Journal of Medical Toxicology
https://doi.org/10.1007/s13181-026-01134-8

ORIGINAL ARTICLE

Is Beta Blocker Toxicity Associated With Hypoglycemia?

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Received: 2 January 2026 / Revised: 24 February 2026 / Accepted: 10 March 2026

Abstract

Introduction It is commonly taught that beta blocker toxicity is associated with development of hypoglycemia, however, empirical evidence is lacking. We aimed to evaluate the association of beta blocker toxicity with hypoglycemia.

Methods We performed a retrospective cohort study of cases reported to a regional poison center from 1/1/2018 to 1/1/2025. Beta blocker toxicity was defined as cases coded as beta blocker exposure with documented bradycardia. Cases of acetaminophen or selective serotonin reuptake inhibitor (SSRI) overdose were selected as controls. Primary outcome was development of hypoglycemia defined as glucose concentration less than 70 mg/dL. A pre-planned subgroup analysis was performed for all groups, comprised of patients meeting the following criteria: single substance exposures, no documented diabetes, and no documented insulin administration. Descriptive statistics were performed, and relative risk for hypoglycemia was calculated between beta blocker and control groups.

Results There were 225 cases that met inclusion criteria for beta blocker toxicity, with 1,586 acetaminophen cases, and 1,955 SSRI cases included as controls. The relative risk of hypoglycemia for beta blockers compared to acetaminophen at any time was 1.44 (95% CI: 0.74, 2.80; $p=0.38$) and 1.74 (95% CI: 0.89, 3.38; $p=0.15$) compared to SSRI. No differences were identified between groups when evaluating the initial presenting glucose or in the pre-planned subgroup analysis.

Conclusions There was no significantly increased risk of hypoglycemia in beta blocker poisoned patients compared to controls. This suggests that hypoglycemia would not be an expected finding in beta blocker toxicity.

Keywords beta blockers · hypoglycemia · toxicity

Introduction

Beta adrenergic antagonists are a commonly prescribed class of medications frequently implicated in drug toxicity or overdose. In the 2023 annual report by America's Poison Center (APC), beta blockers were the seventh largest substance category responsible for fatalities, involved in 3.88% of all poisoning related deaths and 2.45% of single substance exposure deaths [1]. In overdose, beta blockers cause bradycardia and hypotension which can lead to cardiovascular collapse. It is frequently taught that beta blocker overdose is responsible for producing hypoglycemia, with mention in many prominent textbooks across disciplines including Emergency Medicine, Medical Toxicology, and Critical Care [2–5]. However, empirical evidence of this phenomenon is lacking.

In chronic therapeutic use, beta blockers have been associated with hypoglycemia, most commonly in pediatric patients [6, 7]. The proposed mechanism is interference with gluconeogenesis with episodes of hypoglycemia over prolonged therapeutic courses [8]. Whether the same mechanism might be invoked in the setting of acute toxicity is unclear. Empirical evidence evaluating the frequency in which hypoglycemia occurs in the setting of beta blocker

Supervising Editor: Jennifer Love, MD

Data in this study were previously presented in abstract form at the 2025 North American Congress of Clinical Toxicology (NACCT) in Chicago, Illinois.

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Published online: 28 March 2026

toxicity would clarify the toxicological syndrome and inform laboratory monitoring requirements in patients presenting with acute toxicity. We aimed to evaluate the association of hypoglycemia with acute beta blocker toxicity.

Methods

Design and Setting

We performed a retrospective cohort study at a single regional poison center from 1/1/2018 to 1/1/2025. This study was considered exempt from our Institutional Review Board as non-human subject research.

Patient Selection

Cases were identified using America's Poison Center (APC) substance coding. The case group consisted of all beta blocker cases that were bradycardic and had at least one documented blood glucose. Bradycardia was defined as heart rate < 60 bpm in adults, and per Pediatric Advanced Life Support criteria in pediatric patients which is in accordance with APC coding definitions. We included two control groups: acetaminophen (APAP) cases and selective serotonin reuptake inhibitor (SSRI) cases. These two groups were chosen because they would not be expected to cause hypoglycemia in acute overdose and are common exposures. Inclusion criteria for APAP cases were documentation of at least one blood glucose and treatment with N-acetylcysteine (NAC). N-acetylcysteine treatment was chosen as an inclusion criterion as a marker of significance of the exposure. Inclusion criteria for SSRI cases were presentation to a health care facility and at least one blood glucose measurement.

