1-1

The Failings of the NHS Can't Explain the Dramatic Rise in Heart Failure Deaths

NICK RENDELL 15 JULY 2023

Deaths from heart failure in the 15 weeks from week ending March 24th 2023 to the end of June 2023 were 27% higher than the level expected for the same period in 2020. Why?

The Office for Health Improvement and Disparities has produced the data for England. They show that from week 12 to week 26 of 2020 there were expected to be 16,752 heart failure deaths. Whereas in 2023 from week 12 to week 26 17,825 deaths were expected. For some reason we expect 6% more people to die of heart failure now than we did three years ago, even more of a surprise when you consider that the 'pandemic' was supposed to have cut a swathe through the 'dry tinder'! In the event 21,222 people died from heart failure in England during this period in 2023.

Figure 1 shows weekly deaths from heart failure since week 12 of 2020. You can see, with or without Covid linked deaths, there has been a significant and sustained increase.

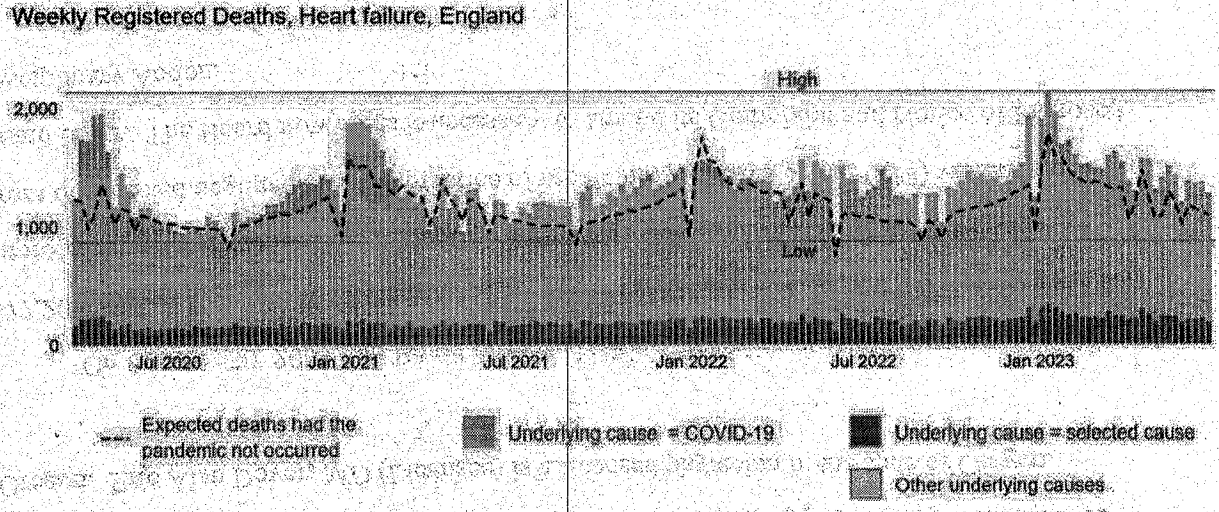

Figure 1

An article published in the *Telegraph* by consultant cardiac surgeon Julian Gaer focuses on the failings of the NHS to provide timely and effective treatment for cardiac patients. It's an interesting read but, to my mind somewhat partial, though he's very clear about where the blame lies:

I do blame a system that has allowed us to reach the point of having dangerous shortages of permanent skilled staff and dilapidated facilities habitually operating at 100% of theoretical capacity (something the NHS hierarchy persists in believing demonstrates value-for-money). I blame the fact that the U.K. has fewer hospital beds per capita than all but five of 38 OECD countries (Mexico, Costa Rica, Colombia, Chile, Sweden). France has three times more hospital beds per capita than the U.K. and Germany four times. Germany has 29 intensive care beds per 100,000 population, whereas the U.K. has seven. Little wonder therefore that Germany recorded just over 2,000 Covid deaths per million of the population, for our 3,000-plus.

So, according to Mr. Gaer it's all about money and resources. But, let's just see where Mr. Gaer gets his numbers from in relation to Covid deaths in Germany and the U.K.

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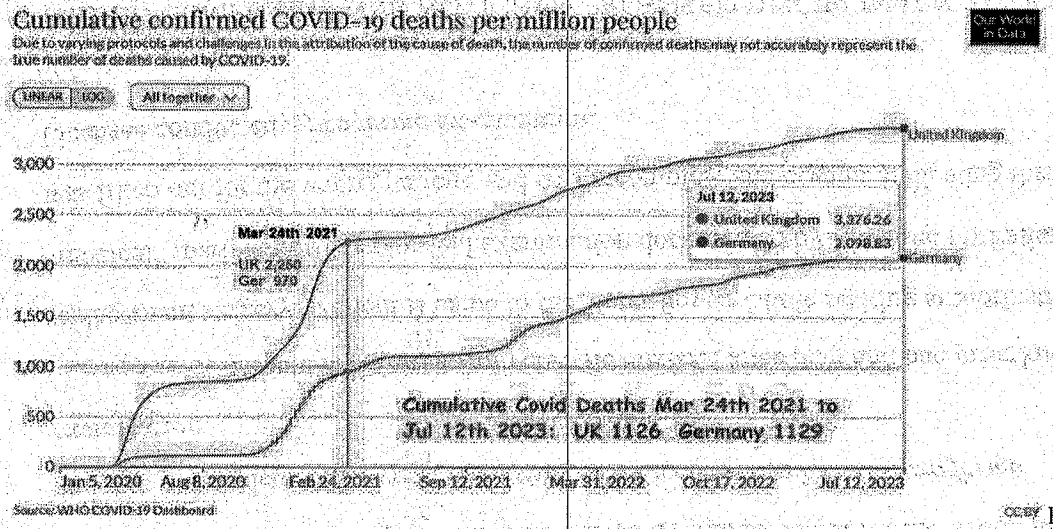
Figure 2 is taken from Our World in Data and shows cumulative Covid deaths from the beginning of January 2020 to July 12th 2023, the day before Mr. Gaer's article was published.

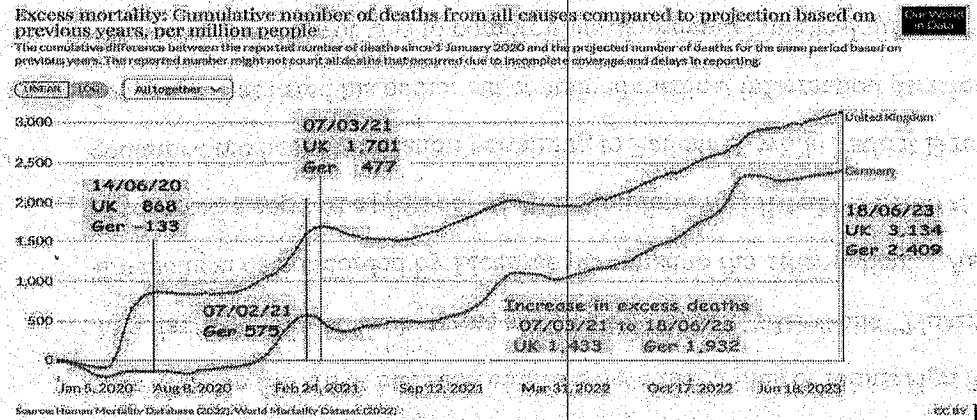


CC BY Figure 2

You can see that, as Mr. Gaer asserts, Covid deaths in Germany over the past three-and-a-half years are recorded as about 2,000 per million compared to the U.K.'s 3,376 per million. However, this variance is wholly due to the course of the pandemic from March 2020 to March 2021, by which point all the variance we see now had already occurred. Is he claiming that Germany's relatively low number of deaths during the first year of the pandemic was wholly down to its treatment protocols, higher number of ICU beds and the organisation of its healthcare systems? If that's the case, why did Germany's Covid deaths match ours for the subsequent two-and-a-half years? Except for Sweden and Belarus, all European countries followed the same public health policies but with hugely variable outcomes, Finland saw one sixth of the Covid deaths that Germany did. Was this because Finland's health service was superior to Germany's? Of course not.

Rather than just looking at Covid deaths let's look at all-cause excess deaths and compare the U.K. to Germany. Most experts agree that all-cause deaths is a far better measure of how a healthcare system works, rather than just the narrow focus on Covid deaths.

Again, a chart from Our World in Data illustrates perfectly that since the beginning of 2020 cumulative excess deaths in the U.K. stand at about 3,134 per million, whereas in Germany they're 725 per million lower at 2,409 per million. However, all this difference and more was accounted for in the first Covid wave. By June 14th 2020 cumulative all-cause excess deaths in the U.K. were 1,001 per million higher than in Germany (868 – 133 = 1,001).



CC BY Figure 3

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Since the U.K.'s March 7th 2021 pandemic peak, all-cause excess deaths in the U.K. have been lower than in Germany. While we've recorded 1,433 per million excess deaths the Germans have seen 35% more at 1,932 per million. To what does Mr. Gaer attribute the failure of Germany's healthcare system over this period?

It doesn't look like it's British exceptionalism that accounts for the relative performance of the U.K. or German healthcare systems. In the same way that when you look a little more closely the apparent variance between Germany and the U.K. becomes obscured, so it is with excess heart deaths. Mr. Gaer would appear to attribute the rise in heart failure deaths to NHS failings in treatment and delivery. If this were the case, why don't we see the same thing with cancer deaths?

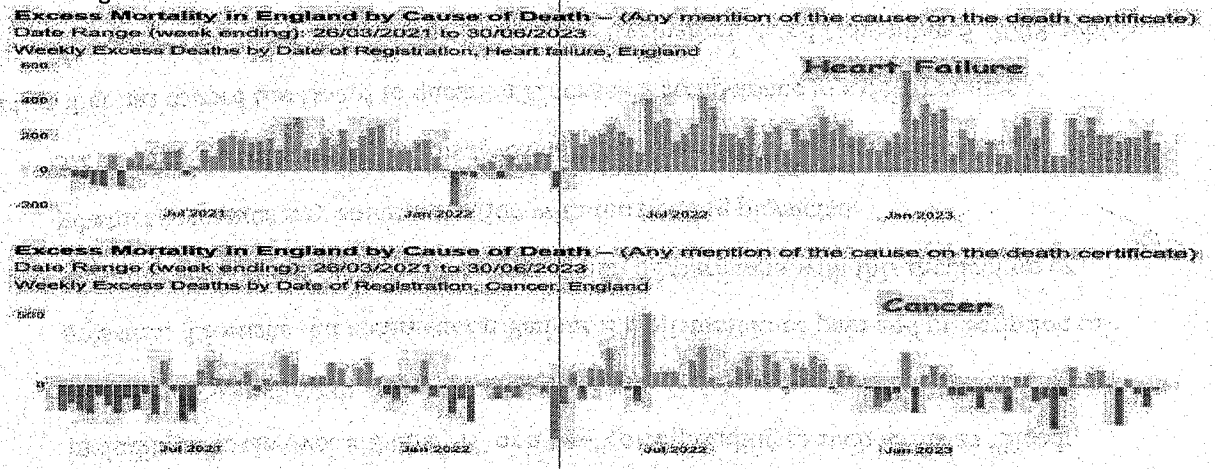


Figure 4

Figure 4 reproduces charts from the Office for Health Improvements and Disparities showing excess deaths from heart failure and cancer from week ending March 26th 2021 to the end of June 2023. Cancer deaths are tracking the expected rate while heart failure deaths are dramatically elevated.

