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**EDITORIAL**

Li P, Zhang HP. From surgery to recovery: Measuring success through quality of life and functional improvements after cardiac surgery. *World J Cardiol* 2025; 17(2): 100213 [DOI: [10.4330/wjc.v17.i2.100213](https://doi.org/10.4330/wjc.v17.i2.100213)]

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Luo LY, Yang TS, He Z, Lin L, Luo XL. Comprehensive understanding of a rare disease: Cardiac metastatic tumor, a double-center 10-year case review. *World J Cardiol* 2025; 17(2): 101851 [DOI: [10.4330/wjc.v17.i2.101851](https://doi.org/10.4330/wjc.v17.i2.101851)]

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## Risk stratification for future cardiac arrest after COVID-19 vaccination

Peter A McCullough, Nicolas Hulscher

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### Abstract

Unheralded cardiac arrest among previously healthy young people without antecedent illness, months or years after coronavirus disease 2019 (COVID-19) vaccination, highlights the urgent need for risk stratification. The most likely underlying pathophysiology is subclinical myopericarditis and reentrant ventricular tachycardia or spontaneous ventricular fibrillation that is commonly precipitated after a surge in catecholamines during exercise or the waking hours of terminal sleep. Small patches of inflammation and/or edema can be missed on cardiac imaging and autopsy, and the heart can appear grossly normal. This paper reviews evidence linking COVID-19 vaccines to cardiac arrest where unfortunately the majority of victims have had no antecedent clinical evaluation. We propose a comprehensive strategy for evaluating cardiovascular risk post-vaccination, incorporating detailed patient history, antibody testing, and cardiac diagnostics in the best attempt to detect abnormalities before sudden cardiac death. This approach aims to identify individuals at higher risk of cardiac events after COVID-19 vaccination and guide appropriate clinical management. It is prudent for each primary care physician to have a pre-established plan when addressing this issue in their practice.

**Key Words:** COVID-19; mRNA vaccines; Myocarditis; Cardiac arrest; Risk stratification; Cardiovascular safety

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**Core Tip:** This study reviews evidence linking mRNA vaccines to cardiac pathology and proposes a comprehensive risk stratification approach involving patient history, antibody testing, and cardiac diagnostics. By identifying high-risk individuals through measurable endpoints like spike protein exposure and cardiac biomarkers, this approach seeks to guide clinicians in addressing the risks of myocarditis, arrhythmias, and cardiac arrest post-vaccination. Implementing this framework in primary care settings may improve cardiovascular outcomes and reduce preventable deaths.

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## INTRODUCTION

We continue to observe coronavirus disease 2019 (COVID-19) vaccinated persons suffer cardiac arrests since the inception of the mass vaccination campaign in late 2020[1-5]. **Figure 1** illustrates the likely mechanisms. Both Pfizer-BioNTech (BNT162b2) and Moderna (mRNA-1273) mRNA have been found in human heart muscle at autopsy[6]. Spike protein has been stained in endomyocardial biopsy samples of young men suffering from COVID-19 vaccine-induced myocarditis[7]. Victims have been found to have circulating Spike protein but ineffective antibodies, likely IgG4 subclass, that fail to neutralize Spike protein and allow its assault on the heart[8]. Positron emission tomography data have revealed a shift from free fatty acid metabolism to glucose metabolism in the hearts of the majority of individuals who have received a COVID-19 vaccine[9]. The positron emission tomography pattern resembles global ischemia. This could be due to vaccine Spike protein hemagglutination in myocardial capillaries or cellular changes in mitochondrial respiration and substrate metabolism[10]. Small patches of dysfunctional, inflamed, or scarred myocardium are sufficient to serve as a nidus for re-entrant ventricular tachycardia that can degrade to ventricular fibrillation and lead to cardiac arrest[11]. A surge in catecholamines (epinephrine, norepinephrine, and dopamine) that can occur during sports or the waking hours of sleep (3 AM to 6 AM) may trigger reentrant ventricular tachycardia or spontaneous ventricular fibrillation leading to a cardiac arrest in patients with COVID-19 vaccine subclinical myocarditis[12].

