

FloTrac®-guided haemodynamic resuscitation for profound cefuroxime-induced anaphylactic shock after induction of anaesthesia for cervical spine surgery: a case report

Kamarol Salleh

Universiti Sains Malaysia

Wan Mohd Nazaruddin Wan Hassan

nazarudin@usm.my

Universiti Sains Malaysia

Case Report

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Abstract

Background

Perioperative anaphylaxis is a life-threatening emergency requiring rapid haemodynamic intervention. Diagnosing anaphylactic shock following anaesthesia induction is challenging, as it mimics more common intraoperative complications and occurs amidst simultaneous exposure to multiple potential triggers. This report details the presentation of this critical condition and highlights how advanced functional haemodynamic monitoring via the FloTrac® system can reveal the exact real-time physiological severity of distributive shock, thereby guiding highly individualised, goal-directed resuscitation.

Case:

A 44-year-old female with well-controlled hypertension underwent elective cervical spine surgery. Minutes after anaesthesia induction and cefuroxime administration, she hyperacutely developed severe bronchospasm, elevated peak airway pressures (40 cmH₂O), cutaneous flushing, and unmeasurable non-invasive blood pressure. Despite discontinuing anaesthetics and administering salbutamol and phenylephrine, profound distributive shock persisted, with an arterial line registering a nadir of 38/23 mmHg. Following emergency stabilisation and abortion of the surgery, she was transferred to the surgical intensive care unit requiring high-dose epinephrine and norepinephrine infusions. Advanced functional haemodynamic monitoring via the FloTrac® system revealed severe capillary leak (initial stroke volume variation [SVV] of 30%). FloTrac-guided SVV successfully titrated aggressive volume expansion, while real-time systemic vascular resistance index (SVRI) and cardiac index (CI) parameters precisely guided vasopressor and inotrope titration to resolve the distributive shock and normalise systemic vascular resistance. By post-admission day 3, metabolic acidosis and lactatemia cleared, vasopressors were discontinued, and the patient was successfully extubated. Subsequent intradermal allergy testing confirmed a definitive, selective hypersensitivity to cefuroxime.

Conclusion

The diagnostic complexity of post-induction anaphylaxis often delays targeted treatment amidst multiple drug exposures. However, utilising real-time functional haemodynamic monitoring using the FloTrac® system provides the immediate physiological precision required during severe distributive shock. Continuous tracking of SVV, SVRI, and CI directly guides successful, goal-directed fluid and vasopressor titration to optimise patient outcomes in life-threatening perioperative emergencies.

Introduction

Perioperative anaphylaxis is a rare but catastrophic emergency, occurring in 1 in 10,000 to 1 in 30,000 anaesthetics [1]. Intravenous cephalosporins, such as cefuroxime, are widely used for surgical site infection prophylaxis but can trigger severe immunoglobulin E (IgE)-mediated immediate hypersensitivity reactions that rapidly progress to profound distributive shock [2]. The primary pathophysiology of anaphylactic shock involves widespread mast cell and basophil degranulation [3]. This degranulation releases potent vasoactive mediators like histamine and tryptase, causing profound, immediate endothelial nitric oxide-mediated vasodilation [4]. Simultaneously, an acute increase in capillary permeability leads to a massive capillary leak, extravasating up to 35% of intravascular volume

into the interstitial space within minutes [3, 4]. During general anaesthesia, identifying this crisis is exceptionally difficult because surgical drapes conceal classic cutaneous signs like urticaria, leaving unexplained profound hypotension as the primary clinical indicator [1, 2].

Standard static blood pressure monitoring reflects pressure rather than volumetric flow, which fails to capture the true underlying functional changes of massive vasodilation [5]. Relying solely on mean arterial pressure (MAP) can delay targeted fluid volume resuscitation or cause the mis-titration of potent vasoactive medications, leading to volume overload or inadequate myocardial perfusion [5, 6].

To overcome these limits, goal-directed hemodynamic management utilising minimally invasive pulse contour analysis has emerged as a crucial tool [6]. The FloTrac® system provides real-time arterial waveform analysis to calculate dynamic parameters, including stroke volume variation (SVV), systemic vascular resistance index (SVRI), and cardiac index (CI), without requiring manual calibration or a central venous catheter [6, 7]. While validated for high-risk surgical cases, its real-time application in titrating aggressive fluid resuscitation and vasopressors during acute intraoperative anaphylaxis remains underreported [7].