Figure 5 compares heart failure deaths with that other big killer, dementia and Alzheimer's. Surely, if the NHS is letting down heart failure patients it must also be failing these patients?

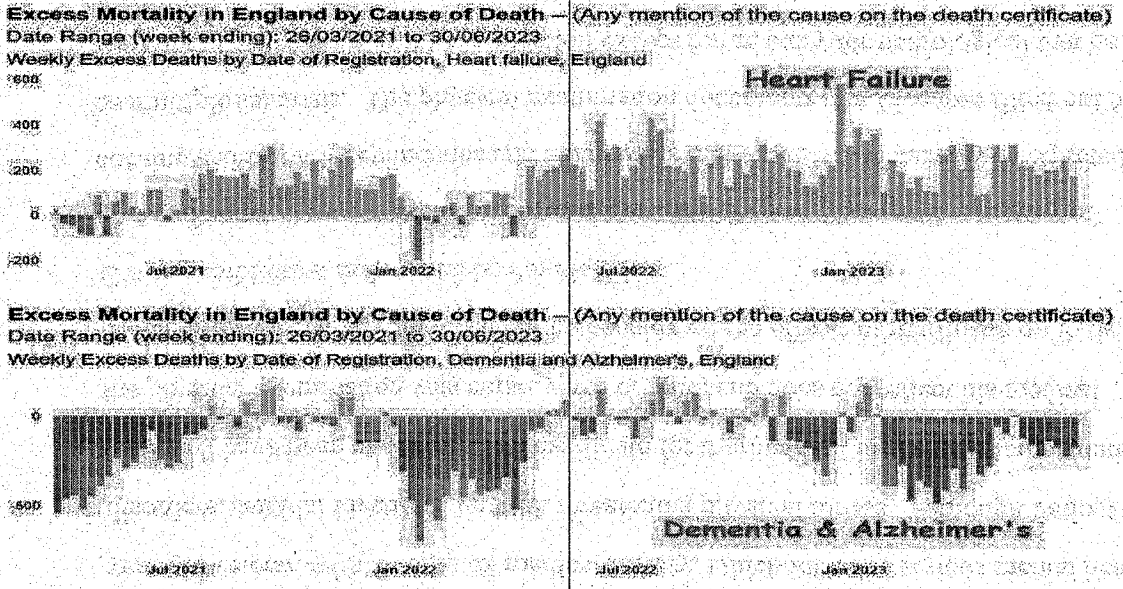


Figure 5

1-4

It would seem not. Over the past two years or so dementia and Alzheimer's deaths have been well below the expected rate.

Figure 6 illustrates another issue that's been too much ignored. Since the beginning of the pandemic, with the exception of the two short spikes in April of 2020 and January of 2021, excess deaths in hospitals haven't been exceptional. However, excess deaths 'at home' shot up and have consistently stayed high.

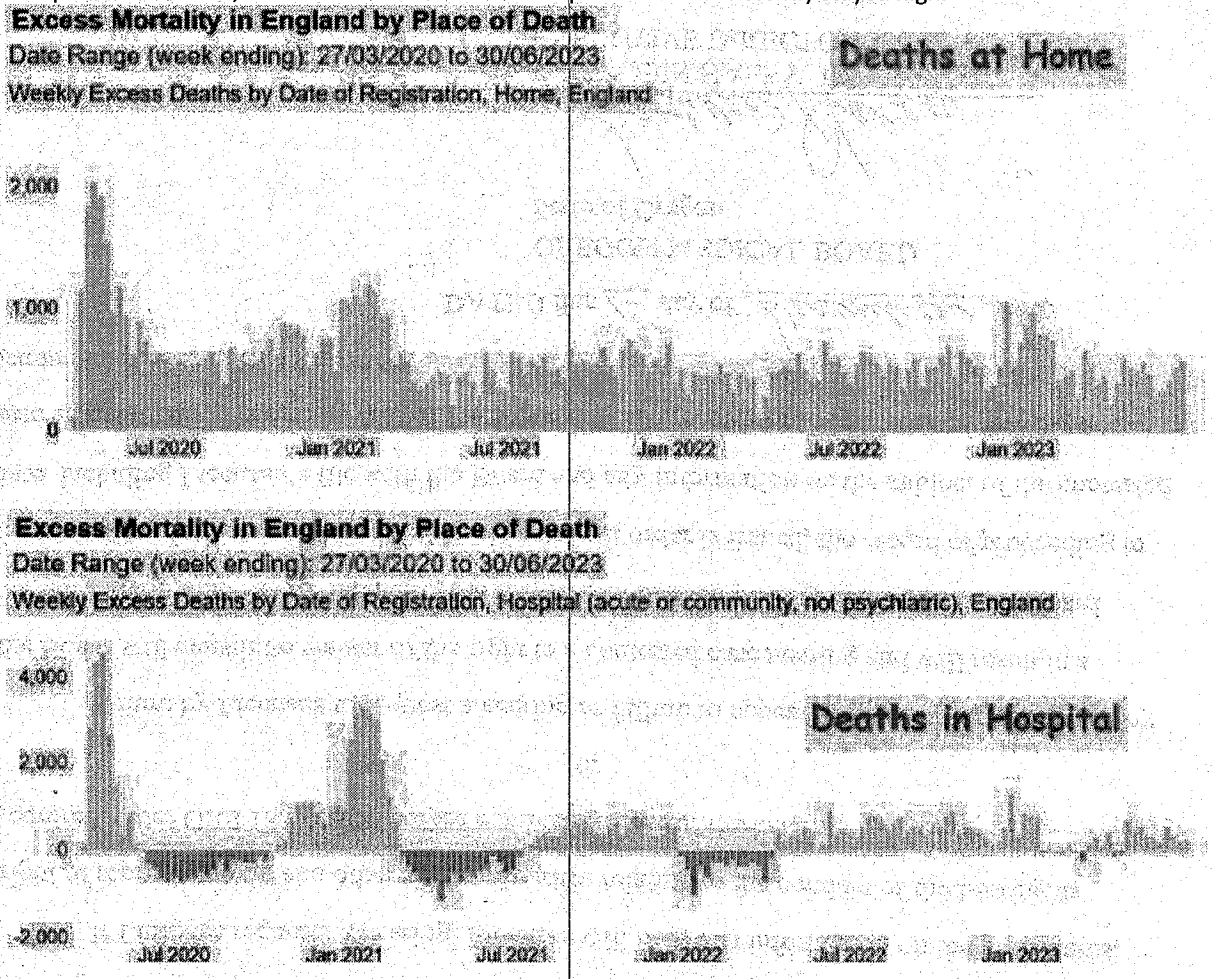


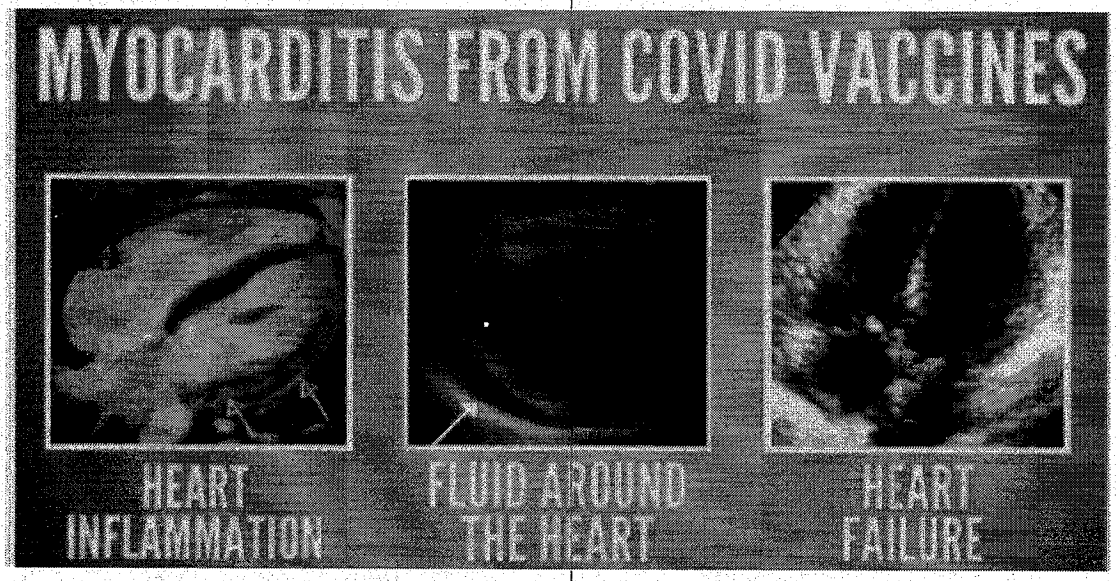
Figure 6

Whatever is causing the alarmingly elevated rate of heart failure deaths, it is not currently causing elevated deaths from cancer or dementia and thus it is hard to see how the primary driver can be the failings of the health service, however acute. For the same reason it is hard to see how the explanation can lie in an ageing population. Something else has seriously impacted on heart health in particular over the past three years.

2-1

Leaked Report Confirms Myocarditis Cases Spiked in US Military Following Forced COVID Shots – As Gateway Pundit Has been Reporting now for Over 2 Years

Jim Hoft Jul. 22, 2023



Back in September 2021 the Gateway Pundit reported on a shocking new study that was conducted by researchers at Canada's University of Ottawa Heart Institute found that one out of every thousand(1/1000) mRNA Covid-19 vaccinations causes heart inflammation(myopericarditis) to develop rapidly in otherwise healthy individuals.

The study looked at over 32,000 individuals who had received either the Pfizer-BioNTech or Moderna vaccines and monitored them for heart-related conditions between June 1, 2021, and July 31, 2021.

32 of the study subjects were admitted into the hospital with heart and chest-related symptoms.

"There were 15,997 doses of Moderna vaccine, and 16,382 doses of Pfizer vaccine administered over the study period, for a total of 32,379 doses. Note that these numbers represent a mixture of first and second doses.

Therefore, if our cohort captured all cases in the Ottawa area, then the incidence of myocarditis would be 0.1% of all vaccine doses (32 cases/32,379 doses x 100), or 10 cases of myocarditis for every 10,000 doses of vaccine."

1-32

Researchers found that the symptoms show up extremely quickly after vaccination, usually after the patient's second dose. On average, people who were affected developed the condition after just 1.5 days.

They also found that men are at a much greater risk than women – only 2 women experienced heart inflammation.

Now a new leaked military study finally admits that myocarditis cases spiked following the forced COVID shots in the US Military by at least 151%.

The Epoch Times reported:

Cases of myocarditis soared among U.S. service members in 2021 after the COVID-19 vaccines were rolled out, a top Pentagon official has confirmed.

