Cerebral Vein Thrombosis after Spinal Anaesthesia with Pregnancy

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Thrombosis of cerebral veins or thrombosis of cortical and deep veins that empty into the sinuses can cause serious neurological syndromes. Cerebral vein thrombosis is seen, especially between the ages of 20-35. The predisposing factors in 80% of patients can be determined. Pregnancy, postpartum period and spinal anaesthesia are among the predisposing factors. Diagnosis of the disease is difficult due to the variety of clinical signs and symptoms. Headache is the most common reason for admission to the hospital. Thromboembolic events in pregnancy are an important reason of maternal morbidity and mortality. Most cases of cerebral venous thrombosis in pregnancy occur in the postpartum period. Confusion, convulsions and respiratory arrest occurred in 37-year-old female patient after 3 hours from operation. Cerebral vein thrombosis was diagnosed, owing to laboratory and neuroradiological findings. In this article, we have emphasised the importance of clinical evaluation of pregnant patients with cerebral vein thrombosis after spinal anaesthesia.

Key Words: Pregnancy, cerebral vein thrombosis, spinal anaesthesia

Introduction

Cerebral vein thrombosis (CVT) is a condition that is rarer than arterial occlusive diseases of the brain. It is responsible for 1-2% of all the strokes in adults (1, 2). For diagnosis, non-invasive and highly sensitive diagnostic methods such as magnetic resonance imaging (MRI), unenhanced cranial computed tomography (CT) and unenhanced two-dimensional time-of-flight (TOF) MRI venography and CT venography are used. There are several risk factors for CVT (Table 1). Spinal anaesthesia is also one of the rare risk factors. After lumbar puncture, CVT may occur in 0.2-3.5% of cases (1).

In our study, we present a CVT case that emerged after caesarean section under spinal anaesthesia.

Case Presentation

Caesarean section under spinal anaesthesia was performed in a 37-year-old female patient who was informed regarding spinal anaesthesia and gave written consent preoperatively. In the third postoperative hour, nausea, vomiting, generalised tonic-clonic convulsion and respiratory arrest developed. The patient was intubated and taken to the intensive care unit. The patient had unremarkable history, except for previous caesarean section. Physical examination indicated that she was unconscious, with a Glasgow coma scale of 4, and had pupillary anisocoria. Light reflex was negative for both papillary and other system examinations were normal. Cranial CT result of the patient who was haemodynamically stable revealed oedema in both hemispheres. Anti-oedema treatment and low-molecular-weight heparin treatment were initiated. The patient was extubated in the 24th hour after hospitalisation. The patient with whom complete cooperation could not be established but whose respiratory pattern and haemodynamic parameters were stable was transferred to the neurology clinic. She developed severe headache, right hemiplegia, amnesia and agitations that occurred from time to time during in follow-ups in the clinic. In cranial MR images, lesions that were consistent with cerebral cortical venous infarct were observed at different locations in the left cerebral hemisphere. On the fourth day of hospitalisation, she developed herpes labialis infection. Considering that the infarct area at temporal lobe localisation in the repeat MRI images might be herpes viral encephalitis, anti-viral treatment was administered to the patient. When she was evaluated using MRI and high-resolution TOF MR venography,

Table 1. Predisposing factors for CVT

Local reasons	<ul style="list-style-type: none"> • Sinus trauma • Regional infections (meningitis, sinusitis, otitis, tonsillitis, mastoiditis, etc.) • Spinal anaesthesia
Systemic reasons	<ul style="list-style-type: none"> • Pregnancy and postpartum conditions • Oral contraceptive use • Malignancy (leucemia, lymphoma) • Hypercoagulable states (Factor V Leiden mutation, Protein C and S deficiency, Anti-trombin III deficiency, Prothrombin gene mutation) • Cyanotic congenital heart disease • Cachectic infants • Sickle cell anaemia • Anti-phospholipid antibody syndrome • Rheumatic diseases (Behçet's disease, lupus, sarcoidosis, vasculitis, nephrotic syndrome) • Primary and secondary polycythemia • Thrombocytosis • Paroxysmal nocturnal haemoglobinuria • High body mass index • Dehydration, hyperviscosity
Idiopathic reasons	No reason in 25% of patients
CVT: cerebral venous thrombosis	

cortical venous infarct areas and filling defect in the trolard vein were detected (Figures 1 and 2). Heparin at $18 \text{ U kg}^{-1} \text{ hr}^{-1}$ infusion was administered to the patient who was diagnosed with CVT. The patient showed a good general condition and was discharged with anti-coagulant treatment on the 15th day. The amnesia complaint continued for about 1 week after discharge. In polyclinic control after 3 months, neurologic examination included weakness of the lower leg at the right side of the patient, and she was walking with steppage gait at her foot. Polyclinic controls of the patient performed by the neurology clinic are continuing.

