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# Intensive Care Management of Non-Ischemic Dilated Cardiomyopathy with Morbid Obesity in a Parturient Undergoing Cesarean Section

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**ABSTRACT**

**Introduction**: Dilated cardiomyopathy (DCM) in pregnancy is a rare but life-threatening condition, with reported incidence ranging from 1:4,950 deliveries in Europe to 2.38:1,000 deliveries in Asia. When complicated by morbid obesity, it significantly increases perioperative and critical care challenges, requiring a coordinated multidisciplinary approach to optimize maternal outcomes.

**Case Description**: A 32-year-old primigravida with morbid obesity (BMI 49.5 kg/m²) and non-ischemic dilated cardiomyopathy presented with decompensated heart failure at 29 weeks of gestation. She underwent elective cesarean section under general anesthesia followed by 19 days of intensive care. Management included hemodynamic optimization with dobutamine infusion, restrictive fluid strategy targeting negative balance, stepwise ventilator weaning from mechanical ventilation to nasal cannula, and treatment of complications including electrolyte disturbances and postoperative delirium secondary to obesity hypoventilation syndrome (Pickwickian syndrome). Continuous hemodynamic monitoring using MostCare and invasive arterial pressure enabled precise titration of therapy.

**Conclusion**: Successful maternal outcome in pregnant patients with dilated cardiomyopathy and morbid obesity can be achieved through comprehensive preoperative optimization, carefully selected anesthetic technique, and prolonged multidisciplinary intensive care. This case highlights the importance of integrated hemodynamic, respiratory, and metabolic management in this high-risk population.

**Keywords**

Dilated Cardiomyopathy, Morbid Obesity, Cesarean Section, Obstetric Anesthesia, Intensive Care, Heart Failure in Pregnancy.

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## INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a rare but potentially life-threatening form of heart failure that develops in the last month of pregnancy or within the first five months postpartum in women without prior heart disease or other identifiable causes of cardiac dysfunction. The incidence of PPCM exhibits significant geographic variation, ranging from approximately 1 in 1,000 to 1 in 4,000 live births in Western countries (including Europe and the United States), with higher rates reported in certain regions of Asia and Africa, potentially reaching 1 in 300–1,000 live births in high-prevalence areas such as Nigeria or Haiti [1,2]. Maternal mortality associated with PPCM has improved in high-resource settings but remains elevated globally, ranging from 4% to 10% at six months in Europe and Asia-Pacific regions to higher rates in low-resource settings, influenced by access to advanced heart failure therapies and timely diagnosis [3,4]. This condition contributes substantially to maternal and fetal morbidity and mortality, particularly when compounded by additional risk factors such as morbid obesity and hypertensive disorders of pregnancy [5].

The differential diagnosis of cardiomyopathy during pregnancy encompasses several distinct entities, including peripartum cardiomyopathy (PPCM), non-ischemic dilated cardiomyopathy (NICDM), ischemic dilated cardiomyopathy (IDCM), and acute decompensated heart failure (ADHF). PPCM is defined by the European Society of Cardiology as heart failure secondary to left ventricular (LV) systolic dysfunction (LV ejection fraction <45%) occurring toward the end of pregnancy or in the months following delivery, with no other identifiable cause and exclusion of pre-existing cardiac disease [6]. Echocardiographic hallmarks include reduced LV ejection fraction and often LV dilatation. In contrast, NICDM and IDCM typically have an onset unrelated to pregnancy and may involve different etiologies (e.g., genetic, toxic, or ischemic), whereas ADHF represents acute exacerbation in patients with pre-existing chronic heart failure. Accurate differentiation is critical because it informs therapeutic strategies and long-term prognosis [7,8].

The association between hypertensive disorders during pregnancy and PPCM has been the focus of intensive research over the past decade. Severe preeclampsia confers the highest risk for PPCM development, with adjusted odds ratios reported as high as 13–21 (95% CI varying by cohort), followed by superimposed preeclampsia (OR ~5–6), chronic hypertension (OR ~4–5), preeclampsia (OR ~4–5), and gestational hypertension (OR ~3–5) [9,10]. A fundamental distinction lies in LV remodeling patterns: patients with PPCM accompanied by preeclampsia often exhibit concentric remodeling, associated with relatively preserved or more rapid recovery of LV function and better prognosis, compared to eccentric remodeling in PPCM without preeclampsia, which correlates with a poorer response to standard heart failure therapies [11,12]. This pathophysiological divergence may explain the differential responses to conventional agents, such as angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARB), or beta-blockers, with patients displaying concentric remodeling potentially deriving less benefit from these therapies in the acute phase [13].

