PLEASE UNBLOCK MY HEART!!!

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INTRODUCTION

Drug overdose appears to be a challenging dilemma to emergency residents. Over ingestion of cardiotoxic agents can only be revealed by focused history and examination. Hence treatment is tailored according to the pharmacological effects of the cardiotoxic drug ingested. Bedside focused cardiac ultrasound may reveal findings of right ventricular dysfunction.

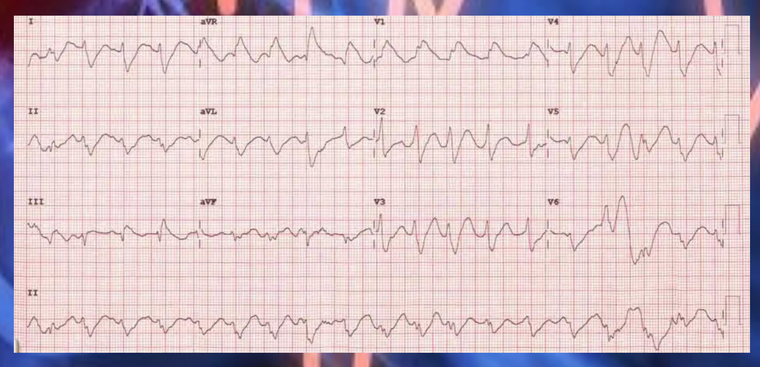
Hereby we describe a case of beta blocker toxicity which posed right ventricular dysfunction and the reasons resulting to it.

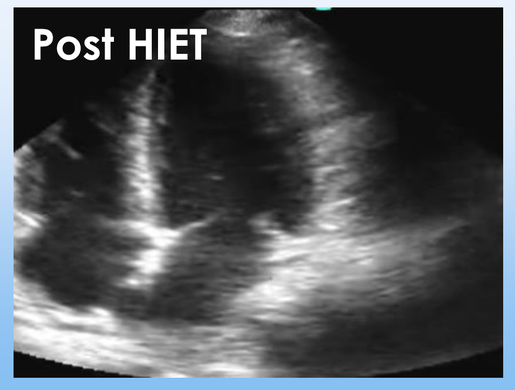
CASE REPORT

A 29 years old lady with underlying history of hyperthyroidism had been brought to our emergency department after ingested 80 tablets of propranolol. Patient was brought to a private medical center, intravenous infusion of noradrenaline was started in view of haemodynamic instability then was transferred to us for continuation of care. We received the patient 1.5 hours post ingestion. Our assessment on arrival, patient appeared drowsy, lethargy and unresponsive to call. BP and HR was normal on IVI Noradrenaline. GCS was E2V2M5 (9/15) with pupils of 2mm/2mm equal and reactive. Capillary blood glucose was 6. We had intubated the patient for airway protection. Electrocardiography had no evidence of heart blocks, but had prolong QT interval and broad QRS complexes. Bedside ECHO revealed moderate contractility, apical wall hypokinesia, no pericardial effusion, dilated right atrium and ventricle with thickened right ventricle, moderate tricuspid regurgitation, right ventricular systolic dysfunction and pulmonary artery hypertension. High Insulin Euglycemic Therapy (HIET) was adhered in the resuscitation of this patient. Subsequently we had referred to the ICU team for continuation of care. After the patient was out of the acute phase, the right ventricle had returned to the normal non dilated state.

DISCUSSION

Propranolol, a first generation non selective beta-1 and beta-2 receptors that pose negative inotropic, chronotropy and dromotropy. In acute toxicity, propranolol inhibits beta-2 receptors on pulmonary vasculature results in increased pulmonary vascular resistance and right ventricular pressure. Sustained elevation of pulmonary vascular resistance results in increased RV pressure overload, causing ventricular interdependence. Hence leading to hypotensive state. This is depicted on our bedside echocardiography assessment. Our patient had a transient right ventricular dysfunction and pulmonary hypertension which had reversed to normal after high dose insulin which is a potent anabolic hormone. Besides improving cardiac contractility, insulin attenuates pulmonary hypertension by inhibiting endothelin-1 which is a potent vasoconstrictor.





CONCLUSION

HIET remains as a paramount saviour in beta blocker toxicity due to its attributes of not only improving cardiac contractility but also relieving high pulmonary vascular resistance.

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