International Journal of General Medicine

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CASE REPORT

Venoarterial Extracorporeal Membrane Oxygenation as an Effective Therapeutic Support for Refractory Cardiac Arrest in the Setting of Spinal Anesthesia: A Case Report and Literature Review

This article was published in the following Dove Press journal:
International Journal of General Medicine

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Abstract: Cardiac arrest is the most serious event among the complications associated with spinal anesthesia. Spinal anesthesia reduces the release of catecholamines and impairs neuroendocrine response following cardiac arrest, which contributes cardiopulmonary resuscitation (CPR) more difficult. Venoarterial extracorporeal membrane oxygenation (VA-ECMO) may be a bridge to provide a more effective and durable mechanical solution under this extremely critical condition. This study reports a 50-year-old man who was scheduled to undergo surgical great saphenous vein varices under spinal anesthesia. A sudden cardiac arrest occurred after spinal anesthesia. Standard CPR was performed and large doses of vascular drugs are administered, but the effect of resuscitation was still poor. We fastly initiated VA-ECMO to provide cardiopulmonary support for this refractory cardiac arrest. Fortunately, the patient was successfully resuscitated with complete recovery. In summary, standard CPR might more difficult during spinal block anesthesia. Quick-started VA-ECMO is a potential option under this situation, which protects the patient from further harm from repeated prolonged CPR, refractory hypotension and deteriorated desaturation, and therefore benefit for patient in this critical condition.

Keywords: cardiac arrest, spinal anesthesia, cardiopulmonary resuscitation, extracorporeal membrane oxygenation

Introduction

Spinal anesthesia is an anaesthetic technique that injects local anaesthetics in the subarachnoid cerebrospinal fluid surrounding the spinal cord of the body to block nerve function. It is an integral part of the daily routine of countless anesthesiologists. However, the benefits of spinal anesthesia in patients are accompanied with some complications. Among them, cardiac arrest is the most serious event. It was reported that the cardiac arrest after spinal anesthesia was ranging from 2.73 to 6.4 per 10,000 with a survival rate from 20% to 77%.[1] Spinal anesthesia can reduce the release of catecholamines and impair neuroendocrine response, which results in standard Cardiopulmonary Resuscitation (CPR) more difficult.[2] In clinical, Extracorporeal Membrane Oxygenation (ECMO) can provide effective support for refractory circulatory shock.[3,4] We report a patient with refractory cardiac arrest

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http://doi.org/10.2147/IJGM.S285939

was successfully resuscitated using Venoarterial Extracorporeal Membrane Oxygenation (VA-ECMO) in the setting of spinal anesthesia.

Case Report

A 50-year-old man was scheduled to undergo surgical ligation, stripping and foam sclerotherapy of great saphenous vein varices under spinal anesthesia. The patient had no history of hypertension, coronary artery disease, epilepsy or allergic reaction. Physical cardiopulmonary examination before surgery was unremarkable. Preoperative electrocardiogram and laboratory test of blood were also unremarkable.

