Case Report

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Middle Cerebral Artery (MCA) Bifurcation Aneurysm Clipping Under Opioid Free Anesthesia: A Case Report

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Abstract

Opioid free anesthesia in Neurosurgery is an evolving concept in light of recent era. Classic convention of using opioid as a cornerstone for provision of analgesia during neurosurgeries is now being shaken off by the ongoing emerging evidence. The present case report aims to bring out the successful usage of opioid free anesthesia during aneurysm clipping surgery. We discuss in detail the various pros and cons including pitfalls in the literature review alongwith this case report.

Keywords: Opioid free anesthesia; Dexmeditomidine; Surgical plethysmographic index; Aneurysm.

INTRODUCTION

Despite widespread attention to the hazards of opioid agents, opioid misuse remains a leading cause of accidental death.[1,2] The first episode of opioid consumption can be traced to the peri-operative period.[3] These risks mandate strategies to minimize and eliminate opioid exposure wherever possible. There are multiple opportunities for the anaesthetist, surgeon and institution to reduce opioid exposure and minimize patient harm. The best characterized clinical strategies include the use of multimodal analgesia and enhanced recovery after surgery (ERAS) initiatives to standardize care and improve outcomes while providing satisfactory peri-operative pain control. Opioid-free anesthesia (OFA) has recently been receiving interest as a potential strategy to fill this knowledge gap. It is a technique in which no intraoperative opioid is administered via any route, including systemic, neuraxial, or tissue infiltration. Initially pioneered forbariatric surgery, the technique relies on combinations of non-opioid drugs like propofol, dexmeditomidine, lidocaine, magnesium and ketamineto produce anesthesia, sympatholysis and analgesia.[4,5]

The intracranial aneurysms pose a requirement of prompt surgical or neuro-interventional management in view of risks of re-bleeding and vasospasm. Initial medical measures after aneurysmal bleed include mild sedation and analgesics for anxiety and headache, prevention of arterial blood pressure elevation and avoidance of anti-platelet agents.[6] Blood pressure frequently rises precipitously as a result of the initial intracranial hemorrhage, due to Cushing response, although unbearable headache and anxiety may also be contributing factors. Due to these conventions, many anaesthesiologists refrain from adopting

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opioid free anesthesia regimens for cerebral aneurysmal clipping.[7] In this case report, which meets the requirements of the **CARE (Case Report)** guidelines, we describe the successful use of A for MCA bifurcation aneurysm clipping.

Stem Case

The patient was a 39-year-old male having a MCA bifurcation aneurysm of WFNS (World Federation of Neurosurgical Societies) grade 1 and Claasen grade 3. He was a known hypertensive, on irregular medication. The patient presented on day 3 of ictus. Cerebral Magnetic resonance angiography (MRA) demonstrated MCA bifurcation aneurysm (fig. 1). Pre-operatively the patient received acetaminophen 1g iv. The baseline vitals recorded by GE Carescape B650 monitor (Finland) were BP = 183/102 mmHg, HR= 76 /min, SpO2= 99%. In order to ensure balanced anesthesia for induction and maintenance of transmural pressure gradient across the aneurysm, we decided to use etomidate 0.3 mg/kg i.v, vecuronium 0.15 mg/kg iv, lidocard 1 mg/kg iv and dexmeditomidine infusion. Firstly, balanced intravenous crystalloid solution (lactated Ringer's solution) was administered (8–12 ml/kg/hr) after securing a large bore venous access after prior infiltration of skin with local anesthetic. Dexmeditomidine infusion was initiated @ 1 mcg/kg/hr for 10 minutes followed by subsequent administration of etomidate 0.3 mg/kg iv, vecuronium 0.15 mg/kg iv, lidocard 1 mg/kg iv bolus. During this period of anesthetic induction 100% oxygen was being given to the patient through facemask (6 L/min). The anesthetic plane was further deepened with concomitant administration of sevoflurane at 2 MAC (minimum alveolar concentration). Patient underwent smooth induction and intubation. Dexmeditomidine infusion was subsequently titrated as per the mean arterial pressure within ± 20% of each patient's baseline value and the surgical plethysmographic index (SPI) value (which served as a surrogate for adequacy of analgesia). Patient was ventilated on pressure controlled mode using Air: Oxygen mixture along with sevofluraneuntil 1 MAC, on a Drager Fabius XL anesthesia workstation. Mechanical ventilation was achieved with 1:1 mixture of oxygen:air ($FiO_2$0.5) with a tidal volume of 6–8 ml/kg and respiratory rate of 11–14 titrated to an end-tidal carbon dioxide between 30–34 mm Hg. Antiemetic therapy with ondansetron (6mg) was provided.

