Hyperglycemic Hyperosmolar State: A Diabetic Emergency

Rachel Fisher

Otterbein University, rachel.fisher@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn

Part of the Endocrine System Diseases Commons, Medical Pathology Commons, and the Nursing Commons

Recommended Citation


https://digitalcommons.otterbein.edu/stu_msn/118

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Master of Science in Nursing (MSN) Student Scholarship by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact shickey@otterbein.edu.
Hyperglycemic Hyperosmolar State: A Diabetic Emergency
Rachel Fisher, RN
Otterbein University, Westerville, Ohio

Introduction

Hyperglycemic hyperosmolar state (HHS) is an acute complication associated with diabetes mellitus. HHS has been known by several other names in the past. These labels include Hyperosmolar Nonketotic Coma, Hyperosmolar Hyperglycemic Nonketotic Coma, and Hyperosmotic Hyperglycemic Nonketotic Syndrome (Hackel, 2014). HHS is characterized by severe hyperglycemia, profound dehydration, and is often accompanied by altered mental status. Less than 1% of diabetes-related admissions are due to HHS, but mortality rates are estimated to be 10% to 20% (Lenahan & Holloway, 2015). HHS typically affects the older adult population, with the average age of HHS patients being 60 years (Collopy, Krebs, & Snyder, 2013). The patient with HHS commonly has type 2 diabetes (Pasquel & Umpierrez, 2013). Up to 40% of those affected have never been diagnosed with diabetes prior to developing HHS (Collopy et al., 2013).

HHS is a medical emergency. The incidence of HHS is expected to rise in the future due to the increase in the occurrence of type 2 diabetes (Lenahan & Holloway, 2015). It is important for healthcare providers to recognize HHS in order to provide appropriate care for the patient and reduce the mortality rates associated with this diabetic complication.

Patient Presentation

The patient with HHS presents with altered mental status, blood glucose level of >600 mg/dl, and with no or minimal ketones present in the urine (Van Ness-Ottnann & Hack, 2013). Poor skin turgor and elevated serum sodium are present due to the profound dehydration caused by severely elevated glucose blood levels (Van Ness-Ottnann & Hack, 2013). The patient may exhibit polyuria and polyphagia, but these compensatory mechanisms may possibly be blunted by the severe dehydration and altered mental status that are characteristic of HHS (Hackel, 2014). Because of the endogenous insulin present in type 2 diabetics, adipose tissue is not broken down for energy, and therefore metabolic acidosis does not occur (Pollack & Funk, 2013).

Signs & Symptoms
• Blood glucose level >600 mg/dl
• Polyuria
• Polyphagia
• Poor skin turgor
• Drowsiness
• Confusion
• Coma

Pathophysiological Process

In type 2 diabetes mellitus, the body tissues become increasingly resistant to the effects of insulin, which leads to a relative insulin deficiency (Van Ness-Ottnann & Hack, 2013). There is usually a precipitating event, such as an infection (i.e., pneumonia, urinary tract infection) or medication side effect, that initiates hyperglycemia in the patient (Hackel, 2014). The body responds to the stress by releasing hormones such as catecholamines, cortisol, glucagon, and growth hormone that cause increased glucose levels (Pasquel & Umpierrez, 2014). Insulin resistance causes inadequate use of the glucose by the peripheral tissues, which also contributes to the rising hyperglycemia (Pasquel & Umpierrez, 2014). Increased blood glucose levels cause an increase in the osmolality of extracellular fluid, which leads to a shift in fluid from the cells to the extracellular space (Pasquel & Umpierrez, 2014). As the glucose level rises, the kidneys attempt to filter the glucose from the blood to the urine via osmotic diuresis (Hackel, 2014). The cells become dehydrated, the blood becomes concentrated, and the lack of fluid restricts the ability of the kidneys to rid the body of excess glucose (Hackel, 2014). Thirst is triggered in an attempt to counteract the dehydration (Hackel, 2014). The alteration in electrolytes due to the dehydration leads to the mental status changes (ranging from stupor to coma) associated with HHS (Pasquel & Umpierrez, 2014).

Figure 1. Pathophysiology of HHS (Zeitzler, Haq, Rovenstine, & Glauser, 2011, p. 19)

Significance of Pathophysiology

The endogenous insulin present in patients with HHS allows the process to progress over the course of days to weeks (Hackel, 2014). This insidious onset enables the body to adapt to the hyperglycemic state (Hackel, 2014). Because of this adaptation, the patient may not seek medical care until they are critically ill (Hackel, 2014). This failure to seek care is compounded by the mental status changes that accompany HHS (Hackel, 2014).

Implications for Nursing Care

During the acute phase of HHS, neurological assessments must be completed and treatment is to keep the blood glucose level between 250 and 300 mg/dl, until HHS is resolved (Pollock & Funk, 2013). After the resolution of HHS, the focus of nursing care should be education. Newly diagnosed diabetics will benefit from speaking with a diabetic educator. Since diabetes is a chronic disease process important to health care providers and their patients.

References


Additional Sources