Hemisphere cerebral infarction after total laparoscopic hysterectomy in the Trendelenburg position
−A case report−

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Perioperative stroke can lead to mortality or serious disability and usually occurs in patients undergoing cardiac, vascular, or neurologic surgery; it is rare in gynecological surgery. We report the case of a patient who suffered life-threatening cerebral infarction after elective laparoscopic hysterectomy. During the surgery, the patient was placed in the Trendelenburg position. On postoperative day one, the patient was diagnosed with right hemisphere cerebral infarction; brain computed tomographic angiography showed proximal right internal carotid artery occlusion. Decompressive craniectomy was performed to resolve brain swelling, but the patient died 10 days later. **(Anesth Pain Med 2016; 11: 362-365)**

Key Words: Cerebral infarction, Laparoscopic hysterectomy, Stroke, Trendelenburg position.

The occurrence rate of perioperative stroke, which can lead to morbidity and mortality, is 0.05−7.4%. Risk factors include age, previous stroke history, atrial fibrillation, vascular disease and diabetes mellitus [1]. The incidence rate of perioperative stroke varies depending on the type of surgery performed, ranging from 0.8−9.7% in cardiac and carotid surgery to as low as 0.08−0.7% in urological and gynecological surgery [2,3]. Massive hemispheric infarction constitutes 5% of all ischemic stroke and is associated with 50−80% mortality rates [4]. We report the case of a patient who suffered fatal brain edema

due to massive hemispheric cerebral infarction caused by internal carotid artery occlusion after total laparoscopic hysterectomy (TLH) in the Trendelenburg position.

CASE REPORT

A 58-year-old woman (155 cm, 83 kg) diagnosed with cervical cancer was scheduled for TLH with bilateral salpingo-oophorectomy. Her history included hypertension, dyslipidemia, and diabetes mellitus. One month prior, she underwent echocardiography and coronary angiography because of angina symptoms; there were no specific findings. Her vital signs were stable before surgery (blood pressure 148/77 mmHg, heart rate 85 beats/min). Electrocardiogram (ECG) revealed left axis deviation and T wave abnormality. Laboratory test results were as follows: Hemoglobin (Hgb) 13.8 g/dl, hematocrit 40.5%, and platelet count of 297,000. HbA1c and fasting blood glucose were 7.1% and 203 mg/dl, respectively. Pulmonary function testing found an FVC of 2.94 L (104%), FEV1 2.26 L (109%), and FEV1/FVC 77%.

Before the induction of general anesthesia, a central venous catheter was inserted in the right internal jugular vein and ECG and pulse oximetry monitoring were performed. After preoxygenation, anesthesia was induced with propofol and rocuronium and then maintained with desflurane (5−6%) and remifentanil. An arterial catheter was inserted in the right radial artery for continuous blood pressure monitoring and blood sampling of arterial blood gas analysis (ABGA). During surgery, the patient was placed under lithotomy and in the Trendelenburg (20°) position; 12 mmHg of intraperitoneal pressure with CO_2 gas was applied for laparoscopic procedure. Before placement in the Trendelenburg position, central venous pressure (CVP) was 18 cmH$_2$O; forty minutes after the change

in position, CVP was increased to 28 cmH2O. During the surgery, mean arterial blood pressure was maintained above 70 mmHg. Initial ABGA results at FiO2 0.5 were pH 7.47, PO2 108 mmHg, PCO2 33 mmHg, and Hgb 10.2 g/dl. ABGA results two hours later at FiO2 0.5 were pH 7.33, PO2 87 mmHg, PCO2 46 mmHg, and SpO2 96%. Positive end expiratory pressure (PEEP) of 5 cmH20 was applied. However, arterial oxygenation was not improved until the end of surgery (PO2 82–89 mmHg). Therefore, the patient was not extubated and was sent to the intensive care unit (ICU) for ventilatory support. The total operation time was three hours and thirty minutes and total anesthesia time was four hours. A total of 3,400 ml of fluid was infused and patient urine output was 150 ml.

In the ICU the patient was sedated with dexmedetomidine and placed on ventilatory support. The ventilator was set to simultaneous intermittent mandatory volume mode with FiO2 0.6, respiratory rate 12, tidal volume 450 ml, and PEEP of 5 cmH2O. The pupil reflex of the patient was normal in both eyes. Initial ABGA results were pH 7.287, PO2 72.7 mmHg, and PCO2 41.1 mmHg. Three hours later, blood pressure dropped to 82/40 mmHg, and the CVP value was 9 cmH2O; 500 ml of normal saline and 0.1 µg/kg/min of norepinephrine were infused. Six hours later, as blood pressure normalized, norepinephrine infusion was stopped. Ten hours later, ABGA results at FiO2 0.4 were pH 7.427, PO2 129.5 mmHg, PCO2 31.6 mmHg, and SaO2 98.7%. Fourteen hours later, pupil dilatation was observed in the left eye. Brain CT angiography revealed an infarction in the right hemisphere with proximal right internal carotid artery occlusion (Figs. 1-3). Emergency decompressive craniectomy was performed to relieve intracranial

pressure as brain swelling worsened. After craniectomy, the patient was in a comatose mental state and did not respond to conservative management. On postoperative day 10, the patient died from continued brain swelling.

