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Anesthetic Management for Elective Cesarean Section in a Pregnant Woman With Hypertrophic Obstructive Cardiomyopathy and a Marked Family History: A Case Report Focusing on Efficient Hemostasis Using Fibrinogen Concentrate

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Abstract

Anesthetic management for cesarean section (CS) in patients with hypertrophic obstructive cardiomyopathy (HOCM) requires strict hemodynamic control. A 32-year-old pregnant woman with HOCM, whose mother had died of HOCM and postpartum hemorrhage following CS, was scheduled for an elective CS. Combined spinal-epidural anesthesia was performed with a continuous norepinephrine infusion. Pre-induction echocardiography confirmed the improvement of left ventricular outflow tract (LVOT) obstruction following volume loading. During intraoperative skin-to-skin contact, an infusion failure caused transient hypotension, which was promptly detected via continuous arterial pressure monitoring and corrected.

Postoperatively, the patient developed postpartum hemorrhage with pre-disseminated intravascular coagulation (pre-DIC). To rapidly correct severe hypofibrinogenemia and break the bleeding cycle, fibrinogen concentrate was prioritized, while fresh frozen plasma (FFP) was combined for comprehensive coagulation factor replenishment and hypovolemia management. Postoperative atrial fibrillation (AF) was resolved with intravenous amiodarone, and she was discharged on postoperative day (POD) 16. In high-risk patients with HOCM, dynamic strategies integrating real-time cardiac assessment, targeted coagulation factor replacement, and continuous arterial pressure monitoring may contribute to successful anesthetic management.

Categories: Obstetrics/Gynecology, Cardiology, Anesthesiology
Keywords: acute obstetric coagulopathy, cesarean section (cs), fibrinogen concentrate, hypertrophic obstructive cardiomyopathy (hocm), postpartum hemorrhage

Introduction

Hypertrophic obstructive cardiomyopathy (HOCM) is an inherited myocardial disease characterized by a dynamic left ventricular outflow tract (LVOT) pressure gradient [1]. This gradient increases with reduced preload, reduced afterload, and enhanced contractility, exacerbating systolic anterior motion (SAM)-associated mitral regurgitation and causing unexpected circulatory collapse [2,3]. Anesthetic management for cesarean section (CS) in patients with HOCM requires utmost care, as anesthesia-induced vasodilation, supine hypotension, and acute hemorrhage drastically alter preload and afterload [4].

Although previous reviews have documented the safety of neuraxial anesthesia in HOCM parturients [5], most literature focuses primarily on routine anesthetic choices or immediate delivery outcomes, with limited attention to intraoperative continuous troubleshooting and postoperative complications. Here, we report a scheduled CS in a patient with HOCM with a high-risk family history: according to family reports, her mother died one day after CS due to postpartum hemorrhage, precipitating acute HOCM-related heart failure. Preoperative volume loading guided by real-time transthoracic echocardiography (TTE) ensured stable anesthesia induction, while meticulous intraoperative monitoring allowed prompt detection and treatment of transient hypotension. Furthermore, prompt administration of fibrinogen replacement for postpartum hemorrhage effectively managed obstetric coagulopathy, avoiding excessive fluid resuscitation and circulatory overload. This case highlights the utility of a seamless, multidisciplinary approach spanning from careful intraoperative anesthetic management to precise postoperative hemorrhage control in high-risk patients with HOCM.

This article was previously presented as a meeting abstract at the 31st Annual Meeting of the Japanese Society of Cardiovascular Anesthesiologists on September 20, 2026 (Nagasaki, Japan).

