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Ectopic Atrial and Ventricular Beats and Paroxysmal Atrial Fibrillation Following SARS-CoV-2 Spike Protein Exposures

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ABSTRACT

A 57-year-old male medical worker recorded over six thousand 30-s rhythm strips for a period exceeding five years and spanning the entire COVID-19 pandemic. Clear peaks were observed in the frequency of ectopic heart beats following each spike exposure. These were predominately premature ventricular contractions (PVCs) following the mRNA vaccines and premature atrial contractions (PACs) following the virus exposures. The initial peak occurred 4 months post-mRNA vaccination gradually decreasing to 2 weeks of postvirus exposure after 4 years of monitoring. The detection of the viral exposures was made by a double peak pattern in the resting heart rate over a 7–12-day period and later confirmed on three occasions by elevated SARS-CoV-2 IgG titers. These patterns strongly suggest that COVID-19 antibodies to the spike proteins are cross-react with the cardiac conduction system, causing the ectopic beats. These PACs progressed to paroxysmal atrial fibrillation 6 weeks of postantigen exposure in two cases suggesting an autoantibody origin for paroxysmal atrial fibrillation.

1 | Introduction

Ectopic heart beats are very common and generally considered benign. They appear in three premature forms, atrial, junctional, and ventricular contractions, with premature atrial contractions (PACs) being the most common [1]. Premature ventricular contractions (PVCs) are usually benign but can indicate an underlying cardiac pathology in some individuals [2]. Ectopic heart beats are thought to be connected to many heart arrythmias with areas of fibrosis from microinfarcts being proposed for the possible origin for many of these ectopic beats [3]. Furthermore, a common arrhythmia atrial fibrillation has also been reported to be triggered by PACs [4]. Due to the very common nature of these arrhythmias, this area of cardiology remains of interest to the general medical community [5].

Arrhythmias and ectopic heart beats are also common among athletes; therefore, this is a topic of interest to exercise physiologists. An increased risk of cardiac ischemia is sometimes seen with an increase in exercise-induced PVCs [6]. Furthermore, another study suggests that PVCs occurring during recovery from exercise are more likely to be associated with long-term cardiac mortality [7]. Therefore, cardiologists generally recommend further testing to rule out coronary artery disease whenever PVCs are observed during exercise [6, 7].

During the COVID-19 pandemic, increased global rates of myocarditis, pericarditis, and cardiomyopathy were reported after receiving the COVID-19 vaccinations [8]. Furthermore, cases of myopericarditis increased after the release of the mRNA COVID-19 vaccines [9]. Interestingly, free spike protein was detected in the serum of young adults and adolescents who developed post-mRNA vaccine myocarditis providing some insight into the possible etiology of this disease [10]. Additionally, Krauson et al. have found vaccine mRNA in heart tissue with

surrounding inflammation [11], and similarly, Baumeier et al. have found small patches of myocardium that stain positive for vaccine spike protein in young men with COVID-19 vaccine myopericarditis [12]. Increased rates of cardiac arrhythmias have also been reported in COVID-19 viral infections, with some suggesting that myocardial inflammation as a possible mechanism. However, in fatal cases of COVID-19, myocardial SARS-CoV-2 and myocarditis have not been found. Inflammation is believed by some researchers to remodel the electrophysiological and structural model leading to these arrhythmias [5, 13].

During the progression of the COVID-19 pandemic, milder cases of viral infections became more common with many people developing asymptomatic COVID-19 infections [14]. This led some researchers to develop alternate techniques for detecting asymptomatic COVID-19 infections such as heart rate data from "Fitbit" wristbands. One group developed a technique which is essentially a spectral analysis of the heart rate time dependence for detecting these infections. This group demonstrated that a 100% sensitivity rate and 90.6% specificity rate are possible from "Fitbit" heart rate data [15]. Resting heart rate patterns are used in this case report to identify asymptomatic COVID-19 viral infections and identify the exact date of the change in the heart rate from the baseline data [16, 17].

