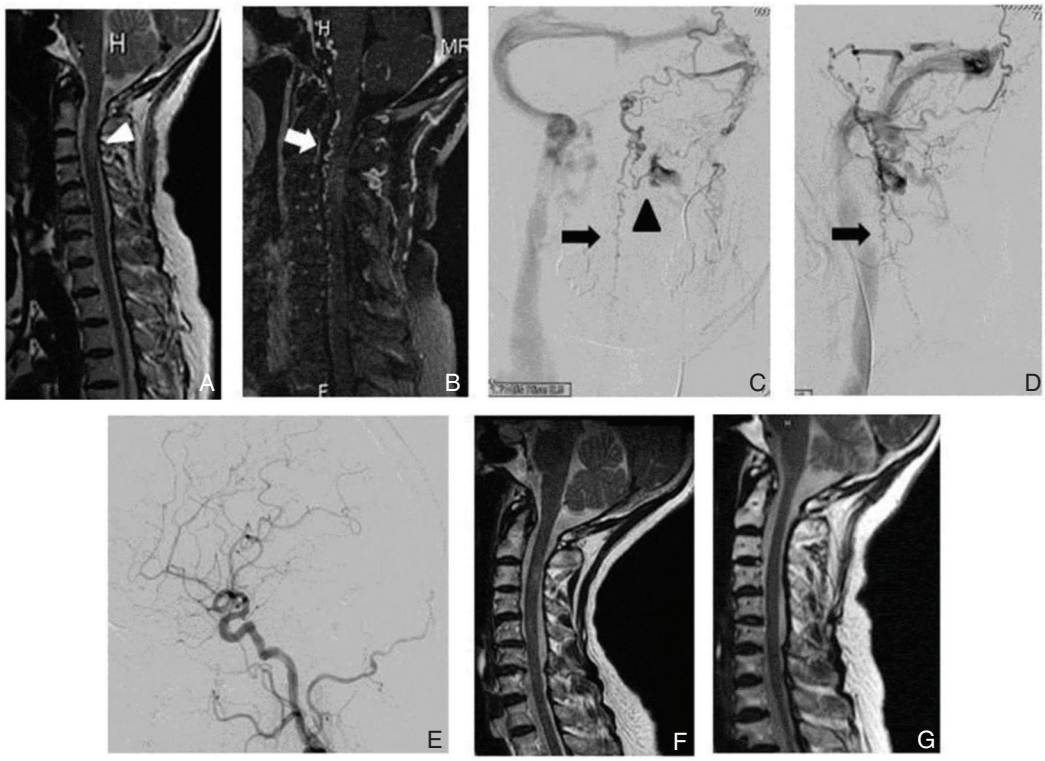

Fig. 1 A: Spine sagittal T_2-wighted MRI showing an high intensity in dorsal surface between C2–C4 levels (white arrowhead). B: Sagittal gadolinium-enhanced cervical MRI shows the perimedullary vein (white arrow). C, D: Left external carotid angiograms, anteroposterior (C) and lateral (D) views, shows a left tranverse-sigmoid sinus dural arteriovenous fistula supplied by the left middle meningeal artery and occipital artery. Venous drainage is contralateral transverse sinus and anterior spinal perimedullary vein through superior petrosal sinus (black arrow). Other drainage route in C-2 level is internal vertebral venous plexus and radiculospinal vein through radiculomedullary vein (black arrowhead). E: Left common carotid angiogram (lateral view) after embolization shows no contrast filling of the dural arteriovenous fistula. F: 1 month after embolization, T_2-weighted MRI of cervical spinal cord showed the cervical high intensity region was improved gradually. G: Cervical high intensity region was disappeared 1 year after embolization. MRI: magnetic resonance imaging.

embolization, the left external carotid angiography showed obliteration of the fistula and disappearance of the spinal perimedullary venous drainage. Three months postoperatively, a cervical MRI with T_2WI revealed a remarkably decreased extent of high intensity. The symptoms of dysesthesia and hypalgesia improved gradually (Fig. 1E–G).

