



Myocarditis After Viral Illness and After Vaccination: Distinguishing the Two in a Sceptical Consultation

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Abstract

Myocarditis can follow either a viral illness or an mRNA COVID-19 vaccine dose, and both possibilities now regularly arise in the same clinic visit. This review sets the incidence and prognosis data for each side by side. A Nordic cohort of 23.1 million residents found an adjusted incidence rate ratio for myocarditis after a second mRNA-1273 dose of 6.57 (95% CI 4.64-9.28) overall and 13.83 (8.08-23.68) in men aged 16-24, versus 1.75 (1.43-2.14) and 5.31 (3.68-7.68) respectively for BNT162b2. A US PCORnet analysis of over 45 million patients found cardiac complications consistently more common after SARS-CoV-2 infection than after vaccination in every age and sex stratum studied: in men aged 18-29, 63.7 cases per 100,000 after infection versus 8.4 per 100,000 after a second dose. A self-controlled case series in 50 million people in England found 10.2 excess myocarditis/pericarditis cases per million after infection in 16-39-year-olds, rising to 18.1 per million in adults 40 and older, against 3.5 to 4.5 excess cases per million after a booster dose in the younger group. Prognosis also differs: in a Hong Kong cohort of 104 post-vaccination and 762 viral myocarditis cases, six-month mortality was 1.0% versus 11.0% (adjusted hazard ratio 0.1), and heart failure developed in 1.9% versus 12.2%. These are genuinely different risk-benefit pictures, not two versions of the same story, and a clinician facing a sceptical patient needs absolute numbers, not slogans, to represent either fairly. This review sets out those numbers and a practical, non-dismissive approach to the consultation in which they come up.

Keywords: Myocarditis; COVID-19 vaccines; mRNA vaccines; Risk communication; Vaccine hesitancy; Pharmacovigilance

Introduction

Myocarditis -- inflammation of the myocardium, usually lymphocytic, most often triggered by a viral illness or by the immune response that follows one -- was a familiar if uncommon diagnosis long before 2020, defined in detail by the European Society of Cardiology's own position statement on its aetiology, diagnosis, and management [2]. What changed with the COVID-19 pandemic is that a clinician now regularly

faces two distinct routes to the same diagnosis in the same patient population: myocarditis following SARS-CoV-2 infection itself, and myocarditis following an mRNA COVID-19 vaccine dose, a signal first described in case reports and formally reviewed by the American Heart Association within months of the vaccines' rollout [1].

This review sets the incidence, relative risk, and prognosis data for these two routes side by side, drawing on national

pharmacovigilance and regulatory analyses, two large self-controlled and cohort studies spanning tens of millions of people, and a direct comparative outcomes study, to give a cardiologist the numbers needed for an honest, non-dismissive conversation with a patient who has heard about vaccine-associated myocarditis and wants to know what it means for them specifically.

The clinical stakes of getting this comparison right run in both directions. Understating vaccine-associated myocarditis risk to a sceptical patient erodes trust the moment they read a different number elsewhere; understating post-infection myocarditis risk leaves the comparison that matters most to an individual deciding whether to vaccinate -- the risk of one route versus the other -- unstated. Absolute risk framing, rather than relative risk alone, has itself been shown to shift how patients weigh this trade-off, which is a further reason to have the actual numbers, not just the qualitative direction, ready at the bedside [9].

Material and Methods

This is a narrative review drawing on the American Heart Association's mechanistic and surveillance summary of vaccine-associated myocarditis, the European Society of Cardiology's position statement on myocarditis generally, the CDC's Advisory Committee on Immunization Practices update and its companion VAERS case-series analysis, a Nordic cohort study of 23.1 million residents, a self-controlled case series of 50 million people in England, a PCORnet cohort comparison of post-infection and post-vaccination cardiac complications in the United States, a Hong Kong comparative prognosis study, and a risk-communication trial addressing vaccine hesitancy directly, identified through the sources cited here and citation tracking between them, without a pre-registered protocol or systematic search string. All rates, incidence rate ratios, percentages, and confidence intervals reported here are taken directly from the cited sources; none has been invented, estimated, or extrapolated.

Results

How large is the vaccine-associated signal on its own terms

The first formal regulatory signal came from VAERS surveillance reviewed by the CDC's Advisory Committee on Immunization Practices in June 2021: among roughly 296 million mRNA doses administered by 11 June 2021, reporting rates were 40.6 cases per million second doses in males aged 12-29 years and 4.2 per million in females of the same age band, falling to 2.4 and 1.0 per million respectively in adults 30 and older [3]. Figure 1 sets these four strata out directly. Within the 12-29 age band, the signal was concentrated further: 62.8 cases per million second doses in males 12-17 years and 50.5 per million in males 18-24 years, with 92% of the 323 cases meeting the case definition presenting within 7 days of vaccination and 76% occurring after the second dose [3].

