

CASE REPORT

Sequential Spinal Anesthesia and Dexmedetomidine-Ketamine Infusion Preserving Spontaneous Ventilation in a Super-Obese Parturient (BMI 63.7 kg/m²) Undergoing Emergency Cesarean Section: An Observational Analytic Case Study

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ABSTRACT

Background. Cesarean delivery in super-obese parturients (body mass index [BMI] ≥ 50 kg/m²) carries a high risk of airway failure and hemodynamic instability, particularly with preeclampsia. When surgery outlasts a single-shot spinal anesthetic, conversion to general anesthesia adds a 'cannot intubate, cannot oxygenate' hazard.

Objective. To describe the cardiorespiratory course of a planned transition from spinal anesthesia to a dexmedetomidine-ketamine infusion in a super-obese parturient undergoing emergency cesarean section.

Methods. Single case report (CARE guideline). A 38-year-old woman (157 kg, 157 cm, BMI 63.7 kg/m², STOP-BANG 5, Mallampati class III) with preeclampsia and fetal hypoxia was followed across five operative epochs (T0 to T4) and postoperative surveillance (T5).

Results. Single-shot spinal anesthesia gave surgical conditions for 120 minutes. When the block regressed, dexmedetomidine (0.5 mcg/kg loading, then 0.5 mcg/kg/h) and ketamine (0.5 mg/kg loading, then 0.5 mg/kg/h) were started and spontaneous ventilation continued. During uterine atony with 2,000 mL blood loss, mean arterial pressure fell to 71.7 mmHg (−37.7% from 115.0 mmHg at baseline) and recovered with crystalloid, colloid, two units of packed red blood cells, uterotonics and norepinephrine, without intubation. The 4,025-g neonate recovered and was discharged on day 10. The mother had no hypoxemic episode, needed under 5 morphine milligram equivalents and was discharged on postoperative day 5.

Conclusion. In this patient, a planned spinal-to-infusion sequence was followed by spontaneous ventilation without airway instrumentation during a prolonged hemorrhagic cesarean delivery. This single observation needs confirmation in larger series.

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1. Introduction

Obstetric practice is facing a rise in maternal adiposity. Parturients with a body mass index (BMI) of 50 kg/m² or more are classed as super-obese.^{1,2} Maternal obesity disturbs several organ systems and erodes homeostatic reserve. A routine operative delivery can become a dangerous perioperative event.^{3,4} In the super-obese parturient, the normal adaptations of pregnancy are distorted by the mechanical and metabolic load of excess adipose tissue.^{5,6} The respiratory system is affected most. The gravid uterus pushes the diaphragm upward and the thoracic and abdominal pannus adds weight on top of it, so functional residual capacity (FRC) can fall by up to 50% below the non-pregnant baseline.^{7,8} In the supine position, closing capacity often exceeds FRC during tidal breathing, and the result is dependent micro-atelectasis, ventilation-perfusion mismatch and an intrapulmonary right-to-left shunt.^{9,10} Maternal oxygen consumption rises to nearly

twice that of lean non-pregnant women. The safe apnea time shrinks to about 60 to 90 seconds.^{8,11}

The cardiovascular system is under comparable strain. Super-obese parturients run a high-output, hyperdynamic circulation, with expanded plasma volume, chronic eccentric left ventricular hypertrophy and raised myocardial wall tension.^{5,12} Superimposed preeclampsia with severe features adds endothelial dysfunction, widespread arteriolar vasospasm and a marked rise in afterload.^{13,14} The myocardium must contract against high systemic vascular resistance (SVR). Subclinical diastolic dysfunction lies underneath. Ventricular filling therefore responds sharply to shifts in preload and afterload.^{5,13} Endothelial injury also increases capillary permeability, which raises the risk of pulmonary edema during intravenous fluid administration, and it promotes consumptive coagulopathy and thrombocytopenia.^{13,14}

Acute fetal hypoxia demands immediate surgical delivery and close coordination among several teams. Choosing the

anesthetic for a super-obese parturient is among the hardest decisions in obstetric anesthesia. Airway failure is common. Redundant pharyngeal soft tissue, a neck circumference above 50 cm, limited atlanto-occipital extension and a small mouth opening together predict a difficult airway.^{15,16} Large multicenter audits show that obstetric patients have a disproportionately high incidence of 'cannot intubate, cannot oxygenate' events, aspiration pneumonitis and hypoxemic cardiac arrest during emergency rapid sequence induction of general anesthesia.^{16,17}

International guidelines therefore recommend regional anesthesia first, so that tracheal instrumentation is avoided and the maternal airway reflexes stay intact.^{15,18} Neuraxial technique is still difficult in the super-obese patient. Surface landmarks are obscured, the skin-to-subarachnoid depth can exceed 8 to 10 cm, and the cephalad spread of hyperbaric local anesthetic is unpredictable because epidural veins are engorged and the lumbosacral cerebrospinal fluid volume is reduced.^{15,18} When fetal distress forces speed, single-shot spinal anesthesia is still the quickest dense and dependable option. It has one fixed limit. Its effect lasts only about 90 to 120 minutes and cannot be extended.¹⁵

An emergency cesarean section in a super-obese patient can run long. Dense adhesions from earlier surgery, a difficult extraction through a thick abdominal wall, or postpartum hemorrhage from uterine atony may all prolong it, and the single-shot block then wears off. The usual response is an emergency conversion to general anesthesia, and that conversion happens under the worst conditions. The patient is draped and supine with an open abdomen. She may be unstable from bleeding, and she may be hypoventilating or aspirating, which makes intubation a crisis.^{11,17} Halogenated volatile agents add a second problem. They relax the myometrium in a dose-dependent way, which worsens atony and speeds blood loss.^{19,20} A contingency strategy, planned in advance, that can rescue a regressed neuraxial block without emergency intubation has been little described.

We therefore describe a sequential approach used in one patient: an initial single-shot spinal anesthetic with a planned transition to total intravenous anesthesia (TIVA) based on dexmedetomidine and ketamine. Dexmedetomidine is a highly selective α -2A adrenergic receptor agonist that acts mainly in the pontine locus coeruleus. It produces cooperative, sleep-like (non-rapid eye movement) sedation. Medullary ventilatory drive and upper airway muscle tone are largely intact.^{21,22} Ketamine is a non-competitive antagonist at the phencyclidine site of the N-methyl-D-aspartate (NMDA) receptor, acting in the dorsal horn and thalamocortical networks, and it gives strong somatic and visceral dissociative analgesia.²³ The two drugs may offset each other's side effects. Dexmedetomidine dampens ketamine-induced tachycardia and emergence agitation, while ketamine counters dexmedetomidine-induced bradycardia and vasodilation.^{24,25} Dexmedetomidine is reported to preserve or increase myometrial contractility. Ketamine has concentration-dependent effects on uterine contractility in vitro (an increase at low and a decrease at high concentrations in rat uterus), so a large effect on the response to exogenous uterotonics is not expected at clinical doses.^{26,27}

The objective of this case report is to describe the longitudinal cardiorespiratory course, hemodynamic

stability and preservation of spontaneous ventilation in a 38-year-old super-obese parturient (weight 157 kg, BMI 63.7 kg/m²) with preeclampsia. She underwent a four-hour emergency cesarean delivery complicated by a 2,000-mL postpartum hemorrhage. Her course was recorded across five operative epochs (T0 to T4) and postoperative high-dependency unit surveillance (T5), with percentage changes from baseline.

2. Methods

2.1. Report design and reporting guideline

This is a report of a single patient, prepared in accordance with the CARE (CAse REport) guideline.^{29,30} The CARE checklist is provided as a supplementary file. The report is organized around five operative epochs (T0 to T4) and a postoperative surveillance epoch (T5), which allow hemodynamic, ventilatory and hemostatic changes to be followed from baseline. No inferential statistics were applied, because a single patient has no group variance.

2.2. Setting

The patient was managed in the Department of Anesthesiology and Intensive Therapy at Dr. Moewardi Regional General Hospital, a tertiary academic referral center affiliated with the Faculty of Medicine, Universitas Sebelas Maret, Surakarta, Central Java, Indonesia. The case unfolded over the emergency admission, a four-hour operation, 24 hours of high-dependency unit (HDU) care and five days of ward recovery. Follow-up continued to day 10 for the neonate and to 6 weeks for the mother.

2.3. Patient information and case selection

The patient was a 38-year-old woman (gravida 2, para 1) at 37 weeks and 1 day of gestation who was transferred as an emergency. She fulfilled the institutional criteria for high-acuity obstetric anesthesia surveillance: maternal age ≥ 18 years, extreme BMI (≥ 50 kg/m²), American Society of Anesthesiologists (ASA) physical status IV E (life-threatening emergency), and comorbid cardiopulmonary or metabolic disease that called for a specialized anesthetic bridging plan. Her comorbidities were type 2 diabetes mellitus and controlled asthma. She had undergone a low transverse cesarean section four years earlier for cephalopelvic disproportion. On arrival at the emergency obstetric triage unit, body weight and height were measured with calibrated scales and anthropometric devices (weight 157.0 kg, standing height 157.0 cm; BMI 63.7 kg/m²). The case was selected because extreme super-obesity, preeclampsia and acute fetal compromise coincided with surgery that ran far beyond the pharmacological window of a single-shot spinal anesthetic, and because the planned response avoided general anesthesia.

2.4. Case timeline

The chronology is set out in Table 1 using relative time. Operative time is counted from the start of spinal anesthesia (00:00). Epoch T0 was the preoperative baseline at emergency admission. Epoch T1 covered spinal anesthesia onset and the stable surgical period (00:00–02:00). Epoch T2 covered spinal block regression and the planned TIVA transition (02:00–02:40). Epoch T3 covered severe postpartum hemorrhage and resuscitation (02:40–03:30). Epoch T4 covered hemostatic stabilization, abdominal closure and resolution (03:30–04:00). Epoch T5

was postoperative HDU surveillance, from postoperative day (POD) 0 to POD 5.

Table 1. Clinical timeline of the case: events, investigations and interventions from emergency admission to last follow-up, using relative time.

