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Continued overleaf

Lower urinary tract symptoms and urinary incontinence

Introduction

Lower urinary tract symptoms (LUTS) is a global term which includes symptoms caused by failure to store urine and empty the bladder, and post-micturition symptoms. The term LUTS was initially coined in 1994. Before that urinary symptoms were frequently attributed to a particular organ such as the prostate [1]. Since then organocentric terms such as 'prostatism' have become increasingly obsolete and the International Continence Society (ICS) in 2002 formally classified LUTS into storage, voiding and post-micturition symptoms [2]. This approach acknowledges the long held notion that "the bladder is an unreliable witness" [3] and relies instead upon a more descriptive approach, with no underlying aetiology or specific organ assumed to be responsible for the symptoms experienced.

Storage LUTS

Storage LUTS include urinary incontinence, urinary urgency, urinary frequency and nocturia. These tend to be more bothersome and socially unacceptable than voiding or post-micturition LUTS [4]. Overactive bladder (OAB) syndrome is a condition which encompasses these storage symptoms. It is defined by the presence of urinary urgency, and frequently associated with detrusor overactivity (DO) on urodynamic investigation [2]. Storage symptoms are also often associated with urinary tract infection, bladder carcinoma or bladder stones, and these should be excluded before starting treatment for LUTS [5].

The move away from an organcentric approach to LUTS recognises that systemic, metabolic or hormonal conditions as well as cardiac and respiratory disease may lead to LUTS. Nocturia in particular is frequently attributable to such conditions [6], and should be borne in mind when assessing patients with storage symptoms. A simple frequency/volume chart is usually sufficient to make the diagnosis of nocturnal polyuria (more than 33% of the 24-hour urinary output being produced at night). In men the traditional assumption was that storage symptoms such as frequency and nocturia were prostatic in origin whereas similar symptoms in women were thought to be related to bladder dysfunction; but surveys suggest that men are as likely as women to suffer with OAB.

The recently reported EPIC study assessed the prevalence of LUTS in more than 19 000 adults in 5 countries [7]. It showed that OAB was present equally in both sexes with an overall prevalence of 11.8%, and that the prevalence of all LUTS increased markedly with age. Surprisingly, storage

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symptoms were found to be twice as common as voiding symptoms in men (storage = 51.3% and voiding = 25.7%). Yet current treatment strategies for male LUTS tends to focus on the prostate and bladder outlet. These findings have implications regarding the management of LUTS in men, and there is growing interest in the use of therapies which were previously predominantly used to control storage symptoms only in women. Anticholinergics for example have been avoided in the treatment of male LUTS because of a presumed risk of acute urinary retention. However, a large proportion of men with LUTS may benefit from them, and research suggests that there is little increase in the risk of developing acute urinary retention with anticholinergics [8].

Urinary incontinence

Urinary incontinence remains a socially stigmatised condition, yet the prevalence is extremely high; 35% of respondents in a survey of 17 000 women reported some incontinence in the previous month [9]. Lower urinary tract dysfunctions such as OAB or a weakened pelvic floor are much more likely to result in incontinence in women as a result of weaker sphincteric mechanisms and the possibility of damage to them by childbirth. An increasing number of men also present nowadays with urinary incontinence owing to the increasing use of radical prostatectomy to treat organ confined prostate cancer [10]. Urinary incontinence is usually classified as either stress urinary incontinence (SUI) where leakage occurs with increases in intra-abdominal pressure, or urgency urinary incontinence (UUI) with leakage preceded by urinary urgency; any condition that weakens the sphincteric mechanisms or pelvic floor will make either condition more apparent. Mixed urinary incontinence (MUI) is where both SUI and UUI co-exist.

Highly effective treatments, with reported efficacies of about 90%, are now available for both forms of urinary incontinence. The development of minimally invasive tape and sling procedures in the last decade has revolutionised the treatment of SUI [11], and although not licensed for urological use yet, botulinum toxin injections to the bladder is causing a similar revolution in the treatment of refractory UUI [12]. They have resulted in a dramatic decrease in the number of open procedures used to treat urinary incontinence, akin to the effect that medications such as alpha-antagonists and 5-alpha reductase inhibitors have had in reducing the number of transurethral prostatectomies performed for BOO secondary to benign prostatic hyperplasia (BPH).

