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Letter to the Editor
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Acute Myocardial Infarction within 5 Days after COVID-19 Vaccination: Three Case Reports from a Regional Tertiary Center

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INTRODUCTION

Several studies have reported the development of acute coronary syndrome (ACS) following COVID-19 vaccination.[1-3] In addition, a study based on the World Health Organization (WHO) database reported that 0.33% of all cardiovascular adverse events following COVID-19 vaccination were acute myocardial infarction (AMI). The elderly (> 75 years) are at higher risk of AMI, cardiac arrest, and circulatory collapse.[4] Herein, we report three consecutive cases of AMI within 5 days after vaccination for COVID-19 (AstraZeneca).

CASE REPORT

Case 1

The first patient was a 78-year-old woman who had had type 2 diabetes mellitus (DM) for 20 years and chronic renal failure under hemodialysis in last 1 year, and was regularly followed at a hemodialysis center. She was sent to our emergency department with chest pain and dizziness for 2 hours. She had received a COVID-19 vaccination (ChAdOx1 nCoV-19 vaccine, AstraZeneca) 3 days ago. On examination, her blood pressure was 99/42 mmHg, and her heart rate was 50 beats/min. A 12-lead electrocardiogram (ECG) showed ST elevation in inferior leads II, III, aVF (Figure 1A) suggesting acute ST elevation inferior wall myocardial infarction (MI).

She received emergency coronary angiography (CAG) which showed three-vessel disease with distal atherosclerosis and coronary spasm in the posterolateral branch of the right coronary artery and preserved coronary blood flow of left coronary arteries (Figure 1B, C), and subsequently underwent balloon angioplasty without stenting (Figure 1D). The peaking cardiac enzyme levels were creatine phosphokinase (CPK) 1052 IU/L, serum creatine kinase-MB (CK-MB) 161 IU/L and cardiac high-sensitivity troponin-T (Tn-T) > 10000 ng/L. Post-percutaneous coronary intervention ECG showed evolutional change at inferior leads (III, aVF). 2D echocardiography showed good left ventricular contractility [left ventricular ventricular ejection fraction (LVEF) 67%] without regional wall motion abnormalities. She was discharged smoothly five days later.

Case 2

The second patient was an 89-year-old man with a history of hypertension and type 2 DM who had been followed at a local medical clinic and presented to our emergency room with progressive short of breath for 2 days. He had received a COVID-19 vaccination (AstraZeneca) 3 days before this presentation. On examination, his vital signs were stable and a chest X ray showed cardiomegaly and lung edema.

ECG showed sinus rhythm with intraventricular conduction delay (Figure 1E). 2D echocardiography showed impaired LV contractility (LVEF 40%) with regional hypokinesia of the anteroseptal wall, anterior wall and apex. The CPK, CK-MB and Tn-T levels were 592 IU/L, 32 IU/L and 436 ng/L respectively. The N-terminal pro-brain

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natriuretic peptide (NT-pro-BNP) level was 19055 pg/mL, and the D-dimer level was 6270 ng/mL. He was diagnosed with acute non ST-elevation MI. CAG showed three-vessel disease with the culprit lesion at left anterior descending artery (Figure 1F). Coronary artery angioplasty with stenting of the culprit vessel was performed (Figure 1G), and he was discharged 20 days later under stable condition.

Case 3

The third patient was an 87-year-old woman who presented to the emergency room with chest pain and progressive shortness of breath for days. She had had colon cancer a long time before this presentation with no evidence of recurrence. She denied any chronic disease such as DM or hypertension. She had received a COVID-19 vaccination (AstraZeneca) 5 days before this presentation. On examination, she was hemodynamically stable, and an ECG showing ST elevation in precordial leads V2-V4 with a QS pattern (Figure 1H) suggestive of recent ST elevation anterior wall MI. The CPK, CK-MB and Tn-T levels were 1656 IU/L, 36 IU/L and 214 ng/L respectively. 2D echocardiography showed impaired LV contractility (LVEF 28%) with regional akinesia of mid to apical septal, anteroseptal, anterior walls and apex. A chest X-ray revealed bilateral pleural effusion (Figure 1I). The blood glucose, urea, creatinine, alanine aminotransferase, aspartate aminotransferase, electrolyte parameters, C-reactive protein, procalcitonin, and hemoglobin levels were all within normal ranges. The NT-proBNP level was 15408 pg/mL.

The patient did not receive CAG due to personal concerns. Dual antiplatelets, anti-anginal drugs were given, and sacubitril/valsartan and diuretics were subsequ-