Time point	Clinical event	Investigation and result	Intervention
Year –4	Low transverse cesarean section for cephalopelvic disproportion	—	—
Day 0, T0 (emergency admission)	Emergency transfer at 37 weeks + 1 day; acute labor, severe preeclamptic hypertension, pathological cardiotocogram (Category III)	BP 155/95 mmHg (MAP 115.0), HR 98/min, RR 22/min, SpO ₂ 96% (room air); platelets 120,000/μL; AST 58, ALT 64 U/L; proteinuria 3+; Hb 11.8 g/dL; fibrinogen 380 mg/dL	Multidisciplinary huddle; spinal anesthesia chosen with a planned infusion contingency
Operative minute 00:00 (T1)	Single-shot spinal anesthesia (L3–L4, sitting)	Skin-to-subarachnoid distance 8.6 cm; sensory block T4 at 12 min; MAP 91.7 mmHg, HR 82/min	Hyperbaric bupivacaine 0.5% 10 mg + fentanyl 25 mcg; ephedrine 10 mg in total; left lateral tilt
Minute 00:35	Delivery of a male neonate, 4,025 g	Apgar 4, 5 and 7 at 1, 5 and 10 min	Neonatal resuscitation (positive-pressure ventilation); oxytocin 20 IU infusion
Minutes 02:00–02:40 (T2)	Spinal block regressed to T10 with pain during pelvic traction	RASS –2 to –3; RR 14–16/min; SpO ₂ 98% (nasal oxygen 4 L/min); MAP 96.7 mmHg	Crisis huddle; dexmedetomidine and ketamine loading and infusion from 02:05
Minutes 02:40–03:30 (T3)	Uterine atony; blood loss 2,000 mL	MAP 71.7 mmHg, HR 108/min, shock index 1.14; Hb 7.6 g/dL; platelets 84,000/μL; fibrinogen 185 mg/dL; lactate 3.4 mmol/L	Methylergometrine, misoprostol, tranexamic acid; crystalloid 2,500 mL, colloid 500 mL, 2 units PRBC; norepinephrine
Minutes 03:30–04:00 (T4)	Hemostasis; closure at 04:00	MAP 88.0 mmHg; GCS 15, RASS 0; urine output 450 mL (cumulative)	Ketamine stopped at 03:40; dexmedetomidine reduced to 0.3 mcg/kg/h
POD 0 (T5)	HDU surveillance for 24 h	SpO ₂ 97–99% on room air; no desaturation; NRS 2–3 at rest	Paracetamol and ketorolac; enoxaparin 12 h after hemostasis
POD 1	Ward care	Hb 9.8 g/dL; platelets 105,000/μL; fibrinogen 290 mg/dL; lactate 1.5 mmol/L	Opioid-free analgesia
POD 5	Maternal discharge	Hb 10.9 g/dL; platelets 168,000/μL; AST 28, ALT 31 U/L; lactate 0.9 mmol/L	Enoxaparin continued for 6 weeks
POD 10	Neonatal discharge	Weaned to ambient air; feeding well since day 3	—
6 weeks	Maternal follow-up	BP 120/78 mmHg	Enoxaparin course completed

Notes: Times are relative to the start of spinal anesthesia (operative minute 00:00); POD, postoperative day. Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; BP, blood pressure; GCS, Glasgow Coma Scale; Hb, hemoglobin; HDU, high-dependency unit; HR, heart rate; MAP, mean arterial pressure; NRS, numeric rating scale; PRBC, packed red blood cells; RASS, Richmond Agitation-Sedation Scale; RR, respiratory rate; SpO₂, peripheral oxygen saturation.

2.5. Clinical findings and diagnostic assessment

Preeclampsia with severe features was diagnosed according to international criteria.^{31,32} The supporting findings were persistent systolic blood pressure $\geq$ 155 mmHg, diastolic pressure $\geq$ 95 mmHg, dipstick proteinuria of 3+ and biochemical evidence of hepatic injury (aspartate aminotransferase [AST] 58 U/L, alanine aminotransferase [ALT] 64 U/L). Airway geometry was assessed by bedside examination (Mallampati classification,^{33,34} neck circumference, atlanto-occipital extension, thyromental distance and inter-incisor opening), and obstructive sleep apnea risk by the STOP-BANG questionnaire.^{35,36} Fetal status was assessed by continuous external cardiotocography. A Category III tracing (recurrent late decelerations with persistent bradycardia) was taken to indicate acute fetal hypoxia and to require abdominal delivery within a 30-minute decision-to-incision window.³⁷

2.6. Therapeutic intervention

After a rapid huddle among the senior obstetric, anesthesiology and neonatology teams, single-shot spinal anesthesia was chosen as the primary technique so that airway instrumentation would be avoided. A pre-planned

contingency was agreed: if the block regressed, anesthesia would be continued with an opioid-sparing intravenous regimen of dexmedetomidine and ketamine rather than by conversion to general anesthesia. The spinal anesthetic was 10 mg of hyperbaric bupivacaine 0.5% with 25 mcg of preservative-free fentanyl at L3–L4. The planned infusion regimen was dexmedetomidine 0.5 mcg/kg over 10 minutes followed by 0.5 mcg/kg/h, and ketamine 0.5 mg/kg followed by 0.5 mg/kg/h. In this report the regimen is called TIVA, as in the submitted text; at these doses, with a spinal block that had regressed only to T10, it corresponds to sedation-analgesia supplementing the block rather than general anesthesia. Postpartum hemorrhage was to be treated with a stepwise uterotonic protocol, tranexamic acid, volume replacement and vasopressor support. Postoperatively, the plan comprised HDU surveillance, multimodal opioid-sparing analgesia and extended thromboprophylaxis.

2.7. Outcome measures and follow-up

Observation began when the patient entered the operating theatre and continued through 24 hours of HDU care and five days of ward recovery. Measurements were captured continuously and logged every 5 minutes. Monitoring

comprised multi-lead electrocardiography, continuous pulse oximetry, invasive radial arterial pressure through a 20-gauge catheter zeroed at the right atrium, and automated oscillometric cuff readings with a conical bariatric thigh cuff as verification. Sedation depth and arousal were scored serially with the Richmond Agitation-Sedation Scale (RASS)³⁸ and the Bispectral Index. Respiratory mechanics and gas exchange were assessed with serial arterial blood gases, continuous side-stream capnography adapted to a high-flow oxygen mask, and respiratory rate. Cumulative fluid input, crystalloid and colloid volumes, estimated blood loss and hourly urine output (through an indwelling temperature-sensing Foley catheter) were recorded. Venous blood was drawn at baseline (T0), at peak hemorrhage (T3), on POD 1 and on POD 5 for complete blood count, coagulation profile, electrolytes, renal function, hepatic transaminases and lactate.

The outcomes of interest were: (1) avoidance of endotracheal intubation with preservation of spontaneous ventilatory drive throughout the procedure; (2) a mean arterial pressure (MAP) within a target window of 65 to 100 mmHg without prolonged vasopressor dependence; (3) control of postpartum hemorrhage, with reversal of uterine atony under non-volatile intravenous anesthesia; (4) postoperative analgesia quality, measured by numeric rating scale (NRS) pain scores and cumulative 24-hour opioid use in morphine milligram equivalents (MME); and (5) neonatal viability and recovery, assessed with 1-, 5- and 10-minute Apgar scores, umbilical cord blood gas analysis and time to clinical resolution in the neonatal high-care unit. Secondary safety outcomes were maternal postoperative respiratory depression, surgical site infection, venous thromboembolism and satisfaction with participation in childbirth.

Data are presented descriptively against standard normative physiological and biochemical reference intervals. Percentage change from baseline was calculated as $\Delta\% = [(T_n - T_0) / T_0] \times 100\%$. Trajectory plots and multi-panel time-series figures were produced in Python 3.11 with Matplotlib and NumPy and rendered at 300 DPI.

2.8. Ethics and consent

The CMHC Research Center Ethics Committee, Indonesia, approved the report (protocol number 2024/7183). The work followed the Declaration of Helsinki and the International Council for Harmonisation Good Clinical

Practice guidelines.³⁹ Written informed consent was obtained from the patient and her legal surrogate for observational recording, laboratory profiling and photographic documentation of her perioperative course, in line with the publication ethics recommendations of the International Committee of Medical Journal Editors (ICMJE) and the Committee on Publication Ethics (COPE).⁴⁰ She was told that all clinical data, time-series physiological values and diagnostic material would be fully anonymized before analysis and publication, with institutional protection of her privacy. Every photograph and diagnostic schematic was checked against international privacy requirements, and facial or otherwise identifiable landmarks were either excluded or permanently covered with solid black bars.

3. Results

3.1. Presentation and baseline findings

A 38-year-old multigravida (gravida 2, para 1) at 37 weeks and 1 day of gestation came by emergency transfer to the tertiary labor and delivery suite. She had acute labor onset, severe preeclamptic hypertension and a pathological fetal cardiotocogram (Table 1). Her calculated body mass index was 63.7 kg/m² (weight 157.0 kg, height 157.0 cm), which is above the World Health Organization class III obesity threshold (BMI ≥ 40 kg/m²) and in the super-obese range (BMI ≥ 50 kg/m²). Physiological reserve was profoundly depleted. Four years earlier she had undergone a low transverse cesarean section for cephalopelvic disproportion, so extensive intra-abdominal adhesions and a slow surgical entry were anticipated. On admission, continuous fetal heart rate monitoring showed severe persistent bradycardia (baseline 95 to 105 beats/min) and repetitive late decelerations unrelated to maternal contractions. These met Category III criteria for acute fetal hypoxia and called for surgical delivery within 30 minutes.

At Epoch T0 she was hypertensive. Systolic pressure was 155 mmHg, diastolic pressure 95 mmHg and mean arterial pressure (MAP) 115.0 mmHg. She also had sinus tachycardia of 98 beats/min and a respiratory rate of 22 breaths/min. Pulse oximetry on ambient air read 96%, which fits basal micro-atelectasis from cephalad displacement of the diaphragm by the pannus and gravid uterus. The baseline anthropometric, airway and cardiorespiratory findings are summarized in Table 2.