Systematic, progressive changes in the frequency of ectopic heart beats and the time delay of the peak of these ectopic beats relative to the dates where the resting heart rate started to increase are presented. The patterns with each progressive exposure to the COVID-19 virus strongly suggest an autoantibody adaptive immune origin for these ectopic beats [18]. When the frequency of PACs reaches a critical amount threshold, episodes of lone atrial fibrillation are precipitated.

2 | Case Report

In 2019 prior to the COVID-19 pandemic, a 57-year-old male medical worker with a 16-year history of well-controlled Type II diabetes mellitus was referred to a cardiac clinic for evaluation and treatment of symptomatic paroxysmal atrial fibrillation and atrial flutter. He was initially treated with flecainide and Eliquis therapy and later referred to an electrophysiologist for a cavotricuspid isthmus ablation at the age of 57.7 years. This ablation was chosen because the atrial fibrillation patterns were frequently preceded by atrial flutter rhythms suggesting that the atrial flutter may be disintegrating into atrial fibrillation. Furthermore, a Fourier spectral analysis of the atrial fibrillation patterns revealed 150 and 300 bpm spectral components suggesting atrial flutter as a possible trigger [19]. Following the ablation, the flecainide therapy was discontinued but the Eliquis anticoagulation therapy was continued. In the event, the arrhythmias did not resolve, a pulmonic vein isolation ablation procedure could be considered in a future ablation. As a result, this individual began an aggressive monitoring of his heart rhythm for any reoccurrence of arrhythmias. With the exception of two lone atrial fibrillation episodes at the age of 60.7 and 62.4 years, the atrial fibrillation and flutter episodes were mostly eliminated [19]. At the age of 62.2 years, he contracted a mild influenza A(H3N2) infection followed by a brief reoccurrence of the paroxysmal atrial fibrillation episodes 6 weeks of post-infection. This event was intermittent lasting from 12 s to one hour per 24 h period over a 2-week period. He was vaccinated with the annual trivalent influenza vaccine that reportedly does not fully protect from the influenza A(H3N2) strain [20]. Interestingly, a large study of 11,374 patients in Taiwan did reveal a 18% increased rate of atrial fibrillation following influenza infections [21].

During a period exceeding five years between ages 57 and 62 years, this individual accumulated over six thousand 30-s rhythm strips with multiple recordings every day. Interestingly, this period also spanned the entire length of the COVID-19 pandemic. Patterns in the frequency of PACs and PVCs began to emerge during the COVID-19 pandemic. He also received the first two COVID-19 mRNA Moderna vaccine doses at age 59 years in January 2021 (Lots 039K20A and 004M20A). An increase in the SARS-CoV-2 IgG titer did occur as a result of those mRNA injections.

He along with 800 other workers in his clinic and regional VA hospital contracted a symptomatic COVID-19 Omicron infection in January 2022. This was believed to be the Omicron BA.1 strain because several hospital workers tested positive for that strain. As shown in Figure 1, he observed a double peak increase in resting heart rate over a ten-day period from the Omicron BA.1 infection at age 60 years. Over the course of the pandemic, he observed seven more double peak increases in resting heart rates with no symptoms. He received titers after three of those events showing that the titers were off-scale, suggesting frequent exposure to these viruses in his clinic environment. In each event, the most prevalent strains circulating in the community per CDC data are the strains denoted in the figures. At the age of 62.2 years, he contracted a mild influenza A(H3N2) infection followed by a reoccurrence of paroxysmal atrial fibrillation 6 weeks later at age 62.4 years. Unlike the COVID-19 Omicron infection, he only observed a single-peak increase in resting heart rate peaking at 4-5 days. In the case of COVID-19, the double-peak pattern in the resting heart rate may be associated with the ACE-2 receptors being located in both the respiratory and vascular systems with the immune response releasing interleukins separately from both systems [18].

