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Case Report

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Hemorrhage in sinus vein thrombosis: the "cashew sign"

Şeyda Çankaya[1], Ahmet Özşimşek[1], Ece Özdemir Öktem[1], Hatice Lakadamyalı[2], Burak Yuluğ[1]

[1]Department of Neurology, Faculty of Medicine, Alanya Alaaddin Keykubat University, Alanya, Turkey
[2]Department of Radiology, Faculty of Medicine, Alanya Alaaddin Keykubat University, Alanya, Turkey

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Corresponding Author: Burak Yuluğ, burak.yulug@alanya.edu.tr

ABSTRACT

Sinus vein thrombosis (SVT) has many clinical heterogeneity and diversity in misdiagnosis and inappropriate treatment due to many etiologic factors. The main factors for diagnosing SVT are good clinical skill and a good interpretation of the radiologic image. Intracerebral hemorrhages are quite common in sinus vein thrombosis (SVT). The morphology of these hemorrhages may vary from small localized juxtacortical lesions and subarachnoid hemorrhages to large hemorrhagic infarcts.[1] In this presentation, we aimed to emphasize venous hemorrhage and the "cashew sign" seen in SVT.

Keywords: Sinus vein thrombosis, cashew sign, hemorrhage

INTRODUCTION

Venous sinus thrombosis (VST) is a rare cause of ischemic stroke. Nowadays, increased awareness of this disease and advancements in imaging methods have facilitated the detection of more cases. Its incidence is reported to be 0.2-1.2 cases per 100,000 individuals per year.[2] Most ischemic strokes have arterial origins, while venous strokes constitute only 1% of all strokes.[2] It can occur in all age groups, but it is more common in newborns and childhood than in adults. There is no gender difference in children and older age groups; however, in the young adult age group (20-35 years), it is three times more common in women than in men.[3] This gender disparity is mainly attributed to additional risk factors such as pregnancy, the postpartum period, and oral contraceptive use.[3] Despite being a potentially fatal condition, the prognosis for VST is generally favorable.

CASE

A 43-year-old woman applied to the emergency department with a headache since last week and left-sided numbness and weakness for the previous three days. She had a history of thalassemia minor. A neurological examination revealed left hemiparesis (motor strength 4/5) and Babinski's sign on the left side. There were no meningeal irritation signs. Diffusion-weighted brain magnetic resonance imaging (DWI) revealed diffusion restriction with nodular infarction on the parasagittal area in the right frontal region (Figure 1A and Figure 1B). Computed brain tomography (CT) revealed hemorrhage in the right parietal region (Figure 2A). Computed Tomography Angiography (CTA) was normal. Brain magnetic resonance imaging (MRI) showed the presence of edema with T2 hyperintensity in the right frontal parasagittal area and hemorrhage in the parietal region (Figure 2B). Contrast-enhanced MR venography (MRV) revealed areas compatible with thrombus, showing filling defects in the superior sagittal sinus that are consistent with SVT (Figure 3). In the secondary intensive care unit, low molecular weight heparin, anti-edema, and warfarin therapy were started. Also, topiramate has been added to the treatment for headache. In the neuroradiology council, the images were evaluated, and it was concluded that hemorrhage resulted from venous hemorrhage. On CT and MR images, venous hemorrhages called "cashew sign" with high specificity for SVT were identified (Figure 2). Her headache was relieved with treatment, and warfarin was started on the 3rd day of hospitalization with daily INR control. No positive vasculitis markers were detected in the patient. Department of Haematology suggested no additional recommendation for thalassemia minor. The patient's severity of headache decreased, and she was discharged on the 10th day of hospitalization with warfarin.

