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The effect of type 2 diabetes mellitus (DM) on bladder contractility in diabetic men

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***Abstract*---**Aims: To clarify the effect of DM type 2 on bladder contractility in type 2 diabetic patients. Methods: Patients (60 adult male patients) with lower urinary tract symptoms (LUTS) underwent a detailed urodynamic investigation. Urodynamic findings were classified as diabetic cystopathy [DC, characterized by impaired bladder sensation, increased post-void residual urine (PVR) and increased bladder capacity and decreased bladder contractility], detrusor overactivity. Results: All the patients in the diabetic group had type 2 diabetes mellitus for average 14 years and only 9/60 patients(15% of the diabetic group) has weak detrusor contractility their BCI less than 100. The rest of diabetic group 51/60 (85%) had good detrusor contractility. This means that diabetes (even with long duration>10 years) does not affect the detrusor contractility in our diabetic patients. 30 patients of the group (50%) had increased cystometric capacity (>450 CC),12 patients (20%) had reduced cystometric capacity (<250CC) while the rest of the patients(18 patients i.e. 30%) had average cystometric capacity(300 :450CC). 30 patients of the group (50%) had increased post voiding residual urine volume (PVR)(PVR up to 500 CC),while 30 patients of the group (50%)had normal PVR(<100CC). 15 patients (25%) of the group had delayed sensations while 45 patients (75%) had normal sensations. Uninhibited detrusor contractions were found in 36 patients of the group (60%).while 24 patients (40%) had no uninhibited detrusor contractions.

***Keywords*---**bladder dysfunction, diabetic cystopathy, type 2 diabetes mellitus, urodynamics.

Introduction

Bladder dysfunction (BD; diabetic cystopathy) in diabetes mellitus (DM) can occur in a number of ways, from impaired detrusor contractility to detrusor overactivity. Diabetic cystopathy (DC) as described by Frimodt-Moller in 2004 is mainly characterized by the steady increase in post-void residual urine (PVR) and in bladder capacity with diminished bladder sensation and decreased bladder contractility. It is a progressive condition that begins with the loss of viscerosensory innervation of the bladder which can be related to diabetic peripheral neuropathy[1]. The patients lose their ability to record the "desire to void" which usually occurs at the filling volume of 300–400 ml in healthy subjects[2] . Thus, micturition reflexes are delayed and an asymptomatic increase in bladder capacity and urinary retention occur [3]. DC likely represents end stage bladder failure. Presumably it is not the most prevalent type of BD in DM, because some clinical studies reported bladder overactivity as the most frequent finding, affecting 39–61% of diabetic patients. Diminished bladder contractility has been found less often [4].

DC is one of the urologic complications of DM. Further, there is a well-established correlation between the duration of DC and the presence and severity of peripheral neuropathy. The correlation between DC and diabetic nephropathy is poor and it is not found to be correlated with diabetic retinopathy [5.] In contrast to Goldman HB and Appell R A, Kebapci found that the presence of diabetic retinopathy increased the probability of BD via PVR. Since patients with developing PVR are prone to recurrent UTI and deterioration of the upper urinary tract, PVR is a key factor in differentiating between the incipient stage of DC and advanced stage [5] .

The diagnosis of DC was established according to the following criteria:

(1) Increased maximal bladder capacity (up to 500 ml);
(2) Impaired bladder sensation (volume at first desire to void > 150 ml);
(3) Decreased bladder contractility (a flat trace on cystometry);
(4) PVR exceeds 100 ml.[4].

A number of clinical studies have reported detrusor overactivity as the most frequent finding, ranging from39% to 61% of diabetic patients [6]. Detrusor overactivity can be also due to BOO or neurologic disease and moreover, it is common among elderly incontinent subjects. Starer et al. reported that 25% of diabetic patients with a history of cerebrovascular disease presented detrusor overactivity during cystometry. They examined elderly diabetic nursing home patients (mean age, 80 years) with previous cerebrovascular accidents. They reported that carbon dioxide cystometry revealed detrusor instability in 61% of patients with urinary incontinence, frequency, urgency, and nocturia[4]. In the absence of neurologic disease other than diabetic neuropathy, among patients with BD, we found 25% of men have isolated detrusor overactivity. Within the male population the most important differential diagnosis and the one that