Table 2. Baseline anthropometric, airway and preoperative cardiorespiratory parameters in a super-obese parturient (BMI 63.7 kg/m²).

Clinical or physiological parameter	Observed value	Reference interval	Physiological derangement and risk implication
Maternal Age	38 years	18–35 years (standard obstetric range)	Advanced maternal age; elevated preeclampsia risk
Obstetric Profile & Parity	G2P1, 37 weeks + 1 day	Term pregnancy (37–42 weeks)	Previous cesarean section scar; acute urgent labor
Maternal Weight	157 kg	50–75 kg (ideal maternal weight)	Massive physical mass; severe logistical positioning challenges
Maternal Height	157 cm	150–170 cm	Short stature amplifying body mass index
Body Mass Index (BMI)	63.7 kg/m ²	18.5–24.9 kg/m ² (normal)	Class III obesity (BMI ≥ 40 kg/m ²) and super-obesity (BMI ≥ 50 kg/m ²); reduced FRC
Mallampati Airway Classification	Class III	Class I–II	High risk of difficult direct and video laryngoscopy
Cervical Neck Circumference	> 50 cm	< 40 cm	Predictor of difficult mask ventilation and tracheal intubation
Cervical Spine Mobility	Severely limited extension	Full extension (> 35°)	Impaired sniffing position alignment; rapid desaturation
Thyromental Distance	5.5 cm	> 6.5 cm	Short mandibular space; acute anterior laryngeal angle
Inter-incisor Mouth Opening	3.0 cm	> 4.0 cm	Limited oral corridor for airway instrumentation
STOP-BANG Score	5 / 8 (High OSA Risk)	0–2 (low risk)	Severe risk of perioperative upper airway collapse and apnea
Pre-existing Comorbidities	Type 2 diabetes mellitus, controlled asthma	Absence of chronic disease	Elevated surgical site infection and bronchospasm hazards
Primary Obstetric Diagnosis	Preeclampsia with severe features	Normotensive pregnancy	Systemic endothelial vasospasm, leaky capillaries, high SVR
Urgent Surgical Indication	Acute Fetal Hypoxia (CTG Category III)	Reassuring fetal status	Emergency time pressure; precludes awake fiberoptic setup
Systolic Blood Pressure (SBP)	155 mmHg	100–120 mmHg	Preeclamptic arteriolar vasoconstriction
Diastolic Blood Pressure (DBP)	95 mmHg	60–80 mmHg	Elevated systemic afterload and ventricular wall stress
Mean Arterial Pressure (MAP)	115.0 mmHg	70–93 mmHg	Severe hyperdynamic vascular state; preload sensitivity
Baseline Heart Rate (HR)	98 beats/min	60–90 beats/min	Compensatory tachycardia in high-output obesity state
Respiratory Rate	22 breaths/min	12–18 breaths/min	Tachypnea compensating for elevated oxygen consumption
Peripheral Oxygen Saturation (SpO ₂)	96% (ambient air)	97–100%	Subclinical dependent atelectasis and V/Q mismatching
Baseline Platelet Count	120,000 /μL	150,000–450,000 /μL	Mild gestational/preeclamptic thrombocytopenia
Hepatic Transaminases (AST / ALT)	58 U/L / 64 U/L	AST < 35 U/L, ALT < 40 U/L	End-organ hepatic involvement secondary to preeclampsia

Notes: Values were obtained during multidisciplinary preoperative assessment on emergency admission. Abbreviations: BMI, body mass index; CTG, cardiotocography; DBP, diastolic blood pressure; FRC, functional residual capacity; HR, heart rate; MAP, mean arterial pressure; OSA, obstructive sleep apnea; SBP, systolic blood pressure; SpO₂, peripheral oxygen saturation; STOP-BANG, snoring, tiredness, observed apnea, high blood pressure, BMI, age, neck circumference, gender; SVR, systemic vascular resistance.

3.2. Diagnostic findings

The airway examination found several extreme predictors of difficult mask ventilation, videolaryngoscopy and tracheal intubation (Figure 1). Mallampati Class III was recorded; at maximal effort only the base of the uvula and the soft palate could be seen. Neck circumference exceeded 50 cm. Redundant pretracheal adipose tissue and a

prominent dorsal cervical fat pad were present. Atlanto-occipital extension was severely restricted (< 20° from neutral), so the oral, pharyngeal and laryngeal axes could not be aligned. Thyromental distance was 5.5 cm and inter-incisor opening 3.0 cm. The STOP-BANG score was 5 out of 8, indicating a high risk of obstructive sleep apnea and pharyngeal collapse.

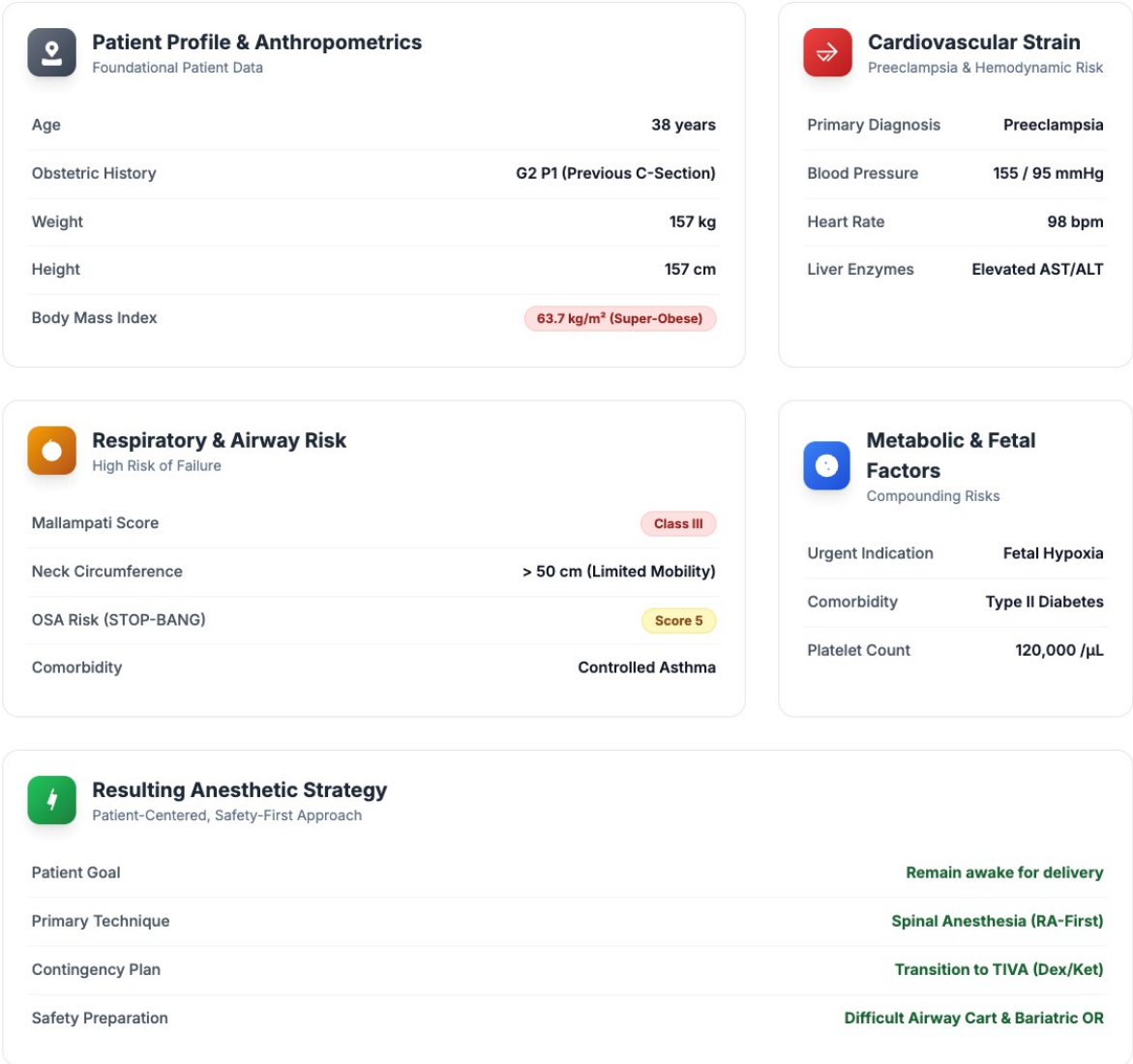


Table 3. Longitudinal hematological, coagulation and biochemical laboratory values across perioperative epochs.

Laboratory parameter	Reference interval	Baseline (T0)	Hemorrhage (T3)	POD 1	POD 5	Δ% (T3 vs T0)
Hemoglobin (g/dL)	11.5–15.5 g/dL	11.8	7.6	9.8	10.9	–35.6%
Hematocrit (%)	36.0–46.0%	35.4	22.8	29.4	32.7	–35.6%
Platelet Count ($\times 10^3/\mu\text{L}$)	150–450 $\times 10^3/\mu\text{L}$	120	84	105	168	–30.0%
White Blood Cells ($\times 10^3/\mu\text{L}$)	4.5–11.0 $\times 10^3/\mu\text{L}$	10.4	14.8	12.2	8.6	+42.3%
Prothrombin Time (PT, sec)	11.0–13.5 sec	12.1	15.4	12.8	11.9	+27.3%
Activated Partial Thromboplastin Time (aPTT, sec)	25.0–35.0 sec	28.4	38.6	30.2	27.5	+35.9%
Plasma Fibrinogen (mg/dL)	300–600 mg/dL (pregnancy)	380	185	290	395	–51.3%
Aspartate Aminotransferase (AST, U/L)	10–35 U/L	58	72	44	28	+24.1%
Alanine Aminotransferase (ALT, U/L)	10–40 U/L	64	81	52	31	+26.6%
Blood Urea Nitrogen (BUN, mg/dL)	7–20 mg/dL	14.0	18.0	15.0	12.0	+28.6%
Serum Creatinine (mg/dL)	0.50–1.10 mg/dL	0.82	1.05	0.88	0.76	+28.0%
Random Blood Glucose (mg/dL)	70–140 mg/dL	148	172	134	118	+16.2%
Serum Lactate (mmol/L)	0.5–1.6 mmol/L	1.2	3.4	1.5	0.9	+183.3%

Notes: $\Delta\% = [(T3 - T0) / T0] \times 100\%$. T0, preoperative baseline; T3, peak hemorrhage; POD 1, first postoperative day in the high-dependency unit (T5a in the submitted table); POD 5, fifth postoperative day before discharge (T5b). Acute coagulopathy and transaminase elevation had resolved by POD 5. Abbreviations: ALT, alanine aminotransferase; aPTT, activated partial thromboplastin time; AST, aspartate aminotransferase; BUN, blood urea nitrogen; PT, prothrombin time.

