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.

Key words
Urinary retention, women.

Introduction
Urinary retention (UR) is defined as the inability to achieve full bladder drainage by voluntary micturition and is classified into acute (complete inability to urinate) 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.

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 (PH). Women on the contrary, especially in older ages, present a number of 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. A considerable rate of older women present cystoscopic findings of urinary obstruction like trabeculations and bladder diverticula 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 signs. In particular, the presence of a clear anatomical impediment does not constitute a common finding though obstructive symptoms, such as poor or intermittent urinary flow, terminal dribbling or post-micturition dribbling, are commonly 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: urinary retention, female 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 reports⁶. 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 1:13⁵. García-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 followed⁶. 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 determined⁷. 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 causes⁶. Interestingly, studies before 1980-90, when Fowler’s syndrome was established, consider 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 undetected⁷.

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 however they can also be classified as iatrogenic⁸. The most common neurological problems associated with female UR are MS, lumbar injury and diabetes mellitus⁸. 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
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.

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. The same females—especially the elderly who have more frequent LUTS symptomatology—are more often do not exhibit CUR frequently. Valentini et al. performed 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.

2.2 Psychogenic causes
UR may occur—single or concomitant to micturition difficulty and perineal pain—in terms of somatoform disorders. These mental disorders are exhibited as somatoform disorders. Their main characteristic is the presence of physical symptoms that can neither
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. 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. 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, 2) serotonergic neurons engage in controlling the lower urinary tract, 3) older (tricyclic) as well as recent (selective serotonin reuptake inhibitors – SSRI) antidepressants have been associated with UR incidents 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. 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\textsuperscript{23}. This medication competes the muscarinic acetylcholine receptors effect and does not induce Ca\textsuperscript{2+} 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\textsuperscript{23}. 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\textsuperscript{23}. In most cases though, micturition resolves upon medication interruption and this implicates antimuscarinics to a great extent\textsuperscript{23}. Opioids have been associated with UR in case reports and in clinical trials. In their majority, opioids were administered for operative analgesia. The postoperative UR impact was estimated at 5\% whereas predisposing factors were diabetes mellitus, intraoperative atropine administration and prolonged hospitalization\textsuperscript{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\%\textsuperscript{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\textsuperscript{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 micturition reflex. They do not however increase sphincter tone\textsuperscript{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\textsuperscript{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 \mu-opioid receptor antagonist. Postoperative UR - surgery independent - is more likely to develop following epidural morphine and less likely after intravenous or intramuscular administration\textsuperscript{32}. Prolonged peripheral nerve blockade (PNB) shows the smallest frequency of postoperative UR (15.8\%); epidural analgesia shows the highest (48.1\%)\textsuperscript{33}. Generally, UR resolves spontaneously 24-48 hours after urinary catheter removal\textsuperscript{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. Female neoplasia-induced UR incidence is not known but is estimated to less than 10% of the total. Anterior vaginal wall or uterus prolapse-induced UR incidence is not reported. In the first case, possible bladder neck displacement causes dysuria and incomplete bladder voiding whereas the second may induce acute as well as chronic UR. UR induced by gallstones, female bladder anatomical anomalies and haematocolpos is likewise unusual.

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. Symptoms may last for up to a month. The Marshall-Marchetti-Krantz technique produced higher rates of overcorrection and UR. In less than 10% of the cases, dysfunctional micturition and UR are observed in the initial postoperative 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 emptying usually resolves quickly (median period 9 days). 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. 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. Intravesical botulinum toxin (BT) injection for the treatment of OAB bears a significant risk of UR which increases by repetition of injections.

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 exhibits as painless and with large post-void residual volume AUR. Commonly, manifestation of dysuria or other discomfort at the lower urinary tract prior to UTI is rare and most females who will finally be diagnosed with this syndrome will not able to determine its onset. Epidemiological data are not available but it has been reported that 40% affects women with polycystic ovary syndrome (PCOS). This associates UR to sex-linked hormonal disorders; mechanism is also unreported. It has been presumed that changes in the hormonal system occurring upon menarche may affect the ion channels in skeletal muscles promoting ephaptic nerve transmission. Particularly, urethral sphincter become-
mes overactive and hypertrophic and reacts excessively to direct stimulation.

2.7 Urethral conditions
Urethral conditions may induce female UR. Their aetiology varies but the obstruction mechanism is shared. Mostly marked urethral conditions are urethral prolapse, abscesses and cysts and congenital anomalies. Urethral prolapse primarily affects young girls whereas urethral caruncle primarily affects women in menopause. Their aetiology relates to supportive tissue impairment and simultaneous intrabdominal pressure (IAP) increase (other causes may apply) which is transferred to the urethra mucus via the genitourinary diaphragm. In girls, prolapse manifestation is a result of constipation, tenesmus, diarrhea, forcible stretching or vigorous cough. In older females, urethra prolapse and caruncle are induced by injuries. Localized pressure causes vascular stasis, oedema and localized stenosis. Urethral glands abscesses and cysts are formed as a consequence of acute bacterial infection especially when the pores are obstructed. Female urethra stenosis is managed as a common condition and typically does not cause acute obstruction. It results from a complicated injury and in particular of lymph nodes granulomatous inflammation. The most frequent congenital anomaly of the female urethra is hypospadias. As the external urethral orifice extrudes to the upper vaginal wall, it is not associated with CUR except for extremely rare cases.