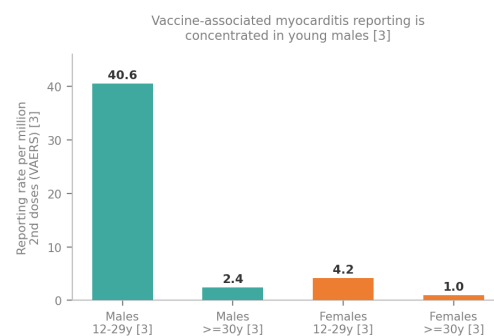


Figure 1: CDC/ACIP VAERS-based myocarditis reporting rates per million second doses, by age band and sex, through 11 June 2021 [3].

A subsequent, more granular analysis of 1,626 VAERS reports meeting case criteria between December 2020 and August 2021 refined the age pattern further: reporting rates per million second doses reached 70.6 in males 12-15 years and 105.9 in males 16-17 years, before falling to roughly 52.4-56.3 in males 18-24 years [4]. The peak in mid-adolescent boys rather than in the youngest eligible age band is a genuine, replicated feature of this signal, not an artefact of one dataset.

Two large studies outside the United States, using different designs, converge on the same qualitative picture with independently estimated relative risks. A Nordic cohort study linking vaccination and hospital records for 23.1 million residents of Denmark, Finland, Norway, and Sweden estimated an adjusted incidence rate ratio for myocarditis after a second homologous BNT162b2 dose of 1.75 (95% CI 1.43-2.14) overall, rising to 5.31 (3.68-7.68) in men aged 16-24; for mRNA-1273 the corresponding ratios were 6.57 (4.64-9.28) overall and 13.83 (8.08-23.68) in the same young male stratum, translating to 4 to 7 excess events per 100,000 vaccinees for BNT162b2 and 9 to 28 per 100,000 for mRNA-1273 within a 28-day window [5]. A self-controlled case series covering roughly 50 million people in England found a similar pattern for booster (third) doses in

16-39-year-olds: a relative incidence of 4.38 (2.59-7.38) for BNT162b2, equivalent to 3.5 excess cases per million, and 7.88 (4.02-15.44) for mRNA-1273, equivalent to 4.5 excess cases per million, in the six days after the booster [6]. Figure 2 places all of these relative-risk estimates on one comparative scale.

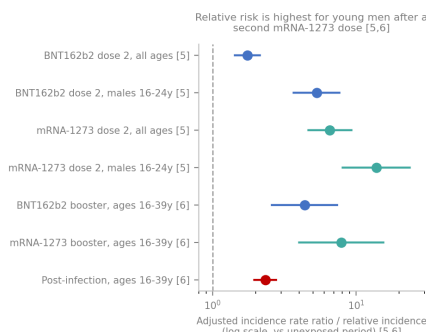


Figure 2: Adjusted incidence rate ratios and relative incidences for myocarditis: mRNA vaccine doses (Nordic cohort, England self-controlled case series) versus post-infection risk (England self-controlled case series) [5,6].

How the vaccine-associated signal compares with the infection-associated one

The same England self-controlled case series that estimated booster-dose risk also estimated post-infection risk directly, in the same population and with the same method: a relative incidence of 2.33 (1.96-2.76) in the 1-27 days after confirmed SARS-CoV-2 infection in 16-39-year-olds, equivalent to 10.2 excess myocarditis/pericarditis cases per million, rising to 18.1 excess cases per million in adults 40 and older, a materially higher excess than the 3.5 to 4.5 per million seen after a booster dose in the younger group [6]. A separate PCORnet analysis spanning January 2021 to January 2022 in the United States reached the same qualitative conclusion using an entirely different design -- a direct head-to-head comparison of cardiac complication rates after infection versus after vaccination within the same health-system network -- and found infection-associated risk higher than vaccination-associated risk in every age and sex stratum studied [7]. In males aged 18-29, cardiac complications occurred in 63.7 per 100,000 after infection versus 3.6 per 100,000 after a first dose and 8.4 per 100,000 after a second dose (risk ratio 7.6, 95% CI 3.7-15.7, comparing infection with second-dose vaccination); in males 30 and older the gap widened further, 63.0 per 100,000 after infection versus 1.2 per 100,000 after a second dose (risk ratio 50.8, 26.7-96.4) [7]. Figure 3 traces this divergence across age bands: post-infection risk stays roughly flat and high from adolescence into adulthood, while post-vaccination risk falls steeply with age.

