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Tozinameran

Vaccine-induced pneumonitis: case report

A 65-year-old man developed pneumonitis following vaccination with tozinameran for immunisation against COVID-19.

The man was admitted due to diffuse ground-glass opacity (GGO) and bilateral lung infiltrates on chest X-ray. Five days prior, he had received the first dose of tozinameran 0.03mg vaccine [Pfizer-BioNTech COVID-19 vaccine; route not stated]. Two days after the vaccination, he developed a low-grade fever. Cough and shortness of breath occurred the next day. Local side effects of vaccine was observed, and he did not receive any antipyretics. He had history of hyperlipidaemia, myocardial infarction and hypertension, which was well-controlled with medications including carvedilol, aspirin, rosuvastatin and enalapril [enalapril maleate]. The medications had not been changes for several years, and he was not taking any supplements. Nine years prior, he underwent percutaneous coronary intervention. He was ex-smoker (7.5 pack-years). At the first visit, an increase in RR to 24 breaths/minute was observed, and the arterial blood oxygen saturation was 85% (on room air). The other physical features and vital signs were normal. A CT scan showed patchy GGO with interlobular thickening and crazy-paving pattern. Left atrium enlargement and minimal bilateral pleural effusion were noted without lymph node swelling. Laboratory test revealed neutrophilia and leukocytosis. Other than increased CRP and LDH levels, no signs of acute heart failure, mycosis, connective tissue disease or vasculitis were observed. An ECG was normal, and ultrasonic echocardiogram demonstrated slight left atrium enlargement and normal wall motion with left ventricular ejection fraction of 65%. His serum brain natriuretic peptide level was 58.2 pg/mL. Two days after hospitalization, bronchoalveolar lavage from the right B3 and B5 with sodium chloride [saline] was performed. The recovery rate of BAL fluid was insufficient (17.2%), which was probably due to peripheral air way obstruction. As he was taking antiplatelet drug, lung biopsy was not performed. Total cell count in the BAL fluid was 0.20 × 10⁵ /mL. The cell differential count in the BAL fluid was eosinophils of 7%, lymphocytes of 14%, neutrophils of 78% and macrophages of 1%. Malignant cells or infectious organisms were not observed. His CD4+/CD8+ ratio was 0.62. Deterioration of respiratory status and chest X-ray findings was observed. After bronchoscopy examination, he needed high-flow nasal oxygen therapy.

The man was treated with methylprednisolone immediately following the bronchoscopy examination, followed by prednisolone without any diuretics. A rapid improvement was observed in his symptoms and laboratory and radiographic abnormalities. Within 15 days, systemic corticosteroids were tapered. Two weeks after the discontinuation of corticosteroids, drug sensitivity testing was done. Drug lymphocyte stimulation test was negative for COVID-19 vaccine with stimulation index of 113%. Similarly, patch test was also negative. Intradermal test (IDT) on the volar region of the forearm with COVID-19 vaccine (0.02mL) and sodium chloride 0.9% [normal saline] as negative control. At 48h, IDT demonstrated positive reaction with a wheal (larger than 30mm in diameter) and rash, although he was negative at 20 minutes. Ultimately, he was diagnosed with COVID-19 vaccine-induced pneumonitis based on the clinical course and delayed IDT reaction. No other trigger except vaccine was detected. Cardiogenic pulmonary oedema secondary to acute lung failure should be considered as differential diagnosis based on CT findings, but clinical course, serological testing and ultrasonic echocardiogram findings were not consistent with severe cardiac dysfunction. His appointment for the second dose of the COVID-19 vaccine was cancelled to avoid possible reoccurrence.

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