Cotrimoxazole-induced hypoglycemia in outpatient setting

In February 2013, a 60-y-old woman with a 10-y history of type 2 diabetes was referred to our outpatient endocrinology and diabetes clinic with uncontrolled blood glucose. She had routine timely follow-up and her blood glucose was well controlled using 10 units of long-acting insulin per day. She also received 80 mg aspirin per day. She explained that she was doing well with this regimen until she developed dysuria and malodor urine without fever. After obtaining urine analysis and culture, she was diagnosed with a urinary tract infection (UTI) by a general practitioner and treated with cotrimoxazole 480 mg (400 mg sulfamethoxazole/80 mg trimethoprim) tablets twice daily. Three days after starting cotrimoxazole; she experienced two episodes of symptomatic hypoglycemia with tremor, sweating, and fatigue without alteration in mental status. The concomitant capillary glucose checked by glucometer at home was 60 mg/dL (Fig. 1). She did not return to the hospital but discontinued her insulin afterward. Laboratory data including renal and liver function tests were absolutely normal and she did not use any other drugs or herbals except cotrimoxazole. The patient was maintained on insulin therapy, again reaching the initial doses during 1 wk. She did not report any other episode of hypoglycemia at her 6-mo follow-up.

Cotrimoxazole is a commonly prescribed antibiotic that is widely used for treatment of UTIs, uncomplicated sinusitis, and bronchitis. Cotrimoxazole is generally well tolerated; however, it has its own complications, as with any other medication.

Fig. 1. Alterations in blood glucose during cotrimoxazol therapy.

Hypoglycemia in the context of cotrimoxazole therapy has been rarely described in patients with or without AIDS presenting with Pneumocystis jiroveci pneumonia [1]. Hypoglycemia also has been reported in patients with UTIs who are given cotrimoxazole [2]. Nearly all of the previously reported patients have had concomitant risk factors for hypoglycemia, such as using propoxyphene, sulfonylurea, or repaglinide [3]. This is mainly supposed to be secondary to the inhibitory effects of trimethoprim and sulfamethoxazole on cytochrome P450 (CYP) isoforms, CYP2 C8 and CYP2 C9, leading to increased plasma concentrations of these oral hypoglycemic agents [4]. However, it should be remembered that sulfamethoxazole can induce pancreatic insulin release by binding to islet cells and mimicking sulfonylurea action when administered in high doses intravenously or in patients with acute or chronic renal failure [5].

Hypoglycemia can occur spontaneously in patients with liver diseases, malnutrition, renal impairment, shock, hypopituitarism, adrenal insufﬁciency, and in those receiving some medications [6,7]. Presence of these risk factors will increase risk for hypoglycemia in patients being treated with cotrimoxazole [8]. Here, we reported a diabetic woman with cotrimoxazole-induced hypoglycemia in an outpatient setting. The patient is unique because she was on insulin therapy and had no other risk factor for development of hypoglycemia. Her renal and hepatic functions were completely normal; she did not suffer malnutrition and did receive oral cotrimoxazole for UTI. The important issue that should be considered by clinicians is that cotrimoxazole may induce hypoglycemia even in an outpatient setting and in patients on insulin therapy. This potentially fatal complication should be remembered when prescribing this antibiotic.

References


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