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A Review of Acute Complications of Hyperglycaemia

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Hyperglycemia can cause serious metabolic emergencies, such as diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS). Both conditions are caused by absolute or relative insulin deficiency associated with excessive counter-regulatory hormones (glucagon, growth hormone, cortisol, catecholamines) during illness [1].

DKA tends to be common in young people with type one diabetes, whereas HHS is more common among older patients.

Overlap between these two conditions can occur in insulin dependent type two diabetes. Some patients with HHS, especially when there is severe dehydration, can present with mild or moderate acidosis that is mainly due to hypoperfusion/lactic acidosis. On the other hand, some patients with Type one diabetes can present with some features of HHS, such as severe hyperglycaemia and renal failure [1].

Diabetic ketoacidosis

DKA is an acute life-threatening complication of diabetes, characterized by the triad of hyperglycemia (> 11), metabolic acidosis (decreased pH <7.3 and bicarbonates <15), and increased total body ketone concentration. DKA can be the first presentation of Type one diabetes and can occur in up to 25% of cases of Type two diabetes. In addition, DKA is a common complication in patients with known type one diabetes, where it may be the consequence of poor compliance with insulin treatment, acute illness, or malfunction of diabetes care equipment. Mortality associated with DKA is predominantly related to the occurrence of cerebral oedema, which occurs in 0.3–1% of patients, whereas only a minority of deaths in DKA is due to other causes [2].

Early identification and treatment of DKA are essential to minimize the associated morbidity and mortality. Treatment of DKA requires strict monitoring with correction of hyperglycemia, acidosis and ketosis, and replacement of fluid, electrolyte losses and insulin infusion of 0.1 unit/Kg/hour.

Another important action in the management of DKA is the identification and treatment of precipitating events like infection for example [2].

Hyperosmolar hyperglycemic state

HHS is the most serious acute hyperglycemic emergency in patients with Type two diabetes. Diagnostic criteria for this condition are glucose level > 30 and increased effective plasma osmolality >320 mOsm/kg, in the absence of ketoacidosis. The incidence of HHS is estimated to be <1% of hospital admissions of patients with diabetes and the associated mortality is 10–20%. There are no good data from randomized studies on the best management of HHS, which has been mainly extrapolated from studies of patients with DKA. In HHS, the goals of initial fluid therapy are to expand the intra- and extravascular volume, restore normal renal perfusion, and promote a gradual decline in serum sodium concentration and osmolality [2].

Patients with new hyperglycemia had higher in-hospital mortality rate and worse functional outcome than patients with a prior history of diabetes and subjects with normoglycemia. In addition, patients with new hyperglycemia had an increased length of hospital stay, were more likely to require admission to an ICU, and were more likely to require transfer to nursing home facility at discharge. Patients with newly diagnosed hyperglycemia were more likely to be admitted to the ICU, had a longer length of hospital stay, and were less likely to be discharged home [3].

Stress Hyperglycemia

Defined as a transient increase in blood glucose concentration during acute physiological illness, represents two distinct populations: those with undiagnosed diabetes or impaired glucose tolerance, and those who develop hyperglycemia as the result of the severe stress and increased counterregulatory hormones. Although the underlying mechanisms for the development of stress hyperglycemia are not fully understood, several potential mechanisms have been proposed. These include increased substrate availability in the form of lactate, increased gluconeogenesis and decreased glycogenolysis due to increased secretion of counterregulatory hormones (catecholamines, cortisol, and glucagon), and peripheral insulin resistance which is exacerbated here with the use of steroids.

Although in such patients, the high morbidity and mortality relate to the associated illness precipitating the stress, hyperglycemia itself may contribute to morbidity by creating a toxic cellular effect, causing intracellular and extracellular dehydration, inducing electrolyte abnormalities, and depressing immune function.

Our study did not address the question of whether treatment of hyperglycemia may reduce the high morbidity and mortality associated with hyperglycemia in patients with and without a history of diabetes. Recently, Van den Berghe et al. reported that among mechanical ventilated adult patients admitted to an ICU, strict normalization of blood glucose levels (4.5–6.1 mmol/L) with continuous infusion of insulin compared with a restrictive insulin regimen to maintain blood glucose levels between 10–12 mmol/L resulted in reduced hospital morbidity and mortality. The intensive insulin schedule significantly reduced ICU mortality by 43% (death odds ratio, 0.52; range, 0.33–0.81), hospital mortality by 34%, mean ICU stay by 22%, and incidence of bacteraemia and haemodialysis by 50%. These results suggest that strict glucose control in hospitalized patients with hyperglycemia is warranted [4].

The Normoglycemia in Intensive Care Evaluation-Survival Using Glucose Algorithm Regulation (NICE-SUGAR) trial compared the effectiveness of a strategy of intensive glucose control (blood glucose target 81–108 mg/dl or 4.5–6.0 mmol/l) to conventional glucose control (blood glucose target ≤ 180 mg/dl or 10.0 mmol/l) in 6,104 medical and surgical patients admitted to adult ICUs in 42 hospitals across four countries. Compared to conventional glucose control, this study found that intensive glucose control increased the risk for severe hypoglycaemia (blood glucose ≤ 40 mg/dl or 2.2 mmol/l; 6.8% versus 0.5%, $p < 0.001$) and 90-day mortality (27.5% versus 24.9%, $p = 0.02$). The results of this large pragmatic trial suggest that blood glucose control using conventional targets (≤ 180 mg/dl or 10.0 mmol/l) should be the preferred approach for adult medical and surgical patients admitted to ICU.

In conclusion, in-hospital hyperglycaemia is a common finding and should be considered an important marker of poor clinical outcome and increased mortality, in patients without a history of diabetes. Patients with new hyperglycemia admitted to critical care areas or to general medical and surgical wards had a significantly higher mortality rate and lower functional outcome than patients with a known history of diabetes or normoglycemia. We strongly recommend that all hospitalized patients should be screened for hyperglycaemia and glucose levels are kept between 7-10 mmol/L during acute illness.

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