There were 275 cases of myocarditis in 2021—a 151 percent spike from the annual average from 2016 to 2020, according to Gilbert Cisneros Jr., undersecretary of defense for personnel and readiness, who confirmed data revealed by a whistleblower earlier this year.

The COVID-19 vaccines can cause myocarditis, a form of heart inflammation that can lead to mortality, including sudden death. COVID-19 also can cause myocarditis.

The diagnosis data comes from the Defense Medical Epidemiology Database.

Mr. Cisneros provided the rate of cases per 100,000 person-years, a way to measure risk across a certain period of time. In 2021, the rate was 69.8 among those with prior infection, compared to 21.7 among members who had been vaccinated.

The Gateway Pundit has reported on this horrible vaccine response in young adults for years now, since September 2021:

3-1

US Military Confirms Myocarditis Spike After COVID Vaccine Introduction

JUL 22, 2023 Zachary Stieber

Cases of myocarditis soared among U.S. service members in 2021 after the COVID-19 vaccines were rolled out, a top Pentagon official has confirmed.

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Mr. Cisneros provided the rate of cases per 100,000 person-years, a way to measure risk across a certain period of time. In 2021, the rate was 69.8 among those with prior infection, compared to 21.7 among members who had been vaccinated.

"This suggests that it was more likely to be [COVID-19] infection and not COVID-19 vaccination that was the cause," Mr. Cisneros said.
No figures were given for members who had been vaccinated but were also infected. The total rate, 20.6, also indicates that some members weren't included in the subgroup analysis.

Sen. Ron Johnson (R-Wis.), who has been investigating problems with the database, questioned how the military came up with the figures.
"It is unclear whether or how it accounted for service members who had a prior COVID-19 infection and received a COVID-19 vaccination," Mr. Johnson wrote to Mr. Cisneros.

Department of Defense (DOD) officials didn't respond to a request for comment.

Mr. Johnson asked for the information no later than Aug. 2.

Dr. Peter McCullough, a cardiologist and president of the McCullough Foundation, looked at the newly disclosed data.
"The large increase in myocarditis cases in our military in 2021 was most likely due to ill-advised COVID-19 vaccination," he told The Epoch Times via email, pointing to a study from Israel that found no increase or myocarditis in COVID-19 patients.

Some other papers have found COVID-19 vaccines increase the risk of myocarditis. COVID-19 has been linked elsewhere to myocarditis, although the vaccines have never prevented infection and have become increasingly ineffective against it.

The military encouraged COVID-19 vaccination after U.S. regulators cleared the vaccines for use in late 2020. Military officials were among the first in the world to raise concerns about myocarditis after vaccination and published an early case series of 22 previously healthy members who suffered myocarditis within four days of receiving a COVID-19 vaccine. U.S. officials have since said the vaccines definitely cause myocarditis.

U.S. Defense Secretary Lloyd Austin mandated the vaccines in 2021, a requirement that remained in place until Congress forced its withdrawal.

Repeated Changes

Military officials have struggled to provide accurate data on 2021 diagnoses.
Whistleblowers revealed in 2021 that myocarditis, as reflected in the Defense Medical Epidemiology Database (DMED), had soared to 2,868 percent higher than the average from 2016 to 2020. They downloaded the data in August 2021.

The number of 2021 myocarditis diagnoses, though, had plummeted from 1,239 to 263 when the data was downloaded later, prompting concerns of manipulation.

Military officials said they reviewed the data and found it was "faulty." They said the data for the years 2016 to 2020 were "corrupted" during a "database maintenance process," which resulted in the display of only 10 percent of the actual medical encounters for that time period.

Officials told Mr. Johnson in 2022 that the problem had been fixed. The fix significantly changed the records. Instead of a 2,181 percent increase in hypertension in 2021, for instance, the increase was just 1.9 percent. Female infertility, instead of increasing 472 percent, increased 13.2 percent.

3-2

The updated percentages, though, were called into question when another whistleblower looked at the database in 2023 and found they were different.

Testicular cancer, initially pegged as increasing 369 percent, was placed at 3 percent by the military. But the actual increase was 16.3 percent, the whistleblower found. Pulmonary embolism was among the other conditions that occurred more often in 2021 than the military had conveyed.

The whistleblower alerted Mr. Johnson, the top Republican on the Senate Subcommittee on Investigations, who asked military officials for answers.

Mr. Cisneros acknowledged that the data given to the senator was incomplete. He said the change stemmed from December 2021 figures not being available when the corrected data was offered. There was a data "lag by about three months," meaning the data wasn't available in February 2022, when officials provided Mr. Johnson with the corrected data, Mr. Cisneros said.

Pentagon officials replicated the analyses from the whistleblower and found the data "are similar" to the data the whistleblower sent to Mr. Johnson, Mr. Cisneros said.

Military officials hadn't previously mentioned any data lag previously while communicating with Mr. Johnson or the public, and they didn't incorporate the available data when they sent him another missive in mid-2022.

"Without the whistleblower's disclosure, I doubt DOD would have ever acknowledged that it provided incomplete information to my office in February 2022 and again in July 2022," Mr. Johnson said.

He said the DOD had demonstrated "a complete disregard for transparency" and urged officials to make clear whether it has investigated whether any of the medical conditions for which diagnoses spiked are associated with the vaccines.

4-1

Swiss study: heart injuries from COVID vaccine 3000x higher than thought

DAVID STROM July 26, 2023

It is a small study, but a very disturbing one.

We keep being told that injury to the heart from the COVID vaccine is very rare, but a study done in Basel Switzerland indicates that the rate of subclinical myocarditis after the COVID vaccine is hardly rare at all.

In fact, in a study with only 777 participants with a median age of 37--all medical professionals getting the COVID vaccine–the incidence of elevated cardiac enzymes 3 days after injection was pretty substantial, at almost 3%.

The CDC did a study and from that, they claimed the rate was 0.001%, or one out of 100,000.

2.8% is a lot higher than 0.001%. Another 0.3% had "probable myocarditis," putting the total at over 3%. That is 3000 times higher than the US government claimed.

In this small study, nobody had serious complications, but with a myocarditis complication rate of 3%, you would have to expect that giving out hundreds of millions of doses is a pretty risky proposition.

I think we all knew that already, but this study seems to put the nail in the coffin of "vaccine injuries are super rare" from COVID-19 shots.

Oops. Who could have guessed?

One oddity was that the rate of myocarditis among the participants was heavily weighted toward women, not men. That could be an artifact of the sample, or it could indicate that women are more likely to get a complication, but the complications are more likely to be serious among men.

One reason the researchers posit for the vast difference between their results–which are based upon blood tests looking for cardiac enzymes in all participants–and the commonly asserted claim that vaccine-induced myocarditis is rare is that the only cases that are diagnosed without looking specifically for it are severe.

In other words, most people don't go to the doctor until there is a serious problem, so many people suffer from myocarditis without ever getting diagnosed.

This suggests that there is a very large group of people who were afflicted but never treated. This in most cases would not be a huge problem, as the inflammation resolves on its own, but in some cases, actual damage to the heart was done without it ever being caught.

Another variable, not mentioned, is that myocarditis complications are more common in young men, and this study skewed both female and middle-aged professionals. Given the cohort studied, one would expect them to be not entirely representative of the population as a whole. They are likely wealthier, healthier, and moderately older than the population as a whole.

In any case, this study sheds quite a light on just how deceptive the CDC, the FDA, and NIAID have been about vaccine safety. And also how intentionally ignorant they have chosen to be. This was not a complicated study to do. The researchers chose a cohort easy to recruit, tested them both before and after vaccination to create a baseline and comparison, and analyzed the data.

Easy peasy. Not even that costly. If you wanted to know the actual numbers of people with heart damage post-vaccine, this was an easy-to-construct and interpret study, and you can get results very quickly.

Why didn't our public health officials do it then? Why did it take a hospital in Switzerland to come up with the idea and execute it? In 2023, no less. **Being off by a factor of 3000 is a big deal.** It is a very big deal.

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5-1

Sex-specific differences in myocardial injury incidence after COVID-19 mRNA-1273 booster vaccination

European Journal of Heart Failure (2023)

Aims to explore the incidence and potential mechanisms of oligosymptomatic myocardial injury following COVID-19mRNA booster vaccination.

Methods and results: Hospital employees scheduled to undergo mRNA-1273 booster vaccination were assessed for mRNA-1273vaccination-associated myocardial injury, defined as acute dynamic increase in high-sensitivity cardiac troponin T(hs-cTnT) concentration above the sex-specific upper limit of normal on day 3 (48–96 h) after vaccination without evidence of an alternative cause. To explore possible mechanisms, antibodies against interleukin-1receptor antagonist (IL-1RA), the SARS-CoV-2-nucleoprotein (NP) and -spike (S1) proteins and an array of14 inflammatory cytokines were quantified. Among 777 participants (median age 37 years, 69.5% women), 40 participants (5.1%; 95% confidence interval [CI] 3.7–7.0%) had elevated hs-cTnT concentration on day 3 and mRNA-1273 vaccine-associated myocardial injury was adjudicated in 22 participants (2.8% [95% CI1.7–4.3%]). Twenty cases occurred in women (3.7%[95% CI 2.3–5.7%]), two in men (0.8% [95% CI 0.1–3.0%]). Hs-cTnT elevations were mild and only temporary. No patient had electrocardiographic changes, and none developed major adverse cardiac events within 30 days(0% [95% CI 0–0.4%]). In the overall booster cohort, hs-cTnT concentrations (day 3; median 5, interquartile range [IQR] 4–6 ng/L) were significantly higher compared to matched controls (n=777, median 3 [IQR 3–5]ng/L, p<0.001). Cases had comparable systemic reactogenicity, concentrations of anti-IL-1RA, anti-NP, anti-S1,and markers quantifying systemic inflammation, but lower concentrations of interferon (IFN)-λ1(IL-29) and granulocyte-macrophage colony-stimulating factor (GM-CSF) versus persons without vaccine-associated myocardialinjury.

Conclusion: mRNA-1273 vaccine-associated myocardial injury was more common than previously thought, being mild andtransient, and more frequent in women versus men. The possible protective role of IFN-λ1(IL-29) and GM-CSFwarrant further studies. Among 777 participants who received a COVID-19mRNA-1273 booster vaccination, 40 (5.1%; 95% confidence interval [CI] 3.7–7.0%) had an elevated Hs-cTnT concentration on day 3, of whom 22 (2.8% [95% CI1.7–4.3%]) were adjudicated as mRNA-1273 vaccine-associated myocardial injury. Females (n=20, 3.7% [95% CI 2.3–5.7%]) had a higher incidence than males (n=2, 0.8% [95% CI 0.1–3.0%]). Hs-cTnT elevations were mild and decreased from day 3 to the follow-up visit in all but one case. No participants developed major adverse cardiac events within 30 days. Participants with mRNA-1273 vaccine-associated myocardial injury had comparable systemic reactogenicity, but lower concentrations of interferon(IFN)-λ1(IL-29) and granulocyte-macrophage colony-stimulating factor (GM-CSF) versus participants without vaccine-associated myocardial injury..

