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Superior Sagittal Sinus Thrombosis and Transient Ischemic Attacks : Possible Mechanism

Clinical manifestations of superior sagittal sinus (SSS) thrombosis are nonspecific but characterized by headache, papilledema, seizures, focal deficits, progressive coma and death. Recurrent transient focal neurologic deficit is an extremely rare manifestation in superior sagittal sinus thrombosis and the mechanism is unknown. A 45-year-old man presented with headache for two weeks and four episodes of transient (5-10 minutes) right or left hemiparesis for two days. Magnetic resonance image and transfemoral cerebral angiography revealed superior sagittal sinus thrombosis with numerous prominent collateral venous channels. There was no parenchymal lesion. After four days of heparinization, no further transient focal neurologic deficits developed. Follow-up angiography showed partial recanalization of the SSS. Possible mechanism of transient ischemic attacks in this patient is thought to be a transient functional disturbance due to a temporal reduction of tissue perfusion in the process of operating fully-enough collateral channels.

Key Words : *Sinus thrombosis, superior sagittal sinus; Ischemic attack, transient*

Dae Il Chang, Sang Soo Yoon, Kyung Cheon Chung

Department of Neurology, College of Medicine, KyungHee University, Seoul, Korea

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Address for correspondence
Dae Il Chang, M.D.
Department of Neurology, KyungHee University Hospital, Hoegi-dong, Dongdaemoon-gu, Seoul 130-702, Korea
Tel : +82.2-958-8497, Fax : +82.2-958-8490
E-mail : dichang@khmc.or.kr

INTRODUCTION

Transient ischemic attack (TIA) is extremely rare as a manifestation of superior sagittal sinus (SSS) thrombosis. Only two patients (1), to our knowledge, were identified from a Med-Line computer search from 1966 through 1996. Both patients had partial occlusion of the SSS manifestated by unilateral repeated transient deficits (dysphagia and/or paresthesia which could be interpreted as either TIA or seizures). In our patient, the SSS was completely occluded and transient right- or left-sided hemiparesis was noted alternately. We present a possible mechanism of transient focal neurologic deficit in this patient with the SSS thrombosis.

CASE REPORT

A 45-year-old man was admitted with recurrent transient hemiparesis. The episodes, which were three right-sided weaknesses and one left-sided weakness alternately, lasted about five to ten minutes. The attacks developed suddenly, and the paralysis of the arm and leg was noted as flaccid. He remained conscious without seizure activity during these episodes. He had no past medical histories of infection, trauma, heart disease, malignancy, dehydration, orogenital ulcer, and drugs, but had been treated with diet for diabetes. He had a severe headache for two weeks before admission. Brain computed tomography

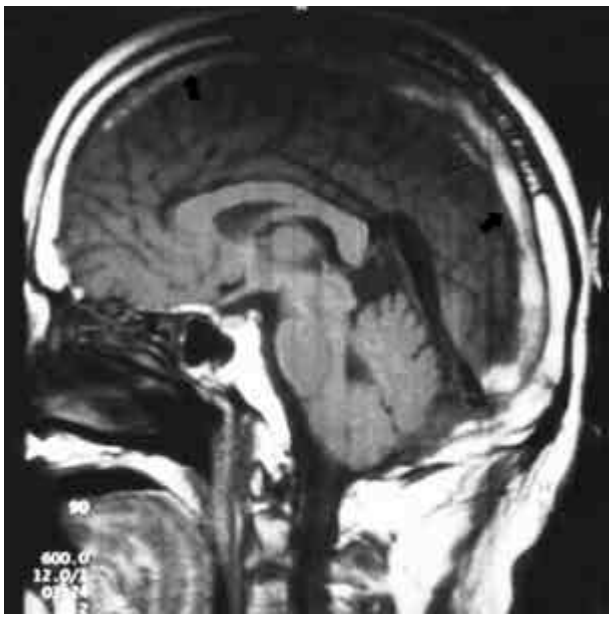

Fig. 1. Sagittal precontrast T-1 weighted MRI shows the clot within the SSS (arrow) and a thrombosed cortical vein (open arrow).

