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Liudmila Chernetska

Hypoglycemia: Turning the tide on crashing blood glucose

By Sarah Beattie, DNP, APRN-CNP, CDCES

Abstract: Primary care NPs are central to the management of diabetes mellitus, which carries with it the risk of hypoglycemia. Fully understanding risk factors, prevention strategies, and treatment assist in reducing hypoglycemic events. This article details hypoglycemia, risk factors for hypoglycemia, prevention strategies, and appropriate treatment plans.

Affecting approximately 37.3 million people in the US, diabetes mellitus is a complex chronic illness that can create management challenges for both primary care NPs and patients.[1] With the projected incidence of diabetes continuing to increase, healthcare providers must be well versed in the condition's care and management. An often-underappreciated potential consequence of diabetes management is hypoglycemia. Armed with information and experience, the primary care provider is perfectly poised to dispel the misconception that hypoglycemia is an unavoidable consequence of diabetes treatment and shift to an approach that includes prevention, early recognition, and treatment. This article aims to inform the NP of the

Keywords: blood glucose, blood sugar, continuous glucose monitoring (CGM), diabetes, glucagon, hypoglycemia, hypoglycemia unawareness, hypoglycemic events, insulin, insulin secretagogues, patient education, prevention, type 1 diabetes mellitus (T1DM), type 2 diabetes mellitus (T2DM)

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burden of hypoglycemia among adult patients with diabetes as well as the latest advances in treatment options for hypoglycemia.

Case study

Mrs. Blake is a 75-year-old Black female who has had type 2 diabetes mellitus (T2DM) since age 52. She is at the primary care office with her husband for her 3-month follow-up visit for her diabetes. She is currently taking insulin glargine U-100 30 units subcutaneously daily, dulaglutide 3 mg subcutaneously once weekly, metformin 500 mg P.O. twice per day, dapagliflozin 10 mg P.O. daily, and insulin lispro U-100 10 units subcutaneously at dinner. Her medical history includes chronic kidney disease (CKD) stage 3a with mild microalbuminuria, obesity, heart failure with preserved ejection fraction, hypertension, and hyperlipidemia. She is overall sedentary. Although she continues to work on improving her diet, she finds it hard to access healthy foods and enjoys drinking soda pop with dinner. She has no known history of hypoglycemia, telling you, "I don't worry about lows because my blood sugar has never been low before."

Prevalence and pathophysiology

Hypoglycemia is defined as a blood glucose level of 54 to 69 mg/dL (Level 1) or less than 54 mg/dL (Level 2).[2] Level 3 hypoglycemia occurs when a hypoglycemic event in an individual results in an altered mental or physical state that requires them to receive assistance from others.[2] Hypoglycemia is often an unintended consequence of diabetes treatment; its prevalence, though often underestimated due to a lack of documentation around hypoglycemic events, has been noted to be nearly 20% in those with type 1 diabetes mellitus (T1DM) and nearly 10% in those with T2DM.[3]

When blood glucose drops below 70 mg/dL, the neuroendocrine system is activated, which increases the level and prompts food-seeking behavior, and neuroglycopenic and adrenergic symptoms such as tremors, blurred vision, diaphoresis, and behavior changes occur.

In individuals without diabetes, to increase blood glucose and achieve euglycemia, the counterregulatory hormonal response to hypoglycemia entails a cascade of events, including the decrease of insulin production by pancreatic beta cells; the increase in glucagon production by pancreatic alpha cells; hepatic gluconeogenesis (the creation of glucose by breaking down other substances such as amino acids) and glycogenolysis (the breakdown of glycogen into glucose); and release of epinephrine, cortisol, and growth hormone.

In people with T1DM and T2DM, the counterregulatory hormone response to hypoglycemia is impaired. In T1DM, the total loss of beta cell function results in a limiting effect on the alpha cells of the pancreas, reducing their ability to secrete glucagon in response to hypoglycemia. With a longer duration of T1DM and with an increased frequency of hypoglycemic events, release of counterregulatory hormones (epinephrine, cortisol, and growth hormone) also becomes impaired.[4] In those with T2DM, residual beta cell function allows for an intact but diminished alpha cell and glucagon response to hypoglycemia. When taking exogenous insulin or insulin secretagogues, the body is dependent on the peak and duration of medication in a hypoglycemic event. Over time, as T2DM progresses and beta cell function continues to decline, the release of glucagon and other counterregulatory hormones becomes impaired, and hepatic gluconeogenesis and glycogenolysis also are diminished. Therefore, the risk of hypoglycemia is higher with a longer duration of T2DM.[5]

