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LONG-TERM USE OF HIGH-DOSE LOPERAMIDE LEADING TO CARDIAC ARREST AND VENTRICULAR ARRHYTHMIAS

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Learning Objectives: Loperamide, an over-the-counter antidiarrheal, is considered safe at labeled doses. Since 2014 numerous case reports on loperamide abuse and overdose have been reported, many resulting in death. In June 2016, the FDA issued a statement regarding the risk for severe cardiac toxicity with high doses of loperamide. We describe a patient who experienced cardiac arrest twice and multiple episodes of ventricular arrhythmias associated with loperamide.

Methods: A 46-year-old female was admitted to the medical intensive care unit following witnessed cardiac arrest at home due to overdose of unknown origin and QTc prolongation. Toxicology screen was positive for oxycodone. Within hours of admission, the patient developed torsades de pointes (TdP), a second brief cardiac arrest with compressions and ROSC, and received magnesium sulfate 14 grams total during the first 12 hr of admission. Ventricular arrhythmias recurred, and isoproterenol was initiated and continued for 24 hr. On hospital day (HD) 2, EKG was concerning for acute STEMI, and patient was taken to catheterization lab where no coronary abnormalities were identified. On HD 3, the patient's significant other reported that for the past year he purchased two-thousand loperamide 2-mg tablets every 2 weeks for the patient to alleviate opioid withdrawal symptoms. On HD 3, the patient transferred to the floor. Upon interview, she reported learning about loperamide for opioid withdrawal symptoms from an internet search. She titrated up to taking loperamide 30 to 60 tablets at a time at least twice a day and oxycodone when available from an illicit source. On HD 9, the patient had 12-beat run of TdP that spontaneously resolved. No QT prolonging medications were identified, adding concern for an underlying congenital arrhythmia disorder. On HD 15, an implant dual chamber (ICD) was placed, and the patient discharged to a skilled nursing facility on HD 16.

Results: This case report adds to the growing body of literature regarding loperamide misuse. It also highlights the importance of a thorough medication history for over-the-counter products and raises awareness of an unexpected drug of abuse.

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EJECTION FRACTION IS A POOR INDICATOR OF VENTRICULAR CONTRACTILITY IN ACUTE HEART FAILURE

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Learning Objectives: Peripheral veno-arterial extracorporeal membrane oxygenation (VA ECMO) is now the first line device for refractory acute cardiogenic shock. Precise evaluation of left ventricular (LV) function in patients under assistance is crucial, in particular during weaning, and is based to a large degree on LV ejection fraction (LVEF). However, the use of this load dependent parameter is questionable. Indeed, LVEF does not reflect intrinsic LV contractility but is the result of interaction between preload, contractile function and afterload.

Methods: We report the case of a 54 y/o man who presented a cardiogenic shock following coronary artery by-pass. He was supported with femoro-femoral VA ECMO as a bridge to recovery (or transplantation). After 3 days of circulatory support, since we observed a normalization of blood lactate, an improvement in hemodynamics, and a normalization of liver and renal functions, the question of VA ECMO weaning was raised. VA ECMO was maintained because of LVEF less than 20%, according to recommendations. LV function assessed with LVEF was compared to LV elastance (Ees) derived from pressure-volume (PV) loops (i.e., LV intrinsic contractility). LV systolic pressure was derived from femoral arterial line while LV volume was derived from transthoracic echocardiography. VA ECMO pump speed was changed to induce small preload variations, allowing to calculate precisely Ees. The afterload imposed to the left ventricle was assessed with the arterial elastance, Ea (mm Hg/ml), characterizing arterial properties including wall stiffness, compliance, outflow resistance and wave's reflections. The ratio of Ees to Ea was defined as the LV-arterial coupling. Recordings were performed every day. Between day 0 and day 3, LVEF decreased from 17 to 14%, while Ees increased from 0.51 to 1.09 mm Hg/sec. This decrease in LVEF was explained by Ea increase from 2.63 to 4.45 mm Hg/sec.

Results: Changes in loading conditions may mask changes in LV contractility, when assessed with load dependent parameters like LVEF. Discrepancy between LVEF and LV intrinsic contractility is explained by LV–arterial coupling. The lower the LV contractility, the more important the influence of loading conditions on LVEF. Using only load dependent parameters like LVEF may lead to improper evaluation of LV contractility in acute heart failure, and, in particular, in VA ECMO decisions.

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