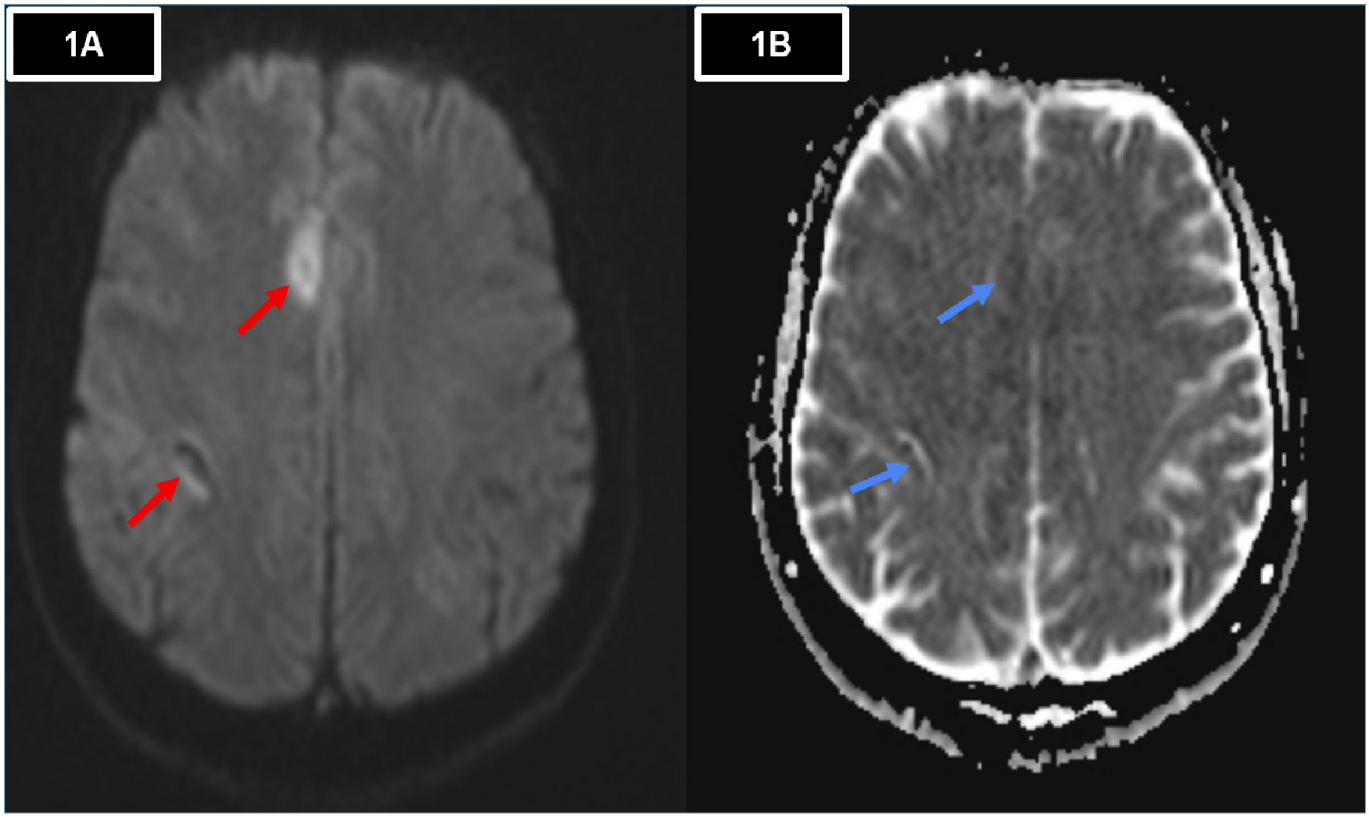

Figure 1.