Data Collection

For each case, patient age, weight, sex assigned at birth, management site, medical outcome, clinical effects, therapies provided, documented blood glucose measurements, and documented vital signs were collected. Medical outcome, clinical effects, and therapies provided were reported in accordance with APC coding guidelines. Hypoglycemia was defined as serum blood glucose < 70 mg/dL in accordance with APC coding definitions. Initial blood glucose and vital signs were defined as the earliest documented in the poison center medical record. The lowest documented blood sugar was defined as the lowest blood glucose measurement recorded in the poison center medical record at any time. The primary outcome for this study was development of hypoglycemia. The secondary outcome was median serum glucose concentration.

Statistical Analysis

Descriptive statistics were performed reporting median and interquartile ranges (IQR) for demographic data as well as rates of different clinical interventions performed in each group. Relative risk of hypoglycemia was calculated comparing the experimental group to each control group separately. Due to the retrospective nature of the study with fixed sample sizes, an a priori power calculation was not performed. Effect sizes were estimated using 95% confidence intervals (95% CI). SciPy was used for statistics calculations [9]. A pre-planned subgroup analysis was performed to reduce confounding causes of hypoglycemia. For this, we excluded any cases from the initially defined cohorts that received insulin while in the hospital, had documentation of diabetes mellitus, and that were documented as polysubstance ingestions. An additional subgroup analysis was performed for all pediatric patients with documented age < 18 years old, as pediatric populations have been identified in prior studies as being at greater risk for development of hypoglycemia while taking beta blockers [6, 7]. The same statistical techniques were performed for each subgroup as per the entire cohort.

Results

We identified a total of 225 beta blocker cases that met inclusion criteria during our study period. For the control groups, 1,586 total APAP cases and 1,955 total SSRI cases met inclusion criteria. Descriptive statistics for the experimental groups and control groups as well as the pre-planned subgroups are found in Table 1. The beta blocker group was older and had a higher proportion male than both control groups. Median number of blood glucose values obtained in each group was one (Beta blocker IQR 1–2, max 7; APAP IQR 1–2, max 10; SSRI IQR 1–1, max 8). Additionally, the beta blocker group had lower heart rates, which is expected as bradycardia was an inclusion criterion, as well as lower blood pressure with a higher proportion of patients requiring vasopressor therapy. These differences remained in the subgroups as well.

For our primary outcome, there was no increased risk for development of hypoglycemia amongst patients with beta blocker toxicity as compared to either patients with APAP or SSRI toxicity (Fig. 1). The relative risk of hypoglycemia between the beta blocker group and the APAP group based on initial glucose value was calculated to be 0.24 (95% CI 0.33–1.75, $p = 0.21$) and at any time to be 1.44 (95% CI 0.74–2.80, $p = 0.38$). The relative risk between the beta blocker group and the SSRI group based on initial glucose value was calculated to be 0.29 (95% CI 0.04–2.15, $p = 0.32$)