Risk factors

The risk factors of hypoglycemic events are complex and often multifactorial, with two or more risk factors compounding to create an overall increased risk. The use of pharmacologic agents with known risk of hypoglycemia increases the odds of a hypoglycemic event. Additionally, though not fully discussed in this article, tighter glycemic index goals during pregnancy can substantially increase the risk of hypoglycemia for patients with diabetes and gestational diabetes, which are often treated with insulin as the standard medical therapy. Renal function also plays a significant role in hypoglycemia risk. Decreased renal clearance increases the availability of many drugs within the body. If a drug with a high risk for hypoglycemia is used in patients with CKD, particularly in those with stage 3 or above, then the risk of hypoglycemia is exponentially higher.[6] Increased age as well as altered cognitive status also create an increased risk for hypoglycemia, as these patients may not be able to communicate the early signs of hypoglycemia, thereby leading to delayed recognition and treatment.

Glucose variability increases the risk of hypoglycemia. Increased glucose variability in a patient entails wide fluctuations in their blood glucose levels, ranging

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from severe hyperglycemia to the hypoglycemic range, that typically occur multiple times throughout the day and night. The more time a patient spends in a hypoglycemic state, the less aware of hypoglycemia they become, resulting in a condition known as hypoglycemia unawareness.[7] In turn, hypoglycemia unawareness further increases the risk of hypoglycemia.

Racial disparity studies in diabetes care and management have noted a higher risk of hypoglycemia in Black adults compared with adults of other races.[2] This racial disparity in hypoglycemia risk is multifactorial. Though the exact physiologic mechanisms are unclear, evidence suggests that Black adults have decreased insulin clearance compared with adults of other races.[8] Nonphysiologic variables including food insecurity, lack of access to healthcare, housing insecurity, and other social determinants of health may contribute to increased risk.[9] Implicit bias among healthcare providers has also been attributed to poor health outcomes in the Black community.[10]

Hypoglycemia risk varies among the available pharmacologic options for diabetes. *Table 1* provides an overview of pharmacologic therapy options for diabetes, their hypoglycemia risk, the mechanisms behind their hypoglycemia risk level, and clinical considerations in view of risk. Insulin and insulin secretagogues (sulfonylureas and meglitinides) are medications that carry a high risk of hypoglycemia. When used together, insulin and insulin secretagogues carry the highest risk of hypoglycemia.[11] The remainder of the pharmacologic agents used to treat diabetes may not cause hypoglycemia alone, though they can induce hypoglycemia if used with the aforementioned agents.

Certain nondiabetes drugs and supplements can also carry hypoglycemia risk. Prescription drugs that can carry hypoglycemia risk include fluoroquinolones, sulfamethoxazole, angiotensin-converting enzyme inhibitors, beta-adrenergic receptor blockers, quinine, salicylates, tyrosine kinase inhibitors, and disopyramide. Other commonly used agents that can cause hypoglycemia include ethanol, bitter melon, fenugreek, ginseng, ivy gourd, and L-carnitine.[12]

Consequences

In the short term, if not recognized and treated, hypoglycemia can be fatal. In the longer term, hypoglycemia carries an increased risk of mortality through factors such as increased risk of cardiovascular disease.[13] Hypoglycemic events also lead to ED visits, lost productivity at work, and a fear of future additional hypoglycemic events.

Treatment

Hypoglycemia, at any stage, requires early identification and swift treatment. The treatment for hypoglycemia for a patient who is alert and oriented is to consume 15 grams of rapid-acting carbohydrates. Any one of the following items would satisfy this 15-gram requirement:

- Glucose tablets (about 4 tabs, depending on brand)
- Glucose gel (1 tube)
- 4 oz of fruit juice
- 4 oz of soda pop (not diet)
- Small amount of candy (usually one small pack, with the amount depending on carbohydrate content)

After consumption of 15 grams of rapid-acting carbohydrates, the patient should wait 15 minutes before rechecking their blood glucose. This process is referred to as the 15/15 rule. The treatment process should be repeated if necessary until blood glucose stabilizes above 70 mg/dL. After blood glucose stabilizes, the patient should consume a snack or a meal. Consuming carbohydrates combined with fat, higher amounts of protein, or fiber can lead to delayed absorption of carbohydrates and prolonged hypoglycemia.[2]

Nonphysiologic variables including food insecurity, lack of access to healthcare, housing insecurity, and other social determinants of health may contribute to increased risk of hypoglycemia.

