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Case Report

Deep venous sinus thrombosis with right thalamic infarction in a young patient after COVID-19 vaccination ☆

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ABSTRACT

Cerebral venous thrombosis is a rare illness, it compromises 0.5% of cases of cerebrovascular diseases globally. The condition can be treated if discovered and treated properly and quickly. With many known risk factors and in recent times with invent of the COVID-19 vaccine, there have been reported incidences of vaccination being implicated in cerebral venous sinus thrombosis. We report an unusual case of an adolescent female with imaging findings of deep cerebral venous sinus thrombosis and right thalamic infarction after recent vaccination against COVID-19. Laboratory results revealed microcytic hypochromic anemia. Further imaging was done which included a non-contrast CT head, magnetic resonance imaging, and magnetic resonance venography leading to a diagnosis of thrombosis of deep venous (galenic) system with vasogenic edema in bilateral thalami and left caudate nucleus with areas of infarction in the right thalamus. She was treated with subcutaneous low molecular weight heparin (Enoxaparin) and discharged on the third day under oral dabigatran and oral iron.

Introduction

Cerebral venous thrombosis (CVT) is a rare illness that represents approximately 0.5% of cases of cerebrovascular disease globally. The condition can be treated if discovered and treated properly and quickly [1]. Diagnosis is often difficult clinically because of non-specific symptoms like headache, focal neurological deficits, and decreased level of consciousness. Thus imaging is critical to diagnosis, and radiologists must be well-acquainted with findings that raise suspicion of CVT so that appropriate treatment (anticoagulation therapy) can be

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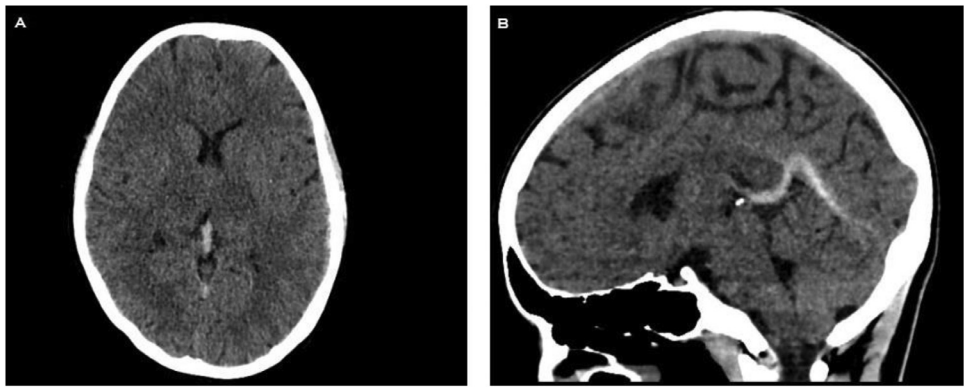

Fig. 1 – (A) Axial noncontrast CT image showing linear hyperdensities in the region of internal cerebral vein and vein of Galen. Bilateral thalami are ill-defined and hypodense with loss of interface between deep gray matter and internal capsule giving the appearance of a disappearing thalami. (B) Sagittal reconstructed images show linear hyperdensity in the region of internal cerebral veins, vein of Galen and straight sinus.

initiated at the earliest to avert complications and possibly death. CVT can strike anyone at any age, however, it is more prevalent in newborns and young people (20-40 years of age). It is also more common in women, with risk factors like pregnancy, postpartum status, and the use of oral contraceptives contributing to the occurrence. Other causes include hematological disorders, hypercoagulable states, certain malignancies, and infective processes (ie, mastoiditis, sinusitis). However, recent case reports are suggesting the COVID-19 vaccination shows a predisposition to venous sinus thrombosis.

We herein present an unusual case of a young female presenting with deep cerebral venous sinus thrombosis after receiving the vaccination against COVID-19.