Discussion

CVT is especially seen between the ages of 20 and 35 years. It has been shown that many factors play a role in etiopathogenesis. Predisposing factors can be detected in 80% of patients (Table 1). CVT is difficult to diagnose due to the variety in clinical symptoms and findings. The most common reason for admission due to CVT is headache (80-95%). Depending on the location of thrombosis, focal neurological signs such as hemiparesis, diplopia, aphasia, nausea and changes in consciousness and focal or generalised epileptic seizures that indicate that brain parenchyma is involved in the disease and papillary oedema may also develop in patients (1, 3).

The incidence of thromboembolic disease related to pregnancy is 0.13%, and in developed countries, it constitutes 10% of maternal morbidity and mortality despite treatment.

Figure 1. TOF MR Venography image: Apparently on the left and parietal lobe, multiple cortical thickenings and heterogeneous staining consistent with cortical venous infarcts were observed TOF: time-of-flight; MR: magnetic resonance

Venous thrombosis risk increases by 5-6 times in pregnancy. In addition, most of the symptoms of CVT occur in the postpartum stage in pregnancy. Cortical venous thromboses that cause cerebral infarct or bleeding are complications

Figure 2. Diffusion MR image
MR: magnetic resonance

of pregnancy and generally form in the first postpartum 3 weeks. In addition to these, dural puncture also establishes a ground for CVT. Hereditary thrombophilia such as factor V Leiden mutation, prothrombin gene mutation, absence of anti-thrombin III, absence of protein C and/or protein S and hyperhomocysteinaemia are responsible for 50% of the thromboembolic events in pregnancy (3, 4).

Post-spinal anaesthesia CVT is seen at a rate of 0.1-0.5%, and it is generally related to underlying predisposing factors. Endothelial damage that develops after the tension in cerebral veins during dural puncture and venous stasis that develop due to venous dilatation may cause the development of sinus vein thrombosis. Moreover, lumbar drainage performed during the application of spinal anaesthesia contributes to CVT progress (5).

CVT is seen most commonly (72%) in superior sagittal sinus and second most commonly (70%) in lateral sinuses. Although unenhanced cranial CT is the first choice as an imaging method, MRI and high-resolution TOF MR venography are the most reliable methods for diagnosis and follow-up. Other diagnostic methods are cerebrospinal fluid (CSF) examination, electroencephalography (EEG) (75% abnormal and non-specific changes) and brain scintigraphy with isotope (6).

Classical treatment of CVT includes anti-coagulation and support treatments. For support treatment, anti-convulsants and treatments for decreasing brain oedema are suggested (7). The use of anti-coagulants is controversial due to the bleeding risk of haemorrhagic infarcts. However, despite the increase

in intracranial bleeding risk, heparin is the first choice of anti-coagulant treatment even in the presence of intracranial bleeding. If the patient's condition worsens despite sufficient heparinisation, urokinase can be given as thrombolytic treatment with selective catheterisation (8). In CVT, recanalisation often occurs in the first 4 months after anti-coagulant treatment, and therefore, this treatment must be performed for 3-6 months. Consciousness defect and papilla oedema at the beginning, acute onset, the presence of intracranial bleeding or cerebral infarct, being accompanied by seizures, male sex, being older than 37 years, babyhood, the presence of malignancy, high D-dimer levels and the presence of neurologic deficits were identified as poor prognostic factors (9). In our case, among the bad prognostic factors, acute onset, mental fog, generalised tonic-clonic convulsion, right-sided hemiplegia retrograde dominant in the leg and anterograde amnesia and cerebral infarct were present.

In early periods, when diagnostic difficulty and, accordingly, delayed initiation of treatment were experienced, the mortality rate of CVT reached up to 30-50%, while mortality rates decreased to 6-10% with early diagnosis and anti-coagulant treatment at present (9). In a study investigating the long-term prognoses of patients who underwent CVT, it was detected that 85% of patients did not have neurological disability, 12% had recurrent convulsion, 14% had non-cerebral thrombosis and 11% had CVT again. Patients who had recurrent CVT were seen in the first year. None of the patients had CVT recurrence in their next pregnancy (10).

Uluğ et al. (11) reported CVT that was diagnosed in a 31-year-old female patient 5 days after caesarean section. Yücel et al. (12) diagnosed a 24-year-old male patient with CVT, who had undergone pilonidal sinus surgery with spinal anaesthesia 15 days ago.

In our patient, pregnancy, postpartum period and spinal anaesthesia were the predisposing factors. Despite the existing indicators of poor prognosis, such as deteriorated clinical course in a short period, changes in consciousness, generalised convulsion and respiratory arrest, the symptoms regressed with diagnosis, proper supportive treatment and heparin treatment.

Conclusion

For clinical problems with various neurologic complaints that developed in the postpartum period, especially in high-risk patients such as those who are pregnant and underwent the application of spinal anaesthesia, CVT should be a considerable cause, and it should be considered that delayed diagnosis and treatment can negatively affect prognosis.

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