Diagnosing anaphylactic shock following multiple-drug administration during anaesthesia induction remains challenging due to atypical presentations that mimic common differential diagnoses. This case report details an uncommon presentation of perioperative anaphylactic shock during cervical spine surgery, later identified as secondary to cefuroxime. We highlight the utility of the FloTrac® system, which provided real-time tracking of hemodynamic severity and guided successful, goal-directed resuscitation.

Case report

A 44-year-old female with a history of hypertension was scheduled for an elective anterior cervical discectomy and fusion at the C5–6 level due to chronic radiculopathy. She reported a 1-year history of left upper limb numbness and a 4-month history of severe right upper limb pain (visual analogue scale score: 8/10) that limited her daily activities and was refractory to oral analgesics. Cervical magnetic resonance imaging demonstrated a C5–6 right posterolateral disc protrusion causing moderate central stenosis and direct impingement of the right C6 exiting and C7 intraspinal nerve roots.

Preoperative evaluation cleared the patient for surgery. Her hypertension was well-controlled with oral telmisartan/amlodipine (80 mg/5 mg, once daily), classifying her as American Society of Anesthesiologists (ASA) physical status II. Airway assessment predicted a non-difficult intubation. Neurological examination was consistent with the cervical pathology, demonstrating 3/5 motor strength in the left C5–C7 distributions and 5/5 strength on the right. Upper extremity sensation was intact, and the remainder of the systemic physical examinations, baseline laboratory investigations, chest radiograph, and electrocardiogram were normal. She reported no known drug or food allergies.

On the day of operation, anaesthesia induction was achieved using intravenous (IV) fentanyl 100 µg, propofol 120 mg, and rocuronium 50 mg. The trachea was successfully intubated on the first attempt via video laryngoscopy using a 7.0-mm endotracheal tube (ETT) secured at 20 cm. Maintenance of anaesthesia was initiated with sevoflurane and subsequently transitioned to target-controlled total intravenous anaesthesia (TIVA) with propofol and remifentanyl to facilitate intraoperative neuromonitoring. Prior to the TIVA transition, a partial test dose of IV cefuroxime (from a 1.5-g total planned dose) was administered, followed shortly by the remaining dose once TIVA target concentrations were reached.

Minutes later, the patient's peak airway pressure abruptly increased to 40 cmH₂O, accompanied by bilateral generalised rhonchi. This prompted the immediate administration of 10 puffs of salbutamol via the ETT. Simultaneously, non-invasive blood pressure and oxygen saturation signals were lost. Serial boluses of IV phenylephrine (100 µg) yielded no hemodynamic improvement despite halting the TIVA infusions. The carotid pulse was barely palpable despite a monitor-registered sinus tachycardia of 120 beats/min. Cutaneous flushing was observed on both upper extremities without a maculopapular rash. Recognising severe perioperative anaphylactic shock, additional staff were mobilised, and multiple IV epinephrine boluses (100 µg) were administered. An emergency arterial line revealed profound hypotension at 38/23 mmHg. Due to extreme hemodynamic lability, continuous infusions of epinephrine and norepinephrine were initiated, alongside IV hydrocortisone 200 mg and chlorpheniramine 10 mg. Frequent premature ventricular contractions (PVCs) emerged on the monitor, and an arterial blood gas (ABG) analysis confirmed severe mixed acidosis: pH 7.11; partial pressure of carbon dioxide (PaCO₂), 63.2 mmHg; partial pressure of oxygen (PaO₂), 76 mmHg; bicarbonate (HCO₃⁻), 16.2 mM/L; and lactate, 4.6 mM/L. Consequently, IV 8.4% sodium bicarbonate (50 mL) and magnesium sulphate (2.47 g) were administered. A subsequent episode of supraventricular tachycardia was successfully managed with carotid massage. The surgical procedure was aborted, and the patient was transferred to the surgical intensive care unit (SICU).

