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NEPHROLOGY - REVIEW

Nocturia: its characteristics, diagnostic algorithm and treatment

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Abstract
Nocturia is the complaint that an individual has to wake up at night one or more times to urinate. It is a frequent condition among older adults and entails detrimental effects with regard to sleeping, sexual activity, comfort, depression, mental function and vitality. It is clinically important to distinguish it from global polyuria, defined as a urinary rate ≥ 125 ml/h (3000 ml/day), as well as from nocturnal polyuria, which is an abnormally large volume of urine during sleep associated with a decreased daytime urine production. A Frequency Volume Chart (FVC), overnight water deprivation test with renal concentrating capacity test, and the nocturnal bladder capacity index are some of the methods that help establish the underlying pathology of this condition and hence define an adequate treatment plan.

Keywords Polyuria · Nocturnal polyuria · Diagnosis

Introduction

There is no normal value of daily urinary excretion of water and electrolytes; however, there is an expected timing and urinary volume for healthy individuals. In this sense, daily diuresis in adults individuals on a habitual western diet is usually 1–1.5 l (about 60 ml/h) during day time, and it decreases to 36 ml/h at night [1, 2]. Consequently, diuresis is classically defined as insufficient diuresis (oliguria) when the urinary rate is ≤ 17 ml/h (400 ml/day), while as excessive diuresis (polyuria) when the urinary rate is ≥ 125 ml/h (3000 ml/day) [1–3]. Moreover, there is a physiologic criterion to evaluate diuretic volume, which is based on the determination of the patient's daily urinary osmolar excretion or *osmolar diuresis* (OD). Since urea is the main osmol in urine, urea excretion rate mainly depends on protein intake, and age in healthy people. Urea urinary excretion and, consequently urinary osmolarity, are usually higher in older individuals compared to younger individuals. This phenomenon has been attributed to reduced urea reabsorption capability in aged renal tubules [1].

OD is based on the fact that there is a close relationship between the osmol amount that the organism has to excrete daily in urine and the concentration of urinary osmoles, resulting in the urinary volume required to achieve the total osmolar excretion. This relationship is expressed by the following equation [4]:

$$\text{OD} = \text{daily urinary osmolar excretion} \,/\, \text{daily urinary osmolar concentration}$$

Thus, for example, according to this equation, since an individual on a western diet has to excrete 900 mOsm/day in urine, if his urinary osmolarity is 400 mOsm/l, then his osmolar diuresis should be 94 ml/h, that is 2250 ml/day.

$$2250\ \text{ml/day}\ (94\ \text{ml/h}) = 900\ \text{mOsm/day} / 400\ \text{mOsm/l}$$

Based on the example mentioned above, the appearance of a urinary rate > 104 ml/h (2500 ml/day) would be interpreted as polyuria; on the other hand, the urinary rate < 84 ml/h (2000 ml/day) would be interpreted as oliguria, regardless of the classic criterion used to define polyuria (≥ 3000 ml/day) or oliguria (≤ 400 ml/day). Additionally, it is clinically important to distinguish between the different

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types of polyuria. Global (whole day) polyuria is different from nocturnal polyuria, which may be one of the possible causes of nocturia, which consists of waking from sleep to void. These concepts are furthermore thoroughly analyzed to acknowledge their differences and characteristics.

Nocturia: definition and inducing mechanisms

Nocturia is the complaint that an individual has to wake up at night one or more times to urinate, and each micturition must be preceded and followed by sleep. Only two or more times of nocturnal waking are sufficient to negatively affect the patient's quality of life [3–10]. Nocturia is a multifactorial disorder, mainly caused by different mechanisms, such as polyuria (global or nocturnal), primary sleep disorders, peripheral edema-inducing diseases, altered bladder storage capacity with or without lower urinary tract symptom (LUTS) including benign prostatic obstruction (BPO), overactive bladder (OAB), and combined mechanisms. The evaluation of these mechanisms is very important for determining the etiology, to prescribe an adequate treatment [4, 10–12].

