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Cerebral Venous Thrombosis Associated with Maxillary and Ethmoid Sinusitis
—A Case Report—

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We herein report a young patient with cerebral venous thrombosis(CVT) with clinical and neuroradiological findings of the left maxillary and anterior ethmoid sinusitis. Serial brain MRIs showed cerebral venous infarct and thrombosis in the superior sagittal sinus(SSS). MR angiography demonstrated nonvisualization of SSS and bilateral transverse sinus. According to our knowledge, CVT associated with maxillary and ethmoid sinusitis has been reported very rarely. High index of suspicion and neuroimaging studies, especially brain MRI, and conventional or MR angiography are very important for the early diagnosis of CVT.

Key Words : *Cerebral venous thrombosis, Maxillary sinusitis, MRI, MR angiography*

INTRODUCTION

Cerebral venous thrombosis(CVT) is a potentially fatal disorder secondary to many underlying etiologies(Bousser et al., 1985 ; Gates, 1986). Because of its frequently misleading presentation, its unpredictable course, and certain treatment problems, CVT remains a challenge for the clinician. Paranasal sinusitis, especially sphenoid sinusitis(Lew et al., 1983 ; Sofferman, 1983 ; Kibblewhite et al., 1988 ; Goldman et al., 1993), is rarely reported to cause CVT. However, according to our knowledge, there is few, if ever, reportsof CVT associated with maxillary and ethmoid sinusitis.

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CASE REPORT

A 32-year-old man with generalized tonic seizure was taken to the emergency room. He had enjoyed good health until six months prior to admission, when he suffered from purulent nasal discharge with foul odor and intermittent nasal stuffiness in the left nose. However he had not undergone any proper management. He denied any past medical history of orogenital ulcer, skull fracture, otitis, or dental infection. He was neither alcoholic nor diabetic.

Two days before admission, he felt a persistent bilateral throbbing headache in the frontal area with copious purulent rhinorrhea. In the morning on the admission day, during sleep, he developed generalized tonic seizure lasting about three minutes. There was postictal confusion, severe headache and irritability for about two hours. The MRI(1.5 Tesla unit) of the brain revealed severe inflammatory reaction in the left maxillary sinus(Fig. 1A) and mild enhancement in the left anterior ethmoid sinus(Fig. 1B). In addition, there was a high signal lesion in the right frontal cortical