A patient who is unable to eat or drink rapid-acting carbohydrates should be treated with glucagon. Glucagon is a hormone found in the alpha cells of the pancreas that stimulates hepatic glycogenolysis and gluconeogenesis. These processes increase glucose levels in the blood, thereby correcting hypoglycemia. As previously discussed, endogenous glucagon production is absent or dysregulated in patients with diabetes, resulting in an inability to rely on the body to

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Table 1. Diabetes pharmacology: Hypoglycemia risk and considerations[16,17]

Medication	Hypoglycemia risk	Mechanism of hypoglycemia risk	Hypoglycemia-related clinical considerations
Insulin (basal, prandial, mixed, or basal-bolus therapy)	High	Allows the uptake of glucose from the blood into cells Inappropriate dosing or combining with other hypoglycemia-inducing agents potentiates insulin effect	Avoid use in combination with sulfonylureas or meglitinides Exercise caution in prescribing in older adults and patients who have CKD or who have hypoglycemia unawareness Insulin therapy is recommended for treatment of diabetes during pregnancy. Due to the tight glycemic control needed in pregnancy, hypoglycemia risk is high.
Sulfonylureas (glyburide, glimepiride, glipizide)	High	Causes insulin secretion from pancreatic beta cells regardless of blood glucose levels Has a long duration of action	Avoid in older adults and in patients who have CKD or who have hypoglycemia unawareness Avoid use in combination with insulin therapy
Meglitinides (repaglinide, nateglinide)	High	Causes insulin secretion from pancreatic beta cells regardless of blood glucose levels Has a fast onset and short duration of action	Avoid in older adults and patients who have hypoglycemia unawareness Exercise caution in prescribing to patients with CKD history Avoid use in combination with insulin therapy
Metformin	Low	Lowers hepatic glucose production and improves hepatic insulin sensitivity Mild insulin sensitizer at peripheral muscle cells	Hypoglycemia risk increases when used with insulin, sulfonylureas, or meglitinides Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications
SGLT2 inhibitors (bexagliflozin, canagliflozin, dapagliflozin, empagliflozin, ertugliflozin)	Low	Inhibits glucose resorption in the distal tubule of the kidney, lowering the threshold for glucosuria and natriuresis. This increases the glucose-lowering effect independent of insulin.	Hypoglycemia risk increases when used with insulin, sulfonylureas, or meglitinides Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications
GLP-1 RAs (dulaglutide, exenatide, semaglutide, liraglutide, lixisenatide)	Low	Activates GLP-1 receptors to increase endogenous insulin production, decrease glucagon secretion, and slow absorption of glucose from the gastrointestinal tract, all in a glucose-dependent manner	Hypoglycemia risk increases when used with insulin, sulfonylureas, or meglitinides Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications
GIP + GLP-1 RA (tirzepatide)	Low	Activates GLP-1 and GIP receptors to increase endogenous insulin production, decrease glucagon secretion, and slow absorption of glucose from the gastrointestinal tract, all in a glucose-dependent manner	Hypoglycemia risk increases when used with insulin, sulfonylureas, or meglitinides Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications

(Continues)

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Table 1. Diabetes pharmacology: Hypoglycemia risk and considerations[16,17] *(continued)*

Medication	Hypoglycemia risk	Mechanism of hypoglycemia risk	Hypoglycemia-related clinical considerations
DPP-4 inhibitors (sitagliptin, saxagliptin, linagliptin, alogliptin)	Low	Inhibits DPP-4, preventing breakdown of GLP-1 and GIP and creating more available circulating GLP-1 and GIP to reduce glucose in a carbohydrate-dependent manner	Hypoglycemia risk increases when used with insulin, sulfonylureas, or meglitinides Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications
TZDs (pioglitazone, rosiglitazone)	Low	Activates PPAR gamma to increase gene expression, increasing insulin sensitivity in adipose tissue and muscle Decreases hepatic glucose production	Hypoglycemia risk increases when used with insulin, sulfonylureas, or meglitinides Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications
Alpha-glucosidase inhibitors (acarbose, miglitol)	Low	Delays carbohydrate digestion and intestinal absorption of glucose, lowering postprandial hyperglycemia, and lowers postprandial insulin levels	Hypoglycemia risk increases when used with insulin or sulfonylureas Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications If hypoglycemia occurs, it must be treated with glucose. Alpha-glucosidase inhibitors delay the absorption of sucrose, which will not be effective in treating hypoglycemia.
Amylin mimetic (pramlintide)	Low	Diminishes postprandial glucagon secretion and increases satiety Acts synergistically with prandial insulin, potentiating its effect	Hypoglycemia risk increases when used with prandial insulin Low hypoglycemia risk when used alone or with other low hypoglycemia risk medications

Abbreviations: CKD, chronic kidney disease; SGLT2, sodium-glucose cotransporter 2; GLP-1 RA, glucagon-like peptide-1 receptor agonist; GIP, glucose-dependent insulinotropic polypeptide; DPP-4, dipeptidyl peptidase-4; TZD, thiazolidinedione; PPAR, peroxisome proliferator-activated receptor.

