Images in Clinical Medicine

Bithalamic Infarct — Sequel of Vein of Galen and Internal Cerebral Vein Thrombosis

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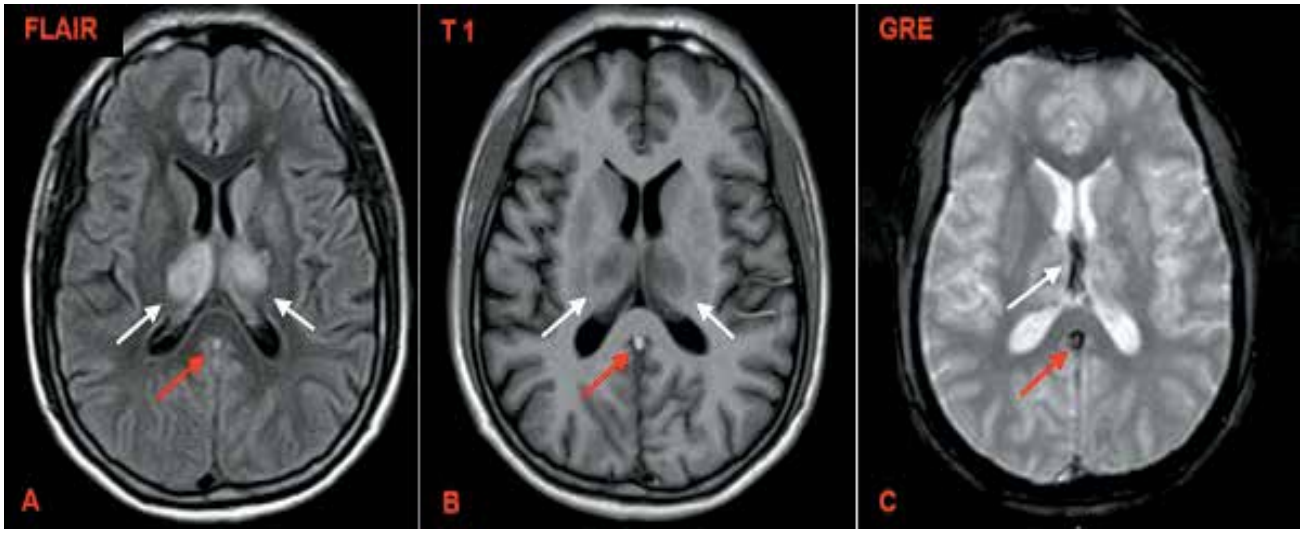

Fig 1. MRI of brain, axial FLAIR image, showing bithalamic infarct (white arrows).The high signal focus posterior to splenium of corpus callosum represents thrombosed vein of Galen (red arrow).

Fig 2. Axial T1 image, showing corresponding low signal in bilateral thalami (white arrows) and high signal focus at the thrombosed vein of Galen (red arrow).

Fig 3. Axial GRE image, showing signal voids in thrombosed internal cerebral veins (white arrow) and vein of Galen (red arrow).

A 46-year-old gentleman presented with frequent vomiting and generalized weakness for one week. He had been diabetic and hypertensive for three years and was on regular oral medications with periodic follow-up. There was no history of fever, headache, blurred vision, seizure, abdominal pain, hematemesis, melena, diarrhea or constipation. On examination, his muscle power was 4/5 in all four limbs and sensation was diminished in both lower limbs. Jerks were normal and plantar response was bilaterally flexor. No neck rigidity was present. GCS score was 15/15. He was hemodynamically stable, with normal pulse, BP and temperature, without any signs of dehydration or other abnormalities in general examination. There was no abnormality in chest, cardiac or abdomen examination. He was advised to have an MRI of brain. Non-contrast T1, T2, FLAIR, GRE and DWI images were obtained in axial, coronal and sagittal planes by PHILIPS Achieva 1.5 Tesla MRI. MRI revealed acute ischemia in bilateral thalami, left caudate nucleus and left putamen, evidenced by diffuse T1 hypointensity and T2/FLAIR hyperintensity, with high signal on DWI. Smaller similar acute ischemic foci were present in bilateral periventricular deep white matter. There was no hemorrhage, mass effect, midline shift or ventriculomegaly. As we were analyzing the images, on GRE we could see 'blooming', i.e., signal void along vein of Galen and internal cerebral veins, indicating thrombosis. We checked and compared with other sequences and there was corresponding hyperintensity, i.e., absence of normal flow void along those veins in T1, T2 and FLAIR images. Bithalamic altered signal

change also occurs in viral encephalitis or metabolic/ hypoxic encephalopathies, but as we already detected the thrombosed veins, we finally concluded with the diagnosis of a case of vein of Galen and internal cerebral vein thrombosis, resulting in acute venous ischemia in bilateral thalami, left caudate nucleus, left putamen and bilateral periventricular deep white matter. Due to these obvious MRI findings, no further contrast was administered and MR venogram (MRV) was not done.

The internal cerebral veins are paired veins, formed near interventricular foramen of Monro. They run parallelly along 3rd ventricle up to posterior to splenium of corpus callosum, where they receive basal veins of Rosenthal and form a short trunk, named vein of Galen, also known as great cerebral vein. These anatomic landmarks are crucial to detect presence of any signal change, i.e., venous thrombosis or malformation. The internal cerebral veins, basal veins of Rosenthal and vein of Galen are together called deep cerebral veins (DCV). Only 1–2 % of strokes are due to venous thrombosis, and DCV thrombosis constitutes about 10% of venous strokes.[1] There is a wide spectrum of causes and risk factors, commonly diabetes, pregnancy, peripartum period, oral contraceptives, extensive trauma, widespread sepsis, vasculitis, collagen diseases, coagulopathies etc. However, in 15% cases, there is no definite causative or predisposing factor.[2] Patients of any age can present with DCV. There is slightly more prevalence in females and the elderly.[3] The clinical presentation of DCV thrombosis is non-specific, and patient can present with short history of headache, nausea, vomiting, aphasia, convulsion, deteriorating consciousness, or even sudden coma and death.[4] MRI and MRV are the best imaging tool for early diagnosis of DCV thrombosis and its sequel.[5] On GRE sequence of MRI, 'blooming', i.e., signal void along DCV indicates thrombosis.[5,6] There is corresponding hyperintensity, i.e., absence of normal flow void along DCV in T1, T2 and FLAIR images.[5,6] MRV clearly delineates presence of thrombus in DCV or elsewhere in cerebral venous sinuses, since non-visualization of DCV in MRV is always abnormal.[7] Digital substraction angiography (DSA) is preserved for intervention and can be considered when MRV findings are equivocal.[3] Bithalamic ischemia is the commonest sequel of DCV thrombosis.[5,6] Parenchymal hemorrhage can be seen in one-third cases, possibly due to continued arterial perfusion in areas of cell death.[5,6] Initial heparin, followed by warfarin to maintain an INR between 2 to 3 is the mainstay of DCV thrombosis treatment. rTPA and endovascular intervention can be considered in acute potentially reversible cases. Mannitol and dexamethasone can be used to reduce cerebral edema. Underlying causes and risk factors of hypercoagulability should be searched and correctable causes should be treated accordingly. Any delay in diagnosis of DCV thrombosis can be lethal and mortality rate increases several fold if anticoagulation is not started initially.[8] We hereby present this case with a view to emphasizing that DCV thrombosis, if present, can be precisely detected in plain MRI of brain, even without any contrast or without MRV. Hence, during analyzing MR images, a high level of suspicion and careful scrutiny at the sites of DCV are necessary whenever there is bithalamic ischemia.

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