frequently coexists with DC is BOO [4]. It is commonly related to BPE, but bladder neck stenosis or fibrosis can also be the cause of BOO. In the study conducted by kebapci et al in 2007, 25% of men with Bladder Dysfunction (BD) and 30% of men with DC presented BOO secondary to BPE. None of their patients had bladder neck stenosis or fibrosis. Diabetes primarily affects detrusor function by affecting bladder sensation, while BPE primarily affects bladder outlet resistance. Furthermore, experimental studies indicate that diabetes and obstruction affect different populations of visceral afferents supplying the bladder. Therefore, distinguishing classic DC from neural plasticity accompanying obstruction due to BPE is needed as a first step in improving the clinical approach to this condition [4]. In men, LUTS and BPE increase rapidly with age starting at about age 50 years. DM tends to occur in the older population, thus concurrent factors such as BPE may contribute to voiding dysfunction in these patients. Although DM is a well-defined disease, DC often develops insidiously and may be asymptomatic. Moreover, 25–50% of patients were found to have LUTS when they carefully asked [1].

The risk of BD increased with duration of diabetes because men with diabetes longer than 9 years. In other words DC established in at least 8–9 years after the diagnosis of type 2 DM which was an important parameter to inform our diabetic patients [4]. Clinically, the diagnosis of diabetic cystopathy is most readily made with urodynamic study [6]. The most common urodynamic findings include elevated residual urine volume, impaired bladder sensation, involuntary detrusor contractions, increased cystometric capacity and decreased bladder contractility. Cystometry may show detrusor areflexia, which is usually found in patients with an impaired sensation of bladder filling [1].Detrusor overactivity is also common in patients with DM. Other aspects of the severity of DM, e.g. duration, glycaemic control a, microvascular complications resulting in damage to innervation of the bladder, have been suggested as possible mechanisms for incontinence [4].

Patients and methods

This study was conducted on 60 male Diabetic patients in Urodynamics Unit, Urology Department, faculty of medicine, Cairo University from July 2017 till May 2019. 60 male patient aged from 40 years to 80 years were studied in urodynamics unit all of them had type 2 diabetes mellitus .each patient was evaluated through uroflowmetry, Ultrasound for prostate size and post voiding residual urine (PVR) ,PSA and IPSS Score then invasive urodynamics (pressure flow study) was done.

Invasive urodynamics (pressure flow study) was done for every patient: rectal pressure was assessed using rectal catheter connected to pressure transducer, intravesical pressure was assessed by introduction of urodynamics catheter 6 French (Fr) into urethra. The aim of the study to clarify the effect of DM on the urinary bladder during both filling phase (cystometric capacity ,sensations, presence of detrusor instabilities)and voiding phase (voiding pressure ,urine flow and post voiding residual urine volume(PVR) which was measured by ultrasound).

Statistical methods

Data were coded and entered using the statistical package for the Social Sciences (SPSS) version 25 (IBM Corp., Armonk, NY, USA). Data was summarized using mean, standard deviation, minimum and maximum in quantitative data and using frequency (count) and relative frequency (percentage) for categorical data. Urodynamic results were given as numbers.

Results

This study was conducted in Urodynamics Department in Urology department, faculty of medicine, Cairo University, from July 2017 to May 2019.
Sixty diabetic candidates were enrolled in the study; they were in diabetic for average 14 years. their age ranges from 40 to 70 years (mean=55 years ±12),they were diabetics for 4 to 27 years with mean 14(±5.7) years, their IPSS Score ranges from 3 to 25 with mean 10 (±5.7),their prostate size ranges from 22 to 95 grams with mean 54 (±20) grams, their PSA ranges 0.28 to 5 mean 3(±0.7) ng/ml, their free uroflow ranges from 6.6 ml/s to 22 ml/s with mean 14 (±4.6) ml/s.