After the patient coming into the operation room, an standard American Society of Anesthesiologists (ASA) monitoring was placed. Meanwhile, an intravenous cannula was inserted at the left arm to establish an infusion access. The patient's vital signs before spinal anesthesia are as follows: Non-invasive blood pressure (NIBP): 123/68 mm Hg; Heart rate (HR): 86 beats per minute; Saturation of pulse oxygen (SpO2): 97%. The patient then was placed in the lateral decubitus on the operating table with the assistance of one nurse. The puncture area was strictly disinfected and covered with a sterile sheet. After anesthetizing the puncture site with 2% lidocaine 2.5mL, the anesthesiologist performs spinal anesthesia between L3-L4 space with a 25G spinal needle. When positive reflux of clear non-colored cerebral spinal fluid was observed, a single 2.6mL (13 mg) bolus of 0.5% hyperbaric ropivacaine was administered. After the performance completed, the patient was placed supine. No additional sedatives or antibiotics were administrated until this time. Approximately 15 minutes after intrathecal injection, a T8 sensorial blockade level was achieved by pinprick (analgesia) method. The surgeon then was beginning to prepped the operating area. At this time, the patient reported anxiety, vomit and nausea with a heart rate downing (from 84 beats per minute to 67 beats per minute) associated with a hypotension (78/45 mm Hg). We accelerated immediately the infusion of hydroxyethyl starch fluids. In view of the sympathetic nervous system blockade after spinal anesthesia resulting in cardioaccelerator fiber suppression and vasodilatation, atropine 0.5mg and phenylephrine 100 μg (HR was 70 beats per minute at this point) were administered intravenously. As only 15 minutes after spinal anesthesia, we did not place the patient in the "Trendelenberg position" for accelerating venous return to the heart at this time, which may cause a higher level of blockade and make hemodynamics worse. We carefully evaluated the patient again and there was no evidence of flush, urticaria, or facial edema in this patient. However, the patient quickly became unresponsive and the rhythm of ECG became progressively bradycardia to zero with blood pressure and SpO2 unable measure. Cardiac arrest was quickly diagnosed, and standard CPR was immediately performed. Meanwhile, full advanced cardiac life support (ACLS) protocol was initiated. The patient was performed trachea intubation immediately and placed on mechanical ventilation. With the assistance of ultrasound, an internal jugular vein catheterization and femoral artery catheterization were quickly established. Intermittent intravenous epinephrine was administrated for progressive resuscitation. Meanwhile, according to the results of arterial blood gas, sodium bicarbonate was given to regulate the internal environment. However, the heart rate of patient occasionally responded accompanied with obvious arrhythmia, blood pressure only marginally improved. During the code, the accumulated dose of epinephrine was totaling 14 mg, amiodarone 300 mg, atropine 2mg and norepinephrine 4mg. In addition, because local anesthetics-related anaphylactic reaction could not be ruled out, methylprednisolone 80 mg and dexamethasone 10mg were administered intravenously. A total of 2500 mL of crystal fluids and 500 mL of hydroxyethyl starch fluids were used during 55 minutes.

A transthoracic echocardiography (TTE) was rapidly performed (discontinue chest compressions beyond 10s) to evaluate the ventricular dysfunction or thromboemboli. However, in the ventricles and visible pulmonary arteries, there was no direct visualization of thromboemboli. Meanwhile, an emergent Peripheral veno-arterial-ECMO (femoro-femoral configuration) system was successfully established in operation room 64 minutes after cardiac arrest. Blood pressure (90/55 mm Hg) and saturation of oxygen (PaO2 366 mm Hg) were restored with an initial setting of 2490 rpm and a flow of 2.56 L/min of ECMO system. About 55 minutes after the initiation of ECMO supporting, the patient's autonomic sinus rhythm was restored with a measured blood pressure of 110/76 mm Hg. The surgery was canceled and the patient was transferred to ICU for further treatment. The ECMO flow parameter setting ranged from 2.0 to 3.0 L/min in ICU for supporting circulation stability. Meanwhile, for anticoagulation management, heparin was titrated to maintain the activated clotting time (ACT) between 180 and 200 seconds. Laboratory testing showed no significant increase

in serum trypsin (a sensitive and specific marker for anaphylaxis), but a definite increase in the concentration of procalcitonin (PCT) at 0.351 ng/L and C-reactive protein (CRP) at 22.95mg/L, respectively, which are highly suggestive of an systemic inflammatory response. After 2 days of ECMO supporting treatment, the patient's cardiorespiratory status became more stable. The ECMO was successfully removed after comprehensive evaluation by ICU physicians on the third day. The patient remained under monitoring of vital signs in the ICU for another 5 days, then was transferred to the general ward without any sequelae.