Nocturia: risk factors and epidemiology

Several clinical settings, such as obesity, sedentary lifestyle, pregnancy, childbirth, and menopause have been documented as risk factors for nocturia [12]. This condition is an underreported and undertreated condition, and it is the most prevalent lower urinary tract symptom (33%), which is more frequent in older individuals (60%) than in younger ones (15–20%), and is slightly reported more in women (54%) than in men (49%) [10, 12].

In 2006, the Tampere Aging Male Urologic Study (TAMUS) examined 1633 Finish men between 50 and 70 years old, and found a nocturia prevalence of 56%, with 10% more men older than 50 years old voiding at night each year [13]. In 2007, the Finnish National Nocturia and Overactive Bladder (FINNO) study examined 3744 individuals between 18 and 79 years old, and nocturia was reported in 56% of men and 40% of women with overactive bladder [13, 14]. In 2013, a Japanese study examined patients older than 65 years of age, and they documented a nocturia prevalence of 47%, which increased to 50% after 1 year, while the Krimpen study found a prevalence of 34% in people up to 60 years old, and increasing with age thereafter [15]. These various results suggest that nocturia prevalence increases with age and hence is higher in older adults. For instance, in men in their 70 s and 80 s, around 70–90% reported 1 or more voids per night, and 30–60% reported at least 2 voids every night [3]. With regards to women, the prevalence in older women is high. However, this condition can also affect a significant proportion of younger individuals. For instance, a systematic review based on 43 articles found that nocturia prevalence in younger women in their 20–40 years was at least 1 void per night in about 20–44%, and 2 or more voids in about 5–18%. Anyhow, in older women nocturia prevalence was 1 or more voids in about 74–77%, and at least 2 voids in about 28–62%. Finally, it was also reported that a higher prevalence of nocturia was documented in Black and Hispanic women compared to Caucasians. Conversely, socioeconomic factors appear not to be relevant for this condition [16].

Nocturia: clinical implications

Nocturia is annoying and inconvenient for the patient, but its impact on sleep quality is associated with a decrease in quality of life (QOL). Nocturia has also been associated with a significant decrease in health-related quality of life (HRQoL), showing a decrease in several measured dimensions such as sleeping, sexual activity, comfort, depression, mental function and vitality. The degree of discomfort seems to be related to the quality of sleep. Even more, nocturia is the leading cause of sleep disorders in older adults, which can also induce conditions such as impaired physiological nighttime blood pressure dipping, increased sympathetic activity, insulin resistance, obesity, and thus increasing cardiovascular death. Moreover, apart from impaired mood and work performance, sleep disorder can also lead to immune response alterations, poor concentration, falls, fractures, and other accidents related to frequent nighttime voiding and daytime fatigue, which could result in premature death [3, 8–20].

Global polyuria and nocturnal polyuria

Global polyuria (24 h polyuria) is classically defined by a urinary rate $\geq$ 125 ml/h (3000 ml/day), and it can be induced by an excessive urinary excretion of solutes (osmotic polyuria) or an excessive urinary excretion of water (water polyuria).

Both types of polyuria can be distinguished because osmotic polyuria has a urine osmolarity > 300 mOsm/l (e.g., in uncontrolled diabetes mellitus), while aqueous polyuria has urine osmolarity < 300 mOsm/l (e.g., in diabetes insipidus, excessive fluid intake) [1].

Regarding diabetes insipidus, it can be secondary to vasopressin deficiency (central diabetes insipidus) or vasopressin renal resistance (nephrogenic diabetes insipidus).

Among the main causes of central diabetes insipidus are encephalopathy, cranial trauma, neurosurgery, and corticosteroids; while among the main nephrogenic diabetes insipidus are: tubule-interstitial nephropathy, loop diuretics, lithium, and hypercalcemia [1].

Main factors that antagonize vasopressin and promote water diuresis are prostaglandin E2, atrial natriuretic peptide (ANP), electrolytes disorders (hypercalcemia, hypokalemia), and certain drugs (lithium, serotonin reuptake inhibitors, calcium-channel blockers, tetracyclines, rifampicin, amphotericin B, and foscarnet) [2, 10].

Nocturnal polyuria should be distinguished from global polyuria since the former only appears at night. Nocturnal polyuria is the production of an abnormally large volume of urine during sleep (bedtime urinary rate $\geq$ 90 ml/h), and this is offset by a decreased daytime urine production, such that 24-h urine volume remains within normal limits [6, 7]. It should be pointed out that the nighttime urine output does not take into account the last void before sleep but includes the first void of the morning [5].

