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# Imaging findings of an isolated deep cerebral venous thrombosis in the absence of superficial sinus thrombosis

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### *Abstract*

Thrombosis of the deep cerebral venous system in the absence of superficial sinus thrombosis is a very rare disease. The clinical and radiological findings can be diagnostically challenging due to the subtle appearances on computed tomography (CT) scan. Magnetic resonance imaging (MRI) examination is a preferred imaging modality to complement the CT findings for an accurate diagnosis of venous sinus thrombosis. We present a case of this unusual condition which present as unilateral thalamic lesion on CT scan and the role of contrast enhanced MRI with fast spoiled gradient echo (FSPGR) sequence and 3D reconstruction which led to the diagnosis of thrombosis in the deep cerebral venous system.

## INTRODUCTION

Thrombosis of the deep cerebral venous system presenting with bilateral thalamic infarction or oedema in the absence of superficial sinus thrombosis is a rare manifestation.[1,2] The clinical and radiological findings can be diagnostically challenging due to the subtle appearances on brain imaging. We report a case of this unusual condition which present as a mass-like lesion on unenhanced CT scan and the role of contrast enhanced magnetic resonance imaging (MRI) with fast spoiled gradient echo (FSPGR) sequence and 3D reconstruction to confirm the presence of thrombosis in the deep cerebral venous system.

## CASE REPORT

A 37-year-old female tourist from Thailand presented with two days history of acute headache and deterioration of the consciousness level. She was not known to have any underlying medical illness. She was brought to the Trauma and Emergency Department, University Malaya Medical Centre by her accompanying friends when she was found unconscious in the hotel room. Further history could not be obtained from the patient's friends due to language barrier.

Upon arrival, the patient's Glasgow Coma Scale was 8/15. Pupils were 3mm each and reactive bilaterally. Patient was intubated at the resuscitation room. The blood pressure, pulse rate and oxygen saturation were normal. Patient was afebrile. Systemic review of the lungs, cardiovascular and abdominal systems were unremarkable.

The full blood count revealed hypochromic microcytic anaemia with haemoglobin of 69g/L (normal: 120-150), mean cell volume (MCV) of 60 fl (normal: 77-97) and mean cell haemoglobin concentration (MCHC) of 250 g/L (normal: 315-345). Total white cell count was elevated measuring 14.1 g/L $10^9$/L (normal: 4.0 - 10.0) with predominantly neutrophilia of 85.2%. Platelet was also elevated measuring 557 g/L $10^9$/L (normal: 150-400). Renal, liver and coagulation profiles were unremarkable. The cerebrospinal fluid (CSF) and blood cultures showed no growth. The retroviral, hepatitis B, hepatitis C, tuberculosis and connective tissue disease screening were negative.

CT scan of the brain (Siemens Somatom Sensation 16, Germany) revealed an ill-defined hypodense lesion with mild mass effect at the left thalamic region measuring approximately 3.9 x 3.4 cm causing effacement of the third ventricle (Figure 1A). The hypodense lesion also involved the left side of the midbrain inferiorly and left corona radiata superiorly. There was acute hydrocephalus as evidenced by dilatation of the lateral ventricles with periventricular lucencies in keeping with CSF seepage and associated mild cerebral oedema.The fourth ventricle and the basal

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cisterns were preserved. Slight midline shift of 0.3cm to the right was noted. The deep venous system, great vein of Galen and the inferior sagittal sinus appeared hyperdense and distended (Figure 1A). The previously seen hypodense lesion at the left thalamic region showed no enhancement on the following contrast enhanced CT brain, which was performed a few hours later (Figure 1B). There was a filling defect noted within the distended great vein of Galen (Figure 1B). The right thalamus was preserved. Based on these findings, an impression of left thalamic oedema with acute hydrocephalus and possible great vein of Galen thrombosis was made. Differential diagnoses included low-grade tumour or infection with secondary thrombosis of the deep cerebral veins.

Patient was transferred immediately to the Neurosurgery Intensive Care unit for close monitoring and transfused with 2 pint of packed cells. An extraventricular drainage was inserted on the next day of admission.

