DEEP CEREBRAL VENOUS THROMBOSIS IN ADULTS-A STUDY OF THREE CASES

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Abstract:
The classic features of thrombosis of the deep cerebral venous system are severe dysfunction of the diencephalon, reflected by coma and disturbances of eye movements and pupillary reflexes, resulting in poor outcome. Deep cerebral venous system thrombosis is an underdiagnosed condition when symptoms are mild, even in the presence of a venous hemorrhagic congestion. The diagnosis should be strongly suspected if the lesion is in the basal ganglia or thalamus, and especially if it is bilateral. Early identification of thrombosis and treatment with anticoagulants improves the outcome.

Keyword:
Internal cerebral vein, vein of galen, deep cerebral venous thrombosis, computed tomography, heparin.

Introduction:
Cerebral venous thrombosis accounts for 10-20% of all young stroke patients.1 Seizures are more common in patients with parenchymal lesions, sagittal sinus and cortical vein thrombosis. Patients with deep CVT often present with a rapidly declining course, altered level of consciousness, and long tract signs. The purpose of this article is to highlight the characteristic CT and MR imaging findings of deep venous system thrombosis that can lead to a rapid diagnosis.2,3,4 Early recognition of thrombosis of deep venous system is very important as prompt treatment with anticoagulants reduces morbidity and mortality.

Case vignette:
We present three cases of deep cerebral venous thrombosis in our institution seen during 2009-2011. We analyzed the clinical presentation and characteristic neuroimaging finding especially CT brain which pointed towards deep venous system thrombosis. Patients with deep CVT often present with a rapidly declining course, altered level of consciousness, and long tract signs. The purpose of this article is to highlight the
characteristic CT and MR imaging findings of deep venous system thrombosis that can lead to a rapid diagnosis. Early recognition of thrombosis of deep venous system is very important as prompt treatment with anticoagulants reduces morbidity and mortality.

Case vignette:
We present three cases of deep cerebral venous thrombosis in our institution seen during 2009-2011. We analyzed the clinical presentation and characteristic neuroimaging finding especially CT brain which pointed towards deep venous system thrombosis and also report the outcome of these patients following early institution of treatment. Case 1 A 22 year old female in her seventh postpartum day presented to the emergency department with history of acute onset of headache with vomiting for 2 days and altered sensorium for one day. On examination patient was afebrile, comatose and flexing her limbs to painful stimuli. Fundus showed bilateral papilledema and brain stem reflexes were intact. Non contrast CT brain showed bilateral thalamus, basal ganglia hypodensity and bilateral internal cerebral veins, vein of galen and straight sinus hyperdensities suggestive of thrombosis. Case 2 A 28 year old male presented with severe headache of one week duration, followed by progressive decline in consciousness and difficulty in moving his right upper and lower limb. On examination he was afebrile, unconscious, had bilateral papilledema, right plantar extensor and no meningeal signs. Non contrast CT Brain showed bilateral thalamus hypodensity interspersed with left thalamic and internal capsule hyperdensity suggestive of hemorrhage.

Hyperdense signal was seen in bilateral internal cerebral vein, vein of galen and straight sinus suggestive of thrombosis.

Case 3 A 40 year old normotensive female presented with severe headache of two days duration with progressive decline in conscious level. On examination patient was comatose, responded to painful stimuli by moving all 4 limbs with bilateral papilledema with intact brain stem reflexes and no neck stiffness. Non contrast CT brain showed bilateral thalamus and right cerebral peduncle hypodensities mixed with hyperdensities and bilateral internal cerebral veins, vein of galen and straight sinus hyperdensity suggestive of thrombosis of deep venous system with hemorrhagic infarcts in thalamus, internal capsule and cerebral peduncle.

CT brain in all three patients showed bilateral basal ganglia and thalamic hypodensity with bilateral internal cerebral vein, vein of Galen and straight sinus hyperdensity suggestive of deep venous system thrombosis. MRI brain with MR venography confirmed the presence of thrombosis of deep venous system. All three patients were treated with intravenous heparin for 10-14 days and showed good recovery except in one who had residual hemiparesis and facial weakness.