Case Presentation

A 32-year-old pregnant woman (gravida 2, para 1; body mass index: 26.7 kg/m^2) with HOCM was referred for

anesthetic management of a scheduled CS at 36 weeks of gestation. She was diagnosed with HOCM at birth; her family history, obtained via direct clinical interview and patient report, was notable for her mother's death one day after CS, which was secondary to postpartum hemorrhage that precipitated acute exacerbation of HOCM-related heart failure. The patient underwent surgical septal myectomy at age 16 for severe mitral regurgitation and a peak LVOT pressure gradient of 155 mmHg. Postoperatively, the gradient decreased to 30-50 mmHg; her first CS at age 27 was uneventful. During this second pregnancy, high-risk due to twins, she underwent selective fetal reduction at 13 weeks following multidisciplinary consultations. The demised co-twin remained in utero throughout the remainder of the pregnancy. While complete blood counts were monitored regularly (13 times at two- to four-week intervals throughout pregnancy), maternal coagulation parameters were specifically assessed prior to fetal reduction and at 30 weeks of gestation (the final assessment prior to delivery), yielding plasma fibrinogen levels of 203 mg/dL and 259 mg/dL, respectively (Table *1*). Her HOCM-related heart failure was managed with carvedilol (5 mg/day), furosemide (5 mg/day), and spironolactone (5 mg/day). Her brain natriuretic peptide levels ranged between 126 and 406 pg/mL, and she remained asymptomatic during daily activities. Preoperative TTE performed five days before surgery showed a preserved left ventricular ejection fraction of 62.2% and a peak LVOT pressure gradient of 19 mmHg.

Parameter	Reference range	10+5 weeks	13+6 weeks	15+1 weeks	19+6 weeks	24+1 weeks	26+1 weeks	28+1 weeks	30+1 weeks	30+6 weeks	31+6 weeks	32+6 weeks	33+6 weeks	34+6 weeks	36+0 weeks (immediate postop)	100 minutes after ICU admission	Post-fibrinogen (3 g)+ FFP (2 units)	Post-additional FFP (4 units)	POD 1	POD 2	POD 3	POD 4	POD 5
Hemoglobin (g/dL)	11.6 to 14.8	10.0	10.8	11.8	13.3	13.5	13.6	13.1	12.6	12.1	12.0	12.3	12.4	12.3	10.4	10.9	10.5	10.3	9.8	9.3	8.8	10.2	12.1
Platelet count (10^9/L)	158 to 348	209	131	177	161	154	134	155	145	154	136	128	109	110	82	102	96	96	103	118	115	120	156
Fibrinogen (mg/dL)	200 to 400	203	ND	ND	ND	ND	ND	ND	ND	259	ND	ND	ND	ND	176	162	265	288	350	ND	ND	ND	ND
PT-INR	0.85 to 1.10	0.95	ND	ND	ND	ND	ND	ND	ND	0.94	ND	ND	ND	ND	1.01	0.99	0.96	0.88	0.92	ND	ND	ND	ND
APTT (seconds)	24 to 34	28.9	ND	ND	ND	ND	ND	ND	ND	31.5	ND	ND	ND	ND	51.6	48.8	44.2	35.5	35.5	ND	ND	ND	ND
BNP (pg/mL)	<18.4	ND	264.7	ND	203.2	126	ND	130.5	ND	194.5	236.3	187.3	227.8	156.5	ND	ND	ND	ND	772.7	ND	ND	ND	ND

TABLE 1: Maternal blood data from early pregnancy through the perioperative period

Relative hypofibrinogenemia was observed at 10 weeks of gestation (reference values in the table represent non-pregnant adult reference ranges). Following fetal reduction at 13 weeks of gestation, a transient decrease in platelet count occurred, followed by recovery at 15 weeks. Platelet counts subsequently showed a gradual decline as pregnancy progressed. Immediately postoperatively, plasma fibrinogen level was 162 mg/dL, which markedly increased to 265 mg/dL following the administration of 3 g of fibrinogen concentrate and 2 units of FFP.

Reference values are based on the standard ranges at our institution.