Nocturnal polyuria pathophysiology

Diuresis is mainly controlled by two hormones: vasopressin and atrial natriuretic peptide (ANP). Vasopressin release is controlled by an individual's plasma osmolality and volemic status. Since this hormone stimulates water reabsorption from collecting tubules, then any disorder that affects its secretion or renal response induces diluted urine, as it is the case of diabetes insipidus. Vasopressin secretion has a natural diurnal fluctuation, having people without nocturia levels of serum vasopressin peak at night. Conversely, individuals who suffer from nocturnal polyuria do not have this nocturnal vasopressin increase [4, 10]. Besides, overnight melatonin secretion is higher in patients without nocturia than in those with nocturia [10]. On the other hand, conditions such as obstructive sleep apnea, in which there is upper airway obstruction, rises negative intrathoracic pressure. This phenomenon increases venous blood flow to the heart, inducing ANP release from the cardiac atrium and ventricle. ANP inhibits vasopressin secretion, as well as sodium tubular reabsorption, finally promoting nocturnal polyuria. Another cause of nocturnal polyuria secondary to increased ANP levels is congestive heart failure [10].

Secondary causes of nocturnal polyuria, aside from congestive heart failure, include diabetes mellitus, hypoalbuminemia, lower extremity venous stasis disease, cirrhosis, renal insufficiency, which can induce peripheral edema (third fluid space). When patients lie supine at bedtime, interstitial fluid redistributes into the intravascular compartment, and this additional water load on the kidney increases diuresis [4, 10].

Another important cause of nocturnal polyuria is a lifestyle, as it is the case of excessive salt or fluid intake or alcohol or caffeine consumption at night. Finally, the timing of therapy with diuretics or beta-blockers also can induce higher nocturnal urine output [16].

Nocturnal polyuria diagnosis

In 2002, the International Continence Society (ICS) defined that nocturnal polyuria is present when greater than 20% of the 24 h urine volume in young adults, or 33% in individuals older than 65 years old, is produced at night. The latter is known as the nocturnal urine volume (NUV 33) or nocturnal polyuria index (NPI 33) definition.

The NPI is calculated by dividing NUV by the total 24-h urine volume [5, 9]. This ICS definition is simple and easy to use, although it does not take into account the duration of sleep [8]. Blanker et al. proposed the nocturnal urine production 90 definition (NUP 90), which defines nocturnal polyuria as the nocturnal urine production exceeding 90 ml/h (for older men). This value was established since 90 ml/h is two standard deviations above the mean nocturnal urine production of 60 ml/h [5]. According to the NUP 90 definition, nocturnal polyuria is more frequent in men than in women, whereas the opposite was observed in the NUV 33 definition [8]. Finally, it has also been proposed the nocturia index, which is calculated as the nocturnal urine production divided by the functional bladder capacity (FBC), where FBC is the largest single micturition in a typical 24-h period [9].

In patients suffering from nocturia, it was observed that 50% of men had nocturnal polyuria. Similarly, it was also reported that the prevalence of nocturnal polyuria in older men according to NPI 33 is very high, since it ranges from 41.8% in 50–55-year-old men to 56.9% in 70–78 years old men. It has also been documented that 78% of the population suffered from nocturnal polyuria, from which 43% had NPI > 33%, and 69% between 20 and 33%. Additionally, 27% of the sample presented NUP > 90 ml/h. It was also documented that the prevalence of nocturnal polyuria in men according to NUP 33 was > 40% for all age strata, and rising from 41.8% between 50 and 54 years to 56.9% between 70 and 78 years [18]. Curiously, it was also observed that the prevalence of nocturnal polyuria in patients with idiopathic Parkinson's disease was not higher than in the general population at the same age [8].

Nocturnal polyuria can be caused by water diuresis, sodium diuresis or a combination of both. Thus, an analysis of urine and a renal function profile might be a useful adjunct to the Frequency Volume Chart (FVC), a voiding bladder diary in patients with nocturnal polyuria, to further determine the pathophysiology of nocturia in each patient [10].

