Case Report

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Intraoperative Circulatory Arrest during Posterior Spinal Fusion - A Case Report and Serial Review of Five Cases

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Cardiac arrest or circulatory collapse in patients undergoing spinal surgery is a rare but devastating event. Our case-series report showed that the overall incidence of intraoperative circulatory collapse was 0.074% in adult patients who received posterior lumbar surgery. According to our database, patients who developed intraoperative circulatory arrest were typically aged (mean age of 73 years), of low disease severity (ASA PS II-III) and without gender predominance. One patient developed postoperative stroke. The etiology of circulatory arrest during spine surgery is most likely multifactorial, including severe hypovolemia, electrolyte derangement, thromboembolic events, anaphylaxis, and anesthesia medication problems. Prospective clinical studies are needed to investigate the hemodynamics and hormonal hemostasis during posterior spinal fusion surgery.

Key words: circulatory arrest, spinal fusion, intraoperative resuscitation

Introduction

Cardiac arrest in patients undergoing spinal surgery in the prone position is a rare but devastating event that may be related to cardiogenic, hypovolemic, anaphylactic or other etiologies. In this case report, we present a case and serial review of five similar cases to discuss the potential etiologies of acute circulatory arrest during posterior spinal fusion.

Case Report

A 61-year-old man underwent a lumbar spine revision operation due to recurrent lower back pain. His medical history was unremarkable except for a history of an ischemic stroke that resulted in left hemiparesis. Endotracheal intubation general anesthesia was performed with intravenous administration of fentanyl (100 µg), lidocaine (80 mg), propofol (140 mg), and rocuronium (50 mg). The patient was then supported by mechanical ventilation and general anesthesia was maintained with volatile desflurane 6 % (v/ v) in mixed oxygen (FiO₂ 0.6). The mean blood pressure was controlled at approximately 70 mmHg. During the later stages of subcutaneous suturing after fusion instrument implantation, the patient developed an abrupt drop in blood pressure, heart rate, and end-tidal CO₂ levels. Electrocardiogram later showed pulseless electrical activity (PEA). Cardiopulmonary resuscitation (CPR) was started immediately after converting the patient into a supine position. The patient regained spontaneous

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circulation after chest compression and intravenous administration of epinephrine (1 mg). Transesophageal echocardiography showed normal ventricular performance and no signs of wall motion defects or right ventricular failure. Blood samples were collected for cardiac enzyme, D-dimer, tryptase, and whole blood analysis. The patient regained full consciousness and was successfully extubated in the operating room (OR). However, low infusion doses of epinephrine (0.02 µg/kg/min) and norepinephrine (3 µg/min) were needed to maintain optimal blood pressure after extubation and during transfer to the intensive care unit (ICU). These vasoactive agents were tapered off after admission to ICU. The results of serial blood tests showed normal levels of cardiac enzymes and D-dimer; however, elevated eosinophil counts were noted in the blood samples collected at the 30 min and 12 h mark after the event (Table 1). Serum tryptase and IgE levels measured at 6 - 12 h after operation were not elevated. The patient was discharged from our hospital eight days later without any neurological or cardiovascular sequelae.

Case Series Report

A retrospective database review of patients who received lumbar spine surgery in our hospital from Jan 1, 2012 to Jun 30, 2019 was conducted after approval by the institutional review board (IRB) of E-Da Hospital (Approval number EMRP-107-001); the requirement for written informed consent was waived by the ethics committee. A total of 8061 cases of lumbar surgeries were performed during the eight-year study period. After reviewing the hospital database, four other cases who developed intraoperative circulatory arrest during elective lumbar spinal surgeries were identified (Table 1). All patients were in the prone position intraoperatively to receive spinal fusion with instrument implantation.

