Computed Tomographic Evidence of an Extensive Thrombosis and Infarction of the Deep Venous System

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**Background:** Massive bilateral infarcts due to deep venous system thrombosis are extremely rare and poorly documented. We present a case with computed tomographic evidence of both thrombosis and infarction of the whole deep venous system.

**Case Description:** A previously healthy 30-year-old woman who had taken oral contraceptives for 3 years complained of recurrent headaches and transient visual obscuration. Three months later, she suddenly became comatose and was found to have papilledema. She rapidly worsened, became decerebrate, and died within 48 hours. Noncontrast computed tomographic scan showed the spontaneous hyperdensity of the thrombosed deep veins and a massive bilateral centrobasilar hypodensity suggestive of a deep venous system infarction.

**Conclusions:** This case shows that cerebral venous thrombosis can masquerade during 3 months as benign intracranial hypertension and then make a dramatic extension to the deep cerebral veins. It illustrates extensive thrombosis of the deep venous system as a possible cause of rapid coma and decerebration and stresses the importance of computed tomographic scan to show both the thrombosed veins and the venous infarct, which allows delineation of the deep venous system territory. *Stroke 1993;24:744–746*

**Keywords** • cerebral embolism and thrombosis • contraceptives, oral • tomography, emission computed

Cerebral venous thrombosis (CVT) is a rare condition affecting most commonly the superior sagittal and lateral sinuses. Isolated intracranial hypertension mimicking benign intracranial hypertension is a frequent and well-established presentation of superior sagittal or lateral sinus thrombosis. Occasional, thrombosis extends to cerebral veins, leading to focal signs, seizures, or disorder of consciousness. Bilateral thrombosis of the deep venous system (DVS) is exceedingly rare and has been documented mainly in pathological studies, although benign cases have recently been reported. We present a case with computed tomographic (CT) evidence of extensive DVS thrombosis and infarction leading to death in 2 days that was preceded by 3 months of isolated intracranial hypertension, most likely related to sinus thrombosis.

**Case Report**

A previously healthy 30-year-old woman who had taken oral contraceptives for 3 years complained of mild recurrent headache and transient visual obscuration. Three months later she was admitted with a rapidly evolving picture of increasing headaches and obtundation for 24 hours. While in the emergency room, her level of consciousness deteriorated; she became acutely unresponsive, with bilateral extensor plantar responses, episodic decerebrate posturing, and Cheyne-Stokes respiration. Fundoscopy revealed a marked bilateral papilledema. The patient was intubated, and an emergency CT scan without contrast showed bilateral symmetrical hypodensity of the deep central areas of the brain, with abnormally increased density along the course of the left caudate veins (Figure 1). Mannitol and hyperventilation were used, but several hours later both pupils became dilated and nonreactive to light. A repeat CT scan without contrast (Figure 2) carried out 17 hours after admission confirmed an extensive bilateral hypodensity of the basal ganglia, thalamus, and adjacent white matter. The CT scan also showed a prominent spontaneous increased density of the internal cerebral veins, veins of Galen, and straight sinus, suggestive of an extensive thrombosis of the DVS. A postcontrast scan showed no change in appearance. The patient’s condition continued to deteriorate, and she died a few hours later.

Tests to rule out systemic diseases and other causes of venous sinus thrombosis including tests for vasculitis, cardiac disease, otologic examination, coagulation profile, platelet function, and complete blood count were negative or normal.

**Discussion**

The clinical presentation of deep venous thrombosis (DVT) is variable. Dramatic cases involving coma,
decerebration, and a rapid downhill course have long been reported.2–5 Autopsy of such cases has shown massive bilateral hemorrhagic infarction of the DVS territory.2 As with our case, the poor clinical picture correlated with massive infarcts that included central reticular formation, both thalami, internal capsules, caudate, and lenticular nuclei, as well as upper midbrain (Figure 1). Recently, however, attention has been drawn to the occurrence of milder cases associated with limited or unilateral DVT and almost complete resolution with minimal or no neurological deficits.4–7

Causes behind such variability may be difficult to identify. However, certain available evidence suggests that massive DVT may be more liable to occur if the venous drainage of DVS territory is already compromised by obliterated collaterals.8,9 Such is the case when DVT is superimposed on a preexisting CVT. Our patient experienced signs of increased intracranial pressure for 3 months. Although she was not examined at that time, it is highly probable that these were related to CVT affecting the superior sagittal or lateral sinus. Her sudden clinical deterioration associated with the mas-
Areas drained by the Deep Venous System of the brain.

Figure 3. Anatomic representation of areas drained by deep venous system (DVS) of the brain. (Modified from Ono et al.17) Deep structures of the brain in the DVS drainage areas include the deep nuclei, consisting of the caudate and lentiform nuclei as well as the claustrum and amygdaloid nucleus (crosshatched areas); both thalami (crosshatched areas); and white matter, including internal and external capsules (stippled areas). Areas drained by DVS are the same as extent of infarct on computed tomographic scan.

sive central infarction resulted from the extension of venous thrombosis to the straight sinus and the DVS.

Although the course of superior sagittal and lateral sinus thrombosis is usually self-limiting, cases such as ours show that an extension to DVS can occur at any time and should possibly be prevented by adequate management of CVT. It is highly relevant to note here that because superior sagittal and lateral sinus thromboses commonly appear as benign intracranial hypertension, such diagnosis should only be made after safely excluding CVT with proper investigation.10

The diagnosis of CVT is best confirmed by angiography or, better yet, by magnetic resonance imaging (MRI), which is a noninvasive modality that can show flowing versus clotted blood.11 CT scan sometimes can detect almost pathognomonic signs of CVT such as the dense triangle sign and empty triangle or delta sign corresponding to new or old superior sagittal sinus thromboses, respectively,12,13 as well as the cord sign indicating thrombosed cortical veins13,14 and the recently described sign of the congested deep subcortical veins.15

The CT scan appearances of DVT as seen in our patient and described in other reports are quite characteristic5-7,16 and correlate well with the angiography and autopsy findings.5 As the present case illustrates, CT showed clear evidence of both venous infarction and thrombosis in the DVS (Figures 1 and 2). The hypodense central region corresponds with infarction and edema in the anatomic area drained by the DVS (Figure 3). This area includes the deep nuclei, the upper midbrain, and the neighboring white matter. The central spontaneous hyperdensity represents the recently thrombosed DVS and choroidplexus (Figure 2). These CT features enable early confident diagnosis of DVT without resorting to the less readily available angiography or MRI; this is especially important in the clinical setting of massive DVT, presenting as acute life-threatening coma, which limits the application of the latter investigations and demands quick diagnosis.

DVT can complicate sinus thrombosis at any time and may be exhibited as a rapidly progressing coma. The CT findings are highly characteristic. Increased awareness of these facts can be expected to improve the prognosis through early recognition and management and possibly prevention by aggressive management of superior sagittal or lateral sinus thrombosis.

References
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