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CASE REPORT

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Ventricular tachycardia triggered by the first dose of an adenoviral vector-based COVID-19 vaccine in an adult patient with congenital heart disease

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Abstract

A unique adverse event of adenoviral COVID-19 vaccine in an adult patient with congenital heart disease is reported.

KEYWORDS

congenital heart disease, COVID-19, side effect, vaccine, ventricular tachycardia

1 | INTRODUCTION

It is undisputable that vaccines represent the most powerful weapon in the coronavirus pandemic (COVID-19) to hamper the spread of the virus and conceivably mitigate symptoms.[1] However, some quite significant adverse events have been detected following the administration of both recombinant adenoviral vector-based and m-RNA-based vaccines. Indeed, although rare, the former may trigger onset of vaccine-induced prothrombotic immune thrombocytopenia known as VIPIT, while a few cases of myocarditis and/or pericarditis have been highlighted following administration of the latter.[2,3]

Due to steady progress in their care, nowadays the number of adult patients with a congenital heart disease (ACHD) outweighs that of pediatric patients suffering from the same condition.[4] However, significant morbidity and mortality affect ACHD, including life-threatening arrhythmias.[5]

We present a patient with a past medical history of multiple surgeries for congenital heart disease and implantable cardioverted defibrillator (ICD) insertion, who developed ventricular tachycardia (VT) a few hours after injection of the first dose of an adenoviral vector-based COVID-19 vaccine (ChAd0x1 nCov-19 by Oxford-AstraZeneca). The abnormal heartbeat was recognized and stopped by ICD, and normal sinus rhythm restored.

2 | CASE PRESENTATION

We present the case of a 63-year-old woman with a background of ventricular septal defect (operated in 1965 with surgical patch and again in 1994 because of

partial patch detachment), pulmonary valve stenosis (surgically treated by pulmonary valvotomy with outflow tract patch enlargement in 1965), ICD insertion (2002) for sustained VT, ablations for atrial flutter (2007 and 2008) and for persistent VT with conventional entrainment mapping (2015), and normal coronary angiogram (2015), who developed the first recurrence of VT a few hours after injection of a ChAd0x1 nCov-19 Oxford-AstraZeneca vaccine.

At her last examination, approx. 6 months before the reported episode, the patient was in NYHA functional class I. Echocardiography displayed mild bi-ventricular enlargement—attributable to the effect of a residual peri-membranous septal defect (left ventricle) and outflow tract patch enlargement (right ventricle)—and dysfunction (left ventricular ejection fraction 48%. Right ventricular tricuspid annular plane systolic excursion 13), ICD lead crossing the tricuspid valve with valvar leaflets failure in central coaptation, tricuspid valve regurgitation at 3.3 m/sec (in the setting of a residual peri-membranous ventricular septal defect). Cardiac magnetic resonance imaging with late gadolinium enhancement was performed before ICD implantation in 2002, and severe fibrotic infiltration of the heart was reported.

The patient was taking nebivolol 5 mg once daily, furosemide/amiloride hydrochloride 40/5 mg once daily, aspirin 75 mg once daily, esomeprazole 20 mg once daily, and escitalopram 10 mg once daily.

The vaccine was administered in the morning, and no side effects were reported, with the exception of soreness and mild pain at the site of injection in the left arm. During the night, while sleeping, the patient woke up and found herself shaking all over. This was followed by shock. The following day, the patient went to hospital. Blood pressure was 115/79 mmHg, and heart rate was 75 bpm (Figure 1). At ICD check, AAI-DDD mode was confirmed (7278 PROTECTA; Medtronic), together with a lower rate of 50 bpm and upper of 110 bpm. The patient was atrial-sensed, ventricular-paced 87.9% of the time and atrial-paced, ventricular-paced 9.9%. A 24-second episode of monomorphic VT was detected. Anti-tachycardia pacing failed and accelerated the ventricular arrhythmia. The event was terminated by a DC shock (Figure 2). Troponin T was mildly elevated (0.07 ng/L) and normalized in 48 h, which is consistent with the recent DC shock. Cardiac magnetic resonance was not performed since the ICD was not magnetic resonance compatible.

Since then, the patient has been asymptomatic, without any recurrence of ventricular tachycardia so far.

3 | DISCUSSION

The steady progress in cardiothoracic surgery, interventional cardiology, and intensive care medicine has led to ACHD patients living longer, thus resulting in the current number of living ACHD patients outweighing that of pediatric patients suffering from the same condition (70% vs. 30%).[4] However, ACHD patients represent a vulnerable population from an arrhythmic standpoint.

Rhythm disturbances in ACHD are a considerable challenge and an important cause of late morbidity and sudden death. The occurrence of arrhythmia is part of the natural history of certain malformations and may result from scarring from cardiac surgery, both in the atria and ventricles, or be the consequence of an unnatural postoperative history and disturbed hemodynamics. Electrical and mechanical factors interact, and arrhythmia is often the consequence of dilation and/or hypertrophy and fibrosis. Risk stratification remains challenging because of