APTT: activated partial thromboplastin time, BNP: brain natriuretic peptide, FFP: fresh frozen plasma, ICU: intensive care unit, ND: not done, POD: postoperative day, PT-INR: prothrombin time-international normalized ratio

In the operating room, standard monitoring (pulse oximetry, non-invasive blood pressure, and electrocardiogram) and a radial artery catheter were established. Although safe for neuraxial block, this preoperative fibrinogen level was recognized as relatively low for late gestation, prompting precautionary preparation of transfusion products, including red blood cells, fresh frozen plasma (FFP), and fibrinogen concentrate, in anticipation of potential postpartum hemorrhaging and coagulopathy. Following a 500 mL hydroxyethyl starch (HES; 130/0.42) load (our institutional standard fluid for volume loading during CS due to its minimal impact on blood coagulation [6]), TTE confirmed improved SAM and trivial mitral regurgitation (Video *1* and Video *2*). To mitigate sympathetic blockade and vasodilation, sequential combined spinal-epidural anesthesia was planned alongside continuous norepinephrine infusion (0.07 µg/kg/min). An epidural catheter was inserted at T10/11, rather than lower lumbar levels, to facilitate reliable epidural sensory block elevation up to the T4 segment if spinal anesthesia proved partial or incomplete. Although a low-dose spinal block was planned, technical difficulties and anatomical changes of pregnancy raised concerns about incomplete block spread. To avoid inadequate anesthesia and pain-induced sympathetic hyperactivity, we administered our institutional standard dose of 1.6 mL of 0.5% hyperbaric bupivacaine and 20 µg of fentanyl, achieving a T4 sensory level. Total pre-delivery hydroxyethyl starch reached 1000 mL, which, combined with norepinephrine, maintained blood pressure above 100 mmHg. Transient hypotension during fundal pressure and skin-to-skin contact was managed immediately via arterial catheter monitoring (Figure *1*).

VIDEO 1: Transthoracic echocardiography findings five days prior to surgery

The parasternal long-axis view displays a Y-shaped color mosaic jet, reflecting left ventricular outflow tract obstruction and mitral regurgitation associated with systolic anterior motion of the mitral valve, a characteristic finding of obstructive hypertrophic cardiomyopathy. Note the beat-to-beat variability of the mitral regurgitation color jet.

View video here: https://vimeo.com/1232509892?fl=pl&fe=cm

VIDEO 2: Transthoracic echocardiographic findings prior to anesthesia induction, following the administration of 500 mL of hydroxyethyl starch

The parasternal long-axis view displays a color mosaic jet due to left ventricular outflow tract obstruction, whereas the mitral regurgitation has decreased to a trivial level. The left ventricular end-diastolic diameter expanded from 38 mm preoperatively to 40 mm following volume loading.

