



**A Case of Metformin-Associated Lactic Acidosis in the Setting of Hypovolemia**

**Friday,  
October 31, 2014  
1:05-1:15 pm**

*The following abstract will be presented at the Southern Medical Association Annual Scientific Assembly, October 30-November 1, 2014 in Destin, Florida.*

**Author and Co-Authors**

**Layla Abushamat, BA**, Tulane University School of Medicine, New Orleans, LA; **Thankam Nair, MD**, Baton Rouge General Internal Medicine Residency Program, Baton Rouge, LA; **S. Raju Vatsavai, MD, CPPS**, Clinical Assistant Professor, Department of Internal Medicine, Tulane University School of Medicine, Baton Rouge Satellite Campus, Baton Rouge, LA.

**Objectives**

Upon completion of the lecture, attendees should be better prepared to:

- 1) Recognize metformin side effects and their relevance in various settings.
- 2) Distinguish causes of anion gap metabolic acidosis and types of lactic acidosis.
- 3) Review general criteria for diagnosis of metformin-associated lactic acidosis.

**Abstract**

**Introduction:** Diabetes has a high prevalence rate in the United States. If treatment with lifestyle prevention fails, metformin is the typical initial pharmacologic therapy. Awareness of its potential side effects in the setting of an acute disease state is applicable to all patients, regardless of history of renal or liver disease.

**Case Presentation:** A 45 YOM with history of hypertension and type 2 diabetes mellitus, controlled with metformin and benazepril, presented with 2 day history of vomiting and diarrhea. Patient was hypotensive, and tachycardic, but afebrile. He was admitted with wide anion gap metabolic acidosis with severe lactatemia and acute kidney injury. A serum metformin level was significantly above therapeutic range. Urine toxicology, blood toxicology, blood culture, urine culture, chest x-ray and x-ray of the kidneys, ureter, and bladder were all negative.

**Final/Working Diagnosis:** The etiology of his acute kidney injury was likely prerenal due to hypovolemia and hypotension from repeated vomiting and diarrhea, exacerbated by the patient being on an ACE inhibitor. In this setting, metformin levels were able to build up and cause lactic acidosis. His hypovolemia and hypotension led to decreased tissue perfusion, which allowed for further production of lactate.

**Management/Outcome/Follow-up:** The patient was admitted to the ICU but continued to deteriorate, eventually requiring cardiac resuscitation, mechanical ventilation, and treatment with 24 hours of bicarbonate-buffered continuous venovenous hemodialysis with epinephrine and vasopressin support. This case raises practitioner awareness of the potential for metformin-associated lactic acidosis in diabetic patients with no history of liver or kidney disease. In the setting of hypovolemia due to vomiting and diarrhea, metformin can accumulate in the blood stream, especially if the patient is concurrently on an ACE inhibitor. Patients should be warned of this potential risk and seek medical help in acute illness and other conditions that could cause hypovolemia.

**Disclosure**

**Layla Abushamat, BA** – No Relevant Financial Relationships to Disclose  
**Thankam Nair, MD** – No Relevant Financial Relationships to Disclose  
**S. Raju Vatsavai, MD, CPPS** – No Relevant Financial Relationships to Disclose