Case description

A 17-year-old female presented to the emergency department of Bir Hospital, Kathmandu in March 2022 with complaints of acute onset of slurring of speech and decreased level of consciousness. The patient also had a headache for the preceding 7 days which was not completely relieved with over-the-counter medications (ibuprofen and paracetamol). The headache was gradually progressive, throbbing in nature, and not associated with aura, photophobia, phonophobia, fever, nausea, or vomiting. There was no history of ear/nasal discharge, dental infection, cough, sinus pain, recent surgery, or trauma. Further elaboration of history revealed that she had been vaccinated against COVID-19 about 10 days back with Covishield (AstraZeneca). Moreover, there is a history of close contact with family members who tested positive for the COVID-19 Omicron variant, and she developed upper respiratory tract symptoms like cough, loss of smell, and malaise 2 months prior to getting vaccinated however she did not get tested.

At the time of presentation, her National Institute of Health Score Scale (NIHSS) score was 7. Her vital signs appeared to be within normal range. The power in both the upper and lower limb was normal (5/5). The sensory examination was normal and reflexes were unaffected. The plantar response was flexor bilaterally. A thorough cranial nerve and eye examination revealed no abnormalities, and fundoscopy revealed normal findings. The results of the systemic exams were within normal ranges.

Laboratory results showed normal D-dimer levels and factor levels, and while showing negative results for RA factor antibody. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) were raised. The coagulation parameters including factor level, homocysteine, antithrombin III, anti-phospholipid antibody, and anticardiolipin antibody levels were not assessed at the time of admission. Renal function tests, liver function tests, lipid profile testing, and thyroid function tests were normal. The Severe Acute Respiratory Syndrome Coronavirus-2 (SARS CoV-2) virus was not found in a nasopharyngeal swab Polymerase Chain Reaction (PCR). Her serum iron levels were low with peripheral blood smear examination showing microcytic hypochromic anemia.

A non-contrast CT head emergently performed at the Radiology and Imaging department of the same hospital demonstrated linear hyperdensities in the deep venous system (involving internal cerebral veins, the vein of Galen, and straight sinus). It also revealed hypodensities in bilateral thalami with loss of interface between deep gray matter and internal capsule giving the appearance of a disappearing thalamus (Fig. 1). With suspicion of CVT based on the noncontrast CT scan findings, Magnetic resonance imaging (MRI) of the brain with Magnetic resonance venography (MRV) was done the following day.

MRI with MRV study demonstrated altered signal areas in bilateral thalami and left caudate nucleus with mass effect in areas in the thalami causing mild compression of the third ventricle. MRV images demonstrated an absence of flow in the internal cerebral veins, the vein of Galen, and straight sinus which correlated with the findings of the noncontrast CT suggesting thrombosis of the deep venous system. The MR imaging features were thus suggestive of thrombosis of the deep venous (galenic) system with vasogenic edema in bilateral thalami and left caudate nucleus with areas of infarction in the right thalamus (Fig. 2).

