# Successful Resuscitation of Intraoperative Clindamycin Anaphylaxis

**Abstract**

Anaphylaxis is a relatively rare, but potentially fatal event whose prevalence is increasing and whose occurrence tends to be under-reported. Perioperative anaphylaxis incidences range from 1 in 3.500 to 1 in 20,000 surgeries. Mortality tends to be significant between 3 to 9%. The principle agents in the perioperative period tend to be neuromuscular blockers, latex and antibiotics. The World Allergy Organization has attempted to clarify the terminology used in describing anaphylaxis in order to increase awareness and promote better treatment. Clindamycin anaphylaxis tends to be fatal. However, we describe in this article a successful resuscitation to clindamycin induced anaphylaxis which presented initially as profuse bleeding.

**Keywords:** Anaphylactic; Anaphylactoid; Anaphylaxis; World Allergy Organization; Clindamycin; Clindamycin induced anaphylaxes; Resuscitation; Hemorrhage

Case Report

Volume 2 Issue 4 - 2015

**John AD[1]*, Paul Kaloostian[2] and Alp Yurter[3]**

[1]*Department of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, USA*
[2]*Department of Neurosurgery, Johns Hopkins University School of Medicine, USA*
[3]*Department of Biomedical Engineering, Johns Hopkins University, USA*

***Corresponding author:** AD John, Assistant Professor of Anesthesiology and Critical Care Medicine, Johns Hopkins University School of Medicine, Johns Hopkins Medical Institutions, 4940 Eastern Avenue, Baltimore, MD 21224, USA, Email: ajohn1@jhmi.edu

**Received:** May 26, 2015| **Published:** June 18, 2015

**Abbreviations:** WAO: World Allergy Organization; AAAAI: American Academy of Allergy Asthma and Immunology; ACAAI: American College of Allergy Asthma and Immunology; EAACI: European Academy of Allergy and Clinical Immunology; ICAAI: International Collaboration in Asthma Allergy and Immunology

## Introduction

The following is a report of successful resuscitation of Clindamycin induced anaphylactic shock. Anaphylaxis is a relatively rare; potentially fatal event whose prevalence is increasing and whose occurrence tends to be under reported. Perioperative anaphylaxis incidences may range from 1 in 3;500 to 1 in 20;000 surgeries with a mortality ranging from 3% to 9% which is significant [1-9]. Confusion has arisen over terminology with immune medicated reactions termed anaphylaxis whereas non-immune medicated reactions are termed anaphylactoid. Several organizations the World Allergy Organization (WAO); the American Academy of Allergy; Asthma and Immunology (AAAAI); the American College of Allergy; Asthma and Immunology (ACAAI) and the European Academy of Allergy and Clinical Immunology (EAACI) have joined together in collaboration known as International Collaboration in Asthma; Allergy and Immunology (ICAAI). The purpose of this effort is to increase knowledge and awareness of allergic conditions; verify language and promote evidence based treatments [2].

The classic Gel and Coombs classification of immune mediated allergic reactions includes 4 Types [4]. Type 1 (lg E medicated) occurs when a drug – lg E complex binds to mast cells or basophils causing a release of histamine and other inflammatory mediators. This usually presents within minutes to a few hours after drug exposure as urticaria; angioedema; bronchospasm; pruritus and anaphylaxis. Type II (cytotoxic) occurs when specific lg E or lg M antibodies are directed at drug-hapten coated cells. The time course for this reactions if variable and presents as hemolytic anemia; neutropenia and thrombocytopenia. Type III (immune complex) occurs with tissue deposition of drug-antibody complexes resulting in complement activation and inflammation. This usually presents 1 to 3 weeks after exposure as fever; rash; urticaria; vasculitis; arthralgias; and serum sickness. Type IV (delayed cell mediated) results from MCH (major histocompatibility complex) presentation of drug molecules to T cells causing cytokine and inflammatory mediator release. Type IV reactions occur a few days to a week after drug exposure and manifests as a maculopapular rash or allergic contact dermatitis [4].

Anaphylactoid reactions are not based on an lg E or immune medial release of vasoactive mediators from mast cells and basophils; rather they involve a non-immune direct activation of the complement and bradykinin cascades with direct activation of mast cells and basophils. Clinical manifestations between anaphylactic and anaphylactoid are indistinguishable. The World Allergy Organization and constituent organizations want to eliminate the use of lg E immune mediated reaction and non-immune mediated-anaphylactoid reaction; but rather use anaphylaxis to describe the clinical condition [1].