The patient was managed in the SICU under mechanical ventilation for 3 days. Sedation was maintained with IV fentanyl (50 µg/h) and midazolam (5 mg/h). Upon admission, she required high-dose inotropic and vasopressor support with epinephrine (0.35 µg/kg/min) and norepinephrine (0.02 µg/kg/min). Bedside ultrasonography revealed greater than 50% inferior vena cava (IVC) collapsibility and a hyperdynamic myocardium with "kissing ventricles," while the chest radiograph was unremarkable. Initial serial ABGs demonstrated refractory metabolic acidosis with a rising lactate level. At 24:00 hours, the ABG showed a pH of 7.29, PaCO₂ of 33.7 mmHg, PaO₂ of 146 mmHg, HCO₃⁻ of 17.3 mM/L, a base excess of - 10.2 mM/L, and a lactate of 9.2 mM/L.

Hemodynamic stabilisation was achieved using functional cardiac output monitoring guided by the FloTrac® system (EV1000; Edwards Lifesciences, Irvine, CA, USA). Hypotensive episodes were managed by titrating crystalloid fluid volumes and vasopressors based on SVRI and SVV. Baseline parameters during the monitoring initiation were: cardiac output (CO), 4.0 L/min; CI, 2.4 L/min/m²; SVRI, 2,600 dyn·s/cm⁵/m²; and SVV, 30%. Resolving the profound fluid depletion resulting from the distributive capillary leak was designated as the primary therapeutic goal. Guided by SVV, aggressive volume expansion was administered. The SVV required 4 hours to decrease below 15% during the acute stage (Table 1), rose again 3 hours later for a 7-hour duration, and subsequently stabilised (Table 2). SVRI and CO remained optimised throughout the crisis via precise vasopressor titration.

Table 1
Hemodynamic changes on day 1 of admission

TIME	1600	1700	1800	1900	2000	2100	2200	2300	2400
BP (mmHg)	140/90	90/70	85/65	110/70	110/60	100/6	120/70	150/110	130/70
HR (beats/min)	120	110	120	130	110	100	100	110	110
CO (L/min)	4	4.6	2.3	3.6	4.1	7.2	7.6	5.6	6.4
CI (L/min/m ²)	2.4	2.7	1.4	3.5	2.4	4.3	7.2	3.3	3.8
SVRI (dyn·s/cm ⁵ /m ²)	2600	2652	3777	1726	2622	1524	1403	2026	1739
SVV (%)	30	31	23	21	22	6	8	6	25
IV Adrenaline (µg/kg/min)	0.35	0.35	0.35	0.30	0.28	0.28	0.25	0.22	0.20
IV Noradrenaline (µg/kg/min)	0.07	0.05	0.04	0.02	-	-	-	-	-
Fluid bolus (ml)	1000	500	500	500	500	-	-	-	-
Fluid Maintenance (ml/h)	120	120	120	120	120	120	120	120	120

Table 2
Hemodynamic changes on day 2 of admission

TIME	0100	0200	0300	0400	0500	0600	0700	0800	0900
BP (mmHg)	110/60	110/60	110/60	120/70	120/70	120/70	110/60	150/60	160/70
HR (beats/min)	110	110	110	90	90	80	80	80	70
CO (L/min)	4.4	3.8	6.0	6.4	2.8	4.1	4.2	5.0	8.8
CI (L/min/m ²)	2.6	2.3	3.6	3.8	1.5	2.4	2.3	4.8	4.8
SVRI (dyn·s/cm ⁵ /m ²)	1791	2455	1575	1460	2301	1460	1464	1504	1463
SVV (%)	26	23	20	19	36	48	49	5	5
IV Adrenaline (µg/kg/min)	0.2	0.2	0.2	0.2	0.2	0.2	0.17	0.16	0.14
IV Noradrenaline (µg/kg/min)	-	-	-	-	-	-	-	-	-
Fluid bolus (ml)	-	-	-	-	-	-	500	500	-
Fluid Maintenance (ml/h)	120	120	120	120	120	120	120	120	120

By post-admission day 2, metabolic parameters normalised, lactatemia cleared, and the patient's mental status improved to a Glasgow Coma Scale score of E3VTM5 despite a low-dose fentanyl infusion (20 µg/h). Vasopressors were discontinued, and she was successfully extubated to a 60% Venturi mask on day 3. Pre-extubation ABG parameters were: pH, 7.38; PaCO₂, 39.0 mmHg; PaO₂, 119 mmHg; HCO₃⁻, 23.2 mM/L; and lactate, 0.9 mM/L. Dermatology evaluated the resolving cutaneous eruption daily. Due to the domestic unavailability of mast cell tryptase testing, complement profiles (C3, C4), total IgE, and specific IgE via Fluorescent Enzyme Immunoassay (FEIA) were referred to an external reference laboratory. She was discharged home with an allergy alert bracelet and an emergency epinephrine auto-injector prescription.