## RATIONALE FOR RISK STRATIFICATION

Elevated numbers of sudden deaths among athletes after COVID-19 vaccination have raised concerns[13]. Alessandria *et al*[14] amplified these concerns, demonstrating higher all-cause death risks in COVID-19 vaccinated individuals compared to the unvaccinated. Participants that received 2 doses lost 37% of life expectancy compared to the unvaccinated population during follow-up. The largest COVID-19 vaccine safety study to date with approximately 99 million vaccinated individuals found that the risk of myocarditis was significantly elevated after mRNA COVID-19 vaccinations, with the risk being 510% higher following the second dose of the mRNA-1273 vaccine and 186% higher following the second dose of the BNT162b2 vaccine, compared to baseline rates[15]. Hulscher *et al*[16] have demonstrated that cardiac arrest within a few weeks of COVID-19 vaccination is likely caused by vaccine myocarditis with no prior premonitory phase that allows for detection. Rose *et al*[17] found that among individuals with clinical myocarditis shortly after COVID-19 vaccination reported to the Vaccine Adverse Event Reporting System (VAERS), the mortality rate was 2.9%.

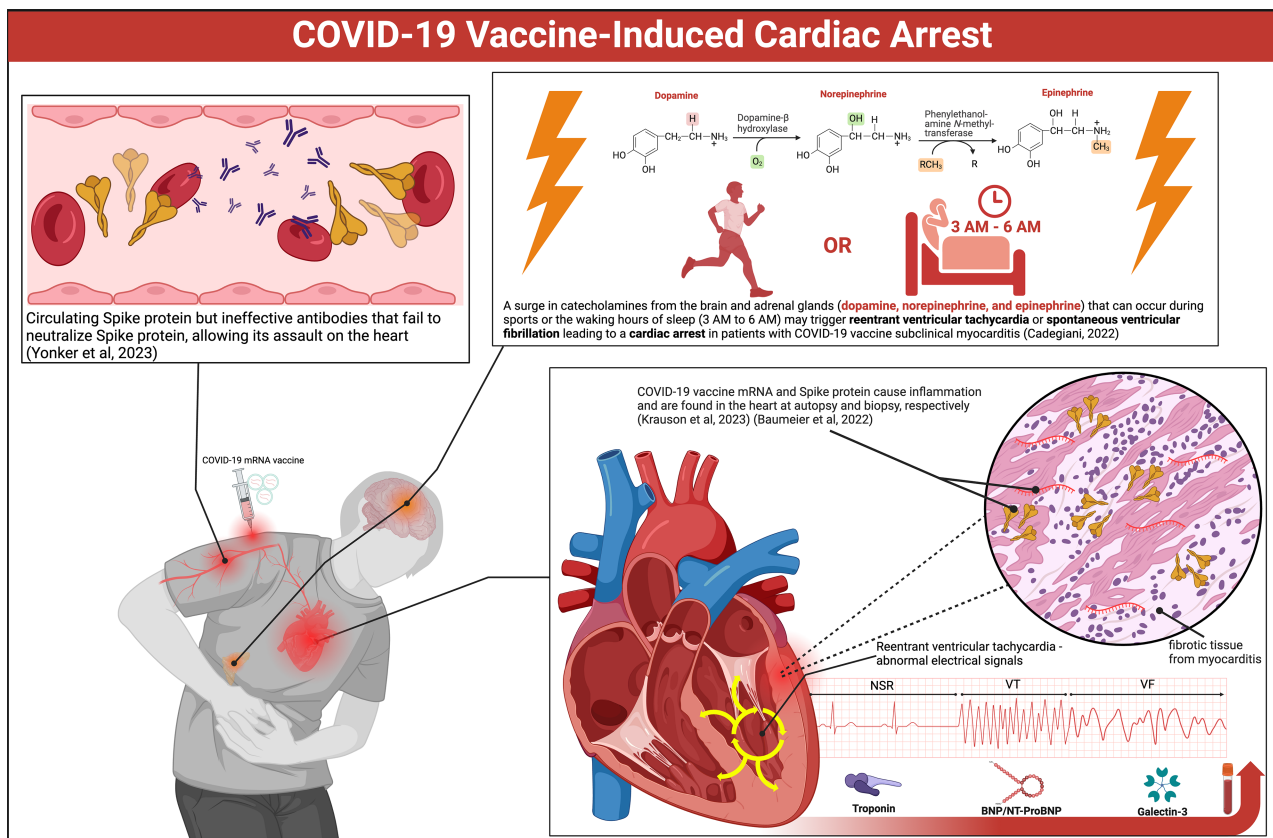
The United States Food and Drug Administration's Center for Biologics Evaluation and Research has established a follow-up period of 5 to 15 years for novel genetic products to monitor for any long-term adverse effects that may emerge in the exposed population[18]. With the passage of time, we have learned much from the evaluation of many vaccine-injured victims who have symptoms months to years after injection[19-21]. Brogna *et al*[22] found vaccine-generated stabilized prefusion Spike protein in subjects up to 6 months after COVID-19 mRNA vaccination. Cardiac abnormalities have been observed for at least a year following the initial diagnosis of COVID-19 vaccine-induced myocarditis, suggesting the potential for long-term effects[23]. As of January 13, 2025, we are unaware of any published risk stratification approach for the prevention of COVID-19 vaccine-induced cardiac arrest. Thus, we propose a comprehensive strategy to address this concerning gap in knowledge.