The severity of anaphylaxis is based on symptoms and is classified in terms of 4 Grades:

Grade 1: Cutaneous signs;

Grade 2: Non life threatening symptoms;

Grade 3: Life threatening symptoms including arrhythmias and bronchospasm;

Grade 4: Cardiac and respiratory arrest [3].

The principle agents causing anaphylaxes in the perioperative period tend to be neuromuscular blockers; latex; and antibiotics. Other agents implicated during general anesthesia

including induction agents; opiates; colloids; antiseptics such as chlorhexidine; non steroidal anti-inflammatory agents; ACE inhibitors and iodinated contrast. Among antibiotics penicillin's; cephalosporin's; beta lactams; metronidazole; vancomycin and clindamycin are principal causes of anaphylaxis [1].

## Case Presentation

The patient is a 30 year old; 6 feet tall; 320 pound white male aviation engineer who one year prior had a 4-Wheel All Terrain Vehicle accident that caused an epidural hematoma which required an L4 - L5 laminectomy and discectomy for decompression; and then a subsequent decompression for recurrent disease. The patient's prior surgical procedures were performed at another institution. Neither the patient nor the operative notes and chart record indicated any intra-operative or post operative issues of concern. During the intervening period the patient has had multiple issues with pain; back and leg instability; difficulty walking; multiple falls; and repeated emergency room and clinic visits for the above. Although the patient's pain was difficult to control; the patient had weaned himself off the prescribed pain regimen of Lyrica; Flexeril; Oxycontin and Dilaudid; and was self treating his pain with only Aleve. The patient was scheduled for elective surgery. Unfortunately; the patient once again fell and now in addition to difficulty walking and falling; the patient was experiencing perineal numbness and difficulty with erection. The patient presented with a T-10 sensory level. The MRI revealed disc protrusion at T6-T7; flattening of the ventral chord without chord edema; a posterior lenticular structure; and severe canal and neural foraminal stenosis at L4-L5. The patient's past medical history is otherwise remarkable for penicillin and codeine allergy; obesity; poorly controlled pain; peptic ulcer disease and appendectomy.

On the day of surgery for T6 posterior thoracic corpectomy with T3-T9 fusion; the patient presented to the operating room in significant discomfort despite having received large doses of oral Oxycontin; and intravenous dilaudid and Fentanyl as part of his baseline pain regimen. The patient was pleasant; but anxious.

Oxygen via nonrebreather face mask and standard ASA monitors including pulse oximeter; end tidal $CO_2$; noninvasive blood pressure; and EKG were placed. The patient's IV from the floor was nonfunctional so an 18g IV was placed in the left antecubital fossa prior to induction. Preoperative vital signs were heart rate of 71 beats per minute; blood pressure of 113/59 mm Hg; respiratory rate of 18 breaths per minute; and an oxygen saturation of 98% on room air prior to the application of oxygen. Induction was carried out with the administration of Lidocaine 100 mg; Propofol 300 mg; and 100 mg of Recuronium as the paralytic. The trachea was intubated without difficulty with an 8.0 ETT; taped at 24 cm. A 20g right radial arterial line was placed aseptically. A right internal jugular cordis (9F) was placed sterilely under ultrasound guidance without difficulty; and with a negative pressure drop test. A chest x-ray was done to confirm central line placement; exclude pneumothorax; and verify endotracheal tube placement. Once confirmation was received from the radiologist; the patient was flipped prone onto the intraoperative CT compatible operating room table. The eyes; endotracheal tube; head and neck; foley; arms tucked at sides; and all pressure points had been properly positioned and verified; the patient was taped on to the operating room table. Vital signs at this point with skin preparation and draping were a heart rate of 74 beats per minute; blood pressure via arterial line of 128/70 mg Hg; Et $CO_2$ of 30; peak airway pressures had increased by 4 in changing from the supine to prone position; temperature was 35.70° C and oxygen saturation was 100% on 100% $FiO_2$ with flows of 2 liters per minute. The inhalational anesthetic was Isoflurane at an expired concentration of 1.0% the dial (inspired) concentration was 1.2%. In preparation for skin incision; infusions of Remifentanil and Ketamine were started for intraoperative pain control. Since the patient was allergic to penicillin and had received Clindamycin on multiple occasions without incident; an infusion of Clindamycin 600 mg was started. All infusions went into a carrier of lactated ringer's solution at a rate of 250 ml per hour into the patient via the cordis.

