Acute Subarachnoid Hemorrhage as the Initial Presentation of Extensive Cortical and Dural Sinus Venous Thrombosis

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Abstract: Cerebral venous thrombosis (CVT) is an infrequent, potentially fatal disease that is treatable if detected early. An initial presentation with subarachnoid hemorrhage (SAH) is very rare. We report a case of a 40-year-old woman that presented with superficial subarachnoid hemorrhage in the right convexity of the brain with extensive thrombosis of the deep and superficial venous system. The non-enhanced CT scan demonstrated increased attenuation of the venous sinuses. Increased awareness of this presentation of CVT can help prevent delays in diagnosis and treatment.

Keywords: subarachnoid hemorrhage, Cerebral venous thrombosis, headaches

1. Introduction

Cerebral Venous Thrombosis (CVT) is a potentially fatal disease but still treatable if detected early. Subarachnoid hemorrhage as the initial presentation of CVT is very rare. Patients with CVT can be difficult to diagnose because of the varied clinical presentation. Hence, neuroimaging is an essential tool for diagnosis of CVT.

2. Case

A 40-year-old women with a history of asthma on albuterol inhalation PRN, back pain and chronic allergic rhinitis, presents with a continuous headache for 6 days. There is no past history of any coagulopathy and the patient has been on oral contraceptive pills for more than one and half years. The patient had a prior bout of headaches two months earlier, which markedly improved with paracetamol. In her most recent bout, she complains of continuous, progressively worsening headaches and associated photophobia. She has no history of fever, neck pain, movement disorder or active nasal problem.

The patient is conscious, alert, with a blood pressure of 117 / 80, temperature of 36.1 C, pulse rate of 98, and 98% oxygen saturation on room air.

There is progressive weakness in the left side of the body. There is left sided lower and upper extremity weakness measuring 3 out of 5. However, high cerebral function and sensation are intact.

A non-enhanced CT scan was requested to exclude intracranial hemorrhage. This demonstrated superficial acute subarachnoid hemorrhage in the right convexity (figure 1A) with normal basal cisterns (figure 1B) and increased attenuation of the veins seen in the superior sagittal sinus (Dense clot sign), straight sinus (Fig 2A), transverse sinuses and internal cerebral veins. Moreover, some of the cortical veins overlying the right cerebral hemisphere also showed increased attenuation (cord sign) (Fig 2-B). A follow up post contrast CT scan demonstrated filling defects in the previous mentioned veins (empty delta sign, Fig 3A-B-C). Therefore diagnosis of CVT was made by the radiologist.

3. Discussion

Acute non-traumatic spontaneous subarachnoid hemorrhage occurs mainly due to ruptured aneurysm in 85% of cases, perimesencephalic hemorrhage in 10% and a variety of rare conditions in 5% of patients. The characteristic hemorrhagic pattern in the previously mentioned two most common causes of non traumatic subarachnoid hemorrhage (95%) classically involves the basilar cisterns.

In this case report, our patient is noted to have an isolated superficial subarachnoid hemorrhage in the right convexity of the brain with normal basilar cisterns. This finding represents the main imaging manifestation of CVT in our patient and is likely responsible for the patient's clinical symptoms. In a previous comprehensive review by van Gijn et al., CVT was not mentioned as a cause of subarachnoid hemorrhage, underscoring its rarity. Diagnostic neuroimaging studies (CT and MRI) are essential in the diagnosis of CVT. Usually CT scan is acquired initially in patients with severe headache, due to its relative low cost, short imaging time, and widespread availability.

We diagnosed CVT with CT scan (without/with contrast). There are indirect and direct signs to diagnose CVT with CT Scan (without/with contrast). In our case, the importance of closely reading the head CT scan is highlighted by the paucity of radiologic findings on non-enhanced CT. Indeed, hyperattenuation is present in only 25% of cases of CVT. Increased attenuation of the venous sinuses can also be seen in patients with dehydration, elevated hematocrit level, subarachnoid or subdural hemorrhage. The presence of these findings with consideration of the patient history encouraged the radiologist to further evaluation of this patient with contrast CT Scan which demonstrated extensive cerebral venous thrombosis.

The precise mechanism of development of SAH in patients with CVT remains unknown. Different pathophysiological
explanations have been proposed: (a) CVT 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. In our case report, the
most relevant mechanism is the last one, as there was
thrombus in the dural sinus with extension to the superficial
cortical veins. Interestingly, as in our case, SAH was
reported mainly in patients with extensive CVT. These
findings suggest that the rarity of SAH is related to the rich
anastomotic network of venous brain drainage, which
precludes occurrence of significant venous hypertension.

4. Conclusion

Subarachnoid hemorrhage as the initial presentation of CVT
is very rare. Increased attenuation of the sinuses and cerebral
veins on non-enhanced CT scan was the only clue for the
diagnosis in our case. The awareness of this rare
presentation of CVT by the radiologist and referring
clinicians may prevent delays in the diagnosis and treatment.

References

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Figure 1-A Non-Enhanced axial CT scan shows acute
subarachnoid hemorrhage overlying the right cerebral
hemisphere convexity.

Figure 1-B Non-Enhanced axial CT scan shows normal
quadrigeminal and interpeduncular cisterns
(Parts of basal cisterns).
Figure 2-A  Non-Enhanced axial CT scan shows increased attenuation of the superior sagittal sinus (Dense clot sign) and the straight sinus.

Figure 2-B  Non-Enhanced Axial CT scan shows increased attenuation of the cortical vein in the right convexity (Cord sign).

Figure 3-A  Enhanced Sagittal CT scan demonstrates extensive filling defects through superior sagittal sinus, straight sinus and internal cerebral veins (Extensive Thrombosis).

Figure 3-B  Enhanced Axial CT scan shows filling defect of the cortical vein in the right cerebral hemisphere convexity.
Figure 3-C  Enhanced Axial and coronal CT scan images demonstrate filling defects in the superior sagittal sinus (Empty delta sign) as well as the transverse sinuses (coronal image) more pronounced on the right.