Case Report

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Intraoperative Anesthetic Management of Bilateral Intraventricular Hemorrhage Secondary to Anterior Communicating Artery Aneurysm Rupture in Hypertensive Patient

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Abstract

Introduction: Primary (IVH) is non traumatic intracerebral hemorrhage confined to the ventricular system which is relatively infrequent, but is more commonly caused by hypertensive (52%) hemorrhagic stroke, followed by trauma (15%), intraventricular arteriovenous malformation (7.5%), coagulopathy (7.5%), diabetic vasculopathy (7.5%), anterior communicating artery aneurysms (3%), and undetectable cause. Clipping of ruptured anterior communicating artery with hypertension possess anesthetic challenges in managing intra-operatively, and when it comes for an ideal neuroprotective agent, barbiturates induced anesthesia was planned.

Case Report: A 70 years old male came to our hospital with history of sudden loss of consciousness and a known case of systemic hypertension for 3 years and was on irregular medications, intubated in emergency medicine department in view of low GCS which was difficult intubation.

CT brain Angiogram was done and diagnosed as ruptured Anterior Communicating Artery Aneurysm and underwent Aneurysmal clipping of anterior communicating artery aneurysm under general anesthesia.

Emergency medications were kept ready. Intensive neuro monitoring, invasive blood pressure monitoring and to decrease the intraoperative intracranial pressure, to preserve autoregulation of cerebral blood flow, barbiturates induced anesthesia were planned (thiopentone as an induction agent and maintained with inhalational anesthetic agent). Patient was shifted to ICU for observation and extubated on next day uneventfully.

Conclusion: We present a successful anesthetic management of hypertensive patient with ruptured anterior communicating artery who underwent clipping of aneurysm. A detailed pre-anesthetic evaluation and proper planning is utmost important to encounter the risk of ischemic injury to brain while clipping of ruptured aneurysmal vessel.

Keywords: Barbiturates induced anesthesia; Hydrocephalus; Hypertension; Intraventricular hemorrhage.

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INTRODUCTION

Primary (IVH) is non traumatic intracerebral haemorrhage confined to the ventricular system which is relatively infrequent, but is more commonly caused by hypertensive (52%) haemorrhagic stroke, followed by trauma (15%), intraventricular arteriovenous malformation (7.5%), coagulopathy (7.5%), diabetic vasculopathy (7.5%), anterior communicating artery aneurysms (3%), and undetectable cause. Clipping of ruptured anterior communicating artery with hypertension possess anesthetic challenges in managing intra-operatively, and when it comes for an ideal neuroprotective agent, barbiturates induced anesthesia was planned.

CASE PRESENTATION

Here we present a 70 year old male who presented to Emergency Medicine Department with a history of sudden loss of consciousness following fall over the ground previous day with one episode of vomiting and no history of seizure.

Patient is a known case of systemic hypertension for 5 years and was on irregular medications (Tablet Amlodipine 5 mg twice a day) with no other comorbidities and no history of any previous surgeries. General physical examination revealed no pallor, icterus, cyanosis, clubbing, lymphadenopathy and edema with blood pressure of 190/120 mmHg, pulse rate of 80 bpm, respiratory rate of 20 cpm and saturation of 68% on room air. At arrival, patient had GCS of E1V1M1 with bilateral pupils 2 mm and reactive to light. Other systemic examination was within normal limits.

In view of low GCS, emergency intubation was done with the help of bougie due to presence of large tongue and CL grade 3 and then shifted to ICU. Patient was on Controlled Mechanical Ventilation mode with FiO_2 of 100%, Tidal volume of 450 ml RR = 12 bpm PEEP = 5 mm Hg.

Patient underwent bedside extra ventricular drainage to avoid the development of hydrocephalus following which he underwent CT Brain Angiography and was diagnosed with Bilateral Intraventricular Hemorrhage secondary to rupture of anterior communicating artery aneurysm.

Routine investigations done including complete hemogram and found to be within normal limit but coagulation profile was altered with increased prothrombin time of 20.3 secs, activated partial thromboplastin time of 33.2 secs and INR of 1.72 for which 4 FFPs were transfused. Renal function tests and liver function tests are done and within normal limits.

Electrocardiogram revealed a sinus tachycardia with tall peaked t waves (right atrial enlargement). Chest X-ray was normal. CT Brain Angiography revealed Saccular aneurysm arising from anterior communicating artery of 5x6 mm and hemorrhage was seen in lateral ventricles, 3rd and 4th ventricles.

On admission physician opinion was obtained for uncontrolled hypertension and was started on Tab. Amlodipine 5 mg 1-0-1, Tab. Telmisartan 40 mg 1-0-0, Tab. Nimodipine 60mg QID, Inj. Levetiracetam 500 mg IV 1-0-1, Inj. Mannitol 100 mg IV 1-1-1-1, Inj. Lasix 20mg IV 1-0-1.

After a thorough preoperative evaluation and written informed high-risk consent, patient underwent aneurysmal clipping for anterior communicating artery aneurysm.

Anesthetic Management:

Right femoral central venous catheterization done alongwith wide bore 18 gauge intravenous cannula. Arterial line was secured on right radial artery for invasive blood pressure monitoring. Strict neuromonitoring with electrocardiography, pulse oximetry and capnography monitoring was done.

