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UPDATE ON INSOMNIA FOR PRIMARY CARE

Introduction

Insomnia is a common clinical issue with varying definitions depending on the source. The DSM-V defines insomnia disorder as one or more of: difficulty initiating sleep, maintaining sleep, or early morning wakening with an inability to fall back asleep.

These problems occur despite adequate time allowed for sleep (7 hours), cause dysfunction, and are not attributed to another disorder. The DSM-V then classifies insomnia as either episodic (at least 1 month but less than 3 months), persistent (lasting 3 months or more) and recurrent (2 or more episodes within a year), and considers potential contributing comorbidities.[1]

According to the International Classification of Sleep Disorders (ICSD-3) classification system, symptoms must occur at least 3 times per week, and insomnia is categorized as either short term or chronic. Short-term insomnia disorder in this paradigm (ICSD-3) occurs when the sleep problems have lasted more than 1 but less than 3 months, while chronic insomnia disorder occurs when symptoms persist for more than 3 months.[2]

Insomnia occurs more commonly in the female sex and in older adults aged >65. Additional risk factors for insomnia disorder include increased arousability, higher levels of body pain, comorbid medical or psychiatric conditions, previous episode(s) of insomnia, and a positive family history of insomnia.[3-5]

An estimated 40% of the Canadian population at some point in their lives has experienced 1 or more insomnia symptoms, with 13.4% of them meeting the criteria for

short-term or chronic insomnia disorder.[3] Once insomnia symptoms meet the criteria for insomnia disorder, chronicity is likely; 86% of patients with insomnia disorder continue to meet the criteria at 12 months, and 66% continue at 3 years. This calls for patient management within a chronic disease treatment model[3], with a focus on long term, sustainable strategies.

Despite being a well-known, pervasive, and ubiquitous disorder in medical practice, insomnia is often accompanied by mixed messages in standard education, and specialist support in Canada is limited. Hence, this article will focus on updated assessment and treatment strategies that primary care practitioners can implement, with a focus on chronic insomnia.

Consequences of Insomnia

While insomnia is frequently viewed as a nighttime sleep problem, 84% of people with insomnia report daytime symptoms such as irritability and increased daytime sleepiness.[6,7] Though often trivialized socially and by the medical system in general, insomnia is also associated with a decreased quality of life, academic difficulties, higher rates of absenteeism/presenteeism, motor vehicle accidents, and workplace disability. Moreover, insomnia is a risk factor for suicide, even in the absence of a mental health condition.[8]

Medically, chronic insomnia is strongly associated with an increased risk of many other chronic diseases, such as cardiovascular disease, chronic pain syndrome, depression, anxiety, diabetes, obesity, and asthma.[9]

Models of Insomnia

The sleep-wake-cycle is regulated by multiple neurotransmitter systems, some of which promote sleep and others that promote wakefulness.[10] Traditional sleep medications, such as benzodiazepines and z-drugs, enhance sleep by amplifying gamma-aminobutyric acid (GABA), a prominent inhibitory neurotransmitter. Wakefulness is thought to involve multiple neurotransmitter systems that are mediated by norepinephrine, serotonin, histamine, and more recently, primarily by the orexin system.[10,11]

It is now clear that a substantial portion of insomnia is not mediated simply by dysfunctional sleep promotion systems. The paradigm has shifted with evidence suggesting "hyperarousal" or "too much wakefulness," as a factor in many types of insomnia, with dysfunction of the orexin system as a key mediator.[11-13] Newly indicated agents are now available in Canada that specifically antagonize the orexin system, blocking central wakefulness-promoting activity.[14,15] These drugs facilitate sleep in a very different and more physiologically natural fashion.

Assessment of Insomnia

When a patient presents with insomnia, a history should be taken of insomnia symptoms including sleep latency, nighttime awakenings, wake time, and the regularity of their sleep pattern. Other associated sleep-related phenomena such as restless legs, snoring, or nighttime behaviours should be gathered. Short-term patient-recorded sleep logs can be very useful for assessing patterns.[9] Often, a comprehensive sleep history may not be realistic in primary care, thus, a summary questionnaire is provided for consideration.[16] As with any chronic disease, several visits may be required to fully assess and treat insomnia.

