

Case Report

Cerebral Venous Infarction due to Internal Jugular Vein Thrombosis: A Case Study and Review of Literature

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Cerebral venous sinus thrombosis (CVST) is an uncommon disease entity. In contrast-enhanced computed tomography (CT) or magnetic resonance venography (MRV) images of the brain, CVST usually manifests as a filling defect of the dural venous sinuses. Brain parenchymal involvement in CVST can be ranged from parenchymal oedema to haemorrhagic infarctions. Though the most frequent cause of haemorrhagic infarction in brain is CVST, other rare causes such as cerebral venous outflow obstruction due to neck vein pathology have been reported. We report a rare case of haemorrhagic brain infarctions secondary to bilateral internal jugular vein thrombosis in a 17-year-old woman, who has presented with worsening headache and seizures. She had high susceptibility of getting venous thrombosis for being a young female on oral contraceptive pills. While reporting a rare cause of cerebral haemorrhagic infarctions, this case report highlights the need for having a high degree of suspicion to diagnose CVST. Further, this case report emphasises the value of prompt and adequate imaging of neck veins if the haemorrhagic infarction presents with patent cerebral venous sinuses.

1. Introduction

Cerebral venous sinus thrombosis (CVST) is an uncommon disease, annually affects nearly 5 per million adults, accounts for 5% of young strokes, and shows female predominance. Early diagnosis and timely treatment of CVST would prevent serious neurological complications. However, a vague presentation of CVST warrants a high degree of suspicion to diagnose early [1].

The CVST manifests as a filling defect of cerebral veins in contrast-enhanced computed tomography (CT) images. Magnetic resonance venography (MRV) is as sensitive as CT venography in detecting CVST, in which CVST again manifests as a filling defect in venous sinus. Though the brain parenchymal involvement is not an essential feature of CVST, if present, brain involvement can be ranged from parenchymal oedema to haemorrhagic infarctions. Pathophysiology of brain parenchymal involvement is explained by poor venous drainage and associated high venous

pressure that leads to poor arterial perfusion [2]. Though the most prevalent cause of poor venous drainage in haemorrhagic cerebral infarction is CVST, other rare causes have also been reported [2].

Here, we report a rare case of bilateral internal jugular vein thrombosis as the cause of poor venous drainage that leads to cerebral haemorrhagic infarctions.

2. Case Report

A 17-year-old married woman presented with three episodes of generalised tonic-clonic seizures. She was confused on admission. The Glasgow Coma Scale (GCS) score was 11: she opened her eyes to verbal command; verbal response was confused; showed withdrawal motor response to pain. She had on and off headaches for the last ten years that worsened to become a persistent headache for the last two weeks. She vomited intermittently for last seven days prior to the admission. Her past medical, surgical, and family histories

were unremarkable with no history of abnormal coagulability, malignancy, trauma, or recent infection. Notably, she did not involve in activities that can cause neck trauma or repetitive neck compressions. She was nulliparous and on oral contraceptives for an unknown duration.

On examination, she was afebrile; vital parameters were normal. Except for a low GCS score as described earlier, general, systemic, and neurological examinations were unremarkable. No localising or lateralizing signs were found; the pupils were equally reacting to light. Since she was confused, cognitive behavioural assessment was not performed. Initial blood investigations were normal. Following initial resuscitation, she was transferred to the Radiology unit for computed tomography scan (CT) of the brain.

Initial assessment was performed with noncontrast CT scan of the brain and has shown a large haemorrhagic infarction in the left frontal lobe anteroinferiorly, with minimal oedema of the surrounding brain parenchyma. Even though there was minimal mass effect on the frontal horn of the left lateral ventricle, no significant midline displacement was found. Additionally, mild cerebral parenchymal oedema was found in the parasagittal region of the right frontal lobe (Figure 1) with no significant mass effect or midline shift. No evidence of extra-axial or intraventricular haemorrhages was found. The density of dural venous sinuses was equal to the density of the basilar artery; no hyperdensity in venous sinuses to suggest acute dural venous sinus thrombosis (CVST).

