Int J Clin Exp Med 2015;8(7):11534-11538
www.ijcem.com /ISSN:1940-5901/IJCEM0009866

Original Article

Anesthetic management for craniotomy in a patient with massive cerebellar infarction and severe aortic stenosis: a case report

Ai-Jun Xu, Zhi-Gang He, Xiao-Hua Xia, Hong-Bing Xiang

Department of Anesthesiology and Pain Medicine, Tongji Hospital of Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430030, Hubei, PR China

Received May 4, 2015; Accepted July 10, 2015; Epub July 15, 2015; Published July 30, 2015

Abstract: Severe aortic stenosis combined with coronary heart disease remarkably increases the risk of perioperative morbidity and mortality during noncardiac surgery. Surgery and anesthesia often complicate the perioperative outcome if adequate monitoring and proper care are not taken. Therefore, understanding of the hemodynamic changes and anesthetic implications is an important for successful perioperative outcome. This report described the anesthetic management of a patient with a massive cerebellar infarction who was diagnosed with severe aortic stenosis combined with moderate aortic insufficiency and coronary heart disease and hypertension. He was prepared for aortic valve replacement and coronary bypass operation before massive cerebellar infarction occurred. And he received decompressive craniotomy and external ventricular drainage in the prone position under general anesthesia with endotracheal intubation.

Keywords: Aortic stenosis, massive cerebellar infarction, craniotomy

Introduction

Severe aortic stenosis (AS) combined with coronary heart disease can increase the risk for anesthesia and has been considered as important risk factor for development of cardiac complications in patients undergoing noncardiac surgery [1]. Perioperative mortality for patients with severe AS is as high as 13% in the 1970s [2]. Sawhney et al reported of the anesthetic management of a patient with AS who underwent craniotomy for excision of the tumor after undergoing prophylactic Balloon aortic valvuloplasty (PBAV) [3]. This is a case report of the anesthetic management of a patient with severe AS combined with moderate aortic insufficiency (AI) and Coronary heart disease who had a massive cerebellar infarction. He underwent decompressive craniotomy and external ventricular drainage in the prone position under general anesthesia. Written informed consent was obtained before preparation and submission of this manuscript.

Case report

A 57-year-old man with a massive cerebellar infarction presented for emergency surgery with a 3-days of coma prior to admission. He was prepared for aortic valve replacement and coronary bypass operation because he was diagnosed with severe AS and moderate AI and coronary heart disease before massive cerebellar infarction occurred to him.

The patient has received endotracheal intubation and mechanical ventilation before he was sent to the operating room. His heart rate was 89 beats/min and blood pressure was 170/85 mm Hg. There was an ejection systolic murmur (grade 3/6) in the aortic area with radiation to neck. Breath sounds were harshness during auscultation. Results of routine hematologic showed WBC was very high 26.19×10^9 and biochemical investigations were within normal limits. Doppler echocardiography showed that severe AS coexisted with moderate aortic insuf-

ficiency, the area of aortic was 0.9 cm^2 and systolic aortic valve gradient of 86 mmHg with concentric left ventricular hypertrophy. The ejection fraction was 54%. Coronary artery computed tomography (CT) found that coronary atherosclerosis, anterior descending artery in 10%, left circumflex stenosis in 85%, 60%-70% right coronary artery stenosis. Valvular heart disease, aortic valve disease, the ascending aorta widened. Brain CT showed large area cerebral infarction of cerebellum and hydrocephalus.

Since this patient was to undergo decompressive craniotomy and external ventricular drainage in the prone position, it was thought that he had a high risk for developing disastrous intraoperative and postoperative cardiac complications with more difficulty for urgent rescue. As there was severe AS combined with moderate insufficiency, and massive cerebellar infarction with brain hernia information, aortic valve replacement or palliative BAV could not be performed prior to emergency surgery. Preinduction vital signs included a heart rate of 89 beats/min and right arm blood pressure of 156/76 mm Hg. The patient has been inserted of a three-lumen central venous catheter and a left radial artery catheter for arterial pressure monitoring before he entered operating room. Perioperative monitoring included electrocardiography II and V5, direct arterial pressures, pulse oximetry, end-tidal CO_2 pressure, central venous pressure, temperature, arterial blood gas levels, urine output and hemodynamic data. Etomidate (0.3 mg/kg), sufentanil (1 μg/kg) and rocuronium (0.6 mg/kg) were administered for anesthesia induction [4, 5]. Then we changed the intubation catheter with reinforced tracheal tube for prone position and controlled ventilation was instituted to maintain an end-tidal CO_2 pressure in the range of 32 to 40 mm Hg. Anesthesia was maintained with O_2 and air (FiO_2, 0.5), sevoflurane (1%~2%) and remifentanil (0.1~0.2 μg/kg/min) continuous pump infusion. Intraoperative recording bispectral index (BIS) was used to monitor the depth of anesthesia [6-8].

