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# Cerebral deep venous thrombosis: case report and literature review

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## Case Report

# Cerebral Deep Venous Thrombosis: Case Report and Literature Review

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## Abstract

The case of a 28 years old woman presenting with headache, drowsiness and vomiting of a short duration, is presented. She was diagnosed as deep cerebral venous thrombosis on the basis of MRI findings. Treatment with heparin gave completed recovery.

## Introduction

Acute cerebral deep venous thrombosis (CDVT) of internal cerebral veins, vein of Galen and straight sinus without associated sagittal sinus thrombosis is a very rare disorder and can be associated with poor outcome.[1] It causes infarction of thalami which can be hemorrhagic. The diagnosis of CDVT based on clinical symptoms is difficult but modern technology of MRI is very useful for its diagnosis. Once CDVT is detected, immediate therapy should be started to avoid a devastating outcome.[2] Prognosis can be favorable with prompt treatment.

## Case Report

A 28 years old female presented with complaints of headache for two days and drowsiness and vomiting for one day. Patient delivered a baby two weeks ago and had post partum hemorrhage. Significant past history was hypertension for seven years. Neurological examination was non focal. EEG showed generalized delta waves suggestive of encephalitis. MRI, including sagittal, axial and coronalT1WI, axial T2WI, coronal FLAIR images was performed. MRI showed hyperintense signals on T2 weighted images (figure 1) in the thalami bilaterally. Venous infarction was suspected and immediately gadolinium enhanced MR Venography was performed. This showed non visualization of internal cerebral veins, vein of Galen and straight sinus (figure 2). These findings were consistent with deep cerebral venous thrombosis. Patient was treated with heparin and recovered from her symptoms. She was discharged from the hospital after a few days. At the time of discharge she had fully recovered without any neurological deficits.

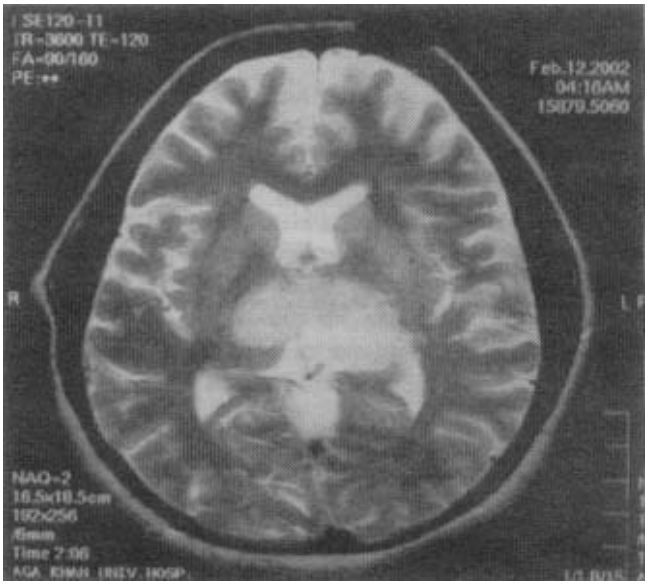


Figure 1. Axial T2 weighted images show abnormal, hyperintense signal involving the thalami bilaterally. There is slightly more marked involvement of left thalamus.

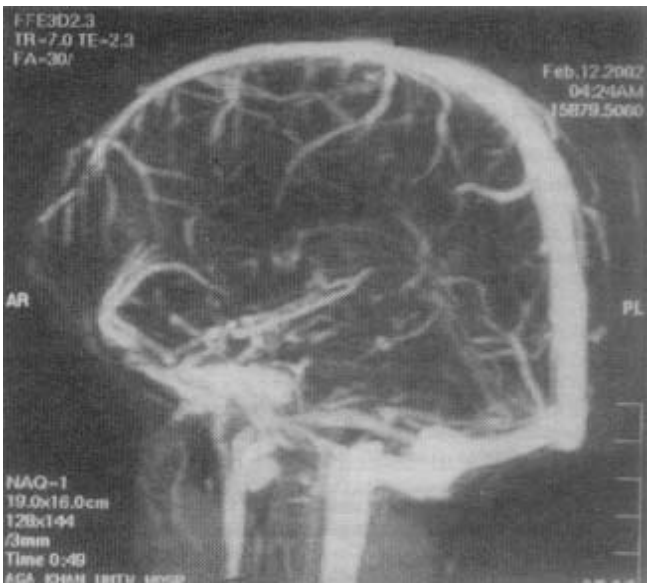


Figure 2. Post gadolinium MR Venograpy demonstrates non visualization of internal cerebral veins, vein of Galen and straight sinus. These findings are consistent with deep cerebral venous thrombosis.

## Discussion

Clinical findings of acute deep cerebral venous thrombosis can be non specific and include, vomiting, change in mental status, alteration of consciousness and coma.[3] Venous infarction of thalami and some times basal ganglia infarcts occur in this condition. Bilateral involvement is typical but unilateral involvement can also be seen.[4] Bithalamic infarcts are usually attributed to thromboembolism of the top of the basilar artery. Apart from the top of the basilar artery syndrome, deep cerebral venous thrombosis should also be considered as a cause of bithalamic infarcts.[5]

Cerebral venous thrombosis may be associated with wide variety of causes.[1] These include use of oral contraceptives, pregnancy, puerperium, dehydration,

hypercoagulable states and infection specially mastoiditis.

The Radiological diagnosis of deep cerebral venous thrombosis may be difficult because characteristic delta sign of superior sagittal sinus thrombosis on contrast enhanced CT scan is not present.[1] Flow related signals in straight sinus on MRI can normally be variable. Hyperdense thrombus can be seen in deep veins on CT scan, which is suggestive of the diagnosis.[1] Magnetic resonance imaging and MR venography are confirmatory.[6] Angiography may still be necessary when diagnosis is not clear.

Diffusion and Perfusion weighted imaging have a role in the diagnosis and assessment of prognosis.[7] Venous obstruction causes venous congestion and disruption of blood brain barrier. Capillary filtration increases leading to cerebral edema. Vasogenic edema was thought to be the type of edema in venous stroke instead of cytotoxic edema as seen in arterial stroke. This concept is reinforced by Diffusion weighted imaging of a patient with venous stroke.[8] In the acute phase value of Apparent Diffusion Coefficient (ADC) is increased. Previous studies of CVT have shown cerebral blood flow reduction well below the threshold of cytotoxic edema formation and decreased ADC within the range of ADC values of pure arterial stroke.[9] In venous thrombosis perfusion of affected brain tissue may still be possible at low flow rates if blood is drained through the collateral pathways. Thus patient still may recover even with significantly decreased ADC value.

Anticoagulation with heparin is the mainstay of treatment even in the presence of hemorrhage.[10] Direct thrombolysis with Urokinase is also useful and lyses of thrombus with persistent recanalization of deep veins can be achieved.[1]

In conclusion early diagnosis of deep venous thrombosis is vital for prompt treatment. When edema or infarcts which may be hemorrhagic, are present in thalami or basal ganglia, one should suspect CDVT. It should also be suspected with non specific symptoms in patients with known risk factors. If detected immediate therapy should be started to avoid a devastating outcome. With prompt treatment prognosis can be favorable as in our case.

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