Objective measurements of sleep can include actigraphy, which measures sleep parameters via motor activity using a non-invasive accelerometer.[17] Actigraphy can supplement an insomnia workup; however, its accuracy may be compromised by medications, other sleep disorders, as well as commercial algorithms including other biological measures (i.e. Fitbit, Oura Ring, and smartphones). Some devices may also inaccurately exaggerate or extrapolate findings, leading to maladaptive cognitive beliefs about sleep. The ongoing use of sleep diaries or actigraphy outside of cognitive behavioural therapy is likely not necessary, may increase patient preoccupation with their sleep, and must always be taken into clinical context.

Another objective measurement is the sleep study. Referral for sleep testing may be necessary for insomnia patients with risk factors for, or symptoms of, other sleep disorders that may be contributing (e.g., sleep apnea, periodic limb movements). It also should be conducted in resistant or chronic cases and for patients on long-term hypnotic therapy.[7,9] In most areas of Canada, home sleep testing is the most accessible first option and is useful to confirm cases of moderate to severe obstructive sleep apnea. However, it cannot comprehensively assess all sleep problems, and milder cases of sleep apnea may be missed. Often, fully observed polysomnography is needed if home testing is negative, or if the patient has not responded to basic treatments.[16]

Comorbidities and Insomnia

Insomnia may occur as an independent disorder, a symptom/risk factor of comorbid sleep, psychiatric and/or medical conditions, or a combination of these. Approximately 75% of people with chronic insomnia have comorbid conditions[18] that must be reviewed and potentially treated. It is important to screen for comorbidities before, after, and during insomnia treatment, especially if the patient does not respond to therapy.[16] The comorbidity list is extensive (**Table 1**) and should be patient targeted (i.e. menopausal issues for women aged 40–60, or urinary issues for men over 60). However, each patient must be reviewed for the most common

Common Comorbidities and Medications Contributing to Insomnia		
Psychiatric Disorders	Mood disorders	Anxiety disorders
	ADHD, PTSD	Alcohol/substance use disorders
Other Medical Disorders	Neurologic (stroke, migraine)	Musculoskeletal (arthritis, fibromyalgia)
	Pulmonary (COPD, asthma)	Endocrine (hypothyroidism, hyperthyroidism, menopause)
	Digestive (GERD, colitis)	Cardiovascular (congestive heart failure)
	Chronic pain	
	Prostate and urinary	
Medications	Antidepressants	Decongestants and antihistamines
	Stimulants	Analgesics
	Antihypertensives	Herbal supplements
	Sedatives	Cannabis, Alcohol, Substances of abuse
	Anti-asthma drugs	

Table 1. Common Comorbidities and Medications Contributing to Insomnia; *courtesy of Atul Khullar, MD, MSc, FRCPC, DABPN (Cert sleep medicine), DABOM, FAASM, and Jennifer Swainson, MD, FRCPC, DABOM.*

Abbreviations: ADHD: Attention Deficit Hyperactivity Disorder, **COPD:** Chronic Obstructive Pulmonary Disease; **GERD:** Gastroesophageal reflux disease; **PTSD:** Post-Traumatic Stress Disorder

Assessment Pearls
Ask about next day functioning
Assess for contributing comorbidities
Establish a timeline and examine the interactions between comorbidities and insomnia
Understand the use of actigraphy and phone accelerometers
Consider sleep studies, being aware that home studies are only useful for confirming moderate to severe sleep apnea and full sleep studies are more definitive

Box 1. Assesment Strategies; *courtesy of Atul Khullar, MD, MSc, FRCPC, DABPN (Cert sleep medicine), DABOM, FAASM, and Jennifer Swainson, MD, FRCPC, DABOM.*

comorbidities, such as mood and anxiety disorders, sleep apnea, and chronic pain. In mental health populations, ADHD, trauma, and bipolar spectrum disorders may also need to be ruled out. Quick patient-rated scales, such as the Patient Health Questionnaire-9 (PHQ-9), Generalized Anxiety Disorder-7 (GAD-7) and the Snoring, Tiredness, Observed, Pressure, BMI, Age, Neck circumference and Gender questionnaire (STOP BANG), can be useful to efficiently assess comorbidities. The Insomnia Severity Index (ISI) can be used in conjunction with comorbidity scales to establish the severity of insomnia. Often, there is a bidirectional relationship between insomnia and many comorbidities, and treating one is likely to improve symptoms of the other.[19]

