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1.5 CEUs

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This lesson has been approved for 1.5 CEUs by both the Canadian Council on Continuing Education in Pharmacy and by l'Ordre des pharmaciens du Quebec. Accreditation of this program will be recognized by CCCEP until November 9th, 2014.

LEARNING OBJECTIVES

Upon successful completion of this lesson, you should be able to:

1. Develop an understanding of the epidemiology and pathophysiology of alcohol dependence.
2. Enhance their knowledge of the pharmacological management of acute alcohol withdrawal and treatment of alcohol dependence.
3. Appreciate the role of the pharmacist in identifying and monitoring acute and chronic outcomes of treatment and recovery.
4. Understand and appreciate the interface between psychiatric co-morbidities and alcohol use and dependence.
5. Be able to recognize the common signs and symptoms of alcohol use disorders in health professionals.

To successfully complete the post-test for this lesson, you may need access to a recent edition (e.g., 2010, 2011) of the *Compendium of Pharmaceuticals and Specialties* (CPS) for additional information.

INSTRUCTIONS

1. After carefully reading this lesson, study each question and select the one answer you believe to be correct.
   For immediate results answer online at www.CanadianHealthcareNetwork.ca.
2. To pass this lesson, a grade of at least 70% (11 out of 15) is required. If you pass, your CEU(s) will be recorded with the relevant provincial authority(ies). (Note: some provinces require individual pharmacists to notify them.)

ANSWERING

For immediate results, answer online at **www.canadianhealthcarenetwork.ca.**

A Pharmacist's Overview of Alcohol Dependence: The Path to Abstinence

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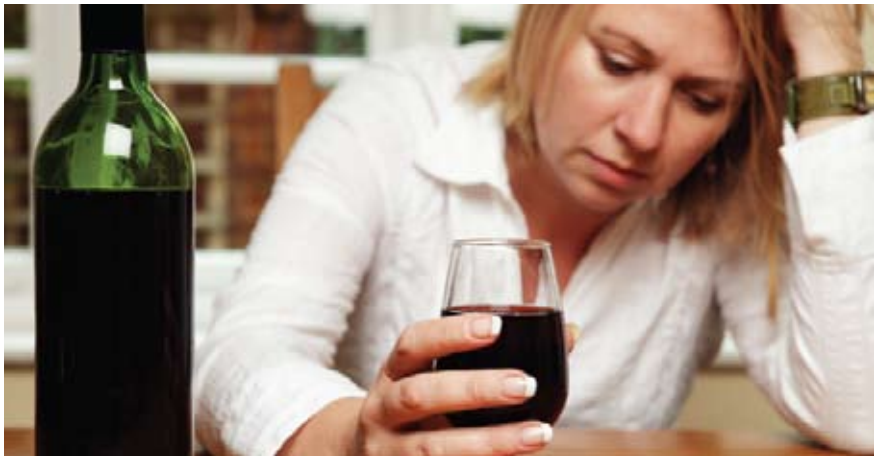

Introduction

Alcohol dependence is a chronic disorder with genetic, psychological and social triggers that often follows a deteriorating and relapsing course.[1] It is a maladaptive pattern of alcohol use that becomes abuse, even in the face of evidence that to continue the actions would cause harm to the drinker or others.[2] Alcohol use, misuse, abuse and dependence are on a spectrum of progressive severity, describing a compulsive pattern of behaviours and actions secondary to an addiction to alcohol.[2]

Without a pharmacological adjunct to behavioural interventions, clinical outcome is poor with up to 70% of patients resuming drinking within one year.[1] This is true even in patients partaking in a residential inpatient rehabilitation program, as they have a one-year abstinence rate of 25% at best.[3]

Prevalence

Alcohol dependence ranks third on the list of preventable causes of morbidity and mortality in the U.S.[1] The U.S. 12-month prevalence of alcohol dependence in 2001–2002 was 3.8%, representing 7.9 million Americans.[4] The lifetime prevalence of alcohol dependence in Western countries is estimated to be 7–12.5%.[5] The lifetime incidence of alcohol dependence in people up to the age of 28 is estimated to be 9.2%.[6] The 2005 Canadian Addictions Survey found that 79.3% of Canadians 15 years of age or older consume alcohol, with 44% of these using alcohol at least once weekly. Moreover, 24.2% of current or former alcohol users stated that their drinking had caused harm to themselves or others at one point.[7]

Risk factors

Alcohol abuse and alcohol dependence are associated with several risk factors including younger age at first drink, male sex, family history of alcohol dependence (genetics), smoking, illicit drug use, and co-existing psychiatric illnesses.[7]

