# Response to: Heparin Reverses Anaphylactoid Shock in a Porcine Model

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## Response to: Heparin Reverses Anaphylactoid Shock in a Porcine Model

*To the Editor:*

A chill went down my spine as I read the brief research report by Heflin et al that appeared in the August 2006 issue of *Annals of Emergency Medicine* describing how heparin rapidly reversed anaphylactoid shock in a porcine model.[1] My reading followed an interesting clinical case of a 54-year-old emergency department (ED) registered nurse who had medicated herself with a diuretic at the beginning of her shift for leg edema. By early afternoon she was complaining of itching and concern of an allergic reaction. Like the terrible patients we health care providers can be, she elected a self-trial of Benadryl rather than check herself in as a patient, as advised. In an already overloaded busy shift she hated, I'm sure, to "bother us." The ED in question is a freestanding facility with no inpatient critical care resources.

A short period thereafter she was found on the ambulance loading dock of the ED with rigors, peripheral cyanosis, difficulty breathing and a pulse oximetry of 80% when put on 2L of $O_2$. Presumptively she was in anaphylactic shock. She did not respond in the first hour to traditional treatment (antihistamines, adrenaline and methylprednisolone). She continued to have dyspnea that required bag assist ventilations and subsequent bipap. Her EKG showed nonspecific ST changes (no elevations) and sinus tachycardia.

The teleintensivist was not convinced that her dyspnea was entirely related to this allergic phenomena since she was not responding to traditional therapy and in fact, her blood pressure was dropping. While still conscious, she adamantly denied chest pain (but did feel like her "bra was strapped on too tight"). Under both the intensivist and the on-call cardiologist's recommendations, a dose of 5000 units heparin IV bolus was initiated on the possibility that her dyspnea and leg edema were of cardiogenic origin and not the originally perceived allergic reaction. Within 10 minutes of heparin bolus our friend and survivor began to improve. Her $O_2$ saturations began normalizing, her discomfort improved and blood pressure stabilized. By the time of her arrival to the accepting facility's critical care bed, she was converted to $O_2$ by nasal cannula. Her subsequent cardiac workup showed no abnormalities of her troponin, echocardiogram, or serial EKGs. Her computed tomographic scan showed no pulmonary embolism and her D dimer was negative.

In discussing the case with the night intern at the time, I said, "Curiously she seemed to turn the corner after the heparin was given; it will be interesting to see if she has cardiac disease." Only days later, I read the article by Heflin et al. I don't have a serum histamine level on this patient so one might respond that it was a "coincidence and only an anecdotal case." But for me, I will use the drug again in a similar situation, and who knows, perhaps our "accidental" case was the first patient trial.

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1. Heflin CR, Brewer KL, Hack JB, et al. Heparin reverses anaphylactoid shock in a porcine model. *Ann Emerg Med*. 2006;48:190-193.

*In reply:*

Several years ago I gave a continuing medical education lecture on anaphylactic shock and discussed a case of intractable shock. During the question period, an otolaryngologist in the audience asked why I had not used heparin as treatment. My reply was that giving heparin for anaphylactic shock was an alien concept to me that is not discussed in the medical texts or medical literature. The otolaryngologist replied that he gets calls from his emergency department (ED) to come and perform tracheostomies on patients with intractable upper airway edema not responding to conventional therapy. He instructs the emergency physician to give a loading dose of heparin. By the time he arrives in the ED, the airway edema has resolved.

The clinical literature about using heparin to treat anaphylactic and anaphylactoid reactions is sparse and anecdotal.[1,2] The basic science literature demonstrates that heparin binds histamine and other mast cell mediators[3,4] and provides a rationalization for efficacy. The encounter with the otolaryngologist and basic science articles motivated the experimental study of the efficacy of heparin for anaphylactoid shock.[5] The resolution of shock after administration of heparin in the porcine model was immediate.[5] My belief is that the resolution of shock after heparin administration in the above case was not coincidental. I agree that using heparin to treat intractable anaphylactic and anaphylactoid reactions is justified when conventional therapy fails.

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## Periorbital Necrotizing Fasciitis and Orbital Apex Syndrome as a Delayed But Emergent Complication of Silicone Nasal Augmentation

*To the Editor:*

Alloplastic nasal augmentation with silicone elastomer is popular in aesthetic rhinoplasty. There are only a few reports with regard to the possible late complications related to nasal alloplasts made of bio-inert silicone material.[1-3] We report a rare case of nasal augmentation with a silicone implant leading to delayed severe paranasal sinusitis, orbital apex syndrome and periorbital necrotizing fasciitis, requiring emergent surgical intervention.

A 45-year-old female woman with type 2 diabetes mellitus, who had undergone rhinoplasty using a silicone implant 15 years ago, presented with a 1-day history of fever and chills. She was noted to have painful erythema and skin pustules over left-sided periorbital and glabellar areas on arrival to the emergency department (ED) (Figure, panel A). Reduced visual acuity, an afferent pupillary defect and ophthalmoplegia were revealed on examination of the left eye. Laboratory testing revealed a neutrophilic leucocytosis and hyperglycemic ketoacidosis. A plain facial radiograpph was unremarkable.

An emergent magnetic resonance imaging (MRI) of the head revealed retention cysts with fluid levels and mucosal thickening over left maxillary and ethmoid sinuses. T2-weighted imaging showed marked increased signal intensity within the orbital apex and impinged left optic nerve (Figure, panel B). Periorbital necrotizing fasciitis with concomitant paranasal sinusitis and orbital apex syndrome was diagnosed. Urgent surgical debridement of the infected necrotic tissue and drainage of paranasal sinuses were performed (Figure, panel C). A mobile silicone nasal prosthesis surrounded by purulent exudates was identified and removed (Figure, panel D). She was empirically started on IV amoxicillin and clavulinic acid. Swab and blood cultures demonstrated growth of oxacillin-sensitive *Staphylococcus aureus* and her therapy was subsequently shifted to IV oxacillin and ceftriaxone sodium. The fasciitis resolved rapidly and the patient remained stable for the duration of her stay in hospital. However, visual acuity impairment and opthalmoplegia persisted over the following 2 months.

Although relatively uncommon, long-term complications related to silicone nasal implant have been observed. Late infection, migration, erosion, extrusion and dorsal nasal cyst formation were reported previously.[2-4] In our case, the patient experienced delayed but severe orbital, periocular and paranasal complications related to the silicone nasal prosthesis, which required a combination of immediate surgical debridement of necrotic tissue, removal of implant, and intensive parenteral antibiotic therapy. Such clinical scenario is rare and has never been reported in the literature.

Silicone-mediated inflammatory responses have been proposed to explain the delayed complications associated with silicone implants. After implantation, small silicone particles