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# When Status Asthmaticus Meets Severe Preeclampsia: A Critical Obstetric Scenario

**Sêtondji Emmanuel Raymond Ahouangansi[1*], Edwige Floriane Mouafo[2], Walamitien Cyrille Toure[3], Zolé Cédick Doh[4], Lossan Herman Kra[4], Aleke Koffi[4], Christian Danielle Chuekam[5], Kuate Christian Kadjé[5], Slim Chettaoui[5], Marc Kognombi[5], Takam Eleonore Sonia Fom[5], Giadjou Erika Vanessa Femtchou[5], Aude Djieumo[5], Patrick Ndjeundo[5]**

[1]Department of Anesthesiology and Critical Care, University Hospital of Angré, Abidjan, Côte d'Ivoire
[2]Department of Anesthesiology and Critical Care, Gynecology-Obstetrics and Pediatrics Pole, University Hospital of Cocody, Abidjan, Côte d'Ivoire
[3]Department of Anesthesiology and Critical Care, University Hospital of Yopougon, Abidjan, Côte d'Ivoire
[4]Department of Anesthesiology and Critical Care, Institute of Cardiology of Abidjan, Abidjan, Côte d'Ivoire
[5]Department of Anesthesiology and Critical Care, Farah Polyclinic, Abidjan, Côte d'Ivoire
Email: *aemmaray15@gmail.com

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## Abstract

**Introduction:** Status asthmaticus during pregnancy is a rare but life-threatening respiratory emergency that can compromise both maternal and fetal outcomes. Its association with severe preeclampsia further complicates management and significantly increases the risk of multiple organ failures. **Case Presentation:** We report the case of a 39-year-old woman with a history of chronic asthma and hypertension, at 31 weeks of gestation, who was admitted to the intensive care unit for status asthmaticus complicated by a cardiopulmonary arrest that was successfully resuscitated. An emergency cesarean section was performed, delivering an extremely preterm infant weighing 800 g, who died on the fourth day of life. The maternal course was complicated by the development of HELLP syndrome with major hepatic cytolysis, thrombocytopenia, acute renal failure, and severe metabolic disorders, consistent with multiple organ failure. Management included lung-protective ventilation, correction of electrolyte and metabolic imbalances, strict blood pressure control, and close multidisciplinary monitoring. Extubation was achieved on day 4, and the patient showed progressive improvement, allowing transfer to a conventional ward on day 8 with full neurological recovery. **Conclusion:** The coexistence of status asthmaticus and severe preeclampsia in the third trimester represents an exceptional and dramatic obstetric emergency, associated with a high risk of multiorgan failure and perinatal mortality. Maternal outcome depends on rapid

and coordinated management, including ventilatory support, strict blood pressure control, timely obstetric intervention, and multiorgan support. This case highlights the importance of a multidisciplinary approach and close follow-up of pregnant women with high-risk asthma.

## Keywords

Status Asthmaticus, Preeclampsia, HELLP Syndrome, Critical Care

## 1. Introduction

Status asthmaticus (SA) is a life-threatening respiratory emergency characterized by severe bronchial obstruction refractory to first-line treatments [1] [2]. In pregnancy, asthma management is particularly challenging due to physiological changes that may exacerbate the disease [1]-[3]. Severe exacerbations increase the risk of maternal-fetal hypoxia, leading to intrauterine growth restriction, prematurity, and increased neonatal morbidity and mortality [1] [2] [4]. Pregnancy may also alter the natural course of asthma, especially in the third trimester, when both the frequency and severity of exacerbations rise [1] [2] [5]. These events are recognized risk factors for maternal complications, notably preeclampsia [4] [6]. In fact, a recent meta-analysis reported that maternal asthma is associated with a significantly increased risk of preeclampsia, with a pooled relative risk ranging between 1.3 - 1.5 compared with non-asthmatic women [7]. Severe preeclampsia is one of the most feared obstetric complications, associated with a wide spectrum of maternal consequences (HELLP syndrome, eclampsia, acute renal failure, multiorgan failure) and fetal outcomes (growth restriction, prematurity, death) [8]-[11]. It remains a leading cause of maternal and perinatal mortality worldwide [11].

The coexistence of SA and severe preeclampsia in pregnancy is exceptionally rare, yet exposes both mother and fetus to extremely poor prognoses due to the synergistic impact on respiratory, hemodynamic, and multiorgan instability [11]. We report the case of a 39-year-old woman, 31 weeks pregnant, with a history of asthma and chronic hypertension, admitted to intensive care for SA complicated by cardiopulmonary arrest secondary to acute respiratory failure and severe preeclampsia. This case highlights the diagnostic and therapeutic challenges of such rare scenarios and underscores the importance of multidisciplinary management involving obstetricians, intensivists, and subspecialists to optimize maternal and fetal outcomes.

