Table 5. Treatment of hypoglycemia in the backcountry

Medication	Dose	Route	Timing	Side effects	Special considerations
Glucose	15−20 g	PO, PR, IV	Repeat dose q15 min PRN for persistent hypoglycemia	Hyperglycemia	Patient must immediately consume meal or carbohydrate snack after resolution of hypoglycemia.
Glucagon	1 mg	IM, "mini-dose" subcutaneous, intranasal	Effective in 10−15 min	Nausea, vomiting	Only if able to take oral glucose. Effectiveness limited in glycogen-depleted patients.

IM, intramuscular; IV, intravenous; PO, oral administration; PR, per rectum; PRN, as needed.

accompanied by hypokalemia, which can last for several hours after blood glucose levels have returned to normal[101] and can cause impaired skeletal muscle contraction, weakness, and potential cardiac arrhythmias.[91]

The primary treatment option for hypoglycemia is glucose repletion. Although common options typically are oral, rectal, and intravenous (IV) repletion, IV repletion should not be relied on as an option in the wilderness setting. Therefore, it is important to assess the severity of a hypoglycemic episode to decide on the safety and utility of oral treatment or whether evacuation is necessary. The basic principles of hypoglycemia management in the wilderness are cessation of insulin treatment and glucose repletion. Glucose repletion can be accomplished either directly or by administration of glucagon, which stimulates gluconeogenesis and glycogenolysis[102] (Table 5).[103,104] High glycemic index carbohydrates are best for oral glucose repletion, and[16] in the wilderness, low-weight options such as sugar packets, sugar cubes, glucose gel, honey, corn syrup, and glucose tablets can be considered. For severe hypoglycemia—defined as severe cognitive impairment requiring external assistance for recovery—glucagon is the first-line treatment. Any glucagon treatment should be followed by carbohydrate intake to prevent rebound hypoglycemia, especially in the setting of depleted glycogen stores.[105,106] Similar to insulin, glucagon should be stored with insulation to avoid extremes of heat and cold. Individuals accompanying those with diabetes should know where glucagon is stored and how and when to administer it. It is important to consider the risk of ketosis if insulin therapy is suspended for more than 2 to 3 h.[107]

Individuals with type 1 diabetes are at risk for delayed nocturnal hypoglycemia following daytime exercise. There are multiple reasons for this phenomenon, including a rise in insulin sensitivity after exercise, increased glucose uptake by skeletal muscles to replenish glucose stores, and impaired counterregulation in response to hypoglycemia.[22] For those with MDI, the risk of nocturnal hypoglycemia may be minimized through approximately 20% reduction of daily basal insulin dose, reduced prandial bolus insulin, and low glycemic index carbohydrate feeding after evening exercise. For those using CSII, basal rate reductions of approximately 20% at bedtime for 6 h after afternoon exercise may limit nocturnal hypoglycemia. Other strategies include a bedtime snack, glucose checks overnight, and/or use of CGM with alarms and automatic pump suspension.[91]

Recommendation. Wilderness athletes with diabetes should have a plan and carry supplies for treating hypoglycemia. They should be prepared to use a glucose repletion and glucagon strategy (**Evidence grade:** 1C).

Wilderness athletes with diabetes should have experience with individualized methods for managing nocturnal hypoglycemia prior to wilderness activity (**Evidence grade:** 1C).

Hyperglycemia

If someone with diabetes is found to be hyperglycemic (plasma glucose level >250 $mg \cdot dL^{-1}$), it is important to determine whether the individual is in an acute hyperglycemic crisis, including hyperosmolar hyperglycemic state (HHS) or DKA. To help differentiate between hyperglycemia and HHS/DKA, individuals with type 1 diabetes should be able to test for blood or urine ketones if they have unexplained hyperglycemia.

Hyperosmolar hyperglycemic state

HHS is a state of progressive hyperglycemia and hyperosmolarity typically seen in individuals with poorly controlled or undiagnosed type 2 diabetes, limited access to water, and a precipitating medical event. The development of HHS is attributed to insulin resistance, deficiency, or both, in addition to increased hepatic gluconeogenesis, osmotic diuresis, and dehydration. The hyperglycemia in HHS is profound, with serum glucose level usually >600 $mg \cdot dL^{-1}$ and extreme dehydration with a fluid deficit of 8 to 12 L.[108]