Cardiac Arrest in a 10-year-old Following Subcutaneous Epinephrine Injection Prior to Excision of a Neck Mass

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Abstract

We present the case of a ten year old male who developed pulmonary edema and cardiac arrest after an inadvertent subcutaneous injection of 100 times the intended dose of Epinephrine, prior to an attempted neck mass excision. The purpose of this presentation is to review the effects and treatment of a toxic dose of Epinephrine. Adrenergic receptors mediate Epinephrine’s effects. Many publications have described instances of cardiovascular collapse and pulmonary edema due to Epinephrine overdose. Treatment is aimed at afterload reduction and maintaining cardiac function.

Following a multidisciplinary Root Cause Analysis we eliminated 30 ml vials of 1:1000 Epinephrine in our perioperative setting.

Case Presentation

A healthy 10 year old male with right neck mass (9mm x 13mm). PMH: pleomorphic adenoma S/P uneventful excision two years prior. Allergies: Amoxicillin

Intraoperative Course

Case proceeded with uneventful induction and intubation. Ancef given per surgeon request at 10:30am. 10 ml of 1:100,000 Epinephrine was injected by the surgeon at 10:35. Dramatic elevation of heart rate and blood pressure, decreased SaO2 (to 94%) and pulmonary edema followed. The case was cancelled. At 10:55 although the EKG revealed sinus rhythm no pulses were palpable (PEA) and CPR was started. CPR was continued for 20 minutes at which point spontaneous circulation returned. Patient was transferred to PICU.

24 hours later the echocardiogram showed normal left ventricular systolic function. The patient was successfully extubated on POD 1. He remained hemodynamically stable. Patient was discharged home on postoperative day 4.

Discussion

Alpha 1 stimulation increases peripheral vascular resistance. Beta 1 stimulation produces increased heart rate, cardiac output myocardial oxygen consumption. Epinephrine, though, is rapidly metabolized and has a short half-life (2 minutes).

Notwithstanding this, life threatening hypertension results from these combined adrenergic receptor effects. The precipitously increased afterload can cause left ventricular failure and pulmonary edema. Prolonged cardiovascular toxicity has been seen in both adult and pediatric populations. Reversible cardiomyopathy, with return of the heart to baseline function 4 to 8 days after Epinephrine over-dose has been described. Some publications also describe a hypersensitivity to Epinephrine when appropriate doses were administered to the patient.

Immediate treatment with an alpha blocker such as Phentolamine is ideal. A short acting beta blocker (Esmolol) might be of use after alpha blockade. Beta blockade without previous alpha blockade results in unopposed alpha adrenergic agonism and severe hypertension leading to cerebral hemorrhage, pulmonary edema, myocardial infarction, ventricular fibrillation and death. Labelol or Calcium Channel Blockers might actually cause cardiac arrest, pulmonary edema and death, by depressing a myocardium struggling to pump blood against a severely increased peripheral resistance. If a beta blocker is administered, the cardiac depressive effect can be reversed with glucagon. We recommend anesthesiologists readily have alpha blocking agents available for surgeries requiring Epinephrine infiltration.

References

1. Anesthesia-related Cardiac Arrest in Children: Initial Findings of the POCA Registry.

Learning Points

- The vasopressor effects of Epinephrine overdose may be counteracted by fast-acting alpha blockers such as Phentolamine, followed by a beta blocking agent such as Esmolol.
- Do not give beta blockers without previous alpha blockade! This results in unopposed alpha adrenergic agonism, severe hypertension, cerebral hemorrhage, pulmonary edema, myocardial infarction, ventricular fibrillation and death.
- Some advocate Labelol or Calcium Channel Blockers to treat hypertension, others feel it causes cardiac arrest, pulmonary edema and death, by depressing a myocardium struggling against increased SVR.

Hospital course

Three hours after cardiac arrest the echocardiogram showed moderately to severe decreased left ventricular systolic function.