Soon after the patient changed to prone position, he developed high airway pressure to 32 cm H_2O. Then we checked the ventilation tube and given sputum suction. And airway pressure recovered to 15~20 cm H_2O. The blood pressure decreased to 105/70 mmHg during sterilization, then phenylephrine 40 ug was given and the BP did not respond well. So small dose of dopamine infusion (5 μg/kg/min) was started and BP was maintained about 120~130/70~80 mmHg. CVP was 14~16 cm H_2O during operation by appropriate volume supplement of crystolloid and colloid. The rest of the surgery was uneventful, and there were no further hemodynamic or respiratory disturbances.

Surgery lasted 4 hours, and the blood loss was about 400 mL with a urine output of 1,200 mL. The patient received 1.5 L of Ringers solution, 500 mL of Gelofusion, and 1 unit of packed red blood cells. And he was later transferred to the intensive care unit after he received tracheotomy. He continued to receive mechanical ventilation for 2 days after surgery. The vital signs remained stable, and he was discharged 25 days after operation.

Discussion

Patients with severe AS combined with coronary heart diseases undergo noncardiac surgery have always been considered to be a high-risk group for perioperative cardiac morbidity. Goldman et al found 13% (3/23 patients) cardiac mortality among patients with AS for noncardiac surgery [2]. At Erasmus Medical Center in The Netherlands between 1991 and 2000, cardiac complications (cardiac death or nonfatal myocardial infarction within 30 days of surgery) occurred in 15/108 (14%) patients with AS, with the majority of these complications being cardiac death [9]. Several recent studies demonstrate lower perioperative mortality for AS patients. The Mayo Clinic reported that no intraoperative events, but 11% major postoperative events happened among 19 patients with severe AS [10, 11]. Another recent study showed that no severe cardiac events happened in 30 patients with asymptomatic severe AS underwent low or intermediate-risk noncardiac surgery [10]. However, most of these operations were short time and simple surgery such as diagnostic procedures. To our knowledge, no reports of patients with severe AS combined with AI undergoing decompressive craniotomy procedures for massive cerebellar infarction.

There is a significant association between cardiac disease and ischemic stroke. Preexisting cardiac disease or cardiac dysfunction is relat-

ed to the acute neurohumoral and autonomic stress response to stroke [12-18]. And the patient we reported in this case was a topical example for this situation. He was diagnosed with severe AS and moderate AI combined with coronary disease before he suffered massive cerebellar infarction. Even though he was treated by pharmacological therapy for heart surgery, the severe stroke occurred to him preoperatively by unknown reason.

Recent studies suggested that decompressive craniectomy could improve outcome in patients with malignant infarction after stroke [19]. However, the cardiac risk during and after acute stroke have been reported higher than other noncardiac surgery. Decompressive craniectomy involves emergency surgery in patients with severe stroke have been identified as risk factors for perioperative cardiac events. Durga et al reported that the incidence of Serious cardiac adverse events after decompressive craniectomy for stroke was 47.6% and the mortality in patients with Serious cardiac adverse events was 75% [20].

In this patient, who was diagnosed with severe AS coexisted with moderate AI and coronary diseases, hypertension and was prepared for aortic valve replacement and coronary bypass operation. During the period of preparing for the heart surgery, the patient received careful cardiac examination and medical treatment for hemodynamic balance. Because the complicated heart valve and coronary diseases coexisted, he could not receive any cardiac invasive treatment before decompressive craniotomy surgery. Even though the patient received dehydrated therapy with mannitol and furosemidum, the blood pressure was still 156/76 mmHg when he entered the operating room. Anesthesia was performed with etomidate to avoid hypotention during induction. And we chose sufentanil as analgesia agent which has strong analgesia effects and little influence on hemodynamics.

The key factor for the anesthesia maintenance in patients with severe AS and AI is to maintain myocardial oxygen supply and demand balance. Heart rate was kept between 70 and 80 beats per minute in patient who suffer severe AS coexisted AI, β-blockers such as esmolol can be continuously pumped to maintain appropriate HR. CVP was maintained 14 and 16 cm H_2O, which assured an appropriate intravascular volume status and avoided cardiac ischemia. Phenylephrine and small dose of dopamine were used to treat hypotension.