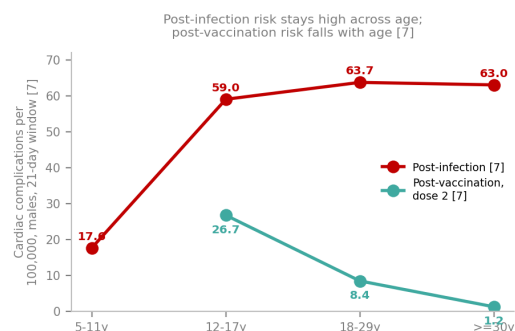


Figure 3: PCORnet cardiac complication rates per 100,000 males, post-infection versus post-vaccination (second dose), by age band, 21-day risk window [7].

The same PCORnet analysis found the same direction of effect, though a smaller absolute gap, in the youngest and in female strata: in males aged 12-17, infection carried 59.0 cases per 100,000 versus 3.3 after a first dose and 26.7 after a second dose (risk ratio 2.2, 1.2-4.0); in females 12 and older across the age bands studied, infection-associated rates of 11.9 to 61.7 per 100,000 exceeded vaccination-associated rates of 0.5 to 6.2 per 100,000 at every dose and age combination reported [7]. Table 1 summarises the design, population, and headline result of the five incidence-comparison studies reviewed here.

Study	Design / population	Headline comparison
Gargano et al [3]	US VAERS, ~296M doses	40.6/M males 12-29y (2nd dose) vs 2.4/M males >=30y
Oster et al [4]	US VAERS, 1,626 cases	70.6-105.9/M males 12-17y (2nd dose), peak at 16-17y
Karlstad et al [5]	Nordic cohort, 23.1M residents	IRR 6.57 (mRNA-1273) vs 1.75 (BNT162b2), dose 2, all ages
Stowe et al [6]	England SCCS, ~50M people	Booster RI 4.38-7.88 (16-39y) vs infection RI 2.33 (16-39y), 2.33-higher in >=40y
Block et al [7]	US PCORnet, direct comparison	Infection > vaccination in every age/sex stratum studied

Table 1: Design and headline result of the five incidence-comparison studies reviewed here [3,4,5,6,7].

The two kinds of number reported across these studies -- an adjusted incidence rate ratio or relative incidence, and an excess-cases-per-population estimate -- answer different questions and both matter in consultation. The relative measure answers how many times more likely myocarditis becomes in the days after the exposure compared with an unexposed period, which is the number that best conveys how concentrated a signal is in a particular age and sex group; the excess-cases measure answers how many additional cases would be expected if an entire population of that size were exposed, which is the number that best answers a patient's more practical question of how likely this is to happen to them specifically [5,6]. A large relative incidence built on a tiny

baseline rate, as in the mRNA-1273 booster comparison, can still translate into a modest absolute number of extra cases per million people, and presenting only the relative figure risks leaving a sceptical patient with an exaggerated sense of their own risk, just as presenting only the excess-cases figure risks understating how unevenly that risk is distributed by age and sex [5,6,7].

Does the mechanism explain the difference, and does prognosis differ too

Figure 4 sets out a practical bedside approach that does not depend on resolving the mechanistic question before acting: standard evaluation with ECG, troponin, and inflammatory markers remains the same regardless of the suspected trigger, and a normal set of these makes myocarditis of either cause unlikely [1,3]. The proposed mechanisms nonetheless differ. Vaccine-associated myocarditis is hypothesised to follow a spike-antigen-directed immune response, with experimental evidence of antibody cross-reactivity against structurally similar self-antigens such as alpha-myosin in a subset of cases, and a possible role for dysregulated innate immune activation in genetically susceptible individuals [1]. Classical post-viral myocarditis, by contrast, is understood to combine direct viral cardiomyocyte injury with secondary immune-mediated damage, often as part of a more systemic illness [2]. Figure 5 sets these two upstream pathways out schematically, converging on a broadly similar lymphocytic inflammatory endpoint on biopsy or MRI despite their different triggers.