Clinically, it can often be difficult to decide whether to treat the insomnia or the comorbidity during a particular visit. Useful guides include establishing a timeline of how the insomnia relates to the comorbidity, whether it remains after treating the comorbidity, or assessing which treatment improves daytime functioning.[20] Often both the insomnia and the comorbidity are treated in parallel. (i.e. instituting Cognitive Behavioural Therapy for Insomnia [CBT-I] strategies while treating major depression and referring for a sleep study). **Box 1** reviews major assessment strategies.

Treatment

Goals of Therapy

The primary goal of any therapy for insomnia is to improve daytime functioning. Realistic treatment goals need to be set that aim to minimize daytime impairment, rather than focusing on sleep performance. A chronic poor sleeper will not become a good sleeper instantly.

Nonpharmacological Treatments

Sleep Hygiene and CBT-I

While good sleep hygiene is a cornerstone of any sleep intervention, it alone is not sufficient to treat chronic insomnia disorders. CBT-I is a first line insomnia treatment that incorporates sleep hygiene, along with stimulus control, cognitive restructuring, sleep restriction, and systemic relaxation.[7,9,21] It is highly effective in a wide variety of clinical settings,[22,23] with stimulus control and sleep restriction potentially being the most effective components.[24,25] CBT-I may be delivered in 2–8 sessions,

Advice for Cognitive Barrier	Elaboration
Keep realistic expectations	Even good sleepers do not always get 8 hours of quality sleep. There are individual differences in sleep requirements.
Do not blame insomnia for all daytime impairments	Consider alternative explanations that might contribute to those impairments.
Do not catastrophize after a poor night's sleep	Insomnia can be unpleasant, but it is not dangerous. The worst outcome: you will be sleepier the next day and sleep more soundly the next night.
Do not give too much importance to sleep	Even if sleep occupies one-third of your life, don't make it the sole focus of your existence.
Develop some tolerance to the effects of insomnia	Rearrange your schedule, but do not cancel planned activities.

Box 2 . Targeted cognitive barriers to sleep; *adapted from Driver H et al. Insomnia in adults and children. Available at: http://www.sleepontario.com/docs/INSOMNIA_BOOK_web.pdf.*

Clinical Tips – Selecting Pharmacotherapy
Consider medications that have demonstrated safety and efficacy for up to 12 months of use (i.e. eszopiclone, lemborexant, and daridorexant).
Tolerance/dependence risks of benzodiazepines and other z-drugs should be considered, but long-term use may be appropriate in some patients .
Risks of weight gain with sedating antidepressants and antipsychotics must be considered.
Fall risks associated with insomnia medications must be weighed; trazodone carries a similar fall risk to benzodiazepines and z-drugs.
For resistant insomnia, DORAs may be used off-label in combination with traditional hypnotics.
The monograph's recommended time-frame between hypnotic administration and driving or operating machinery should be adhered to with initial use. However, habituation to driving effects has been shown with regular use of hypnotics. Off-label sedatives have not been tested for driving safety and may be more deleterious. Substitutes such as alcohol, cannabis, OTC medications, and sleep deprivation itself can significantly impair driving.

Box 3. Clinical tips for selecting pharmacotherapy; *courtesy of Atul Khullar, MD, MSc, FRCPC, DABPN (Cert sleep medicine), DABOM, FAASM, and Jennifer Swainson, MD, FRCPC, DABOM.*

either individually, in groups, or through digital formats. The basic features of CBT-I should be included in any insomnia treatment plan. **Box 2**[26] outlines the major features that can be integrated within a family practice visit.