3.3. Treatment course and outcome

3.3.1. Single-shot spinal anesthesia (Epoch T1)

The patient sat on a heavy-duty bariatric operating table rated for 450 kg. Preprocedural lumbar ultrasonography identified the L3–L4 interspace and measured an acoustic skin-to-subarachnoid distance of 8.6 cm. After sterile preparation, a 25-gauge, 90-mm Quincke needle was advanced in the midline. Clear cerebrospinal fluid flowed freely on the second pass. The intrathecal mixture was 10 mg of hyperbaric bupivacaine 0.5% (2.0 mL) with 25 mcg of preservative-free fentanyl (0.5 mL). It was injected slowly over 30 seconds without barbotage. She was returned to the supine position at once, with a 15-degree left lateral tilt from an inflatable wedge to relieve aortocaval compression. Within 12 minutes of injection, the bilateral sensory block to pinprick reached T4, with a complete motor block of the lower limbs (Bromage score 3).

During Epoch T1 (00:00–02:00), the dense neuraxial block gave operating conditions and circulation that remained within target limits (mean systolic 125 mmHg, diastolic 75 mmHg, MAP 91.7 mmHg, heart rate 82 beats/min). Only modest vasopressor titration was needed: intravenous ephedrine 10 mg in total, in divided 5-mg boluses, to counter venodilation after sympathectomy. Breathing was spontaneous. Arterial oxygen saturation was 99% on 3 L/min of nasal cannula oxygen and the respiratory rate

was 16 breaths/min. Entering the abdomen through the massive pannus was exceptionally difficult. It required a modified Pfannenstiel incision and mechanical cephalad retraction of the 25-kg adipose apron. At operative minute 00:35 a male neonate was delivered, depressed at birth, with a birth weight of 4,025 g (macrosomic). He had moderate birth asphyxia, with Apgar scores of 4 at 1 minute, 5 at 5 minutes and 7 at 10 minutes. The neonatal resuscitation team, stationed nearby, took him at once for positive-pressure ventilation and thermal support. A routine intravenous oxytocin infusion (20 IU in 500 mL lactated Ringer's solution) was then started.

3.3.2. Spinal block regression and transition to dexmedetomidine-ketamine infusion (Epoch T2)

After delivery, the surgeons found dense, vascularized pelvic adhesions fixing the lower uterine segment, the bladder flap and the previous hysterotomy scar. What should have been a standard hysterotomy closure became an extensive four-hour microdissection (the intraoperative timeline is shown in Figure 2). At operative minute 02:00 (120 minutes after induction) the spinal block regressed, and the patient entered Epoch T2. The sensory level fell to T10, and she began to report acute pain and somatic discomfort during deep pelvic traction. General endotracheal anesthesia was not used. Instead, the team held the pre-planned crisis huddle, confirmed her hemodynamic parameters and activated the sequential opioid-sparing TIVA contingency protocol.

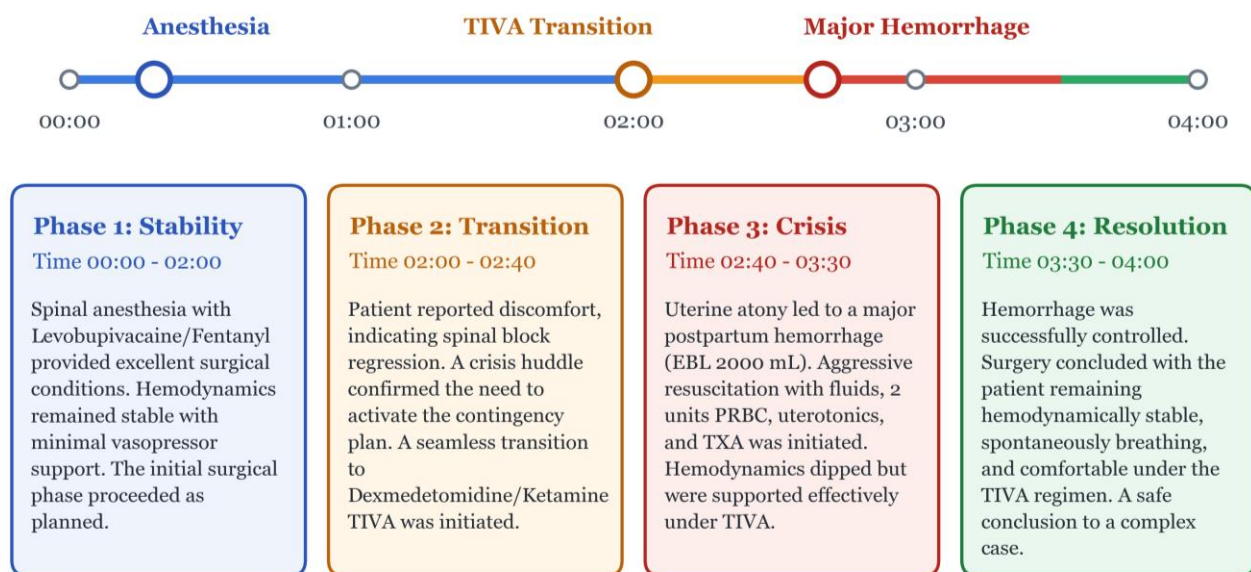


Figure 2. Intraoperative timeline and key events across four phases: Phase 1, stability (00:00–02:00) under single-shot spinal anesthesia; Phase 2, transition (02:00–02:40) triggered by block regression to T10 and initiation of dexmedetomidine-ketamine TIVA after a crisis huddle; Phase 3, crisis (02:40–03:30) with a 2,000-mL postpartum hemorrhage from uterine atony; Phase 4, resolution (03:30–04:00) with spontaneous breathing and stable hemodynamics. The figure was redrawn with the same content. Abbreviations: EBL, estimated blood loss; PRBC, packed red blood cells; TXA, tranexamic acid.

The infusion began at 02:05 (Table 4 details the dosing sequence). Dexmedetomidine hydrochloride was loaded intravenously at 0.5 mcg/kg (80 mcg in total, diluted in 50 mL normal saline) over 10 minutes through a smart infusion pump. A maintenance infusion followed at 0.5 mcg/kg/h (80 mcg/h). Racemic ketamine hydrochloride was given as an intravenous bolus of 0.5 mg/kg (80 mg in total), followed by a syringe infusion of 0.5 mg/kg/h (80 mg/h). Within six minutes of starting both infusions, she was calm and cooperative, with a RASS score of –2 to –3.

Pain complaints and grimacing had stopped. Breathing stayed spontaneous. The respiratory rate settled at 14 to 16 breaths/min, end-tidal carbon dioxide stayed normal (36 to 38 mmHg) and arterial oxygen saturation held at 98% on 4 L/min of nasal oxygen. There was no upper airway collapse, no tongue fall and no need for airway instrumentation. Hemodynamics in Epoch T2 remained steady: mean systolic pressure 130 mmHg, diastolic pressure 80 mmHg, MAP 96.7 mmHg and heart rate 86 beats/min.

Table 4. Sequential anesthetic protocol, dosing and resuscitation pharmacotherapy.

Operative phase and duration	Anesthetic and pharmacological regimen	Agent and target receptor	Dose and administration rate	Pharmacokinetic dynamics and synergism	Clinical endpoint and safety outcome
Phase 1: Neuraxial Blockade (00:00–02:00)	Single-Shot Spinal Anesthesia (Primary Technique)	Hyperbaric bupivacaine 0.5% (10 mg) + fentanyl 25 mcg (2.5 mL total)	Subarachnoid injection at L3–L4 via 25G Quincke needle (sitting position)	Rapid intrathecal uptake; peak sensory block to T4 achieved in 12 min	Successful delivery of 4,025-g infant at 00:35; zero airway instrumentation; stable MAP
Phase 2: Transition Protocol (02:00–02:40)	Total Intravenous Anesthesia (Pre-planned Contingency)	Dexmedetomidine HCl (α_2A agonist) + Ketamine HCl (NMDA antagonist)	Dexmedetomidine: 0.5 mcg/kg loading (80 mcg IV over 10 min), then 0.5 mcg/kg/hr; Ketamine: 0.5 mg/kg bolus (80 mg IV), then 0.5 mg/kg/hr	Central sympatholysis counteracts ketamine tachycardia; profound somatic/visceral analgesia without respiratory depression	Seamless transition upon sensory regression to T10; RASS –2 to –3; spontaneous breathing preserved (RR 14–16 bpm, SpO ₂ 98%)
Phase 3: Hemorrhagic Resuscitation (02:40–03:30)	Multimodal Uterotonic Cascade & Hemostatic Defense	Oxytocin + Methylergometrine + Misoprostol + Tranexamic acid	Oxytocin 20 IU in 500 mL LR; Methylergometrine 0.2 mg IM; Misoprostol 800 mcg rectal; TXA 1 g IV bolus	Synergistic myometrial receptor activation; TXA competitively inhibits plasminogen activation	Arrest of uterine atony; clot stabilization; blood loss contained at 2,000 mL
Phase 3 (continued): Hemodynamic Support (02:40–03:30)	Volume Resuscitation & Vasopressor Titration	Balanced crystalloid + Synthetic colloid + Packed red blood cells + Norepinephrine	Crystalloid: 2,500 mL; Colloid: 500 mL; PRBC: 2 units (~500 mL); Norepinephrine: 0.05–0.1 mcg/kg/min	Restoration of circulating intravascular volume and capillary oncotic pressure; peripheral α_1 -vasoconstriction	MAP defended above 72 mmHg; heart rate controlled (108 bpm); avoidance of hypoperfusion; zero pulmonary edema
Phase 4: Operative Resolution (03:30–04:00)	Sedation Weaning & Emergence Preparation	Dexmedetomidine tapered; Ketamine infusion discontinued at skin closure	Dexmedetomidine reduced to 0.3 mcg/kg/hr; Ketamine stopped at 03:40; cumulative: Dex 140 mcg, Ket 200 mg	Gradual drug clearance; context-sensitive half-time allows clear emergence without delirium	Surgery concluded at 04:00; patient transferred to HDU awake, calm, comfortable, spontaneously breathing, GCS 15

Notes: Abbreviations: GCS, Glasgow Coma Scale; HDU, high-dependency unit; IM, intramuscular; IV, intravenous; LR, lactated Ringer's; MAP, mean arterial pressure; NMDA, N-methyl-D-aspartate; PRBC, packed red blood cells; RASS, Richmond Agitation-Sedation Scale; RR, respiratory rate; SpO₂, peripheral oxygen saturation; TXA, tranexamic acid.