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table 1

Number of drinks considered to be within "safe limits" (1)

For men under 65: *≤4 standard drinks per day and ≤14 standard drinks per week.*
For women under 65: *≤3 standard drinks per day and ≤7 standard drinks per week*
For men and women over 65: *≤3 standard drinks per day and ≤7 standard drinks per week*

*** 1 standard drink = approximately 15 mL of absolute alcohol, 300 mL of beer, 120 mL of wine, 30 mL of 100 proof liquor (50% alcohol)*

table 2

The CAGE Screening Questionnaire(5)

1. Have you felt that you need to **Cut down** on your drinking?
2. Have you ever felt **Annoyed by criticism** of your drinking?
3. Have you ever felt **Guilty** about drinking?
4. Have you ever thought you needed a drink **first thing** in the morning (Eye-opener)?

Interpretation of the Questionnaire:
• Two affirmative answers predict with **94% sensitivity** that the person has an alcohol misuse disorder.

Patient evaluation

Patients with alcohol dependence rarely present to the community pharmacy with overt signs of intoxication such as slurred speech, impaired balance, loss of muscle coordination, erratic behaviour, etc. Patients who self-report ingesting alcohol should be asked: **"How much beer, wine, or other alcoholic beverages do you drink?"** This may be followed by asking the patient if he/she is concerned about their level of alcohol consumption. These questions may open a dialogue with the patient and subsequent actions can be based on the patient's response. The pharmacist can compare the reported alcohol consumption with what is considered drinking within "safe limits" (Table 1). If the patient is exceeding "safe limits," the pharmacist may provide education on recommended levels of alcohol consumption and work with the patient to determine if additional interventions are required.

In order to help gauge the patient's risk for alcohol dependence, a five-minute screening questionnaire called The Alcohol Use Disorders Identification (AUDIT) test could be used.(8) The CAGE screening questionnaire (Table 2) is another validated tool for alcohol dependency risk, consisting of four simple questions with 94% specificity for identifying problem drinkers.(7,9)

Neuropathophysiology

Alcohol can have an impact on various neurotransmitter systems. The pathophysiology of alcohol dependence involves the cortico-mesolimbic-dopamine system and the nucleus accumbens, which is the pleasure-reward system in the brain. These neurological networks govern the reinforcing effects of alcohol and enhance alcohol's abuse liability. The reinforcing/compulsive and "needing" effects of alcohol affect neurotransmitter systems involved with drive, mood and cognition. Several neurotransmitter systems interact and modulate the cortico-mesolimbic-dopamine system, including the dopamine, serotonin, γ-aminobutyric acid (GABA), glutamate, opioid, and cholinergic systems.(1)

Dopamine levels in the mesolimbic system reinforce the acute reinforcing effects of alcohol because dopamine regulates the pleasure-reward system.(10) Reduced activity of the serotonin receptor system is related to decreased impulse control.(10) Alcohol dependence alters the GABA neurotransmitter-receptor complex.(11) GABA is facilitated by alcohol, while the inhibition of GABA is responsible for alcohol withdrawal symptoms and seizures.(10,11) Alcohol inhibits glutamate, which thus inhibits binding to the N-methyl-D-aspartate (NMDA) receptor complex, resulting in a depressive response, and may link to depression.(10,11)

One hypothesis suggests that the opioid system has a modulatory role in the dopaminergic system. Alcohol consumption stimulates the release of endorphins that bind to opioid receptors, stimulating the release of dopamine and activating the pleasure-reward system, which in turn results in the craving for more alcohol.(10) However, others suggest that because the release of endorphins is not sustained with prolonged alcohol exposure, there may be adaptive changes to the neuronal systems in order to overcome the effects of alcohol. Thus, when alcohol is no longer consumed and there is no remaining endorphin release, there may be increased discomfort, discontinuation and increased craving due to previous neuroadaptive change.(12)

The concept of recovery versus relapse

"Recovery" is defined as the period where the alcohol-dependent person ceases alcohol consumption and begins a period of abstinence.(13) "Relapse" is usually defined as the ingestion of any alcohol by a person who has previously been abstinent.(14) However, an opposing view considers "relapse" to have occurred only after a set number of drinks or drinking episodes, while an isolated drink or episode is called a "slip."(14)

Alcohol withdrawal

No set amount of alcohol intake will definitely produce withdrawal symptoms, as there is a wide degree of inter-patient variability. A good history is critical to determine the patient's total alcohol intake. Some studies suggest it is not only the quantity but also the duration of alcohol use that determines the likelihood of developing withdrawal.(11) However, patient self-reporting tends to under-rate consumption and collateral family history may be more accurate.(15) Alcohol intake must be considered in assessment of the patient and selection of pharmacotherapy.