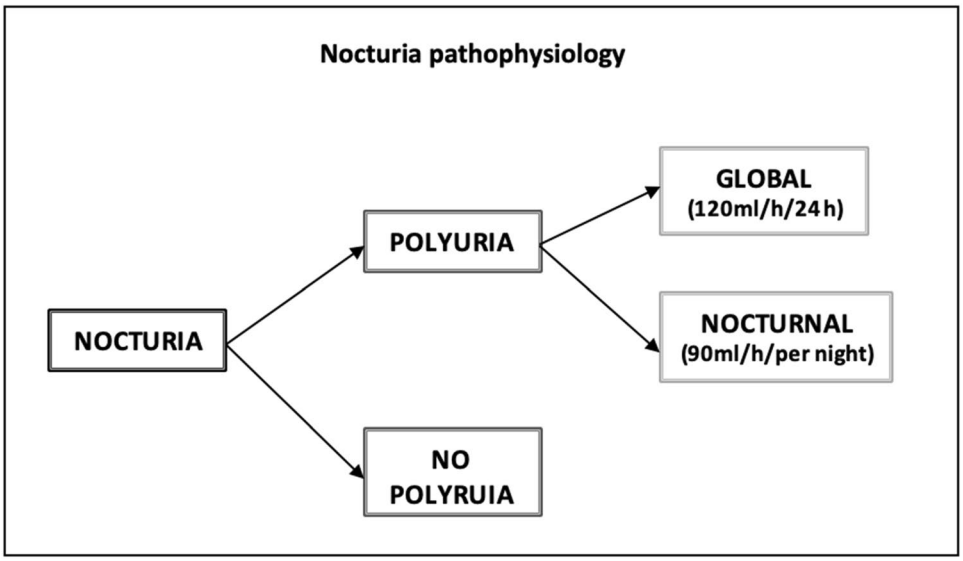

Fig. 1 Nocturia pathophysiology diagnosis algorithm

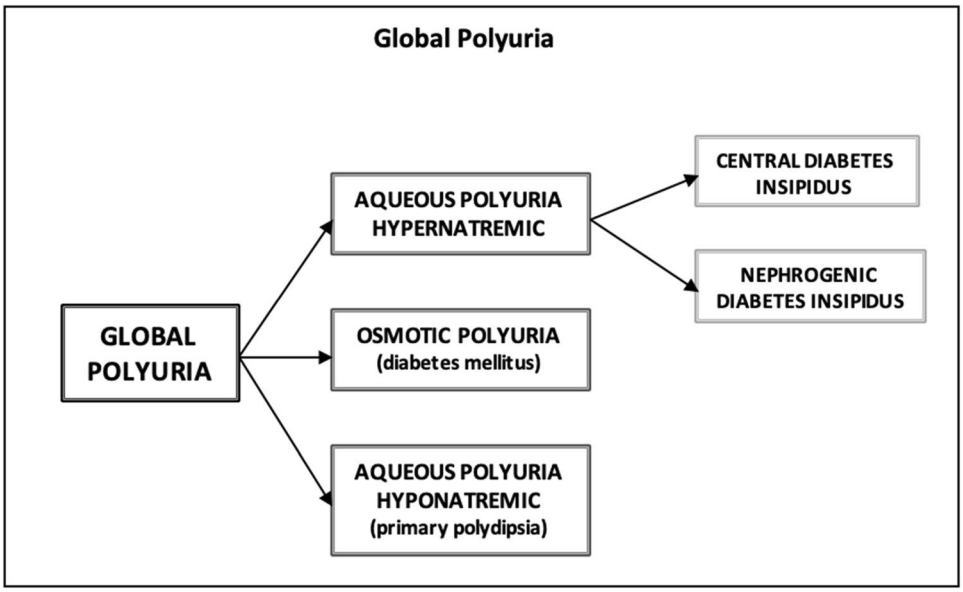

Fig. 2 Global polyuria diagnosis algorithm

In Figs. 1, 2 and 3 an algorithm for nocturia diagnosis is proposed.