## RISK STRATIFICATION APPROACH

When patients are seen in clinical practice for the initial evaluation of cardiovascular symptoms following severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection or vaccination, a proposed approach is outlined in **Figure 2**. COVID-19 vaccine-induced myocarditis is caused principally by the Spike protein[7,8,16,24]. Measures of cytokine activation and inflammation are secondary to Spike protein resident in the myocardium and circulating in blood[7,8].

A detailed SARS-CoV-2 infection and vaccine administration history is essential to the evaluation. Serious COVID-19 requiring hospitalization and mechanical ventilation should be noted. Each SARS-CoV-2 infection and COVID-19 vaccine





**Figure 1** Coronavirus disease 2019 vaccine-induced cardiac arrest. NSR: Normal sinus rhythm; VT: Ventricular tachycardia; VF: Ventricular fibrillation; BNP/NT-proBNP: Brain natriuretic peptide and N-terminal proBNP (Created in BioRender, [Supplementary material](#)).

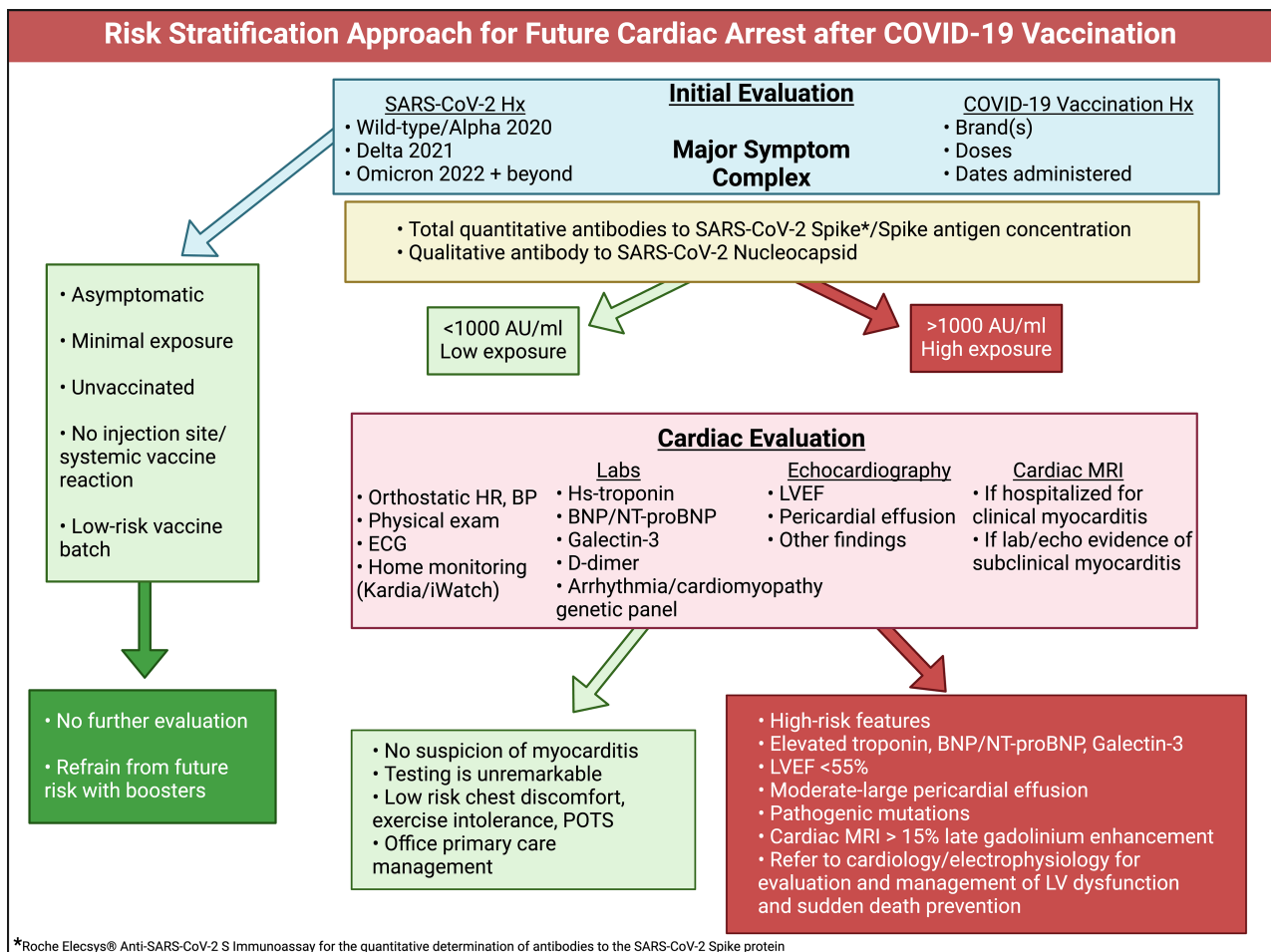
dose can be considered an ‘exposure’. It can be reasonably assumed that the greater the number of exposures, the more likely it is to have large quantities of Spike protein in the body, where natural proteases and lysosomes seem unable to clear it[25].