Table 1 Patient characteristics

	All cases			Single substance, non-DM, no insulin		
	BB	APAP	SSRI	BB	APAP	SSRI
Patient Characteristics						
Total Cases	225	1586	1955	50	758	569
Female, n (%)	127 (56.4%)	1124 (70.9%)	1428 (73.0%)	32 (64.0%)	546 (72.1%)	449 (78.9%)
Age, median [IQR]	55.0 [33.0–67.0]	26.0 [16.0–46.0]	19.0 [15.0–35.0]	38.5 [18.0–61.8]	21.0 [15.0–41.0]	16.0 [14.0–20.0]
HR Initial, median [IQR]	55.0 [49.0–60.0]	85.0 [72.0–99.0]	89.0 [76.0–104.0]	55.5 [48.0–65.0]	84.0 [71.75–97.0]	90.0 [75.0–104.0]
HR Lowest, median [IQR]	51.0 [46.0–55.0]	75.0 [65.0–86.0]	80.0 [69.0–92.0]	49.5 [44.0–53.5]	74.0 [65.0–86.0]	83.0 [71.0–94.0]
BP Initial (SBP/DBP), median [IQR]	116.5/63.5 [98.0–133.0/56.0–74.25]	125.5/72.5 [113.0–138.0/63.0–82.0]	124.0/72.0 [112.0–136.0/63.0–81.0]	119.0/66.0 [102.0–133.0/57.0–76.0]	125.0/73.0 [114.0–137.0/64.0–81.0]	125.0/74.0 [115.0–135.0/65.0–81.0]
BP Lowest (SBP/DBP), median [IQR]	102.0/58.0 [91.0–118.0/52.0–67.25]	115.0/67.0 [105.0–127.0/58.75–77.0]	115.0/67.0 [104.0–127.0/58.0–76.0]	103.0/59.0 [94.0–112.0/56.0–70.0]	115.0/68.0 [106.0–127.0/60.0–77.0]	118.0/69.0 [108.0–128.0/61.0–77.0]
Therapies, n (%)						
Glucagon	75 (33.3%)	8 (0.5%)	27 (1.4%)	15 (30.0%)	0 (0.0%)	1 (0.2%)
Glucose	39 (17.3%)	67 (4.2%)	44 (2.3%)	2 (4.0%)	22 (2.9%)	1 (0.2%)
Insulin	34 (15.1%)	21 (1.3%)	24 (1.2%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Vasopressors	77 (34.2%)	83 (5.2%)	88 (4.5%)	10 (20.0%)	13 (1.7%)	3 (0.5%)
Atropine	53 (23.6%)	10 (0.6%)	19 (1.0%)	11 (22.0%)	0 (0.0%)	0 (0.0%)
Fluids	190 (84.4%)	937 (59.1%)	1248 (63.8%)	40 (80.0%)	385 (50.8%)	272 (47.8%)
Ventilator	26 (11.6%)	91 (5.7%)	98 (5.0%)	1 (2.0%)	14 (1.8%)	5 (0.9%)
Intubation	28 (12.4%)	90 (5.7%)	101 (5.2%)	1 (2.0%)	15 (2.0%)	3 (0.5%)
High Dose Insulin	22 (9.8%)	3 (0.2%)	10 (0.5%)	0 (0.0%)	0 (0.0%)	0 (0.0%)

BB Beta blocker; *APAP* Acetaminophen; *SSRI* Selective serotonin reuptake inhibitor; *DM* Diabetic; *HR* Heart rate