Introduction: Myocardial injury, manifesting clinically as myocarditis, has recently emerged as a possible severe adverse event following the administration of COVID-19 mRNA vaccines occurring mainly in young men a few days after vaccination. Using passive surveillance following vaccination with BNT162b2-mRNA(Pfizer-BioNTech) or mRNA-1273 (Moderna), COVID-19mRNAvaccination-associated myocarditis is currently considered rare.1

However, passive surveillance detects mostly severe cases requiringhospitalization.2,3We hypothesized that COVID-19 mRNA-vaccine-associated myocardial injury following booster vaccination may be much more common, as symptoms may be unspecific, mild or even absent, escaping passive surveillance. Due

5-2

Sex-specific differences in myocardial injury incidence after COVID-19 mRNA-1273 booster vaccination

Natacha Buergin, Pedro Lopez-Ayala, Julia R. Hirsiger, Philip Mueller, Daniela Median, Noemi Glarner, Klara Rumora, Timon Herrmann, Luca Koechlin, Philip Haaf, Katharina Rentsch - July 20, 2023

https://doi.org/10.1002/ejhf.2978

Abstract

Aims

To explore the incidence and potential mechanisms of oligosymptomatic myocardial injury following COVID-19 mRNA booster vaccination.

Methods and results

Hospital employees scheduled to undergo mRNA-1273 booster vaccination were assessed for mRNA-1273 vaccination-associated myocardial injury, defined as acute dynamic increase in high-sensitivity cardiac troponin T (hs-cTnT) concentration above the sex-specific upper limit of normal on day 3 (48–96 h) after vaccination without evidence of an alternative cause. To explore possible mechanisms, antibodies against interleukin-1 receptor antagonist (IL-1RA), the SARS-CoV-2-nucleoprotein (NP) and -spike (S1) proteins and an array of 14 inflammatory cytokines were quantified. Among 777 participants (median age 37 years, 69.5% women), 40 participants (5.1%; 95% confidence interval [CI] 3.7–7.0%) had elevated hs-cTnT concentration on day 3 and mRNA-1273 vaccine-associated myocardial injury was adjudicated in 22 participants (2.8% [95% CI 1.7–4.3%]). Twenty cases occurred in women (3.7% [95% CI 2.3–5.7%]), two in men (0.8% [95% CI 0.1–3.0%]). Hs-cTnT elevations were mild and only temporary. No patient had electrocardiographic changes, and none developed major adverse cardiac events within 30 days (0% [95% CI 0–0.4%]). In the overall booster cohort, hs-cTnT concentrations (day 3; median 5, interquartile range [IQR] 4–6 ng/L) were significantly higher compared to matched controls ($n = 777$, median 3 [IQR 3–5] ng/L, $p < 0.001$). Cases had comparable systemic reactogenicity, concentrations of anti-IL-1RA, anti-NP, anti-S1, and markers quantifying systemic inflammation, but lower concentrations of interferon (IFN)-λ1 (IL-29) and granulocyte-macrophage colony-stimulating factor (GM-CSF) versus persons without vaccine-associated myocardial injury.

Conclusion

mRNA-1273 vaccine-associated myocardial injury was more common than previously thought, being mild and transient, and more frequent in women versus men. The possible protective role of IFN-λ1 (IL-29) and GM-CSF warrant further studies.

Graphical Abstract

Among 777 participants who received a COVID-19 mRNA-1273 booster vaccination, 40 (5.1%; 95% confidence interval [CI] 3.7–7.0%) had an elevated Hs-cTnT concentration on day 3, of whom 22 (2.8% [95% CI 1.7–4.3%]) were adjudicated as mRNA-1273 vaccine-associated myocardial injury. Females ($n = 20$, 3.7% [95% CI 2.3–5.7%]) had a higher incidence than males ($n = 2$, 0.8% [95% CI 0.1–3.0%]). Hs-cTnT elevations were mild and decreased from day 3 to the follow-up visit in all but one case. No participants developed major adverse cardiac events within 30 days. Participants with mRNA-1273 vaccine-associated myocardial injury had comparable systemic reactogenicity, but lower concentrations of interferon (IFN)-λ1 (IL-29) and granulocyte-macrophage colony-stimulating factor (GM-CSF) versus participants without vaccine-associated myocardial injury.

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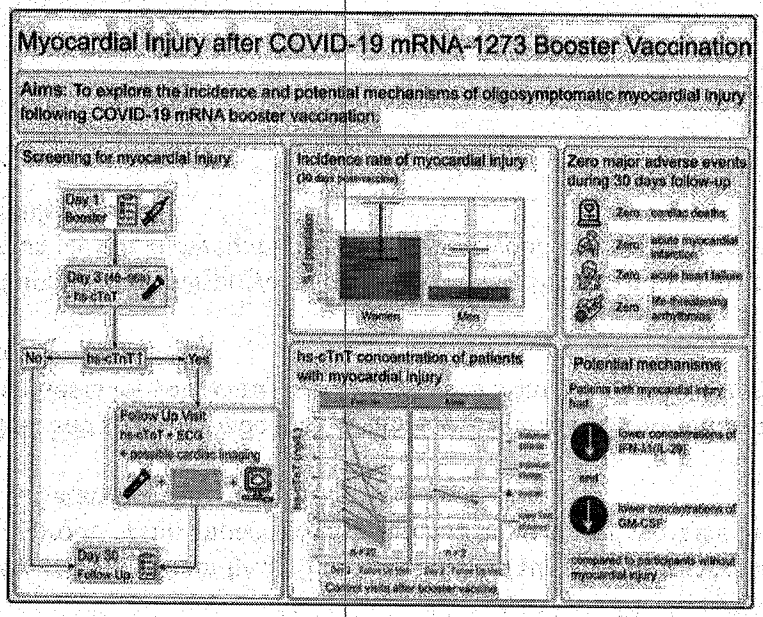

Introduction

Myocardial injury, manifesting clinically as myocarditis, has recently emerged as a possible severe adverse event following the administration of COVID-19 mRNA vaccines occurring mainly in young men a few days after vaccination. Using passive surveillance following vaccination with BNT162b2-mRNA (Pfizer-BioNTech) or mRNA-1273 (Moderna), COVID-19 mRNA vaccination-associated myocarditis is currently considered rare.[1] However, passive surveillance detects mostly severe cases requiring hospitalization.[2,3]

We hypothesized that COVID-19 mRNA-vaccine-associated myocardial injury following booster vaccination may be much more common, as symptoms may be unspecific, mild or even absent, escaping passive surveillance. Due to waning immunity months after mRNA COVID-19 vaccinations there is an apparent need for (repeated) booster vaccinations for billions of people worldwide.[4,5] Thus knowing the true incidence of mRNA-vaccine-associated myocardial injury is of major importance for informed decision-making by patients, physicians and public health authorities.

We therefore conducted a prospective active surveillance study to address this major unmet need. Secondary aims were to provide a 'safety net' for persons identified with COVID-19 mRNA-vaccine-associated myocardial injury to allow early detection and preventive measures to avoid possible aggravation, and to evaluate potential mechanisms underlying COVID-19 mRNA-vaccine-associated myocardial injury.

Methods

Study design and study population

This prospective investigator-initiated industry-independent active surveillance study was approved by the local ethics committee. Employees of the University Hospital Basel, Switzerland, scheduled to receive mRNA-1273 first booster vaccination, and who provided written informed consent, were offered active surveillance. Exclusion criteria were cardiac events or cardiac surgery within 30 days prior to vaccination or patients missing the study visit, therefore missing high-sensitivity cardiac troponin T (hs-cTnT) measurement on day 3.

Active surveillance and laboratory methods

Medical history was assessed on the day of the booster vaccination (day 1). On day 3 (48–96 h) after vaccination, participants were assessed for possible myocarditis-related symptoms and a venous blood sample for the measurement of hs-cTnT (Elecsys,

S-4

sex-specific 99th-percentile of healthy individuals and upper limit of normal [ULN] 8.9 ng/L in women and 15.5 ng/L in men, limit of detection 3 ng/L) was obtained.[6,7] If the hs-cTnT concentration was elevated on day 3, participants were informed, asked to avoid strenuous exercise in order to minimize additional strain of the myocardium and associated cardiomyocyte injury, and offered follow-up including clinical evaluation, a second hs-cTnT measurement, and a 12-lead electrocardiogram (ECG). The follow-up visit was scheduled, if feasible, the next working day. After extensive discussion with the local ethics committee and the COVID-19 task force of the University Hospital Basel, it was prioritized that this study should interfere as little as possible with the motivation of the hospital staff to obtain the mRNA-1273 first booster vaccination and the logistics of booster vaccination itself. Accordingly, blood draws were performed only after the vaccination.

Potential mechanisms underlying vaccine-associated myocardial injury

We evaluated three potential mechanisms of COVID-19 mRNA-vaccination-associated myocardial injury: anti-interleukin-1 receptor antagonist (IL-1RA) autoantibodies,[8] pre-existing vaccine/infection-induced immunity against SARS-CoV-2 (i.e. anti-SARS-CoV-2-nucleoprotein [NP] and -spike [S1]-IgG), and systemic reactogenicity/inflammation. Anti-IL-1RA-, -NP-, and S1-IgG were quantified using the Luminex platform (Luminex Corporation, Austin, TX, USA)[9] (online supplementary methods). Systemic inflammation was assessed by measuring 14 biomarkers using the LEGENDplex™ Human Anti-Virus Response Panel (interleukin [IL]-1β, IL-6, IL-8, IL-10, IL-12p70, interferon [IFN]-α, IFN-β, IFN-λ1[IL-29], IFN-λ2/3[IL-28], IFN-γ, tumour necrosis factor [TNF]-α, interferon gamma-induced protein (IP)-10, granulocyte-macrophage colony-stimulating factor [GM-CSF]), the IL-1RA assay (both Biolegend, San Diego, CA, USA), and C-reactive protein (CRP; Elecsys; ULN 5.0 mg/L).

Adjudication of COVID-19 mRNA-vaccine-associated myocardial injury

Given the general superior sensitivity of hs-cTnT elevations versus the ECG or cardiac imaging for acute myocardial injury,[10,11] COVID-19 mRNA-vaccine-associated myocardial injury was defined as acute dynamic hs-cTnT elevation above the sex-specific 99th perentile ULN (8.9 ng/L in women and 15.5 ng/L in men) on day 3, without evidence of an alternative cause, irrespective of symptoms, ECG, or cardiac imaging abnormalities. In the absence of a baseline hs-cTnT concentration immediately prior to the vaccination, strict criteria were applied in the adjudication of COVID-19 mRNA-vaccine-associated myocardial injury. For the differentiation of acute COVID-19 mRNA-vaccine-associated myocardial injury versus possible chronic preexisting myocardial injury, four criteria were used: first, the extent of the hs-cTnT elevation (the higher the elevation, the more likely acute); second, the extent in the change of hs-cTnT from day 3 to day 4 (the larger the change the more likely acute); third, previous hs-cTnT measurements if available in the medical history of the participants; and fourth, the likelihood for hs-cTnT elevation according to known causes of chronic myocardial injury, including age and preexisting cardiovascular diseases. To emphasize how physicians could miss COVID-19 mRNA-vaccine-associated myocardial injury in women, a sensitivity analysis, using a uniform ULN cut-off (14 ng/L) was used for adjudication. To further verify that COVID-19 mRNA booster vaccination may increase hs-cTnT concentration, hs-cTnT concentration on day 3 in the overall cohort receiving COVID-19 mRNA booster vaccination was compared to matched controls.