3.3.3. Uterine atony and postpartum hemorrhage (Epoch T3)

At operative minute 02:40 (Epoch T3, 02:40–03:30), a postpartum hemorrhage abruptly destabilized the case. It came from acute uterine atony and extensive venous ooze from deep adhesiolysis. The myometrium became completely flaccid. It failed to contract despite the ongoing

oxytocin infusion. Estimated blood loss reached 2,000 mL by the end of Epoch T3, as Table 5 shows across the operative epochs. The hypovolemia produced a sharp fall in pressure. Systolic pressure reached a nadir of 95 mmHg and diastolic pressure 60 mmHg. MAP fell to 71.7 mmHg, a percentage change of $\Delta -37.7\%$ from the T0 baseline of 115.0 mmHg. Heart rate rose to 108 beats/min (+10.2%) and the shock index to 1.14 (+81.0%).

Table 5. Intraoperative hemodynamic, respiratory and fluid values across the operative epochs.

Physiological and fluid metric	T0 baseline (preoperative admission)	T1 spinal block (mean, 0–120 min)	T2 TIVA transition (120–160 min)	T3 hemorrhagic crisis (160–210 min)	T4 resolution (210–240 min)	$\Delta\%$ (T3 vs T0)
Systolic Blood Pressure (SBP, mmHg)	155	125	130	95	120	–38.7%
Diastolic Blood Pressure (DBP, mmHg)	95	75	80	60	72	–36.8%
Mean Arterial Pressure (MAP, mmHg)	115.0	91.7	96.7	71.7	88.0	–37.7%
Heart Rate (HR, beats/min)	98	82	86	108	88	+10.2%
Shock Index (HR / SBP)	0.63	0.66	0.66	1.14	0.73	+81.0%
Respiratory Rate (breaths/min)	22	16	15	18	16	–18.2%
Peripheral Oxygen Saturation (SpO ₂ , %)	96% (Ambient)	99% (Nasal 3 L/min)	98% (Nasal 4 L/min)	97% (Mask 6 L/min)	99% (Nasal 3 L/min)	+1.0%
Arterial PaO ₂ / FiO ₂ Ratio (mmHg)	380	440	425	390	430	+2.6%
Cumulative Crystalloid Infusion (mL)	0	1,000	1,500	2,500	2,800	N/A
Cumulative Colloid Infusion (mL)	0	0	0	500	500	N/A
Packed Red Blood Cells (PRBC, units / mL)	0 / 0	0 / 0	0 / 0	2 units (~500 mL)	2 units (~500 mL)	N/A
Estimated Blood Loss (EBL, mL)	0	300	450	2,000	2,000	N/A
Cumulative Urine Output (mL)	0	150	220	380	450	N/A
Net Intraoperative Volumetric Fluid Balance (mL)	0	+550	+830	+1,120	+1,350	N/A

Notes: T0, baseline before surgery; T1, stable spinal phase (00:00–02:00); T2, spinal regression and TIVA transition (02:00–02:40); T3, hemorrhagic crisis and resuscitation (02:40–03:30); T4, hemostatic stabilization and closure (03:30–04:00). Net volumetric balance = total IV input – (EBL + urine output). The $\Delta\%$ of MAP was recalculated from the tabulated mean (71.7 mmHg). Abbreviations: DBP, diastolic blood pressure; EBL, estimated blood loss; HR, heart rate; MAP, mean arterial pressure; PaO₂/FiO₂, ratio of arterial oxygen partial pressure to fractional inspired oxygen; SBP, systolic blood pressure; SpO₂, peripheral oxygen saturation.

A multi-tiered hemostatic and fluid resuscitation protocol was started without delay. Uterotonic therapy was escalated step by step. Methylergometrine 0.2 mg was injected intramuscularly into the deltoid, misoprostol 800 mcg was given per rectum, and tranexamic acid 1.0 g in 100 mL normal saline was infused over 10 minutes. Resuscitation used rapid-infusion bariatric lines: two 14-gauge peripheral catheters and the 20-gauge radial arterial line. She received 2,500 mL of balanced crystalloid (lactated Ringer's), 500 mL of 4% succinylated gelatin colloid (Gelafusal) and two units of cross-matched packed red blood cells (~500 mL) warmed to 37.0°C. Norepinephrine was started as a peripheral infusion titrated between 0.05 and 0.10 mcg/kg/min and held MAP above 72 mmHg. She kept breathing spontaneously throughout the hemorrhage, without desaturation. SpO₂ was 97% on a simple face mask at 6 L/min, the respiratory rate was 18 breaths/min, and the blood gas showed adequate oxygenation (arterial PaO₂/FiO₂ ratio 390 mmHg) with no hypercapnia or acidemia.

Serial laboratory tests at the height of the hemorrhage captured the effects of acute blood loss and resuscitation. Hemoglobin fell from 11.8 to 7.6 g/dL ($\Delta -35.6\%$), hematocrit from 35.4% to 22.8% ($\Delta -35.6\%$) and platelets from 120,000 to 84,000 / μ L ($\Delta -30.0\%$). Coagulation tests showed consumptive changes. PT lengthened to 15.4 seconds ($\Delta +27.3\%$), aPTT to 38.6 seconds ($\Delta +35.9\%$) and

fibrinogen dropped to 185 mg/dL ($\Delta -51.3\%$). Lactate rose to 3.4 mmol/L ($\Delta +183.3\%$), reflecting transient tissue hypoperfusion. After the full uterotonic cascade, manual bimanual uterine compression and careful oversewing of the placental bed, the myometrium contracted firmly and steadily. Active hemorrhage stopped at operative minute 03:30.

3.3.4. Resolution and closure (Epoch T4)

Epoch T4 (03:30–04:00) brought rapid physiological stabilization and completion of the operation. After definitive surgical hemostasis, the ketamine infusion was stopped at minute 03:40 (cumulative intraoperative ketamine 200 mg). The dexmedetomidine infusion was tapered to 0.3 mcg/kg/h (cumulative dexmedetomidine 140 mcg) to allow smooth cognitive clearing. Total operative duration was exactly 240 minutes (4 hours 00 minutes). Fluid accounting showed cumulative crystalloid of 2,800 mL, colloid of 500 mL, two units of packed red blood cells (500 mL), total estimated blood loss of 2,000 mL and cumulative urine output of 450 mL. The net fluid balance was +1,350 mL, without peripheral or pulmonary edema. At skin closure the hemodynamics were stable: systolic pressure 120 mmHg, diastolic pressure 72 mmHg, MAP 88.0 mmHg and heart rate 88 beats/min. The patient was awake, calm, comfortable and oriented (Glasgow Coma Scale 15, RASS 0). Her airway was patent. She breathed

room air spontaneously. Endotracheal intubation and intensive care mechanical ventilation were avoided.

3.3.5. Postoperative surveillance and outcome (Epoch T5)

Recovery in the HDU (Epoch T5, POD 0 to POD 5) went well for mother and neonate (Table 6 gives the details, and Figure 3 summarizes the maternal-neonatal recovery). During the first 24 hours of HDU care, continuous pulse oximetry and capnography were used with 45-degree semi-upright positioning. They showed no episode of arterial desaturation (SpO₂ consistently 97–99% on room air, no events < 92%) and no upper airway obstruction. Multimodal opioid-sparing analgesia consisted of scheduled intravenous paracetamol 1.0 g every 6 hours and intravenous ketorolac 30 mg every 8 hours. It controlled pain well (NRS 2–3 at rest, 3–4 during movement), and cumulative 24-hour opioid use was zero intravenous morphine (< 5 MME). Venous

thromboembolism prophylaxis began 12 hours after hemostasis, with subcutaneous enoxaparin sodium 40 mg twice daily adjusted for super-obesity and sequential compression boots. It was prescribed for six weeks after discharge. Repeat tests on POD 1 and POD 5 confirmed hematological recovery (hemoglobin 10.9 g/dL, platelets 168,000 /μL, fibrinogen 395 mg/dL, lactate 0.9 mmol/L) and normal hepatic transaminases (AST 28 U/L, ALT 31 U/L). The Pfannenstiel incision healed primarily under negative pressure wound therapy, with no dehiscence or surgical site infection. The neonate was managed in the neonatal high-care unit with non-invasive CPAP and nutritional support. He recovered, weaned to ambient air, was feeding vigorously by day 3 and was discharged healthy on POD 10. The mother went home in good functional condition on POD 5. She was grateful to have stayed awake, free of pain and involved in the birth of her child.

Table 6. Postoperative recovery profile, high-dependency unit surveillance, pain trajectory and maternal and neonatal outcomes.

