Case Report

Dural Sinus Thrombosis with Nonsymptomatic Persistent Falcine Sinus: A Case Report

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A 24-year-old woman was admitted to our hospital after convulsive status epilepticus. A cerebral magnetic resonance venography revealed a persistent fetal falcine sinus. Additionally, the posterior third of the superior sagittal sinus was hypoplastic and the abnormal deep venous drainage was accompanied. These abnormalities had already been detected by magnetic resonance imaging several years ago. In the present scan, we discovered a sinus thrombosis in the hypoplastic superior sagittal sinus. In the cerebral angiography, we observed delayed venous return in the left parieto-occipital lobe and hypothesized that cerebral venous stasis due to the thrombus caused the convulsive status epilepticus. The patient was treated with intravenous administration of heparin along with an antiepileptic drug, and she recovered with no neurological defects. In the present case, the falcine sinus and the anomalous venous return were likely congenital while the status epilepticus was derived from thrombosis in the hypoplastic superior sagittal sinus. Although the falcine sinus functioned as an alternative pathway for the superior sagittal sinus, the hypoplastic superior sagittal sinus itself may also play an important role as a venous drainage channel.

Key Words: Dural sinus thrombosis—falcine sinus—fetal circulation—superior sagittal sinus

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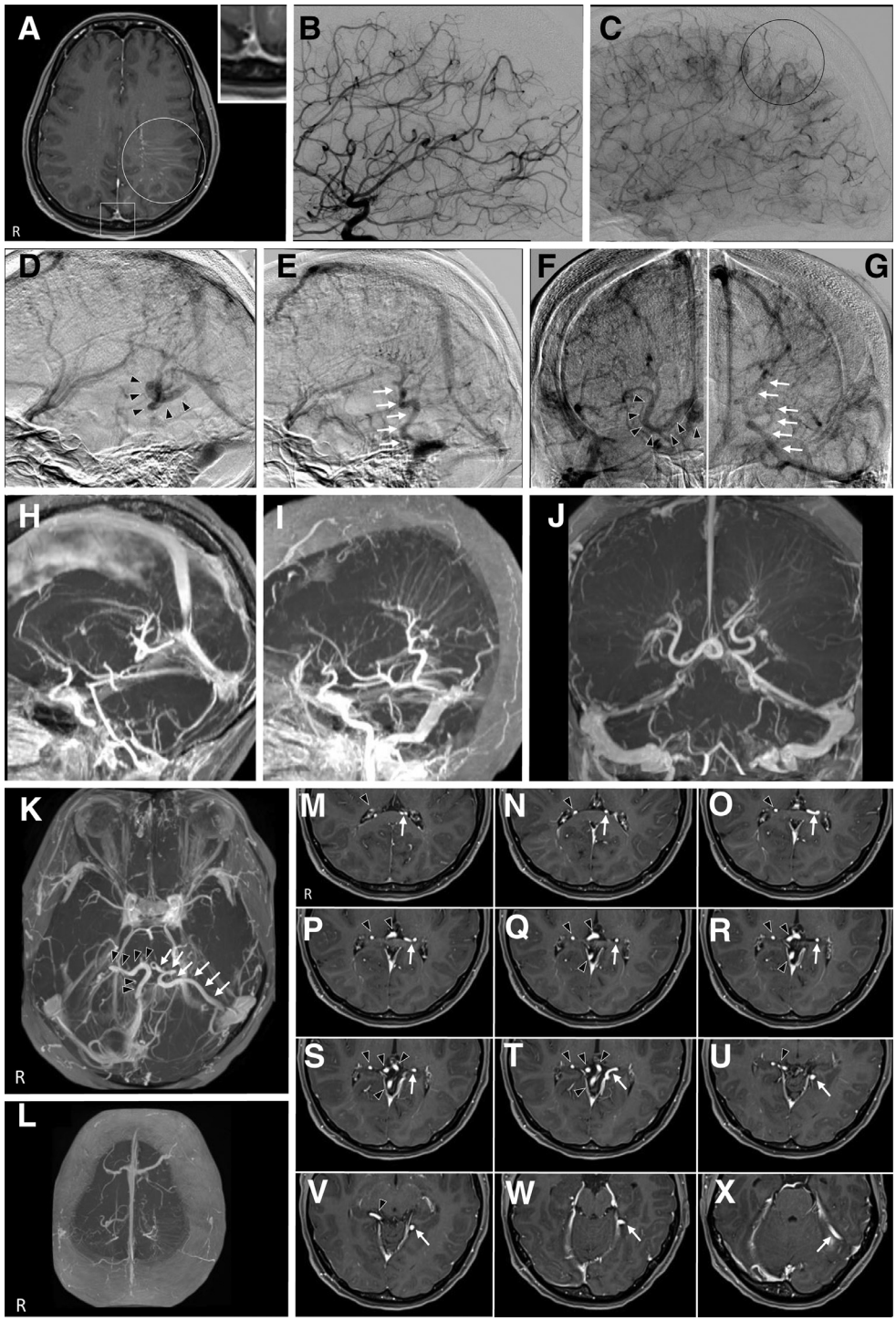