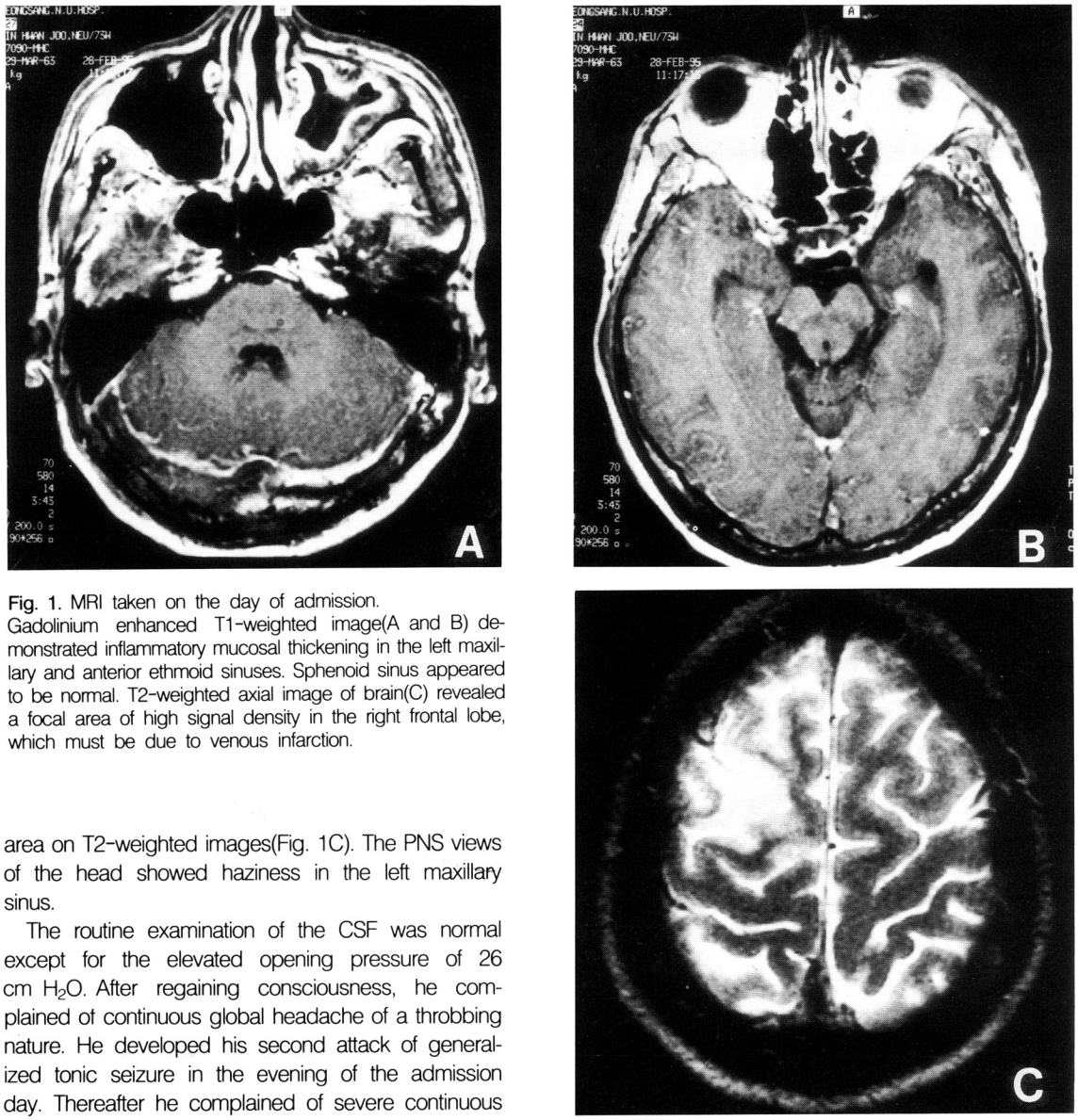
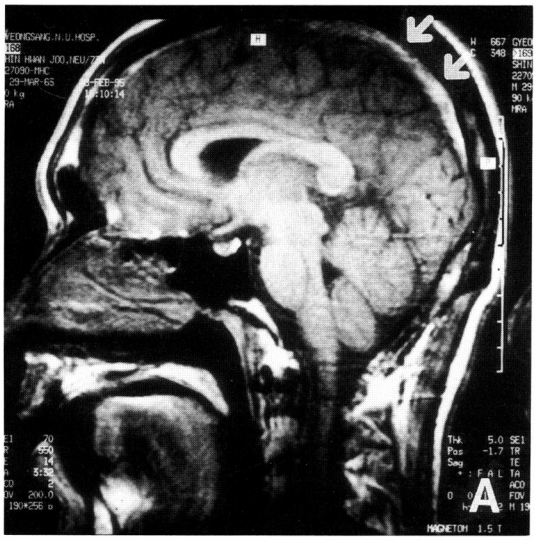


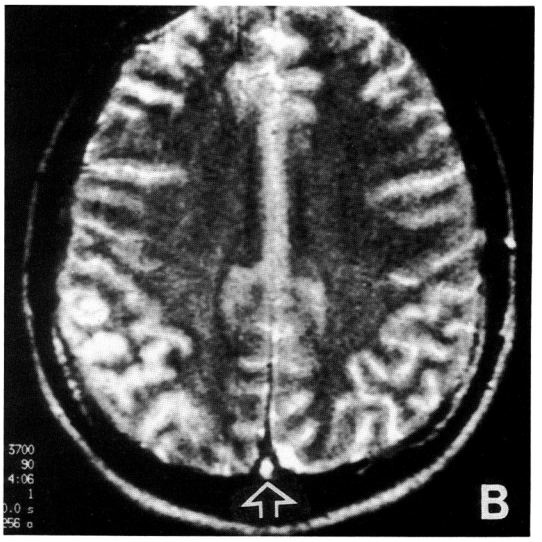
Fig. 1. MRI taken on the day of admission.
Gadolinium enhanced T1-weighted image(A and B) demonstrated inflammatory mucosal thickening in the left maxillary and anterior ethmoid sinuses. Sphenoid sinus appeared to be normal. T2-weighted axial image of brain(C) revealed a focal area of high signal density in the right frontal lobe, which must be due to venous infarction.

area on T2-weighted images(Fig. 1C). The PNS views of the head showed haziness in the left maxillary sinus.