The brain MRI (3T General Electric, USA) which was performed four days later showed an ill-defined heterogenous non enhancing lesion which was hypointense on T1W and hyperintense on T2W/FLAIR sequences, in the left thalamus measuring 2.9cm (AP) x 2.7cm (W) x 2.6cm (H). Similar signal changes were also seen in the right thalamus, left midbrain and the left side of pons with associated surrounding oedema (Figure 2). There were patchy areas of high signal intensity on diffusion weighted imaging (DWI) at b=1000 with corresponding low signal intensity on apparent diffusion coefficient (ADC) map consistent with diffusion restriction. Areas of blooming artefact were observed within this lesion compatible with blood products. Periventricular hyperintense transependymal CSF seepage was present but the degree of hydrocephalus had improved from earlier CT scan. There was hyperintense signal on T1W with blooming artefacts on gradient echo (GRE) seen within the inferior sagittal sinus, great vein of Galen and internal cerebral veins in FSPGR sequence suspicious of venous thrombosis. The parameters for FSPGR were TR = 6.7ms, TE = 1.9ms, FOV = 31mm, matrix = 256 x 256 and thickness = 1.2mm. These findings correlated with the MR venogram findings (Figure 3). MR angiography with contrast revealed patent anterior and posterior circulation arteries. The patient was diagnosed to have deep cerebral vein thrombosis with haemorrhagic venous infarction of both thalami, midbrain and pons.

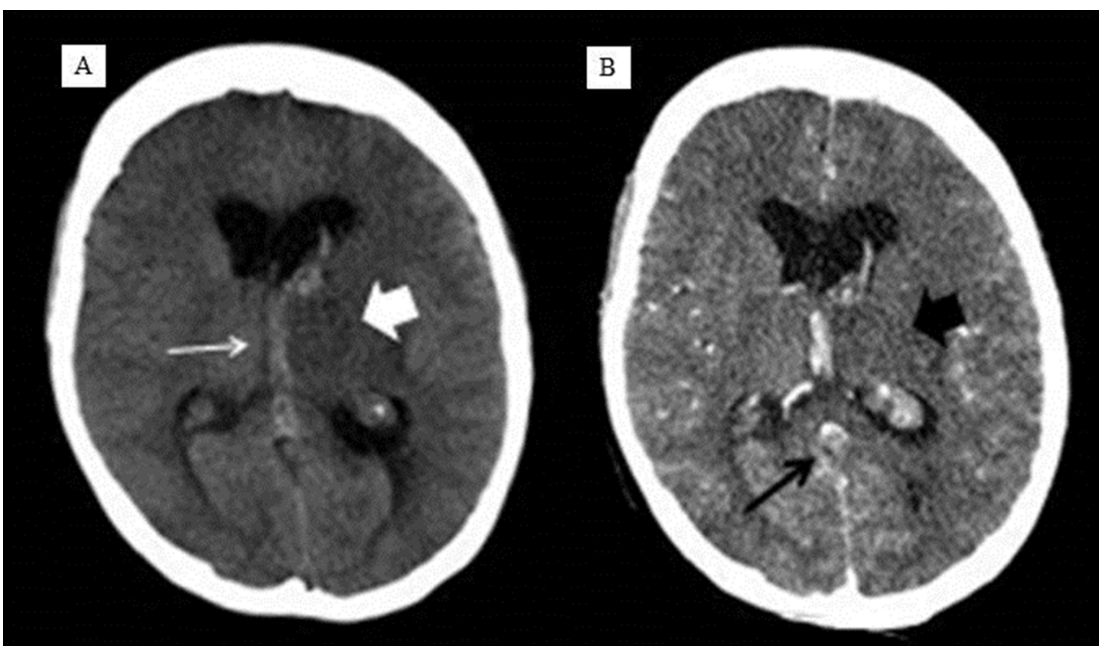


Figure 1. Images of the plain (A) and contrast enhanced (B) CT brain taken at the level of thalamus. The ill-defined hypodense lesion is seen at the left thalamic region (thick white arrow). The internal cerebral vein appears hyperdense and distended (thin white arrow). This hypodense lesion demonstrates no enhancement post contrast administration (thick black arrow). There is small filling defect noted within the distended great vein of Galen (thin black arrow).

