BMC Anesthesiology

CASE REPORT

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Severe stress cardiomyopathy following spinal corrective surgery for scoliosis complicated with pectus excavatum: a case report

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Abstract

Background Stress cardiomyopathy (SCM) is an acute heart failure syndrome characterized by transient, usually reversible left ventricular systolic dysfunction with normal or enhanced basal compensatory wall motion abnormalities involving the left ventricular anterior septum and apex, resulting in a "ballooning" appearance. However, it has rarely been reported in patients undergoing spinal surgery.

Case presentation We report a case of severe stress cardiomyopathy in a scoliosis patient with pectus excavatum who underwent spinal corrective surgery. During the wake-up period, circulatory collapse occurred. After multidisciplinary consultation, the patient was diagnosed with stress cardiomyopathy. At last, she had a good prognosis after a series of treatments including ECMO.

Conclusion Stress cardiomyopathy is a reversible but uncommon condition. It can cause death if it is not diagnosed in time. Consequently, this report should improve the awareness of orthopedists and anesthesiologists for timely identification and management. For patients with potential risk factors, timely preoperative intervention should be performed to reduce the occurrence of stress cardiomyopathy.

Keywords Stress cardiomyopathy, Pectus excavatum, Takotsubo cardiomyopathy, Scoliosis, Veno-arterial extracorporeal membrane oxygenation, VA-ECMO, Intra-aortic balloon pump, IABP, Spinal corrective surgery

Background

Stress Cardiomyopathy (SCM) is an acute heart failure syndrome characterized by transient, usually reversible left ventricular systolic dysfunction with normal or enhanced basal compensatory wall motion abnormalities involving the left ventricular anterior septum and apex, resulting in a "ballooning" appearance. Unlike myocardial infarction, SCM is not caused by coronary artery obstruction. A Japanese scholar, Sato et al. in 1990, first reported this disease. The clinical manifestations are similar to Acute Coronary Syndrome (ACS), leading to frequent misdiagnosis. During the acute phase, it can be complicated by various conditions, including acute heart failure, cardiogenic shock, cardiac rupture,

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thromboembolism, arrhythmia and even death [1]. Its mortality is close to acute coronary syndrome [2]. However, it has rarely been reported in patients undergoing spinal surgery.

Case description

A 16-year-old female patient, 164 cm in height, 42 kg in weight, 15.6 kg/m^2 in BMI (Body Mass Index), ASA (American Society of Anesthesiologists) II level, was admitted to the spinal surgery department due to "asymmetric chest and back protrusion discovered for 1 year". The patient, combined with "pectus excavatum", was generally capable of normal activities, with a MET (Metablic Equivalent)score of above 6. Preoperative arterial blood gas analysis was generally normal ($PaCO_2$ 36 mmHg, PaO_2 92 mmHg, SPO_2 98%).Pulmonary function showed moderate to severe obstructive mixed ventilatory dysfunction (FEV1/FVC 84.5%<92%, MVV(Maximal Voluntary Ventilation) 75.08 L, FEV1 Actual value/predicted value was 56%<80%,FVC Actual value/predicted value was 57%). Electrocardiogram showed sinus bradycardia HR 55 bpm, incomplete right bundle branch block and T-wave changes. The Echocardiogram showed mild regurgitation of mitral and tricuspid valves (EF62.1%). Admission diagnosis: (1) Spinal scoliosis (2) Pectus excavatum (3) Incomplete right bundle branch block. Following communicating with the patient and the patient's family members about surgery-related risks, a planned "thoracolumbar fusion surgery, posterior approach + multi-level vertebral fusion" is scheduled under general anesthesia.

