Radiology Case Reports 19 (2024) 370–373

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Hypodense cerebral venous sinus thrombosis on unenhanced CT: Time-dependent change of CT attenuation in intravenous thrombus ☆

Taiga Takano[a], *Mitsuru Matsuki, MD, PhD*[b,*]

[a] *Fifth-Year Medical Student, School of Medicine, Jichi Medical University, Shimotsuke-shi, Tochigi-ken, 329-049, Japan*

[b] *Department of Pediatric Radiology, Jichi Children's Medical Center, Shimotsuke-shi, Tochigi-ken, 329-049, Japan*

ARTICLE INFO

Article history:
Received 5 March 2023
Revised 17 September 2023
Accepted 18 September 2023

Keywords:
Cerebral venous sinus thrombosis
CT attenuation
Time-dependent change

ABSTRACT

Cerebral venous sinus thrombosis (CVST) is an important cause of stroke, which accounts for 0.5%-1% of all strokes. CVST is principally manifested as headaches, papilledema, psychiatric symptoms, impaired consciousness and seizure disorders, with or without neurological defects.

We encountered a case of CVST after subacute phase, revealing a hypodense thrombus in the superior sagittal sinus (SSS) on unenhanced CT. Retrospective observation of the serial unenhanced CT images taken at another hospital showed that the SSS thrombus changed from high to low attenuation for 10 days.

RBCs dominant CVST at acute phase shows high attenuation on unenhanced CT and is effective for heparin therapy, whereas CVST after subacute phase shows low attenuation on unenhanced CT by the thrombus contents of fibrin, hemosiderin and collagen, and may be ineffective to heparin therapy. Therefore, it is important to accurately identify high attenuation of the CVST at acute phase on unenhanced CT and treat with heparin as early as possible.

Introduction

Cerebral venous sinus thrombosis (CVST) causes reduced outflow of blood and cerebrospinal fluid, which can affect patients leads to venous congestion, infarction and intracranial hypertension. Conventional angiography is generally considered to be the gold standard for the diagnosis of CVST, while recently CT angiography and MR venography plays a significant role in the diagnosis of CVST. However, unenhanced CT is usually the first imaging investigation performed given the nonspecific clinical presentation of the stroke. On unenhanced CT,

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

* Corresponding author.
E-mail address: m18058tt@jichi.ac.jp (M. Matsuki).

https://doi.org/10.1016/j.radcr.2023.09.057

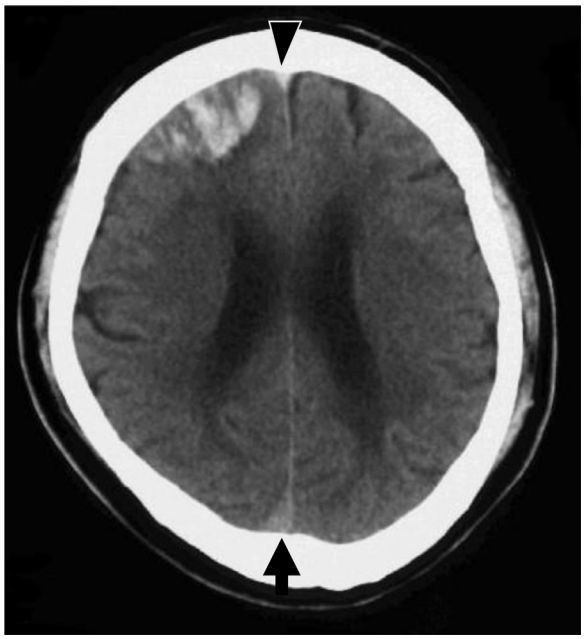

Fig. 1 – Unenhanced CT at admission at day 0. Unenhanced CT image showed subcortical intracerebral hemorrhage with surrounding edema in the right frontal lobe. Hypertensive intracerebral hemorrhage was diagnosed at another hospital. Under our retrospective review, a high attenuation (arrowhead) suggesting thrombosis at acute phase is detected in the anterior portion of superior sagittal sinus. While, the posterior portion of SSS shows a normal ranged attenuation (CT value: 23 HU) (arrow).

CVST at acute phase is commonly identified a high attenuation in venous sinus. While, Niel et al. reported a case with CVST at chronic phase demonstrating a thrombus with low attenuation on unenhanced CT [1].

Herein, we reported a case with CVST after subacute phase demonstrating a thrombus with low attenuation on unenhanced CT, in which the serial unenhanced CT images showed thrombus changing from high to low attenuation for 10 days.

