

Venous Infarction Secondary to Septic Cavernous Sinus Thrombosis

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Abstract

A 65-year-old woman with poorly controlled diabetes presented bilateral miosis, bilateral abducens nerve palsy, and left hemiparesis. On MRI, cavernous sinus thrombosis, subdural empyema and hemorrhagic infarction in the frontotemporal lobe were detected. Cerebral angiogram revealed filling defect in the cavernous sinus with venous congestion but no involvement of internal carotid artery. Postmortem examination demonstrated hemorrhagic infarction in the right frontotemporal lobe as well as hemorrhagic necrosis of the pituitary gland. It should be noted that venous congestion due to cavernous sinus thrombosis may cause these complications.

Key words: cavernous sinus thrombosis, cerebral veins, brain infarction

(DOI: [10.2169/internalmedicine.45.1430](https://doi.org/10.2169/internalmedicine.45.1430))

Introduction

Cavernous sinus thrombosis is a rare but often critical disease which may not only be caused by infectious but also by noninfectious processes, including vascular, traumatic, and neoplastic etiologies (1, 2). Septic cavernous sinus thrombosis was uniformly fatal in the preantibiotic era. Although it is less common in the postantibiotic era, a delay in the diagnosis results in catastrophe (1, 2). In recent years, CT and MR imaging enables us to accurately diagnose cavernous sinus thrombosis by direct and indirect signs (3, 4). Progress in neuroimaging techniques has contributed to an increased recognition of cavernous sinus thrombosis.

Septic cavernous sinus thrombosis has been reported to be accompanied by intracranial complications such as meningitis, brain abscess, and subdural empyema possibly as a result of spread from the same primary focus infection (1). Cerebral infarction has also been reported as a complication

of septic cavernous sinus thrombosis, probably due to narrowing or occlusion of the internal carotid artery (ICA) at the intracavernous portion (5-7).

We herein report a case with septic cavernous sinus thrombosis complicating hemorrhagic infarction in the frontotemporal lobe and hemorrhagic necrosis of the pituitary gland.

Case Report

A 65-year-old woman was referred to a local medical doctor with slowly progressive headache for three weeks. She had a history of hypertension, diabetes, and hyperlipidemia. She received an analgesic, but the symptom became worse. Moreover, she developed a fever and was treated with an antibiotic. Two days later, she developed stupor with left hemiparesis and was transferred to our hospital.

On physical examination, she had a fever of 40°C and nuchal rigidity. Blood pressure was 146/72 mmHg and pulse

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Received for publication May 20, 2005; Accepted for publication October 13, 2005

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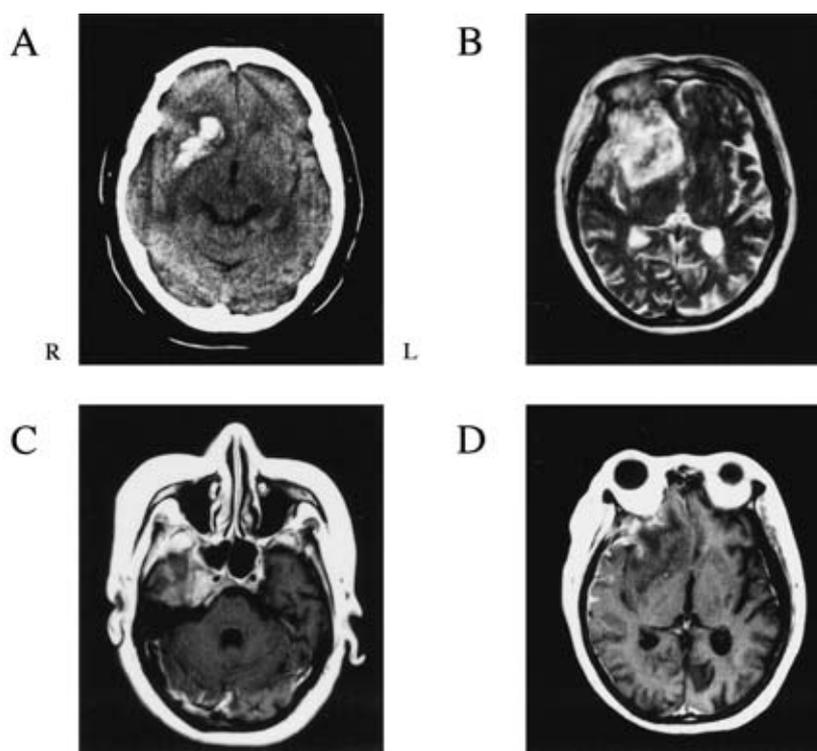


Figure 1. A: Plain CT image demonstrated parenchymal hemorrhage in right frontal lobe. B: T2-weighted axial MR image demonstrated the heterogeneous iso- and hypointensity areas within the high intensity signal in the frontotemporal lobe. C: T1-weighted, contrast enhanced axial MR imaging revealed the abnormal enlargement of the cavernous sinus. D: Subdural empyema was also observed on T1-weighted, contrast enhanced MR imaging.