After fasting and refraining from drinking for 10 h and completed preoperative electrocardiogram monitoring, which showed HR 54 bpm, non-invasive blood pressure 94/63 mmHg, SPO_2 98% (no oxygen inhalation). Following the establishment of peripheral venous access, anesthesia induction was administered after oxygen inhalation by mask at 5 L/min: Midazolam 2 mg, 1% Propofol 60 mg, Sufentanil 20 ug, Vecuronium bromide 6 mg, Dexamethasone 10 mg. Following induction of anesthesia the patient was intubated with a size 7.0 ID endotracheal tube. Maintenance of anaesthesia was achieved by total intravenous anaesthesia with propofol and remifentanil to a target BIS value of 40–60. Fluid infusion was target-guided by PPV (Pulse Pressure Variation) and CVP (Central Venous Pressure). After anesthesia induction, the patient's HR transiently decreased to 39 bpm. After intravenous injection of 0.5 mg atropine, HR gradually increased to 60–70 bpm. Changing to the prone position, the patient's BP dropped significantly, reaching a low of 67/47 mmHg. Norepinephrine 4 μg was given intravenously, resulting in a rise in BP to 90–105/60–70 mmHg. Despite the start of surgery, the BP remained low, fluctuating between 85–95/56–67 mmHg. A continuous infusion of Norepinephrine at 0.1 μg·kg-1·min-1 was administered, and the infusion rate was adjusted based on BP to maintain BP between 100–110/65–75 mmHg and HR between 50 and 70 bpm. Approximately 1.5 h after surgery began, muscle relaxants were discontinued after complete spine exposure. After 4 h from the start of surgery, the bilateral growing rods for spinal correction were successfully placed, so all anesthetics were stopped. The intraoperative arousal was unsuccessful, so the surgery continued. After the skin was sutured, the patient was awakened again and cooperated well with instructions, resulting in a successful awakening. At this time, the BP was 106/70 mmHg, and the HR was 82 bpm. Then,1% Propofol 20 mg was given to deepen anesthesia, and prepared for the change to the supine position. After removing the patient's electrocardiogram (ECG) monitoring and pulse oxygen clip, it was noticed that the patient's ABP suddenly dropped to 24/15 mmHg. Immediately, 12 μg of Norepinephrine was given intravenously followed by changing to the supine position. However, there was no improvement in BP after repositioning. Subsequently, 20 μg of Norepinephrine was administered again, and external chest compression was performed. Epinephrine 0.1 mg was administered intravenously twice in succession, and ECG monitoring and pulse oxygen clip were re-established. ECG showed sinus rhythm, QRS amplitude was small. The BP was 156/119 mmHg; the HR was 142 bpm. The repeated blood gas analysis showed the following results: pH 7.19 ,PaO_2 489 mmHg, $PaCO_2$ 53 mmHg, HCO_3^- 20.2 mmol /L, BE -8.0 mmol/L, Lac 1.1 mmol /L, Na^+ 143 mmol /L, K^+ 4.6 mmol /L, Ca^{2+} 1.06 mmol /L, Glu 7.3 mmol /L, Hb 10.5 g/dL. Then, administered 50 ml of 5% sodium bicarbonate to correct acidosis. Norepinephrine was intermittently administered while simultaneously continuously infused Norepinephrine at 0.3 μg·kg-1·min-1 to maintain BP at 90–105/60–65 mmHg. The total duration of the surgery was approximately 4 h and 35 min. During the procedure, there was a blood loss of 2200 ml, and the urine volume was 800 ml. The patient received 3000 ml of compound sodium chloride solution, 2500 ml of succinylated gelatin, 150 ml of 5% sodium bicarbonate, and 300 ml of 0.9% sodium chloride. Additionally, 1100 ml of leukocyte-reduced red blood cells and 510 ml of autologous blood recovery were transfused. Postoperatively, the patient was transferred to the Anesthesia Intensive Care Unit (AICU) with endotracheal tube for further treatment.