DISCUSSION

Stroke can appear in two forms, ischemic and hemorrhagic, and ischemic stroke has three possible mechanisms; embolism of cardiac or aortic origin, intracranial or extracranial artery thrombosis and systemic hypoperfusion [5]. Coronary artery bypass graft (CABG) is associated with a 1.4–3.8% risk of stroke, while that of general surgery is 0.08–0.7% [2]. Factors
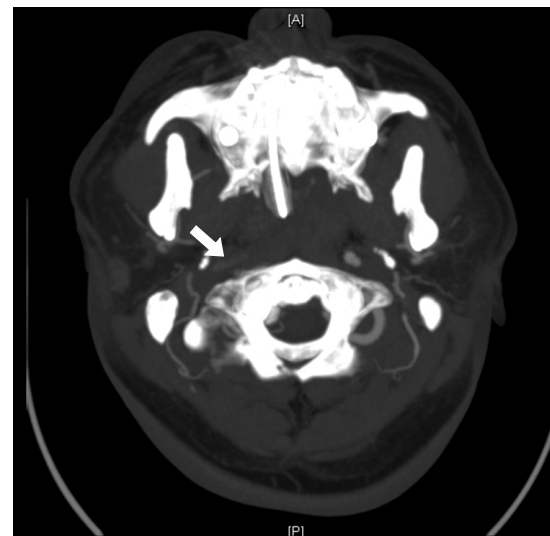
Fig. 2. The right internal carotid artery was not seen at the C2 cervical vertebra level (arrow).
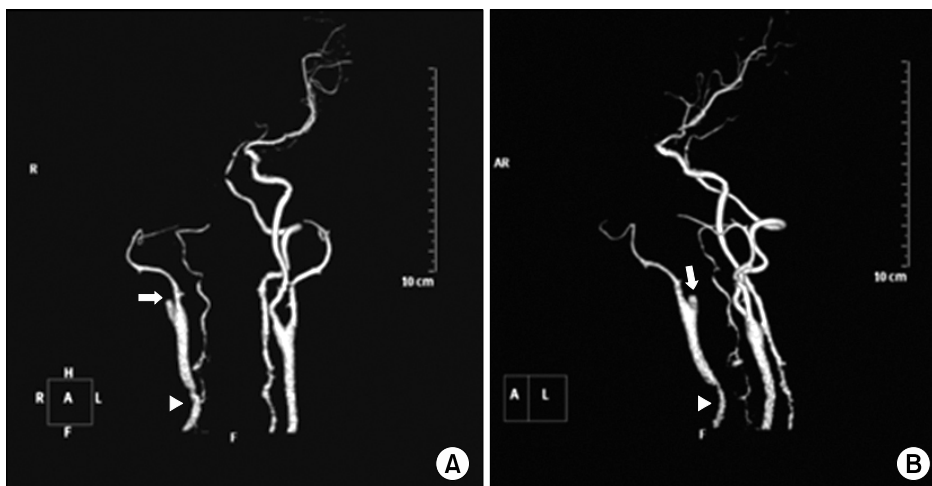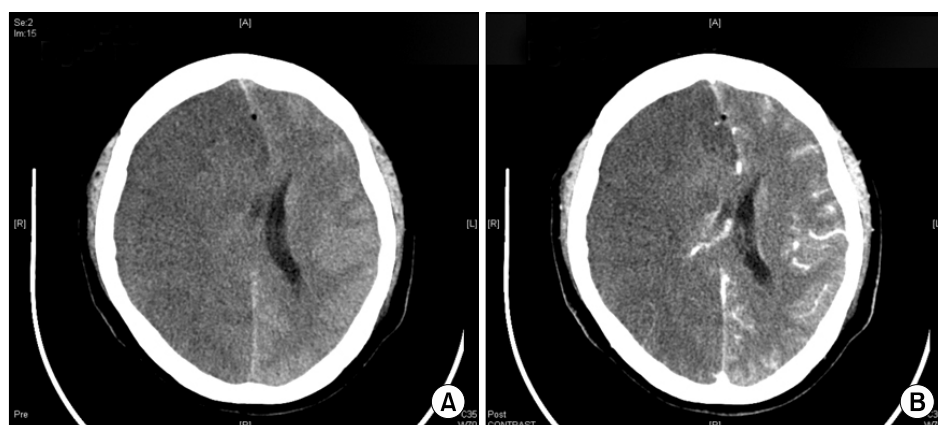
Fig. 1. Computed tomography angiography showing stenosis of the right proximal common carotid artery (arrow head) and total occlusion of the right internal carotid artery at the proximal common carotid artery bifurcation with poorly visualized right anterior and middle cerebral artery (arrow).

Fig. 3. Pre and postcontrast CT showing right cerebral hemisphere infarction with midline shift.

such as general anesthesia, dehydration, bed rest, and stasis in the postoperative period may worsen surgery-induced hypercoaguability and increase perioperative thrombogenic event and stroke risk [2]. After CABG surgery, 45% of embolic stroke occurs during the first postoperative day and the remaining 55% within two days after uneventful anesthesia [2]. Approximately 6% of stroke occurs during surgery. Thus, postoperative events seem to be more important than intraoperative mechanisms.

