Urinary Retention in Women

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Summary

The pathophysiology and epidemiology of urinary retention in women is not well documented. According to the current knowledge the aetiology of this condition appears to be multifactorial. Implicating factors vary and often coexist. They are mainly distinguished in infective, neoplastic, pharmacological, neurological, anatomical, myopathic, functional and psychogenic. Initial management includes bladder drainage if urinary retention is acute or if it is chronic accompanied by obstructive uropathy, infection or pain. Investigations should be initially focused on identifying actual complications and secondly the underlying aetiology.

Keywords

Urinary retention, womenIntroduction Urinary retention (UR) is defined as the inability to achieve full bladder drainage by voluntary micturition and is classified into acute (complete inability to drain) and chronic (incomplete with post-void residual volume). The clinical features of Acute Urinary Retention (AUR) are clearly contrary to those of Chronic Urinary Retention (CUR). Moreover, the absence of a well-defined normal range for the clinical post-void residual volume, the diagnostic setting (ultrasound) and the bladder capacity differentiations make CUR difficult to identify[1].

While UR commonly affects men and especially in older ages, it is relatively unusual in women. In fact, the epidemiology of female UR is not well-documented and its pathophysiology remains unclarified. Indeed, for male patients and especially the elderly, UR is mainly caused by Bladder Outlet Obstruction (BOO) induced by Prostatic Hyperplasia[2] (PH). Women on the contrary, especially in older ages, present a number o various pathological conditions that may contribute to the development of UR such as decreased bladder contractility, poorly sustained detrusor contraction, lack of adequate anatomical outlet or neurological disorder impairing the coordination of the urination process. Thusly, a considerable rate of older women present cysteoscopic findings of urinary obstruction like trabeculitis and bladder diverticulitis non-accompanied by a considerable amount of post-void residual volume; a smaller rate of patients manifest post-void residual volume non-accompanied by obstructive indications. In particular, the presence of a clear anatomical impediment does not constitute a common finding though obstruction symptoms, such as poor or intermittent urinary flow, terminal dribbling or post-micturition dribbling, are comm-

only reported by female patients with UR.

Materials and Methods

Studies published in 1980 onwards were examined via search on MEDLINE, National Library of Medicine and Iatrotek databases. Our initial keywords were: urine retention, woman in combination with: epidemiology, risk factor, treatment. The references in the selected articles were investigated for publications not included in the initial search on the databases.

Results

1. Epidemiological data

The impact of female UR is not well-documented and most information comes from a small case series or case reports[3]. Indicatively and for AUR only, Klarskov et al. conducted a very small prospective study and estimated the annual impact at 7/100.000 whereas its ratio compared to male AUR was estimated at 1:13[4]. Garcva-Fadrique et al. performed a urodynamic study on 202 female subjects exhibiting AUR. Only 28.7% reported urination disorders; the rest 71.3 % had no symptoms. However, only 31.7% had normal urodynamic findings contrary to 32.2% exhibiting decreased detrusor contractility. Complete absence of detrusor contractility was manifested in 18.3%, BOO in 10.4% and decreased pelvic floor relaxation in 7.4% of the cases. Notably, in almost half of all-cause cases (52.4%), normal urination was completely restored and no treatment plan was applied[5]. The retention was finally attributed to neurological causes in 37.1% (10.9% of which was associated to diabetes mellitus), gynecological causes in 9.4% and urological causes in 7.9%. 22.8% of the cases sustained a mixed aetiology and in the rest 22.8% no cause was determined[5]. A respective ratio of neurological and non-urological UR is examined in another small study but a considerable rate of the latter was attributed to psychological causes[6]. Interestingly, studies before 1980-90, when Fowler's syndrome was established, regard urethral and bladder cervix deformities as the main causes of female UR. On the contrary, younger females encounter Fowler's syndrome. For approximately one third of the cases, the cause remains undetected[7].

2. Aetiology

2.1 Neurological causes

Neurogenic lower urinary tract dysfunction (NLUTD) is exhibited in individuals with neurological diseases (Cerebral Vascular Accident-CVA, Parkinson disease, Multiple Sclerosis-MS), lumbar injuries and peripheral neuropathies (like diabetes mellitus and tabes dorsalis). Other conditions associated with NLUTD are spinal lesions (congenital like spina bifida and secondary like cervical myelopathy and lumbar disk herniation) as well as some neoplasms of the Central Nervous System (CNS) (primary or metastatic). Regional anaesthesia and major surgical procedures on the pelvis are also related to NLUTD yet, they can also be classified as iatrogenic[8].

