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CASE REPORT

Sinopharm BBIBP-CorVi and Myocarditis: A Causal Link?

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ABSTRACT
Since the introduction of the SARS-CoV-2 vaccine, several side effects have been reported, including acute myocarditis, which is primarily associated with mRNA vaccines. However, the cardiac side effects of other forms of vaccination against COVID-19 are still unknown.
Here, we present a case of a possible connection between myocarditis and attenuated vaccine Sinopharm BBIBP-CorV. An 18-year-old female patient was admitted to the intensive care unit (ICU) for the management of respiratory distress associated with hemodynamic instability one week after receiving an attenuated vaccine
Myocarditis has been reported as a complication of SARS-CoV-2 mRNA vaccines. Our case report suggests a possible link between myocarditis and attenuated vaccines. Therefore, more research is warranted to determine this causal link.

KEYWORDS: Myocarditis, COVID-19 Vaccination, Attenuated Vaccine, Sinopharm BBIBP-CorV.

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INTRODUCTION

Myocardial injuries have been reported in hospitalized patients with COVID-19 with an incidence rate of up to 36% and are associated with direct myocytes or epithelial cell involvement. Moreover, these injuries may occur during the convalescence phase due to the Multisystem Inflammatory Syndrome in Adults (MISA) [1]. Myocarditis related to SARS-CoV-2 vaccines has been reported in people who received mRNA vaccines [2].

CASE REPORT

An 18-year-old female patient was admitted to the ICU to manage respiratory distress associated with hemodynamic instability one week after receiving an attenuated vaccine (Sinopharm BBIBP-CorV). The RT-PCR for SARS-CoV-2 was negative, with a serology: IgM (-) and IgG (+). Computed tomography (CT) thorax showed multifocal bilateral ground-glass opacities affecting 40% of the lung parenchyma (Figure 1).
On admission, the patient was conscious with a Glasgow score of 15, pale with discolored conjunctiva, and apyretic. Her respiratory rate was 30 cycles/min with crackling on pulmonary auscultation and a saturation of 92% on room air. Her heart rate was120 beats/min, with hypotension at 80/45 mmHg. The electrocardiogram showed sinus tachycardia with an S1Q3 appearance and flattened T waves (Figure 2). The rest of the clinical examination was normal.
The echocardiography showed the following: a dilated left ventricle with global hypokinesia, LVEF estimated at 30%–35% with nondilated right ventricle, absence of paradoxical septum with minimal mitral and tricuspid valve insufficiency, pulmonary artery pressure at 17 mmHg, and TAPSE at 16 mmHg.
Biological investigation revealed the following: Hb, 6.9 g/dl; white blood cells, 25100/mm3; hyponatremia, 127 mEq/l; hypokalemia, 3.1 mEq/l with an alkaline reserve of 6; GOT at 372 and GPT at 99 UI/L; C-reactive protein (CRP), 360 mg/l; urea, 1.37 G/l; creatinine, 40 mg/l; D-dimer, 11300 ng/ml with a troponin rate of 860 ng/ml.
The patient was put under oxygen therapy, diuretics, vasoactive drugs, heparin therapy, and antibiotic and corticosteroid therapy with dexamethasone.
PCR for other viral infections was not available to rule out a viral origin. In addition to her postvaccination symptomatology, the result of the echocardiography, and the very high troponin level, the diagnosis of myocarditis was retained and confirmed by a cardiac MRI (Figure 3).
The progression was marked by a respiratory and hemodynamic status worsening 48 hours after admission,

with signs of respiratory exhaustion and cardiogenic shock necessitating mechanical ventilation under sedation.

On the 5th day of her admission, the patient was extubated and weaned from vasoactive drugs. The echocardiographic control showed the following parameters: nondilated LV without hypertrophy, discrete inferior hypokinesia, and LVEF estimated at 48%.

On the 7th day, the patient was weaned from oxygen and discharged home with a continuous medical follow-up, with no abnormalities.

