Intraoperative Predissection Aneurysm Rupture: No Less than a Catastrophe!

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Anesthetic management for clipping of intracranial aneurysm may be complicated by intraoperative rupture at various surgical stages. Sudden increase in transmural pressure (TMP) caused by either sudden increase in mean arterial pressure (MAP) or decrease in intracranial pressure (ICP) may be responsible for it. Acute hyperventilation causing intraoperative aneurysm rupture has not been reported earlier, even though such a concern exists theoretically.

A 45-year-old man, weighing 60 kg, presented with history of headache, vertigo, and transient loss of consciousness, for the past 3 days. Computerized tomography scan of brain demonstrated subarachnoid hemorrhage (SAH) in interhemispheric fissure. Cerebral digital subtraction angiography revealed anterior communicating artery aneurysm. He had no other systemic illness and all routine investigations were normal. Patient was scheduled for left pterional craniotomy and aneurysm clipping. In the operation theater, noninvasive monitoring was started with 5-lead electrocardiography, automated blood pressure, and pulse oximetry. Anesthesia was induced with fentanyl (2 µg/kg) and thio- pentone (5 mg/kg). Xylocard (1.5 mg/kg) was administered to attenuate the pressor response and tracheal intubation was facilitated with rocuronium (1 mg/kg). Anesthesia was maintained with isoflurane in oxygen and nitrous oxide (1:2) mixture along with intermittent boluses of fentanyl and vecuronium. Mechanical ventilation was adjusted to maintain end-tidal carbon dioxide (EtCO2) around 32 mm Hg. Invasive blood pressure, central venous pressure, and nasopharyngeal temperature were continuously monitored. After craniotomy, the dura appeared slightly tense. Various causes likely to increase ICP were ruled out; for example, hypercarbia (EtCO2 32 mm Hg, PaCO2 33 mm Hg), hypoxia (SpO2 99%), higher concentration of volatile anesthetic (minimum alveolar concentration [MAC]: 1.1), high airway pressure (18 mm Hg) or central venous pressure (CPV) (8 mm Hg), light anesthesia, head down positioning, or excessive rotation of the neck were ruled out. The patient was hemodynamically stable till this stage. By this time, mannitol (1 g/kg) infusion had already been administered over approximately 30 minutes. There was not much improvement even after switching off nitrous oxide, so minute ventilation was increased in an attempt to achieve moderate hypocarbia. Over the next minute, EtCO2 decreased from 32 mm Hg to 27 mm Hg. At this point, the surgeons informed that the dura had become dark in color and the brain had become “stony hard.” During this period, there was no change in blood pressure (►Fig. 1). The surgeons suspected aneurysm rupture and decided to proceed with the surgery. After opening the dura, there was profuse bleeding, thereby blinding the surgeon. Left frontal lobectomy was performed to achieve adequate and urgent exposure. Systolic blood pressure was maintained around 100 mm Hg by administration of crystalloids, colloids, and blood. The ruptured aneurysm was successfully clipped following temporary clip application on left internal carotid artery for 20 minutes. Total estimated blood loss was approximately 3.5 L, which was adequately replaced. Rest of the intraoperative period was uneventful. Postoperatively, the patient was electively ventilated overnight in the neurointensive care unit. The patient was extubated on the first postoperative day and rest of his hospital stay was unremarkable. Patient was discharged on postoperative day 8 with Glasgow Coma Scale score of 15 with slight weakness in right lower limb.

Intraoperative aneurysm rupture is described at three specific periods, that is, early or predissection (7%), during dissection (48%), and during clip application (45%).1 Aneurysm rupture in early or predissection period is mostly due to sudden acute increase in TMP gradient. We excluded all factors which could contribute to raised ICP before resorting to hyperventilation, the effect of which was immediate. This might have increased TMP, resulting in aneurysm rupture. Various factors such as stress response during endotracheal intubation, pin fixation, vibrations of power instruments during craniotomy, change in ICP by mannitol, and various aneurysmal factors have been implicated in early intraoperative rupture of aneurysms.2 In our patient, rupture seems to have occurred after craniotomy as it turned stony hard only after hyperventilation. There are reports of adenosine being successfully used to manage inadvertent intraoperative aneurysmal rupture during dissection or clipping.
phase. But, whether it is safe or has a beneficial role during predissection phase, is not clear. This patient belonged to a good SAH grade and theoretical concerns of hyperventilation have been clearly defined. Yet, to achieve relaxed brain, we resorted to hyperventilation as a quick maneuver to facilitate surgical exposure. However, prompt aneurysm obliteration together with maintenance of normotension during the procedure resulted in good outcome of the patient.

In conclusion, we reiterate the theoretical teaching that during aneurysm surgery, hyperventilation should not be instituted acutely in good-grade patients to decrease ICP, especially before dural opening. If intracranial pressure is to be decreased then utmost care should be taken not to suddenly increase the TMP gradient.

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**Conflict of Interest**
None declared.

**References**


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**Fig. 1** Record of heart rate (HR), mean arterial blood pressure (MABP), and end-tidal carbon dioxide (EtCO₂) at 1-minute interval during the episode of aneurysm rupture. White arrow: acute hyperventilation, black arrow: aneurysm rupture.