After admission to AICU, the patient's vital signs were monitored: BP was 84/45mmHg (under Norepinephrine pump), HR was 91 bpm and pupils on both sides were equal and round, dull to reflect light. The patient's circulatory status remained collapsed, so The patient's BP was maintained by Intermittent injection of Norepinephrine 8 μg, Phenylephrine 40 μg, continuous injection of

Norepinephrine 0.5 ug · kg- 1 · min- 1. Bedside echocardiography was performed immediately to assess cardiac function and volume. Bedside echocardiography showed that the heart cavity was full and the myocardial motion was generally weak, especially at apex and papillary muscles level. Considering the possibility of fluid overload, Dobutamine and Furosemide were given as cardiotonic diuretics, and Norepinephrine was continued to maintain the patient's blood pressure. Four hours after admission to AICU, the patient was awake and could follow the instructions. After sedation, bedside echocardiography was performed again to evaluate the cardiac function and volume, which showed that the basal and apical wall motion of left ventricle was significantly better than before, but the papillary muscle segment wall motion was still poor. The laboratory tests showed that BNP and TNT were within the normal range, and creatine kinase was elevated at 619U/ L. Symptomatic supportive treatment was continued.At night (about 12 h after operation), the CVP suddenly increased and the monitor showed malignant arrhythmia (Fig. 1), accompanied by a sudden drop in BP and undetectable SPO_2. Chest compressions were performed immediately, and Epinephrine 1 mg was injected intravenously twice. After continuous chest compressions for 3 min, sinus rhythm was restored, during which Norepinephrine and Phenylephrine were continuously pumped, then the BP gradually rose to 110–127/90–101 mmHg, HR107-140 bpm. Four minutes later, the vital signs became stable with the support of vasoactive drugs, and the patient was treated with head-cooling cap, mannitol, and steroids. Arterial blood gas analysis, bedside electrocardiogram, and echocardiography were evaluated.The arterial blood gas analysis showed: pH 7.36, PaO_2 96 mmHg, $PaCO_2$ 34 mmHg, HCO_3^- 19.2 mmol /L, BE -5.5mmol/L, Lac 4.3mmol /L, Na^+ 138 mmol /L, K^+ 4.6 mmol /L, Ca^{2+} 1.16 mmol /L, Glu 6.1mmol /L, Hb 11.9 g/dL, SPO_2 97%.Echocardiography showed that the basal wall motion of the left ventricle was acceptable, the motion of other segments of the left ventricle was significantly weakened, the apex was round and dull, and the motion of the right ventricle was generally significantly weakened. We immediately called a cardiologist for an urgent consultation. Combined with the patient's medical history, arterial blood gas analysis, echocardiography, ECG which showed multiple lead T wave inversion and QT interval prolongation, and the circulation remained collapsed. Additionally, high-dose Norepinephrine 0.076 mg·kg-1·h-1 and Phenylephrine 0.72 mg·kg-1·h-1 were required to maintain stable vital signs. Considering the patient had occult prolongation of QT interval and malignant torsades de pointes arrhythmia, the cardiologists suggested that potassium supplement should be used to maintain K+above 4.0 mmol/L, the changes of electrocardiogram should be monitored continuously. Isoproterenol 0.01 mg/h instead of Dobutamine should be used to improve QT prolongation and avoid the occurrence of left ventricular outflow tract obstruction. Meanwhile, we pumped MgSo4 intravenously. Six hours later, the patient's HR gradually increased; the fastest was 151 bpm, the lowest BP was 60/40 mmHg, the arterial blood gas lactate level gradually increased, the highest was 7.3mmol/L, and urine decreased to 20 ml/h. We immediately requested a multidisciplinary consultation from the Department of Echocardiography, Cardiology, and ICU(Intensive Care Unit). At the same time, TNT and BNP were significantly elevated (TNT0.476 ug/L, BNP1360 pg/ml). Echocardiography showed that the basal wall motion of the left ventricle was acceptable, the motion of the remaining wall segments was significantly reduced, the apex was round and obtuse, and the motion of the right ventricular wall was generally significantly reduced, EF21%, SV 13 ml (Fig. 2). The possibility of Stress Cardiomyopathy (SCM) was considered by multidisciplinary consultation. Due to the obvious manifestation of cardiogenic shock and poor cardiac function, this patient was recommended to be treated with Extracorporeal Membrane Oxygenation(ECMO) immediately to improve myocardial function. On the same day, the patient was transferred to the ICU for Veno-Arterial Extra-corporeal membrane oxygenation (VA-ECMO), and a hospital-wide consultation was initiated to determine the next treatment plan, including continuous RRT to remove inflammatory mediators, Milrinone for strengthening the heart, Coenzyme Q10 for nutrition of the myocardium, Piperacillin tazobactam sodium+Vancomycin for empirical anti-infection treatment, Heparin anticoagulation treatment, sedation and analgesia to reduce oxygen consumption, Mannitol to reduce brain edema, infusion of blood products to ensure oxygen supply, and control heart rate. Meanwhile, indwelling pulmonary artery catheter was used to monitor and guide fluid management and shock resuscitation. After the above treatment measures, the patient's spontaneous breathing and circulatory function gradually improved. After 3 days of ECMO treatment, bedside echocardiography showed varying degrees of weakened left ventricular myocardial