Three months after the Omicron BA.1 infection, this worker recognized an increase in frequency of PACs. He also recalled an increase in PVCs starting 1 year earlier that appeared to be resolved by October 2022. As a result, the rhythm strips were reviewed to calculate the actual frequency of PACs and PVCs averaged over each month. Furthermore, the data were sorted into resting and exercise states because the original atrial fibrillation episodes at age 57 were most common immediately following strenuous exercise. The first plot was stunning in that it showed a dramatic increase in the frequency of PVCs following the Moderna mRNA-1273 COVID-19 vaccines and later the frequency of PACs following the COVID-19 BA.1 infection. These data were later extended to the end of December 2024 eight months after he contracted a mild influenza A(H3N2) infection. Figure 2 shows the frequency of PACs and PVCs between July 2019 and December 2024. Locations of where the pandemic started and ended are also indicated as several exposures to COVID-19 strains from the clinic. The COVID-19 strains indicated in the figures were those reported to be most prevalent in the community by the CDC at the time of these exposures [14]. The timing of these exposures was determined from changes in the resting heart rate data from the previous figure.

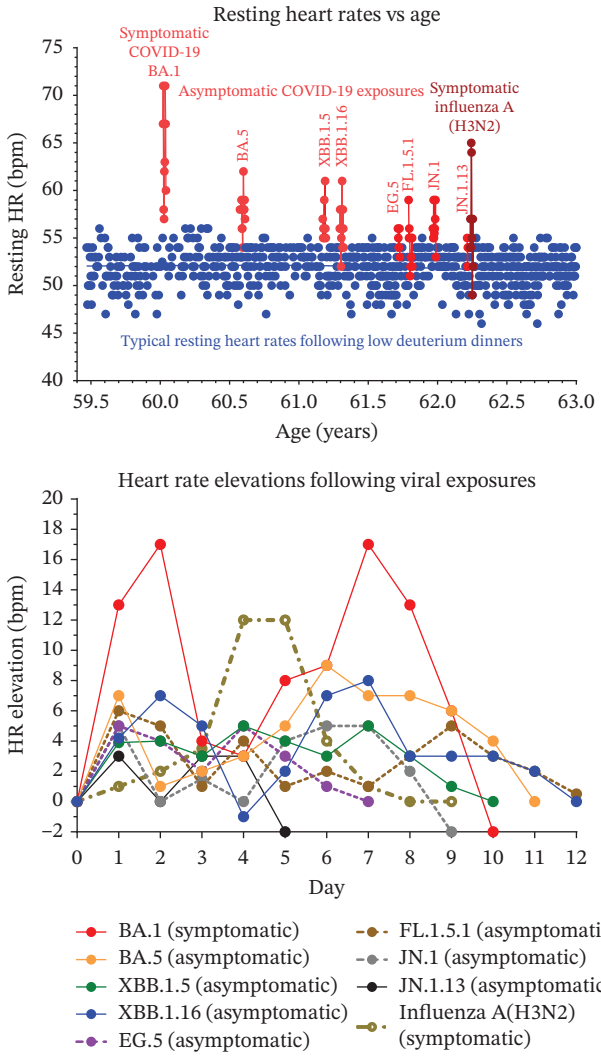

FIGURE 1 | The top panel reveals resting heart rates recorded daily before rising from bed between ages 59.4 and 63 years. Typical heart rates range from 46 to 56 bpm depending on the food eaten for dinner the previous evening. These are recorded as solid blue circles. Heart rates above the expected range occur with certain infections including COVID-19, and these are shown as solid red circles. The first COVID-19 infection, Omicron BA.1, was symptomatic with seven asymptomatic infections over the following 2 years. A symptomatic influenza A(H3N2) infection occurred at the age of 62.2 years is shown as solid rust-colored circles. The lower panel shows the calculated resting heart rate deviations above the expected values for specific meals eaten vs. time following the start of the double-peak heart rate pattern. The increased heart rates ranged from 7 days in length for EG.5 to 12 days in length for FL.1.5.1. The specific COVID-19 strain shown was the dominant strain reported in the community by the CDC at the time. Importantly, COVID-19 IgG titers were measured as off-scale following three of these exposures confirming exposures to this virus commonly occurred in the clinical work environment. Unlike COVID-19, a symptomatic influenza infection A(H3N2) showed a single-peak rise in the heart rate.