The mean age of the four patients was 73 (ranged 54 - 86) years and three of them were females. The preoperative physical statuses of these patients were modest (American Society of Anesthesiologists physical status, ASA PS II-III) without any severely limiting functional conditions. Sudden circulatory collapse developed occurred the operations, and two patients developed cardiac arrest at the end of operation. Massive intraoperative blood loss was not recorded in any of the patients, and none of them had significantly elevated cardiac enzyme levels or a diagnosis of acute ischemic myocardial injury after the operations. Although one patient experienced ischemic stroke, all other patients recovered fully after postoperative care and were discharged from hospital as scheduled. Intraoperative anaphylactic transfusion reaction was confirmed in one patient (Case 3), while more definite etiologies responsible for the event were not found for the other patients. The differential diagnoses of intraoperative circulatory arrest in each patient are listed in Table 1.

Discussion

According to our results, the overall incidence of intraoperative circulatory arrest requiring pharmacologic interventions and/ or cardiopulmonary resuscitation for posterior lumbar spinal fusion surgery was 7.4/10000 (0.074%). Quinn et al. retrospectively analyzed data from the American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) database from 1 Jan 2007 to 31 Dec 2013.1 They identified a total of 127 cases of intraoperative and early postoperative (i.e., within 24 hours) cardiac arrest, giving an incidence of circulatory arrest during and after spine surgery of 0.21%.1 In another case-series report, intraoperative cardiopulmonary arrest occurred in 11 out of 2524 (0.4%) pediatric patients undergoing spinal deformity

Table 1. Patient characteristics and differential diagnosis.

	Case report			Case series		
Case number		2	3	4	\$	9
Gender	M	Ā	M	Н	H	M
Age (years)	61	54	29	84	85	98
$BMI (kg/m^2)$	25.3	32	28.9	20.3	28.5	19.2
Major systemic diseases	Old stroke, HBV	HTN, RA, CKD	HTN, CAD	Nil	HTN, DM, CAD	HTN, DM, CAD VHD, multiple drug allergy
ASA PS	3	2	3	3	3	3
Prior operation	Spinal fusion	FESS	Spinal fusion	Open reduction	Nii	Nil
Levels of spinal fusion	L2-L5	L2-S1	L5-S1	L1-S1	L3-L5	T12-S1
Anesthesia volatile agent	Desflurane	Sevoflurane	Sevoffurane	Sevoflurane	Sevoflurane	Desflurane
Estimated blood loss (mL)	200	850	400	100	300	400
Transfused blood products	PRBC 2 units	PRBC 4 units	PRBC 2 units	PRBC 2 units	PRBC 2 units	PRBC 2 units
Transfused fluid volume (mL)	1800	1300	1950	1000	1550	1000
Clinical presentations of	PEA during wound	Asystole during	Shock after RBC	Asystole during wound	Profound shock	PEA during
circulatory arrest	closure	operation	transfusion	closure	during operation	wound closure
Laboratory and imaging studies	Cardiac enzymes (N) D-dimer (N) tryptase (N) IgE (N) cardiac echo (N)	Cardiac enzymes (N) cardiac echo (N)	Cardiac enzymes (N)	Cardiac enzymes (N) cardiac echo (N)	Thallium scan (N) cardiac echo (N)	Cardiac enzymes (marginally increased)
Outcomes	Good	Good	Good	Good	Cerebral infarction	Good
	Cardiogenic (×)	Cardiogenic (x)	Cardiogenic (x)	Cardiogenic (x)	Cardiogenic (?)	Cardiogenic (?)
Differential diagrams	Anaphylactic (x)	Anaphylactic (?)	Anaphylactic (\forall)	Anaphylactic (?)	Anaphylactic (?)	Anaphylactic (?)
Dinerenuai diagnosis	Neurogenic (?)	Neurogenic (?)	Neurogenic (\times)	Neurogenic (?)	Neurogenic (?)	Neurogenic (?)
	Embolism (x)	Embolism (?)	Embolism (\times)	Embolism (?)	Embolism (×)	Embolism (?)
	Hypovolemic (x)	Hypovolemic (x)	Hypovolemic (x)	Hypovolemic (x)	Hypovolemic (x)	Hypovolemic (×)

