Editorial referring to the paper published in this issue on pp. 366– 374

FUNCTIONAL UROLOGY

Not only diabetic polyneuropathy but also interstitial cells of Cajal dysfunction is a potential mechanism for diabetic cystopathy

Kajetan Juszczak[1,2]

[1]Department of Urology, Rydygier Memorial Hospital, Cracow, Poland
[2]Department of Pathophysiology, Jagiellonian University, Medical College, Cracow, Poland

Canda et al. [1] focused on the changes of interstitial cells of Cajal (ICCs) and neuronal tissue related to diabetes. The results of the current study showed that the number of ICCs in the lamina propria and detrusor muscle is decreased in diabetic patients. This may be an underlying pathomechanism of lower urinary tract symptoms in patients with diabetes. Long lasting diabetes results in different urological complications in over 50% of cases. The most common complications include diabetic bladder dysfunction (DBD), diabetic cystopathy, urinary tract infections and sexual dysfunction. Diabetic cystopathy (DC) was first described by Moller in 1976. DC features consist of an increased urinary bladder capacity, post voiding residual volumes and decreased urinary bladder sensation and motor activity as a result of diabetic polyneuropathy. DBD is referred to a wide range of lower urinary tract symptoms that encompass storage complaints (overactive bladder/detrusor overactivity) and voiding complaints (poor urinary bladder emptying or overflow urine incontinence), as well as other less clinically defined phenotypes such as decreased sensation and increased capacity [2–5].

Past decades have shown an increasing appreciation for the important role of ICCs in gastrointestinal tract motility. Moreover, there is strong evidence of a correlation between some gastrointestinal diseases (e.g. achalasia, diabetic gastroenteropathy, gastro–esophageal reflux disease, gastroparesis, Hirschsprung's disease, etc.) and morphological and functional alterations of ICCs. Multifunctional activity of ICCs has also been described. It is well known that these cells contribute to several functions as follows: 1) pacemaker activity – the generation of electrical potential and its propagation, 2) transduction of efferent inputs from the enteric nervous system, and 3) mechanosensation [6].

Nowadays, considerable progress has been observed in describing the morphology, distribution and physiological properties of ICCs in the urinary bladder. ICCs lie in close proximity to the muscle cells, autonomic nerve endings and urothelial cells. There is increasing evidence that ICCs play a role in urinary tract dysfunction development (e.g. detrusor overactivity, primary obstructive megaureter, congenital ureteropelvic junction obstruction, etc.). ICCs in the urinary bladder are believed to be potent transducers of signals between the autonomic nerve endings and detrusor muscles. These cells may be responsible for generating electrical potentials and for induction of detrusor muscle contractions [7]. Diabetes affects the structure and function of the urinary bladder. Pathological alterations occur in muscles, nerves, urothelial cells and ICCs.

Understanding the nature of the relationship between ICCs and diabetic cystopathy, as well as the underlying mechanism for loss of ICCs is still in progress. Future studies should be performed to better understand these topics, which will in turn clarify the importance of these cells and elucidate the basis of diabetic cystopathy.

10.5173/ceju.2014.04.art11

References

1. Canda AE, Dogan H, Kandemir O, Atmaca AF, Akbulut Z, Balbay MD. Does diabetes affect distribution and number of intestitial cells and neuronal tissue in the ureter, bladder, prostate and urethra of humans? Cent European J Urol. 2014; 67: 366–374.
2. Moller CF. Diabetic cystopathy I: A clinical study of the frequency of bladder dysfunction in diabetics. Dan Med Bull. 1976; 23: 267–278.
3. Daneshgari F, Liu G, Birder L, Hanna–Mitchell AT, Chacko S. Diabetic bladder dysfunction: current translational knowledge. J Urol. 2009; 182: S18–26.
4. Hill SR, Fayyad AM, Jones GR. Diabetes mellitus and female lower urinary tract symptoms: a review. Neurourol Urodyn. 2008; 27: 362–367.
5. Liu G, Daneshgari F. Diabetic bladder dysfunction. Chin Med J. 2014; 127: 1357–1364.
6. Yin J, Chen JD. Roles of interstitial cells of Cajal in regulating gastrointestinal motility: In vitro versus in vivo studies. J Cell Mol Med. 2008; 12: 1118–1129.
7. Juszczak K, Maciukiewicz P, Drewa T, Thor PJ. Cajal–like interstitial cells as a novel target in detrusor overactivity treatment: true or myth? Cent Eur J Urol. 2013; 66: 413–417.

Correspondence
Kajetan Juszczak, FEBU, M.D., Ph.D.
kajus13@poczta.onet.pl