Discussion

Spinal anesthesia is an important anesthesia technique performed widely in clinical practice.[5] Bradycardia and hypotension following spinal anesthesia are mainly due to the effects of spinal anesthesia on the cardiovascular system, which are related to the sympathetic nervous system blockade.[6] Sympathetic nervous system blockade results in parasympathetic nervous system tonus relatively increased, which causes cardiovascular system negative inotropic, dromotropic and chronotropic changes.[7] When cardioaccelerator sympathetic fibers (T1-T4) were blocked, it may result in severe bradycardia and even complete atrioventricular (AV) block.[6,8] Meanwhile, the loss of sympathetic tone results in peripheral vasodilation accompany with a redistribution of blood for splanchnic and limbs beds, which results in a significant reduction in cardiac preload.[2,9]

Both the blockade of cardioaccelerator fibers and reduction of preload cause a negative impact in heart rate after spinal anesthesia. According to the findings of Carpenter et al,[10] sympathetic denervation seems to result in only a 10% drop in heart rate compared to baseline levels while maintaining full preload. Thus, cardiac preload appears to be a more important factor affecting heart rate during spinal anesthesia. Studies have shown that a reduction in preload can result in severe bradycardia by triggering mainly three types of physiological reflex. The first reflex is related to receptors in pacemaker cells. Reduced preload leads to a decrease in atrial filling, causing a decrease in the stretchability of pacemaker cells, which in turn affected the heart rate.[11] The second reflex, the Bayside–Jarvis reflex, involves mechanical receptors in the posterior wall of the left ventricle. It increases parasympathetic nervous system activity and suppresses the sympathetic nervous system, leading to bradycardia, vasodilation, and hypotension.[12] The third reflex is attributed to mechanoreceptors in the right atrium and ventricle, baroreceptors in the right atrium and vena cava.[13,14] Usually, a fast reduction in preload volume can stimulate all those receptors, leading to bradycardia and even CA.

CPR is more difficult in the setting of spinal anesthesia due to sympathetic nervous system blockade resulting in cardioaccelerator fiber suppression and the reduction in preload. Meanwhile, studies also observed that spinal anesthesia can significantly suppress of adrenal gland function, accompanied with the reduction in circulating levels of epinephrine and norepinephrine.[10,15] This catecholamine deficiency is an important mechanism leading to refractory CPR. Studies have shown that the dose of epinephrine required to maintain coronary perfusion pressure between 15 to 20 mmHg during spinal anesthesia may be up to 0.1 mg/kg.[10] The patient in our case, the total dose of epinephrine was up to 14mg; however, the effect of resuscitation was not effective, which suggested that progressive strategies must be enhanced in this refractory situation.

VA-ECMO is a form of temporary mechanical circulatory support and simultaneous extracorporeal gas exchange for cardiorespiratory failure.[16,17] Peripheral VA-ECMO can be quickly initiated percutaneously via femoral artery and femoral or internal jugular vein access. It may be a bridge to provide a more effective and durable mechanical solution under extremely critical conditions.[3] In this patient of our case, we fastly started VA-ECMO to provide cardiopulmonary support, made him avoided to further harm from prolonged chest compression, refractory hypotension, and deteriorated desaturation, which may result in severe myocardial injury, overt systemic inflammatory response, and enormous reperfusion injury for his situation. Fortunately, he was successfully resuscitated without any sequelae. Meanwhile, severe local anesthetics induced-anaphylaxis, toxic reactions to local anesthetics, and pulmonary embolism in this case also required time to diagnose and exclude. Initiation of ECMO in timely for cardiopulmonary support might be a wise choice in such a complex situation.

According to the pathophysiology of cardiac arrest during spinal anesthesia, standard CPR might be more difficult. Quick-started peripheral VA-ECMO is a potential option under this situation, which protects the patient from further harm by deteriorating condition and allows time for other diagnoses and treatments to promote recovery.

Data Sharing Statement

The data that support the findings of this study are available on request from the corresponding author, Hongxuan Pang.

Ethics Statement

Based on the regulations of the Affiliated Hospital of Guilin Medical University, institutional review board approval is not required for case reports.

Consent for Publication

Written informed consent has been provided by the patient and her guardian to have the case details and any accompanying images published.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

There is no funding to report.

Disclosure

The authors declare that they have no conflicts of interest for this work.

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