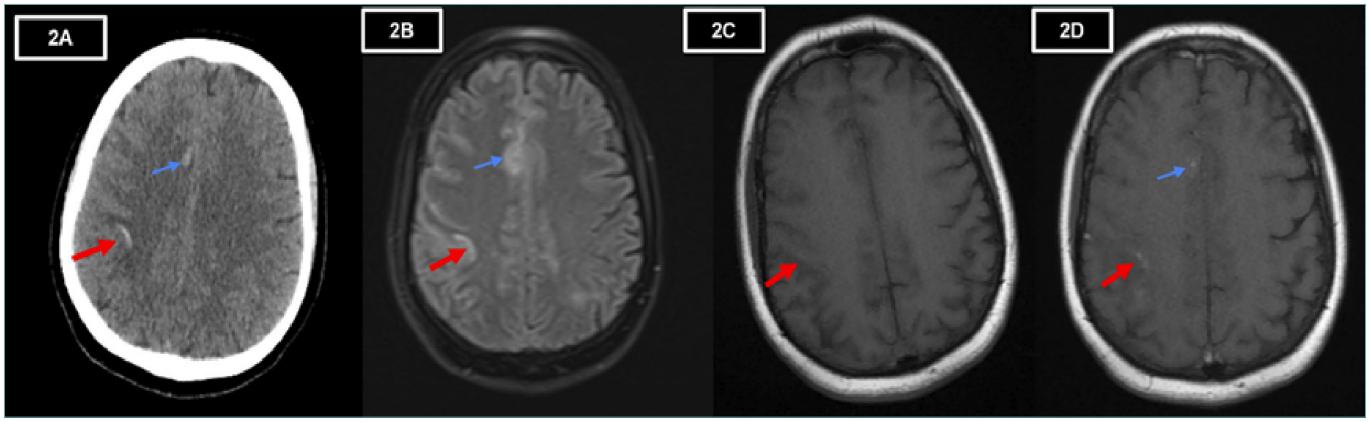

Figure 2.

