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Isolated Superficial Sylvian Vein Thrombosis with Long Cord Sign: Case Report and Review of the Literature

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Abstract

Isolated cortical vein thrombosis (ICVT) is extremely rare. Only single case or small series of ICVT have been reported; clinical details are still uncertain. We report a case of isolated superficial sylvian vein thrombosis with exceedingly long cord sign. A 14-year-old female with severe sudden onset headache visited our hospital. Fluid attenuated inversion recovery and echo-planar T_2* susceptibility-weighted imaging (T_2*SW) showed a long cord sign on the surface of the sylvian fissure. The patency of dural sinuses and deep cerebral veins were confirmed by magnetic resonance venography (MRV), and diagnosis of ICVT was made. She recovered completely without anticoagulant agents. To clarify the clinical characteristics of ICVT, we reviewed 51 ICVT cases in the literature. In many cases, T_2*SW was the most useful examination to diagnose ICVT. In contrast with general cerebral venous thrombosis, MRV and conventional angiography were either supporting or useless. Anastomotic cortical veins were involved frequently; symptoms of gyri around the veins were common. It also suggested that ICVTs of the silent area might have been overlooked because of nonspecific symptoms, and more patients with ICVT may exist. In cases involving patients with nonspecific symptoms, the possibility of ICVT should be considered.

Key words: isolated cortical vein thrombosis, sylvian vein, cord sign, cerebral venous thrombosis, T_2*

Introduction

Cerebral venous thrombosis (CVT) is rare, representing < 1% of all cerebral strokes.[1)] Thrombosis of cortical veins not involving the dural sinuses or deep cerebral veins, i.e., isolated cortical vein thrombosis (ICVT), is extremely rare and reportedly accounts for 6.3% of overall CVT.[2)] Only single cases or small series of ICVT have been reported; clinical details are limited. Usually, definitive diagnosis of ICVT is made by detecting a thrombosed vein, by cord sign or dot sign in imaging; however, this is often not easy because the signs are not as obvious as in CVT cases and are detected in only one or a few modalities/sequences even if many imaging examinations are performed. We report a case of isolated superficial sylvian vein thrombosis with exceedingly long cord sign detected by magnetic resonance imaging (MRI). ICVT with such a long thrombosed vein is rare. We also reviewed 51 ICVT cases and summarized the clinical characteristics of ICVT compared with those of overall CVT.

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Case Report

A 14-year-old female with severe sudden onset headache came to our hospital. Analgesics did not relieve it. Except for slight dizziness, she had no other symptoms. Her past medical history was unremarkable. She denied taking any medications including oral contraceptives. Her familial medical history was negative for hypercoagulable disorders. Neurological examination results were normal. Laboratory examination results were normal including complete cell count, activated partial thromboplastin time, prothrombin time, fibrinogen, plasminogen, antithrombin III, lipoprotein (a), protein C activity, protein S activity, anti-cardiolipin antibody, and lupus anticoagulant.

Brain noncontrast computed tomography (CT), T_1-, T_2-, and diffusion-weighted imaging (T_1WI, T_2WI, and DWI) findings were unremarkable. Fluid-attenuated inversion recovery (FLAIR) showed a long restiform high-intensity lesion on the surface of the sylvian fissure (Fig. 1A, C, D) that started in the left sylvian fissure around the supramarginal gyrus, followed the surface of the sylvian fissure, and ended at the sphenoid ridge. It was comparable to the superficial sylvian vein and continued to the

