Azd-1222

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Fulminant myocarditis: case report

A 63-year-old woman developed fulminant myocarditis following administration of Azd-1222 for COVID-19 vaccination [*route and dosage not stated*].

The woman received the first dose of Azd-1222 [ChAdOx1 vaccine] on 19 June 2021. Following the first dose, she did not experience any side effects. On 04 September 2021, she received the second dose of Azd-1222. After 3h of the second dose, she developed chest pain as an initial symptom. After 5 days, she visited the emergency department of a tertiary hospital for aggravated chest pain, dizziness, nausea, vomiting, diarrhoea and fever which continued for four consecutive days. Echocardiography revealed normal cardiac chamber dimensions with a mildly thick left ventricular (LV) wall with LV ejection fraction of 15%. Doppler imaging showed insufficient transaortic blood flow. A small amount of pericardial effusion was also observed. Creatine kinase myocardial band (CK-MB), high-sensitive troponin I (hsTnI) and N-terminal pro-B type natriuretic peptide were high. Her BP was 71/55mm Hg, heart rate 73beats/min which led to venoarterial (VA) extracorporeal membrane oxygenation (ECMO). Concurrent haemoptysis made her airway insecure. Hence, she was intubated and invasive mechanical ventilation was initiated. Her pulse waveform from the right radial artery became flat. Because of unstable electrical activity, she required a temporary pacemaker for three days. According to previous diagnostic criteria, acute symptoms such as chest pain, dizziness, abnormal ECG, elevated cardiac biomarkers and a thickened LV wall with severe systolic dysfunction facilitated the diagnosis of clinically suspected acute myocarditis. Her cardiac function did not improve, despite eight days of mechanical circulatory support. Chest radiograph showed pulmonary infiltration in the right lower lung field. On the same day, she was transferred to another institution for heart transplantation. Under the VA-ECMO set at the blood flow of 3.4 L/min, vital signs were as follows: mean BP 110 mm Hg, heart rate 58bpm, respiratory rate 21breaths/min and body temperature 36.2°C. An interatrial septostomy for LV decompression was performed. Septostomy was performed via the left femoral vein, due to near-total occlusion of the right femoral vein. Chest radiograph showed rapidly progressing bilateral pulmonary infiltration and the ventilator was set at a positive end-expiratory pressure of 5 cmH2O with a FiO2 of 100% could not maintain a PaO2 of 60 mm Hg without ECMO. The levels of CK-MB and hsTnI remained high. ECG showed a low voltage with a widened QRS. Echocardiography showed an LVEF of 5%, absence of aortic valve opening, and 'spontaneous echo contrast' in the ascending aorta. She was listed for a heart transplant. On the fourth day after listing, a heart from a 17-year-old brain-dead male donor was allocated. A day before heart transplant surgery, her heart stopped with a heart rate of zero on ECG. Within a few hours of the ECG change, her tidal volume also sharply declined from 400 to 180mL. She underwent heart transplantation surgery, the surgery was uneventful until the aortic cross-clamp was released. The total ischemic time was 157min. Several large thrombi occluding the aortic root, right lower pulmonary vein, superior vena cava and inferior vena cava were removed intraoperatively. Biventricular contractility was normal with minimal support from the inotropes. Upon initiation of weaning from cardiopulmonary bypass, the peripheral oxygen saturation decreased with massive sanguineous discharge from the endotracheal tube. Venovenous ECMO was used as a bridge-to-recovery strategy because of the congested lungs. After transportation from the operation room to the intensive care unit, the flooding discharge through the endotracheal tube required suction every 2–5 minutes for 9h. Chest radiograph showed bilateral white-out of the lungs. The pathological findings of the myocardium were consistent with myocarditis. The pathologic examination of the explanted heart showed an inflammatory infiltration predominantly composed of T-cells and histiocytes in all four chambers of the heart. T hese findings were consistent with acute lymphocytic myocarditis. The results of cardiac index and a mean pulmonary artery pressure were consistent with imminent right ventricular failure. The ECMO configuration was changed to veno-arteriovenous (VAV) settings to minimize blood flow to the RV. After several adjustments which included nitric oxide inhalation and alveolar recruitment maneuvers, her vital signs stabilised. On post-operative day 3, VAV-ECMO was changed to VV-ECMO. However, the bilateral damage to the lung parenchyma was more severe than expected, resulting in necrotizing pneumonia. On day 54 from the beginning of the symptoms, she died.

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