INTRODUCTION

Diabetic bladder dysfunction is a very common lower urinary tract pathology, associated with diabetes mellitus. This dysfunction occurred in almost 80% of the patients. Diabetes mellitus is caused by defects in the secretion of the hormone insulin by the pancreas and/or dysregulation of cellular glucose uptake. Glucose is a large hydrophilic molecule. The cellular entry is connected with specific glucose transporter proteins, present on the cell membrane. Therefore Type 1 and 2 diabetics are unable to efficiently transport glucose from the blood into the cell which leads to a state of hyperglycemia. Chronic elevation in cytosolic glucose leads to metabolic abnormalities such as osmotic and oxidative stress. It is believed that those factors cause tissue injury and dysfunction associated with long-term diabetes mellitus.

The urinary bladder is one of the most organs, affected by diabetes mellitus. Many events or conditions can damage nerves that control bladder function, including diabetes and other diseases, injuries, and infections. Alterations in bladder detrusor and innervation have also been reported in diabetes mellitus patients. More than half of men and women with diabetes have bladder dysfunction. Common bladder problems in men and women with diabetes include the following symptoms - urine retention, overactive bladder, poor control of sphincter muscles, urinary tract infections. Diabetes mellitus affects both storage and voiding functions and is characterized by a broad spectrum of symptoms including retention, urgency, frequency, nocturia, and incontinence.

In many cases, in men with LUTS it is difficult to determine whether the symptoms are due to diabetic cystopathy, BPH obstruction or both. Diagnosis is of great importance for selecting therapeutic approach.

PATHOGENESIS

In the first stages of the disease diabetes leads to increase of the osmotic diuresis. The bladder adapts to polyuria by compensatory, increased contractility, but
subsequently decompensates due to the direct effects of chronic systemic hyperglycemia on bladder tissues. The changes in urinary bladder, such as hypertrophy, increased contractility and neurogenic disorders, occur soon after the onset of diabetes mellitus. The decreased peak voiding pressure, evidenced in urodynamic measurements, develops only at a later stage of diabetes mellitus. The factors that contribute to the occurrence of diabetic cystopathy are as follows: the damage on detrusor smooth muscle, bladder vasculature, innervation and the disturbance of the urothelial function.

**URINARY DYSFUNCTION IN MEN**

In men, lower urinary tract symptoms (LUTS) are common, age-related complaints that are often attributed to benign prostatic hyperplasia (BPH). LUTS and BPH increase rapidly with age starting at about age 50 years. Straining, intermittency, postvoid dribbling and weak stream may signify urethral obstruction from BPH. However, among men with diabetes, similar symptoms may also result from bladder dysfunction due to denervation and poor detrusor contractility. Other complex associations of LUTS and BPH among diabetic men include symptoms of urgency, frequency and nocturia that may occur from detrusor overactivity, resulting from BPH, and/or microvascular complications associated with diabetes, increasing hyperactivity of the detrusor. The failure to differentiate LUTS from BPH in studies of diabetic men has contributed to the confusing evidence now seen in the literature. Recent evidence suggests that LUTS may occur more frequently among men with diabetes, with an estimated 25% to nearly twofold increased risk of LUTS in men with diabetes. Additionally, among men with BPH, diabetes is associated with more LUTS symptoms compared with nondiabetic men. Additionally, among men with BPH, diabetes is associated with more LUTS symptoms compared with nondiabetic men.

Physiological, microvascular, and neurological complications of diabetes result in changes that may impair the function of continent mechanisms. That includes damage to the innervation of the bladder, altered detrusor muscle function or urothelial dysfunction. However, despite mounting evidence of a link between diabetes and incontinence, not so much is known about the mechanisms by which diabetes leads to incontinence.

Urodynamics studies on patients with diabetes mellitus and LUTS. (Initial experience)

In a period of 10 months, 08.2013 - 05.2014 we performed uroflowmetry, cystotonometry with electromyography and pressure/flow measurement of 14 patients (men) with LUTS and diabetes mellitus.

