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Original research

Active cocaine use does not increase the likelihood of hyperglycemic crisis

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ABSTRACT

Objective: Hyperglycemic crisis encompasses a group of diabetes emergencies characterized by insulin deficiency with high morbidity and mortality. Cocaine use is increasingly prevalent in the United States and may be associated with increased risk of diabetic ketoacidosis. The objective was to determine if active cocaine use at hospital admission could be considered a risk factor for development of hyperglycemic crisis.

Methods: A retrospective case-control analysis was performed on 950 inpatients with hyperglycemia at an urban academic hospital. Patients admitted with non-emergent hyperglycemia were compared to patients who met criteria for diabetic ketoacidosis (DKA), hyperosmolar hyperglycemic state (HHS), and hyperosmolar ketoacidosis (HK), based on the absence or presence of cocaine metabolites on urine toxicology screen. Outcomes included frequency of cocaine use in patients with DKA, HHS, HK, and non-emergent hyperglycemia; phenotypic characteristics of cocaine users vs. non-users with hyperglycemia; phenotypic characteristics of patients with hyperglycemic crisis vs. non-emergent hyperglycemia.

Results: 950 patients were admitted with hyperglycemia, 133 of which met criteria for hyperglycemic crisis. There was no significant difference in the frequency of cocaine use in individuals with non-emergent hyperglycemia compared to individuals with hyperglycemic crisis (16.9% vs. 17.2%, p = 0.90). 16.9% of patients with DKA, 16.4% of patients with HHS, and 6.4% of patients with HK were cocaine users.

Conclusions: We found no association between active cocaine use at the time of hospital admission and development of hyperglycemic crisis, when compared to non-emergent hyperglycemia. The role of routine screening for cocaine use in patients with hyperglycemic crisis is unclear.

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Introduction

Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) are the most common diabetes emergencies [1]. The overlap syndrome of hyperosmolar ketoacidosis (HK) is less common, but along with HHS, is associated with higher mortality than DKA [2]. These three conditions, which constitute the spectrum of hyperglycemic crisis, result from absolute or relative insulin deficiency in the setting of excess counter-regulatory hormone production [3]. The pathophysiological mechanisms are best described in DKA, in which acute insulin deficiency results in an increase in free fatty acid production. This leads to excess ketone body production, reduced urinary clearance, and resultant metabolic acidosis [4]. HHS is characterized by marked hyperglycemia and hyperosmolarity, the combination of which leads to osmotic diuresis, volume depletion, hyperviscosity and ultimately, tissue hypoperfusion and worsening insulin resistance [5].

Cocaine has previously been described as contributing to worsening hyperglycemia through a combination of direct increase in counter-regulatory hormones and indirect promotion of insulin omission [6–8]. Prior clinical studies of the relationship between

Abbreviations: BMI, body mass index; DKA, diabetic ketoacidosis; HgbA1C, hemoglobin A1C; HHS, hyperosmolar hyperglycemic state; HK, hyperosmolar ketoacidosis; IDR, inpatient diabetes repository; T1D, type 1 diabetes.

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cocaine and hyperglycemic crisis have reported a possible association between patient self-reported and laboratory confirmed cocaine use and subsequent development of DKA. However, a direct association between the two is not well established, given limitations in prior studies, which include variable definitions of hyperglycemic crisis, an exclusive focus on DKA and the fact that concomitant cocaine use has not necessarily been strictly confirmed at the time of admission for hyperglycemic crisis.

In order to address this uncertainty, we conducted a retrospective case-control study to further our understanding as to whether active cocaine use, confirmed through urine toxicology screening at the time of hospital admission, could be considered a risk factor for development of hyperglycemic crisis.

We hypothesized that confirmed cocaine use (urine cocaine metabolites being present at the time of admission with hyperglycemia) would be encountered more frequently in patients admitted with hyperglycemic crisis compared with patients admitted with non-emergent hyperglycemia. Secondarily, we hypothesized that individuals with hyperglycemic crisis who use cocaine might exhibit different phenotypic features, such as more severe hyperglycemia, compared to those not using cocaine. Because so little is known about the role of cocaine in the clinical course of HHS and HK, we also hypothesized that there may be differential effects of cocaine use on the patients' metabolic characteristics depending on whether individuals were diagnosed with DKA, HHS or HK. Through these questions, we hoped to define clinical features that would lead clinicians to suspect cocaine as a contributing risk factor in hyperglycemic crisis.

Methods

We extracted a limited data set from an established, Institutional Review Board-approved inpatient diabetes repository (IDR) of approximately 40,000 individual subjects admitted with hyperglycemia at Boston Medical Center, an urban academic hospital with a large "safety-net population." We identified a sub-group of 950 individuals admitted with both hyperglycemia (blood glucose > 250 mg/dL) and available urine toxicology screens for cocaine between July 1, 2004, and December 31, 2010.

