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Anesthetic anaphylactic shock in an emergency cesarean section: a case report

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Abstract

Background Neuraxial anesthesia is the gold standard for cesarean sections, but general anesthesia is sometimes necessary, especially in emergency cases. Anaphylactic shock due to succinylcholine, a commonly used neuromuscular blocking agent, is rare but life-threatening.

Case presentation A 42-year-old woman with severe preeclampsia and a history of intracranial vascular malformations underwent an emergency cesarean section. Induction with succinylcholine triggered an anaphylactic shock. Immediate intervention with epinephrine and corticosteroids stabilized the patient, allowing the safe delivery of a live infant.

Discussion and conclusion This case highlights the need for rapid identification and management of anaphylactic shock during emergency cesarean sections under general anesthesia, especially in high-risk patients with complex medical histories.

Keywords Anaphylactic shock, Succinylcholine, Cesarean section, Emergency, Case report

Background

Neuraxial anesthesia is the gold standard for cesarean sections, but there are instances where general anesthesia is inevitable, especially in emergency cesarean Sects. [1, 2]. Rapid-sequence induction and intubation (RSII) with cricoid pressure using thiopental and succinylcholine (Suxamethonium chloride) has been the standard practice for general anesthesia in cesarean Sects. [2, 3]. Until recently, succinylcholine at a dose of 1-1.5 mg/kg was the standard agent used for RSII due to its rapid onset, safety profile, and intense neuromuscular blockade [3].

Perioperative anaphylaxis is a real concern for anesthetists and frequently involves neuromuscular blocking agents, with an IgE-mediated mechanism [4]. Anaphylactic shock caused by succinylcholine is rare, occurring at a rate of 1/223,291, according to a nationwide survey in the UK [5]. Despite the use of epinephrine and aggressive fluid therapy in treating severe allergic reactions, approximately 4% of anaphylactic shocks induced by neuromuscular blocking agents result in fatal outcomes [6].

This case report describes a pregnant woman with a history of intracranial vascular malformations and severe preeclampsia (early-onset type) who experienced an anaphylactic shock during an emergency cesarean section, triggered by succinylcholine.

Case presentation

A 42-year-old primigravida with a singleton pregnancy and severe early-onset type preeclampsia was admitted due to poorly controlled blood pressure. The patient's

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height was 158 cm, with a pre-pregnancy weight of 54 kg, and she experienced a pregnancy weight gain of 11 kg. The patient also had fetal growth restriction and significant proteinuria (24-hour urinary protein of 16.12 g). Her medical history included chronic hypertension, intracranial vascular malformations, uterine fibroids, adenomyosis, obesity, and in vitro fertilization-embryo transfer induced pregnancy, with no known drug or food allergies. For the management of severe preeclampsia, the patient was treated with magnesium sulfate to prevent seizures, as well as labetalol and nifedipine to control her blood pressure.

During the hospital stay, at 30^{+3} weeks gestation, the patient noticed a significant decrease in fetal movements. Non-stress testing (NST) indicated a non-reactive pattern, and bedside biophysical profile scoring was 4 (0 for fetal movements, 0 for breathing-like movements), with a single peak in umbilical blood flow, suggesting acute fetal distress. After communicating with the patient and her family, we immediately prepared for an emergency cesarean section. Since this was an emergency cesarean section, the patient did not undergo pre-operative fasting, as would typically be required for elective surgeries. In preparation for the emergency cesarean section, prophylactic cefazolin was administered to prevent infection. Additionally, standard premedication included antacids to reduce the risk of aspiration during general anesthesia.

Upon induction of general anesthesia with propofol and succinylcholine for an emergency cesarean section: Propofol 2 mg/kg (commercial name: Diprivan), Succinylcholine 1.5 mg/kg (commercial name: Anectine or Quelicin), the patient exhibited a sudden and severe drop in blood pressure (BP) from a pre-induction level of 120/90 mmHg to 70/30 mmHg, and oxygen saturation (SpO2) dropped to 69-80%. Heart rate (HR) fluctuated between 62 and 80 beats per minute. During the anaphylactic episode, the patient exhibited typical skin signs of anaphylaxis, including generalized erythema, flushing, and swelling of the neck, chest, and upper limbs, which are consistent with the acute allergic reaction triggered by succinylcholine. The elevated airway pressure (25–30 cmH2O). End-tidal CO2 was low (22–25 mmHg), indicating impaired ventilation.