In invasive urodynamics study, their cystometric capacity ranges from 175 cc to 500 cc with average 379 (±107) cc, their Pdet. at Q max ranges from 30 cm H2O to 175 CmH2O with average 78 (±34)cm H2O, their bladder contractility index(BCI) ranges from 70 to 250 with average 81(±34). All the patients in the diabetic group had type 2 diabetes mellitus for average 14 years and only 9/60 patients(15% of the diabetic group) has weak detrusor contractility their BCI less than 100 . The rest of diabetic group 51/60 (85%) had good detrusor contractility. This means that diabetes (even with long duration>10 years) does not affect the detrusor contractility in our diabetic patients.

30 patients of the group (50%) had increased cystometric capacity (>450 CC),12 patients (20%) had reduced cystometric capacity (<250CC) while the rest of the patients(18 patients i.e. 30%) had average cystometric capacity(300 :450CC). 30 patients of the group (50%) had increased post voiding residual urine volume (PVR)(PVR up to 500 CC),while 30 patients of the group (50%)had normal PVR(<100CC). 15 patients (25%) of the group had delayed sensations while 45 patients (75%) had normal sensations. Uninhibited detrusor contractions were found in 36 patients of the group (60%).while 24 patients (40%) had no uninhibited detrusor contractions.

Discussion

In the previous studies, according to the diagnostic methods, criteria, and the patient characteristics, the frequency of DC varies from 25% to 80% .Frimodt-Moller et al. first reported detailed characteristics of bladder dysfunction in diabetic patients. They characterized DC as loss of sensation detected by elevated bladder perception threshold which was found in 38% of patients [2]. According to the criteria proposed by Kahan et al. which defined DC as an increase in bladder capacity to more than 400 ml with a flat trace on cystometry, 36% of diabetic

patients had DC. Using the criterion that bladder capacity exceeding 500 ml was abnormal; Ueda et al. reported that 32% of diabetic patients had DC [7].

A number of clinical studies have reported detrusor overactivity as the most frequent finding, ranging from39% to 61% of diabetic patients. Detrusor overactivity can be due to BOO or neurologic disease and moreover, it is common among elderly incontinent subjects [6,8],in another study conducted by Starer et al. they reported that 25% of diabetic patients with a history of cerebrovascular disease presented detrusor overactivity during cystometry. They examined elderly diabetic nursing home patients (mean age, 80 years) with previous cerebrovascular accidents. They reported that cystometry revealed detrusor instability in 61% of patients with urinary incontinence, frequency, urgency, and nocturia. In the absence of neurologic disease other than diabetic neuropathy, among patients with BD, we found 25% of men had isolated detrusor overactivity[9].

In men, LUTS and BPE increase rapidly with age starting at about age 50 years. DM tends to occur in the older population, thus concurrent factors such as BPE may contribute to voiding dysfunction in these patients. Although DM is a well-defined disease, DC often develops insidiously and may be asymptomatic.
In our group, detrusor overactivity was the most frequent urodynamic finding (60%) of Bladder Dysfunction. While diabetic cystopathy (increase cystometric capacity) was present in in 50%, delayed bladder sensations found in 25% in our patients.

Although Kebapci et al found that the risk of Bladder Dysfunction (BD) increased in men with diabetes longer than 9 years [4] we found our patients with average duration of DM of 14 years, weak detrusor contractility was found in 15% only of our patients this means that DM even with long period of DM do not affect the detrusor contractility of all of our patients. Since patients with developing PVR are at higher risk to have recurrent UTI and damage to the kidneys, PVR is a key factor in differentiating between the silent stage of DC and advanced stage [10] we found that PVR more than 100 ml was found in 50% of our patients .this percentage are at risk of recurrent UTI and upper tract damage.

Conclusions

In our study the most common Bladder dysfunction (BD) among patients with type 2 DM was detrusor overactivity (was found in 60% of our patients). While diabetic cystopathy (increase cystometric capacity) was present in in 50%, delayed bladder sensations found in 25% in our patients.

The risk of BD increased with duration of diabetes because men with diabetes longer than 9 years. i.e. DC established in at least 9 years after the diagnosis of type 2 DM which was an important parameter to inform our diabetic patients. In our patients the average duration of DM in our patients was 14 years and this long duration affected PVR (50% of our patient had elevated PVR >100 CC) however weak detrusor contractility was found in 15% only of our patients this means that DM even with long period of DM do not affect the detrusor

contractility of all of our patients DC. Characterization of BD was only made using pressure flow study.

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