

PHOTO QUIZ

A 33-year-old woman with severe postpartum headache

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Figure 1: Axial CT¹scan. Figure 2: T2-weighted sequence in MRI². Figure 3: Coronal view of MRV³.

1: Computed tomography, 2: Magnetic resonance imaging, 3: Magnetic resonance venography

Figure 4: T2-weighted sequence of MRI with Gadolinium.

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Case presentation:

Patient was a 33-year-old woman underwent her first time cesarean section combined with spinal epidural anesthesia 5 days before and discharged the day after with good condition. She got severe headache with pain score about 8-9, 2 days after discharge from hospital. Her headache was severe, bilateral, pulsatile and almost likely sudden onset accompanied with nausea that mildly progressed after starting. She went to the hospital, which her delivery was taken; with impression of Post Dural Puncture Headache (PDPH) 10mg IV morphine sulfate was administered totally and sub-

sequently discharged home with relative decreased headache. The day after first headache attack, when she admitted in our Hospital, claimed that could not hold her baby for breast-feeding. In minimental status examination, time disorientation was obvious but orientation to place and person was intact. In motor examination, we found right side hemiparesis and decreased right upper and lower limbs tone. Brain computed tomography (CT) scan and magnetic resonance imaging (MRI) revealed a massive cortico-subcortical hemorrhagic infarction on left parieto-occipital lobes (Figure 1, 2). With attention to significant nonhomogeneous occupying lesion, brain MRI with gadolinium and magnetic resonance venography (MRV) were performed (Figures 3-5).

Figure 5: Flair sequence of MRI with Gadolinium.

What is your diagnosis?



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Diagnosis:

Cortical cerebral vein thrombosis

Cortical cerebral vein thrombosis is a rare but serious emergency manifestation during pregnancy and postpartum that can present by a wide variety of nonspecific symptoms which headache is the most frequent and often the earliest one (1). Headache in the postpartum period can be a manifestation of different diseases including eclampsia, post dural puncture headache (PDPH) and cerebral venous thrombosis (2). The evaluation of postpartum headache should be performed with appropriate method and multidisciplinary approach. Patients without focal neurologic deficit and without findings that are consistent with preeclampsia should be considered initially to have tension-type or migraine headache. Severe headaches resistance to common pain management particularly when accompanied with focal neurologic deficit always require brain imaging (3). During pregnancy, fibrinolytic activity is reduced and on the other hand more coagulation factors are produced, resulting in a physiological hypercoagulatory status in the last trimester of pregnancy and especially the puerperium (4). Furthermore, acute blood loss during delivery, prolonged lying in bed, and postpartum infection sweating, and hyperlipidemia dramatically increase the chance of venous thromboembolism (5). In some studies cesarean section (CS) were mentioned as important risk factor for both stroke and intracranial venous thrombosis. However, it is likely that the dramatic changes occurring after delivery are the most significant factors, intensified by the relatively long time spent in bed after CS and CS itself seems to contribute little to cerebral vein thrombosis (5, 6). Cerebral vein thrombosis induced headache can be of any grade of severity; usually is global, persistent, and has an acute onset. Making correct diagnosis may be so difficult when headache occurs in the absence of any other neurologic sign. Focal or generalized seizures occur in about 40% of the CVT cases. Papilledema, cranial nerve palsies, incomplete hemianopia, hemiparesis, aphasia, various cognitive or psychiatric disturbances and impaired mental status which may fluctuate over days are all count as other clinical manifestations that can be find depend on the location of cerebral lesions and/or the development of raised intracranial pressure. In cases of cortical vein thrombosis, intracranial pressure is not elevated, as it is when the dural venous sinuses are occluded (7). The headache can often be difficult to distinguish from PDPH as it may have a postural

component. Indeed, several cases of cerebral or cortical vein thrombosis have been associated with PDPH, possibly secondary to cerebral vasodilatation after cerebrospinal fluid (CSF) leak and prolonged dehydration (2). Definite diagnosis is based on both clinical manifestations and imaging findings. The diagnosis can be suspected from brain computed tomography (CT) scan, although sometimes it is normal, and be confirmed with conventional angiography or CT angiography or MRI combined with MR angiography of the brain, the latter thought to be the diagnostic procedure of choice. The diagnosis of cortical vein thrombosis is now made by careful examination of the MRV or by the venous phase of the conventional angiogram. Cortical vein thrombosis should be suspected in the situation of multiple hemorrhagic infarctions in one hemisphere without a source of embolism or atherothrombosis (8). Anticoagulation can be effective for the prevention and treatment of extensive cortical vein thrombosis, and should be administered even when thrombosis is complicated by cerebral bleeding. Appropriate anticoagulation is generally not associated with any additional risk of intracranial bleeding (5). In patients with severe involvement of multiple sinuses and veins, anticoagulation alone has limited effects on the lysis of deep cerebral venous thromboses and thrombolytic combined with anticoagulation may be more effective (9). Although early diagnosis and prompt appropriate treatment are strongly associated with a good prognosis, the scarcity and various manifestations of pregnancyassociated cortical vein thrombosis cause that physicians have little understanding of its progression and diagnoses may often be wrong or delayed (5). However, cerebral vein thrombosis remains a potentially life-threatening complication. Markedly impaired consciousness upon presentation, hemorrhage found at the brain CT scan on admission, delay in diagnosis and therefore in the treatment, are poor prognostic factors (7). The lack of required high index of suspicion probably due to the rare incidence of hemorrhagic cortical vein thrombosis, contribute to initial misdiagnosis as a case of intracranial hemorrhage and a delay in the starting the treatment with anticoagulation present in several of the reported cases in the literature, indicating the need for a reminder (10).

Case fate:

MRV (Figure 3) showed decreased signal in left Trolard or superior anastomotic vein. Diagnosis of Cortical cerebral vein thrombosis confirmed based on history, MRI and MRV findings. The patient underwent anticoagulant therapy and discharged 10 days after with warfarin tablet with good condition.



The patient visited after 2 months, had no headache or any neurologic deficit.

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Conflict of interest:

There was no conflict of interest.

Authors' contribution:

All authors contribute in drafting/revising the manuscript, study concept or design, and interpretation of data.

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