Clinical surveillance domain	HDU (POD 0, 0–24 h)	Inpatient care (POD 1–2)	Inpatient care (POD 3–5)	Extended follow-up (POD 10 / 6 weeks)	Clinical benchmark or target
Ventilatory & Airway Stability	Spontaneous ventilation; SpO ₂ 97–99% on room air; zero desaturation (< 92%) episodes; continuous pulse oximetry; semi-upright 45° positioning	Spontaneous ventilation; SpO ₂ 98% ambient; clear breath sounds bilaterally	Unrestricted room-air ambulation; zero dyspnea or wheezing	Complete respiratory recovery; zero delayed airway complications	Zero respiratory depression; effective OSA defense without airway collapse
Hemodynamic Trajectory & SVR	MAP 86–92 mmHg; HR 76–84 bpm; normotensive without inotropic or vasopressor support	BP 130/80 mmHg; MAP 96.7 mmHg; stable on oral nifedipine 30 mg daily	BP 124/76 mmHg; anti-hypertensive therapy successfully tapered	Normotensive at 6 weeks (120/78 mmHg); complete preeclampsia resolution	Hemodynamic homeostasis; prevention of hypertensive crisis or rebound shock
Multimodal Pain Severity (NRS Score)	NRS 2–3 at rest, 3–4 during movement; high comfort	NRS 1–2 at rest, 2–3 during movement	NRS 0–1 at rest, 1–2 during ambulation	NRS 0; fully pain-free	Effective pain control (NRS < 4) enabling early maternal mobilization
Opioid Consumption & Analgesic Regimen	IV Paracetamol 1 g q6h + IV Ketorolac 30 mg q8h; cumulative opioid: < 5 MME (0 mg IV morphine)	Oral Paracetamol 1 g q8h + Ibuprofen 400 mg q8h; 0 MME	Oral Paracetamol as needed; 0 MME	Zero opioid requirement; zero dependence	Opioid-sparing multimodal analgesia (< 15 MME threshold in 24 h)
Venous Thromboembolism Prophylaxis	Enoxaparin sodium 40 mg SC initiated 12 h post-hemostasis; intermittent pneumatic compression boots	Enoxaparin 40 mg SC q12h weight-adjusted regimen	Enoxaparin 40 mg SC q12h continued	Extended enoxaparin 40 mg SC once daily completed for 6 weeks post-discharge	Strict adherence to ASH/RCOG guidelines for postpartum super-obesity thromboprophylaxis
Surgical Wound Healing & SSI	Pfannenstiel incision dry, intact; negative-pressure wound dressing applied; zero drainage	Clean surgical dressing; no erythema, induration, or seroma	Negative pressure dressing removed; clean primary intention healing; discharge on POD 5	Complete wound scar maturation; zero surgical site infection (SSI)	Prevention of superficial and deep incisional SSI in massive pannus
Neonatal Critical Care Outcomes	Male neonate, 4,025 g (macrosomic); APGAR 4 (1 min), 5 (5 min), 7 (10 min); transfer to Neonatal HCU for CPAP and warming	Successful CPAP weaning to ambient air; active suckling reflex	Full enteral breastfeeding established; transferred to mother	Full clinical recovery; discharged healthy on POD 10; normal neurodevelopment	Reversal of intrapartum birth asphyxia; intact neonatal neurological status

Notes: ADR, adverse drug reaction; APGAR, appearance, pulse, grimace, activity, respiration; CPAP, continuous positive airway pressure; HCU, high care unit; HDU, high-dependency unit; MAP, mean arterial pressure; MME, morphine milligram equivalents; NRS, numeric rating scale (0–10); OSA, obstructive sleep apnea; POD, postoperative day; SC, subcutaneous; SSI, surgical site infection; SpO₂, peripheral oxygen saturation; SVR, systemic vascular resistance.

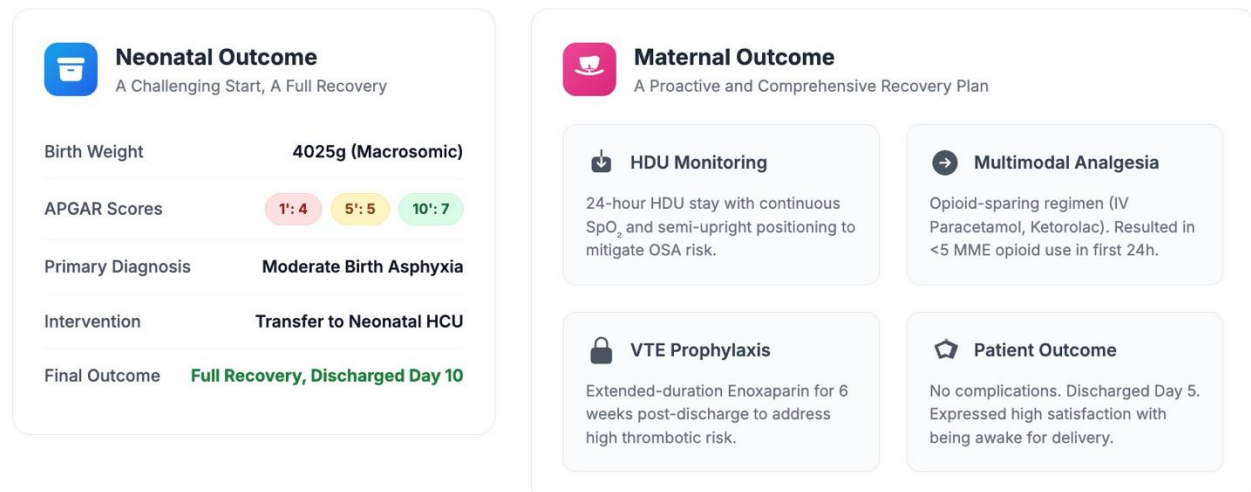


Figure 3. Neonatal and postoperative outcomes. Left panel: neonatal course with birth weight of 4,025 g, Apgar scores of 4, 5 and 7 at 1, 5 and 10 minutes, transfer to the neonatal high-care unit (HCU) for non-invasive respiratory support, and discharge on day 10. Right panel: maternal recovery plan with 24-hour high-dependency unit (HDU) monitoring, multimodal opioid-sparing analgesia (< 5 MME), extended enoxaparin thromboprophylaxis for 6 weeks and discharge on day 5. Abbreviations: MME, morphine milligram equivalents; OSA, obstructive sleep apnea; VTE, venous thromboembolism.

4. Discussion

4.1. Principal lesson of the case

In this super-obese parturient with preeclampsia and fetal hypoxia, a planned transition from a regressing single-shot spinal anesthetic to a dexmedetomidine-ketamine infusion was followed by spontaneous ventilation, without airway instrumentation, through a four-hour operation and a 2,000-mL hemorrhage. The case suggests that exhaustion of a neuraxial block need not be handled as an unforeseen emergency requiring general anesthesia when a contingency plan has been prepared beforehand. It cannot show that the plan caused the outcome, because spinal block, uterotonics, volume replacement, blood products and norepinephrine acted together.

4.2. Comparison with the published literature

Published reports of cesarean delivery in super-obese parturients mostly describe single-shot or catheter-based neuraxial techniques. A single-shot spinal gave surgical conditions in a woman with a BMI of 61.3 kg/m² (hyperbaric bupivacaine 9 mg with fentanyl 20 mcg; block to T8; stable vital signs)⁴¹ and in another parturient with a BMI above 60 kg/m².⁴² Both operations were elective and uncomplicated, whereas our patient needed emergency surgery that outlasted the block. In two cesarean deliveries at BMI 102 and 116 kg/m², incremental dosing through an intrathecal catheter (cumulative hyperbaric bupivacaine 12 mg) avoided a high block and can extend a block as required.⁴³ A catheter technique or a combined spinal-epidural was not used in our patient.

Standard spinal doses carry a documented risk in this population. In 5,015 cesarean deliveries under spinal or combined spinal-epidural anesthesia with hyperbaric bupivacaine of at least 10.5 mg, a high block occurred in 0.6% and was more likely at a BMI of 50 kg/m² or more (odds ratio 6.3; 95% confidence interval 2.2–18.5).⁴⁴ Our patient received 10 mg with fentanyl, reached T4 and had no respiratory compromise. In a retrospective series of 42 cesarean deliveries in parturients with a BMI above 50 kg/m², hypotension occurred in 95.2% and vasopressors were needed in 78.6%; postpartum hemorrhage occurred in 11.9% and body weight correlated with blood loss ($r =$

0.588).⁴⁵ The fall in MAP and the hemorrhage in our patient are consistent with that report.

The closest published parallel is a woman with a BMI of 50 kg/m² and severe sleep apnea who had a combined spinal-epidural anesthetic. Despite a T4 block, prolonged surgery caused upper abdominal pain that needed propofol sedation, which led to respiratory depression managed by adjusting bilevel positive airway pressure.⁴⁶ Our patient had the same problem, a block that did not last for the whole operation, but received dexmedetomidine and ketamine, and no respiratory depression occurred. A report of emergency cesarean delivery in morbid obesity with severe preeclampsia and pulmonary edema underlines the combined risk described here.⁴⁷ The combination of ketamine and dexmedetomidine in cesarean anesthesia has been reviewed narratively, with possible synergy, reduced opioid requirements and enhanced hemodynamic stability, but without pooled data or a formal assessment of bias.⁴⁸ In our search of Crossref and Europe PMC we found no published report of a planned spinal-to-dexmedetomidine-ketamine sequence in a super-obese parturient apart from the earlier brief report of this patient.²⁸ This case adds a documented, planned bridge from a regressed spinal block to an infusion with preserved spontaneous ventilation through a major hemorrhage, supported by a five-epoch hemodynamic, fluid and laboratory record.