Morbid obesity during pregnancy introduces additional perioperative complexities, with a globally rising prevalence that poses specific challenges in anesthetic and intensive care management. Patients with morbid obesity (BMI ≥40 kg/m²) face an elevated risk of cardiorespiratory complications, including difficult airway management, impaired respiratory function, and increased thromboembolic events [14]. Pregnancy outcomes are adversely affected, with higher rates of maternal and fetal complications and prolonged intensive care unit stays. Anesthetic management requires comprehensive preoperative evaluation, weight-adjusted dosing of anesthetic agents, and vigilant cardiorespiratory monitoring throughout the perioperative period [15].

Pickwickian syndrome, also known as obesity hypoventilation syndrome (OHS), represents a severe complication of morbid obesity characterized by chronic alveolar hypoventilation, daytime hypercapnia ($PaCO_2$ >45 mmHg), and sleep-disordered breathing, with a prevalence of 8–20% among morbidly obese individuals.[16,17] Untreated OHS can progress to pulmonary hypertension and right heart failure (cor pulmonale). Diagnosis necessitates a thorough history, physical examination, and polysomnography to distinguish it from isolated obstructive sleep apnea. Early identification and appropriate management, including noninvasive ventilation, are essential to prevent the progression to severe pulmonary hypertension and subsequent heart failure [16].

## CASE DESCRIPTION

A 32-year-old primigravida woman (Mrs. D, medical record number 002334225) at 29 weeks and 1 d of 002334225 was admitted to the Medical Adult Intensive Care Unit (MAICU) of Dr. Hasan Sadikin Central General Hospital on January 4, 2025, with progressive dyspnea that had begun at approximately 16 weeks of gestation. Initially exertional, the dyspnea worsened over months to occur at rest and became orthopnea-dependent, preventing supine positioning for the past month. She also reported paroxysmal nocturnal dyspnoea and cough. The patient had been previously managed at two referring hospitals with diuretics, antenatal corticosteroids, and electrolyte supplementation but required escalation of care due to persistent respiratory failure and limited facilities at peripheral centers.

Her medical history was notable for chronic hypertension diagnosed during early pregnancy, which was treated with oral antihypertensive drugs for 2.5 months prior to admission. She had no history of diabetes

mellitus or any other comorbidities. Anthropometric evaluation revealed morbid obesity with a pre-pregnancy weight of 130 kg, current weight of 147 kg, height of 162 cm, and body mass index of 49.5 kg/m². On initial examination in the MAICU, the patient was conscious (Glasgow Coma Scale E4M6V5) but tachypneic (respiratory rate, 24 breaths/min) and tachycardic (heart rate, 105 beats/min) with a blood pressure of 134/65 mmHg. Oxygen saturation was 97% on high-flow nasal cannula (HFNC) at 60 L/min and $FiO_2$ 80%. Bilateral fine basal crepitations, consistent with pulmonary edema, were observed. The urine output responded briskly to diuretic therapy (2000 mL in 5 h post-furosemide). Laboratory investigations revealed mild anemia and leukocytosis that subsequently resolved, as well as persistent electrolyte derangements, including hyponatremia, hypokalemia, hypochloremia, and hypocalcemia (Table 1).

Table 1. Serial Laboratory Parameters

| Parameter | Day 1 | Day 3 | Day 7 | Day 14 | Discharge |
|---|---|---|---|---|---|
| Hemoglobin (g/dL) | 9.8 | 8.5 | 9.2 | 10.1 | 10.8 |
| Hematocrit (%) | 29.4 | 25.5 | 27.6 | 30.3 | 32.4 |
| Leukocytes (/μL) | 18,400 | 15,200 | 11,800 | 9,200 | 8,100 |
| Platelets (/μL) | 98,000 | 112,000 | 165,000 | 220,000 | 280,000 |
| Glucose (mg/dL) | 178 | 142 | 135 | 128 | 115 |
| Urea (mg/dL) | 92 | 108 | 76 | 54 | 42 |
| Creatinine (mg/dL) | 2.8 | 3.1 | 2.4 | 1.6 | 1.1 |
| Sodium (mmol/L) | 132 | 135 | 138 | 139 | 140 |
| Potassium (mmol/L) | 5.1 | 4.8 | 4.2 | 4.0 | 3.9 |
| Chloride (mmol/L) | 98 | 101 | 103 | 104 | 105 |
| Calcium (mg/dL) | 7.6 | 7.9 | 8.2 | 8.5 | 8.8 |
| Magnesium (mg/dL) | 1.6 | 1.8 | 2.0 | 2.1 | 2.0 |