Skin incision was made almost 2 hours after induction and the heart rate and blood pressure started to immediately decrease on a beat per beat basis. The infusion of Remifentanil was stopped. No effect on the decreasing heart rate and blood pressure. 50 mg of Ephedrine was given; and the Ketamine infusion was stopped. On the operating field it was apparent that there was profuse bleeding on skin incision and initial dissection. Questions were raised as to the possibility of non steroidal or antiplatelet medication. None had been administered in the week that the patient had been in the hospital. Meanwhile a fluid bolus of 1 liter of normal saline had been given via the side port of the cordis. Both the blood pressure and the heart continued to decline. Blood pressure was now 90's/40's mm Hg despite aggressive treatment with 50 mg of Ephedrine 1 mg Phenylephrine; 2 mg of Glycopyrrolate; and 2 mg of Atropine. The Clindamycin infusion was stopped. Surgical bleeding from initial incision and dissection was now in excess of 200 cc. Blood pressure was now 60/32 mmHg heart rate was 50 bpm. Another bolus of 1 liter of normal saline was given as well as 1 mg of Epinephrine; 1 mg of atropine; and 1 g of calcium chloride. All infusions of medications had been stopped for several minutes. The anesthetic agent had been turned off and the expired Isoflurane concentration was now 0.4% and decreasing. Oxygen flows had been increased to 10 liters per minute. Both peak and mean airway pressures had increased by 10. End tidal $CO_2$ was decreasing and the waveform was becoming irregular.

A cardiopulmonary arrest was occurring; the non-invasive blood pressure and pulse ox were not reading with a blood pressure of 34/30 mmHg on the A-line and heart rate of 32 beats per minute decreasing to 28 beats per minute with a wide complex idioventricular rhythm on the monitor. The patient's back was rapidly closed; and a CODE was called. All available operating room staff arrived. During this period another 1 mg of epinephrine; 100 mg of Hydrocortisone; 1 g of calcium chloride; 50 mg of Benadryl; 20 mg of pepcid as well as another 1 liter of normal saline had been given. An epinephrine infusion at 1 mcg/kg/min was started. Both peak and mean airway pressures were declining. Then the heart rate began to improve from

idioventricular in the 30's bpm to sinus tachycardia in the 110's to 120's bpm. There was a corresponding increase in blood pressure from systolic blood pressure in the 30's mmHg to 60's mmHg to 80's mmHg to 120's mmHg. The patient was released from the taped position on the operating room table and flipped supine on the stretcher. Although the patient was moderately edematous there was no urticaria.

Chest compressions were not required. A total of: 4 mg of epinephrine; 3 mg of atropine; 2 g of calcium chloride; 100 mg of hydrocortisone; 50 mg of Benadryl; 20 mg of pepcid; and 3 liters of normal saline had been given. Minute amounts of Remifentanil and Ketamine had been administered; but almost 300 mg of 600 mg Clindamycin had been given. Over the course of the next hour; the epinephrine infusion was decreased from 1 mcg/kg/min to 0.05 mcg/kg/min. The patient was taken to the Neurosciences Critical Care Unit; and was extubated about 8 hours later. There was no neurological sequela.

The events appeared to be an anaphylactic reaction to Clindamycin. In further discussions with the patient; it was revealed that on multiple occasions; the patient would self treat respiratory and skin infections with partial doses of his wife's Clindamycin. Whether sensitivity had developed from recurrent exposures in the past is unknown. Although Clindamycin appeared as the most likely cause of this anaphylactic reaction; it is well known that multiple medications used in anesthesia along with latex exposure may cause anaphylaxis.

The patient had suffered life threatening reaction but without a definitive diagnosis. Despite this; the patient was taken back to the operating room within 36 hours because of the pressing nature of his neurological symptoms. After a thorough discussion with the patient including the significant possibility of death (due to the short time from the first attempt to the second attempt if another unknown allergen was involved); informed consent was obtained and the second attempt was done. In order to have a clear picture of this incident and exclude any possible confounders; the same staff was involved in the second attempt as in the first; along with an identical anesthetic (Table 1) and surgical technique. The patient successfully underwent a T6 posterior corpectomy with T3-T9 fusion without complications on the second attempt. After surgery; the patient awoke without difficulty and had symptom resolution with return of strength and function. The only difference between the first attempt and the second attempt was the antibiotic administered. Clindamycin had been given on the first attempt with anaphylaxis; whereas Vancomycin was the antibiotic given on the second attempt. Thus; it is most likely that Clindamycin was the cause of the anaphylactic reaction.