Nocturia in sleep and mood disturbances

Sleep disturbances secondary to primary sleep disorders (insomnia, sleep apnea, periodic leg movements, narcolepsy), medical conditions (cardiac failure, chronic obstructive pulmonary disease, hyperthyroidism, central nervous system diseases), psychiatric conditions, chronic pain disorders, and medication (corticosteroids, diuretics, alpha-adrenergic antagonists) can induce nocturia [12]. According to the FINNO study, sleep disruption due to obstructive sleep apnea (OSA) with snoring as an indicator, was an important cause of nocturia [4–16]. OSA can induce nocturia through increasing ANP serum levels, and bladder oxidative stress from hypoxic episodes. This mechanism generates detrusor instability by reducing bladder compliance and increasing spontaneous contractions [10]. In 2014 Araujo et al. reported that even though sleep restriction had no association with nocturia, poor sleep quality did show a correlation with this condition, evidencing a bidirectional association between sleep quality and nocturia [21]. Breyer et al. documented that nocturia increased the risk of developing depression, while depression was associated with higher reports of nocturia [22]. These authors established that there is a

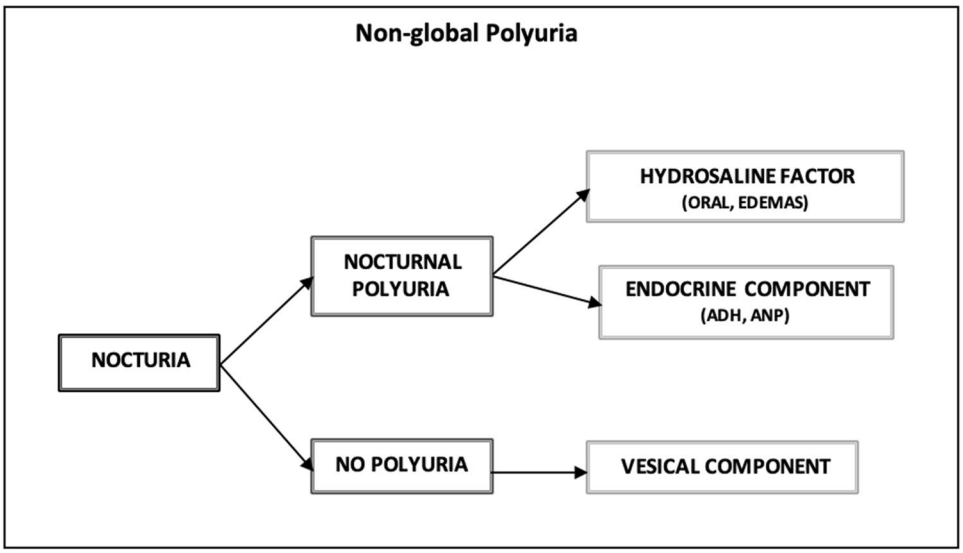

Fig. 3 Non-global polyuria diagnosis algorithm

unidirectional effect of depression on nocturia incidence, higher in men than in women, as untreated depression may lead to nocturia [7].

Nocturia in bladder storage capacity alteration

There are different bladder factors which can induce nocturia in an isolated or combined way:

- Overactive bladder (OAB): this mechanism can be of inflammatory (acute cystitis, benign prostatic hyperplasia) or neurologic (neurogenic bladder) origin. OAB is a condition characterized by urinary urgency, with or without urinary incontinence, usually with increased daytime frequency and nocturia [3, 16].
- Partial bladder obstruction: this mechanism induces the appearance of bladder residue which consequently leads to an increased voiding frequency, even at night (benign prostatic hyperplasia).
- Low bladder compliance: bladder stiffness leads to a reduced bladder capacity which leads to increased voiding frequency during daytime and at night, for instance due to chronic cystitis, pelvic radiation therapy, benign prostatic hyperplasia**.** Combination between bladder dysfunction and polyuric disorders can be frequently found. For instance, 62% of women suffering from overactive bladder (OAB) have underlying nocturnal polyuria [12]. Studies suggest that the functional bladder capacity (FBC), consistent with the studies mentioned previously, decreases with age and is related to the presence of lower urinary tract symptoms [23].