Follow-up

Major adverse cardiac events (MACE) including acute heart failure, cardiac death, life-threatening arrhythmia and acute myocardial infarction (AMI) were assessed at 30-day follow-up.

Matching

To assess cardiomyocyte injury also as a continuous variable, hs-cTnT concentrations on day 3 after vaccination were compared to age-, sex-, history of coronary artery disease/AMI-matched patients (controls) that had presented with acute chest discomfort to the emergency department in a multicentre study (NCT00470587) and were centrally adjudicated as having a non-cardiac cause. A total of 777 booster-vaccinated subjects and 3716 eligible controls (fulfilling inclusion criteria) were identified. Matching was conducted using a nearest neighbour propensity score matching method, without replacement of controls and with a case-to-control ratio of 1:1.[12] For details see online supplementary methods.

Statistical analysis

Continuous variables were reported as median and interquartile range (IQR), categorical variables as counts and percentages. Differences in characteristics between subjects with and without SARS-CoV-2 mRNA-vaccine-associated myocardial injury were assessed using the Mann–Whitney U test for continuous variables, and the Pearson χ^2 test or Fisher exact test for categorical variables, when appropriate. All hypothesis testing was 2-tailed with a significance level of $p < 0.05$. Statistical analyses were performed using R version 4.1.3 (R Foundation for Statistical Computing, Vienna, Austria). Reporting is in accordance with the

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Strengthening the Reporting of Observational studies in Epidemiology (STROBE) statement (online supplementary *Table* **S1**). We did not adjust for multiple testing for the evaluation of different potential mechanisms underlying COVID-19 mRNA-vaccine-associated myocardial injury due to the exploratory nature of the analysis.

Results

From 10 December 2021 to 10 February 2022, 1871 employees of the University Hospital Basel were screened (1294 females [69.2%] and 577 males [30.8%]), of which 835 provided written informed consent to participate in the study, and of these, 777 (93%, 540 females [69.5%] and 237 males [30.5%]) were eligible for analysis (*Table* **1**, *Figures* **1** and **2A**). The median age was 37 years (IQR 30–50), and 69.5% were women. Age-, sex-, and history of coronary artery disease/AMI-matched controls had comparable baseline characteristics (online supplementary *Table* **S2** and *Figures* **S1**–**S3**). A flowchart of the active surveillance programme is depicted in *Figure* **2A** and the *Graphical Abstract*.

Table 1. Baseline characteristics and vaccine-associated symptoms stratified by adjudicated vaccine-associated myocardial injury

Variable	Overall	No vaccine-associated myocardial injury	Vaccine-associated myocardial injury	*p*-value
n	777	755	22	
Age, years, median [IQR]	37 [30–50]	37 [29–50]	46 [33–54]	0.12
Sex, *n* (%)				
Male	237 (30.5)	235 (31.1)	2 (9.1)	0.03
Female	540 (69.5)	520 (68.9)	20 (90.9)	
History of COVID-19 infection, *n* (%)	82 (10.6)	80 (10.6)	2 (9.5)	1
No. of previous COVID-19 vaccinations, *n* (%)				
One vaccination[a]	1 (0.1)	1 (0.1)	0 (0.0)	0.20
One vaccination after COVID-19	37 (4.8)	37 (4.9)	0 (0.0)	
Two vaccinations	714 (92.0)	694 (92.0)	20 (90.9)	
Two vaccinations after COVID-19	24 (3.1)	22 (2.9)	2 (9.1)	
Days since last vaccination, median [IQR]	206.0 [188.0–230.0]	205.0 [188.0–229.0]	222.0 [187.2–253.2]	0.14
History of CAD, *n* (%)	3 (0.4)	3 (0.4)	0 (0.0)	1

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Variable	Overall	No vaccine-associated myocardial injury	Vaccine-associated myocardial injury	*p*-value
History of AMI, *n* (%)	1 (0.1)	1 (0.1)	0 (0.0)	1
History of heart surgery[b], *n* (%)	3 (0.4)	3 (0.4)	0 (0.0)	1
History of myocarditis, *n* (%)	3 (0.4)	3 (0.4)	0 (0.0)	1
History of heart failure, *n* (%)	2 (0.3)	2 (0.3)	0 (0.0)	1
Symptoms following vaccination, *n* (%)				
Chest pain	63 (8.1)	61 (8.1)	2 (9.1)	0.70
Palpitations	70 (9.0)	69 (9.1)	1 (4.5)	0.71
Dyspnoea	23 (3.0)	23 (3.0)	0 (0.0)	1
Fever and/or chills	270 (34.7)	263 (34.8)	7 (31.8)	0.95
Body aches	356 (45.8)	347 (46.0)	9 (40.9)	0.80
Biomarkers				
hs-cTnT (day 3), ng/L, median [IQR]	5 [4–6]	5 [4–6]	13.5 [9–18.8]	<0.001

AMI, acute myocardial infarction; CAD, coronary artery disease; hs-cTnT, high-sensitivity cardiac troponin T; IQR, interquartile range.

[a] The patient with only one previous vaccination had received Johnson & Johnson's Janssen COVID-19 vaccine which is a full primary immunization. According to Swiss authorities, past COVID-19 infection and one vaccination were regarded equivalent to having had two vaccinations (without previous COVID-19 infection) for primary immunization in Switzerland.

[b] History of heart surgery: one bypass surgery, one atrial septal aneurysm and one atrial septal defect.

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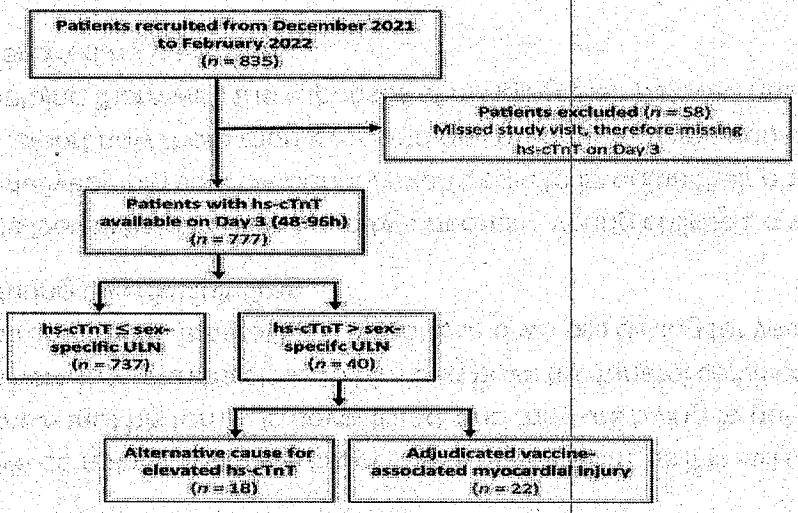

Figure 1

Patient flowchart. Hs-cTnT, high-sensitivity cardiac troponin T; ULN, upper limit of normal.

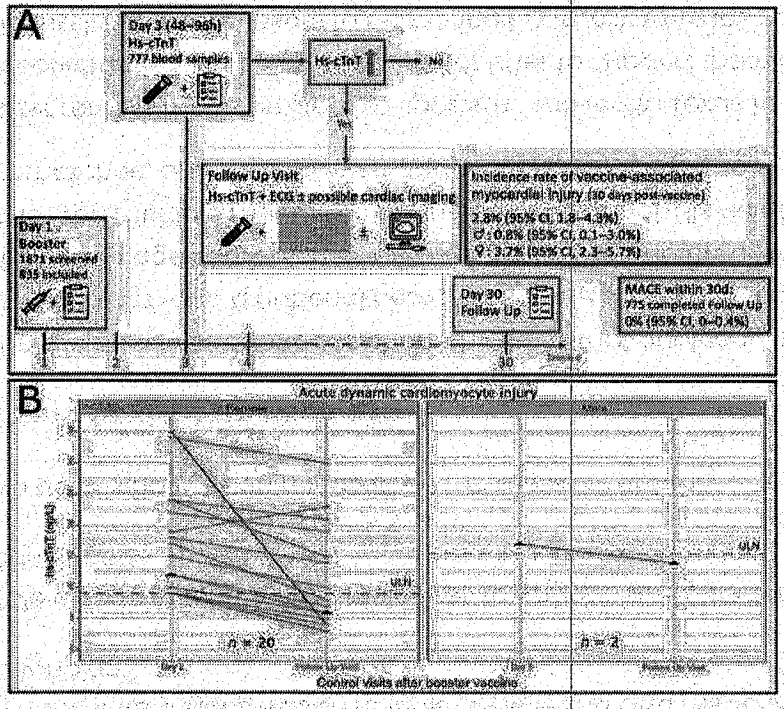

Figure 2

(*A*) Flowchart of the active surveillance programme and incidence of mRNA-1273 vaccination-associated myocardial injury. (*B*) High-sensitivity cardiac troponin T (hs-cTnT) concentrations in patients with mRNA-1273 vaccination-associated myocardial injury. The triangles represent the median, points represent the individual patients, the dashed lines labelled ULN represent the sex-specific upper limit of normal. Both men with vaccination-associated myocardial injury had identical concentrations on day 3 (17 ng/L), therefore only one point is shown. One male patient did not have a follow-up visit, hence only one line is shown. CI, confidence interval; MACE, major adverse cardiac event.

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COVID-19 mRNA-1273 vaccine-associated myocardial injury

High-sensitivity cTnT concentrations (online supplementary *Figure S4*) above the sex-specific ULN were detected in 40 participants (5.1% [95% confidence interval, CI 3.7–7.0%]). In 18 of them (17 women, median age 59 years [IQR 57–60], median hs-cTnT concentration 10 ng/L [IQR 9–11]; online supplementary *Table S3*), an alternative cause was considered most likely (online supplementary *Table S4*). mRNA-1273 vaccine-associated myocardial injury was adjudicated in 22 patients (2.8% [95% CI 1.8–4.3%]), with 20 cases occurring in women (3.7% [95% CI 2.3–5.7%]) and 2 in men (0.8% [95% CI 0.1–3.0%]), with a median age of 46 years (IQR 33–54). This sex difference was statistically significant ($p = 0.03$). On day 3, median hs-cTnT concentration of the 20 women and 2 men with mRNA-1273 vaccine-associated myocardial injury was 13.5 ng/L (IQR 9.0–18.8) (*Figure 2B*). It decreased in all but one patient on the follow-up visit to a median value of 6.0 ng/L (IQR 4.0–14.0), being again in the normal range in half of the participants.