The most common neurological problems associated with female UR are MS, lumbar injury and

diabetes mellitus[6]. However, the exact rate of MS (and Parkinson's disease) inducing UR cannot be precisely defined given that the onset of symptoms in the urinary tract may precede for a mean period of 5 years prior to the disease diagnosis and 2 years prior to the encounter of more specific neurological symptoms[9]. In addition, certain patients, apart from the detrusor hyperreflexia, are referred with a significant post-micturition residual which is usually disregarded[10]. The mechanism of NLUTD in patients identified with Parkinson's disease or MS has not been fully elucidated. Nevertheless, basal ganglia lesions and neurons demyelination realized in these conditions, possibly affect the CNS at several different locations important for the bladder control. This explains the variety of manifestations in the urinary tract system and their early onset. For example, the detrusor hyperreflexia is attributed to cell loss in the brainstem region, whereas incomplete bladder voiding is due to cell atrophy of the Intermediolateral Nucleus[11] (IML). Furthermore, cell loss in the anterior horn in Onuf's nucleus causes urethral sphincter dysfunction[12]. Consequently, it is possible for the patient to present a combination of bladder hyperactivity and sphincter deficiency along with incomplete voiding. In Parkinsonism, outflow obstruction may also be manifested due to impaired relaxation or bradykinaesia of the urethral sphincter[13]; dopaminergeric medication is also responsible for directly impacting the detrusor muscle[14].

Likewise, in diabetes mellitus, the mechanism of urethrovesical dysfunction is not clearly understood. However, peripheral neuropathy, changes in the Autonomic Nervous System (ANS) receptors, change in the micturition reflex at the sacral neurotomes of the spinal cord and detrusor muscle injury have been identified. Main neuropathic changes include nerve loss, axon degeneration and gradual demyelination. These changes are induced by Schwann cell metabolic disorders and changes resulting from chronic glucohemia. Early signs include increased water intake followed by increased micturition frequency and urine volume and are accompanied by detrusor overactivity (DO) (early urodynamic finding). Given the gradual decrease in the bladder's sensation, the progressive decrease in frequency follows together with a parallel increase in the urine volume. The combination of these two along with the decrease in the bladder's contractility encountered much later, cause incomplete bladder outflow and UR. Symptoms commonly appear ten years after the onset of diabetes mellitus[15].

In a large pool of females with UR, none of the aforementioned causes is detected. However, they exhibit detrusor hyperactivity with impaired contractility (DHIC). Some investigators attribute these cases to age-related changes[15]. The same females - especially the elderly whom symptoms at the LUT are more often - do not exhibit CUR frequently. Valentini et al. performed a urodynamic study on 100 symptomatic female subjects aged 80-93 years and found an impact of 15%. Notably, the population study exhibited all-cause detrusor hypoflexia significantly rarely compared to hyperflexia[16].

2.2 Psychogenic causes

UR may occur – single or concomitant to micturition difficulty and perineal pain – in terms of somatoform disorders[17]. These mental disorders are exhibited as somatoform disorders. Their main characteristic is the presence of physical symptoms that can neither be fully explained on the basis of a disease nor can they be interpreted as a substance's action or even be attributed to some other form of mental disorder (eg panic attack). Contrary to the factitious disorders and faking, physical symptoms are not intentional[17]. Psychosocial problems may contribute to urinary retention, single or concomitant to some diseases (e.g. urinary tract infection-UTI). To a great extent, demonstration of UR is associated with a previous incident such as a spouse argument, the death of a loved one etc[6].

2.3 Medication

Prospective studies data suggest that up to 10% of AUR cases can be attributed to use of medication. Occasionally, UR has been reported following the intake of anticholinergic medication (eg antipsychotics, antidepressants and respiratory anticholinergic agents) and after the intake of opioids and other anaesthetics, benzodiazepines, alpha adrenergic receptors, nonsteroidal antiinflammatory analgesics (NSAIA), muscle relaxants and calcium channel blockers (CCB)[18]. It is not clear whether the aforementioned medication causes the UR and the conditions for CUR, single or concomitant to the presence of another agent. However, bladder is susceptible to side-effects of medication like antidepressants, opioids, antipsychotics, antimuskarins and alpha adrenergic receptors, due to the complex neurological control and the frequent excretion of the metabolites of this medication via urine. A further trial suggests the following: CUR is rarely caused by antidepressants as it shown in case reports available in literature. Though this type of medication has many pharmacological actions, its exact role in the urinary tract is not fully known, however, it is supposed to have an alpha-adrenergic action on the smooth muscles of the urethra and thusly it increases the outflow resistance via the urethra[18]. Indeed, the bladder neck and the proximal urethra have alpha adrenergic receptors, which when stimulated, cause smooth muscle fibers contraction. Tricyclic antidepressants (TCAs) like imipramine and doxepin increase urethral resistance but may decrease bladder contractility. It is still under investigation whether UR develops when some other pathological condition acts as a background or whether antidepressants impair detrusor contractility in the long-term. In this case, retention is chronic and possibly realized in various stages associated with serotonin reuptake. The following are indications of the above: 1) long-standing antidepressants administration leads to a decrease in 5-HT2 receptors' number and sensitivity[19], 2) serotonergic neurons engage in controlling the lower urinary tract[12], 3) older (tricyclic) as well as recent (selective serotonin reuptake inhibitors – SSRI) antidepressants have been associated with UR insidents[20] and 4) norepinephrine reuptake

inhibitors (NRI) (thionisoxetine) do not affect bladder capacity or sphincter activity. Yet, the fact that in all cases recorded micturition was restored upon medication interruption limits the above possibility.