Unfortunately, even in well controlled clinical trials of CBT-I, 20–25% of patients do not respond,[27] and 30% drop out.[28] These rates may be even higher in primary care practice due to factors such as a lack of patient motivation, limited access to optimal types of CBT-I, unwillingness to change sleep and daytime behaviours, and patient selection. Patient selection errors can arise from both the type of patient, such as those lacking psychological mindedness and more commonly, the timing of the CBT-I treatment plan, particularly if it is initiated too early when the patient has an active comorbidity that precludes participation. Similar to other chronic diseases such as diabetes or hypertension, if patients do not adhere to recommendations for behavioural management and symptoms persist, pharmacotherapy should be introduced. Patients should not be stigmatized for using medication. Pharmacotherapy can work synergistically with CBT-I, often[29] offering immediate relief. This can enhance the therapeutic alliance and compliance to the CBT-I treatment plan. Similar to most behavioural therapies, CBT-I often shows a transient initial worsening of symptoms and may take 3–4 weeks to demonstrate improved sleep.[22,23,27]

Pharmacotherapy

There is a marked dichotomy between theory and practice regarding hypnotics. Many, but not all medications with a Health Canada–approved indication for the treatment of insomnia are potentially problematic when used long-term. Given the chronic nature of most insomnia cases, long-term medication use may be necessary, especially for patients with significant comorbidities, or a failure/inability to complete nonpharmacologic therapies. A clear rationale, ongoing assessment, and clinical awareness of the risks/benefits of long-term sedative use is essential.[16] Agents with evidence for safety and efficacy in long-term use should be strongly considered. Reluctance to provide patients access to long-term hypnotics may cause marked unnecessary suffering and often leads to unmonitored self-medication with more deleterious over the counter (OTC) medications or substances.

Pharmacotherapy Choices

In Canada, medications approved for insomnia include several benzodiazepines, z-drugs, a very low dose sedating antidepressant, and most recently, dual orexin receptor antagonists (DORAs) (**See Table 2**). Due to stringent exclusion criteria in clinical trials, older agents

Medications	Doses	Half-life (*hrs*)
Benzodiazepines		
Flurazepam (Dalmane)	15, 30 mg	40–250 (75 mean)
Nitrazepam (Mogadon)	5, 10 mg	16–38 (28.8 mean)
Temazepam (Restoril)	15, 30 mg	4–18 (8.8 mean)
Triazolam (Halcion)	0.125, 0.25 mg	1.5–2.5 (2 mean)
Z-drugs (Non-benzodiazepine sedative-hypnotics)		
Eszopiclone (Lunesta) (2020) *	1, 2, 3 mg	6 mean
Zopiclone (Imovane)	5, 7.5 mg	3.8–6.5 (6 mean)
Zolpidem (Sublinox) (2011)	SDT 5, 10 mg	2-3
Low dose antidepressant (wake blocker)		
Doxepin *	3, 6 mg	17 (51 metabolite)
Dual Orexin Receptor Agonist		
Lemborexant (Dayvigo) (2020) *	5, 10 mg	N/A**
Daridorexant (Quiviviq) (2023) *	50 mg	N/A**

Table 2. Medications Indicated for Insomnia in Canada; *courtesy of Atul Khullar, MD, MSc, FRCPC, DABPN (Cert sleep medicine), DABOM, FAASM, and Jennifer Swainson, MD, FRCPC, DABOM.*

*Designates data to support long term use

**Half life is not thought to correlate to clinical efficacy of the DORA

were also studied in insomnia populations without major comorbidities, making their efficacy less representative of most insomnia patients who have major comorbid conditions. Newer agents have long-term data that are more representative of the typical insomnia population.[30-32] Key points for using pharmacotherapy for insomnia are summarized in **Box 3**.

