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1263

ROCURONIUM-INDUCED ANAPHYLAXIS: ANY EVIDENCE FOR SUGAMMADEX?Mohamed Fayed¹, Yousif Makadsi², Fadi Jirjees², Warren Elmer², Asa Gray-buchta² and Donald Penning³¹Henry Ford Hospital, Grosse Pointe Woods, MI, ²Henry Ford Health System, ³Henry Ford Hospital

INTRODUCTION: Anaphylaxis during general anesthesia is estimated to occur around 1 in 3,500 cases. 90% of these cases occur at induction of anesthesia. Neuromuscular blocking drugs are thought to be the most responsible trigger, with Rocuronium being the most implicated drug. Sugammadex is a synthetic modified gamma-cyclodextrin derivative that will incompletely encapsulate Rocuronium, preventing the rocuronium epitope from binding IgE; hence it was thought that its use could decrease anaphylaxis severity. The use of sugammadex in suspected rocuronium-induced anaphylaxis (RIA) is based on personal opinion or experience. Current literature and evidence regarding the use of sugammadex in RIA are based only on case reports or series.

METHODS: We did a literature search in 3 main databases, Medline, Embase, and Web of science. The final total articles were 356 published cases published in the literature.

Two independent reviewers conducted the first screen, and 69 articles with confirmed RIA were selected. These articles were divided into RIA alone in 39 cases and RIA with sugammadex use in 28 cases. We looked at patient characteristics (age, sex, ASA classification), associated symptoms (incidence of bronchospasm or rash, duration of hypotension), discharge disposition to intensive care unit (ICU), and associated morbidity or mortality.

RESULTS: On comparing RIA vs. RIA with sugammadex use, we didn't find a statistically significant difference in patient characteristics, associated symptoms, or discharge disposition to ICU. However, there was a statistically significant difference in morbidity (15.4% vs. 0, p-value 0.03). Mortality was 5% in RIA, and no patient died in RIA with sugammadex use (p-value 0.5). The number need to treat (NNT) analysis showed NNT with Sugammadex to prevent mortality = 20 and NNT with Sugammadex to avoid morbidity is 7.

CONCLUSIONS: Early recognition and treatment of anaphylaxis with Epinephrine is still the mainstay of treatment. Since muscle relaxants, specifically Rocuronium, are the most common cause, we suggest using sugammadex in case of suspected RIA. We recommend using sugammadex in case of cardiac arrest or refractory anaphylaxis as evidence of NNT to avoid organ dysfunction is 7 and potential evidence of mortality benefit. The recommended dose is 16 mg/kg.

1264

EXTRACORPOREAL MEMBRANE OXYGENATION IN MASSIVE PULMONARY EMBOLISM: THE CHALLENGES OF DIAGNOSIS OF PESima Patel¹, Vishal Singh², Jose Maria-Rios³ and Maria carrillo⁴¹Kendall Regional Medical Center, Orlando, FL, ²AdventHealth Orlando, ³AdventHealth East Orlando, ⁴AdventHealth

INTRODUCTION: Pulmonary embolism (PE) is the third most common cause of death after myocardial infarction & stroke. Diagnosis is challenging without a computed tomographic pulmonary angiography (CTPA), & advanced management techniques are difficult to implement without confirmation. We present a patient who was unable to undergo a CTPA, but through a multidisciplinary approach was progressed to veno-arterial extracorporeal membrane oxygenation (VA ECMO).

DESCRIPTION: A 245-kg 30-year-old male was admitted for Ludwig's angina, fiber-optically intubated, taken for incision & drainage, & later successfully extubated. Two days later, he became increasingly hypoxic, leading to reintubation and initiation of a heparin infusion for suspected massive PE. Point of care ultrasound was non-diagnostic due to poor acoustic windows & CTPA unobtainable due to the patient's body habitus. He progressed to obstructive shock & refractory hypoxia. Lower extremity (LE) dopplers were positive for extensive deep venous thrombosis. A multidisciplinary team including critical care, interventional cardiology & cardiothoracic surgery, opted to proceed to the catheterization lab for a transesophageal echocardiogram (TEE) & pulmonary angiography for diagnosis & attempted thrombectomy with VA ECMO as backup. TEE showed a severely dilated right ventricle (RV) with strain. Angiography confirmed extensive PE, but catheter advancement induced ventricular fibrillation & thrombectomy was aborted. Patient was then cannulated for VA ECMO.

DISCUSSION: CTPA is the first line diagnostic technique in patients with PE. Unfortunately, our patient's body habitus limited the ability for this modality. Catheter pulmonary angiography has been considered too invasive solely for diagnosis but has been favored over CTPA when endovascular intervention is intended. Angiography has its limitations, including ease of availability, risk of hemodynamic compromise in severe RV failure & interpretation of imaging. Although this is a concern, it should not deter from making a diagnosis given the case-fatality of an undiagnosed PE. We aim to recognize the challenges in PE diagnosis and to reiterate the importance of a multidisciplinary approach in massive PE to broaden therapeutic options and improve mortality.