Mutism as a Manifestation of Cerebral Venous Thrombosis

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ABSTRACT

The authors describe a case of a 25-year-old-woman with bilateral thalamic lesions due to thrombosis of the deep cerebral venous system who came into the hospital with mutism. The patient was discharged from the hospital on oral anticoagulant therapy and had a complete recovery. The objective of this study is to describe a rare presentation of cerebral venous thrombosis.

Key words. Intracranial thrombosis; stroke; thalamus; mutism

RESUMO

Mutismo como manifestação de trombose venosa cerebral

Relata-se o caso de uma mulher de 25 anos de idade com lesões talâmicas bilaterais por trombose do sistema venoso cerebral profundo, que se apresentou ao hospital com quadro de mutismo. A paciente teve alta em uso de anticoagulante oral e teve recuperação completa. O objetivo do estudo é descrever uma apresentação rara de trombose venosa cerebral.

Palavras-chave. Trombose intracraniana; acidente vascular cerebral; tálamo; mutismo

INTRODUCTION

Cerebral venous thrombosis (CVT) is a rare cause of cerebral vascular disease, accounting for 0.5% of all strokes.1 The clinical manifestations of CVT are highly variable, mimicking many different neurologic conditions. Although rare, mutism has been previously reported as a manifestation of CVT.2-4 We report the case of a 25-year-old-woman with bilateral thalamic lesions due to CVT presenting with mutism.

CASE REPORT:

A 25-year-old-woman was admitted to the hospital because of sudden mutism and apathy upon awakening in the morning. The patient had been feeling well until 10 days earlier, when she reported a global moderate headache. In the following days the headache became severe, accompanied by nausea and vomiting. Her family denied fever, seizures or signs of a systemic disease.

She had no previous history of disease, except for cholecystitis four months earlier, when she was submitted to a cholecystectomy. She began taking combined oral contraception (levonorgestrel 0.15 mg and ethinyl estradiol 0.03 mg) one month prior to admission. Family history was negative for neurological diseases or thrombotic events.

Neurological examination on admission was remarkable for mutism and hypersomnolence. Comprehension was preserved. There were no focal
neurologic deficits or signs of meningeal irritation. Computed tomographic scanning of the brain, without administration of contrast material, revealed bilateral thalamic hypodensities and a hyperdensity in the straight sinus and internal cerebral veins (figure 1). Anticoagulation with enoxaparin was promptly initiated and it was later converted to warfarin.

The patient was submitted to magnetic resonance venography, which revealed abnormal signal and absence of flow in the left transverse sinus, inferior sagittal sinus and straight sinus (figure 2).

Laboratory evaluation was negative for infections, thrombophilia and collagen disease. The only notable laboratory finding was hypochromic microcytic anemia.

The patient had a gradual recovery in the following days, evolving from mutism to aphonya. A follow-up magnetic resonance imaging ten days after admission revealed T2 prolongation in the left thalamus, with normal signal in the right thalamus. She was discharged from the hospital on warfarin. A follow-up visit two months later revealed a complete neurological recovery.

**Figure 1.** Computed tomographic scanning of the brain, without administration of contrast material shows bilateral thalamic hypodensities and a hyperdensity in the straight sinus and internal cerebral veins.

**Figure 2.** Magnetic resonance venography, shows abnormal signal and absence of flow in the left transverse sinus, inferior sagittal sinus and straight sinus.

**DISCUSSION**

Mutism as the presenting symptom of a disease is more commonly associated with psychiatric disorders. Mutism as a manifestation of CVT is rare, with only a few reports in the literature. The most common manifestations of CVT are headache, focal neurologic deficits, and altered consciousness.

The clinical diagnosis of CVT is often challenging and there is usually a delay of seven days from the onset of symptoms to diagnosis. Our patient presented with progressive headache 10 days prior to mutism, which was an early sign of cerebral venous thrombosis, although not specific.

In most patients with CVT, a prothrombotic factor or a direct cause can be identified and it includes the use of oral contraceptive, acquired or genetic prothrombotic conditions, trauma, inflammatory or infectious diseases. The use of oral contraceptive is the most frequent risk factor for CVT in young women. Two case-control studies have demonstrated that the risk of CVT is higher in women taking oral contraceptive. Our patient began taking combined oral contraceptive approximately 20 days prior to symptom onset.

Thrombosis of the deep cerebral venous system can lead to bilateral thalamic lesions, which may
result in complex clinical syndromes. Alterations in arousal, attention, learning, memory, eye gaze, language, spatial cognition, executive function, motivation, and somatosensory and motor functions have all been described in lesions of the thalamic nuclei. The neuroanatomic location of brain lesions causing akinetic mutism includes the frontal lobes, caudate, putamen, mesencephalon and thalamus. The subcortical circuits connecting the frontal lobes to these subcortical structures might also lead to akinetic mutism.

The differential diagnosis of bilateral thalamic lesions also includes thrombosis of the rostral basilar artery, occlusion of the artery of percheron, infectious diseases and tumors such as glioma or lymphoma. In our patient, initial imaging showed bilateral thalamic hypodensities on head CT. The right thalamic lesion regression seen on the MRI probably represents regression of the edema.

The prognosis of CVT is usually good with full recovery in approximately 80% of patients. Predictors of poor prognosis include male sex, older than 37 years, deep cerebral venous thrombosis, infection of the central nervous system, any malignancy, coma, mental status disorder and intracranial hemorrhage on admission.

**REFERENCES**