4.3. Pathophysiological or mechanistic interpretation

The danger in this case came from three conditions acting together: super-obesity, pregnancy and preeclampsia (Figure 4 sets out the risk framework).^{5,6} Super-obesity restructures respiratory mechanics. Fat across the chest wall and abdomen adds restrictive impedance. With the gravid uterus pushing the diaphragm upward, FRC falls substantially.^{7,8} In our patient, closing volume presumably exceeded FRC during supine tidal breathing, which would sustain dependent micro-atelectasis and intrapulmonary right-to-left shunting; this was inferred and was not measured.^{9,10} Maternal oxygen consumption rises in pregnancy and with obesity, and the safe apnea time is short (about 60 to 90 seconds).^{8,11} Even a brief apnea during emergency airway instrumentation could then cause severe desaturation, hypoxic-ischemic brain injury or cardiac arrest.^{49,50}

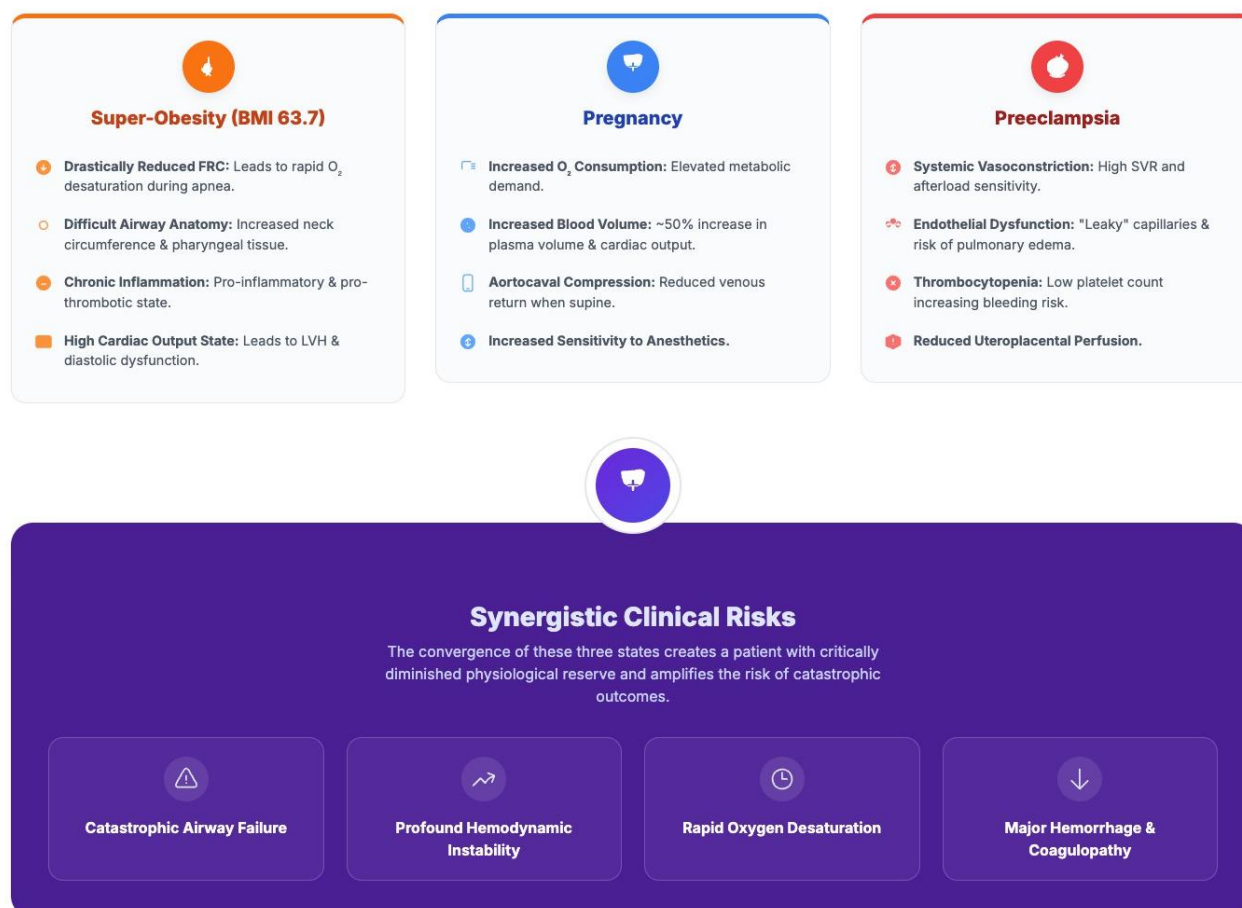


Figure 4. The pathophysiological triad and synergistic risks. Super-obesity (reduced FRC, difficult airway anatomy, chronic inflammation, high cardiac output), pregnancy (increased oxygen consumption, plasma volume expansion of about 50%, aortocaval compression) and preeclampsia (systemic vasoconstriction, endothelial capillary leak, thrombocytopenia, reduced uteroplacental perfusion) combine to give airway failure, hemodynamic instability, rapid oxygen desaturation and major hemorrhage with coagulopathy. This is an interpretative schematic, not patient data. Abbreviations: FRC, functional residual capacity; LVH, left ventricular hypertrophy; SVR, systemic vascular resistance.

The cardiovascular system was also under heavy stress.^{5,12} Extreme adiposity produces a high-output circulation with expanded blood volume and eccentric left ventricular remodeling, which can leave subclinical diastolic dysfunction. Preeclampsia adds widespread endothelial dysfunction, arteriolar vasospasm and a large afterload rise, so the stiff, hypertrophied ventricle must pump against high systemic vascular resistance.^{13,14} The heart becomes very sensitive to changes in volume and vasomotor tone: sudden venodilation or hypovolemia cuts stroke volume sharply, while uncalibrated fluid resuscitation can cause hydrostatic pulmonary edema across hyperpermeable capillaries.^{13,14} Adipose tissue is also an active endocrine organ. Its pro-inflammatory adipokines add to preeclamptic endothelial injury and may promote microvascular thrombosis, consumptive thrombocytopenia (baseline platelets 120,000 / μ L) and hyperfibrinolysis, which raise the risk of severe postpartum hemorrhage.^{1,51}

The airway is the greatest single hazard in such circumstances. Our patient had several anatomical predictors of difficult airway management: Mallampati Class III, a neck circumference above 50 cm, restricted cervical mobility, an inter-incisor opening of 3.0 cm and a STOP-BANG score of 5. Difficult or impossible direct laryngoscopy and mask ventilation were highly probable.^{15,16} National audit data show failed tracheal intubation in roughly 1 in 390 obstetric anesthetics. The risk rises more than ten-fold with morbid obesity and

emergency indications.^{16,17} Had general anesthesia been induced urgently at the two-hour mark, when the spinal regressed, she would have faced a real danger of a cannot intubate, cannot oxygenate arrest, gastric acid aspiration and severe hypoxia during rapid-sequence induction.^{11,17} By avoiding instrumentation and moving to sequential TIVA, we kept her native pharyngeal tone, laryngeal protective reflexes and spontaneous medullary ventilatory drive intact for the whole procedure. No airway morbidity occurred.

The pharmacodynamic properties of dexmedetomidine and ketamine are a plausible explanation for the course, although they were not tested here. The two drugs act through different receptor mechanisms that offset each other (Figure 5 sets these out as a five-tier cascade). Dexmedetomidine is a highly selective α -2A adrenergic agonist (α -2: α -1 selectivity ratio 1620:1) acting mainly on presynaptic α -2 receptors in the pontine locus coeruleus.²¹ Agonism of these G-protein-coupled receptors inhibits adenylate cyclase and hyperpolarizes noradrenergic neurons through inwardly rectifying potassium channels. Sympathetic outflow falls. The result is a sleep-like, cooperative state (non-rapid eye movement type) from which the patient wakes easily to a spoken command.^{21,22} Dexmedetomidine depresses medullary hypercapnic and hypoxic ventilatory drive only negligibly. Upper airway patency is therefore relatively preserved, including in patients with obstructive sleep apnea or obesity.^{52,53} Racemic ketamine, in contrast, gives strong

somatic and visceral dissociative analgesia. It non-competitively blocks the phencyclidine binding site of the NMDA receptor in the dorsal horn substantia gelatinosa

and thalamocortical relay nuclei. Nociceptive transmission and central sensitization are inhibited.²³

