Cerebral Venous Thrombosis Initially Presenting with Acute Subarachnoid Hemorrhage

Ji-Ho Lin[1]*, Shang-Yeong Kwan[2,3], Dean Wu[4]

[1]*Department of Neurology, Tao-Yuan General Hospital, Department of Health, Executive Yuan,* [2]*Pediatric Epilepsy Surgery Group, Taipei Veterans General Hospital,* [3]*National Yang-Ming University School of Medicine, Taipei, and* [4]*Department of Neurology, Min-Sheng General Hospital, Tao-Yuan, Taiwan, R.O.C.*

We report the case of a 44-year-old man with superior sagittal and transverse sinus thrombi, who initially presented with venous subarachnoid hemorrhage over the right parietal sulci. After 7 days of intravenous anticoagulation therapy, his condition stabilized. Cerebral venous thrombosis can be difficult to diagnose and is further complicated when patients initially present with acute subarachnoid hemorrhage. A high index of clinical suspicion is needed to diagnose venous subarachnoid hemorrhage so that appropriate treatment can be initiated as promptly as possible. [*J Chin Med Assoc* 2006;69(6):282–285]

Key Words: angiography, cerebral venous thrombus, subarachnoid hemorrhage

Introduction

Cerebral venous thrombosis (CVT) can be difficult to diagnose, and it is further complicated when patients initially present with acute subarachnoid hemorrhage (SAH).[1] SAH, in these cases, is probably induced by the rupture of a dilated vein associated with superior sagittal sinus (SSS) thrombosis.[2] We present the case of a male patient with SSS and transverse sinus thrombi whose initial presentation was SAH in an unusual location. After treatment with intravenous anticoagulation therapy, the SSS thrombus resolved. However, deep vein thrombosis developed over both lower legs 2 months later, even with continuing oral anticoagulation therapy.

Case Report

A 44-year-old man was sent to the emergency department due to an episode of focal motor seizure of the left arm for several minutes. He had a history of hypertension and hyperlipidemia with regular treatment for 2 years, polio with associated left leg weakness since childhood, and left leg fracture with s/p open reduction with internal fixation 5 years prior to admission. During the 5 days directly preceding admission, he had suffered from a headache over the right parietal region characterized as a "thunderclap". He had no fever. Neurologic examination showed a normal level of consciousness with focal weakness over the left arm consistent with postictal paralysis. A meningismus was found. The rest of the physical examination was unremarkable. Non-contrast brain computed tomography (CT) revealed SAH over the right parietal sulci (Figure 1A). Due to the unusual location of the SAH, CT with contrast was conducted and showed an empty delta sign of SSS thrombosis (Figure 1B). Angiography confirmed the diagnosis of SSS and transverse sinus thrombi (Figure 2). Brain magnetic resonance imaging (MRI) also confirmed the presence of SAH and sinus thrombi (Figure 3).

Coagulation testing including prothrombin time, activated prothrombin time, anticardiolipin antibody titer, antiphospholipid antibody titer, homocysteine titer, and levels of protein C and S, antithrombin III,

*Correspondence to: Dr. Ji-Ho Lin, Department of Neurology, Tao-Yuan General Hospital, Department of Health, Executive Yuan, 1492, Jhongshan Road, Tao-Yuan 330, Taiwan, R.O.C.
E-mail: epilepsy2000@gmail.com • Received: July 20, 2005 • Accepted: January 25, 2006

