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Acute myocardial infarction within 24 hours of CoviShield vaccine

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Abstract

A 65 years old male presented with Myocardial Ischemia after 12 hours of vaccination against SARS-Cov2 (CoviShield, Oxford/AstraZeneca). The patient's angiography showed a complete blockage of the left obtuse marginal artery, which was appropriately managed, and the patient was discharged within three days. As health care workers, it is of utmost importance to be aware of possible serious outcomes of the vaccination. MI can be a serious negative outcome for any vaccine with possibly devastating consequences. Therefore, there is a need for further studies to provide better evidence-based patient care.

Keywords

CoviShield Vaccine; acute myocardial Infarction.

Introduction

Acute Coronary Syndrome (ACS) includes a spectrum of clinical presentations including ST-segment elevation myocardial infarction and Non-ST segment elevation myocardial infarction or unstable angina. It is mostly associated with partial or complete thrombosis of the affected artery and rupture of an atherosclerotic plaque. According to WHO, CVDs (Cardiovascular Diseases) alone affect around three quarters (approx. 13.4 million deaths out of 17.9 million deaths) in the low- and middle-income countries around the world [1].

Case Report

A 65-year-old man, with a history of hypertension, was admitted to the emergency department with sudden onset of chest pain within twelve hours of COVID-19 vaccination (CoviShield, AstraZeneca/Oxford). Shortly before the admission, the patient had profuse sweating and vomiting of five episodes without any history of fever, cough, cold, and shortness of breath. On his arrival in the emergency department, his heart

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rate was between 70-80 beats per minute (bpm), at an average of 73 bpm, respiratory rate was 15 per minute and peripheral oxygen saturation was at 97% on room air. His blood pressure was recorded to be 110/80 mmHg. Examinations of the cardiovascular system and respiratory system revealed normal findings. The patient had a history of hypertension with his blood pressure well-controlled with 5 mg Amlodipine, orally.

With suspicion of myocardial infarction (MI), Electrocardiogram (ECG) was performed, and cardiac troponin was sent. ECG showed a left bundle branch block likely due to ischemia. The initial troponin levels were normal. However, the level after 12 hours of symptom onset, troponin I was positive. The random blood sugar level initially was at 196 mg/dl. However, the results were normal before discharge three days later. Chest X-ray revealed haziness in the left lower lung field. COVID-19 antigen test and PCR were negative. Echocardiography showed a hypokinetic inferior posterior wall with mild Mitral Regurgitation. Other results of blood tests such as lipid profile and renal function test had normal findings. With the diagnosis of Acute Coronary Syndrome (ACS), the patient was sent to the tertiary center for angiography. The angiography showed minor plaque in the proximal part of the right coronary artery, distal right coronary artery, and proximal descending artery. Complete obstruction of the left circumflex artery in the obtuse marginal branch was noted. Percutaneous coronary intervention (PCI), stenting was performed. The postoperative course was uneventful. The patient was managed with blood thinners, anti-hypertensive, Triiodothyronine, a proton pump inhibitor, and supportive treatment. With stable condition after three days, he was discharged on Aspirin, Clopidogrel, Atorvastatin, Telmisartan, and Metoprolol.

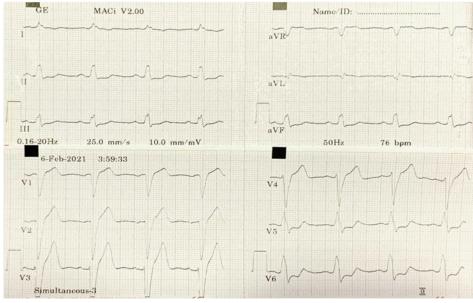


Figure 1: Left bundle branch block

Discussion

World Health Organization (WHO) estimates that 17.9 million deaths are due to cardiovascular diseases (CVDs) in 2019, which represents global deaths of 32%, and in the low- and middle-income countries over three quarters (3/4) of deaths are because of CVDs. The most important risk factors are unhealthy diet, sedentary lifestyle, tobacco, and alcohol use [1]. The presence of risk factors makes the vulnerable plaque to rupture causing the thrombotic occlusion of the coronary artery.

Severe Acute Respiratory Syndrome Coronavirus-2 (SARS-CoV2) is an air-borne virus that is transmitted through exposure to infectious respiratory fluids by direct, indirect, or close contact with the infected ones. COVID-19 vaccine gives acquired form of immunity, introduced after rapid outbreak of COVID-19. There are three mRNA vaccines, nine inactivated vaccines, five viral vector vaccines, and four protein subunit vaccines authorized for emergency use in 2021. Some vaccines like Janssen have reported incidence of thrombosis and thrombocytopenia [2].

As per European Medical Agency (EMA), CoviShield is a viral vector vaccine with rare side effects of blood clots in a blood vessel (1 in 100,000 people) and it has also reported 222 cases of thrombosis among 34 million people who received the vaccine. EMA has reported seven cases of disseminated intravascular coagulopathy and eighteen cases of cerebral venous sinus thrombosis. The relationship between the vaccine and thrombosis with thrombocytopenia has been reported by EMA [3]. Evidence suggests that the first dose of vaccine has more T-cell response that peaks within 14 days of vaccination. With the activation of T cells, CD4+ T-cells produce pro-inflammatory cytokines, interferon-gamma (IFN), and tumor necrosis factor (TNF). The second dose of vaccine has more antibody response with decreased T-cells response. An increase in the pro-inflammatory cytokines may have a possible role in MI after the first dose of vaccination [4]. One study has reported that the incidence of ACS within 24 hours after vaccination is due to its pro-thrombotic effect [5].

In Nepal, the older age groups are prioritized for vaccination, and hence one can expect to see a higher incidence of MI in the age group after COVID vaccination. This case report explores an unfortunate case of Myocardial Infarction after a dose of CoviShield vaccine in a 65-year old man. COVID-19 vaccine that promotes active immunity rarely causes thrombosis that may be immune-mediated, involving platelet-activating antibodies against platelet factor 4, which is ultimately linked to the incidence of arterial and venous thrombosis as well as thrombocytopenia [6,7]. After COVID-19 vaccination, the activation of T and B- cells leads to the release of inflammatory mediators which are responsible for plaque erosion and rupture [8]. Hence, this case does not report that MI is directly caused by COVID vaccination but suggests that MI might be linked to COVID- 19 vaccination. Even though these side effects are considered to occur on rare events, the effects are indeed too dire to ignore which is why further research is needed to test the theory in order to prevent the burden of IHD. Further, the risk of MI does not outweigh the benefits of vaccination.

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