The delayed onset of ectopic heart beats following exposures to COVID-19 spikes, by direct virus contact, suggest an autoantibody origin for these features. The data were replotted as a function of weeks following the exposure date as determined by the start of the double-peak rise in the resting heart rate, as

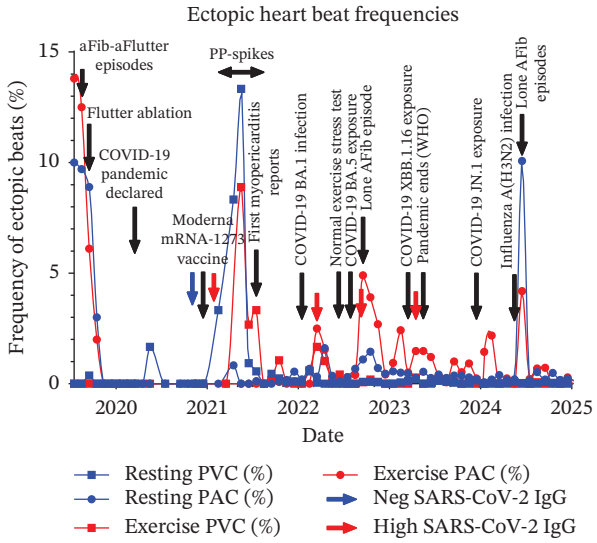

FIGURE 2 | Frequency of ectopic heart beats initially decreased following the cavotricuspid isthmus ablation and later increased following each exposure to the COVID-19 spike protein. The largest increase was in the form of PVCs following the Moderna mRNA-1273 vaccines and PACs following exposures to the various COVID-19 viral strains. Also shown in the figure is a negative cardiac stress test in 2022 and three markedly elevated spike protein IgG titers (red arrows) after three of these exposures confirming frequent exposures to this virus in a clinical work environment. Except for the mRNA vaccine, most of the ectopic peaks occurred on days of strenuous physical activity (red). The vaccine caused a predominately PVC pattern with a slightly higher frequency occurring on rest days (blue) compared to strenuous physical activity (red). The influenza also caused a slightly higher frequency of PACs during rest. The increase in PAC-induced paroxysmal atrial fibrillations following an asymptomatic COVID-19 BA.5 exposure and a symptomatic influenza A(H3N2) infection.

shown in the bottom panel of Figure 1. The data were also summed into weekly segments for better resolution, and all the ectopic beats (PACs and PVCs) were counted together to improve the statistics. These are shown in Figure 3 with best fit curves matched to the data. The most pronounced increase in ectopic heart beats occurred following the second mRNA COVID-19 vaccine dose or 12–16 weeks after the first vaccine dose. The peak in ectopic beats occurred at 12 weeks following the symptomatic COVID-19 Omicron BA.1 infection. Each progressive exposure to a COVID-19 strain in the clinic over the next 2.5 years showed a progressively earlier response strongly suggesting an autoantibody adaptive immune response to the COVID-19 spike proteins. By the time the JN.1.13 strain was circulating, the data were beginning to drop into the background noise. Although exposure to the strains after BA.1 was asymptomatic indicating natural immunity, some notable arthralgia was reported 2 weeks after exposure to the JN.1 strain.

It is possible that the COVID-19 virus and/or mRNA COVID-19 vaccines can potentially damage pacemaker cells. Inflammatory cytokines could also impair cardiomyocytes. The outcome results in "sick" pacemaker cells or cardiomyocytes capable of evoking arrhythmias independent of autoimmunity. These changes could also occur very early in an infection, i.e., first increase in the heart rate, as shown in Figure 1.