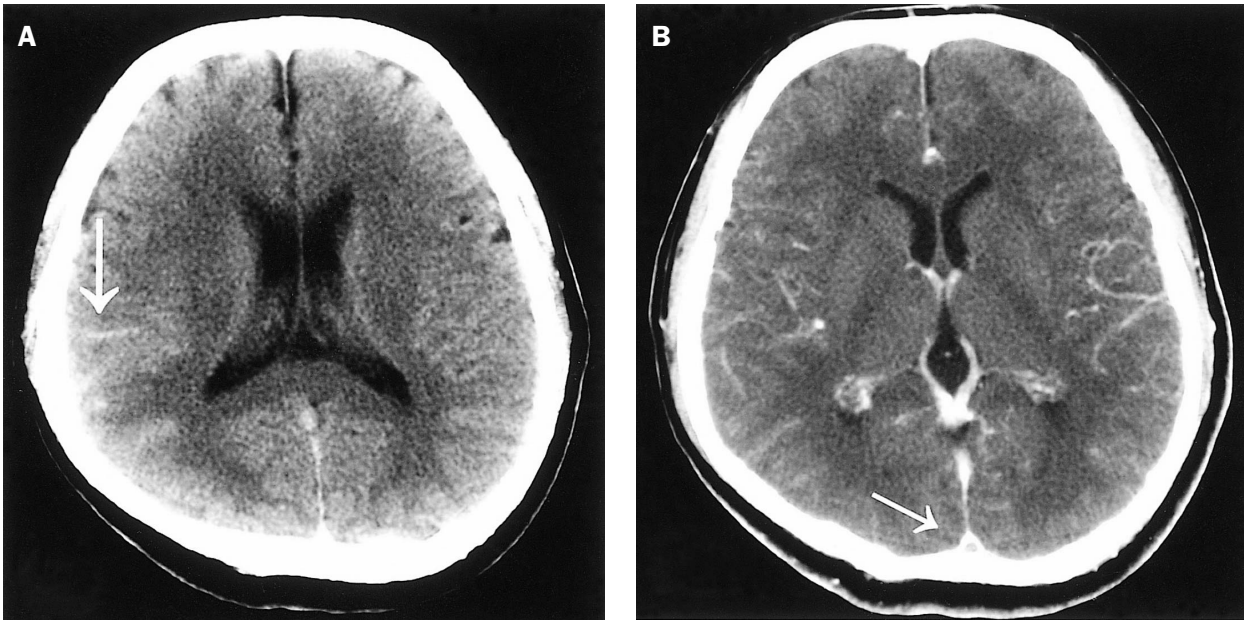

Figure 1. (A) Non-contrast brain computed tomography (CT) showed acute subarachnoid hemorrhage over the right parietal sulci (arrow). (B) Brain CT with contrast revealed an empty delta sign in the superior sagittal sinus thrombus (arrow). Axial contrast-enhanced CT demonstrated a filling defect in the center of the superior sagittal sinus as it approached the confluence of the sinuses.

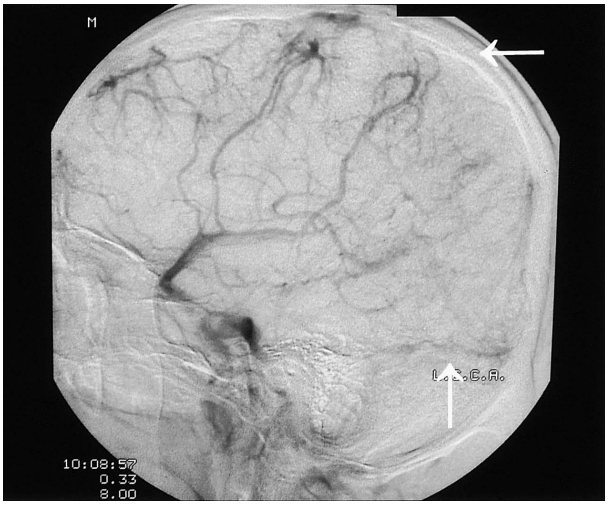

Figure 2. Obliteration of the superior sagittal sinus (horizontal arrow) and left transverse sinus (vertical arrow) with injection into the left common carotid artery.

and fibrinogen were all within normal limits. Intravenous heparin treatment was given under the diagnosis of CVT. Left arm weakness resolved 1 day later. The patient's condition stabilized after 7 days of treatment. Oral warfarin maintained an international normalized ratio (INR) of 2.0–3.0. The patient did well until 2 months after this admission, when he developed deep venous thromboses over both lower legs.