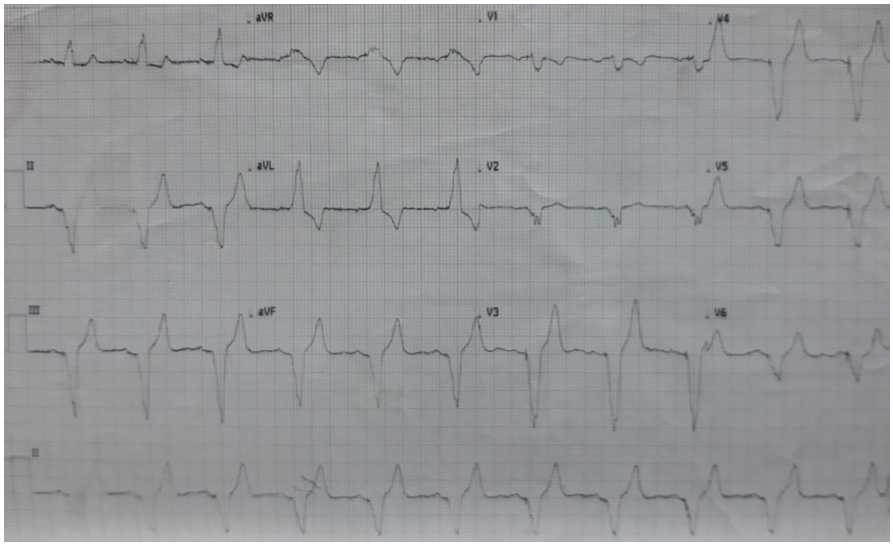

FIGURE 1 ECG showing atrial-sensed, ventricular-paced rhythm at 75 bpm

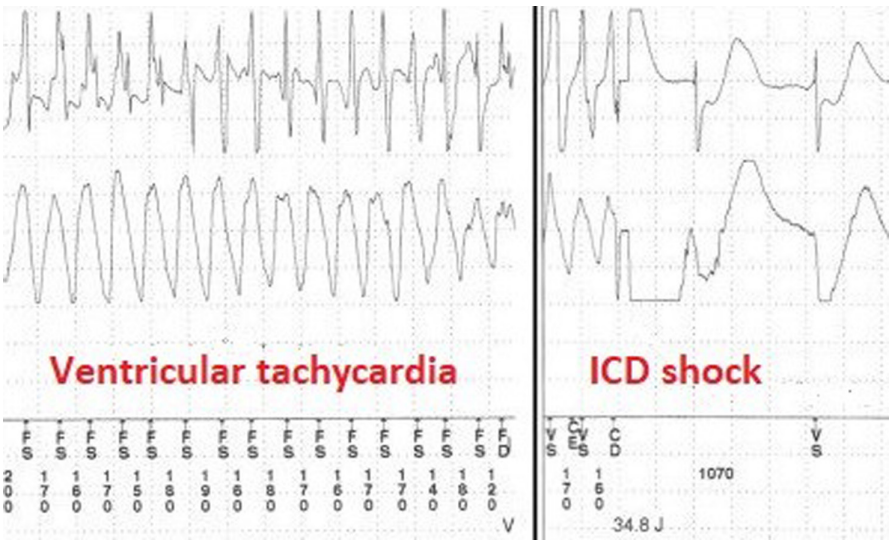

FIGURE 2 Device check testifying the onset of ventricular tachycardia which was stopped by ICD shock

the heterogeneity of the malformations and the surgical approaches.[6]

Ventricular arrhythmias in the ACHD setting include monomorphic VT, polymorphic VT, and ventricular fibrillation. In the presence of surgical scar and patch, as in the here described case, monomorphic VT is the most commonly encountered VT subtype.[6] Of note, during follow-up, at periodical 24-Holter ECG monitoring, only sporadic monomorphic extrasystolic beats were noted, accounting for no longer than 0.4% of the whole registration. The patient heart rhythm was paced most of the time. When he was in sinus rhythm, QTc, JTc interval, and QT dispersion were within the normal range, that is, 0.39, 0.32, and 0.58 ms, respectively.

ACHD seems to represent a population non-overtly susceptible to COVID-19, with very few cases of the disease reported to date.[7] Palpitations, atrial and ventricular premature complexes are not uncommon following COVID-19 vaccination, with only five cases of VT being recorded by the WHO so far, to the best of our knowledge. It is not clear whether these cases were caused by adenoviral vector-based or m-RNA vaccines. However, none occurred in the ACHD setting.[8]

In the case we present, several factors may have concurred together toward producing the harmful arrhythmia: mainly a vulnerable anatomical and electric substrate, mental stress, fever (although not documented), and immunological changes. Moreover, escitalopram and esomeprazole, both taken by the patient, particularly when given in combination, may prolong QT tract and lead to ventricular arrhythmias.[9,10]

As previously mentioned, the most important cardiac adverse event following the administration of adenoviral vector-based ChAd0x1 nCov-19 Oxford-AstraZeneca COVID-19 vaccine is VIPIT. The onset of thrombosis is at unusual sites (cerebral sinus vein and splanchnic vein), mostly in young- to middle-aged women, about 5–14 days after vaccination.[2] Regarding the other up-to-now reported adverse cardiovascular events with this kind of vaccines, they are very rare. Few anecdotal cases of pulmonary embolism, hypertensive crisis, acute coronary syndrome, and stress cardiomyopathy have been reported so far. Currently, the benefits of vaccination outweigh the risks.[11]

In conclusion, this is the first documented case of VT likely triggered by a recombinant adenoviral vector-based COVID-19 vaccine. The arrhythmia occurred in a congenital heart disease patient who was quite vulnerable from an arrhythmic standpoint and predisposed to develop this kind of adverse events.

The underlying mechanisms are purely speculative. There is a remote possibility that coronavirus vaccination might not be directly associated with the development of VT. However, the occurrence of the arrhythmia with the absence of any other obvious cause may suggest that the vaccine can have been a precipitant factor.

Clinicians should keep vaccinating ACHD patients. However, the patients should be closely monitored after vaccination. In fact, although rare, the potentially harmful adverse events triggered by COVID-19 vaccines should continue to be carefully monitored.

AUTHOR CONTRIBUTIONS

PPB involved in design and conceptualization of the case report, clinical care, analysis and interpretation of data, and drafting of manuscript. KM and KPW involved in clinical care, interpretation of data, and revision of manuscript for intellectual content.

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None.

CONFLICT OF INTEREST

None.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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