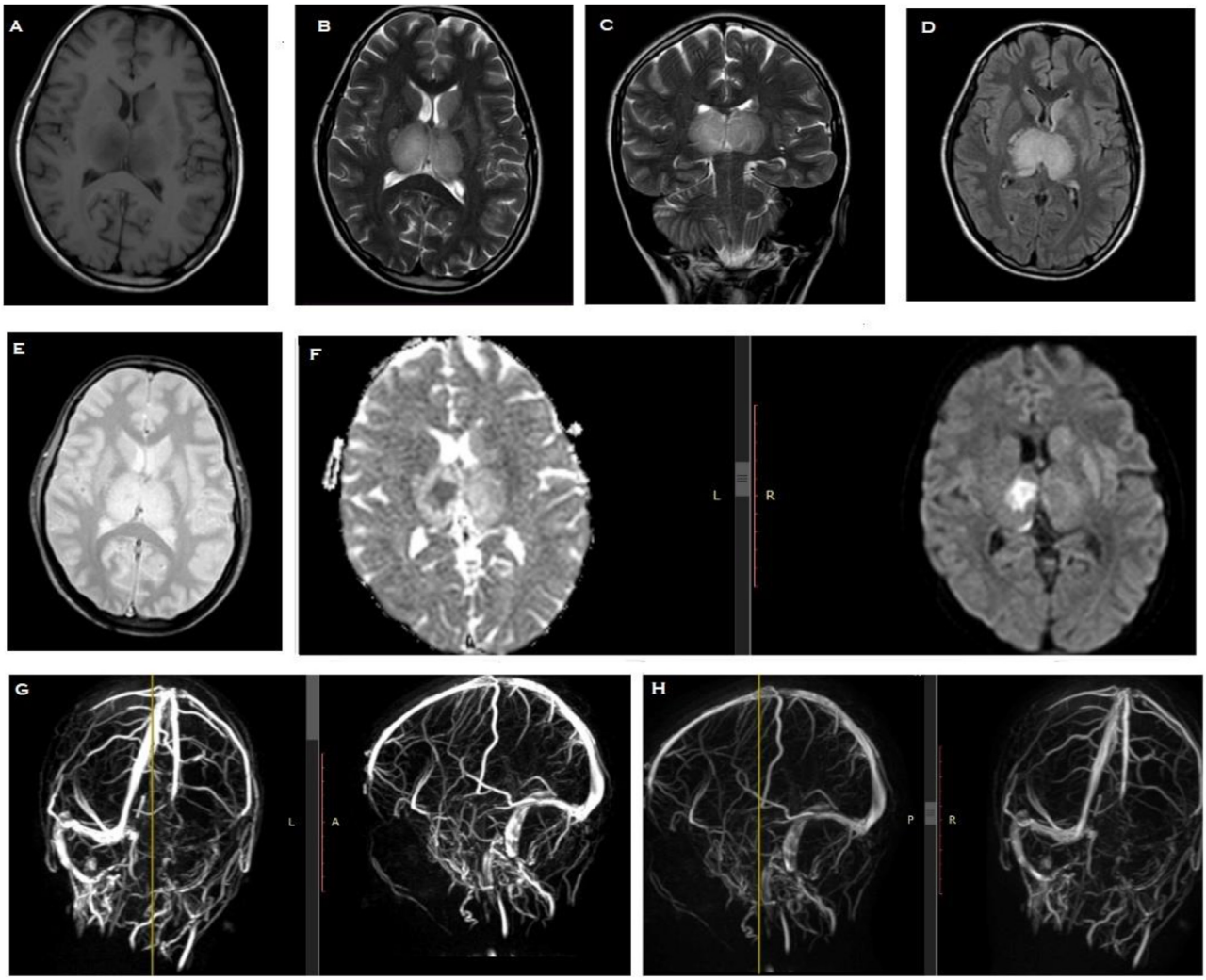

Fig. 2 – (A) Axial T1WI showing hypointensities in bilateral thalami and left caudate nucleus. (B, C) Axial and Coronal T2WI. (D) Axial FLAIR images showing hyperintensities in bilateral thalami and left caudate nucleus. (E) Axial GRE images show no blooming foci in bilateral thalami and left caudate nucleus. (F) Axial DWI/ADC images show restricted diffusion in central portion of right thalamus; however, no diffusion restriction in the left thalamus and left caudate nucleus is present. (G, H) MRV images show nonvisualization of inferior sagittal sinus, internal cerebral veins, vein of Galen and straight sinus along with nonvisualization of left transverse sinus, left sigmoid sinus and left internal jugular vein.

She was admitted to the neurology ward and was treated with subcutaneous low molecular weight (LMW) heparin (Enoxaparin) and monitored with activated partial thromboplastin time (APTT), intravenous dexamethasone, and intravenous pantoprazole. Her symptoms persisted for 2 more days and she was discharged on the third day under oral dabigatran and oral iron. At discharge, the patient was stable with an NIHSS score of 1.