Discussion

The spectrum of clinical presentations of CVT ranges from headache with papilledema to focal deficit, seizures and coma. Up to 75% of cases are characterized by focal neurologic deficit and headache; 30–50% of affected patients present with seizures, often followed by Todd's paresis. Rare but classical clinical pictures are those of SSS thrombosis (4%) with bilateral or alternating deficits and/or seizures.[3]

CVT can be difficult to diagnose because of its wide spectrum of clinical manifestations. Diagnosis may be further complicated when patients initially present with acute SAH.[1] The hemorrhage is probably induced by rupture of a dilated vein associated with SSS thrombosis. The location of SAH from SSS thrombosis is usually different from arterial aneurysms.[2] Angiography remains the diagnostic gold standard.

Management of venous SAH secondary to CVT is also quite different from that of arterial SAH. Treatment of sinus thrombosis with heparin has long been controversial. The benefits of heparin have been demonstrated in a randomized and placebo-controlled trial of 20 patients.[4] In a further placebo-controlled trial, 60 patients were randomized to either low-molecular-weight heparin followed by warfarin, or placebo.[5] The anticoagulated patients had better

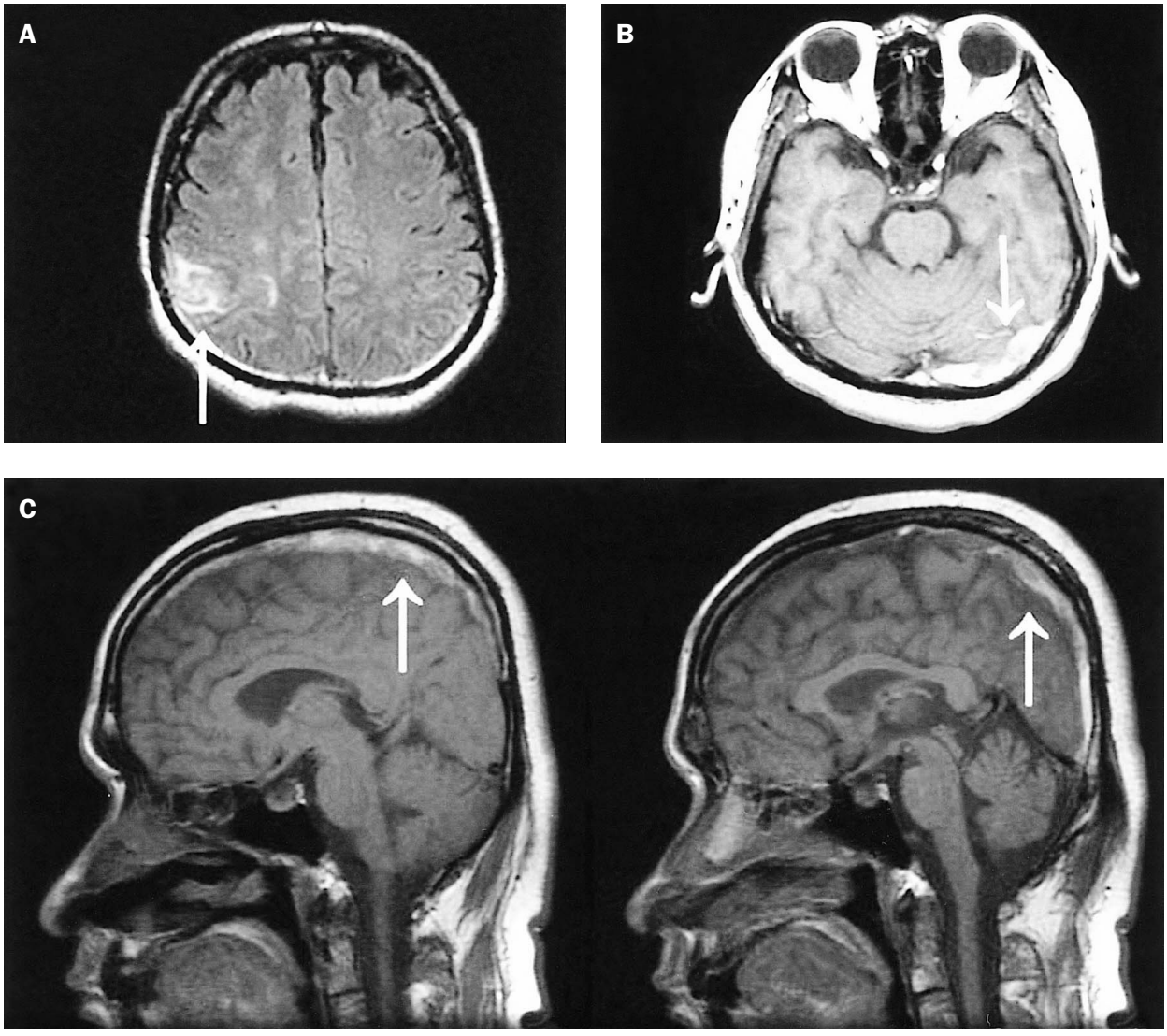

Figure 3. (A) Fluid attenuated inversion recovery brain magnetic resonance imaging (MRI) showed acute subarachnoid hemorrhage over the right parietal sulci (arrow). (B) Left transverse sinus thrombus was shown on T1-weighted MRI with contrast injection (arrow). (C) Superior sagittal sinus thrombus demonstrated on T1-weighted MRI with contrast injection (arrows).

