Gülhane Tıp Derg 2011; 53: 66-68

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Deep cerebral venous thrombosis: a case report

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SUMMARY

In light of its rare occurrence as well as variable and nonspecific clinical presentation, deep cerebral venous thrombosis may be misdiagnosed. We here in present a 29-year-old female patient with cerebral venous thrombosis. Early diagnosis of deep cerebral venous thrombosis may be difficult for the clinicians as its clinical symptoms and signs are similar to central nerve system infections or psychiatric disorders. In the differential diagnosis of acute cognitive impairment, the diagnosis of cerebral venous thrombosis should be considered since its early diagnosis and treatment give a satisfactory response.

Key words: *Bilateral basal ganglia lesions, cerebral venous thrombosis, intravenous heparin, magnetic resonance venography*

ÖZET

Derin serebral venöz tromboz: olgu sunumu

Nadir görülmesi, değişken ve nonspesifik klinik prezentasyonu nedeniyle, derin serebral venöz tromboz tanısında yanlışlıklar olabilmektedir. Bu yazıda serebral venöz trombozlu 29 yaşında bir bayan hasta sunulmuştur. Serebral venöz trombozun erken tanısı, bu hastalığın klinik belirti ve bulguları santral sinir sistemi enfeksiyonu ve psikiyatrik bozukluklara benzediği için, zor olabilir. Akut bilişsel bozulmaların ayırıcı tanısında serebral venöz tromboz tanısı dikkate alınmalıdır, çünkü erken tanı ve tedavi ile tatmin edici cevap alınır.

Anahtar kelimeler: *Bilateral bazal ganglion lezyonları, serebral venöz tromboz, intravenöz heparin, manyetik rezonans venografi*

Introduction

Cerebral venous thrombosis (CVT) presents in a wide spectrum of symptoms and etiological factors. Crawford et al. have classified CVT in two major groups as deep cerebral vein thrombosis and dural sinus thrombosis (1). Deep venous veins are responsible for the venous drainage of both thalami, basal ganglia and diencephalone (2). Thrombotic occlusion of the deep cerebral venous system, the straight sinus, and the vein of Galen causes centrally located and generally bilateral thalamic infarcts (3).

Deep CVT is seen less often than dural sinus thrombosis. Because of the variety of clinical symptoms and subacute or fluctuating onset, deep CVT is often misdiagnosed. The radiologic and clinical findings can be nonspecific and diagnostically challenging (3).

According to the published literature, mortality rate is reduced for patients treated with either intravenous heparin or local thrombolytics (13% compared with 48% in untreated patients) These results increase the importance of early and accurate diagnosis in deep CVT (4).

Here we report a case of deep CVT who had confused initial clinical symptomatology, and was early diagnosed with magnetic resonance venography (MRV) and treated successfully with anticoagulants.

Case Report

A 29-year-old female had experienced headaches for five days and received antibiotic treatment with the diagnosis of “sinusitis” in an outpatient clinic. On the first day of treatment, she had had additional complaints such as fatigue, speech disorders, behavioral changes, sleep propensity and urinary incontinence. She was admitted into our emergency service. Her past medical history was unremarkable. Her vital signs were stable. She was lethargic, and cooperation was limited; she also demonstrated disorientation. Her speech was faltering, and she complained of neck

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Date submitted: June 14, 2010 • **Date accepted:** October 27, 2010

stiffness and urinary incontinence. Strength and sensory examinations were normal, as were deep tendon reflexes and plantar responses.

The emergency service brain-computed tomography was unremarkable. For the differential diagnosis, brain magnetic resonance imaging (MRI), a lumbar puncture (LP), and electroencephalography (EEG) were performed. The cerebrospinal fluid examination was normal, yet the EEG showed irregular generalized background activity with slow wave paroxysms of theta-delta frequency prominent on the anterior area of the bilateral hemisphere. The T2-weighted brain MRI (Figure 1) showed hyper-intense signals in the both thalami, anterior half of the lentiform nuclei as well as in the caput and corpus of caudate nuclei bilaterally, which were thought to be associated with deep CVT. The magnetic resonance venogram indicated the absence of flow voids in the right sigmoid sinus, vein of Galen and internal cerebral veins (Figure 2). Tests for protein C and S deficiency, Factor V Leiden mutation, prothrombin (Factor II) G20210A gene mutation, antithrombin III deficiency, lupus anticoagulant and anticardiolipin antibodies, activated protein C resistance, homocystein level, and antinuclear antibodies were all negative.

Initially, an intravenous bolus of 5000 U heparin was injected, followed by a 1000 U/h intravenous infusion given continuously (aPTT was roughly twice the normal value) based on the diagnosis of deep CVT. Coumadin was started while the patient was still receiving the intravenous heparin. The coumadin dosage was set at INR 2-3 times of normal values.

