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Hyperglycemic Hyperosmolar State: A Diabetic Emergency

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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 hyperglycemic hyperosmolar nonketotic syndrome (Hackel, 2015). HHS is characterized by severe hyperglycemia, profound dehydration, and is often accompanied by altered mental status. Loss of all 1% of diabetes-related admissions are due to HHS, but mortality rates are estimated to be 1% to 20% (Lanham & Holloway, 2015). HHS typically affects the older adult population, with the average age of HHS patients being 60 years (Collop, Krishen, & Snyder, 2013). The patient with HHS commonly has type 2 diabetes (Pasquel, Umpierrez, & Hack, 2013). Up to 40% of those affected have never been diagnosed with diabetes prior to developing HHS (Collop 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 (Lanham & 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 acute 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-Ottmann & Hack, 2013). Poor skin turgor and elevated serum osmolality are present due to the profound dehydration caused by severely elevated blood glucose levels (Van Ness-Ottmann & Hack, 2013). The patient might 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 (Pollock & 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-Ottmann & 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). This results in an increase in the gluconeogenesis and the production of ketones (Van Ness-Ottmann & Hack, 2013). 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).

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). This failure to seek care is compounded by the mental status changes that accompany HHS (Hackel, 2014).

During the acute phase of HHS, neurological examinations must be completed and measures to control and reduce the hyperglycemia should be considered due to the altered electrolyte levels associated with the disease process (McNaughton, Sell, & Sivori, 2011). The altered mental state makes safety a concern, and fall precautions should be implemented. Cardiac monitoring is also necessary because the electrolyte imbalances can lead to life-threatening arrhythmias (McNaughton et al., 2011). Urine output should be closely monitored due to the possibility of acute renal failure from severe dehydration (Van Ness-Ottmann & Hack, 2013). IV fluids should be used to correct the dehydration (McNaughton et al., 2011). Excessive hydration should also be avoided to prevent the development of cerebral edema (Pollock & Funk, 2013). Electrolyte levels will be closely monitored and corrected as needed (Pollock & Funk, 2013).

Blood glucose levels must be monitored frequently as the patient will likely be treated with a continuous insulin infusion (Van Ness-Ottmann & Hack, 2013). Healthcare providers should be aware that the goal of 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 major precipitating factors for HHS are illness and infection, sick day management education is a must (Lanham & Holloway, 2015).

Conclusion

HHS is a potentially lethal hyperglycemic emergency. The insidious onset of the disease leads to the critical state of HHS patients once they present for care. HHS must be carefully monitored and managed in order to prevent complications. The expected increase in incidence of HHS and the high mortality rates associated with it makes understanding the unique aspects of this disease process important to health care providers and their patients.

References


Additional Sources