Figure 1. *A, M-X: Enhanced T1-weighted MRI images. B-G: Internal carotid angiograms. H-L: MIP images of enhanced T1 weighted images. Dural sinus thrombosis is present in the superior sagittal sinus (A; enlargement in right square) and the medullary vein is dilated in the left parieto-occipital lobe (A; circled). Internal carotid angiography does not identify the cavernous malformation and dural arteriovenous fistula (B). Delayed venous return is seen in the left parieto-occipital lobe (C; circled). Anomalous venous drainage is indicated by arrowheads (right hemisphere) and arrows (left hemisphere). Both hemispheres have anomalous venous drainage (right hemisphere, D and F; left hemisphere, E and G). H-J show MIP images associated with the carotid angiograms D-G (H corresponds to D; I corresponds to E; J corresponds to F and G). K corresponds to M-X. In the left hemisphere (E, G, I, J, and K), the medullary veins converge and receive venous flow from the lateral ventricle. Finally, venous return drains into the left transvers sinus. In the early fetal period, venous flow from the choroid plexus drains into the diencephalic vein, which flows into the transverse sinus directly. However, in adulthood, the medullary vein drains into the internal cerebral vein normally. It is thought that the diencephalic vein remains, as an alternative pathway in the left hemisphere, because deep venous flow is returned to the transverse sinus. In the right hemisphere (D, F, H, J, and K), venous flow drains into the adjacent hypoplastic straight sinus. The deep venous drainage is developed behind the brain, but the cortical vein is developed in front of the brain (L). It is suggested that the deep venous drainage develops to compensate for hypoplasia of the superficial vein.*

Case Report

A 24-year-old woman presented at the emergency room with convulsive status epilepticus. Routine laboratory tests revealed that her D-dimer levels were elevated to 7.8 μg/mL (normal range <1.0). Maximum intensity projection images of enhanced T1 weighted images revealed that the posterior part of the superior sagittal sinus (SSS) and the vein of Galen were hypoplastic (Fig 1, H, K) and persistent falcine sinus was observed with anomalous venous drainage (Fig 1H-K). These abnormalities were detected by magnetic resonance image several years ago. We also observed a dural sinus thrombosis in the posterior part of SSS (Fig 1A). A cerebral angiography revealed delayed venous return in the left parieto-occipital lobe (Fig 1C, circle).

Intravenous administration of heparin, followed by warfarin, and the antiepileptic drugs were initiated and the patient was fully recovered. Magnetic resonance image and the cerebral angiography performed after recovery showed that the thrombus remained but increased collateral circulation from the SSS successfully shortened the venous return time.

Discussion

In our case, venous drainage was mainly abnormally achieved by the persistent falcine sinus but not by the SSS. The falcine sinus is a normal embryonic vascular channel that rarely persists after birth.[1] The mesencephalic flexure gives rise to both the falcine sinus and the straight sinus,[2] and the both sinuses are intimately connected with one another.[3] Either the absent or rudimentary straight sinus[4] or the hypoplasia of the posterior third of the SSS could contribute to persistence of the falcine sinus.[3]

An open falcine sinus, encountered after birth, may be congenital or acquired.[5] Congenital persistent falcine sinus is believed to be caused by a mesenchymal disorder, commonly accompanied by vein of Galen malformation.[6] Acquired conditions such as obstruction of the SSS by a mass lesion may lead to recanalization of the falcine sinus.[7]

In our case, the persistent falcine sinus may be congenital as it was detected before the admission event. Two cases of cerebral venous sinus thrombosis with a congenital persistent falcine sinus[1,8] have previously been reported. To our knowledge, ours is the first report of thrombosis in the hypoplastic SSS with congenital persistent falcine sinus. Newly developed thrombosis in the SSS, which had been the unusual alternative pathway of the falcine sinus, resulted in the status epilepticus. These indicate that the falcine sinus functioned as an alternative pathway for the SSS, and that the hypoplastic SSS was also essential for sufficient venous drainage.

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