An extended range total antibody against the Spike protein is a useful proxy for prior Spike exposure. For example, the Roche Diagnostics Elecsys Anti-SARS-CoV-2 Spike assay measures antibodies against the receptor binding domain[26]. Roche Elecsys Anti-SARS-CoV-2 assay has a normal value of < 0.8 U/mL, and in clinical practice, 0-1000 is low risk, and > 1000 indicates higher risk with either multiple infections or COVID-19 vaccine administrations. We have observed that it is not uncommon to find patients with > 25000 U/mL, remaining unmeasurably high even years after vaccination. Antibody concentrations can take at least 12 months to taper off. Thus, a Spike antigen test measurable in whole blood, plasma, or serum is greatly needed to provide a real-time estimate of Spike toxicity.

If (1) the number of Spike protein exposures was low; (2) there were nonserious infections; (3) Spike antibody levels are below 1000 U/mL; (4) minimal or no injection site or initial systemic reaction to vaccination[27,28]; and (5) if the vaccine was obtained from a low-risk batch[29,30] (with zero hospitalizations and deaths found in a VAERS batch analysis[31]), then additional clinical investigation may not be warranted. If there are higher risk features as indicated in the [Figure 2](#), then it is prudent to perform more formal cardiac testing and risk stratification.

It is reasonable to obtain an electrocardiogram and blood testing for high-sensitivity troponin, BNP/NT-ProBNP, galectin-3, and D-dimer. Elevated troponin may indicate ongoing myocarditis[32]. BNP/NT-proBNP are reliable indicators of cardiac pressure/volume overload and predict heart failure[33]. Galectin-3 is a chronic inflammation/fibrosis marker and when elevated predicts future heart failure[34]. D-dimer is a proxy for micro-blood clotting[35] and in our experience < 0.2 is low risk, 0.2-0.5 is moderate, and > 0.5 is high risk for thrombotic events. Patel *et al*[36] demonstrated that 17% of participants exhibited an increase in d-dimer levels from their normal baseline following COVID-19 vaccination. Genetic testing for pathogenic mutations in cardiac ion channels or major proteins is reasonable in patients with high-risk findings or resuscitated cardiac arrest. Mutations in the SCN5A sodium channel have been associated with an increased risk for COVID-19 vaccine cardiac arrest[37].

Point-of-care echocardiography or formal cardiac ultrasound and Doppler can be used to evaluate cardiac structure, function, and presence of pericardial fluid[38]. This package of diagnostics, when results are completely normal, suggests a low risk for future heart failure and cardiac death[39]. Conversely, multiple abnormalities found in electrocardiogram, blood tests, and echocardiography can prompt the use of cardiac magnetic resonance imaging (MRI) with contrast[40]. The MRI is confirmatory for left ventricular function and importantly can detect areas of inflammation/scar with late gadolinium enhancement (LGE)[41]. In other types of cardiomyopathy, LGE ≥ 15% of the left ventricle indicates high risk for cardiac arrest[42].