Discussion

Myelopathy due to venous hypertension of the spinal cord that is induced by a spinal dural AVF is a relatively commonly encountered vascular malformation, and the fistula most often occurs in the thoracic region. Woimant et al. were the first to describe intracranial dural AVFs with perimedullary venous drainage in 1982.[1] Since the first reports, seven cases of transverse-sigmoid sinus dural AVFs with perimedullary venous drainage have been reported.[2–7] The findings of spinal cord swelling with abnormal MRI signals are nonspecific and could be considered to be caused by a tumor, demyelination, myelitis, and/or trauma. Previous reports have emphasized that when symptoms suggest a spinal dural AVF and a spinal angiography is normal, we should examine the intracranial region because Cognard et al. have reported that type V dural AVFs produce progressive myelopathy in 50% of the cases and the other 50% of cases exhibit hemorrhage.[8] Although type V dural AVFs are rare, they are rapidly progressive in 25% of the cases.[9] It was extremely rare that the present case only manifested the sensory disorders of dysesthesia and hypalgesia. Therefore, an atypical dural AVF could lead to a delayed or incorrect diagnosis. The present case had another drainage route into the contralateral transverse-sigmoid sinus besides the spinal perimedullary vein, and therefore could avoid the rapid progression of myelopathy. In short, the presence of several drainage routes could reduce the venous hypertension by dividing the venous flow. Kwon et al. have reported that an intracranial dural AVF with slow venous flow was not visualized with magnetic resonance angiography (MRA) resulting in a false negative.[10] Haryu et al. described contrast-enhanced dynamic MRA was more sensitive and useful in detecting enlarged spinal veins compared to T_2-weighted MRI.[11] By using digital subtraction angiography, visualization of the venous drainage pattern, as well as prompt and correct diagnosis, can be achieved. Therefore, when symptoms and MRI/MRA findings suggest a spinal dural AVF but spinal angiography does not show any abnormality, intracranial angiography should be performed because intracranial dural AVFs (Cognard type V) progressively produce myelopathy.

Aminoff et al. were the first to propose the theory of venous hypertension, and Merland et al. reported that venous hypertension might be produced by any cranial or spinal

dural AVFs in 1974.[12–14] Venous hypertension is considered to result in arterial flow into the spinal perimedullary veins and low perfusion, causing the stagnation of blood flow and ischemia of the spinal cord. Moreover, although the intradural spinal venous system is valveless and the venous perimedullary coronal plexus is in direct continuity with the veins of the posterior fossa, a venous congestive myelopathy is facilitated by the transmission of a high venous pressure to the spinal cord tissue, Suh and Alexander have suggested that there are valve structures between penetrating veins and medium-sized intramedullary veins because of the obstruction in a dye-injection experiment and that the valves of veins in the spinal cord are responsible for the tendency of higher venous pressure in the outer circumference.[15–17] These reasons suggest the possibility that the sensory disorder was dominant in the lower extremities. In this case, the dysesthesia and hypalgesia except for the motor function, could account for the disorder of the nerve fiber of the lateral spinothalamic tract and the dorsal column in the leg area that ran up the outermost region.

In our review of previous reports, 60 cases of intracranial dural AVFs leading to myelopathy have been reported (Table 1).[15,16,18–20] The patients ages ranged from 20 years to 79 years (mean, 57 years), with the male to female sex ratio of the 60 cases being 3:1 (Table 2). Most intracranial dural AVFs with spinal venous drainage cause myelopathy and

