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Cerebral pontine infarction after postpartum hemorrhagic shock

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Abstract

Cerebral pontine infarction is a rare complication of hemorrhagic shock. We report an unconscious 30-year-old woman that was admitted with severe postpartum hemorrhage (PPH). The patient required two surgery to control the bleeding. Focal neurologic deficit was recognized after extubation. Computed tomography (CT) scan showed findings which were consistent with acute right-side pontine infarction. The patient's symptoms improved with antithrombotic therapy and she was discharged on the thirteenth day of hospitalization. A routine stroke rehabilitation program was planned.

Key words: Cerebral infarct, pons, hemorrhagic shock, hypovolemic shock, post-partum hemorrhage.

Introduction

There has been a lack of reports regarding cerebral ischemic events in a case of hemorrhagic shock, particularly in postpartum hemorrhage (PPH). According to the World Health Organization, PPH complicates 10.5% of live births and remains a major cause of maternal morbidity and mortality. (1) Ischemic stroke is a rare (67.1 per 100,000 births) but debilitating complication of PPH which is associated with poorer clinical outcome. It is most commonly caused by embolism and rarely by hemodynamic instability. (2) Brainstem, including pons, is supplied by end arteries arising from posterior circulation without any collaterals. Therefore, these regions are particularly vulnerable to injuries such as hypoxia or thrombotic occlusion. (3) We report a rare case of cerebral pontine infarction after hemorrhagic shock due to PPH.

Case report

An unconscious 30-year-old woman presented to operating room with excessive vaginal bleeding after spontaneous delivery 1 hour ago. The estimated blood loss was 2000 cc. On arrival, she had Glasgow Coma Scale (GCS) of E2M3V3 and vital signs as follows: blood pressure 64/36 mmHg, heart rate 148/minute, respiratory rate 36/minute, peripheral oxygen saturation (SpO2) 95% (room air), and temperature 36.8 °C. Initial physical examination found pallor and cold extremities with prolonged capillary refill time. No uterine contractions were detectable. Routine blood count showed hemoglobin (Hb) level of 4 g/dl. Diagnosis of hemorrhagic shock secondary to post-partum hemorrhage was made. She was given uterotonic agents in conjunction with 2500 cc of crystalloid and 500 cc of colloid, but the bleeding persisted. Emergency subtotal hysterectomy under general anesthesia was immediately performed. Another 1000 cc of blood was lost during the surgery. Intraoperative volume expansion measures included 1000 cc of NaCl 0.9%, 500 cc of colloid, and 500 cc of packed red cells (PRC). Intraoperative blood pressure was maintained by using intravenous (IV) norepinephrine 0.1 μg/kg/minute.

Following surgery, she was admitted to intensive care unit (ICU) and put on a mechanical ventilator (MV). Initial settings were: pressure-controlled

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ventilation mode, respiratory rate 18/minute, positive end-expiratory pressure (PEEP) 5 cmH2O, pressure support (PS) 8 cmH2O, FiO2 40%, and tidal volume (TV) 360 cc. Abnormal hematological profile were as follows: hemoglobin (Hb) 7.2 g/dl, white blood cell (WBC) 16,000/mm^3, prothrombin time (PT) 17.3 seconds, activated partial thromboplastin time (APTT) 58.9 seconds, international normalized ratio (INR) 1.49, and albumin 2.3 g/dl. The patient's postoperative assessment was type IV respiratory failure, hypovolemic shock, sepsis, anemia, and hypoalbuminemia. Ringer lactate 1200 cc/24 hours, PRC 1000 cc, fresh frozen plasma (FFP) 1000 cc, and 4 units of platelets were given to achieve resuscitation target of central venous pressure (CVP) 12-15. The patient was sedated with IV midazolam 0.05 mg/kg/hour. IV norepinephrine 0.2 µg/kg/minute, IV fentanyl 0.05 µg/kg/hour, IV acetaminophen 1 g/8 hours, IV tranexamic acid 1 mg/kg/hour, IV meropenem 1 g/8 hours, IV omeprazole 40 mg/24 hours, and IV vecuronium bromide 2 mg/hour were also administered.

