CASE REPORT

Subarachnoid Hemorrhage as the Initial Presentation of Cerebral Venous Thrombosis

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Abstract

Cerebral venous thrombosis presenting as subarachnoid hemorrhage (SAH) is very rare. We present a woman with thrombosis of the superior sagittal, straight, transverse and sigmoid sinuses who presented with SAH in the right temporal sulcus and bilateral cerebellar sulci. Brain perfusion CT demonstrated a delay of the mean transit time and high cerebral blood volume around the right posterior temporal lobe and cerebellum. These findings were compatible with venous congestion and they suggest the possibility that extension of the dural sinus thrombosis into the superficial veins caused localized venous hypertension with dilatation of the thin, fragile-walled cortical veins which eventually ruptured into the subarachnoid space.

Key words: subarachnoid hemorrhage, cerebral venous thrombosis, headache, low perfusion hyperemia

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Introduction

Patients with cerebral venous thrombosis (CVT) present with a wide spectrum of symptoms and signs (1-3). The clinical manifestation depends on the localization and rate of progression of the thrombus and the extent of venous collateralization. Involvement of more than one sinus causes a broad variety of symptoms, including headache, seizures, focal neurological deficits, lethargy, confusion, or even coma. However, CVT presenting as excessive subarachnoid hemorrhage (SAH) on computed tomography (CT) is very rare (4, 5).

We present the case of a woman patient with thrombosis of the superior sagittal sinus (SSS), straight sinus, transverse sinus and sigmoid sinus who presented with SAH in an unusual location. SAH was associated with seizures caused by the venous infarct. We describe the imaging findings of this patient with CVT presenting as SAH and discuss the mechanisms of development of SAH in association with CVT.

Case Report

A 52-year-old woman with progressive occipital headache, nausea and vomiting consulted a nearby medical institution, where her head CT was unremarkable, and she received an analgesic. She had a history of SAH at the age of 34 years, with surgical clipping of an aneurysm. The analgesic provided little pain relief. Two days later, her headache suddenly worsened and she was admitted to our institution. On admission, she was afebrile and neurological examination was normal, with no evidence of meningismus. Head CT demonstrated SAH in the right temporal sulcus and bilateral cerebellar sulci (Fig. 1A, B). MR Imaging could not be performed due to the presence of the intracranial clip. On admission, cerebral angiography was performed to detect any aneurysm in the intracranial vasculature as the cause of the SAH. The angiography showed no aneurysm, which would have been the more expected and likely cause of the SAH, but instead revealed the thrombosis of the SSS, straight sinus, transverse sinus and right sigmoid sinus (Fig. 1C). Brain perfusion CT performed on the following day demonstrated a delay of the mean transit time (MTT) around the right posterior temporal lobe and cerebellum (Fig. 1D, E). The area with delayed MTT showed a higher cerebral blood volume than the contralateral side. These findings were compatible with “low perfusion hyperemia” (6). She had had two pregnancies, both carried suc-
The blood levels of D-dimer and TAT were elevated (7.6 μg/mL and 9.4 μg/mL, respectively), these levels were normalized two months later. Screening for secondary causes of thrombosis, such as the history of hormone replacement therapy and infections in the ear and nasal cavity, was negative. The best of our efforts yielded no evidence of cancer. Coagulation tests, including the prothrombin time and activated prothrombin time, serum titers of anticardiolipin antibody and antiphospholipid antibody, serum homocysteine titer, and serum levels of protein C and S, antithrombin III and fibrinogen were all within normal limits.

Four days after admission, the patient had her first generalized tonic-clonic seizure, followed by drowsiness. A repeat head CT obtained within a few hours of the onset of the seizure demonstrated a hemorrhagic infarct in the right parietal lobe (Fig. 1F). Intravenous heparin treatment was initiated under the diagnosis of venous infarct caused by CVT; within the next 24 hours, the seizures and drowsiness had resolved. Two weeks before her admission, she had developed dyspnea on exertion; pulmonary perfusion scintigraphy done on day 18 showed wedge-shaped defects in the right middle and left inferior lobes (Fig. 2A, B). Although venous ultrasonography failed to reveal any evidence of deep venous thrombosis, the late phase of a contrast-enhanced CT on day 19 demonstrated thrombosis of the portal and splenic veins (Fig. 2C).