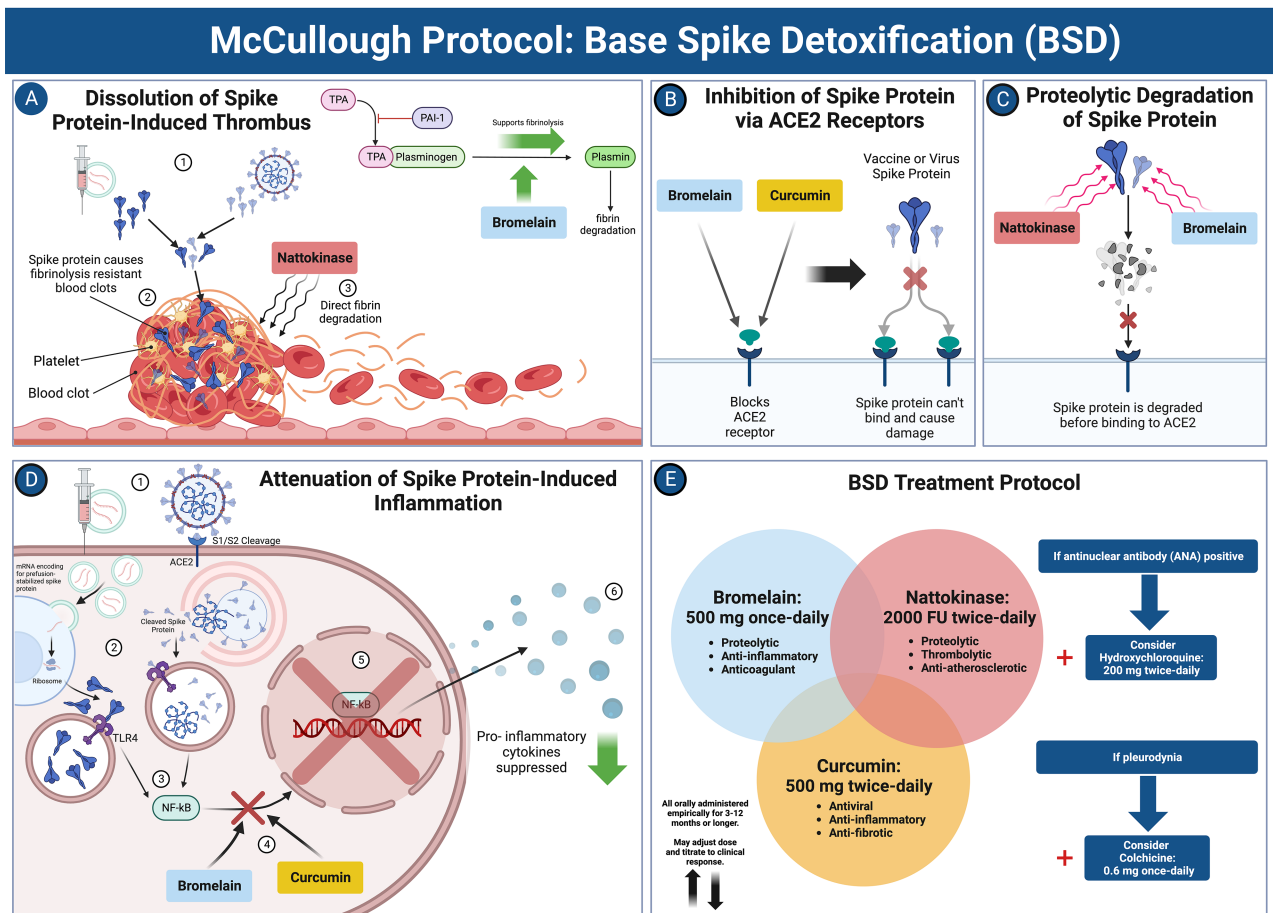
**Figure 2 Risk stratification approach for future cardiac arrest after coronavirus disease 2019 vaccination.** Green boxes indicate clinical features, test results, and patients at lower risk. Red and pink boxes show tests and results indicating higher risk. Hx: History; AU/mL: Antibody units per milliliter; HR: Heart rate; BP: Blood pressure; ECG: Electrocardiogram; Hs-troponin: High-sensitivity troponin; BNP/NT-proBNP: Brain natriuretic peptide and N-terminal proBNP; LVEF: Left ventricular ejection fraction; POTS: Postural orthostatic tachycardia syndrome (Created in BioRender, [Supplementary material](#)).