DORAs

The current evidence indicates that these drugs should be the first line of treatment for chronic insomnia due to their 1-year long-term efficacy data, and the lack of respiratory depression, tolerance, withdrawal, rebound insomnia, signal for falls, and abuse potential.[14,15] A recent meta-analysis of all insomnia pharmacotherapy options looked at their efficacy and safety. As a class, the DORAs were found to be the most preferable treatment in both areas, although this summary of evidence is limited by a lack of head-to-head comparison data.[33]

The major clinical differences between the 2 available DORAs are currently unclear. Lemborexant has been observed to increase rapid eye movement (REM) sleep, whereas daridorexant tends to increase sleep stages more evenly.[14,15] These features may be related to how they antagonize different types of orexin receptors, but the clinical significance remains unknown and requires further research. The clinical response to DORAs does not appear to be a class effect; therefore, if a patient does not respond to or tolerate one, the other should be considered.

These drugs should be considered as centralized sleep-wake stabilizing drugs rather than traditional sedatives. As such, DORAs work differently, possibly requiring 3–6 weeks of regular use to achieve their full effect.[30,32,33] The benefits often include more restorative sleep, the ability to return to sleep after awakening, and improved daytime functioning, rather than "a knock out pill". Patients must be counselled accordingly.

Benzodiazepines

All benzodiazepines, whether indicated for insomnia or not, have sedative and hypnotic properties but differ significantly in their onset, potency, and pharmacokinetics. Benzodiazepines are known to carry significant risks of adverse effects, such as delirium, falls, motor vehicle accidents, complex sleep behaviours, respiratory depression, cognitive impairment, memory issues, abuse, dependence, and withdrawal symptoms with long-term use.[34] These risks are of particular concern for the elderly or medically ill.[35] Although some studies have suggested benzodiazepines and z-drugs increase the risks of mortality and dementia, this has likely been overestimated due to study design; newer data suggest no clear association.[36,37] Risks may be attenuated by short duration and possibly intermittent use.[38]

Z-Drugs

The z-drugs, including zopiclone, eszopiclone, and zolpidem act on the same receptors as benzodiazepines and have similar therapeutic effects for overall sleep. However, they have more limited effects on sleep structure and somewhat fewer adverse effects, including cognitive impairment, dependence, tolerance, and rebound insomnia, especially with eszopiclone.[31,39-42] They have demonstrated 10–40 times less muscle relaxant effect than benzodiazepines,[43] and have not demonstrated any worsening of sleep apnea at recommended doses.[44]

The differences between benzodiazepines and z-drugs as well as among the z-drugs themselves, are related to their relative affinity for the various subunits of the GABA-A alpha receptor.[41] Zopiclone is the most similar to benzodiazepines among the z-drugs, while eszopiclone is the least similar.[41] Eszopiclone appears to have a safer long-term use profile and is likely less problematic than the other z-drugs.[31] It could be considered as a second- or third-line long-term strategy.

In Canada, the monographs for benzodiazepines and z-drugs typically indicate short prescriptions of 7–10 days and no more than 30 days. These guidelines are clearly for short-term insomnia, but they are arbitrarily determined, potentially damaging, and not always evidence-based. Nonetheless, frequent follow-up (3–6 weeks) and limited dispensing (14–60 days) is warranted, particularly at the beginning of insomnia treatment.

Similar to benzodiazepines, zolpidem and zopiclone are usually considered for short-term acute or intermittent use. Long-term use should be considered only for severe or comorbid insomnia cases that have failed other treatments or when these drugs have demonstrated a substantial improvement in daytime functioning.[16]

Low Dose Doxepin

Low dose doxepin (LDD) (3–6 mg) has a reasonable data set supporting its use for sleep maintenance and includes elderly specific data,[45] however, its effectiveness has not translated well to clinical populations. The absorption of LDD is quite variable and is significantly reduced if taken within 3 hours of food intake, and its effects on sleep onset are inconsistent.[45] LDD has limited public coverage in Canada. From a practical standpoint, a slightly higher dose of doxepin (10–40 mg) which is publicly reimbursed, is often used off-label, though it carries a higher potential for side effects.

Off-Label Insomnia Medications

Several medications are commonly used off-label for insomnia (See **Table 3** from[46]). These include sedating antidepressants, antipsychotics, alpha-2 delta ligands (anticonvulsants), and other benzodiazepines. As a rule, these medications have limited evidence supporting their use and have their own deleterious side effects. However, when insomnia is comorbid with another condition or resistant to standard treatments, rational use of off-label agents may be appropriate.[47,48] Public coverage in Canada for safer, approved agents with long-term efficacy is currently limited, which may also lead to the use of off-label agents.