View video here: https://vimeo.com/1232512350?fl=pl&fe=cm

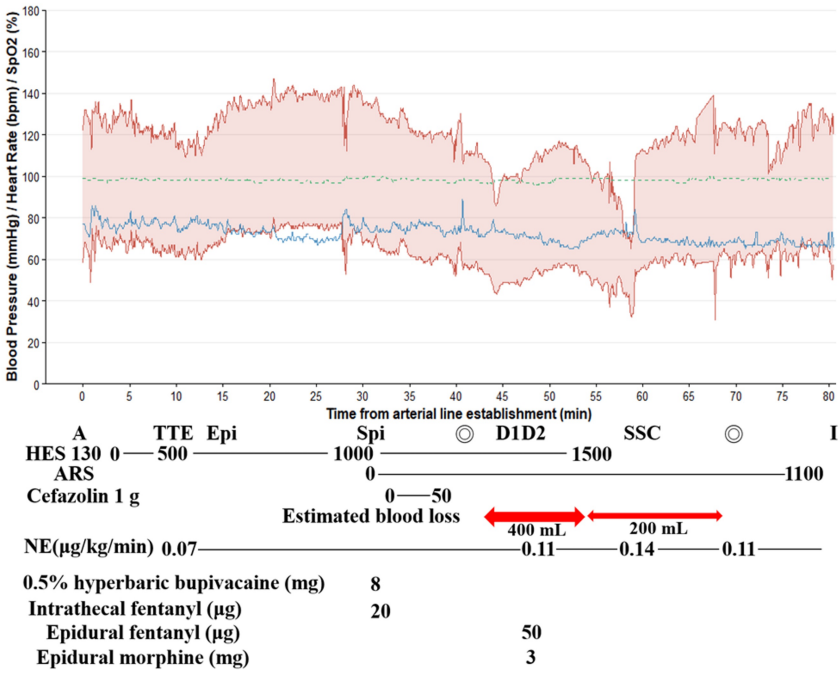


FIGURE 1: Intraoperative hemodynamic trends

Systolic and diastolic blood pressures are shown as a shaded band. The green dashed line represents SpO2, and the blue solid line represents heart rate. The lower panel displays the administration of intravenous fluids, anesthetics, and vasopressors, alongside estimated blood loss.

Key intraoperative hemodynamic events include a transient hypotensive dip (86/42 mmHg) induced by fundal pressure immediately prior to live infant delivery (Marker D1), which recovered to 100/47 mmHg after releasing pressure and titrating NE (0.07 to 0.11 μg/kg/min). During skin-to-skin contact (Marker SSC), positional impairment of the intravenous line led to a secondary acute drop in blood pressure (65/32 mmHg), which promptly resolved to 110/58 mmHg following arm repositioning and further NE titration (0.11 to 0.14 μg/kg/min).

Markers indicate key clinical events in chronological order: A, arterial catheter insertion; TTE, transthoracic echocardiography; Epi, epidural catheter insertion T10/11 interspace; Spi, Spinal anesthesia L3/4 interspace; □, surgery start/end; D1, delivery of live infant; D2, delivery of demised fetus; SSC, skin-to-skin contact; and I, ICU admission.

ARS: acetated Ringer's solution, HES: hydroxyethyl starch, ICU: intensive care unit, NE: norepinephrine, SpO2: transcutaneous arterial oxygen saturation

Following delivery, epidural fentanyl (50 μg) and morphine (3 mg) were administered, and the catheter was removed immediately post-surgery in accordance with our institutional protocol (postoperative fibrinogen: 176 mg/dL, platelet count: 82 × 10^9/L). The total intraoperative fluid volume was 2,650 mL (estimated blood loss: 600 mL, urine output: 830 mL). Anesthesia and operative times were 40 and 30 minutes, respectively. While HES 130/0.42 has a superior safety profile regarding hemostasis compared to older HES

generations [6], potential hemodilution from this pre-delivery 1000 mL load was noted as a background factor regarding the overall dilutional reduction in platelet count and coagulation factor levels, including fibrinogen. The patient was transferred to the intensive care unit (ICU).

Upon ICU admission, uterine atony caused vaginal bleeding. Despite fundal massage and continuous infusions of prostaglandin F2α (600 μg/h) and oxytocin (5 units/h), bleeding continued (estimated loss: 300 mL). Absent clot formation led to a pre-disseminated intravascular coagulation (pre-DIC; score of 6 based on the Japan Society of Obstetrical, Gynecological and Neonatal Hematology for obstetrical DIC criteria [7]) diagnosis, prompting administration of 3 g of fibrinogen concentrate. Concurrently, TTE revealed decreased left ventricular volume and worsening SAM, indicating that volume expansion from subsequent administration of 6 units of fresh frozen plasma (FFP) would be hemodynamically safe and beneficial (Figure *2*). Following fibrinogen administration, clot formation resumed, and hemostasis was achieved using an intrauterine balloon tamponade (Figure *3*). The total postpartum blood loss was 550 mL.