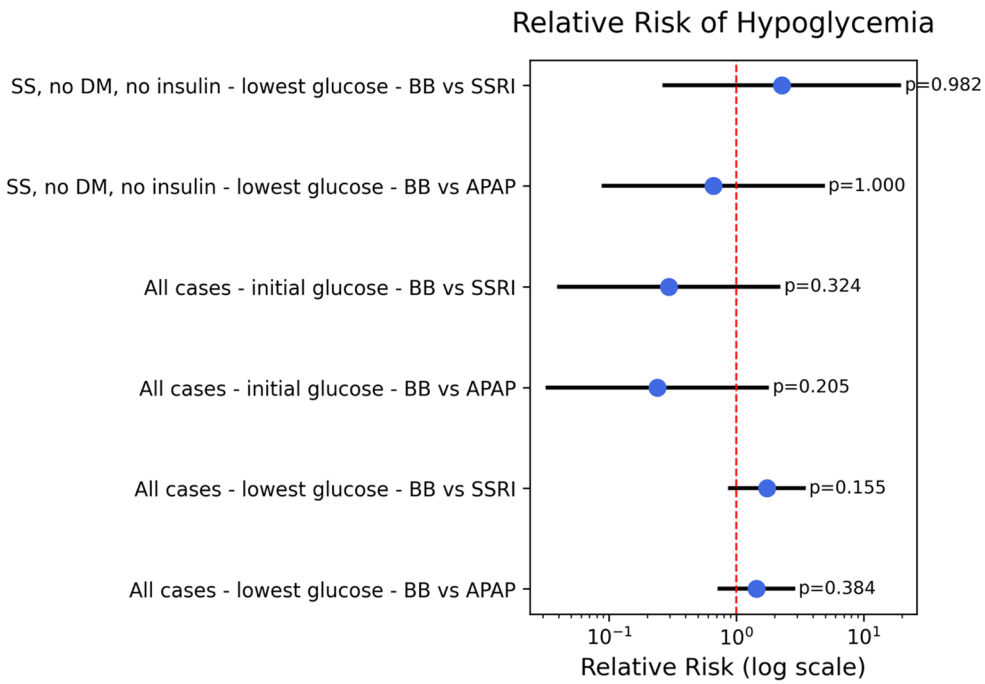

Fig. 1 Forest plot of relative risk of hypoglycemia between the beta blocker (BB) group and each of the two control groups as well as the pre-planned subgroup

and at any time to be 1.74 (95% CI 0.89–3.38, $p=0.15$). Subgroup analysis was consistent with the broader cohort, with no significant difference between groups (Fig. 1). In the subgroup including single substance ingestions only and excluding insulin administration and diabetic patients, the relative risk of hypoglycemia at any time between the beta blocker group and APAP group was calculated to be 0.66 (95% CI 0.09–4.78, $p=1.00$) and the relative risk of hypoglycemia at any time between the beta blocker group and SSRI group was calculated to be 2.28 (95% CI 0.27–19.10, $p=0.98$). The relative risk of hypoglycemia for the initial blood glucose measurement was unable to be calculated for

the subgroup due to no beta blocker subgroup cases having hypoglycemia initially.

The median initial and nadir blood glucose measurements between experimental and control groups were similar (Fig. 2; Table 2), and no differences were identified between groups on subgroup analysis of patients with reported single substance ingestion and no insulin administered (Fig. 3; Table 2).

In the pediatric subgroup, there were 33 beta blocker cases, 510 acetaminophen cases, and 847 SSRI cases (Table 3). Of these cases, none of the beta blocker cases had initial hypoglycemia, and only two (6.1%) patients developed hypoglycemia during their hospital course (Table 3). Of these beta blocker cases, there were 6 (18.2%) patients ≤5 years old. The median and IQR of glucose measurements in the pediatric subgroup were similar between groups in the pediatric cohort (Fig. 4). There was no increased relative risk of hypoglycemia for beta blocker cases in the pediatric subgroup (Fig. 5).

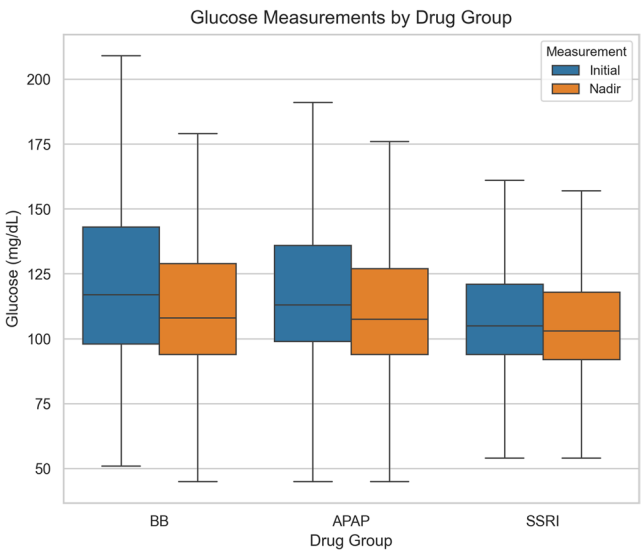

Fig. 2 Distributions of glucose measurements in all groups both initially and lowest measured