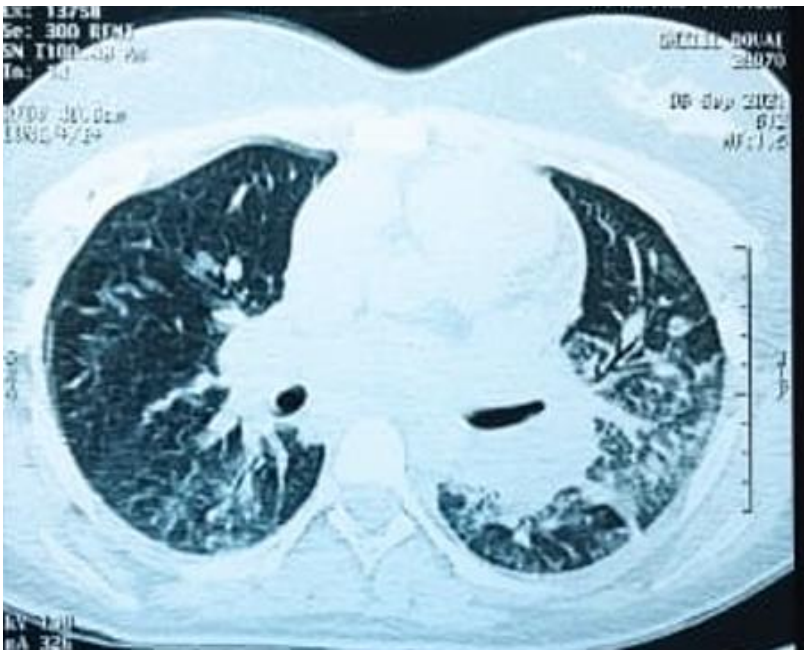

Figure 1: Chest CT showing bilateral frosted glass images

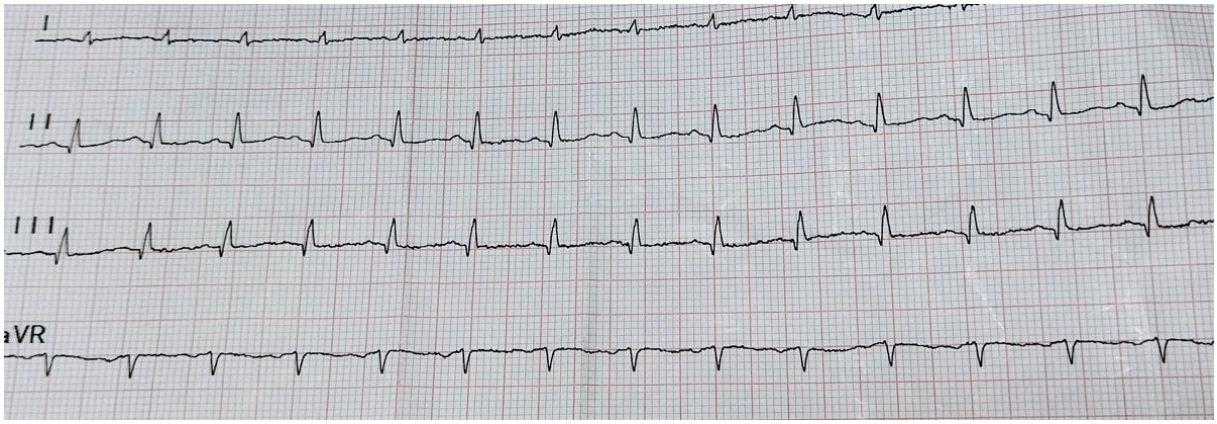

Figure 2: Electrocardiogram showing an aspect of s1q3 with flattened T waves

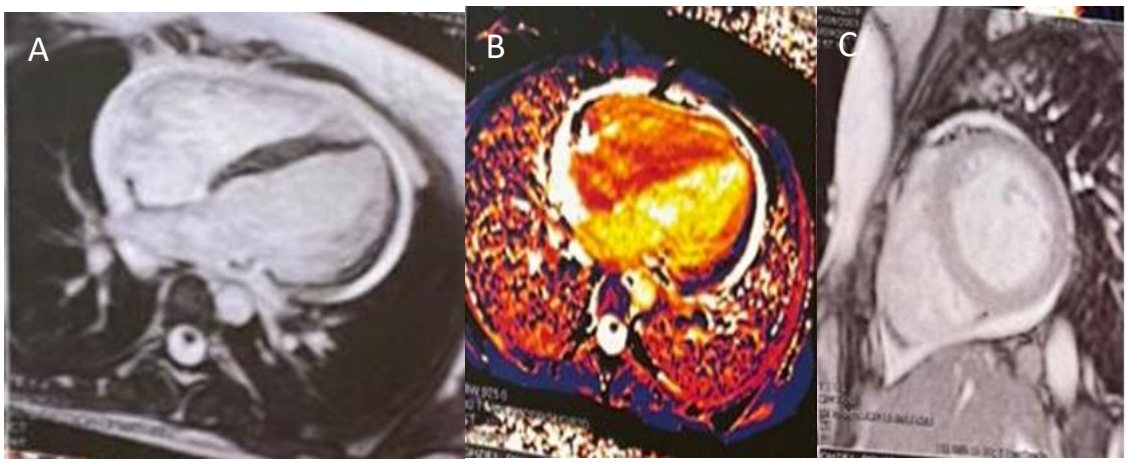

Figure 3: Cardiac MRI showing myocarditis with late intra myocardial enhancement and a thin layer of pericardial effusion at the level of the inferior wall of the LV (red arrow). A & B: cross-section, C: sagittal section.