Voiding LUTS

In the past, voiding symptoms were almost synonymous with the prostate, and more recently with bladder outlet obstruction (BOO). These symptoms include hesitancy, intermittency, slow stream, splitting or spraying stream, straining to void and terminal dribbling. As with storage symptoms there is a move away from an assumed aetiology, for identical symptoms may occur in urethral pathology, eg. strictures, and bladder pathology, eg. poor contractility of the bladder muscle (detrusor failure). The EPIC study confirmed that voiding symptoms have a similar prevalence in both sexes (males = 25.7% and females = 19.5%), confirming that the prostate is not the only possible source of voiding symptoms! [7].

In a large proportion of patients BOO obstruction is the cause of the symptoms experienced, but the 'art' of the diagnosis is in determining which patients do not have simple BOO, and who would benefit from specific therapies. 5-alpha reductase inhibitors or combination therapy will reduce the

size of the prostate, and not only improve symptoms, but will also decrease the risk of developing complications such as acute urinary retention, or the need for prostatic surgery [13]. Only a thorough urological history coupled with symptom score assessment, clinical examination including digital rectal examination, and objective assessments such as uroflowmetry and PSA testing will allow the clinician to determine patients who are at risk of disease progression and those in whom treatment of voiding symptoms alone will be insufficient.

It is misleading to attribute individual symptoms to either sex differences or to a single underlying organ. It is more rational to focus on the presence and severity of symptoms, and to base the therapeutic approach on patients' perceptions, expectations and goals for therapy. We must recognise that symptoms often correlate poorly with objective findings during urodynamic assessments, especially with regard to voiding symptoms. Storage symptoms (OAB) demonstrate a far better association with urodynamically proven detrusor overactivity. Urodynamics are necessary to differentiate between SUI, UUI and MUI. The poor correlation of symptoms with the underlying aetiology emphasises the need to perform objective assessments such as pressure flow urodynamics when invasive treatments are being contemplated following failure of medical management of LUTS, or when there is a doubt regarding the diagnosis.

By challenging the conventional paradigm regarding the aetiology of LUTS in men and women, we will be able to understand better the complex nature of lower urinary tract pathophysiology and to address patients' needs [5], and looking for potential underlying pathophysiological conditions – an approach facilitated by cluster analysis [14,15]. There is now increasing evidence that the metabolic syndrome, associated with obesity and diabetes, may be an important factor in the genesis of LUTS in the male, and the common coexistence of erectile dysfunction [5]. And many clinicians are moving to the use of combinations of therapies such as anticholinergics (traditionally used for overactive bladder dysfunction) with alpha-antagonists (traditionally used for prostate related BOO) [16]. Phosphodiesterase type-5 inhibitors (used in the treatment of erectile dysfunction) have also been shown to improve voiding LUTS [17].

With highly effective medical or minimally invasive treatments being developed to treat LUTS, the incidence of surgery for prostatic and bladder dysfunction has declined dramatically, but there is a continuing need to develop more effective treatments, since these conditions will increase with ageing of the population. Botulinum toxin appears to have a remarkable effect on OAB and DO, and it has also been used in other lower urinary tract conditions such as BPH and painful bladder syndrome. It is likely that novel treatments will lead to further improvement in the management of the highly prevalent and bothersome LUTS.

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Description of angina pectoris, 1926

By angina pectoris I mean an arresting form of cardiac pain, substernal rather than submammary, in which the patient is conscious of a sense of oppression or constriction which may reach a high grade of intensity, up to intolerable anguish. This pain has well-recognised lines of radiation, and is often induced by effort or emotion. There are in addition characteristic associated symptoms not invariably present, notably the sense of impending death and of acute misery accompanied by varying vasomotor disorders such as cold sweats and deathly pallor. These attacks are the response of an inadequate heart to stress, due either to physical effort or emotion. Another potent factor is an exaltation or over-sensitiveness of the central nervous system consequent on prolonged worry, anxiety, or loss of sleep.

John Hay, DL, MD, FRCP (Lond) *Professor of Medicine, Liverpool University: Senior Physician, Royal Infirmary, Liverpool.* In: Modern Technique in Treatment, Published by *The Lancet,* 1926. Volume 2; Page 81.