



Remote Cerebellar Hemorrhage and Iliofemoral Vein Thrombosis After Supratentorial Craniotomy

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Abstract

Introduction Cerebellar hemorrhage following supratentorial craniotomy is rare. Its clinical symptoms are often mild and transient.

Discussion Here, we report a case of cerebellar hemorrhage associated with iliofemoral vein thrombosis as a complication of anterior temporal lobectomy and amygdalohippocampectomy for refractory medial temporal epilepsy.

Keywords Cerebellar hemorrhage · Venous thrombosis · Temporal lobectomy · Supratentorial craniotomy

Introduction

Remote cerebellar hemorrhage (RCH) is a rare and poorly understood complication of supratentorial and spinal neurosurgical procedures. Although its pathophysiology still remains unknown, the substantial loss of CSF volume during surgery, extensive postoperative CSF loss due to

suction drainage, perioperative hypertension, and intraoperative head rotation with obstruction of one jugular vein have all been proposed as a cause of RCH. The typical bleeding pattern includes blood in the sulci of one or both cerebellar hemispheres and vermis facing the tentorium [1].

We report a patient who had RCH and iliofemoral vein thrombosis as a complication of temporal lobectomy and amygdalohippocampectomy for refractory medial temporal epilepsy.

Case Report

A 37-year-old male presented with a seizure disorder starting at age 10 years, characterized by an aura (epigastric sensation, nausea and olfactory hallucinations), followed shortly by a complex partial seizure. He was tried on phenobarbital and phenytoin with unsatisfactory seizure control. A combination of topiramate and carbamazepine led to better control, but they still occurred many times per week especially during sleep. His physical and neurological examinations were normal. Electroencephalography (EEG) demonstrated right interictal temporoparietal slow sharp waves. During his videoelectroencephalographic (VEEG) monitoring, interictal sharp waves and periodic lateralized epileptiform discharges (PLED), and ictal right temporal rhythmic sharp waves were seen. Magnetic resonance (MR) imaging revealed right hippocampal atrophy (Fig. 1a) and increased T2 signal intensity of the right hippocampus (Fig. 1b) consistent with the diagnosis of hippocampal sclerosis.

His preoperative prothrombin time (PT), activated partial thromboplastin time (aPTT), international normalized ratio (INR) and platelet count were within normal limits. He underwent a craniotomy for a right 4.5 cm anterior

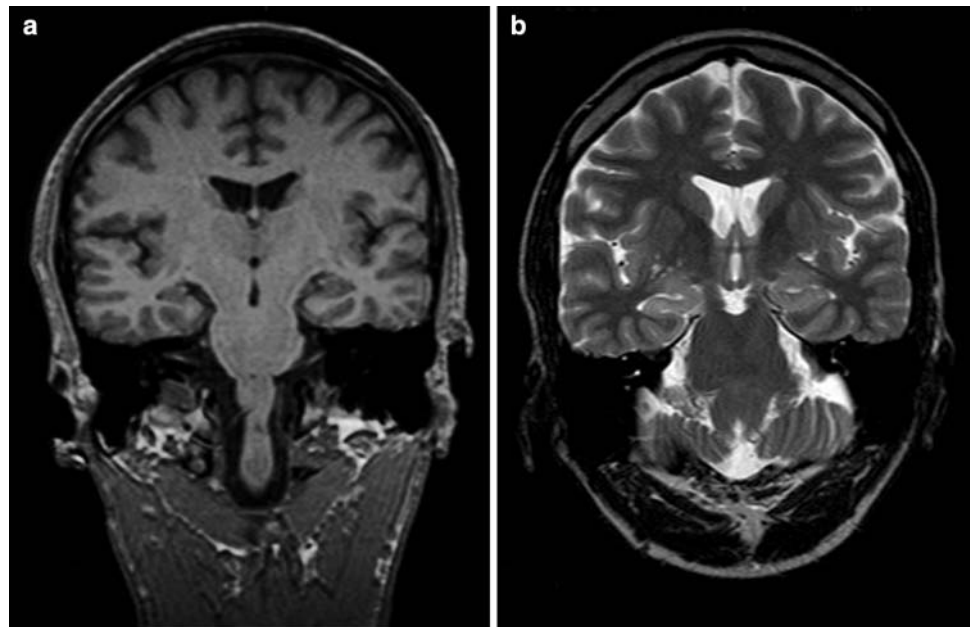
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Fig. 1 Pre-operative MR imaging of the patient. Atrophy (a, coronal 3D T1-weighted (W) gradient-echo image) and hyperintensity (b, coronal T2-W turbo spin-echo (TSE) image) of the right hippocampus suggest hippocampal sclerosis. Please note that increased T2 signal of the right amygdala as well (b)



temporal lobectomy with microscopic amygdalohippocampotomy using the supine position with his head turned to the left. Surgery was completed without complications and subgaleal haemovac drain was placed. About 24 h after the surgery a total amount of 110 ml³ fluid was drained and the drain was removed. The patient had not any evidence of CSF overdrainage. Histological examination of the surgical specimen revealed hippocampal sclerosis.