At the time of discharge from the hospital (day 130, the patient exhibited disorientation, however, follow-up assessment confirmed near-complete neurological recovery within a month. Oral warfarin maintained an international normalized ratio of 2.0-3.0 for the prevention of CVT recurrence.

**Discussion**

The broad variety of signs and symptoms that may be associated with CVT makes the clinical diagnosis of this condition difficult. Headache is the commonest presentation, and although it is usually of gradual onset, it can also be sudden. Other presentations include focal neurological deficits and impairment of consciousness. Forty percent of patients with a CVT present with a seizure. Making a precise diagnosis can be particularly difficult when a patient presents with SAH, because it is one of the rarer presentations of CVT; only about 30 cases have been reported to date (4). Furthermore, a review of the autopsy results of 353 patients who died of SAH did not reveal any case of CVT, suggesting that the association between the two conditions is rather exceptional (5).

The initial symptom in our case was progressive occipital headache. The headache probably reflected elevated intracranial pressure in the posterior cranial fossa with thrombosis of the transverse sinus, as there was no evidence of SAH on initial head CT. CVT is not conclusively excluded by nor-
Figure 2. A, B: Pulmonary perfusion scintigrams on day 18 show wedge-shaped defects of the right middle and left inferior lobes (arrows). C: Late phase of enhanced CT on day 19 demonstrates thrombus of portal and splenic veins (arrows).

The distribution of SAH associated with CVT is usually different from that of SAH of arterial origin, which has a characteristic pattern. The hemorrhage is usually limited by the sulci of the cerebral convexities when associated with SSS thrombosis (7), but it extends beyond the sulci of the cerebellar convexities, sparing the basal cisterns, when it is associated with thrombosis of the transverse/sigmoid sinuses (8). Thrombosis of multiple sinuses produced SAH in the sulci of both the cerebral and cerebellar convexities in the present patient. Such a hematoma distribution is extremely rare.

The precise mechanism of development of SAH in patients with CVT remains unknown; different pathophysiological explanations have been proposed: (a) cerebral venous thrombosis causes a local inflammatory response that increases the vascular permeability allowing for extravasation of blood into the subarachnoid space; (b) venous parenchymal hemorrhagic infarct is a potential complication of CVT and can rupture in certain cases into the subarachnoid space; finally, (c) extension of the dural sinus thrombosis into the superficial veins causing localized venous hypertension with dilatation of thin, fragile-walled cortical veins which eventually rupture into the subarachnoid space (7-11). In the present patient, the CT obtained on admission did not show any venous infarct. However, the perfusion CT on the following day showed delay of MTT and high cerebral blood volume, so called “low perfusion hyperemia” (6), around the right posterior temporal lobe and cerebellum. These findings reflected venous congestion and SAH originated from veins, and suggested that proposition (c) might apply to our case. The present report is SAH secondary to CVT in which cerebral hemodynamics were measured with the perfusion CT. These measurements were useful in the consideration of pathogenesis.

Management of SAH secondary to CVT is quite different from that of arterial SAH. Treatment of sinus thrombosis has long been controversial. The benefits of heparin have been demonstrated in a randomized and placebo-controlled trial of 20 patients (12). In a further placebo-controlled trial, 60 patients were randomized to either low-molecular weight heparin followed by warfarin, or placebo (13). The anticoagulated patients had better outcomes than controls, but the difference was not statistically significant. The investigators suggested that anticoagulation was safe, even in patients with cerebral hemorrhage (13). However, the present case was not intraparenchymal hemorrhage but rather SAH. We considered that heparin deteriorates SAH. So heparin therapy was not started from the admission day.

Conclusion

CVT should be considered in the differential diagnosis of patients presenting with SAH without angiographic evidence of aneurysm. When suspected, primary MR venography or CT venography should be considered in further investigations. Angiography may still be required in certain situations where the clinical index of suspicion is high and CT and MRI are negative.

References