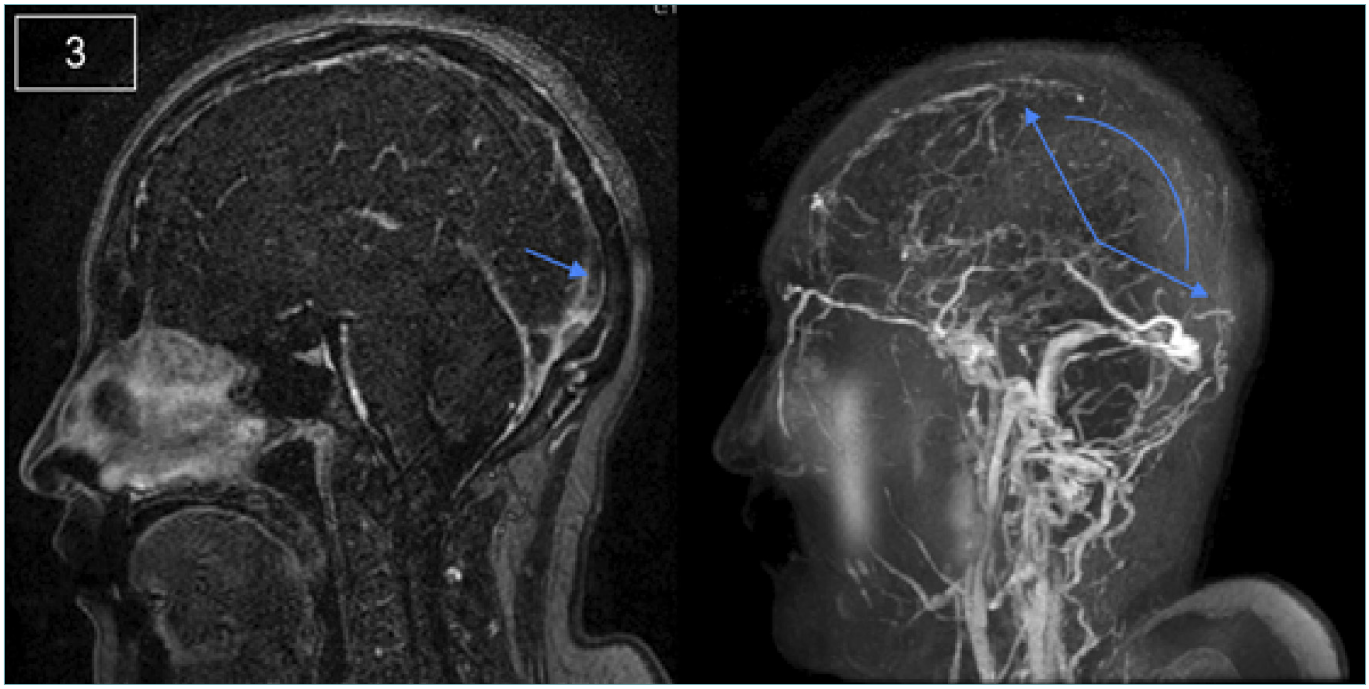

Figure 3.

DISCUSSION

Intracerebral hemorrhage was found in 40% of patients, and juxtacortical hemorrhage in 26% of cases with intracerebral hemorrhage in patients with SVT.[1] In cases of SVT, a concave shape resembling a cashew nut called a "cashew sign" appears at the sulcus base in juxtacortical white matter hemorrhages. The "cashew sign" formed by hemorrhage of the juxtacortical veins is seen on CT and frequently in superior sagittal sinus thrombosis.[4] For non-traumatic SVT, the cashew sign shows relatively high specificity [specificity: 0.98 (95% CI 0.95 -1.0)].[1] Various imaging techniques, primarily non-contrast or contrast-enhanced CT with venous phase acquisition and MRI with MR venography, are employed for the evaluation. CT offers limited insights into the clot, vessels, and parenchyma, with minimal indicators for intracranial hypertension.[5] Conversely, CT venography demonstrates accuracy in diagnosing cerebral sinus thrombosis. In contrast, MRI provides comprehensive information on clot characteristics, vascular structures, parenchymal details, and potential indicators of intracranial hypertension.[5]

In general, symptoms of SVT include nausea, vomiting, headache, visual loss, seizures, and weakness. The diagnosis of SVT is based on radiologic and clinical findings. Symptoms in SVT vary depending on the location of the thrombosis, but the most common symptom is headache. The headache may intensify over days and become intolerable. It can be described as the most severe headache the patient has ever experienced.[6] Our patient also had an unbearable headache. The localization of the sinus thrombosis is a determinant in the clinical presentation. Superior sagittal sinus thrombosis is the most common localisation of SVT, as seen in our case.[7] Dinç et al.[8] presented a case with right-sided hemiplegia, aphasia, and cashew sign on the left frontoparietal area. Our case applied with left-sided numbness consists of a lesion on the right parietal lobe.

Our case had thalassemia minor as a risk factor for SVT. The most important risk factors for SVT are the classical Virchow triad of causes known as blood flow stasis, vessel wall changes and changes in blood content. An underlying cause can be found in approximately 80% of cases. SVT might relate to puerperium or pregnancy in young women.[9] SVT-related strokes account for 27-57% of all ischemic strokes associated with pregnancy.[10,11] Oral contraceptive use and coagulation disorders are important causes. Collagen tissue diseases and inflammatory bone diseases constitute a risk.[12,13] Genetic factors including homocysteine elevation, protein C-protein S-antithrombin 3 deficiency, Factor V Leiden mutation, and prothrombin gene mutations, are responsible for 10-15% of cases.[14] As soon as the diagnosis is confirmed, treatment should be started immediately. Anticoagulation is the main treatment for SVT. Low molecular weight heparin therapy has been found superior to unfractionated heparin.[15] We have started warfarin sodium and low molecular weight heparin therapy after confirming of resorption of hemorrhage with CT.

CONCLUSION

Despite increasing awareness among clinicians, SVT is a complex disease to diagnose clinically and radiologically. This difficulty is because the neurologic picture ranges from objective focal neurologic deficits to subjective symptoms of weakness and headache. Small juxtacortical hemorrhages are characteristic of CVT and are rarely observed in other conditions. Recognizing these hemorrhages can greatly facilitate the diagnosis of CVT for clinicians.

ETHICAL DECLARATIONS

Informed Consent

All patients signed the free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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