Minor withdrawal symptoms, which are

table 3
Clinical Institute Withdrawal Assessment for Alcohol Revised (CIWA-Ar) Scale[11,20]

Scoring 0-7 (least to worst) on the following withdrawal symptoms:

- ❑ Nausea and vomiting
- ❑ Headache
- ❑ Sweating
- ❑ Auditory disturbances
- ❑ Anxiety
- ❑ Visual disturbances
- ❑ Agitation
- ❑ Tactile disturbances
- ❑ Tremor
- ❑ Cloudiness of the sensorium

Interpretation of the Questionnaire:

> 20	Patient may be experiencing severe withdrawal
10–20	Patient may be experiencing moderate withdrawal
<1	Patient may be experiencing mild withdrawal symptoms that may or may not require pharmacotherapy intervention

almost always addressed in the community, include insomnia, tremulousness, changes in blood pressure and heart rate, mild anxiety, anorexia with nausea and vomiting, headache, diaphoresis, and palpitations. These symptoms often occur within six hours of ingesting the last drink.[11] In chronic alcohol users, these symptoms of early withdrawal may occur even when there are still detectable blood alcohol levels.

Moderate withdrawal symptoms present with a more severe presentation of the minor withdrawal symptoms, albeit not to the point in most patients of seizures or delirium tremens (DTs). Clinically there is a rise in systolic and diastolic blood pressures, body temperature, and heart rate. Anxiety and other psychiatric presentations may display in this period, and the patient at this point may require hospitalization.[16-18]

Major alcohol withdrawal symptoms are less common, generally occurring in chronic alcohol users and often requiring hospital interventions. The most problematic major alcohol withdrawal symptom is the grand mal seizure, which may occur within 12–48 hours after the last drink. Alcoholic hallucinations, on the other hand, may develop within 12–24 hours of abstinence. Signs of DTs are exceptionally rare, but may start to develop at 72 hours, most often in long-term, heavy chronic alcohol users who do not get treated for withdrawal symptoms early enough. Symptoms of DTs may persist for five to seven days and include hallucinations, disorientation, fever, tachycardia, agitation and diaphoresis. While infrequent, DTs may be severe, potentially life-threatening, and associated with a five per cent mortality rate.[11]

Alcohol withdrawal management

The treatment of alcohol dependence involves acute management of withdrawal followed by long-term rehabilitation.[19] Early intervention with effective treatment of acute withdrawal symptoms may prevent the development of major withdrawal symptoms, including seizures. While the management of more severe alcohol withdrawal may need to take place in a hospital inpatient setting, management of mild and most moderate alcohol withdrawals often occurs in the community setting. As such, community pharmacists are ideally situated to provide education on medication, addressing potential concerns that might arise, and monitor the patient to ensure positive outcomes.

Should withdrawal symptoms occur, the pharmacist can utilize several tools to assess the severity of symptoms and the need for pharmacological interventions to manage those symptoms, and/or the need for medical detoxification. The most widely used tool in a hospital setting is the Clinical Institute Withdrawal Assessment for Alcohol Revised (CIWA-Ar) Scale (Table 3).[11,20] While this tool may be difficult for community pharmacists to utilize in their practice setting, it is important to be aware of this common scale and understand how it can be used to determine the patient's severity of alcohol withdrawal.

Benzodiazepines are the cornerstone of pharmacological therapy for alcohol withdrawal. The degree of withdrawal the patient experiences—as indicated by the CIWA-Ar scale—defines the dose and frequency of administration. Diazepam and lorazepam are most commonly used, with lorazepam preferred in patients with liver impairment (Table 4).[1,21-23] In certain patient populations with co-morbidities, such as chronic obstructive pulmonary disease, benzodiazepines should be used cautiously due to their potential to induce respiratory depression and the benzodiazepine loading dose may need to be adjusted downwards.[23,24]

While benzodiazepines remain the gold standard for treatment of alcohol withdrawal, treatment of acute alcohol withdrawal may be augmented using dopaminergic agents, such as baclofen[25] or clonidine.[26] Neuroleptics are often used to prevent agitation and attenuate hallucinations and DTs, but they may lower the seizure threshold.[10]

Early intervention may decrease the morbidity and mortality associated with DTs. Patients with DTs and concurrent alcohol-related diseases, such as pancreatitis or cirrhosis, or pulmonary disease, are at especially high risk of morbidity (e.g., changes in oxygen consumption and delivery, respiratory alkalosis, hypovolemia, severe hypophosphatemia, hypomagnesemia) and potential death.[11] These patients must also be monitored for QTc prolongation in hospital, as the further addition of QT-prolonging medications such as quetiapine may increase risk of developing torsades.