and cerebrospinal fluid, performed at a local clinic, were normal. Neurologic examination on admission was normal. Blood pressure was 160/90 mmHg, body temperature was 37℃. The initial complete blood cell count, electrolyte profile, chest X-ray and electrocardiogram were normal. On the second admission day, he was noted to have flaccid paralysis of the right face, arm, and leg and hypesthesia of the right face for about five minutes. On the third admission day, brain magnetic resonance imaging (MRI) showed thrombosis of the SSS and cortical vein without parenchymal lesion (Fig. 1). Transfemoral cerebral angiography revealed complete occlusion of the SSS with numerous prominent collateral venous channels (Fig. 2A, B). Jugular vein Doppler sonography was unremarkable. All extensive laboratory investigations including erythrocyte sedimentation rate, fibrinogen, prothrombin time, partial thromboplastin time, antithrombin III, protein C, protein S, antiphospholipid antibody, protein electrophoresis, rheumatoid factor, antinuclear antibody, liver function tests, renal function tests, and thyroid function tests were normal. On the fifth day after admission, he was noted as having flaccid paralysis of the left arm and leg for about ten hours. Intravenous heparin (1,000 U/h) was initiated on the fourth day of admission for three weeks and oral coumadin thereafter for six months. From fourth day after heparinization, there was no further focal neurologic deficits. Headache subsided two weeks after heparinization. Three weeks after admis-