Bilateral scalp block was given using the injectate mixture of 0.25% bupivacaine and 1% lidocaine. The head was then placed on Mayfield pins and surgery commenced. 3% NaCl 100 ml was used for osmotherapy. However, the neurosurgeon requested for 20% mannitol as the dura was still appearing tense, therefore 100 ml of this solution was subsequently infused. The durotomy was completed and brain appeared lax. The SPI was maintained between 38 to 46 with dexmeditomidine infusion and systolic blood pressure (SBP) was maintained between 142 to 158 mm Hg. Peripheral oxygen saturation (SpO_2) remained stable at 100% throughout the surgery.

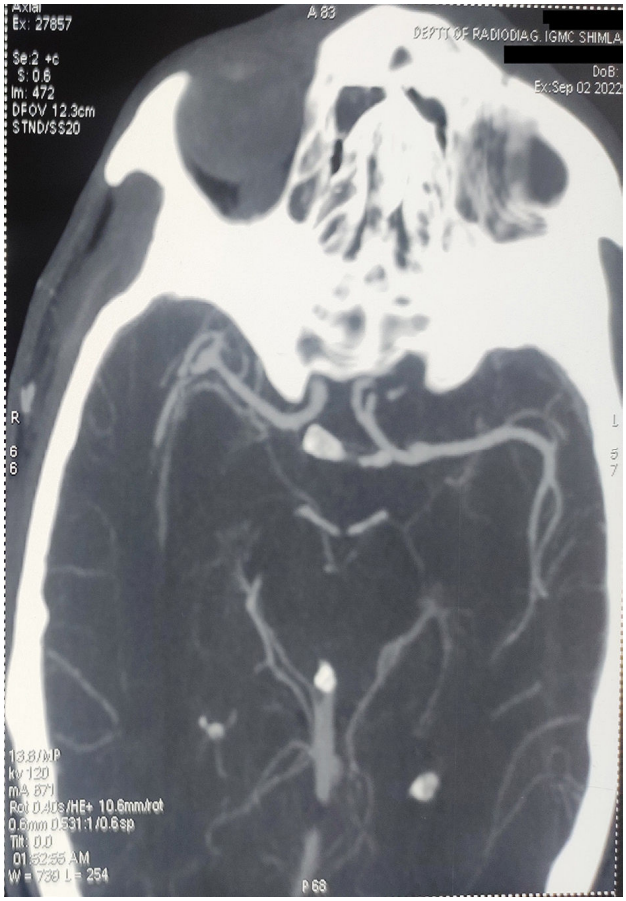

Fig. 1: Correct label is MR angiogram

Dissection was continued until the MCA bifurcation was visualized. A temporary clip was placed for 5 minutes. But the permanent straight clip could not be placed correctly, therefore this temporary clip was removed. After 10 minutes of reperfusion of the vessel, a permanent clip was applied directly on the aneurysm, however it bled after 1 minute of application of the clip. Therefore in order to control the bleed, the surgeon applied another temporary clip on the feeding artery. Thereafter a right angle permanent clip was applied on the aneurysm. Nevertheless the second temporary clip time was 18 minutes.

SPI and vitals remained stable during this period (Table 1). Blood loss of 700 ml occurred during the aneurysmal rupture. One PRBC was transfused. Rest of the surgery went uneventful. After stopping the inhalational agents and dexmeditomidine infusion, the reversal from residual muscle relaxation was ensued using neostigmine. The patient was shifted for neuro-intensive care to ICU. Postoperative pain scores were assessed using Critical Care Pain Observational Tool (CCPOT) (Table 2). The patient was ventilated overnight during which he was sedated with midazolam: morphine infusion. The total post-operative consumption of morphine infused was 22 mg. In morning the sedation was cut off at 6 o'clock and he was given a T- piece trial which he tolerated well. His GCS was $E_4V_5M_6$, at the time of extubation. There was postoperative deficit of left side hemiparesis paresis.