The outpatient FEIA results were ultimately unremarkable. Because the exact trigger remained unidentified at the time of discharge, the patient was counselled on alternative anaesthesia strategies that strictly omitted all drugs administered during the crisis. She was referred to a specialised centre for comprehensive intradermal drug allergy testing, which revealed strong positive results for cefuroxime. Hypersensitivity testing for all other administered anaesthetic agents—including propofol, fentanyl, remifentanyl, and rocuronium—was negative. Subsequent differential antibiotic testing confirmed a selective allergy to cefuroxime that could potentially extend to beta-lactam antibiotics sharing an identical or highly similar R1-side chain (such as ceftriaxone, cefotaxime, cefepime, and ceftazidime).

Discussion

Perioperative anaphylaxis remains one of the most challenging, high-stakes emergencies encountered by anaesthesiologists. This case highlights a catastrophic presentation of immediate IgE-mediated anaphylactic shock triggered by a cephalosporin antibiotic, cefuroxime, administered immediately after anaesthesia induction for elective cervical spine surgery. In such scenarios, the classic diagnostic criteria, including cutaneous signs, are often obscured by surgical drapes or delayed, forcing clinicians to rely entirely on sudden, profound haemodynamic or respiratory collapse as the primary diagnostic indicators [1, 8]. Our case demonstrates the unique clinical utility of minimally invasive pulse contour analysis (the FloTrac® system) in executing a highly tailored, goal-directed hemodynamic resuscitation during severe, refractory distributive shock.

To contextualise this crisis, it is critical to evaluate the broader epidemiological landscape of perioperative hypersensitivity. Globally, the estimated overall incidence of severe perioperative anaphylaxis sits at approximately 1 in 10,000 anaesthetics [1, 9]. While antibiotics represent a rapidly ascending cause—accounting for roughly 46% to 47% of identified culprits in recent large-scale registry audits like the United Kingdom's 6th National Audit Project (NAP6)—neuromuscular blocking agents (NMBAs) remain the leading classic trigger in multiple international databases, responsible for up to 33% to 50% of cases [1, 9, 10]. Within the NMBA category, suxamethonium and rocuronium exhibit the highest relative risk, with suxamethonium twice as likely to cause severe bronchospasm [1]. Conversely, intravenous induction agents such as propofol are rarely implicated, with an estimated incidence of only 1 in 60,000 exposures [8]. Among antibiotics, the highest risk profile belongs to teicoplanin (16.4 per 100,000 administrations) and co-amoxiclav (8.7 per 100,000), which together account for nearly 89% of antibiotic-induced anaphylaxis in Europe [10]. Cephalosporins like cefuroxime represent approximately 10.4% of reported registry cases [11], making its appearance in this case an important reminder of antibiotic liability.

The primary underlying pathophysiology of anaphylactic shock involves widespread mast cell and basophil degranulation. This process releases potent preformed and newly synthesized inflammatory mediators, including histamine, tryptase, and platelet-activating factor [3]. These agents stimulate endothelial receptors, triggering nitric

oxide synthesis and forcing the opening of endothelial cells [4]. This yields a rapid, devastating cardiovascular collapse: profound, widespread vasodilation that drastically reduces systemic vascular resistance (SVR), combined with a precipitous rise in capillary permeability. This "capillary leak" can force up to 35% of the total intravascular volume to extravasate into the interstitial space within 10 minutes [3, 12], inducing severe relative and absolute hypovolemia

Traditionally, clinicians rely on static pressure-based parameters, such as MAP and central venous pressure, to guide resuscitation [5]. However, static pressures do not correlate well with true intravascular volume or functional fluid responsiveness during conditions of extreme vasodilation [13]. Relying solely on a blood pressure cuff or an arterial line pressure trace during an anaphylactic crisis can lead to a dangerous cycle of blind fluid boluses, potentially causing severe hydrostatic pulmonary oedema or abdominal compartment syndrome, or over-titration of vasopressors, which can induce severe myocardial ischemia and malignant arrhythmias [12, 14].

