中文題目：以雙胍類治療的糖尿病人服用磺胺類抗生素導致乳酸血症的案例報告

英文題目：Trimethoprim-Induced Lactic Acidosis in a Diabetes Treated with Metformin

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Introduction: Trimethoprim is an antimicrobial agent that is used extensively in combination with sulfamethoxazole (as TMP-SMX) to treat a variety of common infections. Bacterial cell growth is interrupted through the inhibition of the enzyme dihydrofolate reductase which is responsible for the tetrahydrofolic acid production. Metformin is a commonly used oral anti-diabetic drug that can decrease hepatic glucose production. Among the two drugs, hyperkalemia has been recognized as a common complication of trimethoprim and lactic acidosis is a well-known but rare side effect of metformin. We reported a diabetic patient with long-term metformin treatment who suffered from lactic acidosis when using trimethoprim.

Case report: A 72-year-old female with diabetes for decades was treated with four-combined oral anti-diabetic drugs including glyburide, metformin, pioglitazone, and acarbose. The capillary glucose levels during outpatient-department visits were around 200 mg/dl. However, she suffered from acute gastritis with vomiting. Because of poor oral intake, hypoglycemia attacked and thus she was admitted. After hospital management, she was discharged with metformin and repaglinide for treatment of diabetes. Besides, trimethoprim-sulfamethoxazole (TMP-SMX) was prescribed according to the antibiotic sensitivity test for urinary tract infection with *Escherichia coli*. Two days after discharge, she came back to the emergency department complaining of dyspnea. The blood glucose was 208 mg/dl without ketonuria and potassium level was 4.7 mmol/L. Metabolic acidosis with bicarbonate 16.3 mmol/L and elevated lactate level 3.0 mmol/L (normal range: 0.5~2.2 mmol/L) were noted from blood tests. Lactic acidosis was impressed. Therefore, prescriptions of metformin and TMP-SMX were stopped. Three days later, her lactate level had decreased to 0.5 mmol/L. Because of her past medication history with long-term metformin use, metformin-induced lactic acidosis was less favored. It was suspected that TMP-SMX caused lactic acidosis in the diabetic patient.

Conclusion: As far as we know, this is the first reported case of trimethoprim-induced lactic acidosis. Due to limited references, the mechanism of lactic acidosis caused by trimethoprim is unknown. Also it is not sure whether the occurrence of lactic acidosis is related to the interaction between metformin and trimethoprim. Further study is needed to facilitate better understanding of the drug effects. The case report indicated the importance of awareness for lactic acidosis when prescribing trimethoprim to treat bacterial infections, especially in diabetic patients with metformin treatment.

Keywords: trimethoprim- sulfamethoxazole, lactic acidosis, diabetes, metformin