

Isolated Cerebral Cortical Venous Thrombosis: A Challenging Entity

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Abstract

Cerebral venous thrombosis is one of the most challenging diagnoses in neurology. It can present with localized stroke, intracranial hemorrhage and even subarachnoid hemorrhage. Furthermore, the thrombosis can be in different cerebral veins which can cause it specifically to be misdiagnosed with other pure entities like stroke and intracranial hemorrhage. Herein a challenging case of isolated cerebral cortical venous thrombosis in its early stage is presented. It is argued that gradient echo MRI is crucial in diagnosing cortical vein thrombosis but it has to be evaluated along with other MR diagnostic modalities like MR venography and enhanced MRI images.

Keywords: Cerebral Cortical Vein Thrombosis; Gradient Echo MRI

Introduction

Cerebral sinus venous thrombosis (CVT) is a potentially life threatening and though easily treated entity in the field of neurology [1,2]. CVT diagnosis may be challenging due to its various presentations. The incidence of CVT is 3 to 4 cases per million each year [1,2]. The diagnosis of CVT is first guided by the presence of risk factors for thrombosis modulated by physicians high index of suspicion and is further followed by imaging modalities especially those that focus on the venous sinuses. Besides challenging diagnosis due to presentations, the interpretation of MRI findings is also problematic in case of CVT [3]. In order to interpret the MRI findings in the setting of CVT, venous territories are described which may help guiding the diagnosis in case abnormal MRI findings are localized in these areas [3]. However, pure cortical venous thrombosis may present with unusual findings in MRI which may not be interpreted using known venous territory patterns. In this article a case of cortical venous thrombosis is presented and important diagnostic methods are discussed.

Case Report

A 24 year old woman complaining of resolving nonspecific mild headache started 2 days after first child delivery through cesarean section, presented with 3 episodes of generalized tonic-clonic seizures that were stopped by the administration of intravenous phenytoin, eclampsia was ruled out for her. In physical examination there was upward nystagmus without other signs referable to the posterior fossa cerebral dysfunction. She also had left upper extremity weakness that was further attributed to the orthopedic complications occurred due to trauma during seizures. Other neurological examinations including fundoscopy did not reveal any other abnormalities. In brain computed tomography (CT) imaging there was no appreciable findings. Based on the presenting seizures in the post partum stage and the high rate of (CVT) during this period, magnetic resonance imaging (MRI) and venography (MRV) were requested for the patient which revealed hypersignal changes in left high parietal areas in T2 and FLAIR sequences, gyriform enhancement in post contrast study and diffusion facilitation in diffusion weighted imaging (DWI), all of which indicated subacute infarction (Figure 1A). Brain MRV study however showed that the venous sinuses were patent and furthermore it was reported to be normal by consultant radiologist. In gradi-

ent echo (GRE) MRI study; there were hyposignal areas in left high parietal regions with the configuration of cortical veins (Figure 1B). The abnormal signals were compatible with the above mentioned abnormal signal intensities and enhancement in MRI. All together, the findings were concluded to be due to cortical venous thrombosis that was not involving the major venous structures, this had lead to venous infarction and seizures. The patient was treated with anticoagulants, further studies for hypercoagulable states were performed, however all were negative.