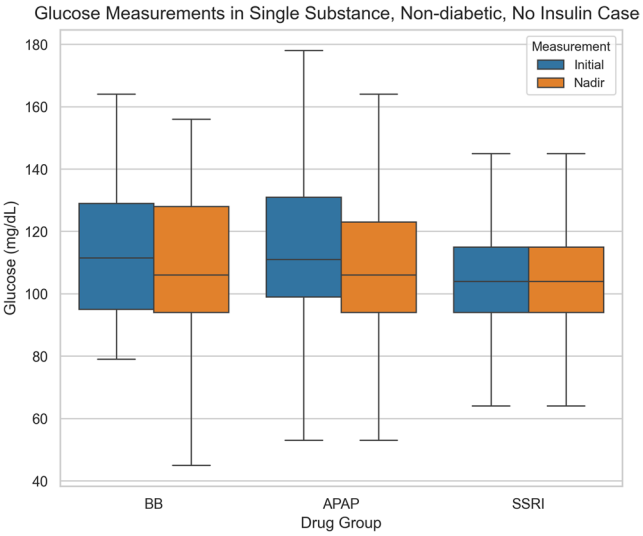

Fig. 3 Distributions of glucose measurements in the pre-planned subgroups both initially and lowest measured

Table 3 Distribution of nadir glucose values among pediatric cases

Drug Class	Total Cases (*n*)	Glucose < 70 (*n*, %)	Glucose ≥ 70 (*n*, %)
BB	33	2 (6.1%)	31 (93.9%)
Acetaminophen	510	8 (1.6%)	502 (98.4%)
SSRI	847	13 (1.5%)	834 (98.5%)

Discussion

In this cohort of patients with beta blocker toxicity reported to a regional poison center, there was no increased risk of hypoglycemia in beta blocker poisoned patients compared to controls. Patients with beta blocker toxicity had similar initial and nadir glucose compared to those presenting with APAP or SSRI toxicity, two conditions that are not associated with hypoglycemia. These findings run contrary to the commonly taught paradigm associating beta blocker toxicity with development of hypoglycemia.

Table 2 Characteristics of glucose for total cohort and pre-planned subgroup

Patient Characteristics	All cases			Single substance, non-DM, no insulin		
	BB	APAP	SSRI	BB	APAP	SSRI
Glucose Initial, median [IQR]	117.0 [98.0–143.0]	113.0 [99.0–136.0]	105.0 [94.0–121.0]	111.5 [95.0–129.0]	111.0 [99.0–131.0]	104.0 [94.0–115.0]
Glucose Lowest, median [IQR]	108.0 [94.0–129.0]	107.5 [94.0–127.0]	103.0 [92.0–118.0]	106.0 [94.0–128.0]	106.0 [94.0–123.0]	104.0 [94.0–115.0]
Initial hypoglycemia, n (%)	1 (0.4%)	30 (1.9%)	30 (1.5%)	0 (0.0%)	15 (2.0%)	4 (0.7%)
Any instance hypoglycemia, n (%)	10 (4.4%)	49 (3.1%)	50 (2.6%)	1 (2.0%)	23 (3.0%)	5 (0.9%)

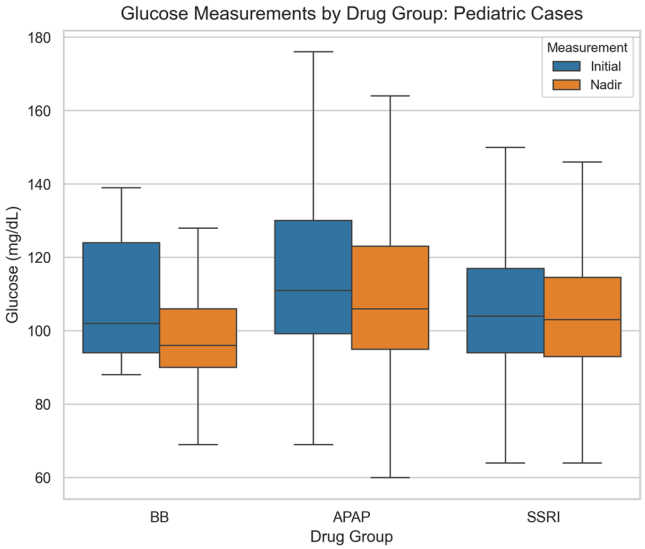

Fig. 4 Distributions of glucose measurements in the pediatric subgroup both initially and lowest measured

