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Review Articles

Cerebral venous thrombosis as a rare cause of subarachnoid hemorrhage: case report and literature review ☆

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ABSTRACT

We report a 48-year-old woman presenting with subarachnoid hemorrhage (SAH) as the first manifestation of superior sagittal sinus thrombosis. In a literature review of 73 cases, SAH associated with cerebral venous thrombosis (CVT) was usually seen at the cerebral convexities. SAH was adjacent to thrombosed venous structures; therefore, the most possible explanation seems to be the rupture of cortical veins due to extension of thrombosis. Computed tomography (CT) was effective for diagnosis of CVT in only 32% of the cases. CVT should be considered when SAH is limited to cerebral convexities and magnetic resonance (MR) imaging with MR venography should be performed.

1. Introduction

Cerebral venous thrombosis (CVT) is a relatively uncommon cerebrovascular disease. Although CVT can occur at any age, it predominates in children and young adults, accounting for 1–2% of strokes [1]. CVT is diagnosed more commonly than previously thought with the advent of accurate noninvasive imaging methods. The estimated annual incidence of CVT is reported to be between two and seven cases per 1 million populations, but it is estimated that five to eight cases may be diagnosed at a tertiary care referral center [2]. CVT is a potentially life-threatening disease, and the clinical diagnosis can be difficult because of a wide spectrum of clinical presentations and numerous causes. There are many predisposing factors for CVT classifying as local (sinus trauma, regional infection, and neoplastic invasion or compression) or systemic (hereditary coagulopathies, peripartum state, dehydration, oral contraceptive use, and hypercoagulable states secondary to malignancy). The etiology is unknown in 25% of cases [1–3].

We report a patient presenting with subarachnoid hemorrhage (SAH) as the first manifestation of superior sagittal sinus (SSS) thrombosis. The presentation of CVT with SAH is very rare. Therefore, we performed a literature review of CVT as a cause of SAH and discussed the preferred imaging modalities in the diagnosis of this presentation.

2. Literature review

The review of the literature included articles published in English from 1996 to 2012. We performed the literature search by using the terms *subarachnoid hemorrhage*, *cerebral venous thrombosis and subarachnoid hemorrhage*, and *cortical vein thrombosis* on PubMed and Medline cross-referencing pertinent articles from initial PubMed and MedLine searches. We also used the translated English abstracts of three case reports with sufficient information which were not available in English [4–6].

3. Case report

A 48-year-old woman with no significant medical history was admitted with a 1-week history of acute onset of severe headache and gait disturbance. Her level of consciousness was normal. She was afebrile, and her neurological examination was normal, with no evidence of meningismus. Magnetic resonance (MR) imaging; T1-weighted, T2-weighted, fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), and susceptibility-weighted imaging (SWI) followed by MR venography (MRV) in two-dimensional (2D) time-of-flight (TOF) mode (1.5 T Espree, Siemens, Erlangen, Germany) were performed on the same day. T1-weighted and FLAIR images (Fig. 1A, B) demonstrated abnormally increased signal intensity in the sulci of the bilateral frontoparietal convexity, better identified on the FLAIR sequence, and SWI (Fig. 1C) showed hypointense signal intensity most compatible with SAH. In addition, there were abnormal hyperintense signals within the SSS on T1-weighted, T2-weighted, and FLAIR images which corresponded to subacute venous thrombosis (Fig. 1A, B, D). MRV confirmed the diagnosis of SSS thrombosis (Fig. 1E). There was

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no restricted diffusion indicative of acute infarct on DWI. MR angiography showed no evidence of an underlying vascular lesion. No predisposing event and risk factors were identified. The patient improved with anticoagulant therapy.

4. Results

We reviewed 38 articles with a total of 73 cases of SAH associated with CVT [4–41]. We essentially focused on diagnostic work-ups and radiological findings of SAH in the setting of CVT besides clinical presentations. We included the location of SAH and CVT and associated findings with respect to diagnostic imaging for this underreported phenomenon. Details regarding demographic features and radiological findings of CVT with SAH are given in Table 1. We are aware that the literature review is limited by a wide range of variability of imaging methods, subjects, interpretations, and quality of images in the reported data for each case. However, we could extract enough information to discuss the preferred imaging modalities in the diagnosis.

Thirty-one (42%) patients were male and 42 (58%) female which are consistent with previous reports with female predominance. The age range was between 1 and 83 years with a mean of 42 years.

The imaging modality for SAH was not available in 1 patient [6]. In 11 of a total of 72 patients, SAH was detected only on MRI, and for 5 of them, only MRI was performed [14,17,24,31]. We noted that SAH was detected on computed tomography (CT) in 61 of a total of 67 cases, whereas initial CT was normal in 4 of these 61 patients [13,28,41]. So, CT failed to detect SAH in only 6 (9%) of the cases [18,20,26,27,34,39]. Initial MRI was ineffective in 1 patient, and diagnosis was made by the subsequent MRI [17]. We could not evaluate the effectiveness of MRI for SAH because the first-line imaging test for SAH was generally CT and the findings for SAH on subsequent MRIs were not mentioned.