Supportive care and interventions to manage dehydration and vitamin deficiencies (thiamine, folic acid and vitamin B12) may also be required. Wernicke encephalopathy results from severe thiamine deficiency and often presents with acute confusion/delirium, ataxia and ocular changes (nystagmus, ophthalmoplegia) during alcohol withdrawal. High-dose intravenous thiamine (250 mg) is required

table 4

Treatment of Alcohol Withdrawal[10-12]

BASED ON CIWA	SYMPTOMS	MEDICATIONS
Mild	SBP 90–150 DBP <100 HR 90–110 Temp 37.7°C Tremulousness, agitation, insomnia	Diazepam PO 5–10 mg PO Q4H prn OR Lorazepam 1–2 mg PO Q4H prn x 1–2 days
Moderate	SBP 150–200 DBP 100–140 HR 110–140 Temp 37.7°C–38.3°C Tremulousness, agitation, insomnia	Diazepam 10–20 mg PO/IM QID prn OR Lorazepam 2–4 mg PO/IM QID prn and taper over 5 days
Severe	SBP > 200 DBP >140 HR >140 Temp 38.3°C Tremulousness, agitation, insomnia, and risk of DTs	Diazepam 10–20 mg PO/IV/IM Q1H prn while awake and titrate to sedation OR Lorazepam 1–4 mg PO/IM/IV Q1H prn

urgently in severe cases of alcohol withdrawal because severe thiamine deficiency induces biochemical abnormalities that can lead to irreversible brain damage and death in 17–20% of people[13] (Table 5).[11,19,21,22] However, mild and moderate cases of alcohol withdrawal can be sufficiently managed with oral thiamine at doses of 50–100 mg/day.[13]

After acute alcohol withdrawal, the patient should consider their future approach to alcohol consumption. One approach is 'abstinence,' which entails the complete elimination of alcohol consumption. An alternative approach is 'harm reduction', in which alcohol consumption is significantly reduced.[10,19] There is much debate as to which approach is preferred.[10,19] However, reduction of alcohol consumption may not address the psychological and reward system patterns (related to dopamine) that occur with chronic alcohol use. Due to the long-term consequences of alcohol dependence and its impact on the patient and family, choosing abstinence, where possible, is preferred.

Alcohol dependence: maintenance therapy

BEHAVIOURAL MODIFICATIONS

Behavioural interventions encourage the patient to set goals to reduce heavy drinking or achieve abstinence. Brief interventions, such as a behavioural compliance enhancement treatment or short-term medical management, may be sufficient to optimize pharmacological treatment such that there may be no need for more intensive psychotherapy or long-term medical management.[1] Intensive psychotherapy has been shown to be less effective than a brief intervention plus placebo. Pharmacotherapy may be instituted, coupled with a brief intervention in a clinician's general practice. Intensive behavioural interventions, typically offered in alcohol treatment programs, are appropriate for those who do not respond to motivational interviewing or risk-benefit open dialogue.[1] Psychotherapy can include motivational interviewing, cognitive behavioural therapy, or a 12-step program (e.g., Alcoholics Anonymous).[19]

Pharmacotherapy

Without a pharmacological adjunct to behavioural interventions, clinical outcome is poor with up to 70% of patients resuming drinking within one year.[1] This is true even in patients partaking in a residential inpatient rehabilitation program, as they have a one-year abstinence rate of 25% at best.[3]

The most promising treatments to reduce heavy drinking or prevent relapse include agents that modulate the cortico-mesolimbic dopamine system through the opioid, glutamate, GABA, or serotonergic systems.[1] Pharmacists should recognize how these medications work, whether by receptor affinity or by decreasing cravings or as a deterrent.

If treatment is successful, the physical consequences of long-term alcohol consumption, as well as psychosocial aspects, marital relationships and work life, should improve.[10] However, the chances for success are enhanced by patient commitment and interventions geared towards abstinence.