Acute stroke impairs cerebral autoregulation and cerebral blood flow is proportional to perfusion pressure. Therefore, the injured brain is prone to damage even without severe hypotension. Before elective surgery, 70% of patients with symptomatic artery stenosis should undergo revascularization such as endarterectomy. However, less than 50% of patients with stenosis undergo carotid endarterectomy [6].

Infarction size is a major determinant of life threatening brain edema. Infarct size greater than 50% of the middle cerebral artery (MCA) territory is a key risk factor of life-threatening brain edema [7,8]. Large MCA infarcts lead to life-threatening edema several days after the onset of stroke, increasing the mortality rate to 78% [4]. According to Kasner et al. [9] predictors of fatal brain edema in large MCA infarction patients include hypertension, congestive heart failure, elevated white blood cell count, early CT hypodensity exceeding 50% of the MCA territory, and CT evidence of involvement of additional vascular territories. Fatal outcomes are expected if there is MCA occlusion or poor collateral blood flow. The cause of death in our patient was compartmental shift and cerebral herniation due to brain edema. It can be difficult to predict life-threatening brain edema in MCA infarction patients and to determine the optimal timing of decompressive surgery. However, the size of the ischemic area may be a factor in clinical decision making [7]. In this case,

the infract spanned the majority of the brain hemisphere, so a high risk of mortality was expected. The patient underwent decompressive surgery to reduce edema caused by cerebral infarction. However, the hemispheric infarction was too large, and thus the patient did not recover.

The timing of stroke occurrence may also affect outcomes. In nonsurgical fields, the mortality rate is 12.6%, while postoperative stroke mortality increases by 26% [10]. The primary causes of mortality in major stroke are delayed diagnosis, cerebral edema and intracranial hypertension [1]. The pathophysiology of stroke is related to inflammation. Inflammatory events outside of the brain play a pivotal role in stroke susceptibility. Inflammatory responses (e.g., expression of IL-1, IL-6, TNFα, C-reactive protein) to surgery and stroke can synergistically amplify plaque rupture, leukocytosis, and platelet activation, leading to increases in stroke mortality of up to 25% [1].

Head-down positioning during surgery increases CVP, interferes with venous return and increases hydrostatic pressure in brain. Steep Trendelenburg position (30°) reportedly increases intracranial pressure (ICP) from 8.8 to 13.3 mmHg [12], while cerebral edema induces an increase in cerebrovascular resistance. Reductions in cerebral blood flow (CBF) may cause impaired perfusion within brain tissue, leading to impaired oxygenation. Arterial pressure also increases, and if the increase in arterial pressure is greater than that of CVP, CBF and cerebral perfusion pressure (CPP) increase. Therefore, it is difficult to predict how such effects influence CBF and cerebrovascular resistance. Steep Trendelenburg positioning (40°) and pneumoperitoneum can cause intracranial hypertension and reduced cerebral tissue oxygen saturation in elderly patients or patients with increased ICP. However, CPP and continuous regional cerebral tissue oxygen saturation may be maintained in these groups [13]. Park et al. [14] reported that

Trendelenburg positioning at 30° slightly increased rSO_2 and did not cause cerebral ischemia. However, if the patient has both systemic hypertension and cerebrovascular disease, cerebral homeostasis may easily break up. Steep Trendelenburg positioning decreases the cardiac index while increasing pulmonary arterial pressure and CVP. After general anesthesia, this positioning may lead to facial and upper airway edema [15]. Even if steep Trendelenburg position does not directly cause cerebral ischemia, it may facilitate the occurrence of stroke if there is vessel stenosis or plaque presence. In our case, the patient had stenosis in the right common carotid artery, so we suspect a plaque was present. Factors such as general anesthesia, Trendelenburg positioning, and increased intrathoracic pressure might have facilitated plaque rupture and formation of a large thrombus that might have caused occlusion of the internal carotid artery. Prompt detection of perioperative stroke is required for early treatment. Therefore, it is important to monitor brain oxygenation during the intraoperative period. Assuming the cerebral metabolic rate is constant, mixed venous oxygen saturation may be an indicator of CBF. CPP below 70 mmHg may rapidly decrease jugular venous bulb saturation. Jugular venous oxygen saturation may be measured via jugular bulb oxygen saturation, but non-invasive regional cerebral saturation monitoring, such as near infrared spectroscopy, which displays balance between cerebral oxygen supply and demand in the frontal lobe, has been used recently. The exact timing of stroke occurrence in this patient remains unclear, as she was under sedation during her stay in the ICU. However, both eyes were isocoric when she first entered the ICU, suggesting that the stroke occurred during the first day after surgery.

In conclusion, even if cerebral perfusion pressure and regional cerebral tissue oxygen saturation are maintained during Trendelenburg positioning, this position may increase the risk of stroke in patients with carotid artery stenosis. Neuromonitoring to detect the blood supply to the brain should also have been applied during the intraoperative period.

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