The imaging modality for CVT was not available in three patients [21,32]. For diagnosis of CVT, MRV or digital subtraction angiography (DSA) was performed in 6 of 70 patients [27]. MRV was done for 25 of a total of 64 patients, CT venography (CTV) for 4 patients, and DSA for 38 of them alone or in combination with other modalities. DSA was the only diagnostic method used for 12 patients for CVT. According to the literature, CT was diagnostic in 21 (31%) of a total of 67 patients for CVT; however, in only 3 of them, CT alone was sufficient for clinical management [13,32]. For the other cases, further diagnostic imaging with MRI, MRV, CTV, and/or DSA was performed to characterize or confirm the extent of the thrombosis. MR imaging was usually performed with MRV and mostly used to detect parenchymal abnormalities; we could not get sufficient information about the effectiveness of MRI without MRV for CVT detection. MR imaging alone was the preferred diagnostic modality for CVT in only 5 patients [8,17,18,26,38]. However, MRI played an essential role in 5 of 10 cases of isolated cortical vein (c) thrombosis, whereas DSA or MRV was normal [14,38,39].

There were no associated findings in 40 (55%) of the 73 patients additional to SAH in the setting of CVT. Nonhemorrhagic infarct (NHI) was seen in 4 patients, venous hemorrhagic infarct (HI) in 11 (15%) patients, and edema (E) in 12 (16%) patients. There was parenchymal hemorrhage (PH) in 8 (11%) patients and subdural hemorrhage (SDH) in 2 patients.

In the review of the prior reports, SSS was the most frequently thrombosed sinus as SSS was the only involved sinus in at least 14 of the 72 cases, with or without transverse sinus (T) in 6 of them [27] and with other sinuses in 30 of them. T was the second thrombosed sinus (at least 35 of the 72 cases) and was usually involved with SSS and sigmoid sinus (S) as S was never alone involved without T. Straight sinus (St) was involved in 10 cases mostly with SSS and/or transverse/Ss. Venous thrombosis of cerebral veins (CVs) including cs, galen, labbe, and trolard was seen in 23 of a total of 70 patients, while c thrombosis was associated with dural sinus thrombosis in 6 patients and was isolated in 10 of them.

The review of the previous reports showed that SAH was usually seen at cerebral convexities but never involved the skull base and basal cisterns in 62 of 65 cases. We noted that SSS thrombosis was usually associated with SAH at frontoparietal convexity, and less commonly at interhemispheric fissure (IF), and sylvian fissure. In sylvian SAH, SSS and transverse/Ss were frequently involved together. Infratentorial (I) SAH was also usually associated with the thrombosis of SSS and/or transverse and Ss. In addition, the parieto-occipital (O) and posterior temporal convexity SAH was related to T thrombosis and paramedian supra- and/or I SAH to St thrombosis. The distribution of thrombosed cerebral venous structures with the location of SAH was shown in Fig. 2.

5. Discussion

In our case, we observed SAH in the sulci of the convexity and subacute venous thrombosis in the SSS as a cause of SAH on MRI and MRV. According to the literature, the distribution of SAH associated with CVT was usually seen at cerebral convexities sparing the skull base and basal cisterns. Although the location of the SAH was variable, hemorrhage was usually adjacent to thrombosed venous structures. Nonenhanced CT (NECT) was effective for the diagnosis of SAH in 91% of the patients and for the diagnosis of CVT in only 31% of the patients.

CVT is a potentially fatal disease, and accurate diagnosis is crucial for prompt appropriate therapy. The main symptoms are headache, partial or generalized seizures, focal neurological deficits, and alteration in mental state [3,7]. Headache is the most frequent presenting symptom in about 90% of patients with CVT and was described as diffuse (D) and progressed in severity over days [42]. De Brugin et al. [8] described 10 patients presented with thunderclap headache mimicking SAH, which suggests a frequency of thunderclap headache of more than 10% in CVT patients.

Clinical data alone are usually not sufficient for a definitive diagnosis of CVT; therefore, imaging plays an essential role to make the diagnosis. Venous thrombi can be detected on CT and MR parenchymal images or with various venographic techniques including unenhanced MRV, contrast-enhanced MRV, and CTV [2].

NECT is the initial imaging examination in most cases of sudden acute neurological symptoms. The CT imaging findings related to CVT include hyperdense thrombus in the occluded sinus, the delta sign and cord sign on NECT, and intraluminal thrombus with enhancement of the dural sinus wall, the empty delta sign on enhanced CT, which is present in 16–46% of cases [1]. These findings have low sensitivity and specificity for the diagnosis of CVT. The delta sign can also be seen in SAH which is not always reliable for diagnosis. In addition, high-velocity venous flow in children and young adults, increased venous attenuation seen in the presence of elevated hematocrit, dehydration, and beam-hardening artifacts from the skull vault can result in a false-positive sign by mimicking a hyperdense venous sinus clot [1,2,7].

Recent reports considered MRI, including MRV, as the technique of choice for definitive diagnosis in all phases and follow-up of CVT. Parenchymal abnormalities such as cytotoxic E, vasogenic E, hemorrhage, and infarct have been reported in as many as 57% of patients with CVT and can be identified more readily on MRI than on CT [2]. In CVT, cerebral E and infarction are usually subcortical and does not conform to an arterial vascular territory as in the arterial stroke. In addition, it should be noted that diffusion abnormalities are variable and often reversible in CVT. Venous obstruction results in increased intracranial pressure and consequently decreased cerebral blood flow. So, first vasogenic E develops with elevated apparent diffusion coefficient (ADC) values; however, areas of decreased ADC, which may also be reversible, may eventually occur, and may be explained by decreased cerebral blood flow with neuronal swelling and membrane pump failure without neuronal death [1,2]. According to