## Discussion

The World Allergy Organization [2,5] defines anaphylaxis as a severe; rapid allergic reaction which may result in death. The diagnosis of anaphylaxis is primarily dependent on clinical criteria; and the clinical criteria used by the World Allergy Organization to define anaphylaxis as the following:

a) A sudden onset of illness (minutes to several hours) with involvement of the skin; mucosal tissue or both; and at least one of the following – either sudden respiratory signs and symptoms; or sudden reduced blood pressure or symptoms of end organ dysfunction. OR;

b) Two or more of the following that occur suddenly after exposure to a likely allergen or trigger: sudden skin or mucosal signs or symptoms; sudden respiratory signs or symptoms; sudden reduced blood pressure or symptoms of end organ dysfunction; sudden gastrointestinal symptoms. OR;

c) Reduced blood pressure after exposure to a known allergen for that patient (minutes to several hours): Adults: systolic blood pressure less than 90 mmHg or greater than 30% decrease from previous baseline [5]. Our patient's manifestation of anaphylaxis was profuse blood loss on skin incision rather than the usual urticarial; and cardiovascular collapse with loss of blood pressure; and increase in mean airway pressure by 10 cm (Table 2).

**Table 1:** Medications given.

| | 1st Attempt | 2nd Attempt |
|---|---|---|
| Antibiotics | Clindamycin[600] ≈ 300 mg/given | Vancomycin 1.5 g |
| Preop Anxiolytic | Midazolam [12 mg] | Midazolam [12 mg] |
| **Intraop** | | |
| Induction | Lidocaine 100 mg | Lidocaine 100 mg |
| | Propofol 300 mg | Propofol 300 mg |
| | Rocuronium 100 mg | Rocuronium 100 mg |
| Maintenance | Oxygen | Oxygen |
| | Isoflurane | Isoflurane |
| | Vecuronium | Vecuronium |
| | Remifentanil (infusion) | Remifentanil (infusion) |
| | Ketamine (infusion) | Ketamine (infusion) |
| | Fentanyl | Fentanyl<br>Dexmedetomidate (infusion)*<br>Hydromorphone*<br>Tylenol (infusion)* |

*At the end of the case for post op pain control.

**Citation:** John AD, Kaloostian P, Yurter A (2015) Successful Resuscitation of Intraoperative Clindamycin Anaphylaxis. J Anesth Crit Care Open Access 2(4): 00064. DOI: 10.15406/jaccoa.2015.02.00064

**Table 2**: Blood Loss

| | | |
|---|---|---|
| 1st Attempt | - Skin incision through muscle | ≈ 400-450 cc |
| 2nd Attempt | - Skin incision through muscle to bone | Minimal |
| | - Total Blood Loss: (For T6, T7 corpectomy, T6, T7 Discectomy and T3 – T9 fusion) ≈ 1400 cc | ≈ 1400 cc |

During anesthesia there are many causes of anaphylaxis. As pointed out by Mertes [1,3] the most common causes of anaphylaxis tends to be neuromuscular blockers (≈ 70%); latex (≈ 12%); antibiotics (≈ 8%); hypnotics (≈ 4%); colloids (≈ 3%); opioids (≈ 1% ) and other agents (less than 3%). Among neuromuscular blockers Rocuronium (≈ 30%); Succinylcholine (≈ 23%); Atracurium (≈ 21%); and Vecuronium (≈ 18%) are the primary offenders [6-8]. With our patient; the agent that caused the anaphylaxis appeared to be Clindamycin; but unfortunately there were other potential causes. Hopkins operating rooms tend to be largely latex free due to the work of Dr. Robert Brown. The same staff using the same anesthetic; using the same surgeons and techniques; under the same conditions was involved in the second attempt. The only difference was the replacement of the antibiotic Clindamycin (the suspected cause of anaphylaxis) with Vancomycin as the antibiotic given. Since the second attempt was unremarkable and uneventful; Clindamycin was most likely the cause of the near fatal anaphylactic reaction.

