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Delayed visual loss following massive hemorrhage during left pneumonectomy: a case report

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Introduction

We encountered a patient with intraoperative cardiopulmonary collapse, lasting for 25 min, as a result of massive hemorrhage that occurred during left pneumonectomy. After the surgery, although no consciousness disorder or quadriplegia was observed, transient delayed visual loss was detected. In this report, we describe this case and discuss the possible reasons for the absence of severe neurological consequences, and the mechanism of the visual loss.

Case report

A 61-year-old man (height, 156.7 cm; weight, 57.6 kg) with a history of left inferior lobectomy for lung cancer, performed 3 years previously, was admitted to our hospital because of tumor recurrence in the residual left superior lobe, and he was scheduled for left pneumonectomy. His surgical history included appendectomy, at 20 years of age, and transurethral prostatectomy, at age 53 years. Preoperative blood examinations and electrocardiography showed no abnormalities.

Anesthesia was induced with thiopental and butorphanol. Vecuronium was given to facilitate tracheal intubation, which was done with a left-sided double-lumen bronchial tube. Anesthesia was maintained with air-oxygen-sevoflurane inhalation, combined with epidural anesthesia. After induction, the left radial artery was cannulated for arterial blood pressure monitoring. The patient was placed in the right lateral position. Two hours after the surgery started, the main left pulmonary artery was damaged, resulting in a massive hemorrhage, of almost 3500 ml, in approximately 5 min. The heart covered by the pericardium was instantly reduced in size. The arterial blood pressure wave complex was flattened, with the mean pressure of 10 to 20 mmHg. Wide QRS was observed on the ECG monitor, indicating electromechanical dissociation. The patient's heart rate fell rapidly, to around 40 beats·min^{-1}, but was not completely arrested. When the arterial blood pressure wave complex became flat, the bleeding points were clamped and ligated. Adequate fluid and blood transfusion was promptly instituted. There was a period of approximately 25 min before the systolic blood pressure was restored to around 90 mmHg. During this interval, ephedrine, 20 mg; methoxamine, 6 mg; epinephrine, 3 mg; 2% lidocaine, 100 mg; and dopamine, 8.6 μg·kg^{-1}·min^{-1} were administered intravenously. When the patient's systolic blood pressure recovered to around 90 mmHg, his pupils were widely dilated bilaterally, with no reactivity to direct and consensual light stimulation. Blood gas analysis conducted immediately before circulatory recovery showed a PaO_2 of 230.7 mmHg (FIO_2, 1.0), $PaCO_2$ of 72.4 mmHg, and base excess (BE) of −17.5 mEq·l^{-1}, which was corrected with sodium bicarbonate. The blood sugar level was 322 mg·dl^{-1}. The bladder temperature was 34.9°C immediately before the cardiovascular collapse, and 33.5°C after cardiovascular recovery, showing mild hypothermia. Subsequently, the patient's systolic blood pressure varied between 80 and 100 mmHg, and his bladder temperature remained around 33°C. Surgery was terminated 3 h after the recovery from cardiovascular collapse. The total blood loss was 7661 ml. Fluid transfusion of 5350 ml and blood transfusion of 22 units of packed red blood cells and 20 units of fresh-frozen plasma were given. The urine volume was 2320 ml.

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After surgery, the patient was immediately transferred to the intensive care unit (ICU), while remaining intubated. On arrival at the ICU, examination of pupils showed miosis and prompt light reflex. Sedation was continued with midazolam and butorphanol. His body temperature was 33.9°C on arrival at the ICU and recovered spontaneously to 37.5°C in the next 6h; controlled hypothermia treatment for neuroprotection was not given. His blood glucose level was 321 mg·dl^{-1} on arrival, and 219 mg·dl^{-1} 5h later, and it was then maintained below 250 mg·dl^{-1}. From the first postoperative day, the blood glucose level was controlled at below 180 mg·dl^{-1}, and $PaCO_2$ was also controlled, at 38 to 43 mmHg. In the morning of the second postoperative day, midazolam and butorphanol were discontinued, and the patient responded when his name was called. The tracheal tube was removed on the third postoperative day. He told the nurses that he wanted to go home at this time, but accepted the situation after explanations were given. He did not complain that he had any visual disturbance. Because his postoperative course was uneventful, he was transferred to the surgical ward on the following day, with no hemodynamic and neurological deterioration.

On the fifth postoperative day, the patient complained of visual abnormality. While the visual acuity for both eyes was 1.0 before surgery, on the 7th postoperative day, he was only able to distinguish his fingers. Apart from the visual loss, no other neurological and no psychotic disorders were detected. A cerebral computed tomography (CT) scan conducted on the 6th postoperative day showed no infarction or ischemic area. In addition, cerebral magnetic resonance imaging (MRI), conducted on the 13th postoperative day, also detected no abnormality. Fundoscopic examination by an ophthalmologist showed no changes in the color or the tone of the optic nerve either at the time of onset of the visual disturbance or on subsequent examinations. The visual disturbance was considered to have been caused by damage to the optic center in the brain. Subsequently, the patient's visual acuity improved. Visual field examination was possible on the 57th postoperative day, and revealed findings of irregular central scotoma. On the 78th postoperative day, the patient's visual acuity had recovered to 0.5 in the right eye and 0.1 in the left eye. He was discharged on the 81st postoperative day. His visual acuity had recovered to 0.8 in the right eye and 0.15 in the left eye on the 92nd day.

Discussion

In the present patient, acute massive hemorrhage during surgery resulted in severe cardiovascular collapse, as shown by the falling of the arterial blood pressure to 10 to 20 mmHg for 25 min. The patient complained of visual loss on the fifth postoperative day. From his clinical course, we initially suspected that cerebral infarction or encephalopathy had occurred after the cardiopulmonary resuscitation. However, in patients with encephalopathy after cardiopulmonary resuscitation, cerebral edema is common, being observed on CT scanning in 80% of the patients after 72h and in 55% after 7 days [1]. However, the present patient had no neurological or psychotic symptoms, except for the delayed visual loss. Furthermore, he had no abnormal CT findings indicative of cerebral edema or cerebral infarction on the 6th postoperative day, and no abnormal MRI findings on the 13th day.

Although a definitive diagnosis concerning the delayed visual loss was not possible, two possibilities can be proposed. Taking into account the patient's good neurological prognosis, as well as the imaging findings, we suggest that the first possibility could have been incomplete cerebral ischemia, in which the cerebral circulation was maintained to a certain extent despite the severe circulatory collapse. The cerebral cortex is one of the sites that are particularly vulnerable to ischemia. According to Gionet et al. [2], the cerebral blood flow does not decrease uniformly in all the cerebral regions in incomplete cerebral ischemia. In our patient, it is possible that the cerebral blood flow decreased predominatly in the posterior lobe or temporal lobe, where the optic center is located, causing the visual disturbance. The fact that the visual disturbance improved subsequently suggests that there was transient dysfunction in the optic center, or that the ischemia-induced neuronal necrosis was compensated by other healthy cells. The absence of abnormalities on fundoscopic examination, and the lack of abnormalities on CT and MRI scans, may be taken as evidence of this possibility.

The second possibility is that, although the brain was protected, the massive hemorrhage during the operation may have caused ischemic optic neuropathy. Visual disturbance as a result of massive hemorrhage is relatively well documented in other countries. Hayreh [3] counted approximately 600 cases concerning visual disturbance following marked and/or recurrent blood loss reported in the literature in hundreds of publications since 1641; this phenomenon was even reported by Hippocrates. Williams et al. [4] reviewed these reports precisely and concluded that ischemic optic neuropathy with visual disturbance may be caused by compromised blood flow in the arteries supplying the optic nerve. Pathologically, such a complication can be classified into two types; anterior and posterior ischemic optic neuropathy. The anterior type, which involves lesions from the optic papilla to the retrolaminar region of the optic nerve, is

characterized by fundal findings that include pale edema of the optic disc and sublimis flame hemorrhage in the disc margin. The prognosis is usually good. In contrast, the posterior type, which involves areas more posterior to the optic chiasma, shows no abnormality of the optic disc shortly after onset, but progresses, with time, to optic nerve atrophy, with little tendency to improvement [5]. Hollenhorst and Wagener [6] reported the characteristics of ischemic optic neuropathy after massive hemorrhage to be as follows: (1) visual disturbance is most commonly caused by gastrointestinal and uterine hemorrhage, in which massive or repeated hemorrhage is common, while visual disturbance rarely occurs after traumatic hemorrhage in healthy subjects, (2) there is a variation, of 12 h to 10 days, in the time lag between hemorrhage and the onset of visual disturbance, (3) the degree of visual disturbance is variable, and visual disturbance resembling glaucoma is commonly present in patients who can be assessed by visual field examination. However, no standardized diagnostic criteria to distinguish between these types of ischemic optic neuropathy have yet been established in the field of ophthalmology. Furthermore, the primary lesions responsible for the development of the visual disturbance after hemorrhage remain controversial.

Although anterior ischemic optic neuropathy has been reported to be the most common form following massive hemorrhage, some cases of posterior ischemic optic neuropathy have also been reported [7]. In the present patient, because tests necessary for a definitive diagnosis of ischemic optic neuropathy, such as fluorescein fundal angiography, ophthalmodynamography, and measurement of blood pressure in the retinal vessels, were not conducted, the presence of posterior ischemic optic neuropathy cannot be excluded. According to the ophthalmologists in our hospital, the presence of anterior ischemic optic neuropathy could be excluded, because of the absence of abnormal fundoscopic findings; therefore, there was a possibility of posterior ischemic optic neuropathy, which generally manifests no abnormal fundal findings. However, because visual acuity improved in the present patient, the possibility of posterior ischemic optic neuropathy, which usually has a poor prognosis, was low, and disturbance in the optic center of the brain was the most probable cause of the visual loss. Nevertheless, a definitive diagnosis concerning the delayed visual loss was not possible.

The present patient recovered with no residual consciousness disorder or paraplegia, despite the prolonged cardiovascular collapse. This favorable outcome was possibly a result of the mild hypothermia during surgery. During the period of the massive hemorrhage, he was already mildly hypothermic, with a body temperature of 34.9°C. Then, the massive fluid and blood transfusion further lowered his body temperature, to 33.5°C. His body temperature on arrival at the ICU was still 33.9°C. Subsequently, it recovered spontaneously, to 37.5°C, in a period of 6 h. Although no controlled hypothermia treatment was conducted; in effect, the mild hypothermia continued for approximately 10 h from its onset. The prolonged mild hypothermia may have protected the brain against transient cerebral ischemia, and might account, in part, for his recovery without residual consciousness disorder or paraplegia. The findings in this patient illustrated the clinical usefulness of mild hypothermia.

The neuroprotective effect of mild hypothermia against ischemia was first proven experimentally by Busto and colleagues in 1987 [8]. Subsequent studies showed that lowering of the body temperature by even 1°C protected the brain [9]. Furthermore, the cerebral protective effect was observed even when hypothermia was initiated after the onset of ischemia [10], and significant brain protection conferred by hypothermia was demonstrated in a cardiac arrest model with whole brain ischemia [11]. Definitive evidence for the mechanism of brain protection conferred by hypothermia is still not available. Conventionally, hypothermia has been regarded to confer neuroprotection by inhibiting the release of glutamate, the excess release of which causes delayed neuronal necrosis. Recently, however, Yamamoto et al. [12] have reported that the neuroprotective effect of mild hypothermia cannot be explained in terms of the suppression of glutamate release during ischemia. On the other hand, Oobashi et al. [13] showed that hypothermia inhibited brain damage in the hippocampus of aged rats through suppressing the excessive efflux of excitatory and inhibitory amino acids. The problem with the explanation in the present patient is that there was continuous hyperglycemia for approximately 8 h after the circulatory collapse, and hyperglycemia is known to be a factor that exacerbates cerebral ischemia. However, Li et al. [14] have reported that the neuroprotective effect of hypothermia surpasses the risk of hyperglycemia. In our patient, the beneficial effect of the prolonged hypothermia probably outweighed the possibility of cerebral damage being caused by hyperglycemia.

In conclusion, we have reported a patient in whom massive hemorrhage during left pneumonectomy resulted in severe cardiopulmonary collapse of 25 min duration. Transient delayed visual loss was observed on the fifth postoperative day, and was probably caused by a disturbance in the optic center of the brain, or by ischemic optic neuropathy resulting from incomplete cerebral ischemia following the massive hemorrhage. Mild hypothermia during surgery possibly protected the brain against ischemic damage and accounted, in part, for the patient's recovery with no severe neurological consequences.

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