CANADIAN DIABETES ASSOCIATION | ASSOCIATION CANADIENNE DU DIABÈTE

Alcohol + Diabetes
Frequency Asked Questions for Healthcare professionals
Nutrition Guidelines Implementation Subcommittee

Question 1:

Why are liquor containing beverages exempted from carrying any nutrition information under the mandatory nutrition labelling regulations?

Answer: Beverages with an alcohol content of more than 0.5% are exempt from carrying any nutrition information. The labeling of alcoholic beverages is a complex matter, with implications for trade, jurisdiction, health, etc. No plans are currently underway in Canada for requiring nutrition labelling on such products.

Reference:

Nutrition Labelling and Claims Regulations - Frequently Asked Questions
http://www.hc-sc.gc.ca/hpfb-dgpsa/onpp-bppn/labelling-etiquetage/regulations_faqs_e.html

Question 2:

How does alcohol contribute to the risk of low blood glucose (hypoglycemia) for someone with type 1 diabetes?

Answer: It happens for a number of reasons:
1. Even small amounts of alcohol may impair someone's ability to detect the onset of hypoglycemia and therefore take appropriate action to correct it.
2. Hypoglycemia may be mistaken for intoxication by others.
3. Alcohol has been shown to impair the hormonal counter-regulatory responses to low blood glucose levels.
4. Small amounts of alcohol can augment the cognitive deficits associated with hypoglycemia in people with type 1 diabetes.
5. Alcohol use may be associated with a delayed effect, increasing the risk of next-day hypoglycemia.

Reference:

Richardson T, Thomas P, Weiss M, et al. Day After the Night Before. Influence of evening alcohol on risk of hypoglyemia in patients with type 1 diabetes. *Diabetes Care*. 2005; 28: 1801-1802.

Updated: January 25, 2006

Question 3:

Can people with type 2 diabetes get DKA?

Answer:

While it normally occurs in people with type 1 diabetes, people with type 2 diabetes can develop it.

DKA (diabetic ketoacidosis) is a life-threatening condition. It results from an absolute or relative insulin deficiency and is characterized by high blood glucose (hyperglycemia), too many ketones (ketosis), an electrolyte imbalance with a low blood pH (acidosis) and dehydration.

DKA may develop very quickly with no prior signs or symptoms. More typically, however, it develops within 24 hours with symptoms of high blood glucose being present for up to several days.

The **symptoms** of DKA include:

- Symptoms of hyperglycemia [polyuria (excessive urination), polydipsia (excessive thirst), blurred vision, weight loss and polyphagia (hunger) if insulin deficiency is present for days or weeks]
- Gastrointestinal symptoms (nausea, vomiting, abdominal pain)
- Respiratory symptoms (an inability to "catch one's breath"; deep, sometimes rapid, breaths)
- Non-specific symptoms (weakness, lethargy, malaise and headache)

While there are no specific **signs** of DKA, a person might suspect DKA if they have the following:

- Hypothermia (although a fever could suggest an associated infection)
- Hyperpnea (deep breaths which reflect a response to acidosis)
- Acetone breath (this ketone, excreted by the lungs, has a fruity odour)
- Dehydration
- Acute abdomen (tenderness; reduced bowel sounds; and sometimes guarding – especially in children). Severe DKA may lead to bowel signs which would normally suggest a surgical emergency – however, these signs disappear after treatment in virtually all cases.
- Changes in mentation (alertness may vary from normal to comatose. The changes correlate best with serum osmolality)
- Decreased reflexes (if serum potassium is affected before or during treatment)

The cause of DKA varies. Infection and illness are leading causes (about 30-40% of cases). These conditions cause a person's body to increase their levels of hormones (glucocorticoids) which cause the liver to produce more glucose. Other hormones (epinephrine and norepinephrine) are also increased causing a breakdown of glycogen into glucose. Insulin deficiency and increased levels of certain hormones that act against insulin (counterregulatory) also lead to the breakdown of triglycerides. The excess free fatty acids

from the triglycerides are converted to ketones while the glycerol is converted to more glucose. Because the "spilling over" of high levels of glucose into the urine brings extra water with it, hyperglycemia leads to dehydration as well as to electrolyte depletion.