outcomes than the controls, but the difference was not statistically significant. The investigators suggested that anticoagulation was safe, even in patients with cerebral hemorrhage.[5] A subsequent study showed no clear benefit of anticoagulant treatment, but the investigators continued to suggest that there was a non-significant trend in favor of anticoagulant treatment.[6] In our patient, the effect of anticoagulation was good. However, bilateral deep vein thrombosis occurred 2 months later, even under adequate oral anticoagulant therapy with INR at 2.0–3.0. Our patient probably had a hypercoagulation trait, even though the results of available coagulation tests were within normal limits.

In conclusion, we suggest that because of the wide various presentations of venous SAH secondary to CVT, a high index of clinical suspicion is needed to diagnose this uncommon condition.

References

1. Oppenheim C, Domigo V, Gauvrit JY, Lamy C, Mackowiak-Cordoliani MA, Pruvo JP, Meder JF. Subarachnoid hemorrhage as the initial presentation of dural sinus thrombosis. *Am J Neuroradiol* 2005;26:614–7.
2. Sasaki A, Nagaseki Y, Kakizawa T, Sasaki H, Fukamachi A, Nukui H. Superior sagittal sinus thrombosis complicated with multiple aneurysms presenting as subarachnoid hemorrhage. *Neurol Med Chir* 1991;31:978–81.

3. Crawford SC, Digre KB, Palmer CA, Bell DA, Osborn AG. Thrombosis of the deep venous drainage of the brain in adults: analysis of 7 cases with review of the literature. *Arch Neurol* 1995;52:1101–8.
4. Einhaupl KM, Villringer A, Meister W, Mehraein S, Garner C, Pellkofer M, Haberl RL, et al. Heparin treatment in sinus venous thrombosis. *Lancet* 1991;338:597–600.
5. de Bruijn SF, Stam J. Randomized, placebo-controlled trial of anticoagulant treatment with low-molecular-weight heparin for cerebral sinus thrombosis. *Stroke* 1999;30:484–8.
6. de Bruijn SF, Budde M, Teunisse S, de Haan RJ, Stam J. Long-term outcome of cognition and functional health after cerebral venous sinus thrombosis. *Neurology* 2000;54: 1687–9.