Editorial Comments — Management of OAB

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Overactive bladder (OAB) is a disorder of the lower urinary tract; it is characterized by urgency, either with (OAB wet) or without (OAB dry) urge incontinence and is usually associated with frequency and nocturia. OAB affects approximated 16% of the population of the United States and Europe. In addition, 53% of Asia women have symptoms of OAB. The symptoms of OAB are not life threatening, but limit physical, social and emotional functioning among both women and men.

Pharmacological treatment with muscarinic receptor antagonists has been the main stay of therapy for patients suffering from this syndrome. It is widely known that the efficacy of these drugs is mediated through blockade of the muscarinic receptors in the detrusor muscle and the urothelium. Historically, the pharmacological site of action for these drugs has ignored the impact of any localized effect on the bladder urothelium. Evidence shows that muscarinic mechanisms are also involved in bladder sensory function, which supports the idea of a localized pharmacological action on the bladder urothelium.

The use of antimuscarinic agents has been expanded from OAB to lower urinary tract symptoms (LUTS) related to benign prostatic hyperplasia (BPH). Recent studies suggest that antimuscarinic agents do not compromise voiding function; furthermore, combined antimuscarinic and alpha-blocking agents provide a better outcome than a single therapy approach when treating patients with LUTS and BPH. Antimuscarinic agents are not always effective with OAB patients and therefore other treatment methods are also used. These include intravesical instillation of vanilloid agents, botulinum toxin injection, neuromodulation and surgery. These approaches have proved to be quite effective among OAB patients who are refractory to antimuscarinic therapy.

Activation of the C afferent fibers is known to play a role in OAB. Desensitization of the C afferent fibers through intravesical instillation of vanilloids such as capsaicin and resiniferatoxin, has proved to be an effective therapy for OAB. Botulinum Neurotoxin A (BoNT-A) is a well known agent for the treatment of muscular hypercontractility. Recently, there has been increasing evidence to support the idea that BoNT-A might also inhibit afferent neurotransmission and have analgesic/anti-inflammatory properties. These latter properties would seem to be independent of BoNT-A's effects on the neuromuscular junction. Such a possible application of BoNT-A to the treatment of OAB have been supported by both preclinical and clinical studies. Neuromodulation modulates the abnormal neural circuitry that controls lower urinary tract function in OAB. Although it is currently unpopular in Taiwan, Western experience in this area might open another window for OAB treatment in Taiwan.

The concepts associated with the various treatment approaches available for OAB are exemplified by five articles in this issue of the journal. These include the effects of antimuscarinics on voiding function, the role of antimuscarinics on neurogenic OAB, the rationale for a combined use of antimuscarinics and alpha blockers, clinical experience treating OAB in Hualien, and treatments available for patients who are refractory to antimuscarinic agents.

I would like to applaud all authors, who should be encouraged to create a wide ranging review for all current concepts on the treatment of OAB.

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