Case Report

Presentation of isolated trolard vein thrombosis with subarachnoid hemorrhage: a case report and review of literature

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ABSTRACT

Cortical vein thrombosis (CVT) is uncommon and life threatening condition. It is usually secondary to the propagation of the clot from dural sinus to cortical vein, but isolated thrombosis of this vein is very rare. Usual symptoms include headache, sensory and motor deficit, seizure, confusion and an increase in intracranial pressure (ICP). Presentation with subarachnoid hemorrhage (SAH) is very rare that has been reported only in the case studies. In this paper, we describe a case of isolated trolard vein thrombosis that present with SAH.

Keywords: Trolard vein, SAH

INTRODUCTION

The clinical presentation of cerebral vein thrombosis is variable. It accompanies with intracranial hypertension, headache, vomiting, seizure and sensory and motor deficit.1 Isolated thrombosis of cortical vein is uncommon and usually is secondary to thrombosis in the dural venous sinuses. Occlusion labbe and trolard veins cause infarction of superior temporal and parietal lobes respectively.1,2 In this paper, we describe a case of isolated cortical vein thrombosis, presented with SAH.

CASE REPORT

A 34 year-old woman was admitted to the neurology service with a thunderclap headache and left hemiparesis. The patient recently started the use of oral contraceptive pill (OCP). Her family history and review of systems were noncontributory. In physical examination, she was oriented to time, place and person. All cranial nerves were intact. Muscle strength was 4/5 over both left upper and lower extremities. In the sensory examination, there was left hemihypesthesia and all DTR was normal.

Figure 1: (A and B): Axial T2 weighted and fluid-attenuated inversion (flair) MRI shows hyper intensity in right front parietal due to edema; (C): Axial T1 weighted shows hyper intensity in right parietal lobe compatible with subarachnoid hemorrhage (SAH); (D): Brain MR venography show defect of trolard vein in leftside.
Fluid-attenuated inversion recovery (FLAIR) and T2 images show increased signal intensity in the right fronto parietal hemisphere compatible with trolard vein thrombosis and T2 weighted imaging, shows focal sulcal hyperintensity at the right parietal convexity in favor of SAH also brain MR Venography show defect of trolard vein in the right side (Figure 1).

All blood tests for hypercoagulability state were normal. Later on the second day of hospitalization, she developed three generalized tonic-clonic seizure that was disoriented and unconscious between them. Levetiracetam 500 mg Intravenous Infusion twice daily for her seizure and anticoagulation with enoxaparin and warfarin for CVT was started. The hemiparesis of left side improved markedly and the patient discharged with warfarin. In follow-up visit, brain imaging show resolution of brain lesion (Figure 2) with complete clinical improvement.

DISCUSSION

Cerebral vein thrombosis is an uncommon and life threatening condition that is fatal if misdiagnosed and don’t treat. The mortality rate range between 5% to 30%. Isolated cortical vein thrombosis is rare. It is usually secondary to the propagation of the clot from dural sinus to cortical vein. The usual clinical manifestations are a headache, seizure, confessional state and sensory and motor focal deficit. The predisposing factor includes birth control pills, pregnancy and postpartum, postoperative state, hypercoagulable conditions, trauma, paranasal infection, dehydration and medications.

In our case, the predisposing cause was birth control pill. The interesting matter in this patient was isolated trolard vein thrombosis without dural sinus vein thrombosis that is very rare. The clinical presentations in our case were headache, hemiparesis, and status epilepticus. A headache in our patient was thunderclap. According to de Bruijn SF et al in a patient with a thunderclap headache, cerebral venous thrombosis (CVT) should be considered if the Cerebrospinal fluid (CSF) pressure is high or if a headache has an unknown cause. Hemiparesis has different etiology in CVT including infarction, parenchymal hemorrhage and brain swelling. It seems that in our patient hemiparesis was due to edema that improves completely after reduction of swelling. The edema is usually cortical and subcortical and not compatible with the special arterial vascular territory. In our patient, Status epilepticus occurred on the second day of hospitalization.

In a study by Masuhr F that assessed the risk of early epileptic seizures in patients with cerebral venous thrombosis, from 194 patients early seizures were occurred in 86 patients (44.3%) and Status epilepticus occurred in 11 patients (12.8%). Digital subtraction angiography (DSA) rarely use for diagnosis due to advance in MR imaging. MRI coupled with MR venogram revealed In T1 weighted imaging, focal sulcal hyperintensity at the right parietal convexity in favor of SAH that surrounded with cortical and subcortical edema without infarction. Presentation with SAH is very rare that has been reported only in the case studies. In a study by Raymond Chang et al report three cases of isolated trolard vein thrombosis with unilateral localized SAH without parenchymal involvement. Anticoagulants have been recommended as the treatment of choice. Conventional or low molecular weight heparin (LMWH) initially then followed by oral anticoagulants for a period of 3–4 months. Eventually, cortical vein thrombosis has different presentation, including cerebral infarction, parenchymal hemorrhage, hemorrhagic infarction, mass-like lesions and rarely SAH, in this condition for accurate diagnosis, we should consider carefully clinical presentation and imaging.

CONCLUSION

We describe a case of isolated trolard vein thrombosis with SAH that dramatically improved after treatment. Although this presentation observed rarely, we must keep in mind it, because early diagnosis and prompt treatment preclude of mortality and morbidity.

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