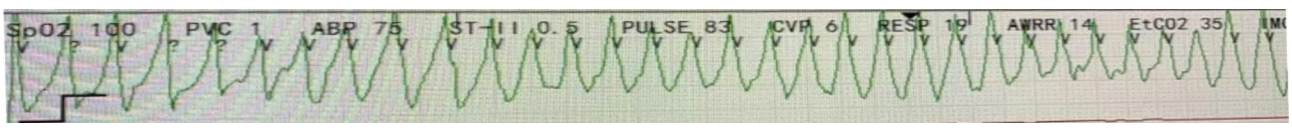
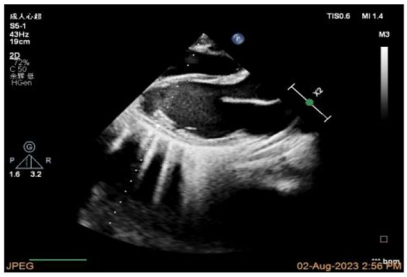
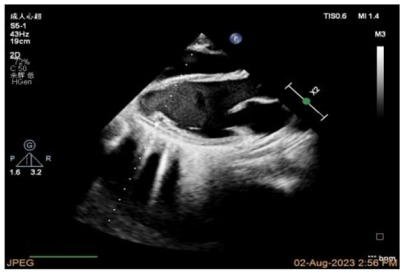


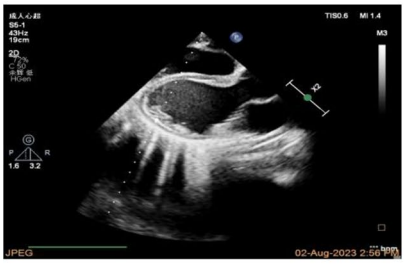
Fig. 1 ECG:1st night postoperatively Torsades de pointes



Diastolic phase1



Diastolic phase 2



Systolic phase 1

Fig. 2 Echocardiography: the apex was obtuse, EF21%, SV13ml

motion, left ventricular dysfunction, left ventricular ejection fraction (EF) value 43%, which indicated recovery of cardiac function and improvement of spontaneous circulation compared with before. ECMO support was withdrawn 8 days later. The patient was extubated 2 days after weaning from ECMO, and vasoactive drugs were discontinued 4 days later. Subsequently, the patient was transferred to the general ward to continue anti-infective, anti-thrombotic and analgesic treatment, and was discharged after 10 days in the general ward. Echocardiography showed a small right heart cavity, EF 52.4%, BNP35.3 pg/ml, and prolonged QTc interval of sinus tachycardia. Telephone follow-up at 1 month and 6 months after surgery showed that the patient recovered well and no adverse events occurred.