correct hypoglycemia. Glucagon is available in intranasal or injectable (I.M. or subcutaneous) formulations and can be prescribed to anyone with diabetes who has a risk of hypoglycemia. Traditionally, glucagon was manufactured in a kit containing a diluent and powder that the caretaker would mix, draw up, and inject into the patient as needed. This cumbersome task during an emergent situation often led to underuse or inappropriate use of the agent. Advancements in technology have allowed glucagon to be packaged in and delivered by autoinjectors, prefilled syringes, and nasal sprays. The newer formulations of glucagon take less time and skill to administer than the traditional kit. The Endocrine Society, in its 2022 clinical practice guideline on hypoglycemia, recommends glucagon preparations that do not require reconstitution as the agents of choice when prescribing glucagon in the outpatient setting.[14]

Education and prevention

Ensuring patients understand their risk of hypoglycemia as well as early symptoms of hypoglycemia can lead to prevention and early identification. Every patient at risk of hypoglycemia should be educated on hypoglycemia symptoms and treatment using the 15/15 rule (see previous "Treatment" section). As with education for many conditions, education on hypoglycemia should be both verbal and written.

Every patient prescribed medications that have a high risk of causing hypoglycemia should

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be provided with a prescription for glucagon and instructions for how to use the delivery device. Referral to a diabetes care and education specialist has proven beneficial in reducing hypoglycemic events and should be an option for all patients at risk of hypoglycemia.[14,15] Other prevention tactics include using longer-acting basal insulin analogues in place of human neutral protamine Hagedorn (NPH) insulin, using rapid-acting insulin analogues in place of regular insulin, and using continuous glucose monitors (CGMs) in place of self-monitoring of blood glucose by finger stick.[2,13] CGM technology assists in reducing hypoglycemia via alarms for rapid decline in blood glucose or alarms for hypoglycemia below a preset threshold, which is typically 70 mg/dL, although the threshold can be increased if needed in patients with a very high risk of hypoglycemia or in those whose blood glucose drops rapidly after nearing 70 mg/dL.

Case study revisited

During your visit with Mrs. Blake, you discuss your concerns regarding her risk of hypoglycemia. You educate her on not only the symptoms of hypoglycemia but also on how to appropriately treat it using the 15/15 rule. You provide her with a prescription for intranasal glucagon and demonstrate to her and her husband how to use the device. You explain when to use glucagon versus when to opt for oral treatment. Finally, you provide Mrs. Blake with a prescription for a CGM, and you refer her to a diabetes care and education specialist for more training.

Three months later, Mrs. Blake follows up stating that she saw the diabetes care and education specialist, has given up soda pop, has increased her physical activity, and has been wearing a CGM. Since making these lifestyle changes, she reports she had a hypoglycemic event during her after-dinner walk, noting that her blood glucose dropped to 62 mg/dL, to which her CGM alerted her, and that she "felt terrible." She was able to treat her hypoglycemia using 4 glucose tablets taken orally. She experienced full resolution of symptoms within 15 minutes and proceeded to eat a snack. Since that time, due to fear of hypoglycemia, she contacted the NP who discontinued the insulin lispro U-100 10 units subcutaneously with dinner and has had no further hypoglycemic events. Due to her diet changes and increased physical activity, her postprandial blood glucose readings are stable. You discuss with her that, given her CKD and basal insulin use, she is still at high risk for a hypoglycemic event. You continue to have her monitor her blood glucose with the CGM, continue to educate her on the treatment of hypoglycemia, and ensure that her glucagon prescription is not out of date at future visits.

Conclusion

Diabetes continues to be a complex chronic illness that is increasing in prevalence, and NPs are at the heart of the diabetes management team. Treating diabetes requires advanced knowledge of patient characteristics and available pharmacologic options and requires the provider to bear hypoglycemia risk in mind. The NP has the power to change the tides of diabetes management, shifting away from the idea of hypoglycemia as an unavoidable reaction to its recognition as a preventable consequence of diabetes treatment. Care plans that include referral to a diabetes care and education specialist and that place an emphasis on early recognition of hypoglycemia symptoms and prompt treatment of hypoglycemia using the 15/15 rule or glucagon administration serve to ensure success and mitigate fears for patients with diabetes. NPs' prescription of long-acting basal insulin analogues over NPH insulin, rapid-acting insulin analogues over regular insulin, CGM technology, and easy-to-use glucagon devices equips the patient with tools to minimize hypoglycemia and maximize quality of life.

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The author discloses no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NPR.0000000000000100

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