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Analysis of Hyperglycemia in Individuals with Poor Health

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Introduction

Observational studies have found that the prevalence of hyperglycemia and diabetes in hospitalised patients ranges from 38 percent to 40 percent, and that 70-80 percent of diabetics with severe diseases or cardiac surgery had hyperglycemia and diabetes. Hyperglycemia (defined as a glucose level >180 mg/dl [10.0 mmol/l]) was found to be prevalent in 32.2 percent of ICU patients and 32.0 percent of non-ICU patients in a 2017 report based on data from point-of-care bedside glucose tests in nearly 3.5 million people (653,359 ICU and 2,831,436 non-ICU) from 575 hospitals in the United States. These figures comprised those with newly diagnosed or stress hyperglycemia, as well as people who had previously been diagnosed with diabetes.

Any blood glucose concentration >140 mg/dl (>7.8 mmol/l) in patients without a prior history of diabetes was defined as stress hyperglycemia or hospital-related hyperglycemia by the American Diabetes Association (ADA) and American Association of Clinical Endocrinologists (AACE) consensus on inpatient hyperglycemia. Despite the fact that stress hyperglycemia usually fades when the acute sickness or surgical stress subsides, a considerable number (up to 60% in some publications) of patients had diagnosed diabetes six to twelve months following discharge. In persons with hyperglycemia but no history of diabetes, HbA1c testing is recommended to distinguish between stress-induced hyperglycemia and previously undetected diabetes [1].

Hyperglycemia Pathophysiology during Illness

Plasma glucose is maintained between 70 and 100 mg/dl (3.9 and 5.6 mmol/l) in fasting people without diabetes through a finely controlled equilibrium between hepatic glucose synthesis and glucose utilisation in peripheral tissues. Because the brain cannot generate or store glucose, maintaining a normal glucose concentration is critical for cardiovascular and central nervous system function.

A dynamic, minute-to-minute control of endogenous glucose synthesis and glucose utilisation by peripheral tissues maintains systemic glucose homeostasis. Gluconeogenesis, also known as glycogenolysis, is a process that produces glucose in the liver and, to a lesser extent, the kidneys [2]. Gluconeogenesis occurs when non-carbohydrate precursors such lactate, alanine, and glycerol are converted to glucose in the liver. Excess glucose is polymerized and stored as glycogen mostly in the liver and muscle. Hyperglycemia results from three factors: enhanced gluconeogenesis, faster glycogenolysis, and decreased glucose consumption by peripheral tissues.

The key pathologic alteration is abnormally elevated hepatic glucose production. Increased hepatic glucose production is caused by increased availability of gluconeogenic precursors such as the amino acids alanine and glutamine, as a result of accelerated proteolysis and decreased protein synthesis; lactate, as a result of increased muscle glycogenolysis; and glycerol, as a result of increased lipolysis; and increased activity of gluconeogenic enzymes (phosphoenol pyruvate carboxykinase, fructose-1,6-bisphosphatase, and pyruvate carboxylase)

The interplay of glucoregulatory hormones — insulin and counter-regulatory hormones – maintains glucose metabolism (glucagon, cortisol, catecholamines and growth hormone) [3]. Insulin regulates hepatic glucose synthesis by inhibiting gluconeogenesis and glycogenolysis in the liver. Insulin increases protein anabolism in insulin-sensitive tissues including muscle, glucose absorption, and glycogen synthesis, while inhibiting glycogenolysis and protein breakdown, depending on the quantity in the circulation. Insulin is also a potent inhibitor of lipolysis, fatty acid oxidation, and ketogenesis.

In addition to glucagon, cortisol, catecholamines, and growth hormone, counter-regulatory hormones (glucagon, cortisol, catecholamines, and growth hormone) play a significant role in glucose production and utilisation control. Because glucagon is the most significant glycogenolytic hormone, it regulates hepatic glucose generation in both normal and hyperglycemic states. Excess counter-regulatory hormones cause altered carbohydrate metabolism by developing insulin resistance, increasing hepatic glucose synthesis, and lowering peripheral glucose consumption during stressful situations. Furthermore, high amounts of adrenaline promote glucagon production while inhibiting insulin release by pancreatic –cells [4].

Hyperglycemia causes an inflammatory response defined by an increase in pro-inflammatory cytokines and oxidative stress markers. TNF-, interleukin [IL]-6, IL1-ß, IL-8, and C-reactive protein levels in the blood are considerably higher two to fourfold in persons with severe hyperglycemia on admission compared to control subjects, and levels return to normal after insulin therapy and resolution of hyperglycemic crises. Increased TNF- causes insulin resistance through altering the regulation of the insulin signalling pathway and at the level of the insulin receptor. Increased quantities of these inflammatory cytokines can enhance insulin resistance by interfering with insulin signalling during acute stressful conditions, according to growing data. TNF- also lowers insulin-stimulated glucose absorption in peripheral tissues by inhibiting insulin-mediated activation of phosphatidylinositol 3-kinase.

Hyperglycemia's Consequences in Hospitalized Patients

The processes underlying hyperglycemia's harmful consequences during acute diseases are not totally understood. According to current evidence, severe hyperglycemia causes impaired neutrophil granulocyte function, high circulating free fatty acids, and overproduction of pro-inflammatory cytokines and reactive oxygen species (ROS), all of which can cause direct cellular damage as well as vascular and immune dysfunction.

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Hyperglycemia is linked to poor outcomes in people who aren't in the ICU but are admitted to general medical and surgery services. Hyperglycemia is linked to poor hospital outcomes in these patients, including longer hospital stays, infections, impairment after release, and mortality. In a study of 1,886 patients admitted to a community hospital, mortality on the general floors was significantly higher in patients with newly (stress) diagnosed hyperglycemia and known diabetes compared to subjects with normal glucose values (10 percent vs. 1.7 percent vs. 0.8 percent, respectively; $p< 0.01$) [5].

Surgical procedures in general Patients who have hyperglycemia during the intraoperative period are more likely to have negative outcomes. High peri-operative glucose levels were linked to an elevated risk of surgical site infection in a comprehensive evaluation of diabetes and the risk of surgical site infection across a number of surgical specialities. In a case-control study, patients undergoing elective non-cardiac non-vascular surgery had higher preoperative glucose levels, which increased the risk of postoperative death. Patients with glucose levels of 110-200 mg/dl (5.6-11.1 mmol/l) and >200 mg/dl (>11.1 mmol/l) had 1.7-fold and 2.1-fold higher mortality, respectively, as compared to patients with glucose levels of 5.6 mmol/l (110 mg/dl).

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