Stress-induced hyperglycemia causes a state of insulin resistance and elevated blood glucose levels

Yang Lu*

Introduction

Hyperglycemia is a common finding in critically ill patients and usually occurs within the first 48 hours after ICU admission in half of the patients. Numerous studies have shown a significant association between ICU and ICU admission blood glucose levels and outcomes. Stress-Induced Hyperglycemia (SIH) causes a state of insulin resistance and elevated blood glucose levels through multiple mechanisms. Counter-regulatory hormones such as catecholamines, cortisol, glucagon, and growth hormone interfere with glucose hemostasis. An increase in inflammatory cytokines also worsens the metabolic environment. Therefore, hepatic gluconeogenesis is unregulated. Glucose uptake by skeletal muscle via glucose transporter type 4 is also impaired. In addition, insulin levels themselves are lowered to combat hyperglycemic conditions. Hyperglycemia has been associated with increased mortality in a retrospective study conducted in the United States, highlighting the importance of tight glycemic control. Moreover, studies show that the SIH condition, not diabetic hyperglycemia, is the primary cause of increased mortality and morbidity.

Description

Achieving adequate glycemic control safely in the inpatient setting requires a coordinated approach by a multidisciplinary team. Processes that include proper diagnosis, optimal ICU management, and robust continuity of care contribute significantly to reducing morbidity risk. Intravenous insulin therapy is the standard treatment for hyperglycemia. Recently, continuous glucose monitoring systems have come into use in hospitals. These are devices that provide continuous blood glucose monitoring without the need for frequent contact by a healthcare professional. Although there is no convincing evidence of improved patient outcomes, the risk of hypoglycemia was significantly reduced. Increased glycemic excursions and the number of hypoglycemic events have also been recognized as leading to poor patient outcomes. The purpose of this review is to outline the management of SIH by discussing in detail possible investigations, insulin needs, and nutritional advances, while highlighting potential complications.

Counter regulatory hormones and proinflammatory cytokines are involved in the metabolic milieu that occurs in SIH. Increased gluconeogenesis and insulin resistance are important factors. Interleukin-1, interleukin-6, and tumor necrosis factor are proinflammatory proteins that cause insulin resistance and suppress concentration-dependent insulin release. Elevated serum levels are associated with insulin resistance, which promotes hyperglycemia by releasing glucose from liver glycogen stores. In addition, hyperglycemia elevates blood levels, presumably through increased production in monocytes. TNF is associated with sepsis severity and is a major mediator in the development of sepsis. TNF, either by itself or by increasing circulating levels of free fatty acids, causes insulin resistance in animals. The hypothalamic-pituitary-adrenal axis is activated in stress-induced hyperglycemia, resulting in increased cortisol secretion from the adrenal glands. Cortisol synthesis is required for hemostasis of cells and maintenance of organ systems. Cortisol, catecholamines, glucagon, and growth hormone are all counter-regulatory hormones that decrease insulin release by increasing the activity of pancreatic alpha cells mediator.

Conclusion

Catecholamines further limit insulin binding, insulin activation by suppressing tyrosine kinase activity, and peripheral glucose uptake by GLUT-4. Similarly, glucocorticoids also limit glucose uptake in peripheral tissues, whereas growth hormone prevents insulin activation at tyrosine residues. Therapeutic interventions such as diet also play an important role in the development of hyperglycemia. SIH is associated with increased gene expression of hepatic glucose-6-phosphatase, a regulated gluconeogenic gene. Critically ill patients have elevated levels of insulin-like growth factor binding protein-1, a liver-derived protein that elongates insulin-like growth factors.

Acknowledgement

None

Conflict of interest

The author has nothing to disclose and also state no conflict of interest in the submission of this manuscript.

Department of Endocrinology, Fujian Medical University, China

Corresponding author: *Yang Lu*

E-mail: *yanglu@123.com*

Received: *30 November 2022, Manuscript No. ajdm-23-85684;* ***Editor assigned:*** *02 December 2022, Pre QC No ajdm-23- 85684 (PQ);* ***Reviewed:*** *16 December 2022, QC No ajdm-23- 85684;* ***Revised:*** *21 December 2022, Manuscript No. ajdm-23- 85684 (R);* ***Published:*** *28 December 2022*