Anaphylactic shock due to succinylcholine was suspected. Immediate interventions included:

- **Epinephrine**: Initial boluses of 20–30 μg were administered intravenously, followed by a continuous infusion of 0.3 μg/kg/min to maintain blood pressure and improve cardiac output.
- **Methylprednisolone**: 40 mg was administered intravenously to reduce inflammation and prevent biphasic reactions. Methylprednisolone is commonly used as an adjunct in anaphylaxis management to reduce inflammation and prevent biphasic reactions [7]. While corticosteroids do not act immediately, they help in long-term control [7].
- **Crystalloids**: Intravenous normal saline was used for rapid volume expansion to correct hypotension.
- **Sodium bicarbonate and calcium gluconate** were also administered to correct blood pH and electrolyte imbalances.

A central venous catheter was placed to allow for precise central venous pressure measurements, rapid fluid administration, and medication delivery. In addition, invasive blood pressure monitoring via arterial line placement was utilized to provide continuous and accurate blood pressure measurements, allowing for real-time assessment of the patient's hemodynamic status. In this case, the patient had a history of intracranial vascular malformations, raising concerns about the potential for an aneurysm rupture when she experienced a sudden and severe drop in blood pressure. Although cardiac arrest was not observed, the significant hypotension prompted immediate concern for possible intracranial bleeding. As a precautionary measure, an ice cap was applied to provide cerebral protection in case the hypotension was secondary to a rupture of the vascular malformation. About 30 min later, the patient's vital signs gradually stabilized with BP at 90–110/60–65 mmHg, HR at 55–60 beats per minute, and SpO2 at 100%.

Simultaneously with the above interventions surgery and resuscitation were conducted concurrently. A lower uterine segment transverse incision was made, and a live infant was delivered, measuring 34 cm in length, and weighing 1210 g, with Apgar scores of 6-8-9, and transferred to the neonatal unit. The patient's intraoperative blood loss was approximately 300 ml. At the moment of birth, the maternal vital signs were as follows: BP 92/61 mmHg, HR 89 beats per minute, SpO2 96%.

Despite the stabilization of her vital signs, the patient required postoperative mechanical ventilation and was transferred to the ICU for further care. The extended ICU observation was primarily necessitated by her severe preeclampsia and the occurrence of anaphylactic shock, both of which significantly increased the risk of postoperative hemodynamic instability. Consequently, her stay in the ICU was prolonged to ensure continuous monitoring and support. During this period, she received fluid management and antihypertensive therapy. Serial blood tests, including serum tryptase measurements, were conducted to confirm the diagnosis of anaphylaxis and to guide further treatment. The patient was successfully extubated on the first postoperative day. After further stabilization and improvement of her condition, she was transferred back to the ward on the third postoperative day to continue antihypertensive treatment and recovery

and was discharged three days later. The infant's neurologic evaluation before discharge was normal, with no signs of neurological deficits.

Discussion and conclusions

Given the sudden onset of hypotension, hypoxia, and bronchospasm, it was essential to differentiate anaphylactic shock from amniotic fluid embolism (AFE) [8]. AFE typically presents with a rapid onset of cardiovascular collapse, respiratory distress, and disseminated intravascular coagulation (DIC). However, in this case: (1) Timing and Triggers: The reaction occurred immediately after the administration of succinylcholine, before the abdominal incision, making drug-induced anaphylaxis more likely. (2) Clinical Presentation: The patient exhibited bronchospasm, a common feature in anaphylactic reactions but less typical in AFE. Additionally, there was no evidence of coagulopathy or bleeding, which are hallmark features of AFE. (3) The patient responded rapidly to epinephrine and corticosteroids, which are standard treatments for anaphylaxis. In contrast, AFE treatment typically focuses on supportive care for respiratory and cardiovascular collapse and managing DIC if it occurs. In conclusion, the clinical presentation and response to treatment strongly supported the diagnosis of anaphylactic shock over AFE. It is worth noting that, although rare, propofol can potentially cause anaphylactic shock. We ruled out propofol-induced anaphylaxis based on the following considerations: Timing: The anaphylactic reaction occurred immediately after succinylcholine administration, while propofol had been given earlier without an immediate adverse response. Clinical Presentation: The characteristic signs of anaphylaxis, including a rapid drop in blood pressure, elevated airway pressure, and generalized erythema with swelling, are more typical of reactions associated with neuromuscular blocking agents like succinylcholine. However, more specific diagnostic tests were lacking at the time.

In the diagnosis of anaphylaxis, several biochemical markers can provide additional confirmation and help guide treatment decisions. The most commonly used marker is serum tryptase, which tends to rise within 30 min to 3 h after the onset of an anaphylactic reaction. Elevated tryptase levels can confirm mast cell activation, which is critical for diagnosing anaphylaxis in uncertain cases [7]. In our patient, tryptase was measured after admission to the ICU, and the results were consistent with anaphylaxis. However, other markers such as plasma histamine and specific IgE to the suspected allergen could further support the diagnosis. Due to the acute nature of the case and the need for immediate intervention, we did not conduct these tests. Future cases may benefit from a more comprehensive biochemical evaluation if the clinical setting allows.

