Background  Management of diabetes can pose challenges as patients approach death. While there is little clinical research to guide decision-making, this Fast Fact reviews important considerations and practical recommendations about diabetes care in patients thought to be in their final few months of life. See Fast Fact #291 regarding hypoglycemia management at the end of life. Note: all guidelines here are empiric and based on published, expert recommendations, including blood glucose goals; all clinical decisions need to be individualized.

Important Considerations

• Tight glycemic control only benefits patients (e.g., prevents long-term complications such as diabetic retinopathy and nephropathy) with prognoses of years. Treatment goals for patients near the end-of-life are to avoid symptomatic hypo- and hyperglycemia and minimize the burdens of diabetes treatment, but not to prevent those long-term complications. Tight glycemic control is not a coherent goal near the end of life, and can be dangerous as it encourages hypoglycemia.

• Both diabetic treatments and uncontrolled diabetes can be burdensome and symptomatic.
  ○ Hypoglycemia from insulin or oral hypoglycemics causes diaphoresis, anxiety/panic, tremors, weakness, palpitations, seizure, coma. Predictable caloric intake, which typically becomes erratic in dying patients, helps prevent hypoglycemia.
  ○ Hyperglycemia, sustained for several days, can cause an osmotic diuresis leading to dehydration, thirst, and polyuria. Sustained, severe hyperglycemia (often >800 mg/dL) causes a hyperosmolar hyperglycemic state (‘HHS,’ also known as hyperosmolar nonketotic hyperglycemia), associated with thirst, dry mouth, lethargy, and coma. HHS typically occurs in insulin-dependent (type 2) diabetics.
  ○ Diabetic ketoacidosis (DKA) from lack of insulin, occurs most commonly in type 1 diabetics, can develop rapidly (less than a day), and causes hyperventilation, nausea, vomiting, abdominal pain and coma. Fundamentally DKA is caused by a lack of insulin (as opposed to the hyperglycemia itself) and is characterized by acidosis. While blood sugars are usually elevated, commonly 300-800 mg/dL, they can be lower than that especially with poor caloric intake.
  ○ Treatment burdens include painful fingersticks and gastrointestinal side effects of oral hypoglycemics.

Patients with an anticipated survival of a few months  Counsel about reducing tight glycemic control and the increased risk of hypoglycemia as oral intake become erratic. Stop A1c testing. There is no clear consensus about blood sugar targets, although the goal is clear: keep blood glucose levels low enough to prevent osmotic symptoms while minimizing the risk of hypoglycemia. Expert recommendations have suggested targeting fasting glucose up to 180 mg/dL (1); however some raise the target to ≤270 and even ≤360 (5, 6). Common sense should guide these decisions (e.g. if a type 1 diabetic has been known to develop DKA with blood sugars as low as 300 mg/dL, the target should be less than that).
  • Type 1 diabetics should continue insulin to prevent DKA, as should type 2 diabetics prone to symptomatic hyperglycemia without insulin.
  • Type 2 diabetics on insulin in addition to oral medications solely for optimal glycemic control (and not to prevent dehydration or HHS) should stop insulin use. If they have any episode of hypoglycemia, doses of oral agents should be halved, with further dose adjustments titrated to meet the aforementioned goals. Glucose checks should occur as infrequently as feasible: for patients at low risk for hyper- or hypoglycemia (e.g. a patient with no history of symptomatic hyperglycemia despite uncontrolled serum glucose in the past), this can be only when symptomatic.

Patients with anticipated survival of weeks (1,5,6)
  • Type 1 diabetics or type 2 diabetics prone to HHS should continue insulin therapy to prevent DKA, assuming that is compatible with their care goals. Generally, insulin doses should be tailored to keep fasting blood sugars >180mg/dL to minimize the risk of hypoglycemia, however this goal
needs to be individualized. Some amount of basal/long-acting insulin is needed. For patients with erratic oral intake, short-acting insulin should only be used when eating, with conservative doses.

- Most type 2 diabetics can stop hypoglycemic agents entirely in the final weeks of life, unless they are prone to HHS; blood sugars can be checked and hyperglycemia treated if symptoms such as thirst, polyuria develop. Long-acting sulfonylureas and metformin in particular should be stopped. If needed, use short-acting sulfonylureas (e.g. glipizide) to minimize the possibility of sustained hypoglycemia. Metformin accumulates with renal failure.

Patients in the final days of life (1,2,5,6) Most patients have decreasing levels and periods of consciousness, and minimal oral intake. All insulin, hypoglycemics, and monitoring can be stopped in type 2 diabetics. DKA can still develop rapidly (<1 day) for type 1 diabetics, and it can be reasonable (and emotionally important for families) to continue insulin therapy with liberal (e.g. <360mg/dL) blood sugar targets. Family/patient preferences and patient level of consciousness should be considered in making a joint decision as to what to do.

Emotional & Communication Considerations For some patients and families, careful attention to diabetes management is an empowering and satisfying health promotion activity, and can continue to be so near life’s end. It may be one of the few health-related activities they can maintain control and mastery over. Suggestions to stop diabetes medications or blood glucose checks can be seen as lack of concern or indifference to a patient’s health. The burden of blood glucose fingerstick checks may be less than the anxiety over not checking. Be careful to frame such discussions therapeutically, clarifying that stopping or adjusting medications is being done to prevent harm and promote patient well-being (2,3).

References

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