INTRACEREBRAL HEMORRHAGE FOLLOWING ENUCLEATION: A RESULT OF SURGERY OR ANESTHESIA?

- A Case Report -

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Summary

Choroidal melanoma is the most common primary intraocular cancer in adults. A sixty-nine years old, hypertensive male with a choroidal melanoma underwent enucleation. After extubation he woke up confused and unconscious. An emergent computed tomographic (CT) scan demonstrated intracerebral hematoma. The underwent repeat surgery in the postoperative first hour, because of left parietotemporal intracerebral hematoma. His neurological state became worse and he died in the eighth postoperative day. Sympathetic stimulation due to extubation, causing increase in the intracranial pressure or uncontrolled hypertension, may be reasons precipitating intracranial hemorrhage.

In patients, who undergo intracranial or intraorbital surgery, had risk factors of intracranial hemorrhage or showed labile blood pressure perioperatively and were confused or unconscious in the postoperatively or had delayed emergence, intracranial hematoma must be suspected.

Key Words: Intracranial hematoma, hypertension, delayed emergence.
Introduction

Choroidal melanoma is the most common primary intraocular cancer in adults. Nevertheless, only about 6 cases per million population are believed to be diagnosed annually in the United States. Until 1970, enucleation was the standard treatment. Intraorbital or intracranial hematoma following intracranial surgery has been reported. Postoperative cerebral hemorrhage has not been reported.

We report a case of cerebral hemorrhage at the same side of enucleation that occurred during surgery, and arising in the postoperative period. This patient had a history of hypertension and pre-existing cardiovascular disease who underwent enucleation for choroidal melanoma and had a fatal outcome.

Case Report

Sixty-nine years old, male patient had gradual loss of vision in the left eye. The diagnosis was choroidal melanoma. Enucleation was planned. Five years previously he had myocardial infarction and his leg was amputated below knee after an accident. He was hypertensive and received antihypertensive medication. Otherwise he was in good health. Electrocardiogram showed a prior inferior myocardial infarction and left ventricular hypertrophy.

Anesthesia was induced with propofol 200 mg, fentanyl 50 µg and was maintained with 50% N₂O, 50% O₂ and 1% isoflurane. Vecuronium 6 mg was administered for tracheal intubation. Before induction systolic blood pressure was 170 mmHg and it fluctuated between 120-170 mmHg during the operation. Oxygen saturation ranged from 95-97%. The operation lasted for one hour. At the end of the operation he was breathing spontaneously. When the surgical procedure was over, reversal of neuromuscular blockade was accomplished with neostigmine 0.05 mg/kg plus atropine 0.01 mg/kg administered IV. Oxygen saturation was 99%. He was extubated as he could not tolerate the endotracheal tube. Following extubation heart rate was 59-78 beats/min,
systolic blood pressure was 165-205 mmHg, respiratory rate was 20 times/min.

Nitroglycerin infusion was started to decrease blood pressure in PACU. He could not respond to vocal stimulations which was attributed to the effect of anesthetic drugs. His oxygen saturation was over 95% with spontaneous breathing but he was found unconscious and confused. Following neurosurgical consultation, a CT scan was obtained immediately. The patient underwent surgery again because of left parietotemporal intracerebral hematoma detected in CT scan (Fig. 1).

During the operation heart rate was 80-100 beat/min, systolic blood pressure was 100-160 mmHg, and oxygen saturations was 97-98%. Operation lasted for 6 hours and 30 minutes. After the operation patient
was admitted to the neurosurgery intensive care unit intubated, and received mechanical ventilation. Extensor response was detected to painful stimulus. His status became worse in the eighth postoperative day and cardiac arrest occurred. He did not respond to resuscitation and was accepted as exitus.

**Discussion**

Spontaneous intracerebral hemorrhage (ICH) is a serious disease despite progress in medical knowledge. ICH appears suddenly without warning, unlike ischemic strokes that are often preceded by a transient ischemic attack. Outcome is determined by the initial severity of the bleeding; mortality and morbidity of ICH are high. So far, treatment regimens are limited.

Prevention of ICH is the most effective approach. Risk factors for ICH appeared to be age, male gender, hypertension, and high alcohol intake. Hypertension is the most important risk factor for ICH. Treatment and management of hypertension remain the most effective prevention strategy for ICH, given both the relatively high prevalence of hypertension within the community and the strong association between hypertension and ICH.¹

Intracranial hemorrhage can be a serious and sometimes fatal complication when it occurs during or after intracranial surgery. Intracranial hemorrhage is likely the result of many predisposing conditions (e.g. surgical hemostasis, venous pressure, coagulation status, genetic predisposition, hemodynamic state).

Acute blood pressure elevations occur frequently prior to postcraniotomy intracranial hemorrhage. Patients who develop ICH are more likely to be hypertensive in the intraoperative and early postoperative periods. Other contributing factors could be tracheal intubation, if it is associated with frequent coughing and straining, which may increase cerebral venous pressure and predispose the ICH. Like tracheal intubation, extubation requires special handling during emergence.
Waga et al, have noted that labile hypertension and intraoperative changes in blood pressure, may contribute to the development of ICH, remote from the operative site following neurosurgical procedures.

In our case, cerebral hematoma was at the same site as that of surgery. There was an intracerebral hematoma in the same side of enucleation at CT which could be contributed either to the surgery of the choroid melanoma itself, or only to the emergence from anesthesia.

Anesthesia could have caused this fatal complication. Before induction systolic blood pressure was 170 mmHg, and intraoperatively it fluctuated between 120-170 mmHg. Other reasons could be the sympathetic stimulation due to extubation, causing increase in intracranial pressure. The use of phenylephrine and cyclopentolare 1% drop in the preoperative period may also be the cause of labile hypertension in our case.

When awakening criteria are not conforming or there is no cooperation after general anesthesia, intracranial hemorrhage is to be suspected. Although our patient was diagnosed earlier, his prognosis was poor. He died in the eighth postoperative day.

Patients who have hypertension, coronary artery disease, perioperative labile hypertension, and surgery planned for intraorbital mass, may carry the risk of intracranial hemorrhage during the postoperative period. Perioperatively and during extubation the anesthesiologist must avoid sudden hypertensive attacks. To protect patient from hypertension, blood pressure must be controlled preoperatively. Surgical stress and hemodynamic response during induction must be controlled with anesthetics or other additional agents. Emergence from anesthesia must be smooth and controlled.

In conclusion, patients scheduled for intracranial or intraorbital surgery and carry risk factors of intracranial hemorrhage, labile tension peroperatively, have delayed emergence and are confused or unconscious in the postoperative period, intracranial hematoma must be suspected and evaluated by CT scan.
References