Discussion and conclusions

Stress Cardiomyopathy (SCM) is an acute heart failure syndrome characterized by transient, usually reversible left ventricular systolic dysfunction with normal or enhanced basal compensatory wall motion abnormalities involving the left ventricular anterior septum and apex, resulting in a "ballooning" appearance. Unlike myocardial infarction, SCM is not caused by coronary artery obstruction. It is also called Takotsubo cardiomyopathy because of changes in the shape of the left ventricle similar to "Takotsubo". In recent years, it has been found that SCM can also be manifested as the involvement of other parts of the left ventricle, the right ventricle or both ventricles and is mostly triggered by stressors, including physical factors or emotional factors [3]. A Japanese scholar Sato et al. in 1990, first reported this disease. Since then, it has been observed worldwide and appeared in various clinical conditions. The clinical manifestations are similar to Acute Coronary Syndrome (ACS), leading to frequent misdiagnosis. In the past, it was considered as a benign and self-limited disease with symptoms of cardiac dysfunction and ventricular wall motion abnormalities recovering within a short time, but it can also have a poor prognosis due to its unique clinical manifestations [4]. During the acute phase, it can be complicated by various conditions, including acute heart failure, cardiogenic shock, cardiac rupture, thromboembolism arrhythmia and even death [1]. Its mortality is close to acute coronary syndrome. SCM accounts for 1-2% of patients with suspected ACS or suspected ST-segment elevation myocardial infarction(STEMI) and occurs predominantly in women [2]. There are few related studies on SCM in China, and the diagnosis rate is low, mainly focusing on case reports.

The pathogenesis of SCM is not fully understood at present. The most widely accepted mechanism is that catecholamine-mediated myocardial stunning and microvascular spasm. Compared with basal cardiomyocytes, apical cardiomyocytes have higher β-adrenergic receptor density and are more sensitive to catecholamine stimulation. Excessive catecholamine makes β2-adrenergic receptor negative inotropic effect mediated by activated G protein, leading to significantly weakened apical myocardial motion, forming the classic

"ballooning appearance". The brain-heart axis theory is another important mechanism of SCM. Complex brain-heart interactions play an important role in this process. It has been shown that patients with SCM show changes in neuronal connectivity in the limbic system regions of the brain associated with stress. This region is important for regulating the body's emotional response and autonomic nervous system function. The ability of a SCM susceptible person to respond appropriately to the precipitating factor may be diminished with a trigger, and on this basis, the imbalance between sympathetic and parasympathetic nervous system can cause patients to develop severe myocardial dysfunction and damage, promoting the development of SCM.

The patient was a young female with no abnormal findings on preoperative cardiac examination. However, during the awakening process after surgery, the BP suddenly decreased, echocardiography showed reduced ventricular wall motion, significantly reduced ejection fraction, and slightly increased troponin and ECG showed prolonged QTc interval, inverted T wave accompanied by malignant arrhythmia. After the surgery, the patient underwent ECMO treatment, improving in cardiac function with recovery of ejection fraction, consistent with the diagnostic criteria and characteristics of SCM. The precipitating factors for the sudden onset of SCM in this patient are difficult to determine but may include: (1) Surgical trauma: The patient underwent major spinal surgery, and during the awakening process with lighter anesthesia, was in a highly stressful state. Catecholamine substances are released in large quantities, resulting in myocardial injury; (2) Prolonged prone positioning during surgery: The patient was in a prone position complicated with pectus excavatum during the whole operation. The corrective segment of scoliosis surgery was located directly above the heart, continuously compressing it, leading to sustained stress on the myocardium (Fig. 3); (3) Intraoperative blood loss and fluid shifts: The patient experienced significant blood loss during surgery, followed by rapid and large fluid infusion in a short period of time, and long-term use of catecholamine drugs such as Norepinephrine; (4) Hypothermia: The perioperative anesthesia period lasted for 4 and a half hours. The extensive surgical incisions, large amount of fluid washouts, and blood transfusion leading to a lower body temperature; (5) After orthopedic surgery for scoliosis, compression of the vessels and mediastinum was relieved, IVC may have been stretched which can lead to increased blood return to the heart. At the same time, massive fluid infusion further increased the load of the heart. This eventually leads to SCM; (6) Preoperative anxiety: The patient may have had preoperative anxiety, which could have led to excessive release of catecholamine under the stimulation of surgical trauma.