In the overall cohort receiving the mRNA-1273 booster, hs-cTnT concentrations (day 3) were significantly higher compared to matched controls (median 5 [IQR 4–6] ng/L vs. 3 [IQR 3–5] ng/L, $p < 0.001$). *Figure 3* illustrates this difference, indicating an overall shift towards higher hs-cTnT concentrations in the booster cohort versus matched controls, for both female (median 4 [3–6] ng/L vs. 2.99 [2.99–4] ng/L) and male (median 6 [5–8] ng/L vs. 4 [2.99–6] ng/L) participants.

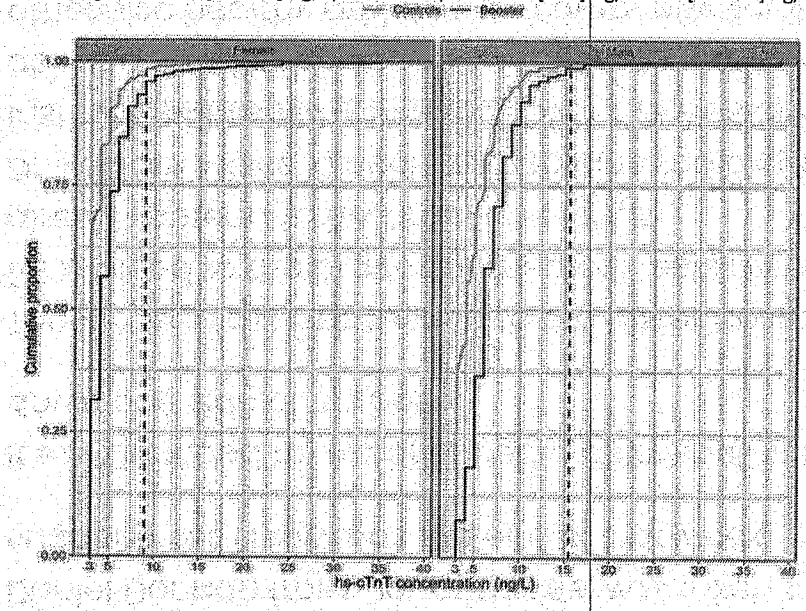

Figure 3

Cumulative distribution curve of cardiomyocyte injury as quantified by high-sensitivity cardiac troponin T (hs-cTnT) concentrations stratified by sex. The dashed lines indicate the sex-specific upper reference limits. Hs-cTnT, high sensitivity cardiac troponin T.

None of the participants with elevated markers of myocardial injury related to mRNA vaccination had a history of cardiac disease (online supplementary *Table S5*). Eleven participants (50%) had unspecific symptoms including fever and chills, two had chest pain, and none had ST-segment depression or T-wave inversion (online supplementary *Table S5*). Pre-defined and prospectively recorded symptoms occurred with comparable frequency in participants developing mRNA-1273 vaccine-associated myocardial injury versus those that did not.

No definitive case of myocarditis was found. However, the two participants (both women) with vaccine-associated myocardial injury and chest pain met the Brighton Collaboration case definition Level 2, indicating probable myocarditis in those patients (0.3% [95% CI 0.1–0.9%]).[13]

Sensitivity analysis

When using a uniform ULN of 14 ng/L, mRNA-1273 vaccine-associated myocardial injury was adjudicated in 14 patients (1.8% [95% CI 1.0–3.0%]), with 9 cases occurring in women (1.7% [95% CI 0.8–3.2%]) and 5 in men (2.1% [95% CI 0.7–4.9%]), with a

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median age of 53 years (IQR 38–56). On day 3, median hs-cTnT concentration of the 9 women and 5 men with mRNA-1273 vaccine-associated myocardial injury was 17.5 ng/L (IQR 15.5–20.5). It decreased in all but one patient on the follow-up visit to a median value of 14.0 ng/L (IQR 10.0–19.0), being again below the uniform ULN in half of the participants (online supplementary *Figure* ***S5***).

Major adverse cardiac events

Thirty-day follow-up was completed in 775 participants (99.7%) and no participant developed MACE (0% [95% CI 0–0.4%]).

Possible mechanisms of mRNA-1273 vaccine-associated myocardial injury

Antibodies against IL-1RA were detected with comparable and low frequency in participants with mRNA-1273 vaccine-associated myocardial injury versus those without (1 in 22 [4.5%] vs. 23/742 [3.1%]; Fisher exact test $p = 0.51$). The plasma levels of IL-1RA were also comparable between the two groups. There was no difference in the magnitude of the anti-S1-IgG and the frequency of subjects positive for anti-NP-IgG (i.e. serological evidence for prior infection with SARS-CoV-2) in participants with mRNA-1273 vaccine-associated myocardial injury versus those without (*Table* **2**). Also, most tested markers of systemic inflammation had comparable concentrations in participants with mRNA-1273 vaccine-associated myocardial injury versus those without. In contrast, levels of IFN-λ1 and GM-CSF were lower in cases with mRNA-1273 vaccine-associated myocardial injury versus those without (online supplementary *Figures* ***S6*** and ***S7***).

Table 2. Inflammatory biomarkers stratified by adjudicated vaccine-associated myocardial injury

Variable	Overall	No vaccine-associated myocardial injury	Vaccine-associated myocardial injury	*p*-value
n	764	742	22	
Antibodies				
Anti-NP (MFI)	138.2 [65.0–322.6]	139.0 [66.0–325.0]	103.5 [33.6–192.8]	0.052
Anti-S1 (MFI)	1641.0 [870.8–3254.0]	1641.0 [877.8–3281.0]	1686.5 [757.5–2614.8]	0.76
Anti-IL-1RA (MFI)	30.8 [23.0–48.1]	31.0 [23.0–48.0]	25.5 [19.5–46.9]	0.31
Systemic inflammation				
IL-1RA (pg/ml)	621.3 [438.0–832.0]	621.3 [440.5–829.1]	605.3 [426.5–895.2]	0.968
IL-1β (pg/ml)	6.8 [3.4–13.2]	6.8 [3.4–13.2]	7.0 [3.6–9.4]	0.57
IL-6 (pg/ml)	1.7 [0.5–3.4]	1.7 [0.5–3.4]	1.5 [0.5–2.7]	0.62
IL-8 (pg/ml)	4.2 [3.1–5.9]	4.3 [3.1–6.0]	3.9 [3.3–5.7]	0.65
IL-10 (pg/ml)	9.8 [3.9–25.8]	9.8 [3.9–25.6]	10.4 [3.2–31.5]	0.91
IL-12p70 (pg/ml)	10.0 [4.8–18.1]	10.1 [4.9–18.2]	8.0 [2.7–14.3]	0.289

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Variable	Overall	No vaccine-associated myocardial injury	Vaccine-associated myocardial injury	*p*-value
CRP (mg/L)	5.5 [2.8–10.2]	5.4 [2.8–10.1]	6.9 [4.3–10.1]	0.28
TNF-α (pg/ml)	5.6 [1.7–17.6]	5.7 [1.7–17.7]	4.1 [1.7–11.9]	0.43
IFN-β (pg/ml)	3.9 [0.8–8.9]	3.9 [0.8–9.1]	3.0 [0.8–6.4]	0.13
IFN-γ (pg/ml)	16.9 [6.4–37.5]	16.9 [6.6–38.0]	15.5 [4.0–30.4]	0.42
IFN-α2 (pg/ml)	2.5 [0.7–5.4]	2.5 [0.7–5.4]	2.0 [1.3–3.8]	0.70
IFN-λ1 (pg/ml)	11.4 [3.8–21.8]	11.8 [3.9–22.3]	5.3 [2.9–10.8]	0.015
IFN-λ2-3 (pg/ml)	7.8 [4.1–12.9]	7.9 [4.2–12.9]	5.5 [3.1–8.6]	0.052
GM-CSF (pg/ml)	2.0 [0.6–4.4]	2.0 [0.6–4.5]	0.6 [0.6–2.9]	0.039
IP-10 (pg/ml)	49.8 [25.8–120.2]	49.8 [25.4–120.8]	49.5 [31.2–78.9]	0.984

In 13 patients (without vaccine-associated myocardial injury) the volume provided to the immunology laboratory was insufficient to measure the inflammatory biomarkers.

CRP, C-reactive protein; GM-CSF, granulocyte-macrophage colony-stimulating factor; IFN, interferon; IL, interleukin; IL-1RA, interleukin-1 receptor antagonist; IP, interferon gamma-induced protein; MFI, median fluorescence intensity; NP, nucleoprotein; S1, spike; TNF, tumour necrosis factor.

Discussion

This prospective investigator-initiated, industry-independent study was performed to test the hypothesis that mRNA-1273 booster vaccination-associated myocardial injury may be more common than currently thought as symptoms may be unspecific, mild or even absent, escaping passive surveillance detecting only hospitalized cases. We report four main findings.

First, our findings confirmed the study hypothesis. mRNA-1273 booster vaccination-associated elevation of markers of myocardial injury occurred in about one out of 35 persons (2.8%), a greater incidence than estimated in meta-analyses of hospitalized cases with myocarditis (estimated incidence 0.0035%) after the second vaccination.[14,15] Elevated hs-cTnT was independent of previous COVID-19 infection or the interval since the last vaccine dose. Among the overall group of participants, hs-cTnT concentration on day 3 after mRNA-1273 booster vaccination as a continuous variable, was significantly higher compared to a well-matched control cohort. Second, all cases were mild with only a transient and short period of myocardial injury (maximum hs-cTnT concentration 35 ng/L). No patient showed ECG changes and no patient developed MACE within 30 days. Potentially, such outcomes were averted by the safety net provided by early detection and early implementation of preventive measures for deterioration including avoidance of strenuous exercise. Notably, systemic reactogenicity (fever, chills, body aches), and chest pain occurred with comparable frequency in participants with versus without mRNA-1273 booster vaccine-associated hs-cTnT elevations. Third, when using sex-specific ULN cut-offs for myocardial injury adjudication, mRNA-1273 booster vaccine-associated myocardial injury occurred significantly more often in women versus men (3.7% vs. 0.8%). This is in striking discrepancy to the sex distribution of vaccine-associated myocardial injury in the setting of clinical myocarditis following the first and second vaccinations detected by passive surveillance, which occurred predominately in young men.[2,3,16] Median age of participants developing mRNA-1273 vaccine-associated myocardial injury was 46 years. Thereby, also the age distribution is different to that of most reported vaccine-associated clinical myocarditis cases.[2,3] When using a uniform (and thereby higher in women and lower in men compared to the sex-specific) ULN cut-off for adjudication, the incidence rate