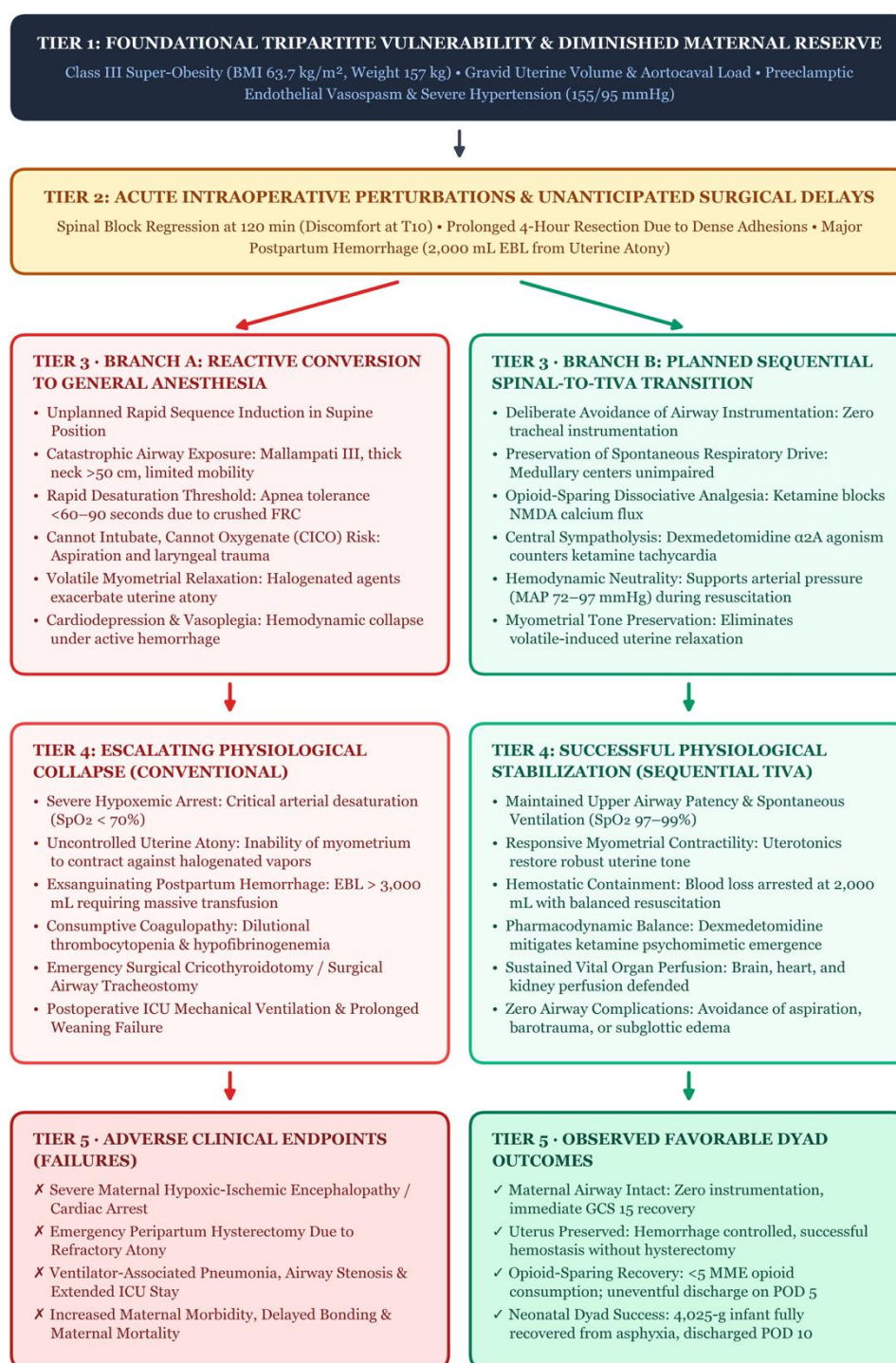


Figure 5. Five-tier cascade comparing reactive conversion to general anesthesia (Branch A) with the planned sequential spinal-to-TIVA pathway (Branch B) in a super-obese parturient with preeclampsia. Tier 1, foundational vulnerability; tier 2, intraoperative perturbations; tier 3, anesthetic branches; tier 4, physiological course; tier 5, clinical endpoints. Abbreviations: CICO, cannot intubate, cannot oxygenate; EBL, estimated blood loss; FRC, functional residual capacity; MAP, mean arterial pressure; NMDA, N-methyl-D-aspartate; POD, postoperative day.

The value of combining the two drugs lies in their opposing autonomic effects, which may cancel each other's side effects. Ketamine usually stimulates central sympathetic outflow. Heart rate, arterial pressure and myocardial oxygen demand rise. Distressing emergence delirium and hallucinations occasionally appear. Dexmedetomidine's central sympatholytic and anxiolytic actions blunt the

hypertension, tachycardia and emergence phenomena and favor a hemodynamically neutral state.^{24,25} In return, ketamine's mild indirect sympathomimetic tone may prevent the bradycardia and peripheral vasodilation that dexmedetomidine often causes. In this patient MAP ranged from 71.7 to 96.7 mmHg across the intraoperative epochs (Table 5) even during acute blood loss. Ketamine is also a

bronchial smooth muscle relaxant, acting through catecholamine reuptake inhibition and calcium channel blockade, and the patient had pre-existing asthma.^{54,55}

One possible advantage of a dexmedetomidine-ketamine regimen in high-risk obstetrics is that it spares myometrial contractility during postpartum hemorrhage. Halogenated volatile anesthetics (sevoflurane, isoflurane, desflurane) have concentration-dependent tocolytic effects. They inhibit myometrial calcium influx through L-type calcium channels and suppress calcium release from the intracellular sarcoplasmic reticulum. At alveolar concentrations above 0.5 minimum alveolar concentration, they relax the myometrium considerably, and the uterus may then resist exogenous uterotonics.^{20,56} Experimental studies suggest that dexmedetomidine does not impair and may increase myometrial contractility, whereas ketamine increased contractions of rat uterus at low concentrations and inhibited them at high concentrations.^{26,27,57} When acute atony caused an estimated blood loss of 2,000 mL, there was no volatile-induced relaxation to oppose the multimodal uterotonic cascade (oxytocin, methylergometrine, misoprostol, tranexamic acid). Firm tone returned quickly. Hemorrhage stopped within 50 minutes, and emergency peripartum hysterectomy was avoided. These are observations in one patient; the contribution of each drug and of the uterotonics cannot be separated.

Maternal-fetal pharmacology deserves a look at placental drug transfer and neonatal outcome. Both drugs are lipophilic. Both cross the placenta. Reported umbilical vein to maternal vein concentration ratios are about 0.76 for dexmedetomidine and 0.85 to 1.20 for ketamine.^{58,59} In our sequential protocol, TIVA began at operative minute 02:05, long after the neonate was delivered at minute 00:35 under spinal anesthesia alone, so the neonate was not exposed to dexmedetomidine or ketamine. The depression at birth (Apgar scores 4, 5 and 7 at 1, 5 and 10 minutes) was probably related to existing intrapartum hypoxia (Category III CTG tracing), placental vascular insufficiency from preeclampsia and a difficult, prolonged extraction through the massive pannus; cord blood gases were not reported. Rapid resuscitation by the neonatal high-care team reversed the asphyxia. The neonate recovered and went home on POD 10.

4.4. Implications for practice

This case illustrates the principles of high-reliability organizations and Crew Resource Management in handling high-acuity, low-frequency obstetric crises.⁶⁰ In Reason's Swiss Cheese model of accident causation, a catastrophe occurs when latent system weaknesses line up with active failures.^{61,62} Our patient had many latent hazards: super-obesity, difficult airway, preeclampsia, a spinal block of finite duration, dense adhesions and uterine atony. The multidisciplinary team anticipated the failure points. It built a shared mental model during preoperative consultation. The team also paused for a structured crisis huddle at the two-hour mark, as the spinal began to regress. Closed-loop communication among the lead anesthesiologist, the primary obstetrician and nursing staff let the TIVA protocol start smoothly, without hesitation or reactive airway instrumentation. Non-technical skills matter.

The postoperative course shows why continuous, protocolized surveillance in the HDU is needed. Super-obese parturients with obstructive sleep apnea are very vulnerable to opioid-induced respiratory depression and

upper airway collapse. The risk is highest in the first 24 to 48 postoperative hours, when sleep is fragmented and sedatives carry over.⁶³ Our multimodal protocol relied on scheduled non-opioid analgesics (intravenous paracetamol and ketorolac). Cumulative 24-hour opioid use was held to zero intravenous morphine (< 5 MME), which was associated with an absence of ventilatory depression, with NRS 2–3 and early mobilization.^{64,65} Super-obese postpartum women also face a steeply increased risk of venous thromboembolism, including fatal pulmonary embolism.⁶⁶ Weight-adjusted enoxaparin has been studied after cesarean delivery in morbidly obese women, and the extended course in this patient covered the most thrombogenic stretch of her recovery. A clinician facing a similar emergency may consider preparing and rehearsing a spinal-to-infusion contingency, with the airway equipment ready and a planned hemorrhage protocol; how transferable this is beyond a team with similar resources is not known.

4.5. Strengths

The record is unusually complete for a case of this kind: measured airway and anthropometric data, a five-epoch hemodynamic, fluid and laboratory series with percentage changes, documented drug doses and times, a neonatal outcome and follow-up to 6 weeks, together with written consent and anonymized figures.

4.6. Limitations

This is a single patient, without a comparator, and cannot establish causation, prevalence or effect. Spontaneous ventilation was maintained while the spinal block had regressed only to T10, so the infusion supplemented a partial block and cannot be credited alone. Several interventions acted at the same time, which confounds any attribution. Airway risk was inferred from predictors, not measured, and the counterfactual (general anesthesia) was not tried. The observation window for the mother ended at 6 weeks and for the neonate at day 10, which is too short to judge neurodevelopment.

4.7. Recommendations for future work

Whether a planned spinal-to-infusion pathway improves outcomes in super-obese parturients would need a prospective registry or comparative series that records airway events, hemodynamics, blood loss and neonatal outcomes, ideally with a predefined protocol and a comparison with combined spinal-epidural or conversion to general anesthesia.

5. Conclusion

In this super-obese parturient with preeclampsia, a planned transition from single-shot spinal anesthesia to a dexmedetomidine-ketamine infusion was followed by preserved spontaneous ventilation and stable recovery through a four-hour operation and a 2,000-mL postpartum hemorrhage, without airway instrumentation. The case supports preparing a spinal-to-infusion contingency for prolonged cesarean delivery in extreme obesity, but one patient cannot show that the plan caused the outcome, and the approach needs evaluation in larger series.

6. Declarations

Ethical approval and consent to participate. This case report was approved by the CMHC Research Center Ethics Committee, Indonesia, under reference number

2024/7183. The work followed the Declaration of Helsinki. Written informed consent was obtained from the patient and her legal surrogate.

Consent for publication. Written informed consent for publication of the clinical details, longitudinal physiological data and anonymized diagnostic materials of this case was provided by the patient. All identifiable features have been removed or permanently obscured.

Availability of data and materials. The datasets generated and analyzed during this report are available from the corresponding author on reasonable request, subject to institutional ethical oversight and patient confidentiality.

Artificial intelligence (AI) use statement. The authors declare that no generative artificial intelligence tool was used in the preparation of this manuscript.

Author contribution statement. A.N. managed the patient, conceived the report, collected the clinical data and drafted the manuscript (conceptualization, investigation, data curation, writing – original draft). A.T.A. provided clinical oversight, co-managed intraoperative hemodynamics and airway strategy and revised the manuscript (supervision, methodology, writing – review and editing). P.P.H. supervised the multidisciplinary obstetric consultation, analyzed the longitudinal data and finalized the manuscript (formal analysis, validation, project administration, writing – review and editing). All authors approved the final manuscript.

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Conflict of interest (competing interests) statement. The authors declare that they have no competing interests.

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