Case report

The patient is a 56-year-old male who was transported to another hospital by ambulance with headache and disturbance of consciousness. Past history was myocardial infarction, hypertension, and diabetes mellitus. Blood pressure was 155/92 mm Hg. Blood analyses showed normal ranged hemoglobin and hematocrit (12.8 g/dL and 42%) and elevated blood glucose (121 mg/dL) and HbA1c (8.0%) levels. Unenhanced CT at day 0 showed subcortical intracerebral hemorrhage with surrounding edema in the right frontal lobe (Fig. 1). Under diagnosis of hypertensive subcortical hemorrhage, glycerol with a compound preparation of glycerin and fructose was injected for the management of increased intracranial pressure and cerebral edema. Symptoms improved for a time. However, headache recurred at day 9. Unenhanced CT showed subcortical hemorrhage in the left frontal lobe. Thereafter, under medication, symptoms resolved and there was no recurrence. Unenhanced CT was performed at day 19, which demonstrated an absorption of the subcortical intracerebral hemorrhage. At day 32, fundus examination was performed for an examination of diabetic ophthalmopathy and revealed bilateral papilledema. Cerebrospinal fluid opening pressure was measured, which revealed slight elevation at 20 cmH_2O. Thereby, he was referred to the neurosurgery department in our institution for further examination of intracranial hypertension. Unenhanced CT examination at day 36 was performed in our institution, which showed low attenuation area after subcortical hematoma absorption in bilateral frontal lobe, and low attenuation in the posterior portion of superior sagittal sinus (SSS)

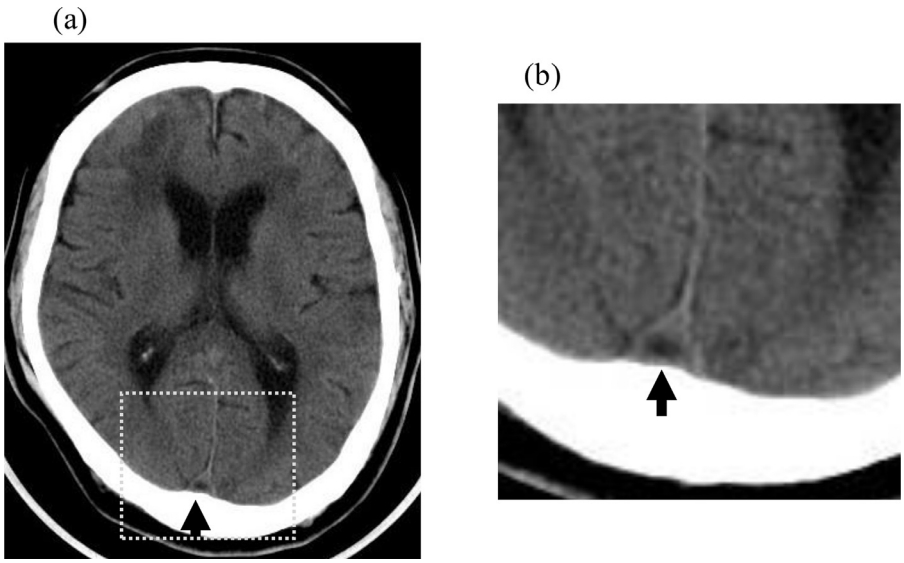

Fig. 2 – Unenhanced CT at day 36. (B) is a magnified view of the dotted line area in (A). Unenhanced CT images show low attenuation areas after hematoma absorption in bilateral frontal lobe and low attenuation (CT value: 11 HU), suggesting thrombosis after subacute phase, in the posterior portion of superior sagittal sinus (SSS).