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of vaccine-associated myocardial injury declined in women and increased in men. Fourth, the predominate mechanisms underlying mRNA-1273 booster vaccination-associated myocardial injury did not seem to include antibodies neutralizing IL-1RA, which were suggested to be involved in the pathophysiology of severe COVID-19 mRNA vaccine-associated myocarditis in young male patients,[8] pre-existing vaccine/infection-induced immunity against SARS-CoV-2, nor systemic inflammation. In contrast, levels of IFN-λ1 and GM-CSF, both modulators of the immune responses to acute viral infection, vaccination, and tissue inflammation, were lower in cases with mRNA-1273 vaccine-associated myocardial injury versus those without.[17-19] However, we did not adjust for multiple testing nor for potential confounders for the evaluation of different potential immunological mechanisms underlying COVID-19 mRNA-vaccine-associated myocardial injury due to the exploratory nature of the analysis and should thus be considered as a hypothesis-generating analysis. IFN-λ limits inflammation-induced tissue damage in viral infections[20] and in models of ischaemic myocardial injury.[21] Whether IFN-λ1 deficiency may reduce myocardial protection and thereby promote vaccine-associated myocardial injury needs to be further investigated. In a phase 3 trial, pegIFN- λ reduced hospitalizations and emergency visits in patients with COVID-19[22] and in a phase 2 study, pegIFN- λ accelerated viral decline in outpatients with COVID-19,[17, 18] thereby further strengthening the rationale of the hypothesis that IFN-λ1 deficiency may be involved in vaccine-associated myocardial injury. GM-CSF exerts pro-inflammatory effects, and both administration and inhibition of GM-CSF are tested as potential therapeutics in COVID-19.[19] Whether low GM-CSF blood levels are a risk factor for immune-mediated cTnT elevations remains to be further elucidated. The significantly higher rate of mRNA-1273 booster vaccination-associated myocardial injury in women versus men may at least partly be related to the higher vaccine dose per body weight or myocardial mass in women and therefore dose-dependent toxic effects. Clinically overt severe vaccination-associated myocarditis may then occur following a second hit, possibly mediated by neutralizing autoantibodies targeting IL-1RA, microvascular thrombosis, or direct cardiac myocyte injury unrelated to inflammation.[8, 23]

Our findings following mRNA-1273 booster vaccination extend and corroborate observations in two recent active surveillance studies after BNT162b2 vaccination.[24, 25] Among 324 healthcare workers (mean age 51 years, 59.2% women) who received a fourth dose of BNT162b2 in Israel, two participants (one woman and one man) developed vaccine-related myocardial injury on day 3 (incidence 0.6%, maximum hs-cTnI concentration 22.1 ng/L). One had mild symptoms including fever and chest pain, one was asymptomatic. Both had a normal ECG and echocardiography.[24] Among 301 adolescents in Thailand (mean age 15 years) receiving the second dose of BNT162b2, five participants (incidence 1.7%), all boys, developed vaccine-related myocardial injury on either day 2 or day 3.[25] One of them had very high hs-cTnT concentrations (593 ng/L) and late gadolinium enhancement indicating myocarditis on cardiac magnetic resonance (CMR) imaging. When comparing these studies, it is important to highlight major differences in the study population and study methodology.

Therefore, the main finding of this study, that subclinical mRNA vaccine-associated myocardial injury is much more common than estimated based on passive surveillance, has been confirmed and generalized in these complimentary cohorts of slightly older healthcare workers in Israel and adolescents in Thailand. Additional active surveillance studies are needed to externally validate two specific findings of this study: the even higher rate of mRNA-1273 booster vaccination-associated myocardial injury overall, and particularly in women. At least in part, these findings seem explained by the use of sex-specific ULN for hs-cTnT in this versus a uniform ULN in the two other studies, as well as using mRNA-1273, which also had resulted in a higher rate of hospitalizations due to clinical myocarditis versus BNT162b2 in prior passive surveillance studies.[2, 3, 26, 27] Of note, mRNA-1273 had also resulted in higher immunogenicity and protection from COVID-19 versus BNT162b2 in large observational studies.[28, 29] Vaccine-related myocarditis has previously been reported following smallpox vaccination with an observed incidence of 16.11/100 000, which was nearly 7.5-fold higher than the expected background incidence.[30] In contrast, myocarditis following other vaccines is rare.[31] Similar to our finding with mRNA vaccination, there is evidence that the frequency of subclinical myocardial injury may also be higher after smallpox vaccination. A study on US military personnel found subclinical cTnT elevations in 2.87% of 1081 smallpox vaccinated subjects, or a 60 times higher rate than overt clinical cases.[32] The same study found no cTnT elevation in 189 subjects vaccinated with the inactivated influenza vaccine. This suggest that vaccine characteristics are relevant for the observed cTnT increase.

The long-term consequences of vaccine-related myocardial injury detected by transient and mostly mild hs-cTnT/I elevations on day 2 or 3 are unknown. Given the small extent of acute cardiomyocyte injury in our study, that is, cTnT levels of about one-fourth of those observed in patients with spontaneous myopericarditis,[10] and its transient nature, good long-term outcomes can be expected. COVID-19 associates with a substantially higher risk for myocarditis that mRNA vaccination,[33] and myocarditis related to COVID-19 infection has shown a higher mortality than myocarditis related to mRNA vaccination.[34, 35] Thus, for the majority of individuals, the overall very favourable risk–benefit ratio of booster immunizations persists.[14, 15, 36-39] However, further studies are needed to assess the impact of mRNA vaccine-associated myocardial injury on the long-term risk of cardiac arrhythmias and heart failure. Also, evidence generated in the perioperative setting should help avoid the over-simplistic

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assumption that the absence of typical chest pain on day 3 after vaccination in most cases would *per se* indicate a favourable prognosis: perioperative myocardial injury not associated with chest discomfort had comparable unfavourable long-term outcome versus perioperative myocardial injury with chest discomfort.[40]

By providing novel insights regarding the incidence, extent, duration, patient characteristics, possible mechanisms, and outcome of mRNA-1273 booster vaccination-associated myocardial injury, this study aims to help patients, physicians, and public health authorities make informed decisions regarding future booster vaccinations.[4] Importantly, this study also may help manufacturers fine-tune the dose and composition of future vaccines.

It is mandatory to put our findings into perspective with the incidence and extent of myocardial injury associated with COVID-19 infection. Before the COVID-19 vaccine were available, the incidence and extent of myocardial injury associated with COVID-19 infection was much higher than observed in this active surveillance study after booster vaccination.[37, 41, 42] Data on the incidence of COVID-19-associated myocardial injury in populations with high immunity against SARS-CoV-2 are not yet available.

Alternative, yet unlikely, contributors to the elevated cTnT in our study include cardiomyocyte injury associated with strenuous exercise, or in the context of a high inflammatory response to the vaccination or a non-cardiac source. While exercise was not restricted between vaccination and first hs-cTnT measurement, none of the detected cases reported strenuous exercise preceding the blood draw on day 3. Importantly, prior exercise was also not restricted among the matched control group, and even strong exercise typically only leads to an increase in hs-cTnT concentration of on average 1 ng/L.[43] Moreover, neither the clinical symptoms (i.e. fever, chills, muscle sore), nor the measured markers of systemic inflammation indicated an overshooting inflammatory response in subjects with hs-cTnT elevation. In contrast to some rather rare chronic active skeletal muscle diseases such as muscle dystrophies, acute skeletal muscle injury, even when as extensive as in patients with rhabdomyolysis, has been found not to be a non-cardiac source of elevated hs-cTnT concentrations.[44-46] Also, interference has been reported as a possible confounding factor for cardiac troponin elevations. However, this issue seems to predominantly affect the current hs-cTnI and not the current hs-cTnT assay.[47] Therefore, the acute dynamic increase in hs-cTnT concentration following mRNA COVID-19 vaccination has to be considered indicating myocardial injury and not secondary to the intramuscular injection and local skeletal muscle injury. Lastly, unknown prior cardiac disease may have been contributing to some of the extent of myocardial injury observed. Therefore, conservative criteria were used for the adjudication of mRNA-1273 booster vaccination-associated myocardial injury and 18 additional patients with hs-cTnT elevation on day 3 were classified as more likely having chronic myocardial injury.

The following limitations should be considered when interpreting our findings. First, to interfere as little as possible with the motivation of the hospital staff to obtain the mRNA-1273 booster vaccination and its logistics, we restricted the study to blood draws after vaccination. Thus, baseline hs-cTnT values were not available. The lack of a baseline hs-cTnT concentration was therefore addressed threefold: (i) by requiring a relevant change in hs-cTnT concentration from day 3 to the follow-up visit as additional criteria to adjudicate mRNA vaccine-associated myocardial injury; (ii) by conservative adjudication in that 18 participants with mild hs-cTnT-elevations on day 3 (17 women, one man), and either no available hs-cTnT concentration at follow-up visit or one with no relevant change, were considered to reflect pre-existing known or assumed cardiac disease rather than mRNA-1273 booster vaccine-associated myocardial injury (although the differential diagnosis in these 18 patients includes persistent vaccine-associated myocardial injury); Thereby, among the 40 participants (5.1%) detected to have increased hs-cTnT concentration on day 3 after mRNA-1273 booster vaccination, only 22 participants (2.8%) were adjudicated to have mRNA-1273 vaccine-associated myocardial injury. For comparison, using the sex-specific 99th percentile as the ULN, among presumably healthy individuals only 1% of persons are expected to have increased levels; (iii) by adding an age-, sex-, and history of coronary artery disease/AMI-matched control group. Despite our efforts to address the lack of baseline hs-cTnT concentration, we may have still misclassified a small number of participants. Future studies using baseline values for adjudicating acute dynamic high-sensitivity cardiac troponin elevation above the sex-specific ULN are warranted to confirm our findings. Second, the time-course of mRNA-1273 vaccine-associated myocardial injury is incompletely understood. Accordingly, by measuring hs-cTnT on day 3 after mRNA-1273 booster vaccination, which was in line with other studies,[24] we might have missed cases that peaked earlier and had already returned to normal on day 3. Third, the 4th universal definition of myocardial infarction states that 'elevated cTn levels may be indicative of acute myocardial injury if the pattern of values is rising and/or falling'.[48] No specific percentage change was proposed, thus in some patients the distinction between acute and chronic was challenging. In those cases, we adjudicated those patients as chronic injury, thus choosing the more conservative approach. Fourth, this study recruited unselected healthcare workers of a university hospital. Thereby, the study population was relatively young and 70% women. Further studies are warranted to extend the findings regarding incidence of mRNA-1273 booster vaccination-associated myocardial injury and 30-day MACE to other populations. Both may differ particularly in older persons with a higher preexisting burden of cardiovascular disease. Fifth, no CMR imaging was performed, as the amount of vaccine-induced cardiomyocyte injury in this study was below the expected limit of detection of CMR for late gadolinium enhancing myocardial

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lesions indicative of myocarditis (usually a hs-cTnT concentration of about 50–100 ng/L).[10, 11] These thresholds were pre-defined in collaboration with imaging experts, but are based on expert opinion rather than large prospective studies. Sixth, it is unknown whether and to what extent early detection and management, such as asking cases to avoid strenuous exercise, contributed to the excellent outcomes at 30 days. Seventh, given the absence of another *in-vivo* technique with comparable sensitivity to hs-cTnT/I regarding acute cardiomyocyte injury, it remains unknown whether mRNA-1273 vaccine-induced myocardial injury resulted in cardiomyocyte cell death and thereby irreversible loss of cardiomyocytes, or sublethal injury. In conclusion, using active surveillance, mRNA-1273 vaccine-associated mild transient myocardial injury was found to be much more common than previously thought. It occurred in one out of 35 persons, was mild and transient, and more frequent in women versus men. Neither anti-IL-1RA, nor pre-existing vaccine/infection-induced immunity or systemic inflammation seemed to be dominant mechanisms of myocardial injury. No participant developed MACE within 30 days.

