Implications of Lower Urinary Tract Symptom Surveillance and Uroflowmetry Screening on Diabetic Bladder Dysfunction

Wei-Chia Lee, M.D.[1,2,3]

Division of Urology[1], Chang Gung Memorial Hospital Kaohsiung Medical Center, Kaohsiung, Taiwan; College of Medicine[2], Chang Gung University, Taoyuan, Taiwan; Graduate Institution of Clinical Medicine[3], College of Medicine, National Taiwan University, Taipei, Taiwan;

E-mail: dinor666@ms32.hinet.net

Type 2 diabetes mellitus has become an important worldwide public health problem because its prevalence has risen steadily during recent decades, especially in Asian and female populations [1]. Diabetic bladder dysfunction (DBD) is characterized by impaired bladder sensation, increased bladder capacity, decreased detrusor contractility, and an increased postvoid residual urine volume [2]. It is frequently not recognized by patients and physicians due to its insidious development and inconspicuous symptoms. Usually, genitourinary dysfunction in diabetic patients has reached an advanced stage by the time urologists are consulted. In addition to DBD, there is a greater incidence of asymptomatic and symptomatic bacteriuria, which can progress to kidney infection and kidney damage [3]. This increase in infection has been attributed to numerous causes, from incomplete bladder emptying to changes in bladder wall components and immune dysfunction. Another important question is whether bladder dysfunction is secondary to an inherent neuropathology induced by diabetes or caused by changes associated with bladder overdistension [3].

In our series of studies [4-6], we have demonstrated a high prevalence (22.2%) of unrecognized DBD in women with type 2 diabetes in the clinic setting [4]. Presentation of lower urinary tract symptoms is also increased in diabetic women, especially nocturia and weak stream [5]. DBD is associated with repeated urinary tract infections and peripheral neuropathy [4,6]. Uroflowmetry parameters were significantly altered in those who had DBD, including bladder voiding efficiency, maximum uroflow, postvoid residual volume and uroflow pattern [6].

Recent evidence has shown that the increased risk of an overactive bladder in diabetic patients is related to peripheral nerve irritation, increased bladder sensation and detrusor overactivity [7]. The presence of an overactive bladder in diabetic patients could be considered a sign of vesical neuropathy [6]. Diabetes induces autonomic nerve dysfunction and up-regulation of the M2 receptor in the bladder. Increased M2 receptors inhibit bladder relaxation and cause an overactive bladder. In addition, diabetes affects the sensory afferent pathway by reducing the nerve growth factor. In the bladder, a deficiency in the nerve growth factor results in increased bladder capacity and postvoid residual volume. Urinary retention and a low functional capacity cause frequency and nocturia. In observing our patients with DBD, we found there were few patients who had a bladder capacity larger than 500 mL. This suggests that overdistension of the bladder is not the dominant mechanism causing DBD.

Generally, diabetic patients evaluated with urodynamic studies are of advanced age and an overlap exists with other age-related urological diseases that have urodynamic consequences. Therefore, the diagnosis of DBD is most readily made with urodynamic testing. However, in the clinic setting, inquiring about lower urinary tract symptoms and a noninvasive determination of the urinary flow rate, combined with measurement of residual urine after voiding by ultrasound, appear to be feasible methods of ruling out bladder dysfunction in diabetic patients. These procedures may be useful in preventing long-term complications that are secondary to DBD. These concepts are particularly important for diabetic patients who have repeated urinary tract infections and neuropathy because there are neurological signs that are directed to DBD.

REFERENCES

1. Gu D, Reynolds K, Duan X, et al: Prevalence of diabetes and impaired fasting glucose in the Chinese adult population: International Collaborative Study of Cardiovascular Disease in Asia (InterASIA). Diabetologia 2003; **46**:1190-1198.
2. Sasaki K, Yoshimura N, Chancellor MB: Implications of diabetes mellitus in urology. Urol Clin North Am 2003; **30**:1-12.
3. Yoshimura N, Chancellor MB, Andersson KE, Christ GJ: Recent advance in understanding the biology of diabetes-associated bladder complications and novel therapy. BJU Int 2005; **95**:733-738.
4. Yu HJ, Lee WC, Liu SP, Tai TY, Wu HP, Chen J: Unrecognized voiding difficulty in female type 2 diabetic patients in the diabetes clinic: A prospective case-control study. Diabetes Care 2004; **27**: 988-989.
5. Lee WC, Wu HP, Tai TY, Liu SP, Chen J, Yu HJ: Effects of diabetes on female voiding behavior. J Urol 2004; **172**:989-992.
6. Lee WC, Wu CC, Wu HP, Tai TY: Lower urinary tract symptoms and uroflowmetry in women with type 2 diabetes mellitus with and without bladder dysfunction. Urology 2007; **69**:685-690.
7. Yamaguchi C, Sakakibara R, Uchiyama T, et al: Overactive bladder in diabetes: A peripheral or central mechanism? Neurourol Urodyn 2007; **26**:807-813.