Three days later, her condition further deteriorated: opened eyes only to pain; produced only incomprehensible sounds; limb flexion observed for pain stimulation; frequent seizure episodes not responded to medications. Hence, she underwent pre- and postcontrast-enhanced CT scan of the brain and the neck. Precontrast CT brain has revealed a new haemorrhagic infarction in the right frontal lobe, while the left frontal infarction has involved in size (Figure 2). The CT venography has shown bilateral internal jugular vein thrombosis that extends from the jugular bulb to the confluence of subclavian veins (Figures 2 and 3). Despite having filling defects in the central part of the veins, periphery of internal jugular veins were opacified with contrast, suggesting partial recanalisation of thrombosed veins. However, the dural venous sinuses and both subclavian veins were patent (Figure 3). Bone window images revealed normal appearance of bilateral styloid processes; there was no impingement of styloid processes on the internal jugular veins.

The diagnosis was made as bifrontal cerebral haemorrhagic infarctions secondary to right and left internal jugular vein thrombosis. Despite adequate treatment, one week after the admission, the patient succumbed due to diffuse cerebral oedema and transtentorial herniation.

3. Discussion

We have presented a rare case of bilateral internal jugular vein thrombosis associated with cerebral haemorrhagic infarctions. Though the frequent aetiology of cerebral haemorrhagic infarctions is dural venous sinus thrombosis, in our case extensive neck vein thrombosis was the aetiology.

Even though cases with CVST propagating into the internal jugular veins and vice versa have been reported [3], isolated internal jugular vein thrombosis manifested as venous infarctions is almost unheard.

The proposed mechanism for cerebral venous (haemorrhagic) infarctions is reduced venous return that leads to high pressure in intracerebral veins. High venous pressure consequently leads to poor arterial perfusion, cell necrosis, and haemorrhages [1, 4]. If a venous infarction is evident in a patient with patent cerebral venous sinuses, other rare causes of reduced venous return, such as neck vein pathology, have to be excluded [4].

Typically, patients with internal jugular vein thrombosis or obstruction present with transient facial oedema, with or without associated neck swelling. However, the majority of neck vein thrombosis present with subtle signs such as pain and swelling at the angle of the jaw [5, 6]. The atypical presentation of internal jugular vein thrombosis in our patient has rendered the diagnosis difficult. Perhaps, worsening of the existing headache had indicated the onset of internal jugular vein thrombosis. At presentation, the thrombi in internal jugular veins were recanalised indicating subacute presentation. For being a young female who was on oral contraceptives, our patient was considered to be at high-risk category to develop venous thrombosis. Imaging studies to evaluate veins particularly are recommended for the patients with headaches of uncertain diagnosis [1].

The incidence of internal jugular vein thrombosis (IJVT) is still unknown, while internal jugular vein catheterisation (incidence 66% of total catheterisations) being the most frequent secondary cause [6, 7]. Malignancy, neck trauma, infections, and hypercoagulable status are considered as the other frequent secondary causes of IJVT [6]. Similar to our case study, the diagnosis of IJVT can be made with CT or MR venogram studies or with venous Doppler study of the neck. Venogram studies report higher sensitivity rates over Doppler studies due to excellent contrast resolution among soft tissue. CT and MR venogram studies report nearly equal sensitivity in detecting CVST. Contrast-enhanced MR venogram said to be with a higher sensitivity compared to former. Although we have reported cerebral venous infarction, as a rare complication of IJVT, pulmonary embolism (10.3%) and post thrombosis syndrome (14.4%) are considered to be the frequent complications of IJVT [2, 6, 8].

CT imaging of the brain reliably differentiates venous infarctions from arterial infarctions. Contrary to arterial infarctions, venous infarctions are frequently associated with parenchymal haemorrhages [2]. The typical cerebral venous infarctions (in noncontrast CT brain) are flame-shaped heterogeneous (mixed hypo- and hyperdense) areas in the brain parenchyma, which are commonly subcortical in position. The hyperdense areas of the lesion represent parenchymal haemorrhages. Arterial infarctions are wedge shaped, involving both cerebral cortex and medulla, which are usually confined to an arterial territory [9]. Differentiation of arterial infarction from a venous infarction is crucial owing to different patient treatment and management protocols [1].