The patient's clinical profile did not improve adequately even on the third day of hospitalization. Abdominal computed tomography (CT) scan revealed active intraabdominal hemorrhage with ascites, thus indicating the requirement of additional intervention to find the source of bleeding. Excision of the left infundibulopelvic ligament and right salpingectomy was performed successfully, in which a pool of 1000 cc of blood and blood clots was found. Postoperatively, the patient's hemodynamic status was stable with blood pressure of 114/76 mmHg and heart rate of 118/minute.

The patient was extubated on the seventh day of hospitalization with GCS of E4M6V5. Subsequent neurological assessment revealed left hemiparesis with motoric strength of 3/5. There was no other neurological abnormality. Non-contrast brain computed tomography (CT) scan demonstrated acute right-side pontine infarction (**Figure 1**). Antithrombotic therapy with dual antiplatelets (oral aspirin 320 mg followed by 80 mg daily, oral clopidogrel 300 mg followed by 75 mg daily) and oral citicoline 500 mg twice daily were then initiated. Her hemiparesis gradually improved and she was shifted to general ward on the tenth day of hospitalization. She was discharged 3 days after with residual symptoms. A routine stroke rehabilitation program was planned to restore normal motoric function.

Discussion

PPH is commonly defined as a blood loss of 500 ml or more within 24 hours after birth. (1) PPH affects 2% of all birth and contributes to 25% of maternal mortality in the world. (4) This patient developed severe PPH (blood loss exceeding 2000 ml) (5) due to uterine atony and was in a state of severe hemorrhagic shock. Hemorrhagic shock results when oxygen delivery is insufficient to meet oxygen demand for aerobic metabolism, with subsequent vital organ ischemia. (6) Takaoka, et al (2004) hypothesized that insufficient brain perfusion triggered by hemorrhagic shock may induce cerebral infarction. (7)

Several protective physiologic adaptations in pregnancy are developed to achieve hemostasis following placental expulsion. However, this mechanism increases woman's risk of thrombosis up to sixfold compared to a non-pregnant woman. Extrinsic and intrinsic factors are involved in activation of clotting factors at the site of injury. The resulting fibrin will stabilize platelet aggregates. In a normal pregnancy, there are increased levels of clotting factors II, V, VII, VIII, IX, X, XII, fibrinogen, von Willebrand factor (vWF), and tissue factor. The increase in clotting factors is a consequence of hormonal changes (particularly estrogen), resulting in a hypercoagulable state. (8) Acute hemorrhage in PPH may lead to trauma-induced coagulopathy (TIC). Activation of coagulation factors in TIC will further increase the risk of thrombosis in this patient. (9) The main causal factor responsible for cerebral infarction in this patient was difficult to ascertain. Isolated hemorrhagic shock is not likely to result in the occurrence of cerebral infarction, (10) hence hypercoagulable state was considered to contribute by precipitating thromboembolic event. This patient's neurological symptoms improved after initiation of anti-thrombotic therapy, suggesting possible thromboembolic origin.

Prothrombin time and activated partial thromboplastin time represent the most common screening tests for coagulation abnormalities, particularly in massive transfusion. The prolongation in PT and aPTT is proportional to the extent of coagulation factor loss and hemodilution. (11) This patient had both prolonged PT and aPTT, which conceivably caused by high-volume resuscitation. Other coagulation markers were not tested due to financial issues. These findings were not consistent with the clinical pathology of ischemic stroke, thus raising the probability that the stroke had ensued even before the first laboratory test was conducted.

As stated by Gillisen, et al (2018), excessive administration of crystalloid fluid may worsen clinical outcome by causing hemodilution, which decrease oxygen-carrying capacity, as well as dilu-

dilutional coagulopathy. (12) Therefore, blood products such as PRC, FFP, and platelets should be utilized concomitantly for massive transfusion protocol in PPH. (13) However, care should also be taken to prevent thromboembolic events induced by multiple large infusions of coagulants such as FFP, platelets, antithrombin III, prothrombin complex, and recombinant factor VIIa. (10) Nevertheless, achieving adequate resuscitation remains crucial in managing patients with hemorrhagic shock. Mechanical ventilation in this patient aimed to prevent secondary brain injury by optimizing oxygen delivery.