The routine examination of the CSF was normal except for the elevated opening pressure of 26 cm H_2O. After regaining consciousness, he complained of continuous global headache of a throbbing nature. He developed his second attack of generalized tonic seizure in the evening of the admission day. Thereafter he complained of severe continuous global headache more throbbing in the frontal area. His headache disturbed his sleep and was aggravated by sitting and standing. However there was no nausea, vomiting, photophobia or visual disturbance. Neurological examination showed no abnormality. Opioids and nonsteroidal anti-inflammatory drugs were administered with only modest effect.

His headache continued day and night and worsened day after day for several days on admission. There were persistent leukocytosis (11,900 to 13,900/mm^3) and elevated sedimentation rates (17 to 26 mm/hr, Westergren method). Coagulation study was normal except for mildly elevated fibrinogen level(285 mg/dl). Viral antibodies(HSV, EBV, CMV), polymerase chain reaction(PCR) for *M. tuberculosis*, bacterial/mycobacterial/fungal smears and cultures showed negative results in the CSF. On the fifth day, the second CSF study revealed normal opening pressure, pleocytosis including both red(178/mm^3) and white(8/mm^3) cells, elevated protein(290 mg/dl),





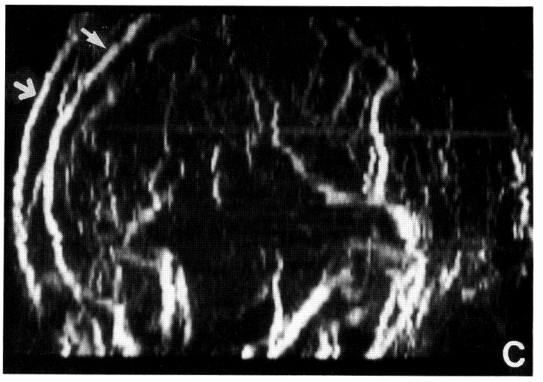


Fig. 2. MRI taken on the sixth day of admission.
High signal intensity in the superior sagittal sinus(SSS) was noted on T1-weighted sagittal(A) and T2-weighted axial image(B). These findings were consistent with dural sinus thrombosis. Lateral projection of MR angiography(C) demonstrated prominent collateral veins (arrows) without visualization of SSS.

and normal glucose level. Microbiological studies of the CSF showed negative findings.

On the sixth day, he became stuporous and then semicomatose accompanied by recurrent generalized tonic clonic seizures and fever over 38℃. The seizures were controlled by intravenous diazepam and phenytoin. Follow-up brain MRI showed high signal intensities in the superior sagittal sinus(SSS, Fig. 2A, B). MR angiography using time-of-flight technique demonstrated nonvisualization of SSS and bilateral transverse venous sinuses(Fig. 2C). Intravenous heparinization was administered without clinical effect. Follow-up CT on the seventh day revealed hemorrhagic infarct and empty delta sign. Conventional angiography was not done.

Thereafter he never recovered his consciousness. On the eighth day, a right parietotemporal lobectomy was done to prevent herniation due to large hemorrhagic infarct and surrounding edema. However postoperative neurological condition did not improve. He passed away on the eleventh day after admission due to cardiopulmonary failure.

