

Editorial

Insulinophobia in the Acute Care Setting – How Sweet it is!

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Abstract

Physicians at all levels of training and experience continue to have an aversion to using insulin to control hyperglycemia. This fear is grounded in the possible development of symptomatic hypoglycemia. Insulinophobia results from a lack of understanding of the time action profile of insulin and the relationships between factors such as infection and other stressors, caloric intake and physical activity, insulin dosing and glycemic control. These aforementioned factors can make blood glucose control difficult to achieve in hospitalized persons with diabetes. Whereas circumstances requiring adjustments to prandial insulin in the acute care setting is recognized, modification of basal insulin remains problematic. The key to overcoming the fear of using insulin in the hospitalized patient is education. Providers must understand the factors/stressors that contribute to poor glycemic control in this population and its consequences. In addition, the pharmacokinetics/pharmacodynamics of insulin and its therapeutic use must be well understood by the provider. Institutional developments of insulin utilization/dosing protocols along with pre-doctoral and postgraduate educational initiatives are needed. These strategies will better position the provider to make appropriate therapeutic adjustments to insulin regimens resulting in improved glycemic control and clinical outcomes in acutely ill persons with diabetes.

Keywords: Insulin; Hypoglycemia; Type 1 diabetes; Type 2 diabetes; Insulin protocols

In my nearly 35 years as a practicing clinical pharmacist and faculty member in an academic Family Medicine residency program, I have been involved in the management of many hundreds of persons with diabetes admitted to our in-patient ward service. Time and experience has taught me one thing, resident physicians and even staff physicians continue to have an aversion to using insulin to control hyperglycemia. Akin to the fear of prescribing opioid analgesics to control moderate to severe pain and the possible creation of a psychologically dependent patient, the fear of using insulin surrounds the potential development of a hypoglycemic patient [1]. The argument may be had that an acute episode of hyperglycemia carries no immediate risks but a hypoglycemic episode is often symptomatic and if severe enough it may even be fatal. Why is this fear so pervasive among providers? Although not grounded in evidence-based research, Insulinophobia most likely results from a lack of understanding of the time action profile of insulin and the relationships between factors such as acute illness and other stressors, food intake and physical activity, insulin dosing and glycemic control. In the hospital setting, persons with diabetes who take insulin are like “fish out of water”. Illness and other stressors, reduced or lack of oral intake and decreased physical activity can wreak havoc on glycemic control. Many providers understand and support the strategy of eliminating or reducing prandial insulin doses when patients are not eating well or when all oral intake has been withheld. Importantly, if measures are not taken to modify prandial insulin orders in these situations, then the providers’ worse fear of inducing hypoglycemia can become a reality. This is especially true for the rapid acting prandial insulin’s (as part, glulisine and lispro).

These newer analogs have faster onset and peak effects coupled with a shorter duration of action compared to the traditional short-acting regular insulin and offer a more physiologic approach to managing postprandial hyperglycemia [2].

The management of basal insulin therapy is an animal of a different color. Basal insulin is used to mimic the 24 hour normal continuous (but pulsate) physiologic insulin secretion. It serves to suppress hepatic glucose production (and resulting fasting blood glucose levels) and controls inter-meal glucose levels. Hospitalized persons with type 1 diabetes who have poor (or no) oral intake may require downward adjustment or discontinuation of their prandial insulin. However, basal insulin is still required to prevent peripheral lipolysis and the subsequent development of diabetic ketoacidosis and its resulting fluid, electrolyte and acid-base disturbances in this patient population. Whether the full or a modified dose of basal insulin should be administered is often left up to the provider’s discretion. No universally-accepted consensus guidelines exist to assist the provider in this decision. That being said, I often find clinicians erring on the side of using less basal insulin than the regularly scheduled dose; again for fear of causing hypoglycemia. Importantly, the provider may fail to recognize that the hospitalized patient with diabetes is often under various stressors that tend to raise blood glucose. Infection, burns, trauma, sepsis, worry, fear, anxiety and even medication (e.g., corticosteroids) can trigger accouter-regulatory hormone response (e.g., cortisol, epinephrine, glucagon and growth hormone) resulting in elevated blood glucose and a worsening of glycemic control. At first this acute elevation in blood glucose levels may seem innocuous. Despite inconsistent findings, it’s understood that inadequate

glycemic control may lead to poor clinical outcomes in post-operative surgical patients and those suffering an acute myocardial infarction or serious infections [3-6].

These same above-mentioned insulin dosing principles also apply to persons with type 2 diabetes receiving insulin. The importance of adequate insulinization carries the same benefits in this population as those with type 1 diabetes namely improved glycemic control. Since the majority of insulin marketed and administered in the United States is for persons with type 2 diabetes, the provider is much more likely to deal with dosing decisions in this population of patients.

Unlike persons with type 1 diabetes, insufficient insulinization generally doesn't result in diabetic ketoacidosis. Most persons with type 2 diabetes still retain some endogenous capacity to secrete insulin from their beta cells. Preserved insulin production and secretion are sufficient to inhibit lipolysis and its resultant hepatic generation of ketones responsible for the development of metabolic acidosis. Importantly, persons with long-standing type 2 diabetes and/or significantly depleted beta-cell mass may develop DKA following significant stressors such as burns, trauma or sepsis. Interestingly, certain select populations (African Americans and Hispanics) with so called ketosis-prone type 2 diabetes may initially present with DKA but may not require long-term insulin therapy [7].

Infection was mentioned earlier as a stressor that could significantly impact an individual's glycemic control. Hyperglycemia has been associated with impaired chemo taxis and phagocytosis by polymorphonucleocytes, elevated levels of inflammatory cytokines and possible effects on T-cell function [3]. Therefore, it's imperative that persons with diabetes suffering from various infections maintain near-euglycemia throughout the course of the illness. Providers fearing hypoglycemia may be reluctant to aggressively manage these patients jeopardizing an optimal clinical outcome or risking treatment failure. I have seen this reluctance in my own clinical practice as well as the benefits derived from improved glycemic control especially in soft tissue infections.

The key to overcoming the fear of using insulin in the hospitalized patient is education. Providers must understand the factors/stressors that contribute to poor glycemic control in this patient population (e.g., acute illness, physical inactivity, dietary changes, etc.) and its consequences. Similarly, the pharmacokinetics/pharmacodynamics

of insulin and its therapeutic use must be well understood by the provider. Insulin protocols can also be developed to assist the provider with dosing decisions in select patient groups including those scheduled for surgery/procedures, receiving continuous enteral/parenteral nutrition and requiring no or limited oral intake [8,9]. In addition, instructional initiatives/curricula addressing these issues should be targeted at the undergraduate medical education level with continuing efforts during residency training and beyond. These strategies will better position the provider to make appropriate therapeutic adjustments to insulin regimens resulting in improved blood glucose control and clinical outcomes in acutely ill persons with diabetes.

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