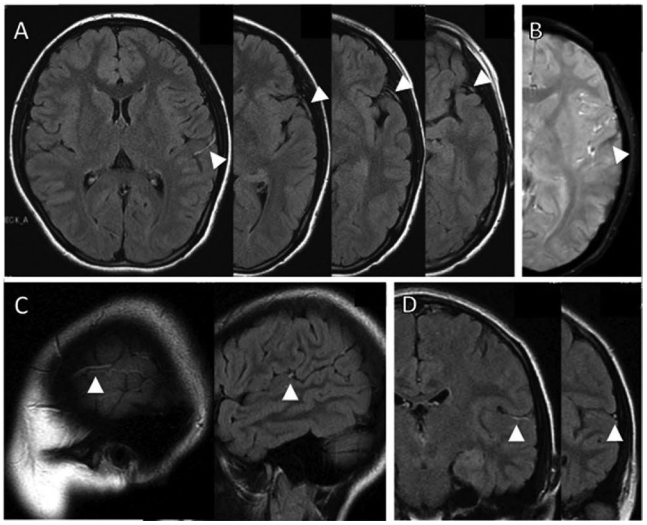

Fig. 1 Fluid attenuated inversion recovery (FLAIR) showed cord sign and dot sign consistent with those of the superficial Sylvian vein. Axial sections (A), sagittal sections (C), and coronal sections (D). Echo-planar T_2* susceptibility-weighted imaging also showed a restiform low-intensity lesion that coincided with the high-intensity lesion in FLAIR (B).

sphenoparietal sinus. The lesion showed a low-intensity signal in echo-planar T_2* susceptibility-weighted imaging (T_2*SW) (Fig. 1B). There was no obvious signal change in the parenchyma suggestive of edema, infarction, or hemorrhage. Magnetic resonance angiography (MRA) and magnetic resonance venography (MRV) detected no structure corresponding to the restiform lesion in FLAIR and T_2*SW. The patency of the dural sinuses and deep cerebral veins was confirmed by MRV, and diagnosis of ICVT was made.

She was treated conservatively without anticoagulant agents. Except for occasional negligible discomfort of the head, she is well without any neurological deficit.

Discussion

We reviewed 51 ICVT cases reported to date including our case (Table 1). The mean age of 51 patients with ICVT was 38.3 years (range, 14–78 years), similar to that of patients with overall CVT (39.1 years),[3] and male:female ratio was 1:2.0 with proportionally more males than in CVT (1:2.9).[3] Coagulation abnormalities were observed in 7 cases, including reduced protein C/S activity,[4] factor V Leiden,[2,5] mutation of methylenetetrahydrofolate reductase,[6] and the 4G/4G genotype of plasminogen activator inhibitor-1.[6] Many patients were suspected to be in a hypercoagulable state because of contraceptive intake,[2,7–12] postpartum status,[2,4–6,13] Behcet's disease,[2] Hodgkin's disease,[11] leukemia,[14] and ulcerative colitis.[15] In total, patients who had coagulation abnormalities represented 44.2% of ICVT. Ten patients (23.3%) had intracranial hypotension syndrome (IHS) (7 after lumber puncture), which was the second largest group with underlying disease.[2,6,7,11,13,16,17] IHS was reported to be a risk factor for CVT.[18–20] The proportion associated with IHS of ICVT was relatively higher than that of CVT (2%).[3] Decreasing blood flow and blood vessel distortion are thought to be causes of CVT with IHS;[21] they also might be causes of ICVT in patients with IHS. Moreover, 6 cases (14.0%) were associated with some kinds of infection.[9,11,22–24]

The most frequent symptoms were seizure and headache observed in 34 (66.7%) and 33 (64.7%) patients, respectively; those reported in overall CVT were approximately 40% and 90%, respectively.[3] Headaches were less frequent in ICVT, possibly because intracranial pressure did not increase as it did in CVT. Other frequent symptoms were motor weakness (19 cases, 37.3%), sensory disturbance (11 cases, 21.6%), aphasia (9 cases, 17.6%), and conscious disturbance (7 cases, 13.7%). Seizure, motor weakness, and sensory disturbance were relatively frequent because the veins around the central sulcus were frequently involved in ICVT.

In some previous reports, the exact veins involved were not mentioned. In such cases, we judged from the report pictures. The Trolard, Labbe, precentral, central, postcentral, and sylvian veins were frequently involved. The vein of Trolard, a.k.a. the superior anastomotic vein, is the largest vein anastomosing the superior sagittal sinus and superficial sylvian vein, and the many of involved veins in ICVT might be vein of Trolard. It could be said that anastomotic veins tend to be involved in ICVT. Another point of view is that ICVT involving anastomotic veins tends to be more easily detected because gyri around anastomotic veins give typical and apparent symptoms. Conversely, ICVTs in other areas, i.e., the silent area, might be overlooked because of nonspecific symptoms. More patients with ICVT may exist.