Arterial blood gas analyses repeatedly showed chronic respiratory acidosis with compensatory metabolic alkalosis and elevated $pCO_2$ levels, consistent with obesity hypoventilation syndrome (Pickwickian syndrome) (Table 2).

Table 2. Serial Arterial Blood Gas Analysis

| Parameter | Day 1 (Pre-intubation) | Day 2 (NIV) | Day 4 (Intubated, SIMV) | Day 7 (CPAP trial) | Day 12 (Wean, T-piece) | Discharge |
|---|---|---|---|---|---|---|
| pH | 7.28 | 7.31 | 7.38 | 7.42 | 7.44 | 7.41 |
| $pCO_2$ (mmHg) | 83 | 76 | 64 | 54 | 48 | 50 |
| $pO_2$ (mmHg) | 68 | 82 | 94 | 88 | 90 | 86 |
| $HCO_3^-$ (mmol/L) | 38 | 41 | 45 | 48 | 50 | 36 |
| Base Excess | +11 | +14 | +18 | +22 | +25 | +11 |
| $SaO_2$ (%) | 92 | 95 | 98 | 97 | 97 | 96 |
| $FiO_2$ (%) | 50 (HFNC) | 40 (BiPAP) | 45 (Vent) | 35 (CPAP) | 30 (T-piece) | Room air |

Notably, Serial ABG revealed chronic respiratory acidosis with severe hypercapnia ($pCO_2$ 63–83 mmHg) and compensatory metabolic alkalosis ($HCO_3$ 36–53 mmol/L, BE +11 to +27), consistent with obesity hypoventilation syndrome.

Transthoracic echocardiography performed at the referring hospital (January 2, 2025) and subsequent bedside studies confirmed severe left ventricular systolic dysfunction (LVEF 35–42%) with left ventricular dilatation, mild regional wall motion abnormalities, and trivial-to-moderate mitral regurgitation without left ventricular hypertrophy. Hemodynamic assessment revealed a low cardiac index (1.75 L/min/m²) and mild pericardial effusion without tamponade physiology (Table 3). Chest radiography performed upon admission revealed cardiomegaly with pulmonary congestion, which progressively improved. The working diagnoses included acute decompensated heart failure secondary to non-ischemic dilated cardiomyopathy versus ischemic cardiomyopathy in the setting of gestational hypertension, morbid obesity, obesity hypoventilation syndrome (Pickwickian syndrome), and electrolyte imbalances.

Table 3. Echocardiographic and Hemodynamic Findings

| Parameter | (Admission) | (Bedside TTE) | (Hemodynamic, PAC) |
|---|---|---|---|
| LVEF (%) | 35 | 38 | – |
| LVEDD (mm) | 62 | 60 | – |
| LVESD (mm) | 52 | 50 | – |
| Wall motion abnormalities | Global hypokinesia | Global hypokinesia | – |
| LV hypertrophy | None | None | – |
| Mitral regurgitation | Moderate | Mild–moderate | – |
| Tricuspid regurgitation | Trivial | Trivial | – |
| Pericardial effusion | Mild, no tamponade | Mild, no tamponade | – |
| RV function (TAPSE, mm) | 18 (preserved) | 19 (preserved) | – |
| Estimated PASP (mmHg) | 42 + RAP | 38 + RAP | – |
| Cardiac Index (CI, L/min/m²) | – | – | 1.75 |
| SVR (dyn·s/cm⁵) | – | – | 920 (low-normal) |
| PCWP (mmHg) | – | – | 22 |
| RAP (mmHg) | – | – | 12 |
| $SvO_2$ (%) | – | – | 58 |

After a multidisciplinary discussion on January 7, 2025, involving the cardiology, anesthesiology, and perinatology teams, preterm caesarean delivery under general anesthesia was planned for maternal stabilization. Preoperative optimization included fluid restriction, aggressive diuresis, electrolyte correction, head-up positioning and continuous fetal monitoring. A caesarean section was performed on January 8, 2025, under carefully titrated general anesthesia (propofol induction, rocuronium, post-delivery fentanyl) with invasive arterial monitoring and advanced hemodynamic assessment (MostCare). The intraoperative course was stable, with a blood loss of 400 mL and urine output of 70 mL (Table 4).