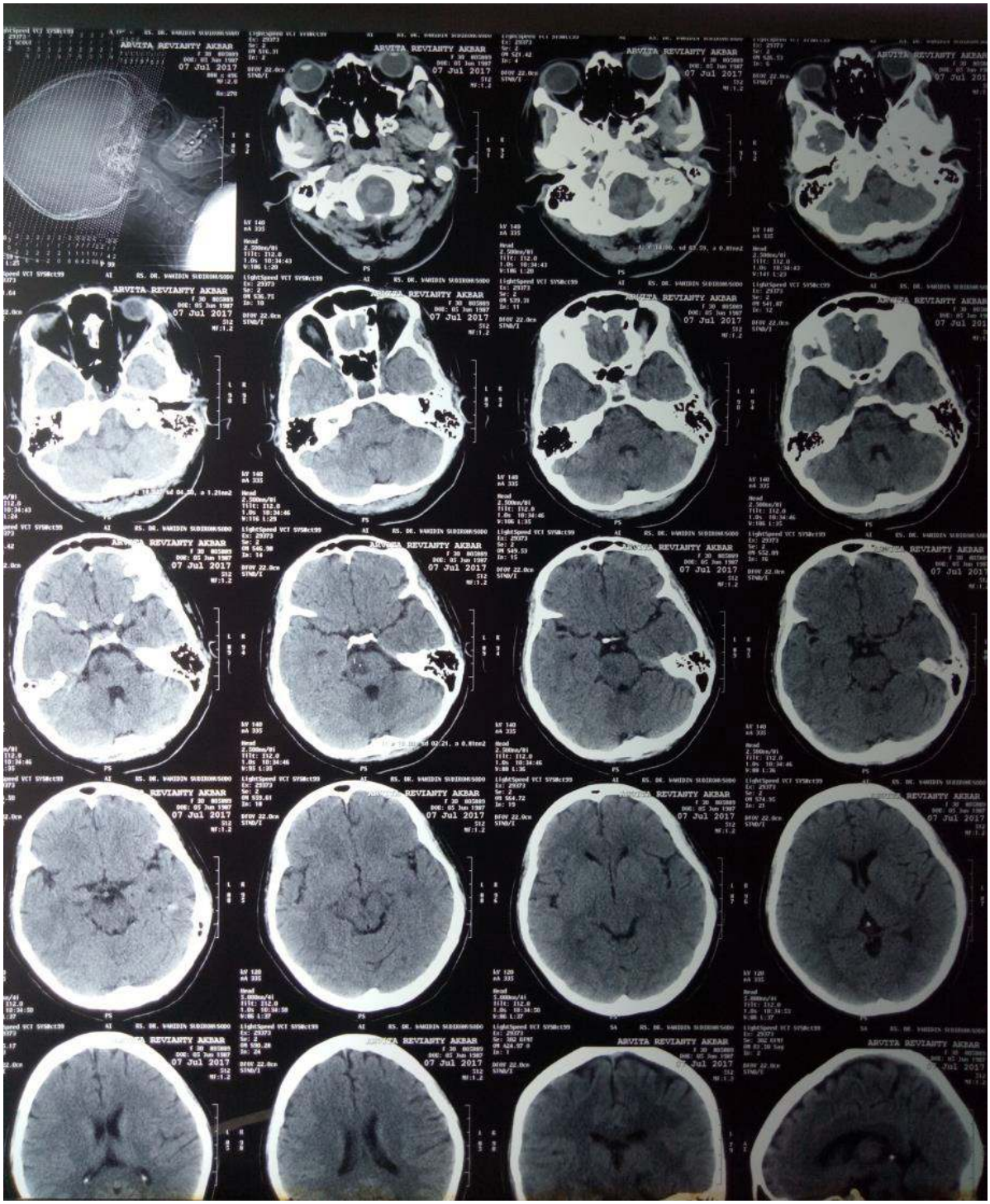
Conclusion

Pregnancy is a state of hypercoagulability that increases the risk of thrombosis. Together with acute and severe hemorrhagic events, they might induce cerebral infarction. Therefore, clinicians should anticipate the incidence of ischemic events to attain early diagnosis and therapy. Optimal management of shock by fluid resuscitation and oxygen therapy is essential to improve clinical outcomes.

Disclosures

The author declares that there is no conflict of interest, and no relevant or material financial interests that relate to this paper.

Figure 1. Non-contrast brain CT scan demonstrating hypodense lesion suggestive of right-side pons infarction



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