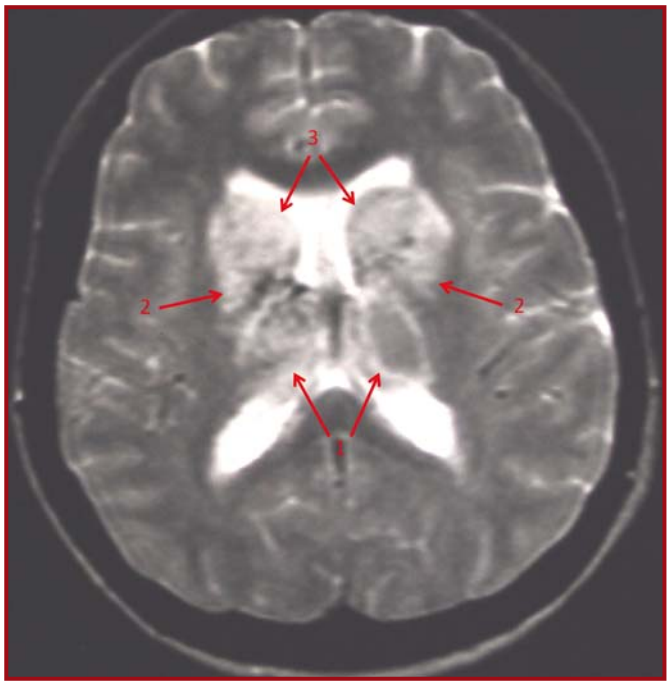

Figure 1. T2-weighted magnetic resonance imaging revealed hyperintense signals in the both thalami (1), anterior half of lentiform nuclei (2), in the caput and corpus of caudate nuclei bilaterally (3)

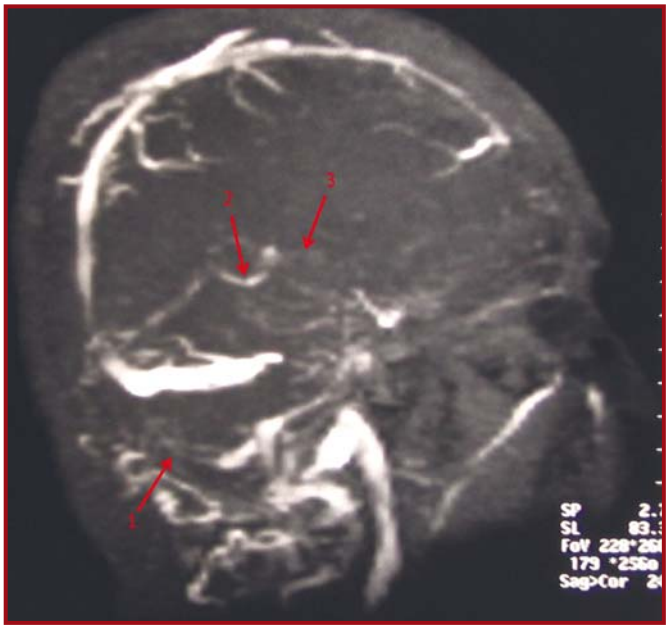

Figure 2. Magnetic resonance venography indicated absence of flow voids in the right sigmoid sinus (1), vein of Galen (1) and internal cerebral veins (3)

Based on the neurological examination and the patient's symptoms, the complaints cleared up within two weeks. The coumadin treatment was discontinued after 3 months. The controls for the neurological examination and MRI studies fell within the normal limits at the 6-month interval.

Discussion

Deep venous systems receive venous inflow from white matter, both thalami, basal ganglia and diencephalone (5). There are a few studies about deep CVT because of approximately 3-8% of cerebral venous thrombosis affect deep venous system (6).

Predisposing factors can be detected in 65-75% of the patients, and 25-35% of the patients are idiopathic (7). CVT may be associated with a wide variety of causes. These include use of oral contraceptives, pregnancy, puerperium, dehydration, hypercoagulable states and infections (especially mastoiditis) (8). In our case, the most common predisposing factors could not be detected and the case was diagnosed as "idiopathic deep CVT" (9).

Clinical symptomatology varies according to the severity of thrombotic process and presence of venous collaterals. Because of thrombosis and endogenous fibrinolysis occurring simultaneously, clinic course shows undulating in most of the patients (60-70%) (6,9,10). CVT is one of the possible diagnoses of headaches as an isolated symptom (11). In our case, headache was

the initial symptom and the patient developed personality changes and lethargy within five days.

Published literature indicates normal CSF examination in 50% of cases, generalized (40-50%) or focal slow (20-25%) wave activity in EEGs and normal brain computed tomography in 25-30% of the cases (9-12). In our case laboratory findings used in the differential diagnosis are consistent with the literature.

It has been reported that MRI and MRV are the best monitoring methods for diagnosis and follow up of CVT (13,14). In the evaluation of bithalamic T2 hyperintensity, MR angiography is a useful adjunct to MR imaging. It helps distinguish between the two vascular causes: deep cerebral vein thrombosis and top of basilar artery occlusion. The confusion with sinus aplasia/hypoplasia (seen as a flow gap on MRV), flow-related artifacts with thrombus and the T2-weighted hypointense signal of deoxyhemoglobin and intracellular methemoglobin with flow void were minimized by using the combination of MRI with MRV (15,16).

Although the use of heparin and oral coumadin treatment has not been determined in a treatment algorithm, their efficacy and safety have been proven by many studies and clinical practice (17). Thus, we used these treatments in our case. Anti-coagulant maintenance treatment was continued for 3 months. Heparin treatment in the acute phase followed by an oral anticoagulant was shown to be safe and efficient to prevent worsening of the disease, recurrence and for quick improvement of neurological symptoms of all treated patients (11).

A meta–analysis revealed a mortality reduction of 14.3% and a risk reduction of 15% in death and dependence with heparin treatment (18). In a study it has been reported that 20% of the patients suffer a recurrence of CVT and 14% a different form of venous thrombosis (19). In our patient, neurological status was stable in a 2-year follow-up period.

Early diagnosis of deep CVT may be difficult for the clinicians as its clinical symptoms and signs are similar to central nerve system infections or psychiatric disorders. In the differential diagnosis of acute cognitive impairment, the diagnosis of CVT should be considered since its early diagnosis and treatment give a satisfactory response.

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