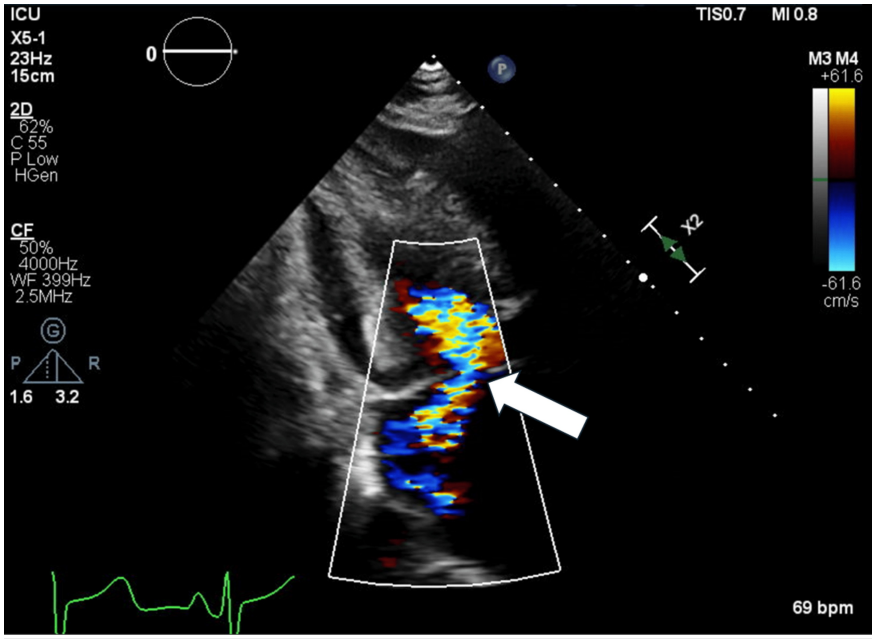


FIGURE 2: Transthoracic echocardiography 90 minutes after ICU admission

Compared with preoperative findings, left ventricular volume is decreased, left ventricular outflow tract stenosis is exacerbated, and mitral regurgitation (white arrow) associated with SAM is worsened.

ICU: intensive care unit, SAM: systolic anterior motion

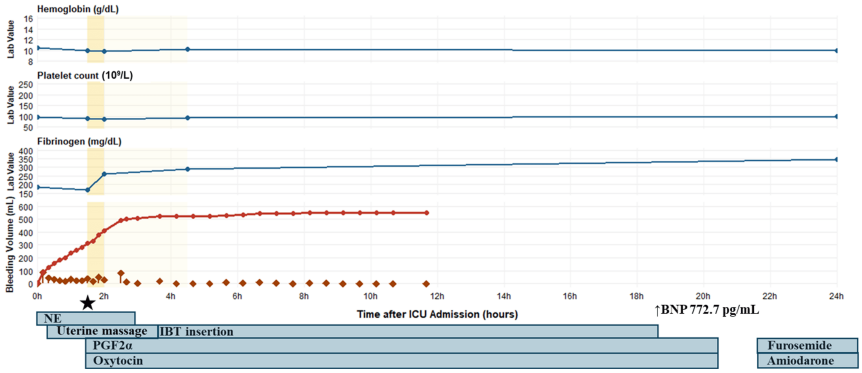


FIGURE 3: Intensive management timeline within 24 hours post-ICU admission

The blue data points represent the test results for hemoglobin, platelet count, and fibrinogen. The red circular data points indicate the cumulative estimated vaginal blood loss over time. The brown diamond-shaped data points represent the blood loss volume at each specific measurement time.

At the time point indicated by the black star (★), the obstetric DIC score was 6 points. The dark yellow band indicates the period during which 3 g of fibrinogen concentrate and 2 units of FFP were rapidly administered over 30 minutes. The subsequent light-yellow band indicates the period during which 4 units of FFP were administered over 2 hours. The grayish-blue bars at the bottom indicate the respective continuous medications and therapeutic interventions.

