Anaesthetic Management of A Patient for Carotid Endarterectomy

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Abstract :

Stroke is one of the leading causes of death in the modern countries. Mainstay treatment for stroke prevention is carotid endarterectomy (CEA). Patients scheduled for surgery often have many associate systemic illnesses which pose a risk of perioperative cardiac and neurological complications. Preoperative evaluation of neurological and cardiac function with optimization of the systemic illnesses is necessary. Ideal anaesthesia technique should provide adequate analgesia, minimal stress response, optimal brain perfusion and oxygenation, optimal hemodynamic and myocardial oxygen balance. Greatest risk in the early postoperative period is new neurological deficit caused by cerebral ischemia and myocardial infarction caused with hemodynamic instability. We are reporting a case for carotid endarterectomy under general anaesthesia.

Key Words : Anaesthesia, Carotid Endarterectomy

Introduction

Stroke is one of the leading causes of death in modern countries. [1] It is caused by carotid artery stenosis with atherosclerotic plaque or atheroma embolization. The mainstay of treatment for stroke prevention is carotid endarterectomy (CEA). It is indicated in symptomatic patients with stenosis 50-69% and in asymptomatic patients with stenosis > 70% if the preoperative risk is acceptable. Although CEA is a preventive procedure, perioperative mortality is relatively high, about 5%. [2] The main risks are cerebral and myocardial ischemia. Cerebral ischemia may occur during temporary clamping of the common carotid artery (while the artery is opened for atherosclerotic plaque removal) producing hypoperfusion, if the collateral blood flow is inadequate.

Case Report

A 48 yr old male ASA risk 3 presented with history of right hemiparesis, slurring of speech since two and a half months. Patient was a known case of hypertension and on Tab. Amlodepin 5mg + Atenolol 25 mg. Patient was on antiplatelet (ecosprin+clopidogrel) since two and a half months which was stopped for seven days before surgery. There was no history of diabetes and ischemic heart disease. On CNS examination patient had left facial palsy with right upper limb power 3/5 and right lower limb power 4/5. Carotid angiography suggestive of 90% stenosis at left carotid bifurcation involving bulb-internal carotid artery origin with good forward flow. Plaque length was 1.2cm. 50% stenosis at the origin of external carotid artery. Left anterior carotid artery shows 60% stenosis. In MRI there were changes of old infarct with gliosis in left tempero parietal lobe and left basal ganglion.

In preanaesthetic examination his heart rate was 60 beats/min; blood pressure 120/90 and SpO_2 98%. On auscultation chest was clear. Electrocardiograph (ECG) and 2D-echocardiography (ECHO) showed normal valvular and ventricular function with 60% ejection fraction. Laboratory tests including complete haemogram, liver function test, renal function test, serum electrolytes and coagulation profile were normal.

The patient was counseled and informed consent was obtained.

In the operation room after taking an intravenous access, routine monitoring in the form of ECG, SpO_2, $EtCO_2$ were applied. Invasive blood pressure monitoring was done by using right radial artery. $EtCO_2$ was monitored after endotracheal intubation. The patient was induced with Inj. Fentanyl 120mcg followed by a induction dose of Inj thiopentone sodium 350mg. Inj Veuronium bromide 6 mg given to facilitate endotracheal intubation. Patient was ventilated with 100% oxygen for 3 mins. Patient was intubated with flexometalic tube no.38.

Anesthesia was maintained with O_2+N_2O+Sevoflurane + Veuronium bromide infusion. MAP was maintained between 80-90 mm of Hg. $EtCO_2$ was maintained 30 mm of Hg. Before clamping of carotid artery, inj. thiopentone sodium 250mg and inj. heparin 5000 units IV were given. Clamping time was 40 mins.

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Intraoperative hypertension was treated with Nitroglycerine (NTG) infusion and Inj.betaloc. Patient was reversed with Inj Neostigmin 3mg, Inj. Glycopyrollate 0.4 mg. Recovery was good with stable hemodynamic without any neurological deficit. There was no significant blood loss during surgery. Post operative hypertension was treated with NTG infusion.

Immediately after surgery he was transferred to ICU. He received analgesia in the form of Diclofenac sodium AQ 75mg i.v. 8 hourly. Patient was continuously monitored in ICU for 24hrs. Patient was discharged on 7th day.

Discussion

Carotid stenosis is caused by the plaques formed at the carotid bifurcation that lead to narrowing of the artery and embolization of thrombus causing transient ischemic attacks or stroke. The risk of stroke has been estimated at approximately 5% per year if the patient is asymptomatic and about 10% per year for a patient who has already had transient ischemic attack. [3] An ideal anaesthetic technique should provide adequate analgesia, minimal stress response, optimal brain perfusion and oxygenation, optimal hemodynamic and myocardial oxygen balance while assuring calm and relaxed patients with good surgical comfort. [4] General anesthesia is recommended for patients with a history of prior cerebral strokes, if the brain collateral circulation is inadequate and for patients with distal stenosis of the carotid artery due to complicated surgical technique. [5] So we gave general anaesthesia to the patient.

During General anaesthesia, a balanced technique is used. Advantages of general anaesthesia include mechanical ventilation with controlled oxygenation and carbon dioxide (CO2) levels, controlled hemodynamics and good surgical conditions. Opioids have an indirect effect on oxygen balance by decreasing the stress response. We have used thiopentone sodium because of its neuroprotective effect. Isoflurane and sevoflurane have the best neuroprotective effect. Although both sevoflurane and isoflurane can provide rapid recovery, sevoflurane produces less vasodilation than isoflurane at the same depth of anaesthesia. [6] Muscle relaxants decrease oxygen consumption in the muscles providing more oxygen to other tissues. Hypocarbia may cause ipsilateral vasoconstriction and extend the area of cerebral ischaemia. Hypercarbia may produce contralateral vasodilatation and cause a steal phenomenon. [7] Based on current evidence, normocarbia is the best policy. It is appropriate to maintain the mean arterial pressure 20% above the preoperative level to maintain the perfusion pressure across the Circle of Willis. Cardiovascular lability in hypertensive patients undergoing anaesthesia and surgery is a well-documented problem. These patients are prone to episodes of hypotension and hypertension in the intraoperative period. [8, 9] Carotid cross-clamping is frequently associated with a marked increase in arterial pressure. Heparin is administered in the intraoperative period before cross clamping to reduce the risk of thromboembolic complication. The greatest risk in the early postoperative period is that of a new neurological deficit caused by cerebral ischemia or myocardial infarction associated with hemodynamic instability. Postoperative hypertension may be a result of carotid baroreceptor trauma and it is more prominent in patients with preoperative hypertension. Both postoperative hypertension and hypotension are common after CEA. O'Conner and Tuman [10] suggested that hypertension is seen in 25 58% of patients after endarterectomy and hypotension in 8 10% of patients.

CEA is a prophylactic operation which only yields benefit if the risks of surgery are less than the risks of medical management. The anaesthetist has an important role in controlling perioperative risk in CEA.

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