OBSERVATIONS

Possible Influence of Ileal Neobladder on Assessment of Urinary C-peptide

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Urinary C-peptide (UCP) is one of the most popular indicators of insulin secretion by pancreatic β-cells (1,2). However, there are no previous reports discussing the relationship between UCP and neobladder, a new, reconstructed bladder using the ileum after radical cystectomy (3,4). We report a patient with neobladder in whom, among parameters of endogenous insulin production, only the 24-h UCP level was extremely decreased.

A 61-year-old Japanese man with diabetes and atrial fibrillation was hospitalized because of left-sided hemiplegia and motor aphasia caused by thrombotic cerebral infarction. The patient had undergone bladder reconstruction with 66 cm of the ileum after radical cystectomy as the result of cancer 10 years ago. Only a lineal operation scar was found on the middle lower abdominal wall. After surgery, insulin therapy had been initiated on the basis of decreased UCP levels and hyperglycemia. However, his glycemic condition on admission was poor (hemoglobin A1C [HbA_{1c}], 10.4%). In addition to overt glycosuria, urinalysis revealed urinary tract infection. Although insulin resistance was absent, assessment of insulin secretory capacity revealed contradictory results: good response to glucagon tolerance test (4.99 ng/mL of serum C-peptide [after 5 min], increase in serum C-peptide of 3.00 ng/mL [Δ5 min]) and normal homeostasis model assessment of β-cell function index (43.7), but extreme decrease in 24-h UCP levels (<0.1–0.2 μg/day) at five different times. Since the patient's endogenous insulin secretion was thought to be reserved, glimepiride (2 mg, o.p.d.) and sitagliptin (50 mg, o.p.d.) were administered, resulting in better glycemic control (HbA_{1c} 7.3–8.2%).

The extreme decrease in UCP levels may be associated with the ileal neobladder. During prolonged contact with the neobladder wall, UCP might be degraded by peptidases present at the brushing boarder of the ileum constituting the neobladder or by bacteria present in the neobladder on the background of a fragile defense system after urinary tract diversion (4) or high susceptibility in diabetic patients. Unlike peptides, e.g., C-peptide that consists of 31 amino acid residues (2), protein is probably not affected by the ileal neobladder, since the patient presented proteinuria (approximately 0.4 g/day). This is because, before peptides are degraded in the small intestine, protein needs to be digested into peptides by gastric acid, trypsin, and proteases in the stomach and duodenum. With recent technical advances in urological surgery, an ileal neobladder can be archived, which leaves only operation scars on the abdominal wall instead of cutaneous urinary stoma. In addition, the patient's quality of life is highly improved (3,4). However, as shown in this case, physicians must still pay careful attention not to overlook urinary tract diversion or neobladder. To confirm degradation of C-peptides in the ileal neobladder, it is necessary to compare the peptide/amino acid composition in the urine before and after neobladder passage or to demonstrate localization and activity of peptidases in the mucosa of an ileal neobladder. The present case demonstrates that UCP evaluation alone is not a reliable parameter in patients who underwent urinary tract diversion involving the small intestine.

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