DISCUSSION

Our patient showed rapidly progressing CVT with early cerebral venous infarct(Fig. 1C) and later hemorrhagic infarction. Diagnostic evaluations for underlying etiology found no significant finding except for the remarkable left maxillary sinusitis and mild left anterior ethmoid sinusitis. Prior to developing seizure, he had had recently exacerbated purulent nasal discharge and headache which highly suggested acute sinusitis. Although autopsy could not be done, pre-mortem diagnosis of CVT was certain in our patient on the basis of the clinical manifestations and courses, CSF findings, and the very characteristic findings of brain CT, MRI, and MR angiography. Persistent leukocytosis, elevated sedimentation rates in the early phase and high fever in the later phase strongly supported

the infectious origin of CVT.

The most common form of septic CVT is cavernous sinus thrombosis usually following an infection of the middle third of the face(Bousser and Barnett, 1993). Other causes of CVT include sphenoid and ethmoid sinusitis, otitis media, dental abscess, HIV and CMV infections(Bousser and Barnett, 1993). We could exclude other infectious causes, except for sinusitis, of CVT in our patient. Noninfectious medical causes of CVT are numerous. Among these, malignancies, inflammatory diseases such as Behcet's disease, and connective tissue diseases are the most frequent-(Bousser and Barnett, 1993).

Sinusitis is not rare among populations. However we do not think the sinusitis and CVT in our patient are coincidental. Initial clinical presentations in our patient were bilateral frontal headache with copious purulent nasal discharge. These highly suggested that the headache was secondary to acute sinusitis. Persistent leukocytosis and elevated sedimentation rates supported acute infection. The negative microbiologic studies of the CSF excluded intracranial infectious source. The seizures which developed two days after headache and rhinorrhea highly suggests cerebral cortical venous infarction.

Among paranasal sinuses, sphenoid sinus infection is occasionally reported to cause CVT(Lew et al., 1983 ; Sofferman, 1983). Because of the close proximity to the cortical venous system, infection from the sphenoid sinus may spread to the intracranial venous system(Lew et al., 1983 ; Sofferman, 1983). In addition, the sphenoid walls can be extremely thin, and sometimes the sinus cavity is separated from the adjacent structure by just a thin mucosal barrier(Silverstein, 1994). However there is no feasible explanation of intracranial spread of ethmoid or maxillary sinusitis yet. We suppose radiologically unidentifiable acute sphenoid sinusitis secondary to maxillary and ethmoid sinusitis might be related with CVT in our patient. Similar case with cavernous sinus thrombosis associated with maxillary and ethmoid sinusitis suggests that cavernous sinus may be another possible route of infection in our patient(Assefa et al., 1994). Spread of infection from the sinus proceeds mainly through the afferent veins to the unvalved cavernous sinus(Assefa et al., 1994). However the exact route of spread from sinusitis into the intracranial venous system in our patient remains obscure. Further study supported by autopsy or biopsy may disclose this route.

MRI offers major advantages for the evaluation of patients suspected of CVT because of its sensitivity to blood flow, its ability to visualize the thrombus itself, and its non-invasiveness(Bousser and Barnett, 1993). Recent MRI study in CVT showed very high sensitivity(96 %) of MRI for the diagnosis of CVT(Dormont et al., 1994). In that study, 50 percents of 53 patients had venous infarction as in our patient. In thrombosed sinuses, three serial types of signal abnormalities were observed : in early stage, isosignal on T1-weighted image and low-intensity signal on T2-weighted image ; in intermediate stage, high-intensity signal on T1- and T2-weighted images ; in late stage, isosignal on T1-weighted image and high-intensity signal on T2-weighted image(Dormont et al., 1994).

Conventional angiography has been the key procedure in the diagnosis of CVT. The partial or complete lack of filling of veins or sinuses is the best angiographic sign of CVT(Bousser and Barnett, 1993). Other angiographic findings include delayed emptying, and collateral venous pathways(Bousser and Barnett, 1993) as in our patient(Fig. 2C). Hitherto only a few cases with CVT were evaluated by MR angiography(Medlock et al., 1992). However MR angiography is an alternative noninvasive means to diagnose and monitor the evolution of CVT and efficacy of therapeutic intervention(Medlock et al., 1992). We suppose that rapidly developing MR angiographic technique with high resolution and less artifact will replace conventional angiography for the diagnosis of CVT in the near future.

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