DISULFIRAM

Disulfiram inhibits aldehyde dehydrogenase, thus preventing the metabolism of acetaldehyde, alcohol's predominant metabolite. The subsequent accumulation of acetaldehyde in the blood causes flushing, sweating, nausea, severe vomiting and tachycardia if a patient ingests alcohol while taking the drug. Disulfiram acts as deterrent to alcohol consumption; since it does not reduce cravings, compliance is key to its success.[1,10,27]

Disulfiram (Antabuse) is no longer commercially available in Canada, but may be prepared by compounding pharmacies. The usual dosage range is 250–500 mg/day.[28] In the absence of alcohol, disulfiram is fairly well tolerated with a garlic or metallic after-taste being the most common complaint.[1,10,27] Alcohol from other sources, such as OTC cough and cold products, mouth washes, hand sanitizers and foods to which alcohol has been added after cooking, may cause this disulfiram reaction.[29]

NALTREXONE

Naltrexone is an opioid antagonist that modulates the subcortico-mesolimbic dopamine system, thus reducing the pleasurable effects and craving for alcohol.[10]

In a recent Cochrane Review, 50 randomized controlled trials (involving 7,793 patients) comparing the efficacy of naltrexone with placebo with respect to reduction in the risk of heavy drinking were analyzed. This analysis found that

table 5

Supportive Care for Alcohol Withdrawal[22]

1. Thiamine 100–250 mg PO/IM/IV daily x 3+ days
2. Multivitamin 1 tablet PO daily
3. Folic acid 1–5 mg PO daily

Monitor liver function tests, bilirubin, complete blood count, electrolytes, serum creatinine, and vital signs

naltrexone reduced the risk of heavy drinking to 83% of the risk in the placebo group, and decreased drinking days by about 4%. Significant differences between naltrexone and placebo were also observed for the secondary outcomes, including: heavy drinking days, amount of consumed alcohol, and gamma-glutamyltransferase levels. No significant difference was observed with respect to naltrexone's effect on probability of resuming alcohol consumption. Therefore, it can be concluded that naltrexone is an effective and safe medication to use to treat alcohol dependence.[30]

The most common adverse effects of naltrexone include nausea, headache, and somnolence. To minimize side effects, naltrexone may be started at 12.5 mg per day, gradually titrating to 50 mg/day for a minimum of two months to minimize the risk of relapse.[1,10] However, the most common starting and maintenance dose is 50 mg/day.

A depot injection is available for patients in other countries, including the U.S., but not yet in Canada.

ACAMPROSATE

Acamprosate is an NMDA antagonist that modulates the dysregulation between excitatory and inhibitory neurotransmission thought to result from chronic alcohol use.[27] It inhibits the elevated glutamate transmission and NMDA receptor activation that occur in alcohol dependence and withdrawal.[10] Acamprosate blocks the cravings experienced in the absence of alcohol.

Several European studies have demonstrated acamprosate's efficacy over placebo for the treatment of alcohol dependence, including increased abstinence rates.[1,10] However, one large double-blind American trial found the percentage of abstinent days did not differ significantly across study groups.[31] A second large double-blind American study also found that acamprosate showed no efficacy with or without behavioural interventions.[9] These conflicting outcomes may be secondary to differences in the patient populations, different definitions of relapse and abstinence, and the psychosocial interventions in the American studies that may have masked the effect of the medication.[15,32] The dosing of acamprosate is 333–666 mg three times a day.[33] Acamprosate is generally well tolerated with the most common side effect being diarrhea.[10]

OTHER MEDICATIONS

Several other medications have been used in alcohol abstinence maintenance, although they have not been formally approved by Health Canada for this purpose.

Topiramate reduces the cortico-mesolimbic dopamine system and increases GABA levels via the GABA-A receptor.[1,27] In alcohol abstinence maintenance, it also blocks glutamate, which reduces neuronal excitability.[27] Two large placebo-controlled clinical trials have demonstrated that topiramate (25–300 mg per day, titrated up through gradual escalation) improved all drinking outcomes, including a reduction of heavy drinking and the promotion of abstinence.[1,27] In one 12-week trial, topiramate was shown to also reduce alcohol craving.[34]

Ondansetron is a 5-HT_3 antagonist. Two placebo-controlled trials of ondansetron 4 µg/kg given twice daily reduced the quantity and frequency of alcohol intake over six and 12 weeks. However, long-term efficacy, safety and tolerability have yet to be established.[10]