The baseline laboratory values upon ICU admission were as follows: Hb, 10.5 g/dL; platelet count, 97 × 10⁹/L; and fibrinogen, 176 mg/dL. Following the administration of 3 g of fibrinogen concentrate, the plasma fibrinogen level promptly recovered to 259 mg/dL at 2 hours post-admission (shortly after the start of rapid replacement) and further rose to 287 mg/dL at 4 hours, coinciding with the clinical stabilization of bleeding following intrauterine balloon tamponade.

BNP: brain natriuretic peptide, DIC: disseminated intravascular coagulation, IBT: intrauterine balloon tamponade, ICU: intensive care unit, NE: norepinephrine, PGF2α: prostaglandin F2α

On postoperative day 1, the brain natriuretic peptide level increased to 772.7 pg/mL, and the patient developed atrial fibrillation (AF) with hypotension. Sinus rhythm was restored with continuous amiodarone infusion (25.5 mg/h), and volume status was managed with furosemide (6.8 μg/kg/h). Pathological examination of the placenta following delivery revealed extensive intervillous fibrin deposition within the placental territory of the demised fetus, without evidence of chronic placental abruption or overt tissue necrosis. The patient was discharged from the ICU on day 6 and from the hospital on day 16 postoperatively. Catheter ablation was performed 9 months postoperatively. She has remained free from arrhythmia recurrence and heart failure worsening up to 20 months after surgery.

Discussion

Anesthetic management for pregnant patients with HOCM requires maintaining systemic vascular resistance and adequate preload [8]. While phenylephrine has been used during CS in HOCM [9], its associated reflex bradycardia can compound spinal anesthesia-induced bradycardia, potentially reducing cardiac output. We therefore selected norepinephrine infusion, which effectively maintains maternal heart rate and cardiac output during CS [10]. Although norepinephrine possesses weak β1-adrenergic agonist activity, which could theoretically exacerbate LVOT obstruction, the patient's baseline β-blocker therapy (carvedilol 5 mg/day) was continued perioperatively, mitigating potential β1-stimulated hypercontractility and tachycardia. Our hemodynamic targets were strictly defined as maintaining systolic blood pressure above 100 mmHg (or within 20% of baseline) and maintaining heart rate between 60 and 80 beats/min to optimize diastolic filling without triggering dynamic outflow tract obstruction.

Regarding the modified anesthetic plan, our initial low-dose sequential CSEA was altered due to spinal puncture difficulties. Because incomplete blockade causing intraoperative pain could trigger sympathetic hyperactivity and critically exacerbate HOCM, we administered our institutional standard dose to ensure reliable anesthesia. Although gradual dose titration was bypassed, the epidural catheter served as an immediate safety backup.

Intraoperative skin-to-skin contact during CS requires unique vigilance regarding patient positioning and the safety of intravenous lines and monitoring equipment [11]. In our case, an unexpected infusion failure occurred because elbow flexion while holding the infant kinked the norepinephrine line, causing transient hypotension. However, continuous arterial pressure monitoring enabled immediate detection and correction before circulatory collapse occurred. This experience underscores that in high-risk patients with HOCM with narrow therapeutic margins, skin-to-skin contact may carry risks if performed routinely; adhering to line vigilance [11] and utilizing invasive arterial monitoring are advantageous for ensuring safety under constant supervision.

While long-term in utero retention of a demised co-twin following selective reduction lacks robust established evidence, it may theoretically induce low-grade, subclinical consumptive coagulopathy, potentially contributing to the patient's relatively low baseline fibrinogen level (259 mg/dL) at late gestation (30 weeks). Notably, placental pathology demonstrated extensive intervillous fibrin deposition within the demised co-twin's placental territory, supporting our hypothesis of localized, ongoing fibrinogen consumption during the antenatal period. This pre-existing vulnerability, combined with subsequent uterine atony and hemodilution, likely accelerated the rapid development of severe postpartum hypofibrinogenemia.

