MANAGEMENT OF CEREBRAL VENOUS SINUS THROMBOSIS IN PREGNANCY: A CASE REPORT

Gilbert Tangkudunga, Ricky Koharb, Corry Mahamac, Mieke Kembuand

sinapsunsrat@gmail.com

aDivision of Interventional Neurology, Department of Neurology, Prof. Dr. R. D. Kandou Hospital, Manado, North Sulawesi, Indonesia;
bDepartment of Neurology, Faculty of Medicine Sam Ratulangi University, Prof. Dr. R. D. Kandou Hospital, Manado, North Sulawesi, Indonesia;
cDivision of Neuropediatrics, Department of Neurology, Prof. Dr. R. D. Kandou Hospital, Manado, North Sulawesi, Indonesia;
dDivision of Neurovascular, Department of Neurology, Prof. Dr. R. D. Kandou Hospital, Manado, North Sulawesi, Indonesia

ABSTRACT
We report a case of cerebral venous sinus thrombosis (CVST) in pregnancy. A 26-years-old female with G2P1A0, 30-31 weeks of gestation without regular antenatal care, presented with seizure and unconsciousness, had been admitted to Obstetrics and Gynecology Department. Patient were diagnosed with eclampsia and HELLP syndrome and managed by their protocols. The further management was done an emergency cesarean section. Hemiparesis on the right side was emerged thereafter. The patient then consulted to Neurology Department. The brain non-contrast CT (NCCT) scan was revealed an intracranial hemorrhagic (sICH score=5) and suspected of CVST. The further management was heparinization with strictly controlled of aPTT. The further evaluation of NCCT scan revealed a resolution of intracranial hemorrhagic followed by improvement on neurological deficit and clinical symptoms of HELLP syndrome. Cerebral angiography was revealed a cortical vein thrombosis. Oral anticoagulant was administered, and modified rankin scale (MRS) after being discharged from the hospital was 0.

Keywords: management, cerebral venous sinus thrombosis, pregnancy

INTRODUCTION
Cerebral venous sinus thrombosis (CVST) is a rare condition, causing stroke with the incidence of 0.5-1% of all stroke. One third cases may be intracerebral hemorrhagic and potential to death. Pregnancy is one of the important etiological factor. During late pregnancy and puerperium, CVST can be an important cause of stroke (1). CVST in pregnancy basically have minimal morbidity and mortality, thus the treatment must be aggressive (2,3).

CASE REPORT
A 26-years-old female with G2P1A0, 30-31 weeks of gestation without regular antenatal care, presented with seizure. Patient referred from district hospital, had been admitted to Obstetrics and Gynecology Department, then consulted to Neurology Department. Seizures had started 3 hours prior to admission. Patient was suffered by headache prior to seizure. After the seizure the patient becomes unconsciousness. Blood pressure measured was 200/120 mmHg. Urinalysis showed proteinuria of +4. Patient then administered initially with magnesium sulfate, methyldopa, nifedipine and dexamethasone. Physical
examination on admission revealed a severe condition. The conscious state was soporous, and she had a GCS of 11/15 (E3V3M5), with blood pressure of 160/100 mmHg (MAP of 120 mmHg). Patient also has edema on her extremities. Laboratory tests on admission showed a leukocytosis and thrombocytopenia. The patient was diagnosed with eclampsia and HELLP syndrome, and further management was done an emergency cesarean section. The patient thereafter treated in ICU on sedation with comatous state. Additional laboratory test showed elevated on liver enzymes, hyperbilirubinemia, hypoalbuminemia, hyperuricemia and elevated creatinine level. A day after cesarean section, hemiparesis on the right side was noted. Her neurological examination revealed comatous state with a GCS of 7 (E2VtM4). Patient was planned of non-contrast brain CT-scan (NCCT) by Neurology Department, and revealed an intracranial hemorrhagic (sICH score=5) and suspected of CVST. The further management was heparinization with strictly controlled of aPTT. The further evaluation of NCCT scan revealed a resolution of intracranial hemorrhagic followed by improvement on neurological deficit and clinical symptoms of HELLP syndrome. Cerebral angiography was revealed a cortical vein thrombosis. Oral anticoagulant was administered, and modified rankin scale after being discharged from the hospital was 0. Three days after discharge from hospital, patient was tested of D-Dimer and showed normal result.

**DISCUSSION**

Pregnancy is a condition of hypercoagulable state (4). The pregnancy-related disorder such as preeclampsia, eclampsia and HELLP syndrome are a group of disorders related to endothelial damage triggered by pregnancy (5). Pregnancy and postpartum period are the high risk phase for many complications, because of redisposition of maternal cardiovascular circulation especially during third trimester and the first four weeks of postpartum (6). Hormonal changes during pregnancy and puerperium increase risk of venous thromboembolism include CVST. The incidence of venous thromboembolism during pregnancy and puerperium has been estimated about 5.5-6 times higher than in general female population (7). CVST occurs in pregnancy with delayed and difficulty on diagnosis or even misdiagnosed, delayed treatment in timely manner; thus potentially threatening the life of pregnancy-related CVST patient. Early and appropriate identification of this case can provide better clinical outcomes. Life threatening complications can be prevented if early detection and treatment done properly. Prompt treatment of CVST with anticoagulants in therapeutic doses leads to reversal of symptoms in most cases. The management of cerebral cortical vein thrombosis usually has good outcomes with anticoagulation therapy. Not all studies reported specifically on outcomes of anticoagulation treatment, because the majority of patients in most studies were treated with intravenous unfractionated heparin (UFH) or low-molecular-weight-
heparin (LMWH) at the time of diagnosis, with eventual use of vitamin K antagonists. The management of cerebral deep vein thrombosis may not recover with anticoagulation therapy only. Anticoagulation alone may not dissolve a large and extensive thrombus. Incomplete recanalization or persistent thrombosis may explain this phenomenon. Recanalization rates may be higher for patients who receive thrombolytic therapy. Many invasive therapeutic procedures have been reported to treat CVST include direct catheter chemical thrombolysis and direct mechanical thrombectomy with or without thrombolysis (8).

![Fig. 1. Brain NCCT before heparinization revealed a hemorrhage on left parietal lobe and cisterna magna, and brain NCCT day 6th after heparinization revealed an absorption of hemorrhage.](image1)

**Fig. 1.** Brain NCCT before heparinization revealed a hemorrhage on left parietal lobe and cisterna magna, and brain NCCT day 6th after heparinization revealed an absorption of hemorrhage.

![Fig. 2. Cerebral angiography revealed a normal internal carotid artery (ICA) injection during arterial phase (A), then on capillary phase (B) revealed a slow flow on one third of middle and posterior territory of middle cerebral artery (MCA). On venous phase (C) revealed a slow flow on middle and posterior sinus.](image2)

**Fig. 2.** Cerebral angiography revealed a normal internal carotid artery (ICA) injection during arterial phase (A), then on capillary phase (B) revealed a slow flow on one third of middle and posterior territory of middle cerebral artery (MCA). On venous phase (C) revealed a slow flow on middle and posterior sinus.

**Statement of Ethics**
The authors have no ethical conflicts to disclose.
Disclosure Statement

The authors have no conflicts of interest to declare

REFERENCE:


