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Improvement of hypoxia during early surgery for ruptured intracranial aneurysm: a retrospective evaluation

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Introduction

Early surgery for ruptured intracranial aneurysm has been recommended to prevent rerupture of the aneurysm and cerebral vasospasm [1–4]. We occasionally encounter hypoxic patients in this early period for whom we would hesitate to use general anesthesia. On the basis of our experience, however, we judged that hypoxia would improve during surgery, so that surgery would not need to be postponed. In the past five years, we have performed surgery on patients under general anesthesia, even when the patient had hypoxia. In this study we retrospectively evaluated changes in the oxygenation of the arterial blood of hypoxic patients during early surgery, and found that it had a beneficial effect on the hypoxia.

Patients and methods

This retrospective study was conducted on patients who were treated between 1992 and 1997. Out of a total of 112 patients who underwent early surgery for ruptured intracranial aneurysm, 14 patients (12.5%) whose PaO_2/FIO_2 was less than 300 mmHg after induction of general anesthesia were analyzed. The demographic data are shown in Table 1. Patients with a past history of apparent respiratory or cardiac failure and those whose gastric contents had been aspirated were excluded from this study.

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Thiamylal or propofol was used for the induction of anesthesia, and vecuronium with cricoid pressure facilitated tracheal intubation. General anesthesia was maintained with isoflurane and 50% O_2/N_2O. When percutaneous oxygen saturation decreased to less than 95%, the inspired oxygen concentration was elevated. After induction, mechanical ventilation at a tidal volume of 7–10 ml·kg^{-1} and a ventilation rate of 10–15 breaths·min^{-1} was applied to result in an end-tidal CO_2 value of 25–30 mmHg. An arterial catheter was inserted to monitor blood pressure and collect the arterial blood. Twenty minutes after mechanical ventilation, arterial blood gas was analyzed. Acetate Ringer's solution was infused and 0.6–0.8 g·kg^{-1} of mannitol was administered before the dural incision. Blood transfusion was necessary for only one patient during surgery due to massive blood loss. Until the clipping of the ruptured aneurysm, systolic arterial pressure was maintained between 80 and 100 mmHg. Arterial blood gas analysis was performed before closing the scalp after clipping.

Data are shown as means ± SD. The results were analyzed by the Wilcoxon signed-rank test, and $P < 0.05$ was considered significant.

Results

Twelve of 14 patients showed various degrees of infiltration shadow by chest X-ray on arrival, and none of the patients showed atelectasis (Table 1). Arterial blood gas analysis in defined oxygen concentrations was performed on 7 of the 14 patients in the emergency room, giving a result of 272.0 ± 70.0 mmHg of PaO_2/FIO_2. During surgery, the PaO_2/FIO_2 of two patients was slightly depressed and that of one patient showed almost no changes. The PaO_2/FIO_2 of the other 11 patients improved during surgery. The PaO_2/FIO_2 increased significantly from 242.9 ± 40.2 mmHg after the induction of anesthesia to 338.4 ± 60.8 mmHg

Table 1. Demographic data

Patient no.	Age (yr)/sex	WFNS	Site of aneurysm	Chest X-ray on arrival	Time to surgery (h)	Hemorrhage (ml)	Crystalloid infusion (ml)	Blood transfusion (ml)	Duration of surgery (min)	GOS
1	59/M	V	A Com	Bil. diffuse infiltration	5	400	1750	0	279	SD
2	76/M	IV	A Com	Rt. focal infiltration	6	2700	2450	2640	170	SD
3	72/M	V	A Com	Bil. diffuse infiltration	8	150	1500	0	269	Death
4	72/F	II	A Com	Bil. slight infiltration	5	100	600	0	206	GR
5	47/F	II	MC	Bil. diffuse infiltration	3	120	900	0	172	GR
6	71/F	IV	IC	Rt. focal infiltration	12	500	1350	0	285	MD
7	66/F	V	IC	Bil. focal infiltration	7	527	950	0	305	MD
8	64/F	IV	AC	Rt. focal infiltration	5	210	750	0	382	GR
9	55/M	V	A Com	Bil. diffuse infiltration	6	100	1850	0	247	GR
10	61/F	I	MC	Almost normal	3	150	950	0	197	GR
11	62/F	V	MC	Bil. diffuse infiltration	3	50	490	0	173	VS
12	76/F	I	MC	Almost normal	12	80	1000	0	170	SD
13	44/M	III	A Com	Bil. diffuse infiltration	12	40	950	0	252	MD
14	48/M	IV	A Com	Bil. diffuse infiltration	4	100	850	0	150	MD
Mean	62.4				16.5	373.4	1167.1		232.6	
SD	10.7				3.3	299.2	547.1		66.9	

WFNS, World Federation of Neurosurgical Societies grading scale; GOS, Glasgow outcome scale; A Com, anterior communicating artery; MC, middle cerebral artery; IC, internal carotid artery; AC, anterior cerebral artery; SD, severe disability; GR, good recovery; MD, moderate disability; VS, persistent vegetative state.

Fig. 1. Changes of PaO_2/FiO_2 in individual data and mean ± SD. There was a statistically significant increase

before closure of the scalp (Fig. 1). There were no cases of aggravation of hypoxia or respiratory failure in the intensive care unit. Two patients who showed no improvement of PaO_2/FiO_2 during surgery recovered gradually from hypoxia after surgery and were extubated the next day. Although some cases still showed infiltration shadow on the chest X-ray the next day, most patients' X-rays became clear within several days.

Discussion

The present study revealed that almost all of the patients with hypoxia recovered from the hypoxic state during surgery, and after surgery none of the patients had respiratory failure. One patient died of extensive cerebral infarction and brain death caused by severe vasospasm. The incidence of hypoxia in patients with acute subarachnoid hemorrhage caused by ruptured intracranial aneurysm has been reported to be around 20% [5]. Although the cause of hypoxia is unclear, neurogenic pulmonary edema (NPE) is one of the conditions related to hypoxia. The following are considered to be possible causes of NPE: pulmonary capillary hyperpermeability neurally evoked by a

disorder of the central nervous system and/or elevation of intracranial pressure [6–8], hydrostatic pulmonary edema evoked by centralization of the circulatory blood after release of large amounts of cathecolamines [8–10], and cardiogenic pulmonary edema caused by myocardial impairment as a result of release of large amounts of cathecolamines [11]. Although hypoxia tends to occur more in comatose patients than in conscious patients, we occasionally see hypoxia in patients with relatively good consciousness [12], as described in this study. We defined hypoxia in this study as PaO_2/FIO_2 less than 300 mmHg after tracheal intubation and ventilation. Since patients who had apparent atelectasis or who had been aspirated were excluded, hypoxia in most of the patients was probably caused by NPE.

In patients with NPE or in a hypoxic state, surgery is usually postponed until hypoxia improves, in spite of the higher risk of rerupture and cerebral vasospasm. Appropriate respiratory care and control of intracranial pressure often improves hypoxia within several days. Therefore, improvement of hypoxia during early surgery for ruptured intracranial aneurysm may be part of the natural course of recovery. The evidence that hypoxia improved during surgery, however, suggests that anesthesia and/or craniotomy itself has a beneficial effect on the hypoxic state. This effect could be the result of suppression of cerebral metabolism by a volatile anesthetic, depression of intracranial pressure by craniotomy, and/or relaxation of sympathetic nervous activity by removal of the subarachnoid hemorrhage. Mechanical ventilation during general anesthesia leads to sufficient alveolar ventilation and allows rapid recovery from the hypoxic state. Aggravation of the hypoxia leading to a vicious circle of NPE and hypoxia can be prevented.

In summary, early surgery for ruptured intracranial aneurysm in patients with preoperative hypoxia, probably due to NPE, was performed, resulting in the improvement of hypoxia. The appropriate respiratory care, general anesthesia, and removal of the hematoma might have contributed to the recovery from NPE in our patients.

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