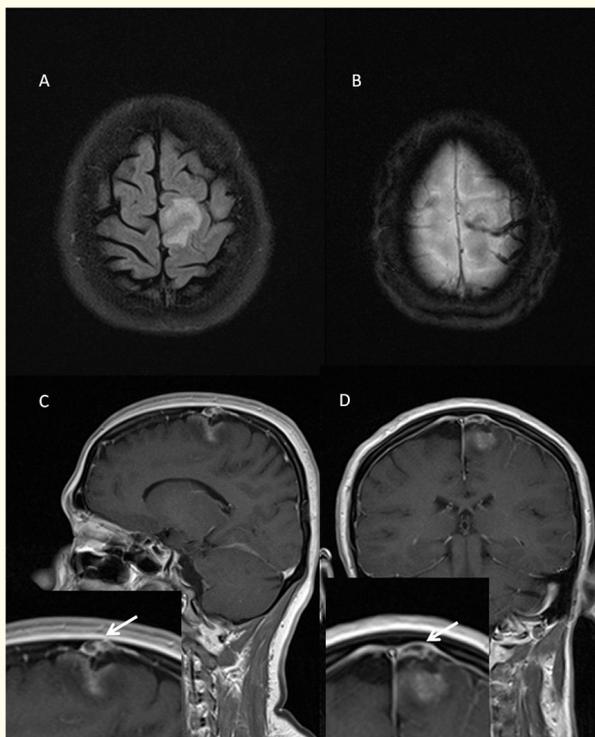


Figure 1: A) Unilateral high parietal signal change in FLAIR. B) GRE signals indicating thrombosis in the configuration of cortical veins. C, D) engorged and defected filling of cortical vein in T1 enhanced MRI images. (arrows in the below magnified images).

Discussion

Isolated cortical vein thrombosis is a rare variety of CVT that is predominantly described in case reports and case series [4,5]. The rarity of this type of CVT increases its diagnostic difficulty and hence results in the delayed initiation of therapy with subsequent devastating consequences.

In the present case, although unilateral high parietal hyperintensities in MRI images were in the territory of superior sagittal sinus drainage, the absence of bilateral findings decreased the probability of CVT in upper venous structures. The only modality capable of diagnosing this disease in this case was GRE pointing to the vital role of this modality in the diagnosis of pure cortical CVT without superior sagittal sinus involvement [6].

The images of the present case were then retrospectively reviewed in search for other signs related to the confirmed left cortical vein thrombosis trying to describe possible helpful findings in other MRI modalities. It was found that there were minor findings in other imaging studies that were difficult to appreciate without access to GRE study (Figure 1C and 1D). These include engorged unilateral cortical vein in the subarachnoid space with filling defect in contrast enhanced frontal view of MR study. Asymmetrical cortical veins originating from two sides of superior sagittal sinus compatible with the engorged vein can also be fairly appreciated in MRV study. The only clue for the guide of diagnosis and search for these tips is physician's high index of suspicion based on clinical grounds and risk factors. The described engorged cortical vein sign in the frontal view of enhanced MRI study is not well appreciated before and is proposed to be helpful besides GRE for isolated cortical vein thrombosis. It is also demonstrated from this case that diffusion facilitation (hypersignal DWI and ADC map) that was interpreted as cerebral infarction, may indicate venous infarct and perhaps venous congestion in case of cortical CVT. Complex diagnostic CVT scenarios are those without bilateral lesions in imaging studies or those early CVTs with no appreciable imaging findings or even those with MRI studies full of nonspecific and old abnormal signal intensities which make it impossible to differentiate abnormal findings related to CVT from nonspecific ones. Functional studies like computerized electroencephalographic analysis of brain symmetry index can be helpful as an adjunct diagnostic modality in these cases [7].

Conclusion

CVT can mimic other ischemic and hemorrhagic entities in neurology like intracranial hemorrhage and subarachnoid hemorrhage. In case of isolated cortical venous thrombosis there is no bilateral lesions, something that is considered to be a crucial clue in the diagnosis of CVT in imaging studies. With its decisive therapy consisting of anticoagulation, which is devastating in cases of pure intracranial hemorrhage, proper early diagnosis of CVT is critical. The presence of CVT as an underlying pathology must be considered in patients with risk factors of thrombosis and in particular in post partum young adults or those under estrogen therapy. Gradient echo MRI must be included in the standard diagnostic protocol for CVT, along with MRV and contrast enhanced MRI. Any simple ischemic or hemorrhagic cerebral entity can have an underlying CVT etiology, therefore any simple cerebral vascular entity must be under clinical and when necessary imaging control to enable the early diagnosis of CVT in patients with risk factors of thrombosis.

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