Discussion

Bilateral thalamic infarcts are uncommon, accounting for 22%-35% of all thalamic infarcts [2]. The thalamus usually has dual arterial contributions from both the anterior and posterior intracranial circulations. The thalamotuberal arteries, which arise from the posterior communicating artery via the anterior circulation, supply the anterior thalamus. Thalamoperforators, arterial branches of P1 segments of the posterior cerebral arteries, supply the paramedian thalamic and rostral midbrain areas. The circulatory supply of the paramedian thalami was described by Percheron, with Type I being the most common variant, where a perforating artery arises from each P1 segment.

Occlusion of the distal top of the basilar artery (top of the basilar syndrome) could result in bilateral thalamic infarction from occlusion of the paramedian perforating branches. Basilar artery occlusion presents with infarcts of not only the bilateral thalami but also of the posterior cerebral, superior cerebellar artery, and pontine territories. Similarly, anomalies in both thalami could be explained by an obstruction of the

artery of Percheron which requires careful investigations of the arterial system to confirm the occlusion. Another imaging observation in the artery of Percheron infarct is a V-shaped hyperintense signal intensity on axial DWI and FLAIR images on the pial surface of the midbrain [3].

Involvement of the deep venous system (the internal cerebral veins, vein of Galen, and straight sinus) presents as a hyperintense T2/FLAIR signal involving the thalami and bilateral thalamic infarcts. Bilateral basal ganglia may also be affected by venous hypertension or infarction [2].

Different kinds of viral encephalitis (WNV encephalitis, JEV encephalitis among others) may also show thalamic involvement. Toxoplasmosis infection is seen as multifocal lesions in the basal ganglia, thalami, and gray-white matter junction, however, unlike in DCVT, lesions in the toxoplasmosis show ring and nodular enhancement. Various metabolic disorders can also present with T2/FLAIR hyperintensity in basal ganglia. Wernicke's encephalopathy, caused by thiamine deficiency, can present with T2 hyperintensity in the medial thalamus, periaqueductal area, mammillary bodies, and tectal plate. Wilson disease is a condition characterized by copper accumulation due to a lack of ceruloplasmin and manifests as T2 hyperintensity in the putamen, globus pallidus, caudate nucleus, and ventrolateral portion of the thalamus. Similarly, some tumors like gliomas, neurocytomas, and germinomas can be located in the thalamus. These neoplasms frequently exert a mass effect, cause compression of nearby structures, and can enhance upon contrast administration [1].

Recently, there have been reports of vaccine-induced thrombotic thrombocytopenia (VITT) which is characterized by thrombosis and thrombocytopenia that occurs 4-30 days after vaccination with (AstraZeneca), Ad26.COV2.S (Janssen), BNT162b2 (Pfizer-BioNTech) or mRNA-123 (Moderna). In the *New England Journal of Medicine*, 3 distinct case series of 39 patients with VITT were first documented in association with the ChAdOx1 nCoV-19 vaccination. The vaccine had been given to these patients 5-24 days before they presented. At the time of presentation, all patients had a negative SARS-CoV polymerase-chain-reaction assay [4]. Although the cause of VITT is unknown, almost all patients were found to have high levels of antibodies to platelet factor 4 (PF4)-polyanion complexes as determined by ELISA.

The patient was started on anticoagulation LMW heparin (Enoxaparin) after diagnosis. European guidelines recommend treating acute CVT with therapeutic doses of heparin followed by oral anticoagulants for 3-12 months [5]. Our patient had a significant improvement in her symptoms with anticoagulation in just 3 days whereby she was discharged. On discharge, she was started on oral dabigatran including oral iron supplements for iron deficiency anemia.

Furthermore, thalamic infarction after CVT is uncommon, with only a few cases reported in the literature. Therefore, it is crucial to distinguish between arterial occlusion and venous occlusion because arterial occlusion necessitates rapid thrombolysis while venous occlusion necessitates anticoagulation [6].

Conclusion

There is a multitude of explanations for bilateral thalamic lesions, and understanding related imaging findings can help reach the diagnosis. With recent reports of COVID-19 vaccination-related cases of cerebral venous sinus thrombosis, one should be careful about including the history of COVID-19 vaccination in search of the risk factors.

Availability of data and materials

Data have been presented in the text.

Patient consent

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

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