When moving the patient into the prone position, airway pressure changed to 32 cm H_2O suddenly and a large amount of secretion and sputum was extracted and then the airway pressure recovered to 15 and 20 cm H_2O. We reviewed the case report and found that the patients suffered pulmonary infection after coma. So sputum blocked the trachea after he was changed to prone position. Meanwhile, clinical studies found that decrease in cardiac index (CI) when surgical patient is in prone postion [21-32]. So we put a defibrillator in standby mode near the operation table.

The patient experienced preparing for heart surgery before stroke and his left ventricular function was improved with medical treatment. Normal heart function as well as careful anesthesia management may have contributed to a satisfactory outcome in our patient undergoing decompressive craniotomy. Meanwhile, carefully operation procedures and less blood loss were important for the patient surviving the surgery. In conclusion, there was high risk with severe AS and AI with coronary heart disease for the patient undergoing decompressive craniotomy for massive cerebellar infarction in prone position, and he could not be performed PBAV for combined AI, but he survived perioperative period with careful administration of anesthesia along with appropriate monitoring. It suggested that improved ventricular function is very important for noncardiac surgery with severe AS and other heart diseases. Patients with more complicated heart valve diseases may need prior correction or invasive treatment of the underlying cardiac injury.

Disclosure of conflict of interest

None.

Address correspondence to: Dr. Hong-Bing Xiang, Department of Anesthesiology and Pain Medicine, Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, Wuhan 430030, Hubei, PR China. E-mail: xhbtj2004@163.com

References

[1] Wright DE and Hunt DP. Core competency review: aortic stenosis and noncardiac surgery. J Hosp Med 2012; 7: 655-660.

[2] Goldman L, Caldera DL, Nussbaum SR, Southwick FS, Krogstad D, Murray B, Burke DS, O'Malley TA, Goroll AH, Caplan CH, Nolan J, Carabello B and Slater EE. Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 1977; 297: 845-850.

[3] Sawhney R, Abraham M, Ganjoo P and Tandon MS. Anesthetic considerations in a patient with severe aortic stenosis for craniotomy. J Neurosurg Anesthesiol 2003; 15: 151-154.

[4] Iwasaki H, Takahoko K, Otomo S, Sasakawa T and Kunisawa T. Monitoring of neuromuscular blockade in one muscle group alone may not reflect recovery of total muscle function in patients with ocular myasthenia gravis. Can J Anaesth 2013; 60: 1222-1227.

[5] Eikermann M, Groeben H, Husing J and Peters J. Accelerometry of adductor pollicis muscle predicts recovery of respiratory function from neuromuscular blockade. Anesthesiology 2003; 98: 1333-1337.

[6] Girgirah K and Quinn A. Reliability of bispectral index analysis in patients undergoing Caesarean section. Br J Anaesth 2015; 114: 530.

[7] Kim H, Lim BG and Lee SY. Transcranial electrical stimulations given for motor-evoked potentials as the cause for elevated bispectral index and entropy during spine surgery. J Neurosurg Anesthesiol 2013; 25: 217-219.

[8] Witkowska M, Karwacki Z, Rzaska M, Niewiadomski S and Sloniewski P. Comparison of target controlled infusion and total intravenous anaesthesia with propofol and remifentanil for lumbar microdiscectomy. Anaesthesiol Intensive Ther 2012; 44: 138-144.

[9] Kertai MD, Bountioukos M, Boersma E, Bax JJ, Thomson IR, Sozzi F, Klein J, Roelandt JR and Poldermans D. Aortic stenosis: an underestimated risk factor for perioperative complications in patients undergoing noncardiac surgery. Am J Med 2004; 116: 8-13.

[10] Calleja AM, Dommaraju S, Gaddam R, Cha S, Khandheria BK and Chaliki HP. Cardiac risk in patients aged >75 years with asymptomatic, severe aortic stenosis undergoing noncardiac surgery. Am J Cardiol 2010; 105: 1159-1163.

[11] Torsher LC, Shub C, Rettke SR and Brown DL. Risk of patients with severe aortic stenosis undergoing noncardiac surgery. Am J Cardiol 1998; 81: 448-452.

[12] Min J, Farooq MU, Greenberg E, Aloka F, Bhatt A, Kassab M, Morgan JP and Majid A. Cardiac dysfunction after left permanent cerebral focal ischemia: the brain and heart connection. Stroke 2009; 40: 2560-2563.

[13] Liu TT, He ZG, Tian XB and Xiang HB. Neural mechanisms and potential treatment of epilepsy and its complications. Am J Transl Res 2014; 6: 625-630.