ASA PS: American Society of Anesthesiologists physical status classification system, BMI: body mass index, CAD: coronary artery disease, CKD: chronic kidney disease, DM: diabetes mellitus, FESS: functional endoscopic sinus surgery, HBV: hepatitis B virus carrier, HTN: hypertension, N: within normal limits, PEA: pulseless electrical activity, PRBC: packed red bleed cell (150 mL per unit), RA: rheumatoid arthritis, RBC: red blood cells, VHD: valvular heart disease, x: excluded, ?: undetermined, \(\psi\$: most

correction surgery.² Since our case-series study only included adult patients who developed circulatory arrest during surgery, a lower incidence than that previously reported appears to be reasonable.

Analysis of the ACS-NSQIP found that patients with ASA PS \geq 4, advanced age (> 60 years), dependent physical status, and chronic kidney insufficiency were associated with a high risk of developing perioperative circulatory arrest in spine surgery. In comparison to those who did not develop circulatory arrest, the perioperative mortality rate was significantly higher in the arrest group (1.2 % versus 61.8 %). Characteristic analysis of our study population showed that the patients who developed intraoperative circulatory arrest were older (mean age of 73 years) and less ill (ASA PS II-III) than those who did not. Furthermore, our database did not show any in-hospital mortality after spine surgery, except for one patient who developed postoperative stroke. Our study also suggests that the incidences of intraoperative circulatory arrest were similar between both genders.

The etiology of circulatory arrest during spine surgery is usually multifactorial, including severe hypovolemia, electrolyte derangement, thromboembolic events (i.e. myocardial infarction and pulmonary embolism), anaphylaxis, and anesthesia medication-related problems.3 Other uncommon mech-anisms have also been proposed, such as a trigeminocardiac reflex and dysregulation of the brain-heart connection during spine surgery.³ Catecholamine surges due to an imbalanced autonomic system are also known to cause surgical stress that induces cardiac arrhythmias, myocardial injury, and Takotsubo cardiomyopathy. 1,4 Furthermore, dural tear during spine surgery can cause a sudden decrease in cerebrospinal fluid pressure and traction of the cauda equina, leading to vasovagal reflex stimulation and subsequent development of severe bradycardia or cardiac arrest.3

At our institute, prone-positioned patients were supported using the Modified Wilson type frame to reduce intraabdominal pressure and enhance the return of venous blood flow.⁵ There was also no record of massive intraoperative blood loss in any of our patients. Therefore, the occurrence of severe hypovolemic shock was unlikely in our case series. Cardiogenic shock secondary to intraoperative myocardial necrosis was also excluded as none of these patients had significantly elevated cardiac enzyme levels or newly developed cardiac wall motion abnormalities after operation. In our case series, one patient developed a grade III hypersensitivity reaction to red blood cell transfusion. However, anaphylactic reactions cannot be ruled out in the other patients as a complete allergy investigation was not performed. In fact, metallic prosthesis and the coating materials,6 hemostatic agents⁷ or other medical care products⁸ are known to induce severe hypersensitivity reactions during operation. In addition, we speculate that a dysfunctional autonomic reflex or compromised circulatory reserve due to depletion of endogenous catecholamines or glucocorticoid may contribute to this intraoperative event. Therefore, more comprehensive monitoring techniques are required to show the subtle homeostatic changes of the autonomic nervous system throughout the perioperative period to allow timely intervention.

Conclusion

The development of acute circulatory arrest during lumbar spine surgery is catastrophic. Not only does the resuscitation process in prone position pose a significant challenge but intraoperative circulatory arrest also increases risk of major morbidities and in-hospital mortality. Although there has been a considerable number of case and case series reports in the literature, the exact pathological mechanism underlying this intraoperative event

remains unclear, particularly the contributions of autonomic dysregulation and depletion of endogenous stress-response hormones. Prospective clinical studies are needed to investigate the hemodynamics and hormonal homeostasis during posterior spinal fusion surgery.

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