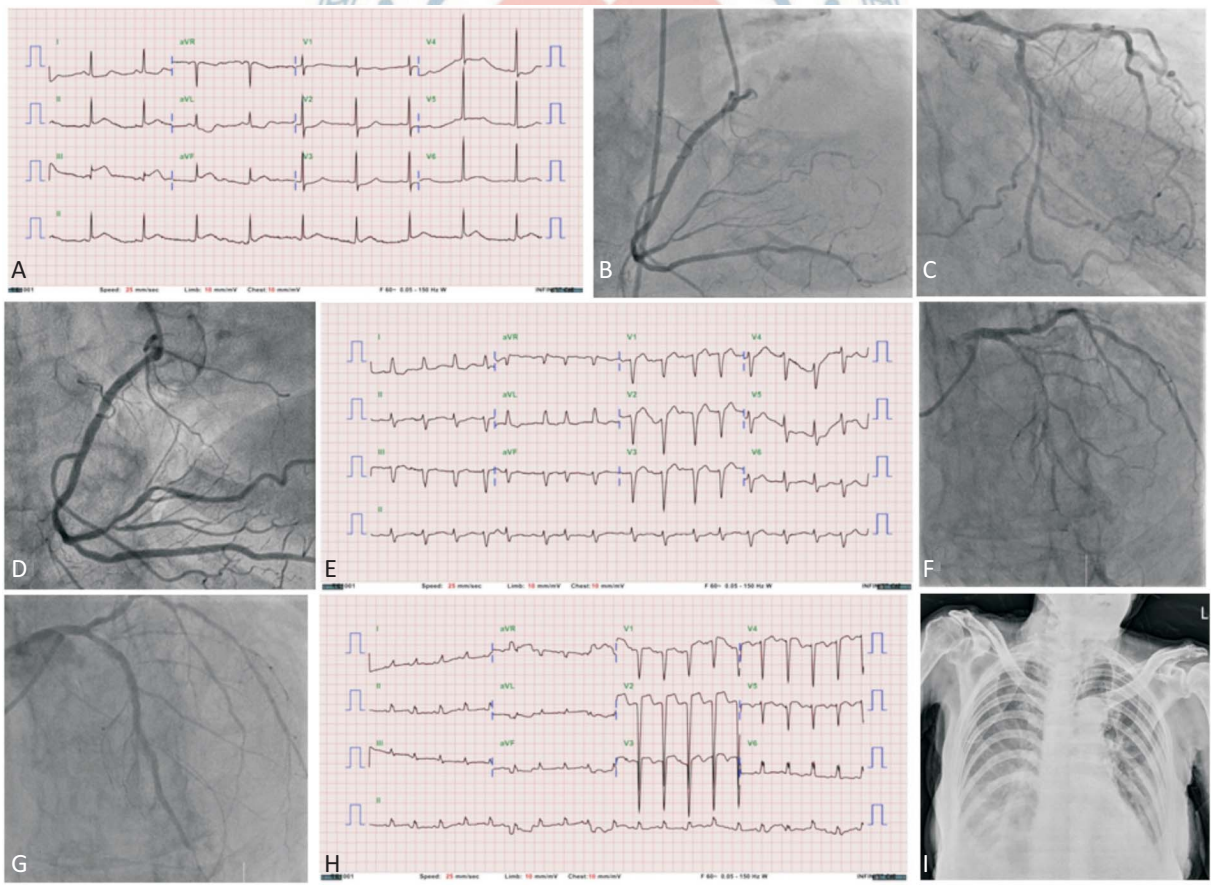

Figure 1. *(A)Twelve-lead electrocardiography (ECG) showed ST-elevation in leads II, III, and aVF. (B) Coronary angiography (CAG) showed distal atherosclerosis and coronary spasm in the posterolateral branch of the right coronary artery (RCA). (C) Preserved coronary blood flow of left coronary arteries. (D) After percutaneous coronary intervention in case 1. (E) ECG showed sinus rhythm with intraventricular conduction delay in case 2. (F) CAG showed 3-vessel disease with the culprit lesion at the left anterior descending artery in case 2. (G) After percutaneous coronary intervention in case 2. (H) ECG showed ST elevation in V2-V4 with a QS pattern. (I) Chest X-ray showed cardiomegaly and pulmonary edema.*

ently added for post-MI heart failure. The patient's hospital course was otherwise uneventful, and she was discharged home 7 days later.

DISCUSSION

The possible relationship between the vaccination and the development of ACS is unclear. There are several hypotheses. First, Greinacher et al. suggested vaccine-induced prothrombotic immune thrombocytopenia, which clinically mimics autoimmune heparin-induced thrombocytopenia.[5,6] Vaccine-induced immune thrombotic thrombocytopenia (VITT) is characterized by a low platelet count, and high levels of antibodies to platelet factor 4 (PF4), as identified by enzyme-linked immunosorbent assay, and platelet activation, which, when tested, was enhanced by addition of PF4.[6,7] Second, Boivin et al. proposed demand-supply mismatch in a frail heart post-vaccination.[1] In addition, it could be vasospastic allergic myocardial infarction in response to a vaccine, termed as Kounis syndrome (KS).[8] Type 1 KS includes patients with normal coronary arteries without predisposing factors for coronary artery disease. Type 2 KS is defined as the presence of coronary spasm due to inflammatory mediators together with the erosion or rupture of a pre-existing atherosclerotic plaque.[9]

In this report, we presented three cases with AMI early after the first dose of a COVID-19 vaccine (AstraZeneca). According to the National Health Insurance Administration Ministry of Health and Welfare, over 13,000 people have MIs per year in Taiwan. Two of our cases had chronic illness (hypertension, DM, chronic renal failure), and one had cured colon cancer. None of them had received a cardiovascular evaluation before the vaccination. None were current smokers and none had received anti-platelets or statins before AMI. There was no significant thrombocytopenia or evidence of venous thromboembolism. Therefore, VITT is less likely. The causes of AMI in our cases may be a contributing factor and could be related to a stress-induced increased myocardial demand, plaque rupture, or be classified as type 2 KS. As elderly adults have priority for the COVID-19 vaccination, the cardiologists should pay particular attention to cases of older patients with AMI after COVID vaccination. These three cases should not dampen enthusiasm for vaccinations, but should raise awareness of the possibility of myocardial ischemia in patients in older patients with multiple co-mobility.[4]

LEARNING POINTS

Although cardiovascular events have been reported in patients after receiving a COVID-19 vaccine, the causality has yet to be established. In older patients with multiple comorbidities, a careful cardiovascular risk assessment and proper treatment before receiving the COVID-19 vaccination are highly recommended, and sustained monitoring of these adverse events is also strongly suggested.

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DECLARATION OF CONFLICT OF INTEREST

All the authors declare no conflict of interest.

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