The association between beta blocker use and hypoglycemia is predicated on studies in pediatric patients chronically taking therapeutic doses of beta blockers. Holland et al. describes three pediatric patients aged 5 weeks to 18 months started on propranolol hydrochloride for treatment of infantile hemangioma [10]. All three developed hypoglycemia with nadir glucose ranging from 19 mg/dL to 55 mg/dL. All three were on therapeutic doses of propranolol and had no other features of beta blocker toxicity. Multiple additional reports and series describe hypoglycemia in this patient population as well as those on long-term beta blockers for management of congenital long QT syndrome, lending credence to this association [6, 7]. This is also supported by a pharmacovigilance study by Carnovale et al. in 2021 which found an overall rate of hypoglycemia of 1.75% of all patients on therapeutic beta blocker therapy with increased risk in the pediatric subgroup [11]. The proposed mechanism for beta blocker induced hypoglycemic episodes is interference with gluconeogenesis and glycogenolysis [8]. It follows that a young pediatric population with limited glycogen stores available would be the most susceptible to these metabolic effects of beta blockers.

It is less clear how this paradigm applies to patients acutely experiencing beta blocker toxicity. The median age of patients in the beta blocker arm of this cohort was 55 years, and only six patients were under five years of age. From a national perspective, in 2023, pediatric patients less than five years accounted for 21% of all single substance beta blocker calls [1]. Love et al. performed a prospective study on this high-risk age group of six months to six years old exposed to beta blockers [12]. They found that there were no instances of hypoglycemia as well as no instances of shock, arrythmia, or symptomatic bradycardia. This suggests that very few patients with beta blocker toxicity have a baseline physiology that is particularly susceptible to developing hypoglycemia while exposed to a beta blocker.

We identified two studies which specifically evaluated association of beta blocker toxicity and hypoglycemia. Lionte et al. looked at both a retrospective population and a prospective population of acutely poisoned patients [13]. In their retrospective cohort, 912 cases of acute beta blocker poisoning were identified, with only 1% of these cases found to have hypoglycemia. These results generally agree with what we found in our study. In the prospective portion of their study, 122 acutely beta blocker poisoned patients were identified, and 27 (11.63%) patients were noted to have a hypoglycemic measurement in the emergency department. However, in the prospective portion of their study, there is variation in the timing of blood glucose measurements, a unique method of quantifying hypoglycemia using a glucose tolerance test, and a lack of details regarding patients' toxicity symptoms or management, making direct comparison of their prospective results to ours not possible.

Gokalp et al. looked at rates of hypoglycemia in a pediatric cohort of acutely beta blocker poisoned patients [14]. This is a retrospective case-control study that compared acute beta blocker poisoned patients to acute SSRI poisoned

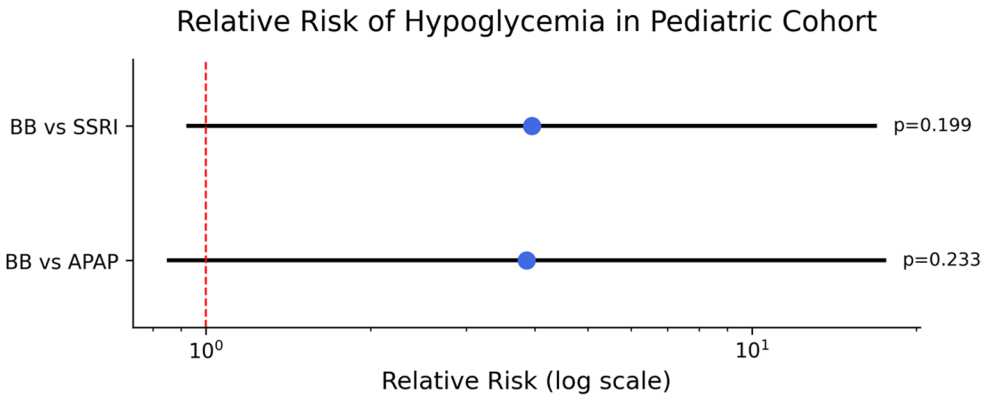

Fig. 5 Relative risk of hypoglycemia at any time between the beta blocker group and the two control groups in the pediatric subgroup