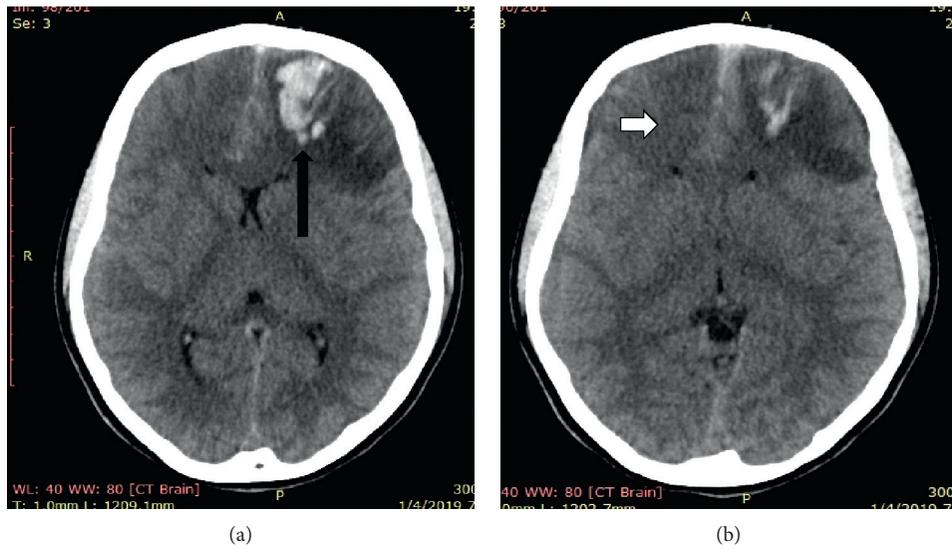


FIGURE 1: Noncontrast CT brain at presentation, indicating (a) haemorrhagic infarction in the left frontal lobe (black arrow) and (b) focal cerebral oedema in the right frontal lobe (white arrow).

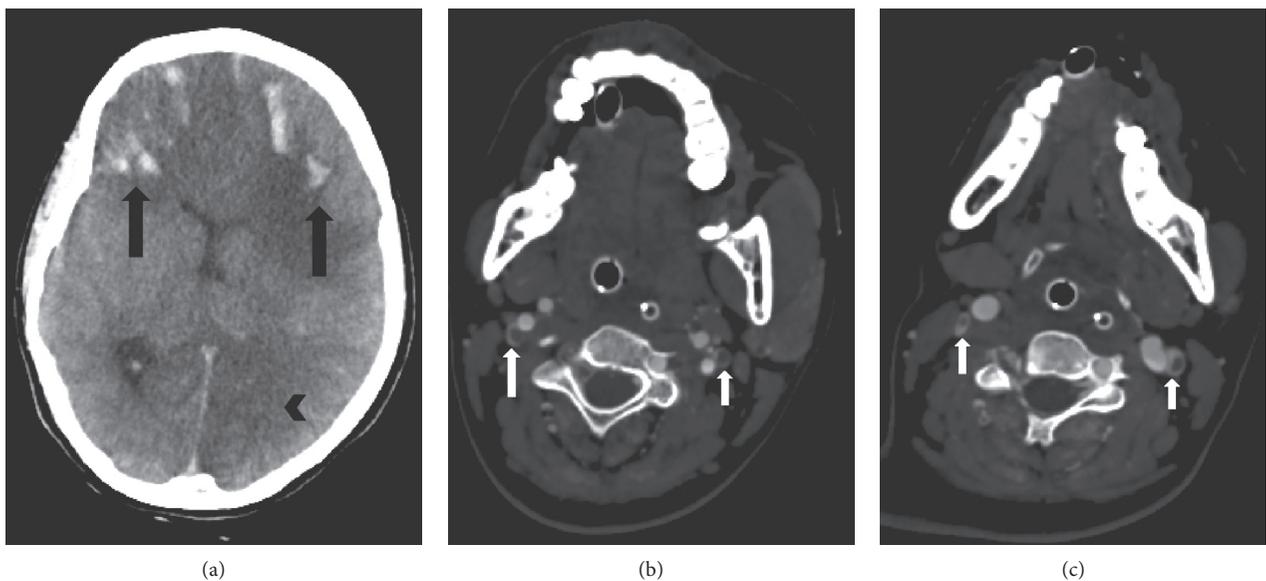


FIGURE 2: Follow-up contrast-enhanced CT brain and neck, indicating (a) haemorrhagic infarctions in both frontal lobes (black arrows); oedema in the left occipital lobe with loss of grey white demarcation (arrow head). (b, c) Contrast-enhanced CT brain and neck, showing filling defects in both internal jugular veins suggesting venous thrombosis (white arrows).

The prognosis of CVST is multifactorial. As in our case, the venous thrombosis that present with haemorrhagic infarction delineates an unfavourable outcome. Other factors associated with poor prognosis are old age, deep cerebral vein thrombosis, associated cerebral infection, or a cancer [1]. Such patients need intense monitoring and treatment to prevent short- and long-term sequel [2].