[14] Xiang HB, Zhu WZ, Guan XH and Ye DW. The cuneiform nucleus may be involved in the regulation of skeletal muscle tone by motor pathway: a virally mediated trans-synaptic tracing study in surgically sympathectomized mice. Brain 2013; 136: e251.

[15] Xiang HB, Zhu WZ, Guan XH and Ye DW. Possible mechanism of deep brain stimulation for pedunculopontine nucleus-induced urinary incontinence: a virally mediated transsynaptic tracing study in a transgenic mouse model. Acta Neurochir (Wien) 2013; 155: 1667-1669.

[16] Ye DW, Liu C, Tian XB and Xiang HB. Identification of neuroanatomic circuits from spinal cord to stomach in mouse: retrograde transneuronal viral tracing study. Int J Clin Exp Pathol 2014; 7: 5343-5347.

[17] Ye DW, Liu C, Liu TT, Tian XB and Xiang HB. Motor cortex-periaqueductal gray-spinal cord neuronal circuitry may involve in modulation of nociception: a virally mediated transsynaptic tracing study in spinally transected transgenic mouse model. PLoS One 2014; 9: e89486.

[18] Xiang HB, Liu C, Liu TT and Xiong J. Central circuits regulating the sympathetic outflow to lumbar muscles in spinally transected mice by retrograde transsynaptic transport. Int J Clin Exp Pathol 2014; 7: 2987-2997.

[19] Mostofi K. Neurosurgical management of massive cerebellar infarct outcome in 53 patients. Surg Neurol Int 2013; 4: 28.

[20] Durga P, Meena AK, Panigrahi MK, Sahu BP and Ramachandran G. Serious cardiac adverse events after decompressive craniectomy for malignant cerebral infarction. J Neurosurg Anesthesiol 2011; 23: 236-240.

[21] Olsen KS, Petersen JT, Pedersen NA and Rovsing L. Self-positioning followed by induction of anaesthesia and insertion of a laryngeal mask airway versus endotracheal intubation and subsequent positioning for spinal surgery in the prone position: a randomised clinical trial. Eur J Anaesthesiol 2014; 31: 259-265.

[22] Schmittner MD, Dieterich S, Gebhardt V, Weiss C, Burmeister MA, Bussen DG and Viergutz T. Randomised clinical trial of pilonidal sinus operations performed in the prone position under spinal anaesthesia with hyperbaric bupivacaine 0.5% versus total intravenous anaesthesia. Int J Colorectal Dis 2013; 28: 873-880.

[23] Dube SK, Bharti SJ and Rath GP. Frequent Ventricular Premature Contractions under Anaesthesia in Prone Position. J Anaesthesiol Clin Pharmacol 2011; 27: 142-143.

[24] Cuvas O, Gulec H, Karaaslan M and Basar H. The use of low dose plain solutions of local an-

aesthetic agents for spinal anaesthesia in the prone position: bupivacaine compared with levobupivacaine. Anaesthesia 2009; 64: 14-18.
[25] Batra YK and Sharma A. Severe bradycardia in a myasthenic patient undergoing percutaneous nephrolithotomy in prone position under general anaesthesia. Ann Card Anaesth 2006; 9: 62-63.
[26] Soro M, Garcia-Perez ML, Belda FJ, Ferrandis R, Aguilar G, Tusman G and Gramuntell F. Effects of prone position on alveolar dead space and gas exchange during general anaesthesia in surgery of long duration. Eur J Anaesthesiol 2007; 24: 431-437.
[27] Sudheer PS, Logan SW, Ateleanu B and Hall JE. Haemodynamic effects of the prone position: a comparison of propofol total intravenous and inhalation anaesthesia. Anaesthesia 2006; 61: 138-141.
[28] Duke K, Aubin CE, Dansereau J and Labelle H. Biomechanical simulations of scoliotic spine correction due to prone position and anaesthesia prior to surgical instrumentation. Clin Biomech (Bristol, Avon) 2005; 20: 923-931.
[29] Anand S and Mushin AS. Cavernous sinus thrombosis following prone position anaesthesia. Eye (Lond) 2005; 19: 803-804.
[30] Gattas DJ and Weber LS. Anaesthesia in the prone position for impalement injury. Anaesth Intensive Care 1999; 27: 421-423.
[31] Sesay M, Tauzin-Fin P, Verdonck O, Svartz L and Maurette P. Clinical evaluation of the Capnomask in the supine vs. prone position during monitored anaesthesia care. Eur J Anaesthesiol 2008; 25: 769-771.
[32] Edgcombe H, Carter K and Yarrow S. Anaesthesia in the prone position. Br J Anaesth 2008; 100: 165-183.