At present, there is no clear standard for the management of SCM, and most of the treatment is symptomatic and supportive, including the use of angiotensin-converting enzyme inhibitor (ACEI), angiotensin II receptor blocker (ARB), β-receptor blocker and diuretic [5], but the evidence of their efficacy and prognosis is insufficient. Because catecholamine overdose is the core mechanism of SCM, there is controversy over whether to use catecholamine drugs in patients with SCM-induced cardiogenic shock. Some studies indicate that catecholamine use is an independent risk factor for mortality due to SCM in hospitalized patients [6, 7]. Therefore, some researchers advocate prioritizing the use of non-catecholamine inotropic agents such as milrinone, vasopressin, and levosimendan in such cases. Levosimendan, in particular, is a potent phosphodiesterase III inhibitor with a mechanism different from traditional positive inotropic agents. It doesn't act on adrenergic receptors but rather enhances the sensitivity of contractile proteins to calcium ions by binding to cardiac troponin C in a

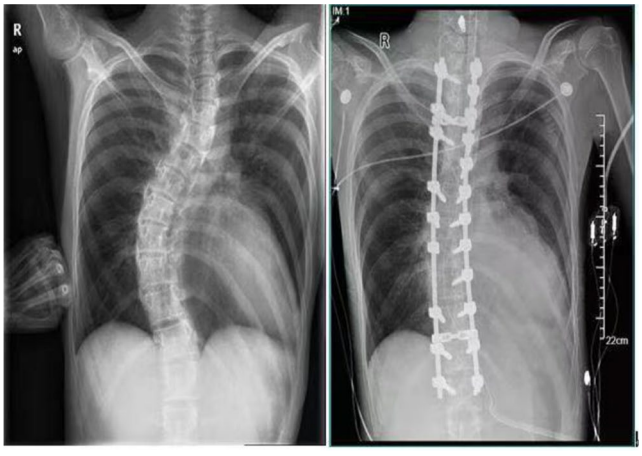


Fig. 3 Pre and post-operative anteroposterior radiographs of the whole spine, Cobb angle 52°

calcium-dependent manner. This enhances myocardial contractility without affecting ventricular relaxation, aiding in the faster recovery of patients with SCM. In addition, it can induce vasodilation in systemic and coronary artery resistance vessels as well as systemic venous capacitance vessels by opening ATP-sensitive potassium channels in vascular smooth muscle [8]. However, the patient was under 18 years old, making levosimendan contraindicated, so milrinone was used to improve the patient's cardiac dysfunction. Additionally, some studies support using mechanical devices for circulatory support, such as Intra-aortic balloon pump (IABP), extracorporeal membrane oxygenation (ECMO), or temporary left ventricular assist devices, to minimize or avoid the use of inotropic agents. A meta-analysis by Silvia Mariani et al. [7, 9] found that in SCM-induced cardiogenic shock, early use of mechanical devices for circulatory support should be considered. Moreover, there is also increasing evidence that early use of V-A ECMO can improve the survival rate and quality of life [7, 10]. With effective ECMO support, the patient's condition is controlled and the prognosis is good.

In contrast to other patients with SCM, this patient was a young child with pectus excavatum before surgery and developed severe myocardial dysfunction after spinal correction surgery, with EF21% on echocardiography. After a series of supportive treatments such as ECMO, the patient returned to normal, and the echocardiographic ef was significantly improved. Spinal surgery is associated with high blood loss. It is normal to use catecholamines. Therefore, for such patients, we should be alert to the occurrence of SCM.