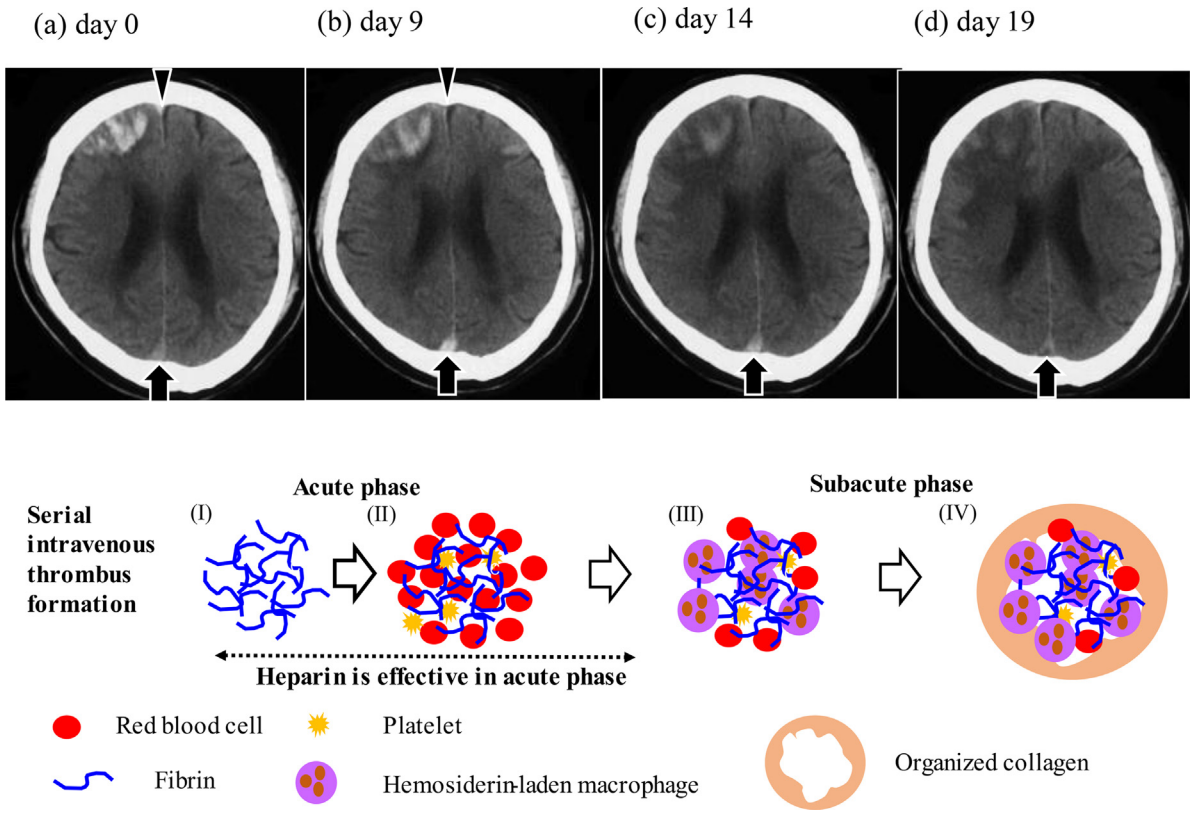

Fig. 3 – Serial unenhanced CT at another hospital and serial intravenous thrombus formation. Serial unenhanced CT images (A-D) show a decreasing of high attenuation in the anterior portion at day 9 (arrowheads (A), (B)) and a high attenuation (CT value: 72 HU) in the posterior portion (arrow (B)) of superior sagittal sinus (SSS) at day 9, and thereafter, the thrombosis decreases in attenuation (arrow (C)) (CT value at day 14 and 19: 48 HU and 14 HU). Serial intravenous thrombus formation (I-IV) corresponding serial CT images is as follows. At acute phase, thrombin creates a loose fibrin network (I), and red blood cells (RBCs) and a few of platelets become trapped within the fibrin network, and RBCs dominant thrombus is formed (II). The fibrin component at an acute phase is continuously being remodeled, undergoing both polymerization and degradation. This makes the acute thrombus highly sensitive to anticoagulation, which promote fibrin breakdown. At subacute phase (III), hemosiderin-laden macrophages can be gradually identified, as significant RBC breakdown occurs. Then, the thrombus appears organized 7-10 days after presentation, classically beginning at the periphery of the thrombus and spreading inward (IV). From the foregoing, RBCs dominant CVT at acute phase shows high attenuation on unenhanced CT and is effective for heparin therapy, whereas CVT around 10 days after presentation shows low attenuation on unenhanced CT by the thrombus contents of fibrin, hemosiderin and collagen, and may ineffective to heparin therapy.

(CT value: 11 Hounsfield unit (HU)) (Fig. 2). We suspected SSS thrombosis after subacute phase. SSS thrombosis was confirmed by conventional angiography at day 38.

We retrospectively reviewed the previous and serial CT images examined at another hospital. Unenhanced CT at day 0 (Figs. 1 and 3A) showed a high attenuation in the anterior portion of SSS, suggesting a SSS thrombosis at acute phase, which was considered to induce subcortical intracerebral hemorrhage. Posterior portion of SSS showed a normal attenuation (CT value: 23 HU). Unenhanced CT at day 9 (Fig. 3B) showed a decreasing of high attenuation in the anterior portion and an appearance of high attenuation in the posterior portion of SSS (CT value: 72 HU). Subsequent sequential unenhanced CT examinations (Fig. 3B-D) showed decreasing attenuation of the thrombosis in the posterior portion of SSS (CT value at day 14: 48 HU (Fig. 3C)). Unenhanced CT examination at day 19 (Fig. 3D) showed a low attenuation (CT value: 18 HU), suggesting a thrombosis at subacute phase.