Baclofen is an agonist at the presynaptic GABA-B receptor that suppresses the mesolimbic dopamine system neurons. It is a promising medication for reducing alcohol intake and craving, as well as for enhancing abstinence in alcohol-dependent patients.[20] It also has a place in therapy in those patients with underlying liver impairment.[20] Side effects include headaches, insomnia, nausea, hypotension, urinary frequency and, rarely, excitement and visual abnormalities. A discontinuation syndrome may present with hallucinations, anxiety, perceptual disturbance, and extreme muscle rigidity, with or without spasticity. To avoid this, baclofen should not be abruptly stopped but gradually tapered.[1]

Alcohol dependence and psychiatric co-morbidities

Schizophrenia, major depressive disorder and anxiety may co-exist with excessive alcohol consumption. It may be difficult to determine if alcohol dependence is the root disorder or a symptom of a pre-existing psychiatric co-morbidity. Treatment and awareness of these underlying psychiatric conditions may have a beneficial, but not "curative" effect in both reducing alcohol consumption and increasing retention in treatment programs.[10] As discussed above, changes in dopamine levels (e.g., diminished dopamine turnover) may correspond with decreases in serotonin activity, which is linked to depression.[10,35,36] Serotonergic dysfunction may be linked to decreases in long-term success in treating alcohol dependence.

Depressive symptoms also occur frequently in those with alcohol dependence.[1] They are especially common during the withdrawal period and the accompanying depressive symptoms (e.g., insomnia, anxiety, dysphoric mood) may complicate the course of treatment and recovery.[1] Pharmacists should watch for these depressive symptoms, including suicidal ideation, in patients recovering from alcohol dependence.[1]

Pharmacotherapy of concurrent alcohol dependence and depression

SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRIS)

SSRIs increase serotonin levels by inhibiting serotonin reuptake at the synapse. SSRIs may indirectly improve patient outcomes in alcohol dependence treatment (e.g., abstinence) by increasing serotonin levels, thus alleviating underlying depression.[10] Fluoxetine, sertraline, and citalopram have been studied in clinical trials, with sertraline also impacting on dopamine as well as serotonin. However, the evidence to support their efficacy is weak.[10]

QUETIAPINE

Quetiapine is an atypical antipsychotic with multiple receptor affinities (predominately serotonin and dopamine), which may be beneficial in alcohol dependence treatment.[27] One hypothesis suggests quetiapine's benefit is secondary to the structural similarity of one of its metabolites to methadone, which may subsequently act as a pleasure-reward modulator.[37] Additional effects of quetiapine on drinking outcomes may be related to its beneficial effects on mood, anxiety and sleep, which may help alleviate protracted withdrawal symptoms as well as address co-morbidities.[3] The efficacy of quetiapine in treating alcohol dependence was demonstrated in a small placebo-controlled trial, but further research is warranted.[38]

Communication skills in alcohol dependence

Counselling the patient with alcohol dependence requires awareness on the part of the pharmacist to ensure that any barriers—including environmental, personal biases, patient, time and behavioural—are removed or minimized. These barriers contribute to 90% of the nonverbal communication that is transferred to the patient. We must be aware of our own belief systems and cultural awareness about alcohol use prior to engaging in dialogue.[39] Awareness of this phenomenon will diminish the chance of "scolding" or "lecturing" the patient, actions that add to their guilt, and shame, which may increase the risk of failure of treatment. Empathy and empowerment are keys to prevent adding further "***dis-ease***" to the disease of alcohol dependence. Helpful resources for pharmacists and patients/families are presented in Table 6.

table 6

Additional Resources for Pharmacists and Patients

Health Canada – Alcohol & Drug and Prevention Publications
A useful website for pharmacists containing information about youth, seniors and special populations with alcohol and substance abuse, including rehabilitation.
www.hc-sc.gc.ca/hc-ps/pubs/adp-apd/index-eng.php

Alcoholics Anonymous/Al-Anon/Alateen
Resources for patients and family members seeking support while drinking or when in recovery.
www.alcoholics-anonymous.org; www.al-anon.alateen.org/

Centre for Addiction and Mental Health (CAMH)
A comprehensive resource for patients interested in learning more about alcohol dependence including diagnosis, impacts, rehabilitation and recovery.
www.camh.net/About_Addiction_Mental_Health/AMH101/top_searched_alcohol.html

National Institute on Alcohol Abuse and Alcoholism
A resource for healthcare professionals including clinical guidelines, pamphlets, brochures and research for helping patients with alcohol dependence.
www.niaaa.nih.gov/Pages/default.aspx