Patient was pre-medicated with Inj. Glycopyrrolate 0.2 mg and Inj. Fentanyl 100 mcg. Induction with Inj. Thiopentone 250 mg IV over 5 minutes minutes followed by infusion dose was started at 4 mg/kg/hour it is titrated according to mean arterial pressure of the patient and facilitated with muscle relaxant Inj. Vecuronium. Anesthesia was maintained with isoflurane, oxygen, nitrous oxide and vecuronium.

Intraoperative blood pressure was 190/120 mmHg initially for which Inj. Nitroglycerin was commenced at 10 mcg/min (0.6 ml/hr) initially and titrated according to clinical response of the patient.

Blood loss was managed intraoperatively with crystalloids, 1 haemacel, 2 packed cells, 2 FFP and patient was hemodynamically stable throughout the intra-operative period and aneurysmal clipping was done successfully.

Patient was shifted with endotracheal tube in-situ to ICU for elective ventilation. Post-operative monitoring of vitals was done in ICU and ventilation was weaned down overnight. Post-

operative period was uneventful and hence patient was extubated the next day morning.

DISCUSSION

Intraventricular haemorrhage (IVH) is a recognized entity of intracranial haemorrhage.

Spontaneous also known as Primary (IVH) is non traumatic intracerebral haemorrhage confined to the ventricular system, occurs predominantly in men over 40 years of age. It is relatively infrequent, comprising only 3.1% of all spontaneous intracranial hemorrhages, but is more commonly caused by hypertensive (52%) hemorrhagic stroke, with high rates of death and disability. The appropriate treatment is not clear and the prognosis is variable with mortality rates reported as 40%-83%.

CT Angiogram or MRI Angiogram identifies the source of bleeding in about 60% of cases.

In the absence of specific treatment (*i.e.*, Extraventricular drainage (EVD), IVH is associated with a 78% risk of death and a 90% risk of poor outcome and so EVD is mandatory for the patients with IVH to reduce the risk of acute hydrocephalus.

The treatment is aimed primarily at neuroprotection and understanding the physiological implications of the surgical procedure.

Intraoperative thiopental administration significantly reduces the post-operative neurological complications in patients undergoing surgical clipping for aneurysmal rupture.

There are several proposed mechanisms for cerebral protection of thiopental.

Barbiturates (Thiopentone) induced anesthesia preserves autoregulation of cerebral blood flow by adose dependent reduction in CBF and $CMRO_2$ until the EEG becomes flat. At the point of isoelectrical EEG, no further $CMRO_2$ reduction occurs despite of further increase in barbiturate dose. The maximal thiopentone induced $CMRO_2$ decrease is 55 to 60%. Thus, with barbiturates, functional depression appears tobe coupled with reduction in CBF and $CMRO_2$. ICP is reduced by barbiturates, possibly because of reduction in CBF & CBV. This effect is used during the treatment of raised ICP in head injured patient as well as induction of anesthesia in patients with decreased intracranial compliance.

They mainly act at the post synaptic GABA receptors of the CNS synapses by keeping open the chloride channel which in turn produce conduction blockade due to the chloride ion flow producing hyperpolarization, barbiturates increase the duration of this open state of chloride channel whereas benzodiazepines increase the frequency of chloride channel opening. Secondly, patients with refractory elevated intracranial pressure (RICH) due to traumatic brain injury (TBI) may have improved long term outcome when barbiturate coma is added to their neurointensive care treatment. This phenomenon is also called an inverse steal or Robin Hood effect as cerebral perfusion to all parts of the brain is reduced (due to the decreased cerebrovascular response to carbon dioxide) allowing optimal perfusion to ischemic areas of the brain which have higher metabolic demands, since vessels supplying ischemic areas of the brain would already be maximally dilated because of the metabolic demand.[1-4]

Cardiac evaluation and liver function test was done to look for normal heart function as thiopentone is cardiotoxic which reduces the cardiac output and also it gets metabolized in liver.

The goal is to maintain ICP <25mmHg and to achieve therapeutic EEG response (burst suppression) or BIS value of 10-20 and SR of 60-80%.[5-6]

Continuous EEG monitoring, arterial blood pressure and ECG monitoring and plasma K+: Refractory hypokalaemia has been reported in patients receiving Thiopental infusions. The fall in serum K+ is thought to be due to metabolic changes within the brain. There is potential for severe rebound hyperkalaemia when the thiopental infusion is ceased, so supplement potassium cautiously, if ECG changes indicate is needed.

Nitroglycerin may reduce BP in both ischemic stroke and intracerebral hemorrhage in ultra-early, early, and subacute phase. NTG is a nitric oxide donor with other properties, including an antiplatelet effect and prevention of ischemia induced apoptosis. NTG leads to a decrease in mean arterial pressure and an increase in cerebral perfusion pressure (CPP) without any change in hemispheric CBF.

Post-operatively patient was shifted to Intensive care unit with tube in situ for elective ventilation.

CONCLUSION

In our case report we present a successful anesthetic management of a hypertensive patient who underwent aneurysmal clipping for anterior communicating artery aneurysm by intraoperative thiopentone administration which leads to

reduction in the post-operative neurological complications. A detailed pre-op planning and through understanding of hemodynamic changes is utmost important to overcome the risk of intraoperative events.

Conflicts of Interest: Nil

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