Table 1 Characteristic of reported 60 cases

Case	Age y/o, sex	Symptoms Brainstem sign (+/–)	Feeder/Drainage	Site of shunt	Delay in diagnosis (months)	Outcome
1 (20)	43, M	Tetraplegia, (–)	OA, MHA/Petrosal vein,	Petrous apex	20	GR
2 (20)	68, M	Paraplegia, (–)	OA, MHA/PMV	Tentorium	6	SD
3 (20)	42, M	Paraplegia, (–)	OA, APA/Petrosal vein, PMV	Tentorium	7	MD
4 (2)	35, F	Tetraplegia, (+)	OA, MMA/PMV	Lateral sinus	4	GR
5 (2)	37, M	Tetraplegia, (+)	MMA/Petrosal sinus	Petrous region	9	Death
6 (2)	53, M	Tetraplegia, (–)	MHA/PMV	Tentorium	5	SD
7 (2)	69, M	Paraplegia, (–)	APA, OA/SPS	Petrous sinus	12	GR
8 (2)	68, F	Tetraplegia, (+)	OA, APA, MMA/SPS	Petrous sinus	4	MD
9 (18)	69, M	Paraplegia, (–)	MHA/PMV	Tentorium	Unknown	Unknown
10 (20)	40, M	Paraplegia, (–)	MHA/PMV	Tentorium	12	GR
11 (20)	63, M	Paraplegia, (–)	PMA/PMV	FM	4	GR
12 (20)	74, M	Paresthesia, (–)	PMA/PMV	FM	6	Death
13 (7)	50, M	Tetraplegia, (+)	MHA, OA/SPS, PMV	Petrous sinus	7	GR
14 (7)	71, M	Tetraplegia, (–)	OA, MMA/SPS, PMV	Petrous sinus	4	GR
15 (19)	78, F	Paraplegia, (–)	Unknown/PMV	Tentorium	6	SD
16 (19)	42, M	Paraplegia, (–)	Unknown/PMV	Tentorium	Unknown	GR
17 (20)	31, M	Paraplegia, (–)	MHA/TS, pontomesencephalic vein	Tentorium	4	MD
18 (20)	36, M	Tetraplegia, (–)	MMA, VA/Inferior vermian vein	Torcular region	1	SD
19 (20)	47, M	Tetraplegia, (–)	VA/Inferior vermian vein	Torcular region	12	SD
20 (20)	74, M	Paraplegia, (–)	VA/PMV	FM	Unknown	SD
21 (20)	50, F	Paraplegia, (–)	OA, MMA/Lateral venous sinus	Lateral sinus	6	GR
22 (20)	67, M	Tetraplegia, (–)	APA, OA/PMV	Petrous sinus	6	MD
23 (12)	36, M	Tetraplegia, (–)	VA/PMV	Tentorium	12	SD
24 (4)	69, M	Tetraplegia, (–)	ICA meningeal branch/PMV	Tentorium	36	Death
25 (4)	53, M	Paraplegia, (–)	MMA, ICA meningeal branch/ Petrosal vein, PMV	Tentorium	6	GR
26 (4)	40, F	Tetraplegia, (+)	ICA meningeal branch /SOV, PMV,SPS	Cavernous sinus	12	Death
27 (4)	75, F	Tetraplegia, (+)	MMA/PMV	Petrous sinus	Few days	GR
28 (4)	51, F	Paraplegia, (+)	OA, MMA/right sigmoid sinus	Sigmoid sinus	3	GR
29 (20)	68, M	Tetraplegia, (–)	VA/PMV	CCJ	6	MD
30 (15)	70, M	Dysesthesia, (–)	VA/PMV, AMV	CCJ	27	GR
31 (20)	46, M	Paraplegia, (+)	APA/PMV	Unknown	Few days	GR
32 (13)	65, M	Tetraplegia, (+)	OA, PMA/Petrosal vein	Torcular resion	3	GR
33 (17)	69, M	Paraplegia, (–)	APA, VA/PMV	CCJ	48	MD
34 (17)	53, M	Tetraplegia, (–)	APA/PMV	FM	24	GR
35 (10)	58, M	Tetraplegia, (+)	VA/PMV	CCJ	6	Death
36 (18)	64, M	Paraplegia, (+)	APA/PMV	Tentorium	0.5	SD
37 (6)	71, M	Paraplegia, (–)	ICA meningeal branch/SPS	Petrous sinus	Unknown	MD
38 (6)	47, M	Tetraparesis, (–)	ECA, VA/Petrosal vein	FM	5	SD
39 (6)	58, F	Tetraparesis, (–)	APA/Petrosal sinus	Skull base	Unknown	SD

(*Continued*)

Table 1 (Continued)

Case	Age y/o, sex	Symptoms Brainstem sign (+/–)	Feeder/Drainage	Site of shunt	Delay in diagnosis (months)	Outcome
40 (20)	57, M	Tetraplegia, (–)	APA/PMV	FM	36	SD
41 (20)	79, M	Tetraplegia, (–)	VA, APA, posterior auricular branch/PMV	Skull base	6	MD
42 (20)	58, M	Paraplegia, (–)	MHA/PMV	Tentorium	0.5	GR
43 (16)	68, M	Dysesthesia, (–)	APA/AMV, PMV	CCJ	10	MD
44 (12)	45, M	Paraplegia, (–)	OA, APA/PMV	Tentorium	2	SD
45 (20)	42, M	Tetraplegia, (–)	ICA meningeal branch/PMV	Tentorium	15	MD
46 (1)	60, M	Tetraplegia, (–)	MHA/vein of tentorium, cerebellomedullary fissure	CCJ	1	GR
47 (14)	73, M	Paraplegia, (+)	MMA, OA/sigmoid sinus	Transverse sinus	12	MD
48 (16)	45, M	Tetraplegia, (+)	ICA meningeal branch/PMV	Tentorium	6	MD
49 (20)	68, M	Paraplegia, (–)	ICA meningeal branch/PMV	Tentorium	3	MD
50 (20)	58, M	Tetraplegia, (–)	MHA, APA, MMA/Petrosal vein	Tentorium	3	GR
51 (20)	65, M	Tetraplegia, (–)	Stylomastoid artery/Petrosal vein	Petrous region	12	SD
52 (20)	72, F	Tetraplegia, (+)	Unknown	FM	24	SD
53 (20)	62, F	Tetraplegia, (+)	APA, OA/IPS	FM	Few days	SD
54 (20)	48, M	Tetraplegia, (+)	MHA/PMV	Tentorium	6	SD
55 (20)	62, F	Paraplegia, (–)	VA/PMV	CCJ	9	MD
56 (20)	20, F	Tetraplegia, (+)	MHA/cerebellar vein	Tentorium	0.5	MD
57 (20)	38, F	Tetraplegia, (+)	MMA, OA, APA, MHA, VA/PMV	Transverse-sigmoid sinus	0	MD
58 (20)	70, M	Tetraplegia, (–)	MMA, OA/IPS, anterior condylar vein	ACC	24	SD
59 (6)	69, F	Paraplegia, (+)	MMA, OA, APA/AMV, PMV	Sigmoid sinus	1	SD
60 (20)	58, F	Tetraplegia, (+)	VA meningeal branch/PMV	CCJ	0	MD