In this case, the patient experienced severe SCM postoperatively. After a series of supportive treatments, the ejection fraction returned to normal and myocardial injury markers decreased to normal levels resulting in a good prognosis. However, reviewing the management of this patient, there are still some shortcomings including: (1) The patient's cardiac function deteriorates rapidly after onset, and circulation remains collapsed for a prolonged period, which needs high-dose vasopressor support. Meanwhile, early initiation of cardiac energy support during the early stage may slow down the progression of cardiac dysfunction. Studies have shown that oxidative metabolism disorder is the basis of cardiac dysfunction in patients with SCM [11], so we think that early use of drugs that improve myocardial energy metabolism can benefit the prognosis of patients, but there is still insufficient clinical evidence. (2) Coronary angiography and cardiac magnetic resonance examination were not performed immediately after onset to confirm the diagnosis. In addition, we can use rest myocardial perfusion imaging with SPECT and TIMI Risk Score for early diagnosis and prediction of prognosis [12, 13]. (3) The preoperative electrocardiogram and echocardiography were not fully normal, but further testing was not performed before the procedure, which may have led to an inadequate preoperative assessment of the patient's cardiac function. (4) The preoperative assessment didn't pay attention to the evaluation of the patient's emotional state adequately. Studies indicate that preoperative anxiety is the potential trigger for SCM [14, 15],administering a certain amount of sedative drugs preoperatively to alleviate anxiety. During the operation, adjusting anesthesia depth in real-time based on surgical stimuli can effectively reduce stress responses while maintaining an appropriate anesthesia depth. Especially for patients who need wake-up during scoliosis surgery, adequate analgesia should be provided before awakening to reduce the stress caused by the shallow depth of anesthesia.

In summary, the key points of perioperative anesthesia management of SCM are as follows: Pay attention to the patient's anesthesia evaluation before surgery, identify the high-risk factors of SCM, and formulate individualized anesthesia plans. During the operation, triggered factors were actively removed to avoid all adverse stress reactions and a multi-modal precise management plan was formulated after the operation. Anesthesiologists need to enhance their ability to identify SCM early and formulate a detailed and standardized anesthesia management plan for SCM patients. Multidisciplinary collaboration, early identification, accurate evaluation, correct handling and prevention of complications are crucial for improving the perioperative safety of SCM patients.

Abbreviations

SCM	Stress Cardiomyopathy.
ACS	Acute Coronary Syndrome.
BMI	Body Mass Index.
ASA	American Society of Anesthesiologists.
MET	Metablic Equivalent.
MVV	Maximal Voluntary Ventilation.
ABP	Arterial Blood Pressure.
PPV	Pulse Pressure Variation.
CVP	Central Venous Pressure.
ECG	Electrocardiogram.
Bis	Bispectral Index.
AICU	Anesthesia Intensive Care Unit.
VA-ECMO	Veno-Arterial Extra-corporeal membrane oxygenation.
EF	Ejection fraction.
STEMI	ST-segment elevation myocardial infarction.
ACEI	Angiotensin-converting enzyme inhibitor.
ARB	Angiotensin II receptor blocker.
IABP	Intra-aortic balloon pump.

Acknowledgements
Not applicable.

Author contributions
The designs of anesthesia strategy and anesthesia implementation were by J.Z.and XH.Y.,the management of AICU were by YE.S. ,J.H. and J.X. , and the drafting of the manuscript were by XH.Y.; manuscript review and correction was by ZL.M. ,Z.J. and YE.S.All authors read and approved the final manuscript.

Funding
Not applicable.

Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable. All methods were carried out in accordance with relevant guidelines and regulations.

Consent for publication
Written informed consent was obtained from the patient for publication of the case report and the images.

Competing interests
The authors declare no competing interests.

Received: 19 June 2024 / Accepted: 2 September 2024
Published online: 18 September 2024

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