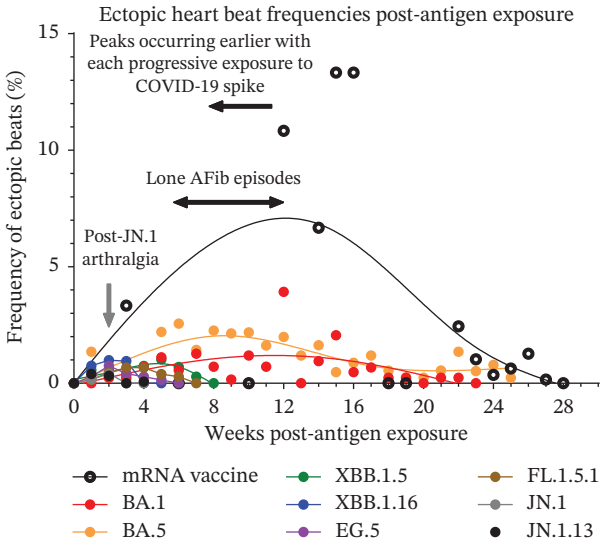

FIGURE 3 | The ectopic frequency of combined PACs and PVCs over a weekly basis following each exposure to the COVID-19 spike. The first exposure to the spike was the Moderna mRNA vaccine which is coded as open black circles. The origin $t = 0$ was set to the day of the first of the two mRNA doses. Eight subsequent exposures are shown in sequential order indicating that the peaks are occurring progressively earlier with each exposure to the spike protein. This strongly suggests an autoantibody adaptive immune response may be responsible for these patterns [18]. The COVID-19 strains shown in the legend were the most prevalent strains reported in the community at the time per the CDC, and the color coding of these strains also match the color coding of the lower panel in Figure 1. Lone atrial fibrillation episodes occurred between 6 and 12 weeks of postantigen exposure to the COVID-19 BA.5 antigen and a symptomatic influenza A(H3N2) infection.

The appearance of atrial fibrillation occurred twice during a five-year period following the cavotricuspid isthmus ablation. A lone episode occurred six weeks after the exposure to COVID-19 BA.5 strain, and paroxysmal AFib episodes reoccurred 6 weeks post-influenza A(H3N2) infection. These are indicated in both Figures 2 and 3. Both events occurred after a rise in the frequency of PACs, suggesting a common etiology for both of these ECG features.

3 | Discussion

Several mechanisms have been proposed for the etiology of atrial fibrillation with autoantibodies targeting the calcium channels within the pulmonary vein for the paroxysmal forms [22–24] and structural remodeling of the atria from hypertension, valvular problems, CHF, smoking, or alcohol use for the sustained forms [5, 25–27]. Genetic mutations in the potassium ion channels have also been reported to increase the risk of lone atrial fibrillation episodes [28]. Researchers have also reported that atrial fibrillation and atrial ectopic beats most frequently originate within the pulmonary veins extending as far as 4 cm from the heart [29]. This region is exposed to all the antibodies travelling within the systemic circulation. The endothelium of the pulmonic vein in this region is also different in that there is an absence of the von Willebrand factor (vWF), unlike in all other veins [30].

There have been multiple reports of increased rates of arrhythmias during the COVID-19 pandemic. During the first year of the pandemic, researchers showed increases in PACs, PVCs, and atrial fibrillation in 186 post–COVID-19 patients. The authors postulated that inflammation and hypoxia may have caused cardiac conduction abnormalities leading to these findings [31]. Furthermore, an increased risk of arrhythmias was also reported in England following either a positive SARS-CoV-2 test or the second dose of the mRNA-1273 Moderna vaccine between December 2020 and August 2021 [32]. Similarly, it was also reported that 254 arrhythmias were among the 17636 reported adverse cardiovascular complications following the COVID-19 mRNA vaccines between December 2020 and January 2022 [33]. A recent review article examines other viruses that are also capable of inducing arrhythmias that have received less attention than COVID-19 [34].

In regards to the COVID-19 mRNA vaccines, there was a case report of a 35-year-old female with an existing autoimmune disorder (low level 1:80 ANA titer) who developed a 6-month history of elevated PVC counts following her second COVID-19 mRNA vaccine dose. She was reported to have 6 genetic markers placing her at a higher risk for developing CVD/diabetes mellitus that were believed to have contributed to her increased PVC count [35]. This same effect occurred in the current case presented here where the individual reported a 16-year history of well-controlled diabetes mellitus who developed PVCs reaching a peak after the second mRNA vaccine dose. As shown in Figure 3, these PVCs started 3 weeks after the first vaccine dose reaching a peak around 16 weeks. Near this peak at 15 weeks, an episode of ventricular bigeminy, Figure 4, was recorded on a Holter monitor. Figure 5 shows the increase in these PVC counts persisted up to 675 days following the first mRNA vaccine dose. Hulscher et al. have described fatal, autopsy-proven vaccine myocarditis in patients with no prior clinical evaluation, implying that the first manifestation of vaccine myocarditis can be sudden death [36].