## 2. Case Presentation

A 39-year-old woman with a history of asthma and chronic hypertension, at 31 weeks of gestation, presented with several days of dyspnea treated with repeated nebulizations, initially considered as asthma attacks. Symptom worsening prompted

referral to the FARAH clinic due to the unavailability of oxygen therapy.

On admission, the patient was comatose (Glasgow Coma Scale 3) with gasping respirations, rapidly followed by cardiopulmonary arrest. Resuscitation achieved return of spontaneous circulation after intubation and central venous access. An emergency cesarean section was performed. Intraoperatively, the course was marked by severe hypercapnia, oxygen desaturation, pulmonary hypertension, and hemodynamic instability (BP 226/157 mmHg, HR 145 bpm). Severe hypertension was managed with a continuous intravenous infusion of nicardipine combined with hyperventilation. A growth-restricted premature infant weighing 800 g (Apgar 5/6) was delivered and admitted to neonatal intensive care. During transfer to the ICU, the patient experienced a second low-flow cardiac arrest, which was successfully resuscitated.

Under lung-protective ventilation ($FiO_2$ 100%), clinical findings included persistent severe hypertension, tachycardia, and bilateral crackles. Laboratory tests revealed severe mixed acidosis (pH 6.85, $PaCO_2$ 84 mmHg), marked hyperlactatemia (10.3 mmol/L), moderate hepatic cytolysis, and leukocytosis. Cerebral CT was normal, while thoracic CT demonstrated diffuse "white lung" patterns consistent with SA complicated by aspiration.

Initial management included neuro-sedation, protective mechanical ventilation, empirical antibiotics (third-generation cephalosporin and metronidazole), repeated nebulizations, and intensive monitoring.

The course was complicated by multiorgan failure characterized by proteinuria, major hepatic cytolysis (AST > 4700 IU/L), thrombocytopenia (71,000/$mm^3$), severe inflammatory response, acute renal failure, and electrolyte disorders, consistent with severe preeclampsia complicated by HELLP syndrome (Mississippi class II, defined by a platelet count between 50,000 and 100,000/$mm^3$) [12]. Echocardiography revealed concentric hypertensive cardiomyopathy, reduced left ventricular ejection fraction (40%), grade I diastolic dysfunction, and narrowing of the aortic isthmus suggestive of secondary hypertension. Hepatic cytolysis and inflammatory markers gradually regressed, while renal function remained impaired but with preserved diuresis. Following progressive drug tapering and prophylactic anticoagulation, extubation was achieved on day 4 with complete neurological recovery. The patient was transferred to a conventional ward on day 8.

Neonatal outcome was unfavorable, with the infant dying on day 4 of life.

## 3. Discussion

The coexistence of SA and severe preeclampsia evolving toward HELLP syndrome and multiorgan failure in pregnancy is exceedingly rare in the medical literature [4] [6]. This case raises several important issues: epidemiological rarity, clinical complexity, multidisciplinary management, pedagogical implications, and physiopathological insights into the interplay among respiratory inflammation, hypoxia, and endothelial dysfunction [3] [4] [6].

**Epidemiological perspective.** Asthma is the most common chronic respiratory

disease in pregnancy, with a prevalence ranging from 4% to 8% depending on populations and diagnostic criteria [3] [4]. Owing to advances in prenatal care, therapeutic education, and effective medications, severe exacerbations requiring ICU admission remain uncommon [1] [4] [5]. Large cohorts suggest that only 0.4% - 1% of pregnant asthmatic women require hospitalization for severe deterioration, with a risk of acute respiratory failure in cases of poor adherence or exacerbating factors (infection, allergens, treatment discontinuation) [2] [5].

Preeclampsia affects 2% - 8% of pregnancies in developed countries and progresses to severe forms with multiorgan involvement in 10% - 20% of cases [8] [9] [11]. The concomitance of SA and severe preeclampsia is thus extraordinarily rare, emphasizing the scientific and clinical relevance of this case [4] [6] [11]. Literature reports are scarce and usually describe poor outcomes, particularly neonatal but occasionally maternal, when management was delayed [4] [6].