The diagnoses of ICVT were based on detection of the thrombosed vein. Cord sign or dot sign with high-intensity T_1WI signal, low-intensity T_2*SW signal, high-intensity FLAIR signal, and high density in noncontrast CT were typical findings of thrombosed vein and observed in 26 (51.0%), 25 (49.0%), 13 (25.5%), and 7 (13.7%) cases, respectively. T_2*SW was reported as the most useful sequence for detecting involved veins in ICVT[2,25] and overall CVT.[26] Actually, T_2*SW detected involved veins with obvious low-intensity signals with high frequency, especially in recent cases. MRV and conventional angiography, the most definitive examinations in CVT, were also performed in many ICVT cases; some showed filling defects and/or slowing blood flow in involved veins. However, these findings are neither objective nor definitive, and their role in ICVT was either supporting or useless; they are useful for confirming patency of the dural sinuses and deep cerebral veins. Parenchymal

Table 1 Summary of isolated cortical vein thrombosis

Case No.	Author (Year)	Age/Sex	Symptom	Underlying condition	Involved vein (Modality/Sequence detected it)	Parenchymal change (Modality/Sequence)	SAH (Modality/Sequence)	Treatment	mRS (Period from onset)
1	Macchi et al. (1986)[27]	31 F	HA, CD, seizure	ND	Lt posterior parietal vein[a] (CT, T_2)	HI (CT, cCT, T_2)	None	ND	ND
2	Yokota et al. (1990)[28]	56 M	CD, MW, seizure, agraphia	None	Lt vein of Labbe (AG)	HI (CT, T_2)	None	ND	ND
3	Vuillier et al. (1996)[5]	26 F	HA, seizure	Postpartum, Factor V Leiden	Rt vein of Labbe (AG)	HI (CT, T_2)	None	AE, HP, AC	0 (3 m)
4	Jacobs et al. (1996)[11]	38 F	Flu-like symptom, VD, SD, MW, seizure	HLA-B27 (+)	Rt postcentral vein[a] (Surgery)	E/I (T_1, T_2)	None	AE	ND
5		42 F	HA, VD, MW, seizure, aphasia	Post-radiculography, OC	Lt central vein[a] (AG)	HI (CT, T_2)	None	AE, HP, AC, steroid	0 (1 w)
6		33 F	HA, MW, MD, seizure, aphasia	None	Rt central vein[a] (AG)	HI (CT, T_1)	None	AE, HP, AC	0 (2 w)
7		33 M	HA, aphasia	Hodgkin disease	Lt postcentral vein[a] (AG, MRA)	HI (CT, T_2)	None	HP, LMWH	0 (3 w)
8	Derdeyn and Powers (1998)[15]	26 F	MW, seizure	Ulcerative colitis, DVT	Lt sylvian vein (T_1, T_2, PD, AG)	HI (T_1, T_2, PD)	None	AE, WF	1 (ND)
9	Rudolf et al. (1999)[12]	41 F	CD, aphasia, seizure	OC	Lt vein of Labbe (Surgery)	HI (CT)	None	HP, osmodiuretics, surgery, WF	2 (1 m 2 w)
10	Minadeo and Karaman (1999)[22]	54 F	HA, N/V, cough, sputum, seizure, MW	Sinusitis	Rt postcentral vein[a] (T_1)	HI (CT, T_1, T_1Gd)	None	AE, HP, coumadin	0 (2 w)
11	Park et al. (1999)[23]	29 M	HA, VD, flu-like symptom, MD	None	Rt vein of Labbe (AG)	Abnl enhacement (T_1Gd)	None	AE, HP, AC	2 (1 y)
12	Cakmak et al. (2004)[29]	78 M	CD, MW, seizure	ND	Lt sylvian vein[a] (T_2*)	None	None	ND	ND
13		38 F	HA, VD	ND	Rt posterior parietal vein[a] (T_2*)	HI (T_2*)	None	ND	ND
14	Chang and Friedman (2004)[9]	29 F	HA, SD, VD, N/V, MW, seizure	OC	Rt vein of Trolard (T_1, PD, MRV)	None	+ (FLAIR)	AE, HP, WF	0 (ND)
15		46 F	HA, VD	Hypertension	Rt vein of Trolard (T_1, PD, MRV)	None	+ (FLAIR)	AE, HP, WF	0 (ND)
16		64 F	HA, neck pain	Rheumatoid arthritis, sepsis	Rt vein of Trolard (T_1)	None	+ (CT, FLAIR)	None	6 (ND)
17	Duncan and Fourie (2004)[10]	21 F	HA, N/V, MW, SD, seizure	Renal vein thrombosis, OC	Lt central vein[a] (T_1, T_2, FLAIR, AG)	E/I (T_2)	+ (CT, FLAIR)	LMWH, WF	0 (9 m)
18	Urban and Müller-Forell (2005)[30]	64 F	Seizure	Cerebral amyloid angiopathy	Rt precentral vein[a] (T_2*, T_1Gd)	E/I (CT, T_1)	+ (T_2*)	AE	ND
19		72 F	HA, SD, seizure	None	Rt central vein[a] (CT, T_2*, MRA)	E/I (CT)	None	AE, clopidogrel	ND

