

[CASE REPORT]

‘Dense Inverted Triangle Sign’ in Cerebral Venous Thrombosis

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Abstract:

The early diagnosis of cerebral venous thrombosis in the emergency department is challenging. A 70-year-old man presented to the emergency department after falling with new-onset convulsions. Brain unenhanced computed tomography (CT) revealed right frontal hemorrhage indicative of traumatic subarachnoid hemorrhage (SAH). Brain unenhanced CT on day 2 revealed increased density in the anterior superior sagittal sinus (SSS), namely ‘dense inverted triangle sign.’ Brain magnetic resonance venography showed a filling defect in the anterior SSS. When interpreting unenhanced brain CT findings in the setting of acute convulsions or cortical stroke, including SAH, cerebral sinus abnormalities near stroke foci should be evaluated carefully.

Key words: computed tomography, superior sagittal sinus, cerebral venous thrombosis, dense sign, subarachnoid hemorrhage

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Introduction

Cerebral venous thrombosis (CVT), a rare type of cerebrovascular disease, is often overlooked because it can mimic other disorders; this ultimately results in a delayed diagnosis (1). In fact, a recent study in a large cohort showed that the mean time from the onset of symptoms to the diagnosis of CVT was 11.8 days (2). Although magnetic resonance venography (MRV) is the gold standard for diagnosing CVT (1), it is not routinely performed unless CVT is clinically suspected (3). Therefore, suspicion of CVT by unenhanced computed tomography (CT) is important.

However, the utility of serial unenhanced CT for diagnosing CVT is not well known. Furthermore, abnormalities of the anterior superior sagittal sinus (SSS) on unenhanced CT are not a well-known finding in CVT.

We herein report a case of CVT that was initially misdiagnosed in the emergency department (ED) as traumatic subarachnoid hemorrhage (SAH) in which a change in the anterior SSS density on brain serial CT was helpful for making a correct diagnosis.

Case Report

A 70-year-old man with a history of diabetes mellitus presented to the ED with new-onset convulsions. His wife had found him lying unconscious and convulsing at home. He had a history of gastric cancer and had undergone total gastrectomy 11 years prior.

On an examination, his blood pressure was 161/80 mmHg. His Glasgow Coma Scale (GCS) score was 7/15 (E1 V1M5); we observed right-sided conjugate eye deviation and generalized tonic-clonic seizures. Laboratory tests revealed the following: white blood cell count, 14,700/ μ L; hemoglobin, 11.4 g/dL; creatinine, 1.08 mg/dL; sodium, 139 mEq/L; potassium, 4.4 mEq/L; glucose, 472 mg/dL; glycohemoglobin, 9.7%; and D-dimer, 4.0 μ g/mL. Arterial blood gas levels were consistent with metabolic acidosis (pH 6.857; bicarbonate, 7.1 mmol/L). Unenhanced brain CT revealed right frontal hemorrhage mainly in cortical areas, indicative of SAH (Figure A). He was diagnosed with diabetic ketoacidosis, status epilepticus, and possible traumatic SAH.

Intravenous (IV) diazepam was repeatedly administered for convulsions, followed by IV fosphenytoin. He was intubated and admitted to the intensive-care unit (ICU) for fur-

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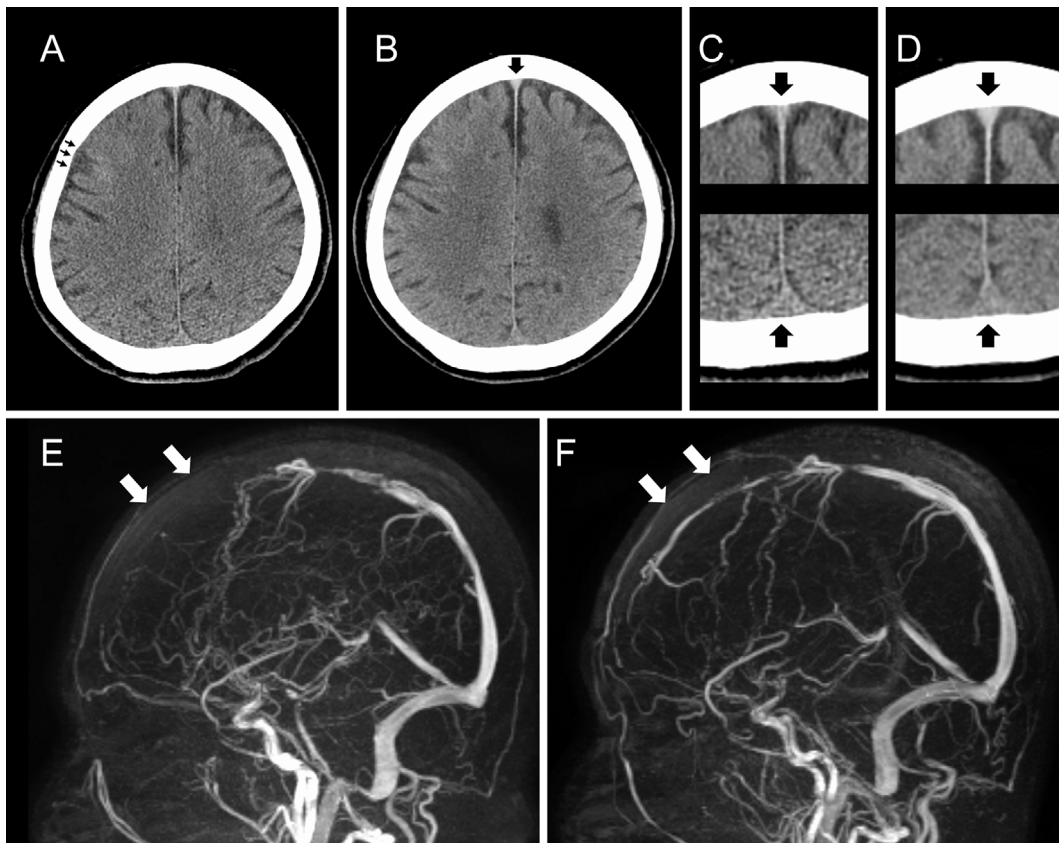


