The Effect of Hypertriglyceridemia on Extracorporeal Membrane Oxygenation

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INTRODUCTION: Extracorporeal membrane oxygenation (ECMO) is commonly used in potentially reversible severe cardiac or pulmonary failure. The use of ECMO is shown to improve survival and recovery in severe acute respiratory distress syndrome (ARDS). Survival rates are reported to be up to 70% in patients requiring ECMO. We present a case of suboptimal oxygenation with ECMO in a patient with severe ARDS secondary to bilateral cytomegalovirus pneumonia.

CASE PRESENTATION: A 22-year-old male with a history of intravenous drug and tobacco abuse presented to the emergency department with a 4 day history of shortness of breath. Initial evaluation was significant for SpO2 of 60% on room air, white blood cell count 6.4 \(10^3/uL\), hemoglobin 15.2 g/dL, sodium level 129 mmol/L, creatinine 3.1 mg/dL, triglyceride level 1277 mg/dL. Arterial blood gas on admission showed pH 7.33, pO2 41 mmHg, pCO2 33 mmHg, HCO3 16 mEq/L. Chest x-ray was consistent with bilateral pneumonia. The patient was put on azithromycin, vancomycin and piperacillin/tazobactam. His blood pressure was maintained with norepinephrine infusion. Blood and sputum cultures, influenza screen, HIV screen, and legionella antigen were negative. Serology for cytomegalovirus was positive. The patient was intubated and mechanically ventilated. The oxygen saturation was low despite high-pressure ventilation and venovenous ECMO was initiated. Post-oxygenation arterial blood gas showed pO2 200-400mmHg while on 100% FdO2 and the pre-oxygenation blood gas sample was grossly lipemic. Insulin infusion was administered. Repeat triglyceride levels was 434 mg/dL. Repeat chest x-ray showed complete bilateral white-out. The patient was made comfort care and expired rapidly after the ECMO circuit was discontinued.

DISCUSSION: There is evidence that ECMO can sequester circulating blood compounds like essential amino acids and lipid soluble vitamins. Ex vivo models showed no significant reduction in triglyceride levels. However, in this case there was a reduction in the triglyceride levels from 1277 mg/dL to 434 mg/dL, which suggests possible sequestration of triglycerides in the circuit oxygenator. This was also associated with suboptimal oxygenation in the post-oxygenation blood gas. Attempts to reduce the lipid levels with insulin infusion did not improve oxygenation. The mechanism of the suboptimal oxygenation is not entirely clear, however, severe hypertriglyceridemia seems to cause partial oxygenation on ECMO.

CONCLUSIONS: Suboptimal oxygenation on ECMO can be caused by severe hypertriglyceridemia.


DISCLOSURE: The following authors have nothing to disclose: Stamatis Baronos, Lauren Krowl, Zabeer Bhatti, Pardeep Masuta, David Landsberg

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