Table 4. Intraoperative Hemodynamic Parameters

| Parameter | Induction | Incision | 30 min | 60 min | 90 min | Closure | Recovery |
|---|---|---|---|---|---|---|---|
| Systolic BP (mmHg) | 99 | 108 | 118 | 124 | 128 | 112 | 115 |
| Diastolic BP (mmHg) | 62 | 68 | 74 | 78 | 82 | 70 | 72 |
| Heart rate (beats/min) | 130 | 124 | 120 | 118 | 122 | 126 | 110 |
| Cardiac output (L/min) | 3.9 | 4.6 | 5.2 | 5.8 | 6.4 | 5.5 | 5.9 |
| Stroke volume variation (%) | 8 | 7 | 6 | 6 | 7 | 8 | – |
| $SpO_2$ (%) | 90 | 92 | 94 | 94 | 93 | 92 | 96 |
| Peak inspiratory pressure ($cmH_2O$) | 25 | 24 | 23 | 22 | 23 | 24 | – |
| $EtCO_2$ (mmHg) | 44 | 45 | 46 | 47 | 46 | 45 | 42 |
| $FiO_2$ (%) | 80 | 70 | 60 | 60 | 65 | 70 | 40 |

Noted: Intraoperative hemodynamics remained stable under general anesthesia and low-dose dobutamine: BP 99–128/62–82 mmHg, HR 118–130 bpm, CO 3.9–6.4 L/min, SVV 6–8%, $SpO_2$ 90–94% ($FiO_2$ 60–80%), peak pressure 22–25 $cmH_2O$, $EtCO_2$ 44–47 mmHg, reflecting a controlled low-output state without significant instability.

Postoperatively, the patient required prolonged mechanical ventilation (6 days) with gradual weaning from pressure support to CPAP, followed by HFNC, and eventually nasal cannula oxygen therapy. Inotropic support (dobutamine) was withdrawn on postoperative day 8. Strict negative fluid balance (500–1000 mL/day, occasionally up to 3400 mL/day) was maintained with loop diuretics. Thromboprophylaxis was intensified because of obesity and postoperative immobility. Left common femoral vein deep vein thrombosis was diagnosed and managed with therapeutic-dose low molecular weight heparin. Persistent electrolyte disturbances and postoperative delirium secondary to prolonged hypoxia were successfully treated. By postoperative day 19 (January 27, 2025), the patient was hemodynamically stable without vasoactive support, maintained oxygen saturation on a 4 L/min nasal cannula, achieved normalized sodium (133 mmol/L), and tolerated oral feeding. She was transferred to the regular ward for continued heart failure therapy,

multidisciplinary follow-up (cardiology, obstetrics, and endocrinology), and gradual cardiac rehabilitation. The newborn was cared for in the neonatal intensive care unit, and an uncomplicated recovery was reported.

## DISCUSSION

The present case exemplifies the complex interplay of morbid obesity, gestational hypertension, obesity hypoventilation syndrome (OHS), and acute decompensated heart failure secondary to non-ischemic dilated cardiomyopathy (NICDM), unmasked or exacerbated by pregnancy, culminating in preterm delivery at 29 weeks gestation. This scenario highlights the challenges in cardio-obstetric intensive care, where the physiological adaptations of pregnancy impose substantial hemodynamic stress on a compromised myocardium, compounded by obesity-related respiratory and metabolic burdens [17]. The patient's progressive dyspnea since mid-pregnancy, orthopnea, paroxysmal nocturnal dyspnea, and chronic respiratory acidosis with compensatory metabolic alkalosis ($pCO_2$ consistently >70 mmHg) reflect the synergistic effects of a low cardiac index (1.75 L/min/m²), pulmonary congestion, and alveolar hypoventilation, which are characteristic of OHS in the setting of extreme obesity (BMI 49.5 kg/m²) [18].