MedlinePlus (National Institutes of Health) – Alcoholism and Alcohol Abuse
An overview of alcoholism and alcohol abuse for patients, including symptoms, causes, diagnosis, treatment, complications, and support groups.
www.nlm.nih.gov/medlineplus/ency/article/000944.htm

The Definition of Addiction
A detailed resource for pharmacists addressing alcohol dependence, withdrawal symptoms, post-acute withdrawal syndrome, and approaches to recovery.
www.addictionsandrecovery.org/definition-of-addiction.htm

Motherisk
A helpful resource and reference with articles for patients and healthcare professionals on the use of alcohol during pregnancy and breastfeeding.
www.motherisk.org/women/alcohol.jsp

Professionals Health Program
A resource for pharmacists interested in learning more about the Ontario Professionals Health Program and its confidential services to assist members suffering from incapacitation, substance use and/or mental health issues.
www.ocpinfo.com/client/ocp/OCPHome.nsf/web/ Professionals+Health+Program

Guidelines for Handling Incapacitated Pharmacists and Pharmacy Co-workers
An Ontario College of Pharmacists document outlining warning signs, duty to report (with scenarios) and the definition and guidelines in defining the "incapacitated pharmacist."
www.phpoma.org/PDF%20files/Pharmacist/ Guidelines_for_Handling_Incapacity.pdf

Monitoring

PHYSIOLOGICAL PARAMETERS

TLab tests such as liver function tests, mean corpuscle volume, serum alcohol levels, urine specific gravity and bilirubin are usually elevated in a patient who chronically consumes alcohol, while serum B12 and folate are commonly depleted. Test results within normal ranges may provide evidence of continued abstinence, although not definitive. This is coupled with the patient and family member's updates to quantify stable recovery. In some cases, random urine toxicology screening may be performed.[40]

PSYCHOLOGICAL PARAMETERS

It is essential for the pharmacist to continually quantify and discuss drinking

behaviours in collaboration with the patient and/or support system and offer positive support. The community pharmacist is well-placed to notice adherence with medication refills, using this interaction to open communication. Integration and collaboration with the patient are critical in order to help set the appropriate goals for the reduction or cessation of alcohol consumption.[1]

Patients and their families must be made aware of the post-acute withdrawal syndrome (PAWS) that may occur within the first two years of abstinence, since the development of PAWS may result in relapse or its symptoms may be confused with symptoms of relapse.[41] Signs and symptoms of PAWS include an inability to think clearly, memory problems, emotional overreaction, emotional instability, sleep disturbances, physical coordination problems, and enhanced stress sensitivity.[41] Maintaining regular sleep patterns, diet, exercise, and social interactions are more effective than using a benzodiazepine. Pharmacotherapy is usually not indicated for PAWS.[41]

Addiction in health professionals

Similar to the general population, evidence of substance abuse in health professionals may include emotional, behavioural or physical changes. Emotional changes include aggression, burnout, anxiety, depression, paranoia and denial. Behavioural effects may manifest as slowed reaction time, impaired coordination, slurred speech, irritability, excessive talking, inability to sit still, limited attention span, lack of motivation or lack of energy. Physical effects may include weight loss, sweating, chills and the smell of alcohol. The health professional is often able to "function" professionally as work is frequently the last aspect of their life to deteriorate. As such, their incapacity may have significantly escalated before it is recognized in the workplace. Work-related warning signs may include: increased disorganization, increased number of prescription errors (dispensing and counselling) or customer complaints, increased absence and decreased focus.[42] Resources to assist incapacitated healthcare professionals are included in Table 6.

Conclusion

Community pharmacists are well-placed to interact with patients with alcohol dependence. Hospital pharmacists may engage in initial dialogue with the patient to determine needs and manage acute withdrawal symptoms. The community pharmacist, through dialogue and documentation of patient-centred goals, may engage in regular follow-up with these patients, including monitoring adherence with prescription refills, especially in the first year. Pharmacists can also address concerns about acute and maintenance pharmacotherapies. They can also direct patients to further education and enhance awareness of psychosocial or behavioural interventions.

Pharmacists can also watch for patients who seek other substances that produce mind-altering effects (i.e., "substitute addictions"). Common substitute addictive medications include, but are not limited to opiates, benzodiazepines, appetite suppressants, cough syrups and antihistamines.[24] Community pharmacists can try to monitor for misuse of OTC agents and identify other potential drugs of abuse by recognizing abnormalities in prescription refill dates or purchase histories.[43] Pharmacists are also well-placed to provide brief psychosocial intervention as well as optimize pharmacotherapy, the two cornerstones of a successful recovery model.