2.8 Intestine conditions
In some females, mainly of young age, with normal urodynamic parameters (SV, EMG) and absence of other obvious cause, the development of UR is associated with chronic idiopathic pseudoobstruction (CIP). This unusual syndrome is characterized by chronic intestinal obstruction in absence of anatomical or mechanical damage. The exact mechanism of concomitant micturition dysfunction is practically unknown even though 10-69% of the patients exhibit dysfunctional micturition. Certain investigators have reported findings from urodynamic studies in children with CIP syndrome such as hypococontractile detrusor, increased capacity and residuals as well as decreased sensation. It is believed that these are attributed to the same myopathy or neuropathy that affects the intestine.

3. Diagnosis and Management
Clinical examination, urine analysis and ultrasound are performed for the determination of UR, its extent and the presence of complications (hydronephrosis, urinary tract infection). Initial management focuses on bladder drainage, especially in the case where the female is symptomatic or in risk of complications. This is achieved by indwelling and intermittent catheterizations. However, apart from bladder decompression, intermittent catheterizations contribute in monitoring micturition function (by measuring post-void residual volume) and in preventing possible morbidity of the indwelling catheter. If reversible causes are detected (e.g., infection, inflammation, prolapse), these can be corrected simultaneously or at a later stage. The rest investigations should focus on indentifying the underlying pathophysiology or the reversible factor inducing
UR. Investigation is based on detailed anamnesis, clinical examination and laboratory, imaging and special tests. Clinical examination should be sequenced by thorough gynaecological and neurological examination. Cystoscopy and urodynamics are required in all other cases for the investigation of the cause in order to opt for the definitive management. Neurological condition induced UR treatment is typically limited to intermittent catheterizations but there are cases where these constitute definitive management such as in psychogenic UR. In drug-induced UR, cause interruption is adequate in resolving micturition in most of the cases. UR treatment caused by anatomical conditions depends on nature and origin and is mainly surgical. In female patients with PBNO, dissection or intraurethral incision are usually advised though published studies demonstrated varied efficacy and SUI is recognized as a significant post-operative complication. On the contrary, alpha-blockers can reduce urethral pressure but their use has not been documented in terms of permanent or temporary treatment (nocatheter trial). Post-SUI operation observed UR does not usually require a second therapy except for temporary catheterization. In contrast, certain surgical treatments for OAB-induced UUI do require restoration. Regarding postoperatively observed UR, this is not well understood but since micturition resolves relatively quickly, no treatment is required. Urethral conditions such as ectropion, abscesses and cysts are surgically managed whereas females exhibiting stenosis undergo urethral dilation as part of obstruction management but no sufficient data document its efficacy. Neuromodulation effectiveness in micturition restoration constitutes the only applicable treatment in Fowler’s syndrome. On the other hand, poor understanding and late-stage diagnosis establish intermittent catheterizations as the applicable treatment for CIP.

Conclusion

Female UR pathophysiology and epidemiology are not well validated. Currently known science presents the condition’s aetiology as multifactorial. Neurological causes are more frequent whereas obstructive are uncommon. Often, implicating factors are concomitant. Careful assessment including detailed anamnesis, physical examination, urodynamic and imaging studies is required to elucidate the condition’s nature and irreversibility.
Περίληψη

Επίσημη Ούρων στη Γυναίκα
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Διεθνής παθοψυχολογία και η επιδημιολογία της επίσημης των ούρων στη γυναίκα δεν είναι επαρκώς τεκμηριωμένες. Σύμφωνα με τα μέχρι τώρα γνωστά, η αιτιολογία αυτής της οντότητας φαίνεται πως είναι πολυπαραγοντική. Οι παράγοντες που εμπλέκονται πιο σημαντικά είναι συγγενείς, νεοπλασματικοί, φαρμακολογικοί, νευρολογικοί, ανατομικοί, λειτουργικοί, μυοηγαθικοί και ψυχογενεικοί. Εάν η επίσημη των ούρων είναι οξεία ή χρόνια που συνδέεται από αποφαστική ουροπάθεια, λοιμωξή ή πόνο, τότε η αρχική αντιμετώπιση της υποκείμενης γυναίκας περιλαμβάνει την κένση και την επιμέλεια της ουροδόχου κύστης. Η διερεύνησή τα πρέπει αρχικά να επαναλαμβάνει στην εντοπιότητα των επικίνδυνων επιπλοκών και έπειτα στην υποκείμενη αιτιολογία.

Λέξεις ευρετηριασμού: Επίσημη ούρων, γυναίκα.

References


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