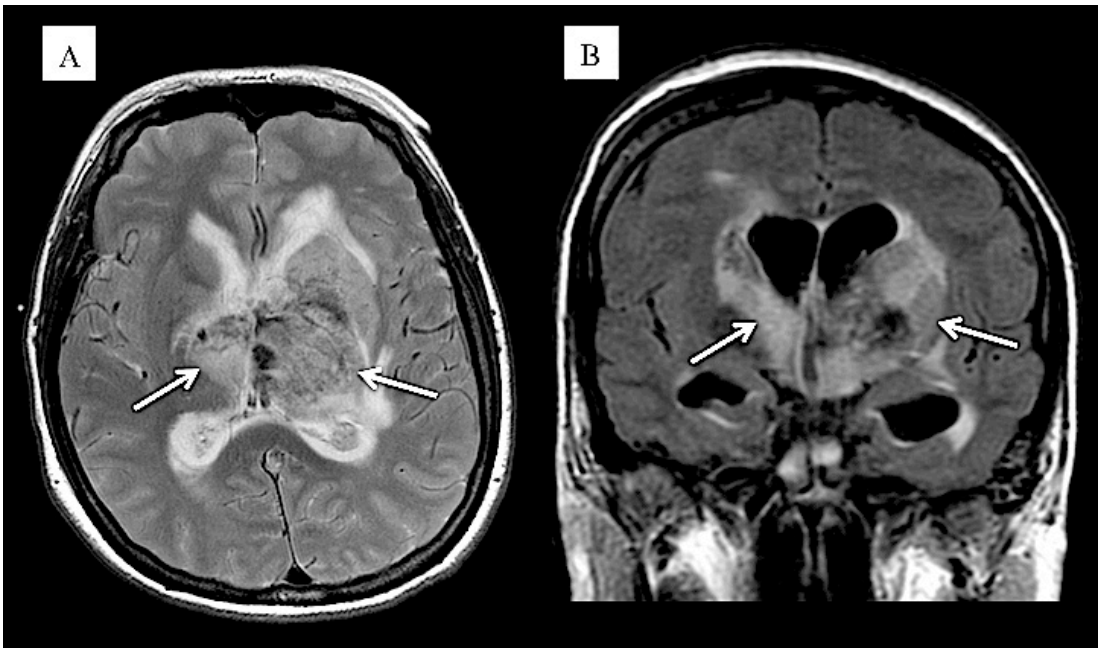


Figure 2. Brain MRI (A) axial T2W (B) coronal T2 FLAIR sequence showing heterogenous mixed signal intensity and cytotoxic oedema within both thalami and left basal ganglia (arrows) with associated mass effect and obstructive hydrocephalus.

Patient was started on subcutaneous Clexane 0.6mg daily as an anticoagulant treatment and was continued with heparin infusion 10 days later. She was ventilated and her general condition remained unchanged for about three weeks of admission until she developed sepsis and hypotension. Clinical and biochemical assessment were consistent with disseminated intravascular coagulopathy (DIVC). She was started on Dopamine infusion and was given DIVC regimes treatment. However, despite treatment and intensive care, she was progressively deteriorated and succumbed to death a week later.

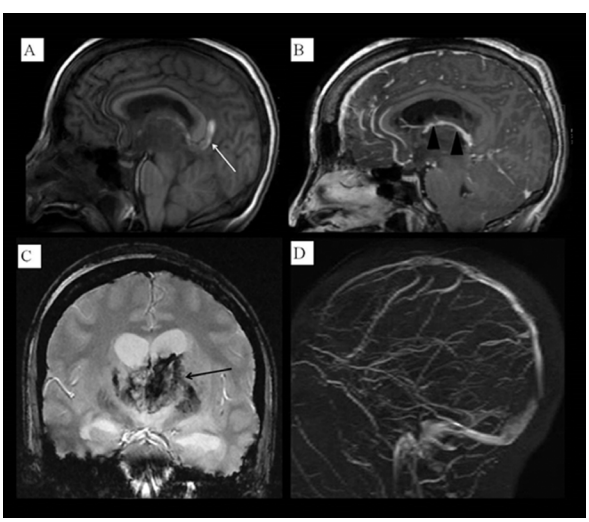


Figure 3. MRI images showing (A) sagittal reconstruction of FSPGR pre Gadolinium, (B) sagittal reconstruction of maximum intensity projection (MIP) FSPGR post Gadolinium, (C) coronal GRE and (D) MRV using TOF reconstruction. Filling defects are seen within the great vein of Galen and internal cerebral veins in FSPGR sequence post Gadolinium administration (black arrow head) and hyperintense signal on T1W pre Gadolinium (white arrow) in keeping with venous thrombosis. GRE image demonstrates areas of blooming artefact in both thalami consistent with haemorrhagic venous infarction (black arrow). MRV TOF showed flow void in the internal cerebral, great vein of Galen and straight sinuses.