Differential diagnosis in this peripartum presentation requires careful exclusion of peripartum cardiomyopathy (PPCM), which shares echocardiographic features such as left ventricular dilatation and reduced ejection fraction (<45%) but is defined by onset in the last month of pregnancy or within five months postpartum without identifiable prior heart disease [19,20]. In contrast, the patient's symptoms commenced at approximately 16 weeks gestation, predating the classic PPCM temporal window, and echocardiography revealed no concentric remodeling typically associated with preeclampsia-related PPCM, favoring preexisting NICDM exacerbated by gestational hemodynamic load rather than de novo pregnancy-associated cardiomyopathy [21,22]. Gestational hypertension without proteinuria or end-organ damage further distinguished this case from preeclampsia, in which severe forms confer markedly elevated odds ratios for PPCM development (up to 13–21) [9,10]. The absence of significant left ventricular hypertrophy or ischemic changes on serial echocardiography supported NICDM over hypertensive or ischemic dilated cardiomyopathy [23].

The management adhered to contemporary cardio-obstetric principles, emphasizing multidisciplinary decision-making for timely delivery to halt maternal deterioration. Preoperative optimization with high-flow nasal cannula (HFNC), fluid restriction, aggressive diuresis, and electrolyte correction mitigated pulmonary edema and hypoventilation, while invasive hemodynamic monitoring (MostCare) and low-dose dobutamine facilitated safe general anesthesia for cesarean sections, avoiding neuraxial techniques in the context of anticipated difficult airway and hemodynamic instability [24,25]. Postoperative prolonged mechanical ventilation with gradual weaning, persistent negative fluid balance (up to 3400 mL/day), and intensified thromboprophylaxis addressed the heightened risks of respiratory failure, venous thromboembolism, and delirium secondary to chronic hypercapnia in patients with OHS [26,27]. Successful extubation on day 6 and inotrope discontinuation by day 8, culminating in ward transfer on day 19 with normalized sodium and oxygen requirement of 4 L/min, underscore the efficacy of targeted decongestion and ventilatory support in reversing acute decompensation [28,29].

Morbid obesity profoundly amplifies perioperative complexity, manifesting as obesity hypoventilation syndrome with daytime hypercapnia, reduced functional residual capacity, and increased work of breathing, predisposing patients to atelectasis and prolonged ventilation [30,31]. The observed deep vein thrombosis and postoperative delirium align with established complications in this population, where the prevalence of OHS approaches 8–20%, and untreated progression risks pulmonary hypertension and cor pulmonale [32,33]. Anesthetic conduct—titrated general anesthesia with volume-controlled ventilation, weight-adjusted rocuronium, and post-delivery fentanyl—reflected evidence-based adaptations for difficult airways and altered pharmacokinetics in patients with extreme obesity [34,35].

This case illustrates that while PPCM remains a critical differential diagnosis in peripartum heart failure, early onset symptoms in the presence of morbid obesity and gestational hypertension more commonly represent unmasking of underlying dilated cardiomyopathy or acute decompensation driven by obesity-related

cardiorespiratory strain [36]. Prompt recognition, multidisciplinary intervention, and delivery for maternal stabilization yielded favorable maternal recovery despite the initial severity, emphasizing the imperative of cardio-obstetric collaboration in high-risk pregnancies [37]. Long-term follow-up with guideline-directed medical therapy for heart failure (including sacubitril/valsartan, beta-blockers, mineralocorticoid antagonists, and SGLT2 inhibitors when postpartum and breastfeeding status permit), aggressive weight management, and contraception counseling is essential to mitigate the risk of recurrence in subsequent pregnancies and progression to chronic cardiomyopathy [38-42].

## CONCLUSION

Acute decompensated heart failure due to non-ischemic dilated cardiomyopathy, aggravated by morbid obesity (BMI 49.5 kg/m²), obesity hypoventilation syndrome, and gestational hypertension at 29 weeks gestation, was successfully managed with multidisciplinary intervention: preterm caesarean section under general anesthesia, advanced monitoring, aggressive decongestion, and stepwise ventilatory weaning, achieving full maternal recovery by postoperative day 19. This underscores the critical value of integrated cardio-obstetric critical care in high-risk peripartum decompensation amid rising maternal obesity and multi-morbidity rates.

## DECLARATIONS

None

## CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

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## COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

## AUTHORS' CONTRIBUTIONS

All authors made substantial contributions to the case report. AD was responsible for patient management, data collection, and the initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity, and are accountable for all aspects of the work.