DISCUSSION

Since the start of the pandemic, SARS-CoV-2 infection has caused illnesses ranging in severity from a simple flu-like syndrome to acute respiratory distress syndrome [3]. This variation in clinical manifestations is mainly due to the tropism of the virus at the ACE2 receptors [4], which is strongly expressed in the lungs, digestive tract, kidneys, heart, gallbladder, seminal glands, and testicles. However, the clinical symptomatology post-SARS-CoV-2 infection does not always correlate with the anatomical distribution of these receptors, whereas COVID-19 causes respiratory, neurological, digestive, cardiological, hepatic, ocular, and even skin damage [5].

COVID-19 vaccination was initiated to halt the dissemination of the virus and, more importantly, prevent serious complications; however, vaccination may have some serious side effects and complications. The following side effects due to the vaccination have been reported—minor effects: pain, swelling, and redness at the injection site, asthenia, headache, myalgia, and fever; serious side effects: myocarditis, mainly described in people who received the mRNA vaccine [6].

Myocarditis is characterized by the presence of an inflammatory infiltrate within the myocardium, which is associated with areas of necrosis of nonischemic origin. The diagnosis of acute myocarditis is based on evocative clinical symptoms (acute chest pain, heart failure, or even cardiogenic shock) and electrical, biological, and morphological criteria (MRI and echography). However, cardiac biopsy, the gold standard examination, is not recommended in current practice since the emergence of cardiac MRI, and it was not possible in our case. Depending on the initial clinical presentation, myocarditis can be classified into four forms: fulminant, acute, chronic, and active or persistent, with very different management procedures and prognoses [8].

Infectious etiologies, particularly viral, are by far the most frequent, mainly parvovirus B19, herpesvirus 6, and influenza viruses [9-11].

Postvaccination myocarditis is not a current topic. However, several cases of myocarditis have been observed after administering many different vaccines, including smallpox and influenza [12-13].

Postvaccine myocarditis was synonymous with mRNA vaccines during this pandemic, a complication seen almost exclusively in men and most often in the younger age groups [14].

In total, 23 cases of acute myocarditis were reported in military personnel, occurring within the first four days after receiving the first dose of an mRNA vaccine (7 Pfizer and 16 Moderna vaccines). All patients were male, with a median age of 25 years. Moreover, 87% of the cases occurred after the second dose of the vaccine, while the remaining patients had COVID-19 within two months before injection [15].

Kim HW has reported another four cases of acute myocarditis a few days after mRNA vaccine administration (2 cases: Pfizer; 2 cases: Moderna) [16]. However, the largest number of cases of myocarditis was reported by the Israeli Ministry of Health, with 62 cases of myocarditis in patients who took the COVID-19 vaccination out of 5 million vaccinated persons. The majority of myocarditis cases occurred following the second dose of mRNA vaccines [7].

The causal relationship between mRNA vaccines and acute myocarditis is widely described in the literature, but no case of myocarditis has been reported following vaccination with the attenuated type of Sinopharm BBIBP-CorV.

Our case presented the picture of fulminant myocarditis after the first dose of Sinopharm BBIBP-CorV vaccine, with negative PCR, positive IgG, and a cardiac MRI conclusive to the diagnosis. Furthermore, the progression was favorable, which can be attributed to the early and prompt medical management, the absence of medical history, the use of vasoactive drugs, and the patient's young age.

In our case, the inability to eliminate other viral infections due to the lack of classical molecular biology techniques and multiplex PCR makes concluding a causal link between the attenuated vaccine and myocarditis challenging. However, this possibility cannot be definitively ruled out given the delay in onset of symptoms, the absence of other diagnoses, and the previously reported association of different types of SARS-CoV-2 vaccine with this complication [12, 13].

CONCLUSION

Myocarditis has been reported as a complication of SARS-COV2 mRNA vaccines. Our case report represents a possible link between the occurrence of myocarditis and attenuated vaccines. Therefore, more studies are warranted to evaluate this link. Finally, we recommend that cardiac assessment be performed systematically in cases of respiratory distress developed after SARS-CoV-2 vaccines to look for myocarditis, which can be a fatal side effect.

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AUTHORS' CONTRIBUTIONS

The participation of each author corresponds to the criteria of authorship and contributorship emphasized in the Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly work in Medical Journals of the International Committee of Medical Journal Editors. Indeed, all the authors have actively participated in the redaction, the revision of the manuscript, and provided approval for this final revised version.

COMPETING INTERESTS

The authors declare no competing interests with this case report.

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PATIENT'S CONSENT

Written informed consent was obtained from the patient for the publication of this case report.

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