Many off-label medications (tricyclic antidepressants [TCA's], mirtazapine, quetiapine, gabapentin/pregabalin) can cause significant weight gain, which can independently decrease sleep quality.[49] Low dose quetiapine (25–100 mg) is commonly used in clinical practice for insomnia, but the supporting data is of poor quality, and it may not help sleep architecture.[50] It should not be used for sleep except possibly in cases of significant comorbidities such as depression or severe generalized anxiety.

Data supporting the use of trazodone (50–200 mg) for insomnia without comorbidities is restricted to small studies.[51] Nevertheless, its widespread use is due to some clinical successes, and support from some experts in the field.[16,52] Though trazodone does not appear to have the memory, abuse, or rebound insomnia issues observed with benzodiazepines or z-drugs, it and other sedating antidepressants can have a similar fall risk.[53] Insomnia doses of trazodone and other sedating antidepressants usually do not have an appreciable antidepressant effect, however, trazodone has several theoretical receptor benefits that could independently promote sleep.[51] It could be considered a second- or third-line treatment option.

Over the Counter Medications

When assessing insomnia pharmacotherapy, a history of OTC medication use for sleep must be reviewed, as this is a common practice due to easy availability and misleading promotion.[54] Melatonin is frequently used by patients, and while it may help certain subgroups of patients with insomnia (i.e. elderly, neurodevelopmental disorders, children, and those with ADHD), it is not recommended overall due to a small and inconsistent overall effect size.[55] Although there is no consistent data indicating a lack of safety with melatonin, the lack of regulation on the purity and manufacturing of melatonin supplements in North America can lead to adverse outcomes, so caution is advised.[56] Magnesium supplementation is known to help overall sleep quality, but does not directly address insomnia.[57] As a rule, OTC sedating antihistamines should be avoided.[16]

Substances

Alcohol is well known as one of the most harmful substances for sleep, yet it remains commonly used, therefore, patients must be asked about their intake.

Drug Class	Reasons for Use	Considerations
Sedating antidepressants: • Mirtazapine (low-dose) • Tricyclic antidepressants (including doxepin >10 mg) • Trazodone	• Insomnia with mood disorder • Mirtazapine may be useful for low appetite or alcohol use issues • Comorbid migraine and other central sensitivity syndromes (TCAs only)	• Next-day sedation and motor restlessness can occur • TCAs also associated with anticholinergic adverse effects • Both TCAs and mirtazapine may be associated with weight gain • Trazodone may be better for sleep maintenance, not overall efficiency
Antihistamines: • Chlorpheniramine • Diphenhydramine • Hydrazine	• Insomnia associated with histamine-mediated sleep disturbance (e.g. allergies, atopic dermatitis) • Very short term • Rarely recommended due to side effect profile	• Excessive risk of daytime sedation, psychomotor/cognitive impairment and anticholinergic toxicity, especially in the elderly.
Anticonvulsants: • Gabapentin, low-dose • Pregabalin, low-dose	• Insomnia associated with centralized pain syndromes (e.g. fibromyalgia, neuropathic pain, restless legs syndrome)	• Weight gain and next-day sedation are common adverse effects • CNS depression and cognitive impairment may also occur
First-generation antipsychotics: • Chlorpromazine, low-dose • Methotrimeprazine • Loxapine	• Insomnia associated with very resistant bipolar disorder or schizophrenia	• Not recommended for insomnia in the absence of comorbidities, due to unacceptable risk of anticholinergic and neurological toxicity
Second-generation antipsychotics: • Olanzapine • Quetiapine • Paliperidone • Clozapine • Brexpiprazole • Risperidone • Lurasidone • Asenapine	• Insomnia with bipolar disorder or schizophrenia (olanzapine, quetiapine, clozapine, paliperidone, risperidone, asenapine, lurasidone) • Insomnia with major depressive disorder (augmentation with brexpiprazole) • Insomnia with generalized anxiety disorder (quetiapine)	• Not recommended for insomnia in the absence of comorbidities, due to unacceptable risk of metabolic syndrome • Metabolic issues and weight gain must be monitored
Other benzodiazepines: • Diazepam • Clonazepam • Lorazepam • Nitrazepam • Alprazolam • Oxazepam	• Insomnia with anxiety disorders or severe hyperarousal • Insomnia associated with restless legs syndrome (clinically done, minimal evidence to support)	