Nocturia: differential diagnosis

Primary polydipsia, central diabetes insipidus, and nephrogenic diabetes insipidus can be distinguished using an overnight water deprivation test and renal concentrating capacity test. The ability to concentrate urine after overnight water deprivation proves that a patient can both produce and respond to vasopressin, indicating a diagnosis of primary polydipsia. If the overnight concentration of urine does not occur, patients receiving desmopressin will concentrate their urine if the kidneys are normally responsive to this hormone, as it happens in central diabetes insipidus. Conversely, failure of urine concentration despite desmopressin administration is typical of nephrogenic diabetes insipidus [10].

A nocturnal reduction in bladder capacity is diagnosed if nocturia presents volumes less than the maximum bladder capacity, and is diagnosed by applying the nocturnal bladder capacity index (NBCI): actual number of voids (ANV) minus the predicted number of voids (PNV): NBCI = ANV—PNV.

ANV does not include the first-morning void after waking. PNV is calculated as nocturia index (NI) minus 1 (PNV = NI – 1).

When ANV exceeds PNV, NBCI is > 0 and indicates reduced nocturnal bladder capacity relative to the patient's maximum 24-h capacity. A NBCI > 1.3 has been suggested as the threshold at which reduced nocturnal bladder capacity contributes to nocturia.

Finally, Nocturnal polyuria index (NPI) is calculated as nocturnal urine volume (NUV); the total volume of urine voided (TVUN) during the night, including the first-morning void, divided by 24-h urine volume (TUV): NPI = TVUN/(TUV).

Nocturnal polyuria is then defined as an NPI of > 33% in adults aged > 65 years (NP 33) and > 20% in adults aged < 25 years, assuming the total 24-h urine volume is normal, with gradations for intermediate age groups. According to the NUP, 90 definition mentioned previously, nocturnal polyuria is defined as nocturnal urine production > 90 ml/h (NUP90), NUV > 6.4 ml/kg and NUV > 0.9 ml/min [10].

For the diagnosis of nocturia it must be taken into account [9–16]:

- A detailed patient history is crucial to diagnose nocturia. Patients should be screened for medical conditions which can induce peripheral edema, symptoms of the lower urinary tract symptoms, neurologic diseases, sleep disorders, depression, and nighttime pain. Information about patterns and types of fluid intake such as 24 h fluid intake, fluid intake at bedtime, caffeine, alcohol or diuretic ingestion at night.
- Physical evaluation, including genital, pelvis, and neurologic examination, particularly testing the anal sphincter tone, bulbocavernosus reflex, sensation on sacrum skin, digital prostate exam**.**
- Laboratory evaluating renal function, serum and urine glucose and electrolytes, urinalysis, and urine culture if urine infection is suspected.
- Frequency volume chart (FVC), which consists of a voiding bladder diary, it is a 24-h urine volume record which includes time and volume of each void and fluid intake during the whole day, and it should be kept at least for three days, ideally including a weekend. Based upon analysis of the 24-h FVC, the patient is categorized as having any of the following: global polyuria, nocturnal polyuria, low bladder capacity, or a combined disorder (Table 1).
- Even though the FVC documents the number of nocturnal voids, it does not document the impact of nocturia on a patient's quality of life. Thus, the Nocturia Quality of Life Questionnaire (N-QoL), and the first uninterrupted sleep period (FUSP) questionnaires can be added to the FVC for this purpose. The N-QoL evaluates sleep, energy, degree of bother and global quality of life. Besides, since the initial hours of sleep are vital to overall sleep quality, the evaluation of the FUSP can also be used to evaluate the impact of nocturia on quality of life.

Nocturia treatment

The treatment of nocturia should be based on its particular etiology. Examples of specific treatments are glycemic control in diabetes mellitus, removal of offending medication in drug-induced polyuria, water restriction and psychotherapy in psychogenic polydipsia, desmopressin in central diabetes insipidus, thiazide and indomethacin in nephrogenic diabetes insipidus, compression stockings and evening leg elevation for peripheral edema, melatonin or hypnotic drugs for sleep disorders, continuous positive airway pressure (CPAP) for OSA, optimal management of inducing edema chronic diseases [10]. Anyhow, as this is a multifactorial condition, one drug may not significantly improve the clinical picture. A review of trials using antimuscarinic drugs for OAB showed no significant symptom improvement [24]. However, there are some recommendations which address this condition