patients presenting to a pediatric emergency department. This study had 40 beta blocker cases and 40 SSRI cases, and they defined hypoglycemia at glucose < 50 mg/dL. They found that there was a statistically significant difference in mean blood glucose concentrations in the beta blocker group at one hour and 24 h post presentation. However, they were not in the hypoglycemic range, with the means being approximately 86 mg/dL at both times in the beta blocker group. In their beta blocker group, there were 11 cases of hypoglycemia, and in their SSRI group, there were 6 cases of hypoglycemia. They report no statistical significance in rates of hypoglycemia at any time point between the beta blocker group or the SSRI group. For the purpose of comparison to our study, we computed the relative risk of hypoglycemia in their beta blocker group compared to their SSRI group using the data provided in their study. The relative risk of hypoglycemia at any time was 1.83 (95% CI 0.75–4.48, $p = 0.274$), which is consistent with the results of our study. Our study strengthens these findings by using a stricter definition of beta blocker toxic patients and examining a larger cohort.

These findings have important clinical implications. First, we propose that routine serial blood glucose monitoring is not indicated for patients with beta blocker toxicity in the absence of symptoms which may suggest the presence of hypoglycemia, such as acute neurological change. Second, we argue that blood glucose cannot be used as a marker to risk stratify severity of beta blocker toxicity. Hyperglycemia is a well-documented clinical manifestation in calcium channel blocker toxicity, and blood glucose level has been shown to directly correlate with severity of calcium channel blocker toxicity [15]. Blood glucose can therefore be used to help differentiate between calcium channel blocker toxicity and beta blocker toxicity, which otherwise have considerable overlap in clinical manifestations. Based on our findings, blood glucose is relatively unperturbed in beta blocker toxicity and would therefore not hold similar predictive value.

There are several limitations of this study. The retrospective nature of this study leaves open the potential for unaccounted confounders, including but not limited to unreported co-ingestants. Poison Center data is passively collected and relies on voluntary reporting, and the patient population included in our cohort may therefore differ from the general population in unforeseen ways. Given the overall median of only one recorded blood glucose value across groups, there are likely many unrecorded glucose measurements due to the nature of using a poison center medical record rather than the primary medical record. In many cases, there is documentation of normal glucose without a value associated as well as not all lab draws or point of care testing getting transcribed into poison center record. However, we suspect that these limitations would be similarly shared across all groups. As we are unable to assess baseline (pre-ingestion) vital signs, we cannot assess the magnitude of heart rate change in the beta blocker patients. It is therefore conceivable that the use of bradycardia as inclusion may have resulted in inadvertent inclusion of patients not experiencing acute toxicity but were instead on long-term and stable pharmacotherapy. Additionally, quantification of serum concentrations to further characterize beta blocker exposure or toxicity was not feasible. Importantly, clinical manifestations were consistent with expected developments from beta blocker toxicity, and the total cohort frequently demonstrated critical illness with substantial minorities receiving some combination of vasopressors, insulin, and/or atropine. In our subgroup analysis, in an effort to exclude possible cofounders, we excluded all patients who were documented to have received insulin. Unfortunately, it is not possible in our records to accurately identify timing of insulin administration in relation to timing of blood glucose measurement, which led to exclusion of all insulin cases in this subgroup. This includes any patient receiving high-dose insulin therapy for beta blocker toxicity, thus potentially excluding the sickest cohort from this subgroup analysis. Finally, as described above, our study has relatively few pediatric patients, with only six patients under five years of age, which is the cohort thought to be at highest risk for acute hypoglycemia. Given very limited number of these high-risk patients, it is possible that this population remains at risk for acute hypoglycemia that could have been missed in our study. While our study addresses a population that is more likely to experience beta blocker intoxication, it would be of interest for future studies to focus on this higher risk subset of patients.

Conclusions

In our cohort of patients, there was no significantly increased risk of hypoglycemia in patients with beta blocker toxicity as compared to APAP and SSRI ingestion cases. This contradicts the commonly taught notion that hypoglycemia would be an expected finding in acute beta blocker toxicity. As such, frequent blood glucose measurements in beta blocker poisoned patients are likely unnecessary in patients who are not exhibiting symptoms of hypoglycemia.

Funding There are no funding sources for this study.

Declarations

Conflict of interest The authors do not have any conflict of interest.

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