According to allergists and immunologists; there is an increase in the incidence of anaphylaxis with a lifetime prevalence of almost 2%; and deaths from anaphylaxis are significantly under reported [1-3]. On post mortem; the acute screen markers of anaphylaxis are not apparent [2-4]. Classical Type I reaction (Anaphylaxis) involves lg E mediated hypersensitivity reaction leading to the release of basophils and mast cell mediators. This usually occurs from repeated exposure to the triggering agent; but may also arise from the initial exposure to a substance which bears a similarity to a triggering substrate; because of the cross reactivity between substrates. A variety of metabolic pathways may trigger the activity and release from mast cell and basophils. In addition; either the classical or alternative (complement) pathways can form an aphylatoxins which stimulate the release of basophils and mast cell substances. In anaphylaxis the mediators released include histamine which leads to vasodilatation; prostaglandins which result in bronchoconstriction and vasodilatation; leukotrienes which lead to edema; vasodilatation and proteoglycans which stimulate the coagulation cascade leading to DIC; arachidonic acid metabolites which result in both vasodilatation and increased capillary permeability; and nitric oxide which results in vasodilatation; increased capillary permeability and hypotension.

There may also be non-immune mediated release of substrates from basophils and mast cells (formerly known as anaphylactoid reaction) which is clinically indistinguishable from anaphylactic reactions [1-8]. As pointed out by Mertes [10]; using a simulator none of 42 anesthesiologists were able to correctly diagnose or treat an anaphylactic reaction during the first 10 minutes; and only 25% were able to do so after prompting by the instructor. This is reflected in the significantly high mortality rate from anaphylaxis (≈ 10%) even when treated and resuscitated in the operating room. Whereas; the complication rate from surgery is less than 0.5% [10-12].

Successful resuscitation of Clindamycin anaphylactic shock in the operating room is extremely rare; most cases proceed to fatality. Why is intraoperative Clindamycin anaphylaxis so deadly? Clindamycin is a Lacosamide antibiotic that acts on the ribosome to inhibit protein synthesis and is primarily active against gram positives and skin flora [13-15]. The most common adverse reaction to Clindamycin is the development of pseudo membranous colitis. Occasionally toxic epidermal necrolysis and Stevens Johnson like syndrome have been reported. Anaphylaxis to Clindamycin; in contrast; is a rare reaction; unlike a penicillin allergy. In the community Clindamycin is used orally or topically for skin conditions. In the hospital; however; Vancomycin was the preferred alternative for penicillin allergic patients [13-15]. The increase in prevalence of Vancomycin resistant enterococcus had led to infectious disease specialist's actively discouraging Vancomycin use. Clindamycin's narrow spectrum and the potential for pseudo membranous colitis in ill hospitalized patients limits its use. However; in the subpopulation of healthy patients coming to the operating room for minor procedures who are penicillin allergic; Clindamycin is a preferred antibiotic; especially in light of its lower cost.

When used intraoperatively; Clindamycin tends to be given in these young or otherwise healthy patients with penicillin allergies for quick or minor procedures. These patients are "healthy" with low resting heart rate and blood pressure; and intraoperatively it is very unlikely that they will have central venous access or invasive arterial blood pressure monitoring. Rapid or bolus administration of Clindamycin in the pre-induction period can produce minutes of confusion as to the cause of the decrease of blood pressure and variability of heart rate response. The initial anesthetic response is that one is witnessing a decrease in blood pressure and change in heart rate from the normal responses to induction agents given to a healthy; compliant; volume depleted patient with low baseline resting blood pressure. Common things are common. It takes several minutes to recycle a blood pressure even up to 10 minutes if a standard non-invasive device is used.

Unfortunately; this is occurring at the same time there is intense operating room activity - the patient is being positioned or prepped; drapes are going up; multiple people are talking; and monitors are routinely being disconnected or alarming. By the time that there is recognition that a catastrophic event is occurring - valuable time has passed up to 10-15 minutes. The abrupt realization that a catastrophic; potentially fatal event is underway in what was to be another normal routine case is a shock to every on the room - regardless of how skilled or clinically competent the involved staff are. In the setting

of complete cardiovascular collapse the need for immediate action and diagnosis; even under optimal circumstances usually means that even the best resuscitation is somewhat belated. This is a possible explanation of why Clindamycin anaphylaxis intraoperatively tends to be so fatal.

Our resuscitation of intraoperative Clindamycin anaphylactic shock was successful. This was due in large part to the presence of experienced faculty; invasive monitoring (arterial line giving beat to beat pulse and blood pressure); the presence of a large bore central line (9F cordis); the separation in time of almost 2 hours from induction; and the early aggressive resuscitation that was done on the prompt recognition that the unexpected increased surgical bleeding on skin incision was a manifestation of anaphylaxis. The other report of successful resuscitation of intraoperative Clindamycin anaphylactic shock was by Dr. Chien-Kun Ting and associates in the Journal of the Chinese Medical Association [16]. They were experienced faculty; with a clear and proximate time course to Clindamycin administration; with both an invasive arterial line and central intravenous access; and they provided definitive prompt and aggressive treatment that resulted in a successful outcome.