Identifying the aetiology of venous infarction is empirical for successful patient management. Frequently the aetiology of venous infarction is CVST. In noncontrast CT brain, CVST manifested as hyperdense dural venous sinus. However, venous sinus hyperdensity was seen only in 25% of

CVST. False-positive venous sinus hyperdensity can be a typical manifestation in dehydration, increased haematocrit, or the presence of concomitant subarachnoid haemorrhages. Similar to our patient, the dehydrated patients have equal densities in both cerebral arteries and veins.

Presence of CVST can be confirmed using a postcontrast CT scan of the brain, preferably in a CT venography [1, 2]. Typically, the location of cerebral haemorrhagic infarction is related to the site of the CVST. Since the parenchymal changes of CVST is often related to the venous drainage territory of the brain, the site of infarction can be used as a guide to locate the CVST. Often bilateral parasagittal

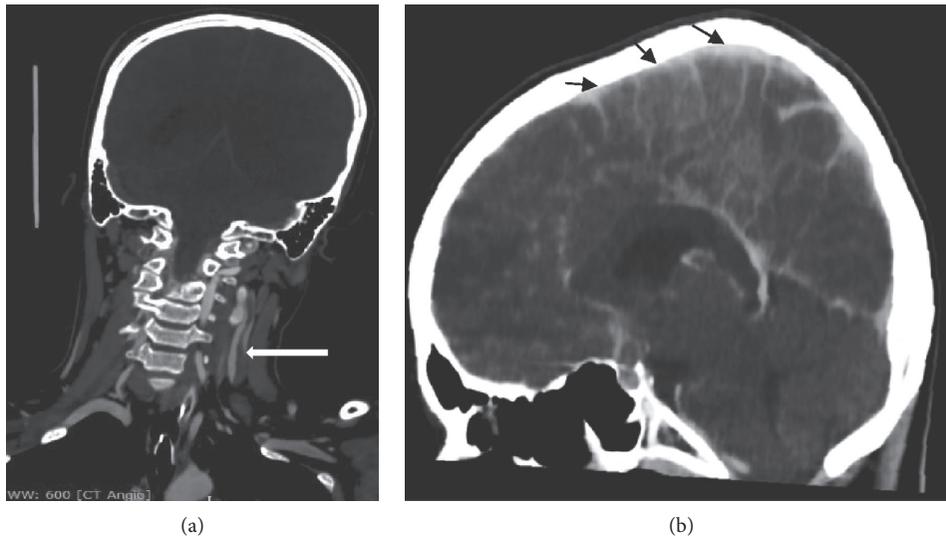


FIGURE 3: (a) Coronal CT scan of the neck demonstrating central thrombus in the left internal jugular vein (white arrow). (b) Sagittal postcontrast CT scan of the brain indicates no thrombosis in the superior sagittal sinus (black arrows).

infarctions represent superior sagittal sinus thrombosis; ipsilateral temporo-occipital and/or cerebellar infarctions may represent transverse sinus thrombosis; and bilateral periventricular, basal ganglia, and thalamic infarctions represent deep cerebral vein thrombosis [1, 10]. Considering all, authors recommend CT or MRI venography of the brain and neck to diagnose or exclude venous thrombosis, for every patient with haemorrhagic infarctions present with patent cerebral venous sinuses. Considering the availability and being able to perform at the bedside, the Doppler study of the neck veins is a complementary option to CT and MR venography. However, the sensitivity of venous Doppler in detecting thrombosed vein is much lower than CT or MR venography [11].

While presenting a rare case of cerebral venous infarction secondary to internal jugular vein thrombosis (IJVT), authors would like to highlight the following limitations. In this case, IJVT is evaluated only with contrast-enhanced CT study, and the majority of radiological features of IJVT would be described if venous Doppler studies of the neck and MR venography are also combined. Confirming the association between venous infarction and IJVT is suggested using an adequate sample of similar cases. Further studies with a sufficient sample size are recommended to evaluate the prognosis and treatment efficacy of IJVT.

4. Conclusion

Since early diagnosis and treatment determine favourable patient outcome, the patients with cerebral haemorrhagic infarctions should image promptly and adequately to diagnose associated venous pathology, such as CVST, jugular vein thrombosis, or any other form of venous outflow obstruction.

Consent

Informed consent has been obtained from the patient's husband.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this report.

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