Table 1 Differences among nocturia, global polyuria, nocturnal polyuria

Urination disorders	Characteristic	Increased voiding frequency at bedtime	24 h urinary Volume	Nocturnal Urinary volume
Nocturia	The individual has to wake up at night one or more times to urinate, and urination is preceded and followed by sleep, regardless of the emitted urinary volume	Yes	Normal or high	Normal or high
Global polyuria	Urinary rate ≥ 125 ml/h (3000 ml/day)	Yes	High	High
Nocturnal polyuria	urinary rate ≥ 90 ml/h at bedtime (≥ 700 ml/bedtime)	Yes	Normal	High

with a comprehensive focus that can improve symptoms and quality of life in patients suffering from nocturia [10, 12, 19]:

- Changes in the patient's life-style: voiding before going to sleep, nocturnal dehydration and the avoidance of caffeine and alcohol intake at least 4–6 h before bedtime.
- Timed diuretic therapy: diuretics are often prescribed for peripheral edema without attention to the time of the day at which they would be most effective. In patients with nocturnal polyuria due to lower extremity fluid reabsorption at night, inadequate diuretic timing could lead to nocturnal polyuria exacerbation. Therefore, it is recommended to administer diuretics during the afternoon, to promote diuresis before bedtime.
- Therapies for treating high post-void residue, reduced bladder capacity secondary to different mechanisms:

  In bladder obstruction due to benign prostate hyperplasia there are different pharmacologic alternatives, alone or combined, such as: alpha blocker (tamsulosin), beta-agonist (mirabegran), 5-alpha reductase inhibitors (finasteride, dutasteride), phytotherapy, and non-steroidal anti-inflammatory drugs (e.g., diclofenac) since prostaglandin inhibition reduces detrusor muscle tone. Finally, there are invasive therapeutic alternatives, such as intraprostatic botulinum toxin injection or prostate surgery [4, 10].

  Data from the Veterans' Administration Cooperative Study Program trial in men aged 45–80 years showed that patients who were on terazosin improved from 2.5 to 1.8 voids, while patients on terazosin + finasteride had a reduction from 2.5 to 2.0 nocturnal voids. The placebo arm showed a reduction from 2.4 to 2.1 voids per night. In the Medical Therapy of Prostatic Symptoms trial (MTOPS) nocturia frequency improved significantly compared to placebo after using the combination of finasteride + doxazosin than doxazosin alone for one year [19–26].

  OAB therapeutic alternatives are: pelvic floor muscle training, muscarinic antagonists (tolterodine—solifenacina—darifenacin—oxybutynin—trospium) used alone or in combination with alpha-blockers, percutaneous tibial nerve stimulation, and intradetrusal botulinum toxin injection.

  Zinner et al. found in 389 (74%) and 134 (25%) male patients with OAB symptoms, that after 12 weeks of treatment based on trospium chloride, the average of nocturnal voids frequency decreased by 0.47 and 0.29 in the placebo group. In addition, several studies in men have reported the positive effect of an antimuscarinic drug (tolterodine) after unsatisfactory response to an alpha-blocker alone [10, 19–25].
- Desmopressin: this drug is indicated in patients suffering from nocturia caused by nocturnal polyuria, who do not have a high nightly fluid intake, and other causes have been excluded. Improvements in many quality-of-life scales have been documented with desmopressin treatment, and a dose–response relationship has been demonstrated with beneficial effects [2]. Cannon et al. have proven a decrease of 0.8 voids per night in men treated with 40 ug desmopressin [6]. Women have been shown to be more sensitive to its antidiuretic effect, and consequently they have an increased risk of hyponatremia. This phenomenon has been explained by the fact that the V2 receptor gene is located on the X chromosome in a region with a high probability of escape from inactivation [12].

Conclusion

Nocturia is a multifactorial disorder which leads to an interrupted sleep, increasing the patient´s morbid mortality. A careful differential diagnosis among its different potential inducing mechanisms should be performed to prescribe its adequate treatment.

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Declarations

Conflict of interest All the authors declare that they have no conflict of interest.

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