**Severity and unique course.** The atypical and dramatic course of this patient was marked by successive acute events: refractory severe asthma exacerbation, cardiopulmonary arrest, emergency cesarean delivery, HELLP syndrome, and multiorgan failure [8] [9]. Despite two cardiac arrests, acute renal failure, hepatic cytolysis, severe hypertension, and coagulopathy, maternal survival illustrates the resilience of the maternal organism under timely multidisciplinary management [9] [11]. While neonatal prognosis was poor due to extreme prematurity and intrauterine growth restriction, maternal recovery represents a positive signal for future similar cases. In addition, the patient's history of chronic hypertension, a well-established risk factor for preeclampsia, likely amplified endothelial dysfunction and contributed to the rapid progression and severity of her clinical course [8].

**Multidisciplinary dimension.** The favorable maternal outcome reflects the rapid mobilization of a multidisciplinary team—obstetricians, intensivists, neonatologists, and organ specialists [7] [8] [10]. This synergy is critical for managing rare, life-threatening maternal-fetal syndromes. Obstetric decision-making (emergency cesarean) limited prolonged fetal hypoxia, although the neonatal outcome remained unfavorable due to extreme prematurity [4] [8] [9]. Maternal outcome benefited from expert intensive care, including lung-protective ventilation, hemodynamic monitoring, metabolic corrections, antibiotics, and justified neuro-sedation [3] [4] [8]. The thoracic CT revealed a diffuse "white lung" pattern, raising the possibility of acute respiratory distress syndrome (ARDS). Differentiating ARDS from severe status asthmaticus in this setting remains a diagnostic challenge, as ARDS is characterized by non-cardiogenic pulmonary edema and refractory hypoxemia, whereas status asthmaticus involves severe bronchial obstruction and dynamic hyperinflation. This overlap illustrates the complexity of respiratory failure assessment in obstetrics.

**Scientific contribution.** This case highlights possible pathophysiological links between severe asthma and preeclampsia:

- *Systemic inflammation*: Severe asthma is associated with increased pro-inflam-

matory cytokines (IL-6, TNF-$\alpha$) that impair endothelial function, a key feature of preeclampsia [3] [4] [6].

- *Hypoxia*: Recurrent hypoxia in asthma exacerbations promotes oxidative stress and the release of anti-angiogenic placental factors, exacerbating the endothelial dysfunction typical of preeclampsia [3] [4] [6].
- *Endothelial dysfunction*: Exacerbated during severe asthma, it may potentiate HELLP syndrome, thrombocytopenia, vascular permeability, and multiorgan failure [6]-[11].

Such associations open perspectives for research on predictive biomarkers, targeted prevention in high-risk women, and potential endothelial-protective therapies [4] [6]. Beyond these general mechanisms, exacerbations of asthma may precipitate maternal hypoxemia and systemic inflammation that amplify canonical preeclampsia pathways. Placental hypoxia activates hypoxia-inducible factors, particularly HIF-2$\alpha$, which upregulate anti-angiogenic mediators such as sFlt-1 and sEng, driving widespread endothelial dysfunction [13]. Experimental data in trophoblasts confirm that hypoxia induces FLT1/sFlt-1 expression via HIF-2$\alpha$ [14], while recent reviews emphasize the synergistic anti-angiogenic effect of sFlt-1 and sEng in preeclampsia [15].

In parallel, severe asthma is associated with HIF-1$\alpha$ signaling and an exaggerated type-2/Th17 cytokine response, which sustains neutrophilic airway and systemic inflammation. A convergent mechanism involves neutrophil extracellular traps (NETs), increasingly recognized as contributors to placental endothelial injury, thrombosis, and inflammation in preeclampsia [16] [17].

Together, these mechanistic insights suggest that maternal asthma may act as a biological amplifier of preeclampsia through shared hypoxia- and inflammation-driven pathways. They also open perspectives for research on predictive biomarkers, targeted prevention strategies in high-risk women, and potential endothelial-protective therapies [4] [6].

## 4. Conclusion

The coexistence of SA and severe preeclampsia constitutes a major clinical and scientific challenge. This case demonstrates that timely multidisciplinary intensive care can temper the severity of such life-threatening conditions, highlighting the importance of surveillance and therapeutic education in high-risk asthmatic pregnant women. It also underscores the need for further research on shared pathophysiological mechanisms involving chronic inflammation, hypoxia, and vascular dysfunction in pregnancy.

## Authors' Contributions

Ahouangansi Sêtondji Emmanuel Raymond: Study conception, critical review, and final approval. Mouafo Edwige Floriane, Touré Walamitien Cyrille, Doh Zolé Cédick, Kra Lossan Herman, Koffi Aleke, Chuekam Danielle Christian, Kadje Kuate Christian, Chettaoui Slim, Kognombi Marc, Fom Takam Eleonore Sonia, Femtchou

Giadjou Erika Vanessa, Djieumo Aude, Ndjeundo Patrick: Critical review and final approval of the manuscript.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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