Questions

Answer online at www.CanadianHealthcareNetwork.ca, CE section, Quick Search CCCEP #1065-2011-340-I-P

1. The 12-month prevalence of alcohol dependence in the United States at any point in time in the general population is:
a) approximately 1%
b) approximately 4%
c) approximately 15%
d) approximately 20%
e) approximately 25%

2. The one-year incidence of relapse without any pharmacological adjunct to brief behavioural interventions is:
a) 60%
b) 70%
c) 80%
d) 90%
e) 100%

3. Which neurotransmitter systems have been implicated in alcohol dependence?
a) opioid
b) dopamine
c) GABA
d) all the above
e) b and c

Case 1: X is a pharmacist who works with you in a midnight community pharmacy. Lately you notice X is acting more "unpredictably"—taking frequent days off due to migraines, claiming nausea and vomiting, and you notice a regular purchase pattern for dimenhydrinate. However, X is always willing to help out, take extra shifts, and stay late…you are unable to pinpoint the root of your unease.

4. Your colleague X is more likely to be:
a) male
b) female

5. What is a risk factor in patients with alcohol dependence?
a) non-smoker
b) female
c) professional
d) family history

6. X is diagnosed with alcohol dependence. When X stops consuming alcohol, X's blood pressure is 160/110 mmHg with a heart rate of 115 and X is also experiencing diaphoresis. X may be classified as experiencing:
a) mild withdrawal
b) moderate withdrawal
c) severe withdrawal

7. X's blood test results on admission reveal elevated liver enzymes indicative of early damage and elevated vital signs. What would be the best pharmacotherapy choice to help manage the acute withdrawal?
a) watch and wait
b) lorazepam 1 mg orally every 4 hours if needed
c) diazepam 5 mg orally every 4 hours if needed
d) lorazepam 2–4 mg orally four times daily if needed
e) diazepam 15–20 mg orally four times daily if needed

8. Supportive interventions for X may include the following:
a) thiamine 100 mg oral daily
b) folic acid 1 mg oral daily
c) multivitamin 1 tablet daily
d) all of the above
e) none of the above

ce lesson

PHARMACY PRACTICE NATIONAL CONTINUING EDUCATION PROGRAM

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9. What symptoms of alcohol dependence may an incapacitated health professional display?
a) emotional symptoms
b) physical symptoms
c) behavioural symptoms
d) all of the above
e) none of the above

Case 2: Y is a 44-year-old woman who has had three episodes of depression and anorexia with concurrent anxiety. She is prone to stopping her antidepressants and favours alcohol use to "solve her problems, same as her father did." She admits she has a problem with alcohol.

10. Would monotherapy with an SSRI "cure" Y's concurrent alcohol dependence and depression?
a) yes
b) no

11. Which SSRI has direct activity on serotonin and dopamine?
a) paroxetine
b) sertraline
c) escitalopram
d) fluoxetine
e) citalopram

12. Given Y's family history of alcohol dependence, which agent for abstinence maintenance therapy would be optimal if they wanted a "deterrence" factor?
a) acamprosate 1 g three times a day
b) naltrexone 5 mg daily
c) disulfiram 250–500 mg daily
d) naltrexone 50 mg daily
e) quetiapine 100 mg at bedtime

13. Six months into recovery, Y returns to your pharmacy complaining of memory problems and irritability. This may be due to:
a) post acute withdrawal syndrome
b) relapse
c) all of the above
d) none of the above

14. What intervention(s) is/are most effective at preventing relapse in this patient?
a) brief psychosocial intervention
b) pharmacotherapy
c) pharmacotherapy and psychosocial interventions
d) residential treatment programs
e) none of the above

15. Y has been stabilized on disulfiram 500 mg daily for the past 12 months, refills have been regular and she reports good adherence and abstinence. However, after a recent cold, she calls complaining of severe nausea, vomiting and flushing. Which may be the cause?
a) mouthwash
b) cough syrup
c) alcohol hand sanitizer use
d) all of the above
e) none of the above

References are available at www.CanadianHealthcareNetwork.ca, CE section.

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THIS MONTH
A PHARMACIST'S OVERVIEW OF ALCOHOL DEPENDENCE: THE PATH TO ABSTINENCE

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All lessons are reviewed by expert pharmacists for accuracy, currency and relevance to current pharmacy practice.

This lesson is valid until November 9, 2014. Information about palliative care may change over the course of this time. Readers are responsible for determining the most current aspects of this topic.

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5. a b c d	**10.** a b	**15.** a b c d e

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