When postpartum hemorrhage occurred, obstetric hemostasis was prioritized to prevent a fatal decrease in preload. However, oxytocin carries the risk of inducing vasodilation and tachycardia, potentially exacerbating HOCM pathophysiology. Ferguson et al. reported a HOCM case during CS [12], where a bolus of 1 unit of oxytocin precipitated a sudden reduction in afterload, followed by a decrease in preload, worsening LVOT obstruction and leading to critical hypotension. Aware of this danger, we avoided bolus administration and implemented a low-dose continuous infusion (5 units/h) to safely achieve uterine contraction, while maintaining afterload and preload through meticulous titration of norepinephrine.

According to obstetric hemorrhage guidelines, normal plasma fibrinogen levels in pregnant women reach approximately 500 mg/dL, whereas FFP contains only about 200 mg/dL. Consequently, relying on FFP is inefficient for correcting severe hypofibrinogenemia without causing massive fluid overload [13]. In contrast, fibrinogen concentrate requires no thawing, enables precise dosing, and increases plasma fibrinogen levels by approximately 100 mg/dL per 4 g (60 mg/kg) dose. Therefore, when functional fibrinogen deficiency is rapidly identified via point-of-care testing, some literature advocates prioritizing fibrinogen concentrate over FFP [14]. In our case, point-of-care testing (e.g., viscoelastic testing) was deferred to prioritize acute hemodynamic control and initial management of uterine atony. However, active non-coagulable bleeding and a low postoperative fibrinogen level (176 mg/dL) indicated a clinical coagulopathy driven by hypofibrinogenemia, justifying the prompt administration of 3 g of fibrinogen concentrate. Given that delayed intervention and subsequent hemorrhagic shock could have catastrophically exacerbated HOCM, early fibrinogen replacement was a reasonable approach aimed at minimizing the risk of severe cardiovascular decompensation. Furthermore, bedside TTE revealed intravascular volume depletion along with exacerbation of SAM and mitral regurgitation (Figure *2*). This confirmed that subsequent FFP administration would be hemodynamically safe and beneficial to restore intravascular volume while simultaneously replenishing other coagulation factors. Overall, this clinical outcome reflects the combined contribution of fibrinogen replacement alongside timely uterotonic administration, FFP, and intrauterine balloon tamponade in achieving comprehensive hemostasis.

Despite this cautious approach, the patient developed AF with hypotension and elevated brain natriuretic peptide (772.7 pg/mL) on postoperative day 1. In HOCM, AF is associated with severe symptoms, functional deterioration, and higher mortality, necessitating aggressive rhythm control [15]. Prompt amiodarone administration restored sinus rhythm, and furosemide facilitated strict volume management. This subsequent development of AF and transient hemodynamic vulnerability underscores the patient's extremely narrow therapeutic window for volume expansion, supporting our rationale for avoiding large-volume FFP resuscitation alone in the acute phase to reduce the likelihood of significant volume overload.

Conclusions

Managing HOCM in obstetric settings requires dynamic, tailored strategies optimizing standard anesthetic and obstetric approaches. Real-time TTE was valuable for verifying intracardiac changes, while continuous arterial monitoring detected immediate instability during procedures such as skin-to-skin contact. For postpartum hemorrhage, early coagulation factor replacement, as part of a multimodal hemostatic strategy alongside timely obstetric interventions, was key to halting the vicious cycle of bleeding and hemodynamic deterioration, thereby preserving adequate left ventricular volume, and avoiding cardiac decompensation. Ultimately, seamless multidisciplinary collaboration among anesthesiology, obstetrics, and cardiology was instrumental in achieving a favorable maternal outcome in this case.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Critical review of the manuscript for important intellectual content: Tokimitsu Hibino, Yusuke Okui, Yoshie Toba, Maiko Ikebata, Hideki Saito, Takeshi Murakoshi

Supervision: Yusuke Okui, Yoshie Toba, Maiko Ikebata, Takeshi Murakoshi

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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