Table 3. Off-Label Agents Commonly Used for Comorbid Insomnia; *adapted from Khullar, A., 2021.*

Abbreviations: TCAs: tricyclic antidepressants

Strategies for Switching Insomnia Therapies
ADD, THEN TAPER
Use if the first drug is somewhat effective or has a risk of withdrawal. Add the new drug to help the patient sleep before taking away the old drug
Use this for initiating DORAs for someone taking other sleep medications
CROSS TAPER
Decrease the first drug and start the second
Use this when switching therapies to reduce side effects.
DIRECT SWITCH
Stop the first drug and immediately start the second
Consider this for same class switches (i.e. DORA to DORA, z-drug to z-drug)

Box 4. Strategies for switching insomnia therapies; *courtesy of Atul Khullar, MD, MSc, FRCPC, DABPN (Cert sleep medicine), DABOM, FAASM, and Jennifer Swainson, MD, FRCPC, DABOM.*

Abbreviations: DORAs: dual orexin receptor agonists

Cannabinoids are not prescribed for insomnia, but may improve sleep quality in those with comorbidities that have evidence supporting the benefits of cannabis, such as chronic pain.[58] The effects of various cannabinoids on sleep are complex, multidimensional, and dependent on the mixtures of the active ingredients, necessitating further study.[58] Self medication with alcohol and/or cannabinoids for sleep often strongly indicates not only insomnia, but also a major comorbidity that must be reviewed.

Switching and Deprescribing (Box 4)

Deprescribing or switching benzodiazepines and z-drugs should be considered for appropriate patients, especially those with advancing age, since side effects such as balance issues and memory complaints become increasingly prevalent.[46]

Plans to taper or switch medications require careful consideration of factors including the duration of medication use, relevant comorbidities, current efficacy, and the level of improved daytime function on the current medication.[59] Indiscriminately restricting, tapering, or stopping stable low doses of benzodiazepines or z-drugs may lead to more harmful behaviours such as increased unmonitored OTC medication or substance misuse.[60] A harm-reduction and cost-benefit approach is essential, as resources for tapering/discontinuation may be limited or the drug may clearly be improving daytime functioning and quality of life.[60]

Patient motivation and alliance is critical in this process. Strategies should include motivational interviewing rather than fear-based reasoning, along with understanding that a complete withdrawal may not be achievable, and that a lower dose can still yield significant benefits. Plan to taper and withdraw hypnotics during a low-stress period. For patients on long-term medications, the tapering schedule should be slow and gradual, often extending over many months.[46]

During the switching process, the patient may require more than one hypnotic, especially when switching from long-term z-drugs and benzodiazepines to DORAs. A common error is the premature withdrawal of the previous sleep medication before waiting 4–6 weeks for more robust efficacy of DORAs. Off-label, smaller doses of sleep medications from different classes may be necessary for patients with severe insomnia or multiple comorbidities and this is a common practice with the DORAs and z-drugs in Japan.[61]

Conclusions

It is important for primary care physicians to be mindful of the potential impacts of insomnia on both patient functioning, and comorbid chronic diseases. Insomnia is not simply an inconvenience; if left untreated, it may contribute to substantial morbidity and even mortality. While screening and taking a history for insomnia can be challenging during a short clinic visit, there are strategies to address this efficiently which can often help with the management of other chronic diseases. Moving beyond sleep hygiene and offering targeted achievable CBT-I strategies and informing patients about local resources is critical and can be accomplished during a short visit. Clinicians should consider the risks/benefits of both indicated and non-indicated pharmacotherapy and become familiar with newer treatment options such as the DORAs, which offer greater safety and efficacy and are designed with potential long-term use in mind.

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