In this study the following criteria were taken into consideration:
- LUTS with IPSS > 8, and Q of L >2
- sterile urine examination
- PSA < 4ng/ml
- prostate rectal examination aimed to exclude prostate cancer
- transrectal ultrasound of the prostate gland >25cm³
- measurement of residual urine (without limitation) results 0 - 60 ml

**RESULTS**

The patients were between the age of 50 - 77. In 2 patients urethral catheter was affixed due to acute urine retention, respectively for a period of 1 and 3 months. Both patients were treated with α-blocker/prostate volume < 25sm³. The other patients were not treated for LUTS. Four of the patients were with insulin dependent diabetes mellitus, ten of the patient were treated for diabetes mellitus by per oral therapy, half of them took more than one medication for treatment of the diabetes mellitus.

Cystotonometry
- Detrusor overactivity without bladder outlet obstruction (two patients). Treatment: M cholinolitic
- Detrusor overactivity and urge incontinence without bladder outlet obstruction (one patients). Treatment: M cholinolitic

Pressure-flow study
- Normal detrusor activity PubMed 14 patients
- Pressure-flow study
  1. BOOI < 20, Q max > 15 ml/sec (8 patients). Treatment: watchful waiting
  2. BOOI 20-40, Q max > 12 ml/s (4 patients). Treatment: a - blocker
  3. BOOI > 40, Q max < 10ml/s (2 patients ). Treatment: TURP (bipolar resection)

**CONCLUSION**

We consider that recognition of urinary incontinence as an important medical problem remains an issue. Diabetes and diabetic bladder dysfunction will rapidly increase with population aging. Physicians should be alert for diabetes cystopathy, because it is often unrecognized and therefore under-treated among men with diabetes.

Future research is needed to identify the risk factors, mechanisms, and most effective treatment of this disorder.
FIGURE 2. UROFLOWMETRY IN PATIENT WITH BPH AND DIABETES MELLITUS

FIGURE 3. PRESSURE FLOW STUDY IN PATIENT WITH BPH AND DIABETES MELLITUS
SUMMARY

Uvod: Dijabetesna disfunkcija mokraćne bešike je veoma učestao problem bolesnika sa dijabetesom. Ova disfunkcija se manifestuje kod 80% bolesnika sa dijabetes melitusom. Učestali simptomi bolesnika podrazumijevaju retenciju, "overactive bladder" skup simptoma, lošu kontrolu sfinktera kao i infekcije urinarnog trakta.

Materijal i metode: U periodu od deset meseci 08.2013.-05.2014. godine radjena je urofloumetrija, cistotonometrija sa elek tromiografijom i pritisak protok merenje na 14 bolesnika sa simptomima donjeg urinarnog trakta i dijabetes melitusom. Kriterijumi uključenja su bili IPSS veće od 8, PSA manji od 4, RT isključen carcinom prostate i veličina prostate veća od 25 cm³.

Rezultati: Bolesnici su bili starosti od 50 do 77 godina. Kod dva bolesnika zbog akutne retencije je postavljen kateter. Tretirani su alfa blokatorima dok ostali bolesnici nisu primali dodatnu terapiju. Četiri bolesnika su imala insulin zavisni dijabetes okolona otnošenje koje je njih deset imalo insulin tretiran oralnim antidiabeticima. Cistotonometrija je pokazala kod dva bolesnika prekomernu detrusorsku aktivnost koja je tretirana M holinoliticima. Kod jednog bolesnika prekomerna detrusorska aktivnost bila je udužena sa ugroženom kontinencijom i tretirana na isti način. Normalna detrusorska aktivnost je zabeležena kod 14 bolesnika na pritisak protok merenju. U 8 bolesnika sa Qmax većim od 15 ml/s tretman je podrazumijevao pravčenje. Kod 4 bolesnika sa Q max većim od 12 ml/s uvedeni su alfa blokatori dok je kod 2 bolesnika sa Qmax manjim od 10 ml/s uradjen bipolarni TURP.

Zaključak: Dijabetes i dijabetesna disfunkcija mokraćne bešike će prati vremenom s obzirom na stariću populaciju. Potrebno je upoznati lekare svih specijalnosti sa dijabetičnom disfunkcijom bešike jer je često neprepoznata i ostaje bez tretmana. Dalja istraživanja u smislu utvrđivanja faktora rizika, mehanizama i najadekvatnije terapije u tim uslovima.

Ključne reči: diabetes melitus, mokraćna bešika, disfunkcija bešike

BIBLIOGRAPHY