rate was 70 beats/min. Fundoscopic examination revealed bilateral mild papilledema. Neurological examination showed disturbance of consciousness (Japan coma scale 30) and bilateral miosis (bilateral 2 mm) with minimal pupillary light response. Right corneal reflex was slightly depressed. She had bilateral lateral rectus muscle palsies indicating bilateral abducens nerve palsies, left facial palsy, and left upper and lower limb weakness. The remainder of the physical examination showed no abnormalities. Laboratory data showed leukocytosis of 10400/ μ L (83% neutrophil) and elevated levels of C-reactive protein (37.8 mg/dl) and glycosylated hemoglobin (HbA1c, 12.3%). Chest X-ray showed cardiomegaly but no other abnormalities. No microorganism could be identified in the blood, urine and cerebrospinal fluid. Cerebrospinal fluid contained 210 white blood cells/ mm^3 (58% neutrophil) with a protein concentration of 215 mg/dL and a glucose concentration of 89 mg/dL (27% of a simultaneous blood glucose concentration). Brain CT showed a high density area in the frontotemporal lobe (Fig. 1A). On MRI, T2-weighted image disclosed a heterogeneous hyperintense area in right frontotemporal lobe (Fig. 1B). Gadolinium-enhanced T1-weighted image revealed enlargement of the right cavernous sinus with a heterogeneous hyperintense mass (Fig. 1C) and a hyperintense area in the subdural space (Fig. 1D). Cerebral angiogram did not show any involvement of the internal carotid artery (Fig. 2A). In the venous phase, the cavernous sinus was not

filled with contrast medium (Fig. 2B).

The respiration rhythm was irregular and her respiration was controlled by the respirator. She was treated with a prolonged course of high dose intravenous cefpirome sulfate, as well as drainage of the sphenoid sinus. However, the patient's situation deteriorated progressively and she eventually died. Autopsy demonstrated chronic inflammation of the meninx and cavernous sinus. Sinusitis involving the sphenoid and ethmoid sinuses was observed and considered to be the primary foci associated with septic thrombosis of the cavernous sinus. Hemorrhagic necrosis of the right frontotemporal lobe and pituitary gland were also seen.

Discussion

It has been reported that cavernous sinus thrombosis is complicated by brain infarction (5, 6, 8-11). The cavernous portion of the internal carotid artery is occasionally narrowed or obstructed which results in a diminished flow in the peripheral lesion (5). Spasm or inflammation of the arterial wall induced by arterial invasion of infection, or both, have been implicated for lesions in the intracavernous carotid artery (6). In the present case, the infarction was accompanied by hemorrhagic change and was not identical to the vascular territory without any significant change in ICA. Recent reports showed that venous sinus thrombosis associated with septic cavernous sinus thrombosis is not restricted to

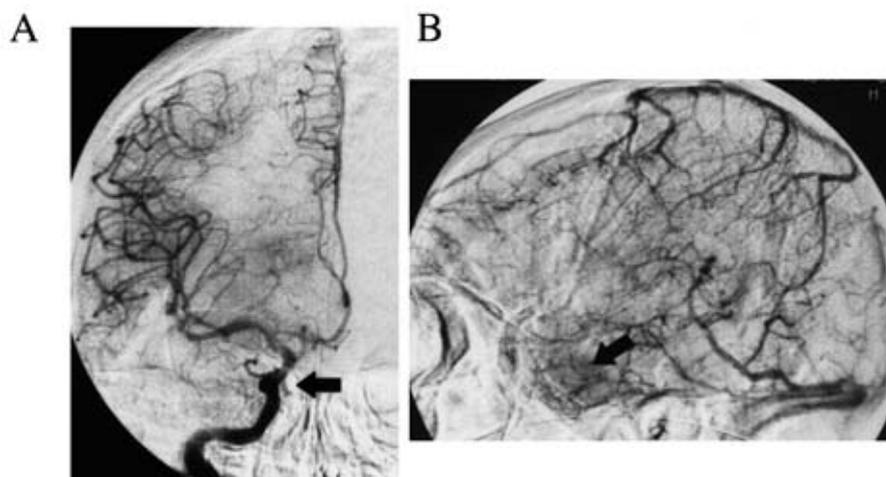


Figure 2. A: Carotid arteriogram did not show any involvement of right internal carotid artery (arrow). B: Cerebral angiogram showed filling defects in the cavernous sinus (arrow) in the venous phase.

the superior ophthalmic vein and is more common than previously assumed (4). The veins and venous sinuses that communicate with cavernous sinus are valveless, and therefore thrombophlebitis may affect the cavernous sinus in a retrograde fashion. Cavernous sinuses drain the blood from the orbits and the anterior part of the base of the brain via the sphenoparietal sinus and the middle cerebral veins. As a result, thrombosis can extend from the cavernous sinus to other dural venous sinuses. Thus, hemorrhagic infarction may have developed due to tributary cerebral venous thrombophlebitis.

Silver et al reported that pituitary insufficiency and the syndrome of inappropriate antidiuretic hormone secretion followed thrombosis of the cavernous sinus (12). Hypopitui-

tarism is a rare complication of cavernous sinus thrombosis, possibly related to infectious necrosis or aseptic infarction of the gland, which can develop more than a year after the initial event (1, 2). In the present case, necrosis of the pituitary gland was observed in the postmortem examination. A mechanism might be that hemorrhagic necrosis of the pituitary gland is also caused by venous congestion of the pituitary gland due to cavernous sinus thrombosis. These rare complications should be taken into consideration in patients with septic cavernous sinus thrombosis.

We are grateful to Dr. Morishige Takeshita for his helpful comments on histopathology.

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