(): reference number, OA: occipital artery, APA: ascending pharyngeal artery, MMA: middle meningeal artery, VA: vertebral artery, MHA: meningohypophyseal artery, PMV: posterior medullary vein, PMA: perimedullary artery, TS: transverse sinus, ICA: internal carotid artery, ECA: external carotid artery, AMV: anterior medullary vein, IPS: inferior petrosal vein, SPS: superior petrosal vein, SOV: superior orbital vein, ACC: anterior condylar confluence, CCJ: craniocervical junction, FM: foramen magnum, GR: good recovery, SD: severe disability, MD: moderate disability.

Table 2 Characteristic of epidemiology and clinical findings in 60 cases

Mean age (y/o)	57
Sex (M:F)	45:15
Location	
Tentorium	20 (33%)
Foramen magnum	8 (13%)
Craniocervical junction	8 (13%)
Petrous sinus	7 (12%)
Transverse-sigmoid	6 (10%)
Torcular region	3 (5%)
Others	8 (13%)
Clinical manifestation	
Tetraparesis	35 (58%)
Paraparesis	22 (37%)
Dysesthesia	2 (3.3%)
Hemiparesis	1 (1.7%)
Brainstem dysfunction	20 (33%)
Prognosis	
Good recovery (GR)	19 (32%)
Moderate disability (MD)	17 (28%)
Severe disability (SD)	19 (31%)
Death	5 (8%)
Delay in diagnosis (mean month)	
Brainstem symptoms (+)	3.9
Brainstem symptoms (–)	11.9
Good prognosis (GR, MD)	10.3
Poor prognosis (SD, death)	11.3

motor function disorder, and the clinical manifestations included paraparesis in 22 cases, tetraparesis in 35 cases, and hemiparesis in one case. Moreover, the clinical outcomes of the reviews were unfavorable: 19 of 60 cases (32%) showed a good recovery, 17 cases (28%) showed moderate disability, and 24 cases (40%) showed a poor outcome or death after treatment. We conducted a quantity comparative statistical analysis using a Student's t-test. All statistical results were considered significant if $P < 0.05$. The analysis was performed with EZR (Saitama Medical Center, Jichi Medical University, Saitama), which is a graphical user interface for R (The R Foundation for Statistical Computing). While it was a small population, the comparison of the delay in diagnosis from onset between the brainstem sign positive group and the negative group showed that brainstem sign positive group had a significantly shorter delay than the negative group did (3.4 months vs. 9.6 months, respectively; $P = 0.021$). However, there was no significant difference between the prognosis and the delay in diagnosis (good prognosis: 10.3 months vs. poor prognosis: 11.3 months, $P = 0.35$). These results suggest that intracranial dural AVFs with brainstem signs experience aggressive progression, but the correlation between the disease duration and prognosis is relatively low in literature review. We speculate that severity of venous hypertension due to venous drainage pattern is an important factor influencing prognosis. However, it is important to arrive at a prompt and accurate diagnosis because early

diagnosis offers the possibility of improving the reversible symptoms and avoiding the poor outcomes.

Conclusion

The clinical manifestations of dural AVFs are related to venous drainage pattern and, not to the fistula location. Therefore, the symptoms of intracranial dural AVFs with spinal perimedullary venous drainage (Cognard type V) are often related to the spinal dysfunction, rather than to the brain. We should perform cerebral and spinal angiography without delay and consider that angiography should be performed as early as possible because the intracranial dural AVFs with slow-flow drainage may produce false negative findings on MRA. Delayed and incorrect diagnosis might result in the poor outcomes of irreversibly severe spinal injury and Foix-Alajouanine syndrome, which is considered to be the result of progressive vascular thrombosis resulting in a necrotic myelopathy due to spinal-continuous venous hypertension.

Conflicts of Interest Disclosure

The authors declare that they have no competing interests and no financial support.

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Corresponding author:
Hisaya Hiramatsu, MD, Department of Neurosurgery, Hamamatsu University School of Medicine, Handayama 1-20-1, Higashi-ku, Hamamatsu, Shizuoka 431-3192, Japan.
✉ jzt00467@nifty.com