Anticholinergic drugs have occasionally been associated with AUR[22]. A randomized, placebo-controlled study investigated, among others, the safety of the antimuscarinic agent oxybutynin, in 65 aged female subjects with intellectual disability (ID) suffering from urge urinary incontinence (UUI). Only one patient (1.5%) complained of UR, which resolved without treatment[23]. This medication competes the muscarinic acetylcholine receptors effect and does not induce Ca2+ release and consequent muscle contraction at the detrusor. It is not established whether detrusor contractility becomes resistant to medication over time, however, the observed URs are more likely to develop due to another condition. The case of increased urethral resistance and/or obstruction is possibly not included in them. Actually, in another randomized controlled trial comparing antimuscarinic tolterodine to tamsulosin, an alpha1 adrenoceptor blocking agent for the treatment of male subjects with lower urinary tract symptoms (LUTS) and overactive bladder (OAB), did not reveal statistically significant differences in the development frequency of UR (tolterodine & tamsulosin 0.4%, tolterodine 0.5%, tamsulosin 0% and placebo 0%). In addition, the development of UR was similar for both men with small and men with large cell prostate cancer[24]. Interestingly, a retrospective study by Roehrborn et al. revealed that the relative risk for AUR in patients receiving antimuscarinics – dose independent – was higher during the early treatment (first 30 days) and in patients with genitourinary system conditions. This shows that in fact, URs do develop caused by another pathological mechanism[25]. In most cases though, micturition resolves upon medication interruption and this implicates antimuscarinics to a great extent[26].

Opioids have been associated with UR in case reports and in clinical trials. In their majority, opioids were administered for operative analgesia. The post-operative UR impact was estimated at 5% whereas predisposing factors were diabetes mellitus, intraoperative atropine administration and prolonged hospitalization[27]. Given that UR patients sustain more than one risk factors, the exact impact of opioids on UR is hard to be assessed but is estimated between 3.8 and 18.1%[27, 28]. Nonetheless, Panicke et al. found that a high percentage (39%) of females manifesting all-cause URs had been administered opioids the previous period[29]. The mechanism is yet unknown and their urinary tract impact is not fully apprehended. Based on up to date knowledge, it seems that they reduce detrusor tone, contraction strength, sense of fullness and at the same time suspend micturiotion reflex. They do not however increase sphincter tone[30]. Experimental studies have found that these effects, irreversible when naloxone is administered, are realized centrally in the brain and the spinal cord with recorded peripheral impacts on the bladder[31]. Rosow et al. proved that some of the above changes

in the bladder activity are partially induced by opioids' peripheral action that can be reversed by methylnaltrexone, a peripherally-acting μ-opioid receptor antagonist. Post-operative UR –surgery independent– is more likely to develop following epidural morphine and less likely after intravenous or intramuscular administration[32]. Prolonged peripheral nerve blockade (PNB) shows the smallest frequency of post-operative UR (15.8); epidural anaelgesia shows the highest (48.1%)[33]. Generally, UR resolves spontaneously 24-48 hours after urinary catheterization removal[34].

2.4. Anatomical causes

Pure urethral obstructions have been established in females exhibiting UR. Though their mechanism is shared, these vary according to aetiology and origin. The most common cause is primary bladder neck obstruction (PBNO) identified in 9-16% of females with obstructive micturition. Malignant neoplasias in the female reproductive system, colon and bladder as well as benign bladder neck masses induce BOO[35, 36]. Female neoplasia-induced UR frequency is not known but is estimated to less than 10% of the total[5]. Anterior vaginal wall or uterus prolapse-induced UR frequency is not reported. In the first case, possible bladder neck displacement causes dysouria and incomplete bladder voiding whereas the second may induce acute as well as chronic UR[37]. UR induced by gallstones, female bladder anatomical anomalies and haematocolpos is likewise unsual[38-40].

2.5 Surgical procedures

Vaginal vault suspension may lead to overcorrection as well as to urethral compression or deformation especially when one of the sutures is placed close to the urethra. Dysuria develops commonly; UR is very rare[41]. Symptoms may last for up to a month[41]. The Marshall-Marchetti-Krantz technique produced higher rates of overcorrection and UR[42]. In less than 10% of the cases, dysfunctional micturition and UR are observed in the initial post-operative period for the restoration of stress urinary incontinence (SUI) by applying tension-free vaginal tapes (TVT). That said, AUR is quite uncommon and complete bladder voiding usually resolves quickly (median period 9 days)[43]. In these cases, the technique is not as much implicated as in anaesthesia, discomfort, localized oedema, haematoma formation and outlet realignment procedure. Overcorrection is mainly implicated when AUR is observed or when symptoms and post-void residual volume remission do not resolve within a month[42].

Augmentation cystoplasties (AC) aim to increase bladder capacity by reducing bladder contractions during filling but they affect bladder contraction and so, some patients present with a considerable post-void residual volume and may manifest UR[43]. Intravesical botulinum toxin (BT) injection for the treatment of OAB bears a significant risk of UR which increases by repetition of injections[44].

2.6 Fowler's syndrome

Fowler's syndrome is defined as UR with large post-void residual volume in young females following menarche. It is detected in UTI screening tests or it