## Summary

We report a successful resuscitation of Clindamycin anaphylactic shock in the operating room. This was due in large part to an attention to detail and a prompt recognition; diagnosis and treatment of an abnormality. The initial manifestation of anaphylactic shock was an unexpected amount of bleeding in the operative field. This led to the rapid diagnosis - anaphylaxis to Clindamycin. Then prompt treatment-cessation of the offending agent (only 300 mg of the 600 mg Clindamycin were given) and aggressive early resuscitation of the anaphylaxis.

## Recommendations

a) Antibiotics should be given slowly by infusion either preoperatively or well after induction. In an era when speed and efficiency are being emphasized administratively; patient safety should not be jeopardized.

b) Prompt recognition; diagnosis; and treatment of anaphylaxis [17,18].

c) Referral to Allergist. Anaphylaxis is a clinical diagnosis and not dependent on the presence of elevated tryptase; histamine; or PAF [2,5].

## References

1. Galvao VR, Giavina-Bianchi P, Castells M (2014) Perioperative Anaphylaxis. Curr Allergy Asthma Rep 14(8): 452.
2. Simons FE, Ardusso LR, Bilò MB, Cardona V, Ebisawa M, et al. (2014) International consensus on (ICON) anaphylaxis. World Allergy Organ J 7(1): 9.
3. Kannan JA, Bernstein JA (2015) Perioperative anaphylaxis: diagnosis, evaluation and management. Immunol Allergy Clin North Am 35(2): 321-334.
4. Reidl MA, Casillas AM (2003) Adverse drug reactions: types and treatment options. Am Fam Physician 68(9): 1781-1790.
5. Simons FE, Ardusso LR, Bilò MB, Dimov V, Ebisawa M, et al. (2012) 2012 Update: World Allergy Organization Guidelines for the assessment and management of anaphylaxis. Curr Opin Allergy Clin Immunol 12(4): 389-399.
6. Simons FE, Ardusso LR, Dimov V, Ebisawa M, El-Gamal YM, et al. (2013) World allergy organization anaphylaxis guidelines: 2013 update of the evidence base. Int Arch Allergy Immunol 162(3): 193-204.
7. De Bisschop MB, Bellou A (2012) Anaphylaxis. Curr Opin Crit Care 18(4): 308-317.
8. Simon FER, Sheihk A (2013) Anaphylaxis: the acute episode and beyond. BMJ 346: 1602.
9. Fuzak JK, Trainor J (2013) Comparison of the incidence, etiology, and management of anaphylaxis over time. Pediatr Emer Care 29(2): 131-135.
10. Mertes PM (2005) Special Report: Recognition and Management of Anaphylactic and Anaphylactoid Reactions during Anesthesia. Anesthesiology News.
11. Hepner DL, Castells MC (2003) Anaphylaxis during the perioperative period. Anesth Analg 97(5): 1281-1395.
12. Mertes PM, Lexenaire MC (2012) Allergic reactions occurring during anesthesia. Eur J Anaesthesiol 19(4): 240-262.
13. Osei DA, Boyer MI (2012) Preoperative antibiotic prophylaxis in the penicillin allergic patient. J Hand Surg Am 37(12): 2623-2625.
14. Stratton CW (1998) Macrolides, Lincosamides, and Streptogramins: New Agents and new roles. Antimicrobics and Infectious Disease Newsletter 17(12): 89-96.
15. Spizek J, Novotna J, Rezanka T (2004) Lincosamides: chemical structure, biosynthesis, mechanism of action, resistance and applications. Adv Appl Microbiol 56: 121-154.
16. Chiou CS, Lin SM, Lin SP, Chang WG, Chan KH, et al. (2006) Clindamycin-induced anaphylactic shock during general anesthesia. J Chin Med Assoc 69(11): 549-551.
17. Schulz CM, Endsley MR, Kochs EF, Gelb AW, Wagner KJ (2013) Situation awareness in anesthesia: concept and research. Anesthesiology 118(3): 729-742.
18. Levy JH, Castells MC (2011) Perioperative anaphylaxis and the United States perspective. Anesth Analg 113(5): 979-981.

**Citation:** John AD, Kaloostian P, Yurter A (2015) Successful Resuscitation of Intraoperative Clindamycin Anaphylaxis. J Anesth Crit Care Open Access 2(4): 00064. DOI: 10.15406/jaccoa.2015.02.00064