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# EPINEPHRINE CAUSES SEVERE LACTIC ACIDOSIS IN A PATIENT WITH SHELLFISH-INDUCED ANAPHYLAXIS

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**PURPOSE:** Epinephrine is the recommended treatment for anaphylaxis. We report a case of anaphylaxis without shock complicated by lactic acidosis after administration of epinephrine. Our goal is to increase clinicians' awareness of epinephrine induced lactic acidosis in anaphylaxis patients.

**METHODS:** A 54-year-old male with history of shellfish allergy presented to emergency room with dyspnea, dysphagia and urticaria, 20 minutes after he consumed an assortment of seafood. Vital signs were: T 97.8, HR 72, BP 132/59 mmHg, RR 24, pulse oximeter 95% on room air. Physical examination showed edema of posterior pharynx, enlarged tonsils, and rash. ABG showed elevated lactate (3.2). EKG showed sinus rhythm with premature atrial complexes. He was medicated with subcutaneous epinephrine, followed by epinephrine drip, diphenhydramine, famotidine, methylprednisolone, and IV fluids. Despite clinical improvement, the lactate level increased from 3.2 to 12.4 (Table 1). We decided to discontinue the epinephrine drip and the lactate level improved to 4.2. Other possible causes of lactic acidosis were ruled out.

**RESULTS:** The patient reported complete symptom relief and left against medical advice.

**CONCLUSIONS:** Epinephrine induced lactic acidosis does not relate to poor clinical outcome. The mechanism of epinephrine induced lactic acidosis in this case presentation is probably related to increased glycogenolysis, glycolysis and lipolysis.

**CLINICAL IMPLICATIONS:** In patients presenting with severe allergic reactions with associated lactic acidosis, a decision to continue or discontinue epinephrine needs to be made depending on the shock status. In the presence of clinical improvement with worsening lactic acidosis, a trial of discontinuation of the epinephrine needs to be considered.

**DISCLOSURE:** No significant relationships.

**KEYWORDS:** lactic acidosis, anaphylaxis, epinephrine

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