For cesarean sections, there is a high risk of complications related to airway management, such as aspiration pneumonia and hypoxia, and a high incidence of difficult or failed intubation [9]. While succinylcholine is traditionally favored for rapid sequence induction due to its rapid onset and short duration, rocuronium presents an alternative for situations where succinylcholine use may be contraindicated, such as in patients with a history of allergic reactions or those at risk of malignant hyperthermia. Rocuronium, when used in combination with a reversal agent like sugammadex, can achieve rapid recovery, making it a viable choice for cesarean sections where neuromuscular blockade is needed. Although rocuronium has a slightly slower onset compared to succinylcholine, its use in rapid sequence induction has gained support due to its safety profile and reversibility [10, 11].

According to a nationwide survey in the UK [5], the incidence of severe systemic allergic reactions to neuromuscular blocking agents is 1/19,070 (95% CI: 1/14,934–1/24,762), with succinylcholine specifically causing reactions at a rate of 1/223,291. In pregnant women, the incidence may be even lower, making such cases exceedingly uncommon. Existing literature suggests that allergic reactions to succinylcholine are more likely in patients with a history of atopy or previous exposure to neuromuscular blocking agents, either through surgery or in an occupational setting [6]. Additionally, cross-reactivity among different neuromuscular blocking agents can occur, potentially increasing the risk of anaphylaxis if there has been prior sensitization to a different agent in this class. Moreover, advanced age and obesity also increase the incidence of allergic reactions to neuromuscular blocking agents [6]. In this case, the patient, a 42-year-old with advanced maternal age, severe preeclampsia and obesity, may have had an abnormal immune response, increasing the sensitivity to the succinylcholine medications. Furthermore, the presence of intracranial vascular malformations necessitates stricter control of blood pressure fluctuations during anesthesia and surgery, adding complexity to the management of anesthesia and surgery, could have made her reactions more sensitive and severe.

Treatment of anaphylaxis during surgery, including cesarean sections, requires immediate intervention to prevent life-threatening complications. The first-line treatment in both pregnant and non-pregnant patients is epinephrine, which rapidly counteracts the vasodilation and bronchoconstriction associated with anaphylactic shock. Other supportive treatments, such as intravenous fluids, corticosteroids, and antihistamines, are used adjunctively to stabilize the patient [12]. The physiological changes during pregnancy, such as increased blood volume, decreased systemic vascular resistance, and

aortocaval compression by the gravid uterus, can complicate the management of anaphylaxis. Pregnant women may have a blunted response to vasopressors, requiring higher doses of epinephrine to maintain hemodynamic stability. Additionally, uteroplacental blood flow is highly sensitive to hypotension, necessitating rapid correction to prevent fetal hypoxia [13]. Therefore, aggressive fluid resuscitation and careful titration of epinephrine are critical in pregnant women compared to non-pregnant patients.

Epinephrine, while lifesaving for the mother, may have transient effects on the neonate. The most common concern is fetal acidosis, which may result from decreased uteroplacental perfusion due to maternal vasoconstriction. However, studies suggest that with appropriate dosing and careful maternal monitoring, the benefits of epinephrine in treating anaphylaxis far outweigh potential risks to the neonate [14]. Neonates delivered following maternal epinephrine use should be closely monitored, but severe adverse effects are rare with proper management [14].

In cases of anaphylaxis during a cesarean section, the timing of neonatal delivery is crucial. Once maternal hemodynamics are stabilized, the delivery of the neonate should proceed promptly to prevent prolonged fetal hypoxia and acidosis. Delaying delivery can lead to worsening neonatal outcomes. In the event of maternal cardiovascular instability, concurrent resuscitation and delivery may be necessary to optimize both maternal and fetal survival [15].

This case underscores the importance of rapid identification and management of anaphylactic shock during emergency cesarean sections under general anesthesia. Continuous monitoring and preparedness for allergic reactions are crucial, especially in high-risk patients with complex medical histories.

Abbreviations

RSII	Rapid-Sequence Induction and Intubation
SpO2	Oxygen Saturation
HR	Heart Rate
ICU	Intensive Care Unit
AFE	Amniotic Fluid Embolism
BMI	Body Mass Index
DIC	Disseminated Intravascular Coagulation

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Author contributions
Rong Zhou and Qi Cao conceived the study and participated in its design and coordination. Shengping Zhou and Hongqin Chen carried out the data collection. Qi Cao drafted the manuscript. All authors read and approved the final manuscript.

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Data availability
The data is available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
The study was approved by the Ethics Committee of West China Second University Hospital. Informed consent was obtained from the patient included in the study.

Consent for publication
Consent to publication has been acquired. The patient gave written informed consent for the clinical details to be published in this study. Moreover, the manuscript does not involve any personal information.

Competing interests
The authors declare no competing interests.

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