Table 1: The essential time points

Time points	B.P (mmHg)	H.R (beats/min)	S.P.I
Baseline vitals	178/100	76	86
Initiation of Opioid free Analgesia	Dexmeditomidine@ 1 μg/kg/hr for ten minutes followed by titration to 0.5 μg/kg/hr		
During mask ventilation	160/90	65	54
During induction	156/88	61	50
During Intubation	159/91	67	53
Immediately after intubation	154/89	65	52
During Scalp block	150/85	63	49
After Scalp block	152/86	60	45
Before positioning	153/88	59	47
During positioning	156/90	56	45
During application of Mayfield Pins	159/90	54	44
During incision	154/87	50	42
During durotomy	143/82	56	45
Application of Temporary Clip	128/76	66	43
Application of Permanent Clip	122/72	68	48
During closure of Dura	132/70	73	51
During closure of Skin wound	136/74	71	55

Table 2: Critical care pain observational tool

Indicator	Description	Score	
Facial expression	No muscular tension observed	Relaxed, neutral	0
	Presence of frowning, brow lowering, orbit tightening and levator contraction	Tense	1
	All of the above facial movements plus eyelid tightly closed	Grimacing	2
Body movements	Does not move at all (doesn't necessarily mean absence of pain) or normal position (movements not aimed toward the pain site or not made for the purpose of protection)	Absence of movements	0
	Slow, cautious movements, touching or rubbing the pain site, seeking attention through movements	Protection	1
	Pulling tube, attempting to sit up, moving limbs/thrashing, not following commands, striking at staff, trying to climb out of bed	Restlessness	2
Muscle tension	No resistance to passive movements	Relaxed	0

Table Cont...

Evaluation by passive flexion and extension of upper limbs when patient is at rest or evaluation when patient is being turned	Resistance to passive movements	Tense, Rigid	1
	Strong resistance to passive movements or incapacity to complete them	Very tense, Rigid	2
Compliance with the ventilator (intubated patients) OR	Alarms not activated, easy ventilation	Tolerating ventilator or movement	0
	Coughing, alarms may be activated but stop spontaneously	Coughing but tolerating	1
	Asynchrony: blocking ventilation, alarms frequently activated	Fighting ventilator	2
Vocalisation (extubated patients)	Talking in normal tone or no sound	Talking in normal tone or no sound	0
	Sighing, Moaning	Sighing, Mmoaning	1
	Crying out, Sobbing	Crying out, Sobbing	2

DISCUSSION

There is accumulating level 1 and 2 evidence to support the use of opioid free anesthesia as an equipotent analgesic to opioid-based analgesic regimens. **Sriganesh *et al.*[8]** compared SPI and biomarkers of surgical stress between fentanyl and dexmedetomidine analgesia in 24 patients aged 18 to 60 years undergoing elective craniotomies for brain tumor resection under general anesthesia. After randomization, they received either fentanyl 1 µg/kg/hriv or dexmedetomidine 0.5 µ/kg/ hr iv infusion as the primary intraoperative analgesic. In the statistical analysis the SPI, surgical stress-cortisol, blood glucose and pH was not significantly different between the two study groups. However postoperative white blood cell count was higher in the fentanyl group. The added advantage of improved postoperative analgesia, shorter duration of stay in the post-anesthesia care unit (PACU) and reduced postoperative nausea and vomiting (PONV) is making this technique popular. It will be indispensible in patients at high risk of opioid-related complications, chronic pain conditions, opioid misuse disorder and obstructive sleep apnoea. To date, there are only sparing reports of OFA for cerebral aneurysm surgery.

Opioids can depress the central respiratory drive leading to post-operative hypoventilation. Hypoventilation can lead to cerebral vasodilatation and increase in intracranial pressure which can result in global hypoperfusion. The incidence of rebleeding from a ruptured aneurysm isaround 30% in the first month following SAH, with the occurrence of 4 to 13.6% in the first 24 hours. Re-rupture is associated with high mortality and poor outcome. Early obliteration of the aneurysm reduces the risk of re-bleeding from the natural course. In patients with significant risk of re-bleeding but with unavoidable delay of definitive treatment, short-term anti-fibrinolytic therapy with tranexamic acid or aminocaproic acid may be considered as a temporary measure to reduce the incidence of aneurysm re-bleeding.

Rebleeding is also common in patients undergoing treatment interventions of ruptured aneurysms. The multicenter study of Cerebral Aneurysm Rupture After Treatment (CARAT) reported an intra-procedure rupture rate of 19% with clipping and 5% with endovascular coiling.[9] Intraoperative rupture of the aneurysm results in high morbidity and mortality. Although intraoperative aneurysm re-rupture is most commonly due to surgical manipulation of the aneurysm, prevention of aneurysm re-rupture by aggressively limiting the degree and duration of systemic hypertension has been one of the major goals for the an aesthesiologist.