Small patches of myocardial inflammation, edema, or fibrosis may not be detectable by axial slices on cardiac MRI or autopsy[43,44]. Thus, the heart may appear normal on post-mortem examination and the final report may indicate death due to “natural causes” in a healthy patient with no antecedent disease. We believe these cases likely represent previously silent subclinical myocarditis and cardiac fibrosis which serves as the substrate for re-entrant ventricular tachycardia that degenerates to ventricular fibrillation and asystole in patients that do not receive prompt defibrillation.

When risk stratification indicates low-risk, primary care office management is suggested with McCullough Protocol: Base Spike Detoxification and adjunctive medications depending on the syndrome (Figure 3)[25]. For subclinical myopericarditis, oral colchicine 0.6 mg twice a day (BID) or once a day is indicated for at least one year[45]. For COVID-19 vaccine-induced postural orthostatic tachycardia syndrome, use of colchicine and nadolol 20-40 mg BID can be helpful [46]. In patients who screen as high-risk, Base Spike Detoxification[25] is also indicated. For those at very high risk for cardiomyopathy and or ventricular arrhythmias, formal cardiology consultation is suggested with the main goals of preventing heart failure and sudden death. Additionally, some patients may require ICD implantation if there are symptomatic arrhythmias, LGE > 15%, or genetic predictors such as pathogenic *SCN5A* mutations[37,47].

While this framework is designed for the general population, it is important to recognize that specific subgroups, such as older adults and individuals with pre-existing cardiovascular conditions, may require additional considerations. Older adults, for example, may benefit from more frequent monitoring and tailored preventive strategies to mitigate heightened risks associated with age-related cardiovascular changes. Similarly, patients with a history of heart disease should undergo customized evaluations and interventions to address their unique vulnerabilities. Incorporating these subgroup-specific approaches will further enhance the practical utility of the proposed framework across diverse patient populations. While the proposed risk stratification framework is already supported by substantial indirect evidence and sound clinical principles, it is important to note that real-world implementation data are currently lacking. This represents an opportunity to validate its effectiveness through future retrospective analyses and prospective clinical studies, which will not only strengthen its utility but also refine its practical application. By bridging this gap, the framework has the potential to become an indispensable tool for improving patient outcomes and guiding evidence-based care.





**Figure 3 McCullough protocol: Base spike detoxification.** A: Dissolution of spike protein-induced thrombus. Nattokinase directly degrades fibrinolysis-resistant fibrin (from spike protein), and bromelain upregulates fibrinolysis; B: Inhibition of spike protein via ACE2 receptors. Bromelain and curcumin block the ACE2 receptor, preventing spike protein from binding; C: Proteolytic degradation of spike protein. Nattokinase and bromelain degrade spike proteins, rendering them inactive; D: Attenuation of spike protein-induced inflammation. Bromelain and curcumin downregulate the nuclear factor kappa B signaling pathway induced by spike protein, leading to the suppression of inflammatory molecules; E: Base spike detoxification treatment protocol. The full treatment regimen and the addition of other compounds based on clinical indication are illustrated. TPA: Tissue plasminogen activator; PAI-1: Plasminogen activator inhibitor-1; ACE2: Angiotensin converting enzyme-2; NF-kB: Nuclear factor kappa B; S1/S2: Spike protein subunits S1/S2; TLR4: Toll-like receptor 4. Citation: Hulscher N, Procter BC, Wynn C, McCullough PA. Clinical Approach to Post-acute Sequelae After COVID-19 Infection and Vaccination. *Cureus* 2023; 15(11): e49204 Copyright ©The Author(s) 2023. Published by Springer Nature[25] (Supplementary material).

## CONCLUSION

In summary, we have proposed a risk stratification approach that addresses the clinical concern of future cardiac arrest following COVID-19 vaccination. The numerous studies highlighting serious cardiovascular safety concerns related to COVID-19 vaccines have raised public and physician awareness. It is prudent for each primary care physician to have a pre-established approach when addressing this issue in their practice.

## FOOTNOTES

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