Figure. Unenhanced brain computed tomography (CT) at first examination (A, C) and on day 2 after hospitalization (B, D). (C) and (D) are enlarged views of the superior sagittal sinus (SSS). CT on day 1 shows right mild cortical subarachnoid hemorrhage (arrows) (A). There is no increased density in the SSS on day 1 (A, C). CT on day 2 shows increased density in the anterior SSS ('dense inverted triangle sign'), but not in the posterior SSS (B, D). Arrows in (B, C, D) indicates SSS. (E, F) Magnetic resonance venography (MRV). MRV on day 2 shows a filling defect in the anterior SSS (arrows) (E). MRV on day 79 shows improvements in this abnormality (arrows) (F).

ther management with IV insulin. Brain unenhanced CT on day 2 revealed an increased density in the anterior SSS that had not been seen on the initial scan (Figure B, C, D). The posterior SSS did not show any increase in density. Brain MRV showed a filling defect in the anterior SSS, indicative of CVT (Figure E). Further laboratory testing showed normal levels of protein C, protein S, antithrombin, and anti-cardiolipin antibodies; however, serum vitamin B12 was low (198 pg/mL; reference range: 233-914 pg/mL), and the serum homocysteine level was high (26.6 nmol/L; reference range: 3.7-13.5 nmol/mL).

CVT was believed to have been the result of diabetic ketoacidosis and hyperhomocysteinemia after gastrectomy. Intravenous heparin was initiated, followed by warfarin and intramuscular mecobalamin. His seizures were effectively controlled by levetiracetam. His GCS score improved to 14/15 (E4V4M6) on day 3 of hospitalization. He was discharged on day 20 without any neurological deficits. MRV on day 79 showed improvement of prior abnormalities (Figure F). The patients had no relapse one year after the onset.

Discussion

Our patient showed an acute convulsive state, and initial CT showed isolated SAH without a high dense sign in the cerebral sinus. The present case highlights two clinically important issues concerning the early diagnosis of CVT. First, CVT can manifest as isolated SAH in unenhanced CT, mimicking other causes, such as traumatic SAH. Second, the dense sign in anterior SSS without a posterior SSS abnormality was only confirmed by follow-up CT in this case.

SAH is thought to be a rare manifestation of CVT (4, 5). Early studies from the 2010s showed that 3%-4% of CVT cases had SAH (6, 7). However, a recent study showed that SAH is not uncommon in CVT; although isolated SAH is rare (4%), 24% of hemorrhagic CVT patients presented with SAH (8). This study also suggests that a combination of hemorrhage subtypes (especially parenchymal bleed with SAH) or the presence of sulcal SAH should raise suspicion about CVT as the cause of intracranial hemorrhage (8). Our case was challenging to diagnose at the initial examination. There were no eyewitnesses for the fall, so his SAH was initially diagnosed as of traumatic origin; however, his falling

could have been caused by convulsion due to CVT. Our case indicates that CVT is an important differential diagnosis of isolated cortical SAH.

As seen in our case, increased density may only be located at the anterior SSS on follow-up unenhanced CT, highlighting a pitfall in evaluating brain imaging in the ED and ICU setting. Although unenhanced brain CT is necessary for the assessment of seizures and unconsciousness, cerebral sinus abnormalities can be easily missed because clinicians may focus on other more apparent abnormalities, such as the evidence of a stroke. Previously, unenhanced CT findings in CVT mainly focused on the posterior SSS. Abnormal findings of posterior SSS have classically been known as a 'dense triangle sign' (increased density in the posterior SSS) on unenhanced CT and as an 'empty delta/triangle sign' (defect of contrast) on enhanced CT (3, 9, 10). A study among Asians showed that the dense triangle sign was positive in more than half of patients with CVT involving the SSS (3). In contrast, our patient did not show the dense triangle sign; however, he showed similar findings only in the anterior SSS. We propose that an abnormal imaging finding in the anterior SSS in patients with CVT be called the 'dense inverted triangle sign.'

In conclusion, partial CVT can manifest a dense sign of restricted cerebral sinus that can be easily overlooked. When interpreting unenhanced brain CT in the setting of acute convulsions or cortical stroke, including SAH, cerebral sinus abnormalities near stroke foci should be evaluated carefully. Physicians should consider follow-up if the etiology is not confirmed at the first examination.

The authors state that they have no Conflict of Interest (COI).

Declarations

The authors declare no competing interests relevant to the content of this manuscript.

Ethical standard statement

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

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