New onset of type 1 diabetes or not taking enough insulin (due to psychological problems, fear of weight gain, fear of hypoglycemia or lack of understanding of sick day management, for example) also commonly lead to DKA. Another potential source of inadequate insulin leading to DKA is related to problems with insulin pumps. These problems can include blockages or crimping of the tubes.

In terms of alcohol and DKA, excessive alcohol intake can lead to poor diabetes control (e.g. problems with medication/blood sugar balance, etc...) which can contribute to DKA. Also, while not directly causative, in some people with type 2 diabetes excess alcohol can worsen dehydrating conditions (e.g. vomiting) and thereby contribute to the development of DKA (particularly when blood glucose levels are elevated as may occur when high sugar/starch alcohols such as home brews are consumed or infections are present, for example).

References:

Personal correspondence: Dr. Stewart Harris, MD MPH CCFP ABPM, The University of Western Ontario

«A Core Curriculum for Diabetes Educators: Diabetes and Complications», American Association of Diabetes Educators, Marion Franz, MS RD LD CDE, editor (2001).

Question 4:

What is the link between alcohol intake and nerve damage (neuropathy)?

Answer:

Alcohol intake is considered one of many risk factors for developing nerve damage or neuropathy, which is one of the complications of diabetes.

If you already have nerve damage (neuropathy), alcohol intake can increase the pain, burning, tingling, numbness and other symptoms found with nerve damage. Significant alcohol intake can lead to further nerve damage.

References:

The American Diabetes Association http://www.diabetes.org/all-about-diabetes/diabetes-news/enews-archive/05-06-04.jsp

The American Diabetes Association http://www.diabetes.org/type-1-diabetes/alcohol.jsp

International Guidelines on the Out-patient management of Diabetic Peripheral Neuropathy. The Medicine Group (Education) Ltd, 1998.

Question 5:

My client has type 1 diabetes. Graduation is coming up and drinking more than the recommended guidelines is a strong possibility. What do I need to discuss?

Answer: An educator should remind the person to:

- Wear identification indicating that the diagnosis of diabetes.
- Inform friends that if the person "passes out" they need to call an ambulance.
- Keep a blood glucose monitor available to check blood glucose.
- Eat sources of carbohydrates to prevent hypoglycaemia.
- Have the adult monitor patient's blood glucose every 2 hours until the patient is awake enough the next day to do so him/herself
- Eat a snack containing sources of carbohydrate before going to sleep
- Take usual bedtime insulin but if forgotten, take it late but decrease dose by 20%.
- Have a responsible person take patient to ER if he/she is vomiting, unable to keep blood glucose >4mmol/L, has a change in respirations, is showing signs of dehydration, is unconscious, or there is a concern about alcohol poisoning. The medical staff must be told that the person has been drinking.
- Get up the next day as close to the usual time as possible, take insulin, eat breakfast and continue monitoring blood glucose.
- Monitor blood glucose levels throughout the day after the drinking occurred and make appropriate adjustments to insulin based on blood glucose results and keeping in mind that hypoglycemia can occur up to 14 hours after alcohol consumption.

Question 6:

Why might Glucagon injection not work as effectively for treating hypoglycemia while alcohol is in the body?

Answer:

Endogenous glucagon is a pancreatic counterregulatory hormone, which is secreted in response to low blood glucose levels. Its main role is to restore low blood glucose levels by generating a ready supply of glucose. It accomplishes this in two ways. Principally, glucagon stimulates the breakdown of liver glycogen stores, converting them to glucose through a process called glycogenolysis. In addition to mobilizing liver glycogen stores, glucagon stimulates hepatic gluconeogenesis through conversion into glucose of gluconeogenic substrates such as alanine, pyruvate, lactate, and glycerol.

Alcohol can interfere with the process of gluconeogenesis. This occurs during the metabolism of alcohol, in which there is depletion of the supply of pyruvate needed for gluconeogenesis. As a result, alcohol by itself may lead to hypoglycaemia or delay recovery from hypoglycaemia.

