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Successful Application of Veno-Venoarterial Extracorporeal Membrane Oxygenation for Acute Exacerbation of Asthma Followed by Stress Cardiomyopathy

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We report a case treated by veno-venoarterial extracorporeal membrane oxygenation (vva-ECMO) for stress cardiomyopathy due to exacerbation of asthma.

A 45-year-old woman with a history of asthma was transferred to our emergency department from a local medical center. She had developed severe dyspnea and wheezing an hour before. At the time of arrival at our hospital, she was already intubated and had been administered systemic corticosteroid. The initial blood pressure (BP) was 140/90 mm Hg, and heart rate was 130 beats per minute. Her arterial blood gas analysis (ABGA) showed PaO_2 18 mm Hg, $PaCO_2$ 88 mm Hg, and SaO_2 9%. She initiated on ventilator care with FiO_2 1.0. Initial transthoracic echocardiography (TTE) showed that left ventricular ejection fraction (LVEF) was 45%. Her electrocardiogram (ECG) was sinus tachycardia without ST-segment abnormalities, and troponin I was 0.11 ng/mL. Computed tomography revealed diffuse tracheobronchial narrowing in both lung fields, which was compatible with acute exacerbation of chronic asthma. Despite 12 hours of ventilator care, hypoxia and hypercapnia developed (PaO_2/FiO_2 73 mm Hg/0.5, $PaCO_2$ 118 mm Hg). Our ECMO team was contacted and decided to apply veno-venous extracorporeal membrane oxygenation (vv-ECMO) via bilateral femoral veins: RotaFlow centrifugal pump (Maquet cardiopulmonary AG, Hirrlingen, Germany), a 20 Fr femoral venous cannula (Fem-Flex II, Edwards Lifescience, LLC, Irvine, CA, USA), a 21 Fr femoral venous cannula (DLP, Medtronic Inc., Minneapolis, MN, USA). ECMO was initiated and gas exchange improved: PaO_2 172 mm Hg on ventilator FiO_2 0.4, $PaCO_2$ 43 mm Hg, peripheral SaO_2 99%.

Thirty minutes after the start of ECMO, arterial BP dropped below 40/20 mm Hg. Despite the high dose of catecholamines, systolic BP was below 60 mm Hg. TTE showed LVEF of 15% and global wall motion abnormality of LV. ECG presented nonspecific T-wave inversion and troponin I was slightly increased (2.18 ng/mL). We postulated that she developed stress cardiomyopathy due to exacerbation of asthma, because she had no clinical history suggestive of acute coronary syndrome. We converted vv-ECMO to vva-ECMO by insertion of an arterial cannula via the left femoral artery: a 16 Fr femoral arterial cannula (Fem-Flex II, Edwards Lifescience), connected to ECMO via the Y-connector.

After 15 hours of vva-ECMO support, follow-up TTE showed LVEF of 35% and mid-ventricular akinesia and apical hypokinesia (Fig. 1). We reverted to vv-ECMO on the 3rd day. On the 4th day, the ECMO was weaned off without significant complications. On the 7th day, she was extubated, and transferred to a general ward the following day. She underwent rehabilitation for 2 weeks, and was subsequently discharged from the hospital.

Asthma has a high prevalence world-wide, and can be fatal in some cases. Asthma with acute exacerbation often leads to respiratory failure requiring mechanical ventilation.[1] vv-ECMO is helpful when a patient has persistent hypoxemia and

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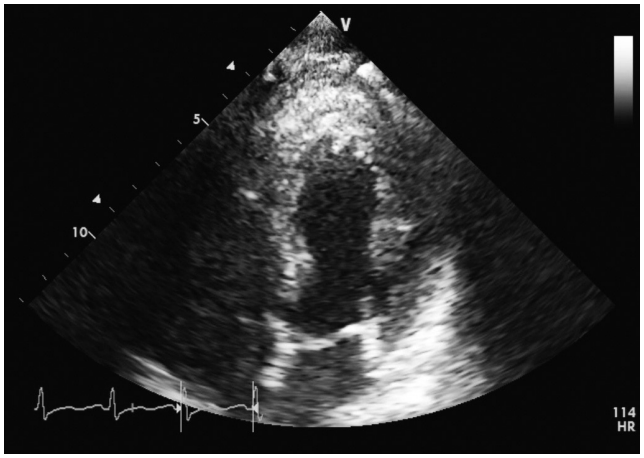

Fig. 1. On the 2nd day ECMO insertion, transthoracic echocardiography (TTE) shows mid-ventricular akinesia and apical hypokinesia.

hypercapnia despite maximal ventilator support.[2] We initially provided our patient with vv-ECMO for respiratory support. After a while, her cardiac function deteriorated rapidly with low BP, low LVEF, global wall motion abnormality of LV, nonspecific T-wave inversion on ECG, and increased troponin I. We considered that she developed stress cardiomyopathy due to severe respiratory failure. Coronary angiography to exclude obstructive coronary disease was not performed, since the patient had no implicating history. She was in cardiogenic shock refractory to fluid and high dose inotropic drugs.

The studies on vva-ECMO are limited. A small-cohort study showed that vva-ECMO appears to further improve survival in the acute respiratory distress syndrome.[3] Because vva-ECMO provides well-oxygenated blood to the systemic and pulmonary circulation, it helps reduce the pulmonary resistance and the existence of intrapulmonary-shunts in the pulmonary circulation. It also supplies sufficient oxygen to the coronary and peripheral organs.

vva-ECMO is beneficial for supporting both cardiac function and respiratory function in acute respiratory failure combined with cardiac failure, as in the current case.

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