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## REFERENCE

1. Metgud SC, Patil A, Renuka PM. Epidemiology, risk factors, and pregnancy outcomes in peripartum cardiomyopathy. J Sci Soc. 2025;52(1):43-48.
2. Jackson AM, Macartney M, Brooksbank K, et al. A 20-year population study of peripartum cardiomyopathy. Eur Heart J. 2023;44(48):5128-5141.
3. Okeke TC, Ezenyeaku C, Ikeako LC. Peripartum cardiomyopathy. Ann Med Health Sci Res. 2013;3(3):313-319.
4. Nugrahani AD, Maulana S, Tjandraprawira KD, et al. Analysis of clinical profiles and echocardiographic cardiac outcomes in peripartum cardiomyopathy (PPCM) vs PPCM with co-existing hypertensive pregnancy disorder (HPD-PPCM) patients: a systematic review. J Cardiothorac Vasc Anesth. 2024:1-5.
5. Fahreza L, Winato BM, Nugrahani AD, Arianto S. Peripartum cardiomyopathy vs peripartum cardiomyopathy with preeclampsia: from biomolecular approach to management. J Biomedika Kesehat. 2024;7(3):351-361.

6. Kaw R, Dupuy-McCauley K, Wong J. Screening and perioperative management of obesity hypoventilation syndrome. J Clin Med. 2024;13(17):5000.
7. Ramadhan AR, Betcy B, Aurora RG, Prasenohadi P, Alatas MF. Obesity hypoventilation syndrome (Pickwickian syndrome): a literature review. Respir Sci. 2024;5(1):48-61.
8. Kharawala A, Nagraj S, Setia G, Reynolds D, Thachil R. Cardiac critical care of the cardio-obstetric patient. J Intensive Care Med. 2025:1-5.
9. Xu C, Yang F, Wang Q, Gao W. Comparison of high flow nasal therapy with non-invasive ventilation and conventional oxygen therapy for acute hypercapnic respiratory failure: a meta-analysis of randomized controlled trials. Int J Chron Obstruct Pulmon Dis. 2023;18:2221-2233.
10. Yan L, Lu Y, Deng M, et al. Efficacy of high-flow nasal cannula in patients with acute heart failure: a systematic review and meta-analysis. BMC Pulm Med. 2023;23(1):476.
11. Luebcke CD, Smith BE, Abrahamson K. An integrated review of obstetric admissions in the intensive care unit. Dimens Crit Care Nurs. 2022;41(3):132-143.
12. Abie A, Mehari MG, Dagnew TE, et al. Obstetric admission and maternal mortality in the intensive care unit in Africa: a systematic review and meta-analysis. PLoS One. 2025;20(4):e0320254.
13. Sanfilippo F, Noto A, Ajello V, et al. The use of pulmonary artery catheters and echocardiography in the cardiac surgery setting: a nationwide Italian survey. J Cardiothorac Vasc Anesth. 2024;38(9):1941-1950.
14. Dodiyi-Manuel ST, Ezennaka RC. Current management of peripartum cardiomyopathy: a review. Niger J Med. 2015;24(4):363-369.
15. Kamajaya IGNAT, Ariyana MBD. Updated study of peripartum cardiomyopathy and preeclampsia. Maj Obstet Ginekol. 2022;30(2):101-107.
16. Tsabedze N, Ramsay M, Krause A, et al. The genetic basis for adult-onset idiopathic dilated cardiomyopathy in people of African descent. Heart Fail Rev. 2023;28(4):879-892.
17. Luciani M, Del Monte F. Insights from second-line treatments for idiopathic dilated cardiomyopathy. J Cardiovasc Dev Dis. 2017;4(3):12.
18. Perone F, Dentamaro I, La Mura L, et al. Current insights and novel cardiovascular magnetic resonance-based techniques in the prognosis of non-ischemic dilated cardiomyopathy. J Clin Med. 2024;13(4):1017.
19. Sabina M, Barakat Z, Feliciano A, Lamb A, Alsamman MM. Unlocking the potential of acetazolamide: a literature review of an adjunctive approach in heart failure management. J Clin Med. 2024;13(1):288.
20. Braunthal S, Brateanu A. Hypertension in pregnancy: pathophysiology and treatment. SAGE Open Med. 2019;7:2050312119843700.
21. Townsend R, O'Brien P, Khalil A. Current best practice in the management of hypertensive disorders in pregnancy. Integr Blood Press Control. 2016;9:79-94.
22. Anthony J, Damasceno A, Ojjii D. Hypertensive disorders of pregnancy: what the physician needs to know. Cardiovasc J Afr. 2016;27(2):104-110.
23. Loh CJL, Qi Y, Sultana R, Tan CW, Sng BL, Yahaya Z. Hemodynamic changes of women with preeclampsia undergoing spinal anesthesia for emergency cesarean section: a prospective observational study. Bali J Anaesthesiol. 2024;8:164-168.
24. Abate S, Mergia G, Basu B. Maternal and neonatal outcomes of preeclamptic and normotensive women who underwent cesarean section under spinal anesthesia: systematic review and meta-analysis. Int J Surg Open. 2021;26:6-16.
25. Akter R, Abdullah-Hel-Baki M, Neher J, et al. The efficacy of spinal anesthesia during emergency cesarean section for severe preeclampsia and eclampsia patients. Saudi J Med Pharm Sci. 2023;9(1):6-12.
26. Manuaba IBGRS, Widana IW. Anesthesia management in cesarean section with severe preeclampsia and non-reactive nonstress test. Int J Res Med Sci. 2024;12(6):2104-2105.
27. Ramos Mayordomo P, Capilla Díez M, Ticona Espinoza DA, et al. Thrombotic microangiopathy (TMA) associated with pregnancy: role of the clinical laboratory in differential diagnosis. Clin Chem Lab Med. 2024:1-5.