## DISCUSSION

Deep cerebral venous system consists of internal cerebral vein, vein of Galen and straight sinus. Thrombosis within the deep venous system usually present as an extension of widespread superficial sinus thrombosis. In an isolated deep vein thrombosis, involvement of thalamus and basal ganglia in the absence of cerebral lobar venous infarction is usually demonstrated.[3] We reviewed the English literature search in Pubmed/ Medline from 1995 to 2015 and found 24 cases of isolated deep cerebral venous thrombosis in case series and reports.[2,4-15] From these cases of isolated deep cerebral venous thrombosis, 15 cases presented initially on CT or MRI imaging as bilateral thalamic infarction, whereby the reminder of the cases presented with unilateral thalamic infarction.

Amongst these reported series, two patients with unilateral deep cerebral venous thrombosis had been diagnosed initially as tumour at the thalamus and basal ganglia on CT/MRI.[9,12] There was also a patient with bilateral deep cerebral thrombosis who also had been misdiagnosed initially as a tumour (other than differential diagnosis of encephalitis and angiitis) at the paramedian thalamus, hypothalamus, internal capsule, midbrain and medial temporal lobe on MRI.[8]

This case presented on plain CT imaging as a hypodense mass-like abnormality at the left thalamic region. Apart from deep cerebral venous thrombosis, other possible differential diagnosis of thalamic oedema or infarction would be arterial occlusion, lymphoma, low grade glioma or infection.[3] One of the features of superior sagittal sinus thrombosis on unenhanced CT scan, will be the 'dense clot sign' that may be seen at the superficial cerebral venous sinuses.[16] In this case, the initial subtle clue would be the hyperdense and distended internal cerebral veins.

Contrasted multi-slice brain CT had revealed a filling defect within the distended great vein of Galen. No "empty delta sign' was appreciated on the superior sagittal sinus. Therefore, these findings could suggest presence of deep venous sinus thrombosis with no evidence of superficial venous sinus involvement. In this case, CT venography protocol should be performed to look for cerebral venous thrombosis. CT venography can provide detailed anatomical depiction of the cerebral venous system superior to the MRI with TOF venography. The accuracy of detection of the cerebral venous thrombosis is at least equivalent to the MRI with TOF venography.[17,18] It has a sensitivity of 75-100% and a specificity of 81-100%. Joo *et al*. in a comparison study reported that multidetector computed tomography venogram (MDCTV) was equal to MRV in its sensitivity for diagnosis of cerebral venous sinus thrombosis.[19]In view of additional radiation dose, repeated CT brain with CT venography protocol was not performed, hence, an MRI examination is recommended at this stage to complement and confirm the diagnosis.

MRI with time-of-flight (TOF) venography and contrast enhanced MRI venography are also common techniques to diagnose cerebral venous thrombosis where contrast enhanced MRI venography provides better depiction of all the venous structures.[18,20]In this case, we utilised thin slice post contrast axial FSPGR sequence with 3D reconstruction to confirm presence of deep cerebral thrombosis and to evaluate the cerebral venous system. The advantages of this FSPGR sequence are better spatial resolution,capability of multiplanar reconstruction postprocessing, can be acquired in a volume acquisition, finer depiction of anatomical relation of the vessels with the surrounding brain structures, thin contiguous images, rapid acquisition time, and the presence of flow-related enhancement. It is also good for functional MRI and showing subtle haemorrhage. In this case, Gadolinium was administrated and 3D reconstruction of FSPGR sequence was performed. The ability of reconstruction into sagittal and coronal views facilitates the exact location of the thrombus in the cerebral venous system. Rapid acquisition time provides feasibility of single breath hold. Complete visualization of cerebral veins and dural sinuses was significantly better accomplished with this technique. TOF venography and contrast enhanced MRI venography in comparison only provide visualization of the veins. Studies have shown that this sequence acquisition with 3D reconstruction and contrast enhancement is superior to MR TOF venography and conventional spin echo technique in the depiction of normal venous structures and the diagnosis of dural sinus thrombosis, and is a potential alternative to DSA.[21,22] The MRI findings of this case helped to confirm the diagnosis of deep cerebral vein thrombosis with haemorrhagic venous infarction of thalami, midbrain and pons.

Hypodense lesions in the thalamic region on CT imaging which do no fit common vascular territories should warrant a search for venous disease.[12] Therefore, in our case, recognition

of hyperdensity at the deep cerebral venous system is helpful to facilitate and suggest the cause of thalamic oedema or infarction seen on an unenhanced CT scan. In this case, an MRI examination was subsequently performed to complement the initial CT scan findings. In our case, we utilized contrast enhanced 3D FSPGR sequence to confirm the diagnosis.

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## DISCLOSURE

Conflict of interest: None

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