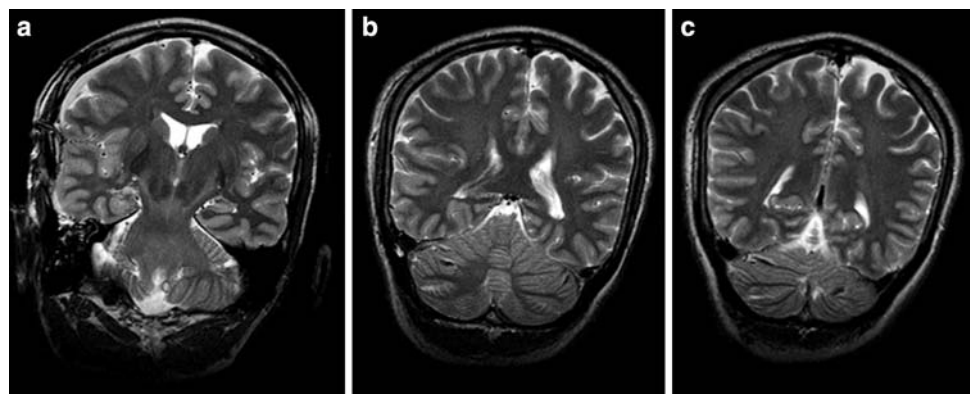
His immediate post-operative MR imaging (5 h after the operation) confirmed total hippocampal resection (Fig. 2a). Additionally, there was a bilateral acute hemorrhage spreading deep into the cerebellar folias, typical of remote cerebellar hemorrhage (Fig. 2b,c). This pattern of hemorrhage has been described as a 'zebra-pattern'. MR angiography of the brain revealed no any other vascular pathology. The patient was stable until the second post-operative day, when he began to complain of left leg pain. At that time his neurological examination was normal, but

his left leg was swollen and red. Doppler ultrasonography was performed and a large thrombus visualized in the iliofemoral vein. No specific treatment was given for the RCH, but he was treated with a low molecular weight heparin and warfarin for his deep vein thrombosis with close monitoring. He was discharged and his neurological examination was normal at 3-month follow up visit.

Discussion

Till date many theories about the pathophysiology of the RCH have been offered. A history of hypertension and transient hypertensive peaks during peri and postoperative period, disturbed blood coagulation, impaired venous drainage due to intraoperative head rotation with obstruction of one jugular vein, large amount of postoperative CSF loss due to suction drainage have all been suggested to

Fig. 2 Immediate post-operative MR imaging of the patient. Coronal T2-W TSE image shows hippocampotomy and local postoperative changes, such as hemorrhagic fluid accumulation (a). Bilaterally, cerebellar acute hemorrhage spreading into the cerebellar folia (b, c)



precipitate RCH [2–4]. Although the mechanism is still unclear, most authors agreed that RCH has a venous origin, because the hematomas are often bilateral almost uniformly located in the upper vermis and in the upper cerebellar folia facing the tentorium where the draining veins of the cerebellar hemispheres are located and are often bilateral [5]. Its incidence is between 0.3% and 4.9% [1] and gender may be a predisposing factor, because 73% of the patients were men [6].

Many authors agree that intraoperative and even more likely postoperative loss of CSF plays a major role at RCH [1]. With the withdrawal of large amount of CSF, the pressure gradient between the supratentorial and infratentorial compartment may change. The negative supratentorial pressure may reduce the pressure within tentorial and supratentorial veins whereas the cerebellar venous pressure may be unchanged. By this suction effect blood may be withdrawn from the infratentorial veins draining the upper cerebellum and vermis. As a result cerebellar capillaries may be traumatized and intracerebellar hemorrhage precipitated.

His second complication was the iliofemoral venous thrombosis. He was 37-year-old, had neither metabolic nor coagulation disorders, as verified by normal platelets count and PT/aPTT values, he was non smoker, was mobilized the first postoperative day and he had no other predisposing factors for deep vein thrombosis. Both complications have a venous origin. We might speculate that a small outflow venous thrombus occluded the cerebellar veins and as a result venous pressure increased and created the venous hemorrhage. This may be a group of patients more inclined to venous thrombosis than others. The most important factor is treatment. Patients with an intracerebral hemorrhage and deep vein thrombosis present a difficult therapeutic dilemma. They risk recurrent fatal pulmonary emboli in the absence of treatment and of recurrent intracerebral hemorrhage if treated with anticoagulants. Previous studies have shown that low dose heparin appears safe in patients with traumatic brain injury [7], and full anticoagulation appears to be safe in patients with intracranial malignancy [8] who develop venous thromboemboli. There is also increasing evidence that treatment with heparin is safe and appropriate in patients with hemorrhagic venous infarction [9]. Another preventative maneuver is inferior vena cava filters in cases where anticoagulation is problematic due to either active bleeding or a high risk of major bleeding.

We treated our patient with a low-molecular weight heparin and warfarin under a close monitoring to avoid pulmonary emboli.

Treatment must balance risk and benefit and must depend on the severity of the cerebellar hemorrhage and resulting complications. Most cases of RCH are benign and can be treated conservatively, but at about 10–15% of patients die or have severe neurological symptoms after RCH [1]. For patients with obstructive hydrocephalus, external ventricular drainage may be beneficial. However, if there is evidence of brain stem compression, decompression should be performed.

In conclusion the mechanism of RCH still remains unknown. Future studies in a large population of patients with RCH may provide more information regarding etiology and treatment of this recently defined condition. Early detection and close monitoring of the patients should help to avoid further aggravation of symptoms.

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