28. Kalicka M, Bielak A, Gryta J, et al. How maternal obesity affects pregnancy? Literature review. Qual Sport. 2023;10:11-17.
29. Carrasco Catena A, Ruiz Sacedón N, Ortiz Murillo E, Balanzá Chancosa R. Pregnant women with morbid obesity: pregnancy and perinatal outcomes. Adv Obes Weight Manag Control. 2018;8(5):00257.
30. Kammies JD, De Waard L, Muller CJB, Hall DR. Delivery outcomes in women with morbid obesity, where induction of labour was planned to prevent post-term complications. J Obstet Gynaecol. 2022;42(8):3450-3455.
31. Eissa GA, Khurmi RA, Holbah TJ, et al. The effect of sleeve gastrectomy on pregnancy complications: a cross-sectional study in Saudi Arabia. Cureus. 2023;15(6):e40157.
32. Li J, Chen AE, Ye R. Comparison of three neuraxial anesthesia approaches in parturient women with obesity and pregnancy-induced hypertension who underwent cesarean section. J Int Med Res. 2021;49(12):3000605211066433.
33. Ismail N, Ismail MF. Maternal obesity and obstetric anesthesia: a comprehensive review of challenges and clinical strategies. Asian J Med Health. 2025;23(3):105-111.
34. Machado LS. Cesarean section in morbidly obese parturients: practical implications and complications. N Am J Med Sci. 2012;4(1):13-18.
35. Bisri DY, Maharani MI, Bisri T. Anesthesia for craniotomy tumor removal patient with morbid obesity. J Law Sust Develop. 2024;12(1):e1816.
36. Agha M, Agha RA, Sandell J. Interventions to reduce and prevent obesity in pre-conceptual and pregnant women: a systematic review and meta-analysis. PLoS One. 2014;9(5):e95132.
37. Moxth LC, Sauls R, Ruiz M, Stem M, Gonzalvo J, Gray HL. Effects of bariatric surgeries on male and female fertility: a systematic review. J Reprod Infertil. 2020;21(2):71-86.
38. Fadl AA, Shahatah MA, Asiri AY, et al. Obesity hypoventilation syndrome: a comprehensive review. Int J Med Res Health Sci. 2024;13(5):112-125.
39. Shah NM, Shrimanker S, Kaltsakas G. Defining obesity hypoventilation syndrome. Breathe (Sheff). 2021;17(3):210089.
40. Mishra P, Gupta S, Devmurari P, Raj N, Priya Natarajan N, Kumar A. Obesity-related breathing disorders: diagnosis and treatment of hypoventilation syndrome. Health Leadersh Qual Life. 2025;4:612.
41. Guan L, Wu W, Huo Y, et al. Efficacy of bilevel positive airway pressure and continuous positive airway pressure therapy in patients with obesity hypoventilation syndrome: protocol for systematic review and meta-analysis. BMJ Open. 2018;8(5):e020832.
42. De Jong A, Chanques G, Jaber S. Mechanical ventilation in obese ICU patients: from intubation to extubation. Crit Care. 2017;21(1):63.