A deep plane of anesthesia needs to be ensured in order to prevent acute increases in blood pressure during stressful events, such as laryngoscopy and endotracheal intubation, head pinning, tissue dissection and dural opening. Meticulous care is needed to control ventilation. For patients without intracranial hypertension, the goal is to keep the patient normocarbic, avoiding decreases in intra-cranial pressure (ICP) before the dura is opened, while employing subsequent mild hyperventilation and mannitol to relax the brain, facilitate surgical exploration and reduce the potential damage from tissue retraction. Local anesthetics can help to avoid acute cardiovascular effects secondary to head pinning and extradural tissue dissection.

During surgical clipping, maintaining adequate blood pressure is important to ensure adequate

cerebral perfusion to reduce injury from ischemic insults. Intraoperative blood pressure is typically controlled to maintain cerebral perfusion pressure between 70 and 90 mmHg. The retractor-induced cerebrali schemia gets accentuated during hypotension. Transient induced hypotension may infrequently be requested by the surgeon immediately prior to clipping the aneurysm to allow for more secure placement of the permanent clip on the neck of the aneurysm. In addition, if intraoperative aneurysm rupture occurs, induced hypotension will decrease the rate of bleeding into the surgical field, thereby helping the surgeon gain better control. In contrast, induced hypertension may be needed to raise blood pressure above baseline levels to augment collateral blood flow, if the surgeon applies temporary clipping of the feeding arteries to reduce the rupture risk of the aneurysm during surgical manipulation of the aneurysm dome.

Although the use of temporary clips decreases the risk of rebleeding during surgical manipulation, this practice results in an area of cerebral ischemia, which can be minimized by using controlled hypertension. Blood pressure management can also be guided by intraoperative neurophysiological monitoring, such as electroencephalography and somatosensory-evoked potential and motor-evoked potential monitoring.

Chakrabarti *et al.*[10] conducted a prospective randomized study on 49 patients undergoing cerebello-pontine angle (CPA) surgeries. The dexmeditomidine group had 25 patients whereas the control group had 24 patients. Anaesthetic induction was standardized in the cohort. Propofol was used for intraoperative maintenance of anesthesia. The BIS was kept between 40 and 60. Intraoperative analgesia was maintained with fentanyl 0.5 µg/kg/hr along with the interventional drugdexmedetomidine (0.5 µg/kg/hour) or the control infusion of normal saline. The patients underwent cranial nerve EMG monitoring, therefore no muscle relaxants were administerd. The data analysis revealed that the total dose of propofol and fentanyl (adjusted for duration of surgery, body weight and number of extra boluses) was significantly lower in the dexmedetomidine group. The recovery from anaesthesia, hypotension, hypertension and tachycardia was similar between the two groups. However, bradycardia was significantly higher in the dexmedetomidine group.

In a systematic meta-analysis by **Peng *et al.*[11]** regarding efficacy and safety of dexmedetomidine as an anesthetic adjuvant for patients undergoing intracranial surgery, 8 RCT were evaluated. Their analysis revealed that patients treated with dexmeditomidine required less intraoperative treatment for hypertension (p=0.001) and hypotension (p=0.05) and less postoperative treatment for hypertension (p=0.01) and tachycardia (p=0.007) compared with placebo. The interventional group had lower mean arterial pressure and heart rate after extubation (p=0.04) and lower postoperative antiemetic requests (p=0.003). This meta-analysis shows evidence that dexmedetomidine as an anesthetic adjuvant during intracranial procedures leads to better perioperative hemodynamic control, less intraoperative opioid consumption and fewer postoperative antiemetic requests.

Kumar *et al.*[12] described a rare case where aneurysmal rupture occurred during meningioma resection. They emphasized on the maintenance of stable transmural pressure and adequate cerebral perfusion pressure as a key goal of neuroanaesthesia. The present case report also justifies the above objective by the use of dexmeditomidine as a part of the OFA in order to maintain optimal anaesthesia and analgesia for aneurysmal clipping.

CONCLUSION

Dexmeditomidine as a part of OFA for aneurysm clipping surgery holds a potential area of interest. With the development of newer pain index monitoring machines like SPI, ANI *etc.*, it has become safer to use OFA in the situations where the traditional mindset has been to use opioid only.

Limitations

This is a case report of single patient. Large study cohort is required to validate the findings of this work.

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