Other researchers have found double proline spike protein fragments, e.g., PP-spike fragments, in the blood of vaccinated individuals between 69- and 187-day postvaccination independent of their IgG antibody titer [37]. Furthermore, these double prolines are found in the mRNA vaccines but not the mRNA of the actual COVID-19 virus [37]. The time of resolution for these PP-spike fragments is very similar to the time of resolution of the PVCs in the 35-year-old female [35], and the current case discussed here strongly suggest that PP-spike fragments directly induced these PVCs. These PVCs suggest that the spike proteins are toxic.

Boros et al. point out that deuterium (heavy hydrogen)-rich prolines and hydroxyprolines in the vaccine-coded spikes resist enzymatic breakdown leading to these long-lasting spike proteins seen in the circulation [38]. Furthermore, ribosomal frameshifting due to the use of pseudouridine-enriched mRNA also caused an unexpected appearance of two higher molecular weight peptides raising concerns about the safety of these mRNA vaccines [38].

These PVCs and other cardiac effects may not have occurred if the mRNA vaccines had been produced from the N protein–coding region rather than the S protein–coding region of the coronavirus genome. Researchers have shown that antiserum can be derived from bat-SL-CoV-ZC45 in laboratory animals [39]. Noting that the nucleocapsid region of the SARS-CoV-2 virus

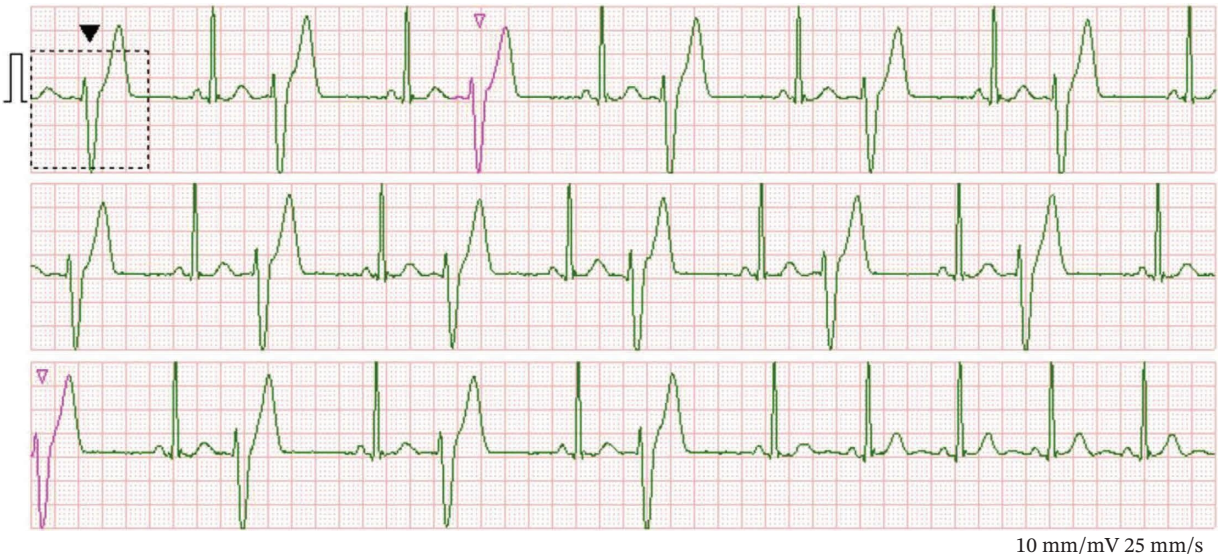

FIGURE 4 | An episode of ventricular bigeminy was recorded on a 24-h Holter monitor 15 weeks after the first mRNA COVID-19 vaccine dose. This episode occurred during the PVC rate peak, May 2021, and lasted 30 s. The individual only reported minimal palpitations. There was no shortness of breath or chest discomfort. This image was captured as a screen dump. The black box at the upper left can be used as a zoom function of any part of this rhythm strip in the Holter software. The two pink sections correspond to the individual pressing a button indicating regions of symptomatic palpitations.

genome matches the bat-SL-CoV-ZC45 genome suggests that these mRNA vaccines produced from this alternate coding region of the genome could have provided immunity to the SARS-CoV-2 virus with less risk of these PVCs and other cardiac arrhythmias [40].