Using this dataset, we performed a retrospective case-control analysis to compare a group of individuals with hyperglycemic crisis to a group of hyperglycemic individuals without crisis. We included individuals between the ages of 18 and 65 (age limits were pre-determined by the available IDR dataset) with blood glucose > 250 mg/dL on emergency department laboratory evaluation with available urine toxicology screens at the time of the admission of interest. We defined a group of 133 individuals from all ethnic groups who met diagnostic criteria for DKA, HHS or HK as the hyperglycemic crisis group (cases). The DKA group was defined by the presence of all of the following criteria: venous or arterial pH < 7.3 and/or serum bicarbonate < 15 mmol/L, ketonuria, and anion gap > 14; the HHS group was defined by both blood glucose > 600 mg/dL and measured serum osmolarity > 340 mOsm/kg H2O or effective serum osmolarity > 320 mOsm/kg H2O; the HK group was defined by venous or arterial pH < 7.3 and/or serum bicarbonate < 15 mmol/L, ketonuria, and measured serum osmolarity > 340 mOsm/kg H2O or effective osmolarity > 320 mOsm/kg H_2O. Effective osmolarity was defined as: 2[measured Na^+(mmol/L)] + glucose (mg/dL)/18. Total osmolarity was defined as: 2[measured Na^+ (mmol/L)] + glucose (mg/dL)/18 + BUN/2.8. It is important to note that based on these criteria, certain individuals may meet criteria for more than one diagnosis and as a result were "double counted" in subsequent analyses. For example, a patient meeting criteria for HK may meet all of the criteria for DKA but if blood sugar is less than 600 mg/dL would not meet all of the criteria for HHS, so would be included in the analyses for both HK and DKA, but not HHS. Individuals with hyperglycemia not meeting the aforementioned criteria for hyperglycemic crisis were designated as subjects for the non-emergent hyperglycemia control group (controls).

We deliberately excluded individuals without available urine toxicology data to ensure that antecedent cocaine exposure was confirmed. Cocaine exposure was defined as the presence of cocaine metabolites on urine toxicology screen on presentation to the emergency department or admission to the intensive care unit. Serum toxicology screens were not included as urine toxicology screening is the routine practice at our institution. The decision to perform toxicology screening was at the discretion of the emergency department and intensive care unit teams.

We performed multiple analyses in this group of hyperglycemic individuals who had undergone urine toxicology testing designed to explore the hypotheses that cocaine use is more likely to be associated with DKA, HHS, and HK than non-emergent hyperglycemia; that cocaine acts as an effect modifier for the relationship between demographic factors and hyperglycemic crisis; and that there may be phenotypic differences between hyperglycemic crisis cocaine users and hyperglycemic crisis non-users.

We first describe the hyperglycemic crisis and non-hyperglycemic crisis groups in terms of demographic characteristics, laboratory values and cocaine use. The categorical variables are presented as counts and percentages, and the continuous variables as means, standard deviations, medians and ranges. Fisher's exact test was used to compare categorical factors across the groups and *t*-test was used to compare continuous variables. We describe the hyperglycemic crisis subgroups – DKA, HHS and HK – in terms of demographic characteristics, laboratory values and cocaine use.

A multivariable logistic regression was used to evaluate the relationship between cocaine use and hyperglycemic crisis controlling for possible confounders: age, gender, race (self-identified by patient), education, primary language, body mass index (BMI) and hemoglobin A1C (HgbA1C). Odds ratios with 95% confidence intervals are reported. To examine potentiation effect modification by gender, race and education, we added multiplicative interaction terms with cocaine use to the model.

To examine potential phenotypic differences between hyperglycemic crisis cocaine users and non-users, we used Fisher's exact test for categorical variables and *t*-test for continuous variables. All analyses were performed using SAS v9.3. P values < 0.05 were considered statistically significant.

Results

Demographics

Of the 950 patients admitted with hyperglycemia and available urine toxicology data between July 1, 2004, and December 31, 2010, 133 patients presented with hyperglycemic crisis. Their clinical characteristics are shown in Table 1. Compared to those admitted with non-emergent hyperglycemia, those with hyperglycemic crisis were younger (44.9 vs. 47.7 years, p = 0.008). There were more men and black patients admitted across both groups, but a higher percentage of patients with hyperglycemic crisis were black compared to those without crisis (61% vs. 48%, p = 0.035). Patients with hyperglycemic crisis were more likely to have worse baseline glycemic control as determined by HgbA1C obtained during admission [11.7% (104 mmol/mol) vs. 10% (86 mmol/mol), p < 0.001] and more significant hyperglycemia on admission (average blood glucose 707.9 vs. 406.5 mg/dL, p < 0.001). The groups did not differ in primary language spoken, education level, BMI, or cocaine use.

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