TITLE:

Aortic Cannula Clot: Devastating Thrombotic Complication on ECMO

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INTRODUCTION:

Management of anticoagulation for ventricular assist devices (VADs) and extracorporeal membrane oxygenation (ECMO) continues to be challenging, with devastating complications described from both hemorrhagic and thrombotic events. Thrombotic events can be related to patient, pump or management factors and thrombus typically occurs at areas where stasis is common. Here we describe a case of thrombosis that occurred unexpectedly at a point of high flow in a patient on therapeutic anticoagulation.

CASE:

A 46 year-old male presented with scapular pain, dizziness and transient blindness to an emergency department where he was diagnosed with an anterior ST-elevation myocardial infarction complicated by complete heart block. He was transferred to our center for emergent cardiac catheterization which revealed total occlusion of the left anterior descending (LAD) artery and right coronary artery (RCA) amongst other lesions and he underwent emergent five-vessel coronary artery bypass grafting (CABG). When weaning off cardiopulmonary bypass, severely depressed left-ventricular function was noted with an inability to maintain an acceptable hemodynamic profile despite maximal inotropic support. At that time, right ventricular systolic function appeared preserved. A Centrimag LVAD was implanted as a bridge to recovery with inflow cannula placed in the apex of the left ventricle and outflow cannula positioned in the aortic arch with flows averaging 3.5 liters/minute. Epinephrine, milrinone, vasopressin, norepinephrine infusions and inhaled epoprostenol were continued. Vasoactive infusions were steadily weaned and an unfractionated heparin drip was started on postoperative day 2. The patient became increasingly hypoxic and oxygenation was supported by optimizing sedation, neuromuscular blockade, positive end-expiratory pressure titration with esophageal manometry and inhaled epoprostenol; however, he continued to deteriorate with a nadir P:F ratio of 100 on postoperative day 3 and an oxygenator was spliced into the LVAD circuit with subsequent improvement in arterial oxygenation. A computerized tomography angiography (CTA) of the chest revealed a left pulmonary artery embolism; of note, simultaneous laboratory testing for antiPF4/heparin antibodies was negative. Unfractionated heparin was continued per our ECMO protocol with a target activated clotting time (ACT) of 180-220 seconds and a goal antiXa assay level of 0.5-0.7 IU/mL. Oxygenation and hemodynamics continued to improve with oxygenation transitioned entirely to the ventilator and routine transesophageal echocardiography (TEE) was unremarkable. On postoperative day 6, however, sudden drops in LVAD flows unresponsive to fluid bolus were noted and subsequently dropped to zero within minutes; fortunately intrinsic LV pulsatile function was sufficient to maintain systemic perfusion. Exploration in the operating room revealed a large clot at the end of the aortic cannula, subsequent wean off cardiopulmonary bypass was uneventful with no further mechanical support deemed necessary. Notably, cannula position appeared optimal and no clot was noted in oxygenator or other parts of the circuit. Further testing revealed presence of antiPF4/heparin antibodies and positive serotonin-release assay testing consistent with heparin induced thrombocytopenia (HIT) despite stable platelet count.

DISCUSSION:

Given therapeutic anticoagulation, appropriate cannula position and unremarkable diagnostic studies, cannula thrombosis at a site of high flow was an unexpected finding in this case. Preexisting pulmonary embolus may be suggestive of underlying hypercoagulability and HIT likely contributed.

REFERENCES:

Birati EY, Rame JE. Diagnosis and Management of LVAD Thrombosis. Curr Treat Options Cardiovasc Med. 2015 Feb;17(2):361.

Blitz A. Pump thrombosis-A riddle wrapped in a mystery inside an enigma. Ann Cardiothorac Surg. 2014 Sep;3(5):450-71

Evans CF, Li T, Mishra V, Pratt DL, Mohammed IS, Kon ZN, Griffith BP. Externally visible thrombus partially predicts internal thrombus deposition in extracorporeal membrane oxygenators. Perfusion. 2016 Nov 22.

Hastings SM, Deshpande SR, Wagoner S, Maher K, Ku DN. Thrombosis in centrifugal pumps: location and composition in clinical and in vitro circuits. Int J Artif Organs. 2016 Jun 15;39(4):200-4.

Pollak U, Yacobobich J, Tamary H, Dagan O, Manor-Shulman O. Heparin-induced thrombocytopenia and extracorporeal membrane oxygenation: a case report and review of the literature. J Extra Corpor Technol. 2011 Mar;43(1):5-12.

Tchantchaleishvili V, Sagebin F, Ross RE, Hallinan W, Schwarz KQ, Massey HT. Evaluation and treatment of pump thrombosis and hemolysis. Ann Cardiothorac Surg. 2014 Sep;3(5):4905.

Williams B, Bernstein W. Review of Venoarterial Extracorporeal Membrane Oxygenation and Development of Intracardiac Thrombosis in Adult Cardiothoracic Patients. J Extra Corpor Technol. 2016 Dec;48(4):162-167.