(*Continued*)

Table 1 (Continued)

Case No.	Author (Year)	Age/Sex	Symptom	Underlying condition	Involved vein (Modality/Sequence detected it)	Parenchymal change (Modality/Sequence)	SAH (Modality/Sequence)	Treatment	mRS (Period from onset)
20	Urban and Müller-Forell (2005)[30]	66 M	Seizure	None	Rt central vein[a] (CT, T_1, PD, AG)	E/I (CT, T_1, PD, T_1Gd)	None	AC	ND
21		72 F	Seizure	None	ND (T_1, FLAIR, T_1Gd)	E/I (FLAIR, T_1Gd)	None	AE, AC	ND
22	Thomas et al. (2005)[24]	23 M	HA, fever, aphasia, agraphia, acalculia	ND	Lt vein of Labbe (T_1, T_2, T_2*, AG)	HI (T_1, T_2, T_2*)	None	ND	ND
23	Rubi and Arjona (2005)[31]	46 M	HA, dysphasia, seizure	ND	Lt temporosylvian vein[a] (CT, T_1, AG)	None	None	ND	ND
24	Wang et al. (2007)[17]	33 F	HA, N/V, SD, seizure	IHS	Lt vein of Trolard (CT, DSA)	None	+ (T_1, FLAIR)	None	0 (6 m)
25	Lai et al. (2007)[16]	45 F	HA, N/V, SD, seizure	IHS	Lt posterior frontal vein[a] (T_1)	E/I (T_2, FLAIR, MRS)	None	HP	0 (1 w)
26	Albayram et al. (2009)[13]	25 F	HA, dizziness, vertigo, seizure, nuchal rigidity	IHS after LP, puerperium	Rt postcentral vein[a] (T_2, MRV)	HI (CT, T_2)	None	HP	0 (6 d)
27	Chakraborty et al. (2008)[8]	28 F	HA, CD	OC	Lt vein of Labbe (T_1, MRV)	E/I (T_2, DWI, ADC, FLAIR)	+ (CT)	ND	ND
28	Rathakrishnan et al. (2008)[4,32]	46 M	Seizure	Temporal AVM	Rt postcentral vein[a] (CT, T_1, T_2*, DWI)	E/I (T_2, DWI, FLAIR)	None	AE	0 (ND)
29	Boukobza et al. (2009)[2]	23 F	HA, seizure	Postpartum	Central vein (T_2*, AG) (T_1 in 5 cases, T_2 in 6 cases, FLAIR in 7 cases, DWI in 3 cases)	E/I (DWI in 5 cases, T_2* in 3 cases)	None	AE, HP, AC	0 (6 d)
30		31 F	HA, MW, seizure	Postpartum, epidural anesthesia	Precentral vein (T_2*)		None	AE, HP, AC	0 (2 m)
31		37 M	HA, ataxia, seizure	Bechet disease, Factor V Leiden	Lt vein of Trolard (T_2*, MRV)		None	AE, HP, AC, steroid	0 (3 m)
32		40 F	HA	Subdermal contraceptive	Small frontal vein (T_2*)		None	HP, AC	0 (1 w)
33		23 F	HA, aphasia	Hyperthyroidism	Vein of Labbe (T_2*, AG)		None	HP, AC	0 (3 m)
34		28 F	HA, MW, seizure	OC	Midfrontal vein (T_2*)		None	AE, HP, AC	0 (1 m 2 w)
35		57 M	HA, aphasia, MW	Meningitis	Lt vein of Labbe (T_2*, AG)	HI (T_1, T_2*)	None	AE, HP, AC, steroid	2 (1 y)
36		46 F	HA, aphasia, MW, CD	OC, IHS	Lt vein of Trolard (T_2*, MRV, AG)	HI (T_1, T_2*)	None	AE, HP, AC, steroid	2 (1 y)
37	Bittencourt et al. (2009)[7]	31 F	HA	IHS after LP, OC	Bil anterior parietal vein	E/I (FLAIR)	+ (FLAIR)	AC	0 (ND)
38	Sharma and Teoh (2009)[33]	38 M	HA, seizure	None	Rt central vein[a] (T_1)	HI (T_1, T_2, T_2*, DWI)	None	AE, HP, coumadin	0 (3 d)
39	Thamburaj and Choudhary (2009)[14]	14 F	HA, SD	Leukemia	Rt postcentral vein[a] (T_1, DWI)	HI (T_1, DWI)	None	Antiedema	0 (ND)

