Massive Propranolol Ingestion Successfully Treated with Very High Dose Insulin Euglycemic Therapy (18u/kg/hr)

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Background

Propranolol is a non-specific beta blocker that is highly lipophilic, which allows it to cross the blood brain barrier. Although still used to treat hypertension and to prevent acute cardiovascular events, it is often used for the treatment of situational anxiety. The lowest reported fatal dose of propranolol is 800mg.

Case Presentation

A 14-year-old female presented to the ED 30 minutes after ingesting 910 mg propranolol, 630 mg fluoxetine, and 8400 mg of quetiapine. Her mental status and vital signs were normal upon ED arrival. After 20 minutes in the ED, she became acutely obtunded and was intubated. Gastric lavage was performed, with return of pill fragments. A nasogastric tube was placed, 100 gm of activated charcoal was administered, and whole bowel irrigation was initiated. CBC and CMP were unremarkable. APAP/ASA/EtOH were non-detectable. Vital signs after intubation were HR 89, BP 89/59, RR 18, and O2 Sat 100%. After transfer to a quaternary care center PICU, vital signs were: HR 82, BP 87/53 mm, RR 16 (mechanically ventilated), normothermic, and O2 Sat 100%.

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High Dose Insulin Euglycemic Therapy May have a continued Dose Response Beyond 10 units/kg/hr

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Case continued

Whole bowel irrigation was stopped on account of absent bowel sounds and abdominal distention. She was hypotensive with a systolic BP in the 80's and an epinephrine infusion was started at 0.1 mcg/kg/min with good response. High dose insulin euglycemic therapy (HIET) was initiated with the goal to stave off cardiovascular collapse and wean pressor requirements. HIET dosing peaked on PICU day 5 at 18 units/kg/hr, with a 50% reduction in vasopressor requirement. HIET and vasopressors were discontinued on PICU day 6. She was extubated neurologically intact, and ultimately admitted to psychiatry. Her peak propranolol level was 803 ng/mL.

Case discussion

We attribute the duration and intensity of our patient's propranolol toxicity to prolonged GI absorption, either from a pharmacobezoar or ileus. Oddly, her 12-hour serum propranolol level of 803 ng/mL is lower than the 2000 ng/mL level previously reported as a threshold for "probable toxicity." It is possible that her fluoxetine or quetiapine co-ingestions exacerbated the propranolol toxicity, or more likely, that 803 ng/ml does not reflect her true peak level. Although never severely bradycardic, her blood pressure was dependent upon HIET throughout her PICU stay, and would exhibit precipitous decline with increased vasopressor requirement whenever HIET weaning was attempted. Likewise, she exhibited almost no vasopressor requirement at HIET 18 u/kg/hr.

Conclusion

Propranolol can exert prolonged toxicity in large overdoses, and HIET is an effective antidote for propranolol toxicity, although higher than usual HIET dosing regimens may be required to manage severe poisonings.