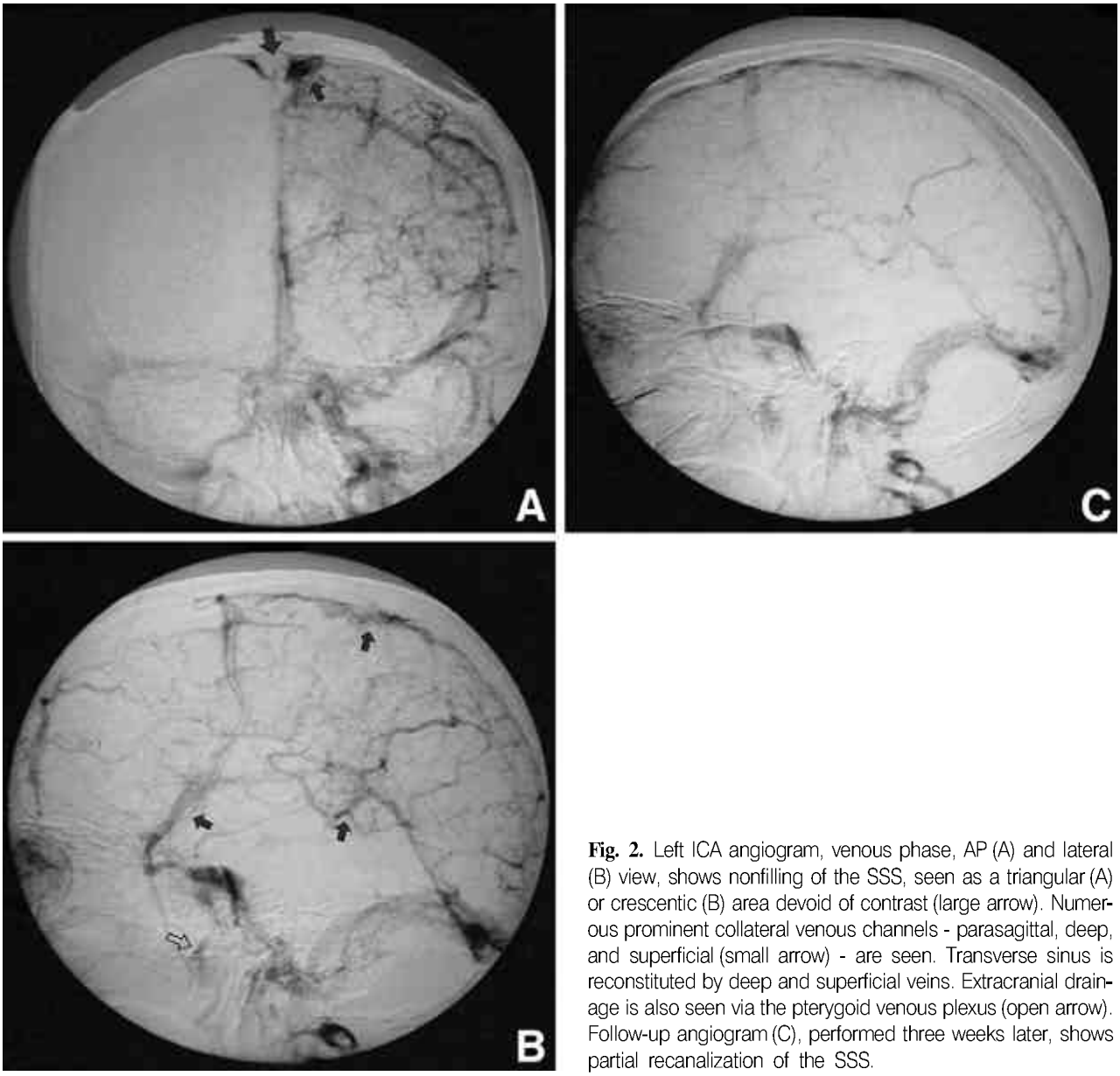

Fig. 2. Left ICA angiogram, venous phase, AP (A) and lateral (B) view, shows nonfilling of the SSS, seen as a triangular (A) or crescentic (B) area devoid of contrast (large arrow). Numerous prominent collateral venous channels - parasagittal, deep, and superficial (small arrow) - are seen. Transverse sinus is reconstituted by deep and superficial veins. Extracranial drainage is also seen via the pterygoid venous plexus (open arrow). Follow-up angiogram (C), performed three weeks later, shows partial recanalization of the SSS.

sion, follow-up angiography showed partial recanalization of the SSS (Fig. 2C). There was no recurrence of episodes in the 12 months of follow-up.

DISCUSSION

Although the causes of TIA in cerebral arterial disease are known to be due to thromboembolism or low flow (2), the pathogenesis of TIA in venous sinus thrombosis has not yet been fully understood.

In this patient, TIAs are due to the SSS thrombosis because; 1) occlusion of the SSS was confirmed by angiography. 2) neurologic deficits were manifested on the right- or left-side, alternately. 3) transient focal neurologic deficits did not recur after anticoagulation. Since there is no parenchymal lesion, the cause of focal neurologic deficit in our patient is thought to be not a structural change of parenchyme but rather a functional and/or metabolic disturbance.

In normal circumstances, the superficial cerebral veins chiefly drain into the SSS and transverse sinus via the anastomotic veins of Trolard and Labbe. In cases of complete SSS thrombosis, the collateral flow proceeds through the sphenoparietal sinus, cavernous sinus, and basal vein of Rosenthal (3) as seen in this patient.

Although collateral pathways in venous sinus disease are generally believed to be well-established because of the slow growth of venous thrombus (4), a discrepant moment of venous drainage can occur before opening of fully-enough collateral channels. Flow dynamic forces may collide at the level of capacitance vessels, and then tissue perfusion pressure may be reduced, which results in neurologic deficits. When the pressure of pre-capacitance vessels far exceeds that of the capacitance vessels, pre-existing venous channels must be operated as collateral drainage pathways. This results in immediate opening of the collateral channels, normalization of low tissue perfusion pressure, and recovery of brain dysfunctions. Transient neurologic deficit, therefore, may be due to a temporal reduction of tissue perfusion amid a maelstrom of the formation of fully-enough collateral channels.

Another possible mechanism of TIA in SSS thrombosis is the seizure phenomenon (4). The symptomatology in this patient, however, is not compatible with this.

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