In this case, the deployment of the FloTrac® system shifted the resuscitation strategy from empirical pressure-based management to a dynamic, goal-directed volumetric protocol. The system uses arterial pulse contour analysis to calculate SVV, SVRI, and CI on a beat-to-beat basis without requiring a central venous catheter or external calibration [6, 7]. Under mechanical ventilation with a regular tidal volume, SVV serves as an exceptionally sensitive indicator of fluid responsiveness [13]. Upon the patient's admission to the SICU, her baseline parameters vividly captured the underlying anaphylactic mechanism: a profoundly elevated SVV of 30% confirmed a state of critical intravascular volume depletion, while bedside ultrasonography corroborated this with "kissing ventricles" and significant inferior vena cava collapsibility.

By prioritising SVV as our primary therapeutic target, we were able to safely execute an aggressive volume expansion program. Tracking the real-time decline of SVV provided a clear endpoint for volume expansion. In our patient, it required four hours of targeted crystalloid administration to reduce the SVV below 15% during the initial acute window. Because the capillary leak phase of severe anaphylaxis can persist for several hours, we noted a rebound elevation in SVV three hours later, which lasted for an additional seven hours. Continuous SVV monitoring allowed us to immediately recognise this secondary volume depletion phase and titrate further fluid boluses pre-emptively, avoiding a recurrence of profound hypotension while simultaneously avoiding volume overload.

Simultaneously, tracking SVRI and CI allowed for the precise, objective titration of our vasoactive and inotropic infusions. Anaphylaxis-induced vasodilation typically demands aggressive alpha-1 receptor agonism to restore vascular tone [8]. However, the excessive administration of epinephrine and norepinephrine can drastically increase afterload, compromise cardiac output and exacerbate lactic acidosis [15]. By monitoring the SVRI (which stood at 2,600 dyn·s/cm⁵/m² at the start of FloTrac® therapy due to the high-dose vasopressor support initiated during transfer) alongside a borderline CI of 2.4 L/min/m², we were able to incrementally wean the vasopressor doses as the intravascular volume was restored via SVV-guided fluid resuscitation. This prevented excessive vasoconstriction, optimised global tissue perfusion, and facilitated the rapid clearance of severe lactatemia (which had peaked at 9.2 mM/L).

Finally, our post-crisis allergy workup reinforces the critical importance of a definitive diagnosis. Cefuroxime is a second-generation cephalosporin, and its cross-reactivity with other beta-lactams is primarily driven by the structural similarity of its R1-side chain [8, 16]. The strong positive intradermal test for cefuroxime in our patient confirmed it as the sole culprit agent, allowing us to safely clear her other induction agents (propofol, rocuronium, fentanyl, and remifentanyl) for future use. Crucially, it enabled us to counsel her against the future use of specific

beta-lactams sharing identical or highly similar R1-side chains, such as ceftriaxone, cefotaxime, and cefepime, while preserving other safer antibiotic alternatives [16].

The primary limitations of our case report include the inability to measure serum tryptase levels during the acute stage of highly suspected anaphylactic shock, as this assay was unavailable at our centre. Furthermore, immediate drug allergy testing could not be performed to identify the specific triggering agents due to local laboratory and diagnostic limitations.

Conclusion

Relying solely on traditional, static pressure monitoring during a severe, acute perioperative anaphylactic crisis can result in delayed, imprecise resuscitation. The integration of advanced, minimally invasive functional haemodynamic monitoring using the FloTrac® system allows clinicians to dissect the complex physiological components of distributive shock in real time. By utilising SVV to direct precise fluid resuscitation and SVRI/CI to titrate vasopressors, clinicians can achieve rapid haemodynamic stability, optimise tissue perfusion, and avoid the catastrophic pitfalls of both under-resuscitation and fluid overload.

Declarations

Ethics approval and consent to participate

Ethical approval was not required for this case report as it involved the use of de-identified patient information, in accordance with institutional and national guidelines.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal

Competing interests

The authors declare that they have no competing interests

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Author Contribution

All authors contributed equally to the patient management, manuscript writing, and final approval of the version to be published.

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Data availability

All relevant data supporting the conclusions of this case report are included within the article.