Unlike the mRNA vaccine inoculations that preceded increases in the frequency of PVCs, direct exposure to the COVID-19 virus led to delayed increases in the frequency of PACs. Figure 3 shows that the peaks in the frequency of PACs occur progressively sooner after each exposure with an overall decrease in amplitude

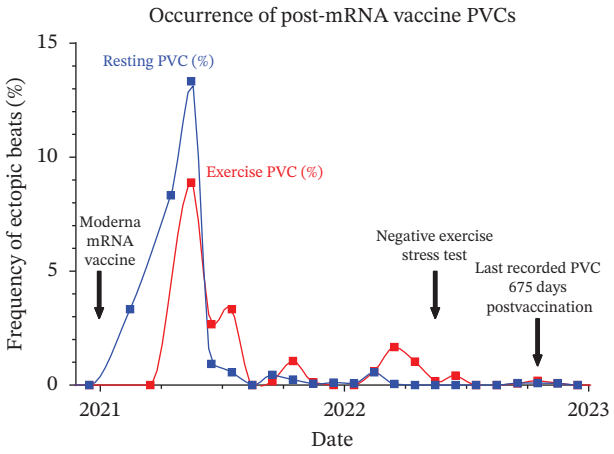

FIGURE 5 | Monthly rates of premature ventricular contractions (PVCs) following the mRNA COVID-19 vaccinations. Both doses were received in January 2021 with the first dose received on the 4th day of the month. Three to six peaks of increased PVC rates occurred with the last recorded PVC occurring 675 days after the first COVID-19 vaccination. These PVCs are believed to be caused by circulating spike proteins induced from the mRNA vaccines. A baseline PVC rate between January 2023 and January 2025 remained at a relatively low rate of 0.049%.

of these rates. These findings are the patterns one would expect from a humoral adaptive immunity response [18]. Furthermore, if a threshold PAC frequency is reached, a lone atrial fibrillation episode can be triggered.

4 | Conclusion

Atrial fibrillation is a supraventricular arrhythmia that adversely affects cardiac functioning. It is the most common cardiac arrhythmia putting individuals at increased risk of cerebral infarcts (stroke), and it increases with age [5]. The data presented in this report show that lone atrial fibrillation can occur when PACs reach a critical amount threshold. The data also suggest a common origin for both of these electrocardiogram features with atrial fibrillation being a sustained form of these PACs. The importance of understanding this mechanism is that it may assist in the development of future treatment protocols for atrial fibrillation.

In this study, the etiology of lone atrial fibrillation in COVID-19 appears to be subclinical myopericarditis (mRNA and spike protein in the myocardium-causing inflammation) with cardiac electrical irritability manifested by frequent PACs and PVCs. It is possible that autoantibodies directed toward viral antigens, i.e., COVID-19 and influenza A(H3N2), later cross-react with the calcium channel gates of the pulmonic vein. This results in premature triggering of these gates leading to an improper depolarization of the atrial conduction system. An autoimmune origin for paroxysmal atrial fibrillation has been suggested by others [22–24], but this study is the first to actually reveal a progressive adaptive immune response to a specific class of viral pathogens.

Fortunately, many supraventricular arrhythmias can be managed with a pharmacologic rate and rhythm control [41]. More invasive treatment procedures to fill or obliterate the left atrial appendage [41] or catheter ablation to electrically isolate the

pulmonic vein from the atrial endothelium [42, 43] are frequently useful in lowering the rate of these arrhythmias.

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Consent

A signed written informed consent form was obtained from the participant prior to the manuscript writing and submission. The participant gave their consent for publication.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data are available upon request from the authors.

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