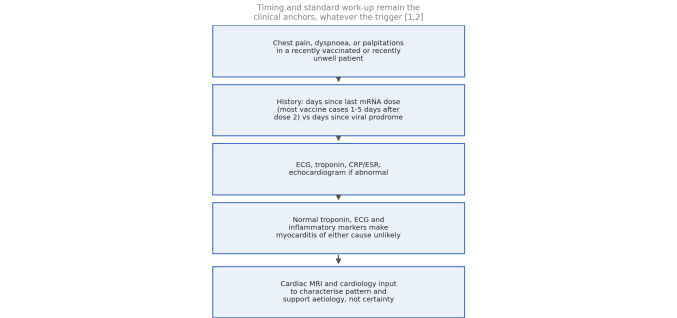


Figure 4: A practical bedside approach to suspected myocarditis, applicable regardless of whether a vaccine dose or a viral illness is the suspected trigger [1,3].

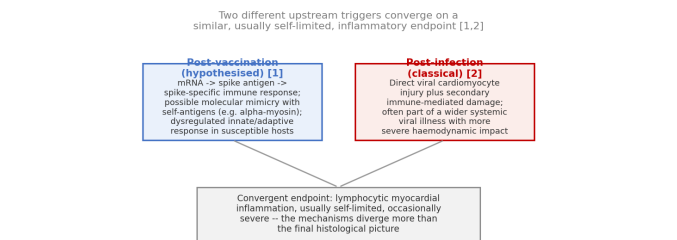


Figure 5: Hypothesised mechanistic pathways for post-vaccination versus post-infection myocarditis, converging on a similar inflammatory endpoint [1,2].

Whether these different upstream mechanisms produce a different clinical course was tested directly in a Hong Kong Hospital Authority cohort comparing 104 patients with post-vaccination myocarditis against 762 patients with viral myocarditis drawn from the same health system's records between 2000 and 2019 [8]. Six-month mortality was 1.0% (1/104) in the vaccine group versus 11.0% (84/762) in the viral group, an adjusted hazard ratio of 0.1, equivalent to a roughly 92% lower risk of death [8]. Heart failure developed in 1.9% (2/104) of the vaccine group versus 12.2% (93/762) of the viral group, and dilated cardiomyopathy in 1.0% (1/104) versus 3.7% (28/762) [8]. Figure 6 sets these three outcomes out together, and Table 2 restates the same comparison in tabular form alongside the adjusted hazard ratio.

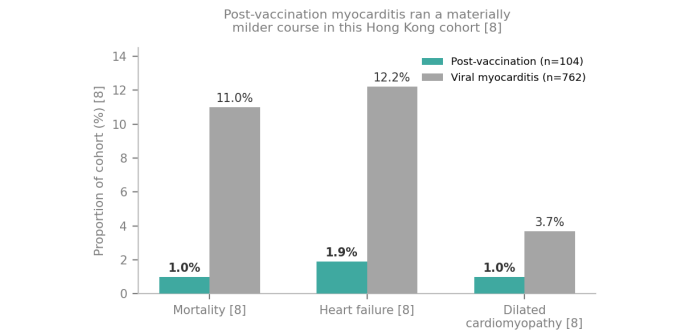


Figure 6: Six-month mortality, heart failure, and dilated cardiomyopathy: post-vaccination versus viral myocarditis, Hong Kong Hospital Authority cohort [8].

Outcome (6 months)	Post-vaccination (n=104) [8]	Viral myocarditis (n=762) [8]	Adjusted HR
Mortality	1.0% (1/104)	11.0% (84/762)	0.1
Heart failure	1.9% (2/104)	12.2% (93/762)	--
Dilated cardiomyopathy	1.0% (1/104)	3.7% (28/762)	--

Table 2: Prognosis comparison between post-vaccination and viral myocarditis, Hong Kong Hospital Authority cohort [8].

A single comparative cohort cannot settle whether this gap reflects the mechanism itself, earlier detection of vaccine-associated cases through active post-authorisation surveillance, or a genuinely less severe historical comparator population -- a point returned to in Study limitations below -- but the size of the difference (roughly an order of magnitude in mortality) makes it unlikely to be explained by ascertainment bias alone.

What strengthens confidence in the qualitative pattern reported here -- infection-associated myocarditis risk exceeding vaccination-associated risk in almost every comparison attempted -- is that it was not produced by one surveillance system using one method. It emerges independently from US passive pharmacovigilance (VAERS) [3,4], from active linkage of national vaccination and hospital registries in four Nordic countries [5], from a self-controlled case series design in England that serves as its own control for unmeasured

confounding [6], from a direct multi-site electronic health record comparison in the United States [7], and from a comparative outcomes cohort in Hong Kong using entirely different data infrastructure [8]. Four different countries, four different data systems, and at least three different epidemiological designs converging on the same direction of effect is a stronger basis for a consultation conversation than any single study, however large, could provide on its own.