References

1. Cook TM, Harper NJN, Farmer L, Garcez T, Floss K, Marinho S, et al. Anaesthesia, surgery, and life-threatening allergic reactions: protocol and methods of the 6th National Audit Project (NAP6) of the Royal College of Anaesthetists. *Br J Anaesth*. 2018; 121. <https://doi.org/10.1016/j.bja.2018.04.001>.
2. Mertes PM, Laxenaire M-C, Alla F. Anaphylactic and Anaphylactoid Reactions Occurring during Anesthesia in France in 1999–2000. *Anesthesiology*. 2003; 99:536–45. <https://doi.org/10.1097/00000542-200309000-00007>.
3. Reber LL, Hernandez JD, Galli SJ. The pathophysiology of anaphylaxis. *Journal of Allergy and Clinical Immunology*. 2017; 140:335–48. <https://doi.org/10.1016/j.jaci.2017.06.003>.
4. Kounis NG, Cervellin G, Koniari I, Bonfanti L, Dousdampanis P, Charokopos N, et al. Anaphylactic cardiovascular collapse and Kounis syndrome: systemic vasodilation or coronary vasoconstriction? *Ann Transl Med*. 2018; 6:332–332. <https://doi.org/10.21037/atm.2018.09.05>.
5. Ackland GL, Brudney CS, Cecconi M, Ince C, Irwin MG, Lacey J, et al. Perioperative Quality Initiative consensus statement on the physiology of arterial blood pressure control in perioperative medicine. *Br J Anaesth*. 2019; 122:542–51. <https://doi.org/10.1016/j.bja.2019.01.011>.
6. Saugel B, Cecconi M, Wagner JY, Reuter DA. Noninvasive continuous cardiac output monitoring in perioperative and intensive care medicine. *Br J Anaesth*. 2015; 114:562–75. <https://doi.org/10.1093/bja/aeu447>.
7. Tsai Y-F, Liu F-C, Yu H-P. FloTrac/Vigileo system monitoring in acute-care surgery: current and future trends. *Expert Rev Med Devices*. 2013; 10:717–28. <https://doi.org/10.1586/17434440.2013.844464>.
8. You B-C, Jang A-S, Han J-S, Cheon H-W, Park J-S, Lee J-H, et al. A Case of Propofol-Induced Oropharyngeal Angioedema and Bronchospasm. *Allergy Asthma Immunol Res*. 2012; 4:46. <https://doi.org/10.4168/aair.2012.4.1.46>.
9. Pitlick MM, Volcheck GW. Perioperative Anaphylaxis. *Immunol Allergy Clin North Am*. 2022; 42:145–59. <https://doi.org/10.1016/j.iac.2021.09.002>.
10. Blumenthal KG, Peter JG, Trubiano JA, Phillips EJ. Antibiotic allergy. *The Lancet*. 2019; 393:183–98. [https://doi.org/10.1016/S0140-6736\(18\)32218-9](https://doi.org/10.1016/S0140-6736(18)32218-9).
11. Mo W, Ouyang M, Yang H, Kong W. Comprehensive Study of Drug-Associated Severe Allergic Reactions: An Analysis of High-Risk Medications and Epidemiological Trends Based on the FAERS Database. *Allergy Asthma Immunol Res*. 2026; 18:242. <https://doi.org/10.4168/aair.2026.18.2.242>.
12. Fisher MMCD. Clinical Observations on the Pathophysiology and Treatment of Anaphylactic Cardiovascular Collapse. *Anaesth Intensive Care*. 1986; 14:17–21. <https://doi.org/10.1177/0310057X8601400105>.
13. Marik PE, Monnet X, Teboul J-L. Hemodynamic parameters to guide fluid therapy. *Ann Intensive Care*. 2011; 1:1. <https://doi.org/10.1186/2110-5820-1-1>.
14. Brown SG. Cardiovascular aspects of anaphylaxis: implications for treatment and diagnosis. *Curr Opin Allergy Clin Immunol*. 2005; 5:359–64. <https://doi.org/10.1097/01.all.0000174158.78626.35>.

15. Gouel-Chéron A, Harpan A, Mertes P-M, Longrois D. Management of anaphylactic shock in the operating room. *Presse Med.* 2016; 45:774–83. <https://doi.org/10.1016/j.lpm.2016.04.002>.
16. Romano A, Valluzzi RL, Caruso C, Maggioletti M, Quaratino D, Gaeta F. Cross-Reactivity and Tolerability of Cephalosporins in Patients with IgE-Mediated Hypersensitivity to Penicillins. *J Allergy Clin Immunol Pract.* 2018; 6:1662–72. <https://doi.org/10.1016/j.jaip.2018.01.020>.