It is important to realize that the behaviour-altering effects of alcohol can also complicate hypoglycaemia by clouding the recognition of hypoglycaemia by the patient and his surroundings, as well as by delaying the treatment of hypoglycemia with prompt oral glucose

supplementation. Alcohol consumption may therefore have serious implications in the case of severe hypoglycaemia, in which a person is unable to self-treat. Usually persons are taught to inject exogenous glucagon s.c. or i.m. as an antidote to reverse severe hypoglycaemia. Though alcohol interferes with gluconeogenesis, it may only pose a *theoretical* concern in the setting of exogenously administered glucagon. This is because of the supraphysiologic blood levels of glucagon achieved following an s.c. or i.m. injection, and alcohol's lack of effect on the glycogenolytic pathway. It is therefore unlikely that alcohol, on its own, would prevent the reversal of hypoglycaemia following the administration of glucagon.

In the setting of chronic alcoholism, however, liver glycogen stores may become depleted secondary to malnutrition (or a reduced supply of substrate). The efficacy of glucagon, as a treatment for reversing severe hypoglycaemia, may therefore be significantly reduced. This is because *both* the glucagon-stimulated metabolic processes for generating glucose, namely glycogenolysis and gluconeogenesis, have been compromised, either indirectly or directly. In such a scenario, the preferred treatment for reversal of severe hypoglycaemia would be the administration of intravenous glucose.

In patients with an absent or diminished endogenous glucagon response to low blood glucose (i.e. type 1 diabetes), reversal of severe hypoglycaemia — particularly when secondary to insulin — may be more difficult to achieve with exogenous glucagon in the presence of alcohol. In this case, treatment with intravenous glucose may be preferred.

References:

Glucagon product monograph, CPS 2005, Compendium of Pharmaceuticals and Specialities, The Canadian Drug Reference for Health Professionals, Canadian Pharmacists Association.

Griffin, JE, Ojeda, SR., editors. Textbook of Endocrine Physiology (3rd edition). Oxford University Press, 1996.

Cryer PE, Davis SN, Shamoon H. Hypoglycemia in diabetes. *Diabetes Care* 2003; 26: 1902-1912.

Rasmussen BM, Lotte O, Schmitz O, Hermansen K. *Alcohol and glucose counterregulation during acute insulin-induced hypoglycaemia in type 2 diabetes. Metabolism* 2001; 50: 451-7.

Bartlett D. Confusion, somnolence, seizures, tachycardia? Question drug-induced hypoglycaemia. *Journal of Emergency Nursing* 2005; 31: 206-8.

Turner BC, Jenkins E, Kerr D, Sherwin RS, Cavan DA. The effect of evening alcohol consumption on next-morning glucose control in type 1 diabetes. *Diabetes Care* 2001; 24: 1888-93.

Question 7:

Can alcohol be consumed when an individual is taking oral hypoglycemic agents or insulin?

Answer:

Risk of hypoglycemia is increased with the use of insulin or insulin secretagogues (i.e., glyburide, gliclazide, glimepiride, chlorpropamide, tolbutamide, nateglinide, repaglinide). Consuming alcohol further increases the risk of hypoglycaemia.

There is no increase in the risk of hypoglycemia with the use of acarbose, pioglitazone, rosiglitazone or metformin. However, many individuals are on combination therapies, which may include insulin and/or insulin secretagogues. In these instances the risk of hypoglycaemia would be increased and consuming alcohol further increases the risk of hypoglycemia.

An increased risk for the development of lactic acidosis occurs if large amounts of alcohol are consumed, either acutely or chronically, while taking metformin. Excess alcohol intake, whether acute or chronic, is not advised when an individual takes metformin.

In the setting of severe hypoglycaemia complicated by concomitant alcohol and insulin secretagogue therapy, exogenously administered glucagon will acutely reverse the hypoglycaemia, but its blood glucose-raising effect may be too short-lived to prevent subsequent hypoglycaemia, which may be more likely to occur with the use of longer-acting insulin secretagogues. In such a case, maintenance of euglycemia may require medical intervention through intravenous glucose until the hypoglycemic effect of the insulin secretagogue has resolved.
Individuals should consult their physician or pharmacist if they require further information.

Reference:

Product monographs, CPS 2005, Compendium of Pharmaceuticals and Specialities, The Canadian Drug Reference for Health Professionals, Canadian Pharmacists Association.