Discussion

Cerebral venous sinus thrombosis (CVST) is a rare cerebrovascular disease that a thrombus forms in the venous sinus. The most common locations of thrombosis are the superior sagittal sinus and transverse sinus. It mostly presents with focal cerebral edema, venous cerebral infarction, and intracranial hypertension. The risk factors are prothrombotic conditions, oral contraceptives, pregnancy, puerperium, dehydration, malignancy, infection, head injury, and mechanical precipitants. Impaired venous return due to CVST causes development

of collateral veins. In the treatment, anticoagulation therapy using unfractionated or low-molecular-weight heparin is recommended. Conventional angiography is generally considered to be the gold standard for the diagnosis of CVST, while recently CT angiography and MR venography plays a significant role in the diagnosis of CVST. However, unenhanced CT is usually the first imaging investigation performed given the nonspecific clinical presentation of the stroke. On unenhanced CT, CVST at acute phase is commonly identified a high attenuation in the venous sinus. The increase in CT attenuation at acute phase is caused by clot retraction, eliminating water and thereby raising the concentrations of red blood cells. Buyck et al. reported that CT attenuation measurement of the venous sinus and CT attenuation to hematocrit ratio are useful in diagnosing CVST at acute phase [2]. And, Virapongse et al. reported that CVST attenuation decreases for approximately 7-14 days [3], at which thrombus cannot be differentiated from normal blood or may even appear low attenuation relative to blood as our case. Niel et al. reported a case with hypodense CVST at chronic phase on unenhanced CT [1]. The pathology literatures report the following [4,5]: At acute phase of intravenous thrombus, thrombin rapidly converts fibrinogen to fibrin, and fibrin polymerization creates a loose fibrin network. Red blood cells (RBCs) and a few of platelets become trapped within the fibrin network, and RBCs dominant thrombus is formed. The fibrin component at an acute phase is continuously being remodeled, undergoing both polymerization and degradation. This makes the acute thrombus highly sensitive to anticoagulation, which promote fibrin breakdown. At subacute phase, hemosiderin-laden macrophages can be gradually identified, as significant RBC breakdown occurs. Then, the thrombus appears organized 7-10 days after presentation, classically beginning at the periphery of the thrombus and spreading inward.

In conclusions, RBCs dominant CVST at acute phase shows high attenuation on unenhanced CT and is effective for heparin therapy, whereas CVST around 10 days after presentation shows low attenuation on unenhanced CT by the thrombus contents of fibrin, hemosiderin and collagen, and may ineffective to heparin therapy. Therefore, it is important to accurately identify high attenuation of the CVST at acute phase on unenhanced CT and treat with heparin as early as possible.

Patient consent

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

REFERENCES

[1] Neal E, Sturgeon J. Hypodense cerebral venous sinus thrombosis on unenhanced CT: A potential pitfall. Report of a case and review of the literature. Radiol Case Rep 2020;15(1):35–8. doi:10.1016/j.radcr.2019.10.012.
[2] Buyck PJ, De Keyzer F, Vanneste D, Wilms G, Thijs V, Demaerel P. CT density measurement and H:H ratio are useful in diagnosing acute cerebral venous sinus thrombosis. Am J Neuroradiol 2013;34(8):1568–72 Epub 2013 Mar 7. doi:10.3174/ajnr.A3469.
[3] Virapongse C, Cazenave C, Quisling R, Sarwar M, Hunter S. The empty delta sign: frequency and significance in 76 cases of dural sinus thrombosis. Radiology 1987;162(3):779–85. doi:10.1148/radiology.162.3.3809494.
[4] Nosaka M, Ishida Y, Kimura A, Kondo T. Time-dependent organic changes of intravenous thrombi in stasis-induced deep vein thrombosis model and its application to thrombus age determination. Forensic Sci Int 2010;195(1-3):143–7 Epub 2010 Jan 8. doi:10.1016/j.forsciint.2009.12.008.
[5] Czaplicki C, Albadawi H, Partovi S, Gandhi RT, Quencer K, Deipolyi AR, et al. Can thrombus age guide thrombolytic therapy? Cardiovasc Diagn Ther 2017;7(3):S186–96. doi:10.21037/cdt.2017.11.05.