EDITORIAL

Solving the LUTS/BPH puzzle

I recently took a three-month sabbatical. Sabbatical is perhaps the wrong word as I fitted in some cycling in France! Most of the time, however, I worked at an NHS hospital in Nottingham, UK.

I was given what, at the outset, appeared a lowly job as an "office urologist", a term in the UK which has been replaced by "diagnostic urologist". Colleagues prepared to do this "sub-specialty" are apparently, for obvious reasons, greatly sought after! Yet, despite the basic nature of the work, I'm embarrassed to admit I learned a great deal from doing the work for two months. The UK was emerging from COVID-19 and there was a cabinet full of GP referrals that had not been attended to! Dealing with this endless stream of referrals was my job.

It's estimated that 41% of men over 50 years in the UK report moderate to severe lower urinary tract symptoms (LUTS).[1] Thus, unsurprisingly, most referrals were for benign prostatic hyperplasia (BPH). I imagined there was nothing I could learn about this bread-and-butter condition. Was I wrong!

Even in the UK there is a great unmet need for surgical management of BPH. Waiting lists were estimated at 9–12 months for a transurethral resection of the prostate (TURP). Thus, part of my role was to assess, stratify and hold most on medical treatment.

I encountered two types of patients in the UK which had an impact on my thinking. Firstly, I was struck by how many men had intractable storage LUTS. Their lives appeared a misery. I'm embarrassed to admit, I may not have perceived the extent of misery in the men I treat at Groote Schuur Hospital.

One metric of this misery is insomnia resulting from nocturia. Speakman et al. have shown that 40% of men with BPH suffer from insomnia, compared to 19% in the general population ($p < 0.001$). Additionally, they have poorer sleep efficiency.[1] Furthermore, LUTS impacts the quality of life of men's partners. Forty-two per cent of partners were tired because of being woken at night and 66% had noticed a worsening sex life.

Most men I encountered were already on combination therapy with many also on mirabegron or solifenacin – all started by the GP, so I often didn't have much value to add. Furthermore, a review of the literature reminded me how little improvement these additional medications add.

A meta-analysis of the addition of solifenacin to alpha-blockers, showed no statistically significant difference in the International Prostate Symptom Score (IPSS), micturition number or urge episodes per 24 hours.[2] Cochrane reviews of the addition of PD5I or desmopressin equally showed very tepid or no impact at all on symptoms.[3,4] Desmopressin is also now being considered a potentially inappropriate medication according to the Beers criteria for medications in older adults.

The second group of patients to impact my thinking was far smaller and should not have alarmed me, but did! These men presented with minimal or no LUTS yet had a 700 ml residual and hydronephrosis!

These men with minimal bother, yet in big trouble, led me to the pioneering work of a urological surgeon from Singapore, Professor Foo.

At medical school, Foo reminds us, we are taught to treat the disease, and not the symptoms. Yet the American Urological Association (AUA) and European Association of Urology (EAU) guidelines still manage LUTS/BPH according to symptoms (IPSS). Likewise, symptom response (IPSS) is the marker of treatment success.[5]

It is true that we mistakenly equate BPH with LUTS. This leads to undertreatment in some with severe bladder outlet obstruction (BOO) with no symptoms and overtreatment in patients with LUTS but no clinical BPH.

Foo introduces the notion of "clinical BPH" – defined simply as a prostate adenoma causing BOO and potentially harming the bladder and kidneys.

Clinical BPH should be more important than symptoms (IPSS) and be given more weight in decision making. Foo also introduces a staging and grading classification of severity, similar to a malignancy.[5] Grading relies on non-invasive ultrasound to measure prostate size and the novel concept of "intravesical prostatic protrusion" (IPP).

IPP introduces another of Foo's insights. He maintains that a small adenoma at the bladder neck (BN) (middle lobe) can cause significant BOO due to distortion of the funnelling effect of the normal BN. By contrast, lateral lobes can grow to a large size before causing BOO by compression. In flow dynamics, compression is less obstructive than distortion.[5]

In conclusion, Foo, in my view, greatly develops our understanding by introducing the notion of *clinical BPH*. It allows a stratification that does not focus on symptoms alone. This novel approach would allow the asymptomatic men I referred to earlier, to be flagged and managed more aggressively.

Lastly, the unmet need for surgical management of clinical BPH demands attention from our community. It demands we strategise how the TURP backlog can be addressed.

One area of promise to help solve this backlog comes from technology. Minimally invasive surgical treatments (MISTs) have recently become available, potentially offering a quick office procedure for clinical BPH. I hope we can devote future space in African Urology to discussing water vapour ablation (Rezum), UroLift and prostate artery embolisation.

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