(*Continued*)

Table 1 (Continued)

Case No.	Author (Year)	Age/Sex	Symptom	Underlying condition	Involved vein (Modality/Sequence detected it)	Parenchymal change (Modality/Sequence)	SAH (Modality/Sequence)	Treatment	mRS (Period from onset)
40	Morris et al. (2010)[34]	75 F	HA, SD, seizure	Hypertension	Rt central vein[a] (CT, FLAIR, T_2*, AG)	E/I (ADC)	None	(AE, aspirin), HP, WF	0 (3 m)
41	Yildiz et al. (2010)[6]	23 M	SD, MW, seizure	IHS after LP, 4G/4G genotype of PAI-1	Bil postcentral vein[a] (T_1, T_2*)	HI (T_2*)	None	HP, WF	0 (ND)
42		24 F	SD, MW, seizure	IHS after LP, Postpartum	Rt central vein, precentral vein[a] (T_2*)	HI (T_2*)	None	AE, HP, WF	0 (ND)
43		34 M	SD, MW	IHS after LP, MTHFR mt.	Lt postcentral vein[a] (T_1Gd)	HI (T_2*, T_1Gd?)	None	HP, WF	0 (ND)
44	Linn et al. (2010)[35]	48 M	HA, MW, fluctuating vigilance	ND	Lt frontal, temporal, parietal veins (T_2*, MRV)	HI (ND)	None	ND	ND
45		60 F	MW, reduced vigilance, seizure	ND	Lt parietal veins (T_1, T_2*, FLAIR, MRV)	None	None	ND	ND
46		29 F	CD, MW, seizure	ND	Bil frontal parietal veins (T_1, T_2*, FLAIR)	HI (ND)	None	ND	ND
47	Rathakrishnan et al. (2011)[4]	54 M	Seizure	None	Rt central vein[a] (T_1, T_2*, MRV)	E/I (T_2)	None	AE, LWNH, clopidogrel	0 (2 y)
48		25 F	SD, seizure	Postpartum, protein S ↓	Lt postcentral vein[a] (T_1, T_2*)	E/I (T_2)	None	AE, HP, WF	0 (1 y)
49		33 M	Seizure	Protein C ↓	Rt postcentral vein[a] (T_1, T_2*, MRV)	E/I (T_2)	None	AE, LMWH, WF	0 (9 m)
50		45 M	Seizure	Protein S ↓	Rt postcentral vein[a] (CT, T_2*, MRV)	E/I (T_2)	None	AE, HP, WF	0 (2 y 4 m)
51	Present case	14 F	HA, dizziness	None	Lt sylvian vein (FLAIR, T_2*)	None	None	None	1 (2 w)