Discussion

Taken together, these data describe two genuinely different risk-benefit pictures rather than two versions of the same story. Vaccine-associated myocarditis is real, concentrated in young men in the days after a second (or booster) dose, more frequent after mRNA-1273 than BNT162b2, and detectable at rates of tens of cases per million doses in the highest-risk stratum [3,4,5]. Post-infection myocarditis is also real, less concentrated by age and sex, and larger in absolute terms across almost every comparison attempted directly against vaccination in the same population [6,7]. Where the two have been compared for clinical course rather than only incidence, the vaccine-associated cases ran a substantially milder course in the one large comparative cohort available [8]. None of these three findings cancels out the others, and a clinician who leads with only one of them -- the vaccine signal alone, or the infection comparison alone -- is not being dishonest, but is giving an incomplete answer to a patient who is entitled to all three.

For the sceptical patient in the room, the operational lesson from the risk-communication literature is that framing matters as much as content: a trial testing a relative-risk framing tool against standard CDC messaging found it shifted stated vaccination intent significantly more than the standard messaging did, without requiring the clinician to minimise the genuine vaccine-associated signal [9]. Practically, this supports giving the patient in front of them the number for their own age and sex band -- not a population-average figure that may over-represent their risk if they are an older woman, or under-represent it if they are a teenage boy -- set beside the equivalent post-infection number from the same or a comparable study, rather than a single relative-risk statistic detached from either denominator. Table 3 collects these age- and sex-specific absolute numbers in one place for exactly that purpose.

Patient profile	Post-vaccination risk (per million, dose 2)	Post-infection risk (per 100,000)
Male, 12-17y	62.8-105.9 [3,4]	59.0 [7]
Male, 18-29y	50.5-56.3 [3,4]	63.7 [7]
Male, >=30y	2.4-12.0 [3,7]	63.0 [7]
Female, 12-29y	4.2 [3]	11.9-61.7 [7]
Female, >=30y	1.0 [3]	11.9-61.7 [7]

Table 3: Absolute risk figures for use directly in consultation, by age and sex; units differ by source (per million doses versus per 100,000 person-time) and are kept as reported rather than rescaled, to avoid implying a precision the underlying data do not have [3,4,7].

This framing should not be used to talk a genuinely hesitant patient out of a legitimate concern. A young male patient asking specifically about second-dose risk is asking a real question with a real, non-trivial answer -- tens of cases per million is not zero, and for a small number of families that number will matter more than any comparison to infection risk. The honest response is not to reframe every such question as a false equivalence with infection risk, but to give the vaccine-specific number plainly, alongside the infection-specific number for the same demographic, and let the patient weigh both against their own circumstances -- prior infection history, occupational exposure, local transmission intensity -- none of which a population-average incidence figure can capture on its own.

Study limitations

This is a narrative review, not a systematic review with a pre-registered protocol, and its quantitative conclusions rest on a small number of large studies rather than an exhaustive search of the myocarditis surveillance literature. The studies compared here use different case definitions, different denominators (per million doses versus per 100,000 person-time), different follow-up windows (6 days, 21 days, 27 days, 28 days), and different populations (US VAERS passive surveillance, Nordic and English linked national registries, a US multi-site electronic health record network), so the absolute numbers in Table 3 are not directly poolable and are reported here as each study defined them, not rescaled to a common metric [3,4,5,6,7].

The single comparative prognosis study reviewed here draws its viral-myocarditis comparator from a 2000-2019 historical cohort in one health system, predating the intensive active surveillance, low diagnostic threshold, and early cardiology involvement that characterised the post-vaccination cases identified from 2021 onward; some of the mortality difference reported may reflect earlier detection of milder vaccine-associated cases rather than a true difference in disease severity at a matched stage of illness, and this review cannot separate those two explanations from a single non-randomised cohort comparison [8]. VAERS-based

reporting rates are also subject to the general limitations of passive surveillance -- under-reporting, reporting-rate changes over time as clinician awareness grew, and no unvaccinated comparator group within the same dataset -- which is why this review leans on the Nordic, English, and PCORnet studies, which used active linkage or a direct comparator group, for the head-to-head comparisons rather than on VAERS rates alone [3,4,5,6,7].