Table showing symptoms, signs and CT brain findings of three patients.
<table>
<thead>
<tr>
<th>Case</th>
<th>Symptoms</th>
<th>Signs</th>
<th>CT Brain</th>
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<tbody>
<tr>
<td>1</td>
<td>Acute headache in 7th postpartum day with vomiting for 3 days Altered sensorium - one day</td>
<td>Albino; unconscious, flexing her limbs to painful stimuli, bilateral papilledema, pupils equal and reacting to light, oculocephalic reflex was intact and no neck stiffness</td>
<td>Hypodensity in bilateral thalamus and basal ganglia with hyperdensity in bilateral IC/VG suggestive of thrombosis</td>
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<tr>
<td>2</td>
<td>Headache - 1 week altered sensorium - 2 days pancytopenia of movements of right upper, lower limbs - 2 days</td>
<td>Comatose, bilateral papilledema, Right hemiparesis, Right plantar extensor</td>
<td>Bilateral thalamus hypodensity with hyperdensity in left thalamus and internal capsule suggestive of hemorrhagic infarct. Bilateral IC/VG, SS hyperdensity</td>
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<tr>
<td>3</td>
<td>Headache - 2 days altered sensorium - 1 day</td>
<td>Unconscious, response to painful stimuli by moving all 4 limbs, bilateral papilledema, no meningeal signs</td>
<td>Bilateral thalamus, right cerebral peduncle hemorrhagic infarct. Bilateral IC/VG, SS hyperdensity suggestive of thrombosis</td>
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</table>

Non enhanced CT brain of case 1 showing bilateral thalamus hypodensity (black arrow) in fig 1 and hyperdensity in bilateral internal cerebral veins (black arrow), vein of galen (horizontal large arrow) and straight sinus (vertical arrow) suggestive of thrombosis in fig 2 Figure 3 Figure 4
MRI brain of case 2 showing hyperintense signal in vein of Galen (large arrow) in fig 3 and straight sinus (black arrow) in fig 4 suggestive of thrombosis.

Discussion
Cerebral venous thrombosis (CVT) has a wide spectrum of clinical manifestations that may mimic many other neurological disorders and lead to misdiagnosis. Cerebral venous thrombosis accounts for 10-20% of all young stroke patients. Headache, generally indicative of an increase in intracranial pressure, is the most common symptom in CVT. Thrombosis of superficial venous system is more common than deep CVT. Approximately 16% of patients with CVT have thrombosis of the deep cerebral venous system. Patients with deep CVT often present with a rapidly declining course, altered level of consciousness, and long tract signs. All our patients presented with acute onset of headache with declining conscious level with or without focal neurological deficit. CT brain in all the three patients showed hypodensity of the bilateral thalamus, and basal ganglia, internal capsule due to oedema or infarction secondary to the venous stasis and hyperdense signal in the straight sinus, vein of Galen and the internal cerebral veins which was suggestive of thrombosis. Crawford et al in his series reviewed seven cases of deep CVT. All patients in their series presented with acute onset of headache, declining consciousness (64%) and long tract signs (61%) which was similar to our patients. Unenhanced CT brain in their series demonstrated hyperdensity along the internal cerebral vein in one patient, in the vein of Galen in all four, and in the straight sinus in two, with bilateral thalamic hypodensity in all patients. This computed tomography findings were similar to our patients.

Based on the CT Brain all our three patients were treated with unfractionated heparin for 10-14 days. All patients showed dramatic improvement with a minimal residual hemiparesis in one patient. MRI brain done later showed lack of flow-void in the deep venous system which confirmed the diagnosis. Van der bergh et al in his series of 4 patients of deep CVT noted similar clinical presentation. They were treated only with anticoagulation. All recovered well with two patients who had residual neurological deficits.

Conclusion
We present these cases of deep cerebral venous thrombosis in order to highlight the classical clinical symptomatology and to stress the need for early diagnosis and institution of anticoagulant treatment based on clinical suspicion and classical computed tomography imaging findings which will improve the clinical outcome in these patients.

References
3. Walter M. van den Bergh, Irene van der Schaaf- The spectrum of presentations of venous infarction caused by deep cerebral vein thrombosis. Neurology 2005;65;192