Abnl: abnormal, AC: anticoagulant, ADC: apparent diffusion coefficient, AE: antiepileptic, AG: conventional angiography, AVM: arteriovenous malformation, Bil: bilateral, cCT: contrast computed tomography, CD: conscious disturbance, CT: non-contrast computed tomography, d: day, DVT: deep venous thrombosis, DWI: diffusion weighted imaging, E/I: edema/infarction, F: female, FLAIR: fluid attenuated inversion recovery, HA: headache, HI: hemorrhagic infarction, HP: heparin, IHS: intracranial hypotension syndrome, LMWH: low molecular weight heparin, LP: lumber puncture, Lt: left, m: month, M: male, MD: memory disturbance, MRA: magnetic resonance angiography, mRS: modified Rankin Scale, MRV: magnetic resonance venography, MTHFR mt: methylenetetrahydrofolate reductase mutation, MW: motor weakness, ND: no data, Nl: normal, N/V: nausea/vomiting, OC: oral contraceptive, PAI-1: plasminogen activator inhibitor, PD: proton density-weighted imaging, Rt: right, SAH: subarachnoid hemorrhage, SD: sensory disturbance, T_1: T_1-weighted imaging, T_1Gd: T_1-weighted imaging with gadolinium enhancement, T_2: T_2-weighted imaging, T_2*: echo-planar T_2* susceptibility-weighted imaging, VD: visual disturbance, w: week, WF: warfarin, y: year. a: We judged from the report pictures.

change was observed in 43 ICVT cases (84.3%) and was high compared with that in CVT (63%).[3] This finding was not thought to mean that parenchymal change in ICVT was more frequent than in CVT but that most ICVT without parenchymal change was overlooked because of the relatively mild symptoms. Hemorrhagic infarction was observed in 21 cases (41.1%), and edema or infarction alone was observed in 21 cases (41.1%). These changes were detected by noncontrast CT, T_1WI, T_2WI, FLAIR, and T_2*SW. For detecting parenchymal changes, T_2*SW was often useful. Subarachnoid hemorrhage (SAH) was observed in 8 cases (15.7%), which was mainly detected by FLAIR. However, the distinction between SAH and thrombosed vein was often difficult and might have been confused in some cases. Reviewing literature revealed that exceedingly long cord sign in our case was extremely rare. Considering relatively mild symptom and absence of parenchymal change in our case, severity of ICVT may not relate to the length of involved vein.

There were 11 cases in which cerebrospinal fluid was studied; 6 cases reported nonspecific abnormalities including increased white blood cells in 3 cases[11,22] and increased protein in 2 cases.[11]

Electroencephalograms were obtained in 17 cases; 11 (64.7%) were normal, and the other had nonspecific abnormal waves on the involved site.[5,11,23,30]

Therapeutic strategies were mentioned in 40 previous cases. Although 33 cases received anticoagulants (82.5%), their outcomes were not different from those of patients without anticoagulants. Surgery was performed in 2 cases; 1 was exploratory craniotomy[11] and the other was cerebral decompression for brain swelling with uncal herniation.[12]

The outcomes of ICVT were generally favorable. Thirty-six reported cases described outcomes and 29 (80.6%) completely recovered. Mostly, symptoms were resolved in a few days or weeks. Overall, patients with good outcomes, apparently corresponding to modified Rankin Scale (mRS) scores of 0–2, accounted for 97.2% of the cases, which is higher than that of overall CVT (87.2%).[36] There were no recurrent cases, but 1 case of ICVT progressed to dural sinus thrombosis. Only 1 patient died from other underlying disease.[9] Therefore, mortality as a consequence of ICVT was 0%; the overall mortality was 2.8%, which was very low compared with that of CVT (mean overall mortality, 9.4%).[36] Abnormal imaging findings in many cases were reversible. In 25 cases with information on follow-up imaging, the abnormal findings in the initial study vanished in 16 cases (64.0%); improvement was observed in the remainder. The fact that abnormal findings are temporary may make diagnosis problematic.

In conclusion, we presented a case of isolated superficial sylvian vein thrombosis with long cord sign. The literature suggested that ICVTs of the silent area might have been overlooked because of nonspecific symptoms, and more patients with ICVT may exist. In cases involving patients with nonspecific symptoms, such as in our case, the possibility of ICVT should be considered.

Conflicts of Interest Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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