Finally, the risk-communication trial cited here tested a specific relative-risk visualisation tool in US survey respondents recruited online in 2022, a design that measured stated vaccination intent rather than a clinic consultation outcome or an actual vaccination decision, and its result should be read as evidence that framing can move stated intent, not as a validated script for the cardiology consultation itself [9]. No study reviewed here directly measured whether presenting both post-vaccination and post-infection myocarditis risk together, in the way Table 3 does, changes what a sceptical patient ultimately decides; that comparison remains a genuine gap in the literature rather than a settled finding this review can report.

Conclusion

Myocarditis after mRNA COVID-19 vaccination and myocarditis after viral, including SARS-CoV-2, infection are both real, both quantifiable, and not interchangeable in either their incidence or their prognosis. The vaccine-associated signal is concentrated in young men in the days after a second or booster dose, larger for mRNA-1273 than BNT162b2, and detectable at tens of cases per million in the highest-risk group; the infection-associated risk is less concentrated by demographic group and larger in absolute terms in almost every direct comparison attempted; and where clinical course has been compared directly, the vaccine-associated cases have run a materially milder course. A cardiologist facing a sceptical patient serves them best not by picking one of these three findings to lead with, but by having the age- and sex-specific absolute numbers for both routes ready, and by treating the patient's question as a real one deserving a complete, undismissive answer.

A final practical note for the consultation itself: none of the numbers above requires the clinician to predict what an individual patient will decide, only to represent both routes fairly. A young man asking why he should accept even a small vaccine-associated risk when he expects to survive infection anyway is asking a coherent question, and the honest reply draws on Table 3 directly -- his post-vaccination risk of 50.5 to 105.9 per million is real and worth stating plainly, but so is his post-infection risk of roughly 60 per 100,000, which is 600 to 1,200 per million on the same scale, and so is the milder average clinical course reported for the vaccine-associated cases in the one large comparative cohort available [4,7,8]. An

older woman asking the same question is in a different position again: her post-vaccination risk sits below 5 per million on every study reviewed here, well under her post-infection risk on the same comparisons, and the size of that gap, not a general reassurance about vaccine safety, is what should carry the conversation [3,7]. Restating the comparison in the patient's own units, rather than leaving her to convert a relative-risk ratio or a rate-per-100,000 into a rate-per-million herself, is a small step that the risk-communication literature suggests makes a measurable difference to how the numbers land [9].

1. Bozkurt B, Hotez PJ, Kamat SA. "Myocarditis With COVID-19 mRNA Vaccines". *Circulation* 144(2021):e123-e135.
2. Caforio ALP, Pankuweit S, Arbustini E, et al. "Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: a position statement of the European Society of Cardiology Working Group on Myocardial and Pericardial Diseases". *Eur Heart J* 34(2013):2636-2648.
3. Gargano JW, Wallace M, Hadler SC, et al. "Use of mRNA COVID-19 Vaccine After Reports of Myocarditis Among Vaccine Recipients: Update from the Advisory Committee on Immunization Practices -- United States, June 2021". *MMWR Morb Mortal Wkly Rep* 70(2021):977-982.
4. Oster ME, Shay DK, Su JR, et al. "Myocarditis Cases Reported After mRNA-Based COVID-19 Vaccination in the US From December 2020 to August 2021". *JAMA* 327(2022):331-340.
5. Karlstad O, Hovi P, Husby A, et al. "SARS-CoV-2 Vaccination and Myocarditis in a Nordic Cohort Study of 23 Million Residents". *JAMA Cardiol* 7(2022):600-612.
6. Stowe J, Miller E, Andrews N, Whitaker HJ. "Risk of myocarditis and pericarditis after a COVID-19 mRNA vaccine booster and after COVID-19 in those with and without prior SARS-CoV-2 infection: a self-controlled case series analysis in England". *PLoS Med* 20(2023):e1004245.
7. Block JP, Boehmer TK, Forrest CB, et al. "Cardiac Complications After SARS-CoV-2 Infection and mRNA COVID-19 Vaccination -- PCORnet, United States, January 2021-January 2022". *MMWR Morb Mortal Wkly Rep* 71(2022):517-523.
8. Lai FTT, Chan EWY, Huang L, et al. "Prognosis of Myocarditis Developing After mRNA COVID-19 Vaccination Compared With Viral Myocarditis". *J Am Coll Cardiol* 80(2022):2255-2265.
9. Byerley CO, Horne D, Gong M, et al. "An effective COVID-19 vaccine hesitancy intervention focused on the relative risks of vaccination and infection". *Sci Rep* 14(2024):7419.

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