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KILL SHOT: Recent Peer-Reviewed Report Finds 1 in 35 People Who Took Moderna COVID Shot Had Signs of Heart Damage

Jim Hoft Jul. 27, 2023

A new study from the **European Journal of Heart Failure** found that 1 in 35 people showed signs of heart damage after taking the Moderna COVID19 vaccine.

The study concluded that mRNA-1273 vaccine-associated myocardial injury was more common than previously thought, being mild and transient, and *more frequent in women versus men.*

Abstract

Aims

To explore the incidence and potential mechanisms of oligosymptomatic myocardial injury following COVID-19 mRNA booster vaccination.

Methods and Results

Hospital employees scheduled to undergo mRNA-1273 booster vaccination were assessed for mRNA-1273 vaccination-associated myocardial injury, defined as acute dynamic increase in high-sensitivity cardiac troponin T (hs-cTnT) concentration above the sex-specific upper limit of normal on day 3 (48–96 h) after vaccination without evidence of an alternative cause. To explore possible mechanisms, antibodies against IL-1RA, the SARS-CoV2-Nucleoprotein(NP) and -Spike(S1) proteins and an array of 14 inflammatory cytokines were quantified. Among 777 participants, median age 37 years, 69.5% women, 40 participants (5.1% [95%CI, 3.7%–7.0%]) had elevated hs-cTnT concentration on day 3 and mRNA-1273 vaccine-associated myocardial injury was adjudicated in 22 participants (2.8% [95%CI, 1.7%–4.3%]). Twenty cases occurred in women (3.7% [95%CI, 2.3%–5.7%]), two in men (0.8% [95%CI, 0.1%–3.0%]). Hs-cTnT-elevations were mild and only temporary. No patient had ECG-changes, and none developed major adverse cardiac events within 30 days (0% [95%CI, 0%–0.4%]). In the overall booster cohort, hs-cTnT concentrations (day 3; median 5 [IQR, 4–6] ng/L) were significantly higher compared to matched controls (n = 777, median 3 [IQR, 3–5] ng/L, p < 0.001). Cases had comparable systemic reactogenicity, concentrations of anti-IL-1RA, anti-NP, anti-S1, and markers quantifying systemic inflammation, but lower concentrations of IFN-λ1(IL-29) and GM-CSF versus persons without vaccine-associated myocardial injury.

Conclusion

mRNA-1273 vaccine-associated myocardial injury was more common than previously thought, being mild and transient, and more frequent in women versus men. The possible protective role of IFN-λ1(IL-29) and GM-CSF warrant further studies.

They forced Americans to take this vaccine to work and attend public events.

Via **Vigilant Fox**.

Peer-Reviewed Study Finds 1 in 35 People Had Signs of Heart Damage After Receiving the Moderna Booster Shot

Think about that: a 1 in 35 risk of heart damage after 3 shots — for something as mild as the flu for most people. This is devastating. pic.twitter.com/vA8Bmc1GCf

— The Vigilant Fox (@VigilantFox) July 26, 2023

Here's how Dr. John Campbell reacted to this news:

Portland Health Care
ooo
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Fax: (503) 408-1606

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"If regulators around the world don't take notice of the information I'm about to give via this paper, then they are at best, in my view, negligent."

Credit: **@TheChiefNerdhttps://t.co/6vZ22zAVil**

— The Vigilant Fox (@VigilantFox) **July 26, 2023**
The Epoch Times reported:

Damage to the heart is more common than thought after receipt of Moderna's COVID-19 booster, a new study indicates.

One in 35 health care workers at a Swiss hospital had signs of heart injury associated with the vaccine, mRNA-1273, researchers found.

"mRNA-1273 booster vaccination-associated elevation of markers of myocardial injury occurred in about one out of 35 persons (2.8%), a greater incidence than estimated in meta-analyses of hospitalized cases with myocarditis (estimated incidence 0.0035%) after the second vaccination," the researchers wrote in the paper, published by the European Journal of Heart Failure.

In a generally healthy population, the level would be about 1 percent, the researchers said.

The group experiencing the adverse effects was followed for only 30 days, and half still had unusually high levels of high-sensitivity cardiac troponin T, an indicator of subclinical heart damage, at follow-up.

The long-term implications of the study remain unclear as little research has tracked people over time with heart injury after messenger RNA vaccination, which is known to cause myocarditis and other forms of heart damage.

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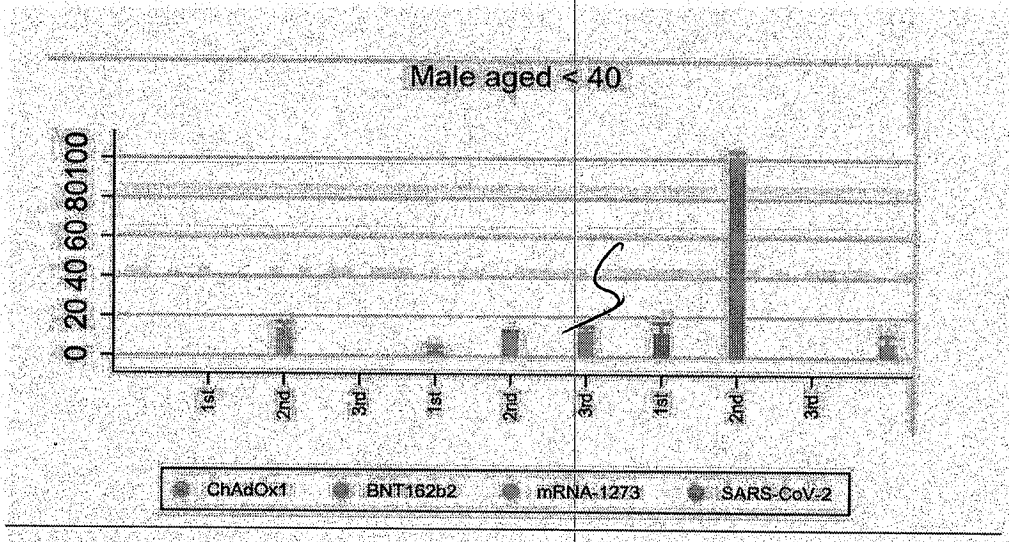
The Truth About Vaccine-induced Myocarditis

As a young male with a platform, it's my responsibility to give voice to the voiceless.

RAV ARORA January 15, 2022

The following essay has been rigorously fact-checked by Stanford medicine professor and infectious disease expert Dr. Jay Bhattacharya. The scientific claims regarding post-vaccine myocarditis are fully in line with the current medical literature. This Substack publication is exclusively devoted to spirituality, mystical experience, and self-actualization. However, I have decided to make an exception and publish a multi-part essay series on the under-recognized truths surrounding vaccine-induced myocarditis. This long-form essay is the first part. I must begin with sharing the straw that broke the camel's back:

A new analysis of relative myocarditis risk by Oxford researchers who published a paper in Nature Medicine on December 14th. The authors find higher rates of vaccine-induced myocarditis than myocarditis from infection in males ages 16 - 39 across multiple vaccine doses: Pfizer dose 2 & 3 and Moderna dose 1 & 2. This graph compares the rates:



However, this analysis understates the vaccine risk in specific demographics. As Dr. Vinay Prasad highlights, if the researchers used more accurate seroprevalence data for viral infection (which would increase the denominator) and analyzed the risk in younger males ages 16-24 specifically (the highest risk group), the risk-benefit ratio would swing further against the administration of the aforementioned vaccine doses in this population.

The government and medical establishment's failure to recognize this basic scientific reality has resulted in numerous young males making irreversible medical decisions resulting in cardiac damage with potential long-term implications. Before even having taken interest in this subject matter, I came to learn of three verified cases of post-vaccine myocarditis in young males requiring hospitalization in my city alone:

A 16-year-old male after dose 2 of Moderna

A 17-year-old male after dose 1 of Pfizer

A 25-year-old male after dose 1 of Moderna

The 25-year-old male, who asked to remain anonymous, was diagnosed with ventricular tachycardia and "high-risk" arrhythmia — dangerously irregular heartbeat which causes the body to receive inadequate oxygenated blood. He spent 5 days in a hospital after enduring throbbing chest pains and difficulty breathing post-vaccination. He now has to take 3 months off from

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work and cannot engage in any form of physical exercise. Doctors have told him even going up the stairs in his house could exacerbate his heart condition.

I personally spoke to him and was devastated by his testimony:

"I felt so pressured to take the vaccine. I wanted to live a normal life and be able to travel where I want to. And now I'm basically unable to do anything without fearing risking my heart condition...my life is ruined for at least the next few months." This 25-year-old man is not part of an exceptionally small minority of vaccine-injured people for whom this side effect could not be anticipated.

As has been long established, myocarditis is the most documented adverse reaction from the Pfizer and Moderna vaccines. A number of studies have established the risk of myocarditis as highly stratified by age and gender. A study from Israel found that males aged 16 to 29 faced the greatest risk, with around 11 in 100,000 males developing post-vaccination myocarditis. A pre-print study last year comparing risks of infection versus vaccination found that boys aged 12 to 15 were four to six times more likely to develop myocarditis from the vaccine than become hospitalized with any Covid-related condition (in the broadest possible sense, including incidental cases — meaning the relative myocarditis risk is likely understated).

The specific point of causality has not been identified by scientists yet, as the vaccines are experimental by nature and their long-term implications are not fully understood. A recent article in the Wall Street Journal compiles the leading hypotheses for what is causing this adverse event. One newly emerging theory relates to the way the vaccine is injected into the body: "The shots are supposed to be injected into the shoulder muscle, also known as the deltoid muscle. If the injection accidentally reaches a vein, it could lead to delivery of some of the vaccine to the heart through blood vessels."

As for the gender-specific risk, some scientists speculate it is due to higher testosterone levels in men:

"That myocarditis appears to happen more among younger males after vaccination than in other age and sex groups suggests a link to the hormone testosterone, which is usually at high levels in younger males, according to researchers. Testosterone may heighten an inflammatory immune response, Dr. Bozkurt said, leading to myocarditis in some male adolescents and young men."

The consistently identified risk in young males across different countries, medical journals, and research institutes warrant serious caution and re-evaluation of fully vaccinating healthy young males — given their extremely low risk of serious illness or death from Covid. Public health officials in Norway, the UK, and Hong Kong have acted with commendable prudence, offering only one dose of the vaccine to young people since myocarditis cases are clustered after the second dose. Other countries such as Finland, France, and Germany have advised against administration of the Moderna vaccine in males under the age of 30 because of higher rates of myocarditis compared to the Pfizer vaccine.

However, both Canada and the United States have adopted a one-size-fits-all policy, making no medically tailored recommendations for teenagers and young adults.

As a 20-year-old healthy male myself, who has suffered from minor heart complications in early adolescence (irregular heart palpitations), I have decided not to take the vaccine. As a result of my personal health decision informed by my physician, my social and physical well-being has been significantly compromised. The Canadian government (both provincial and federal) has implemented coercive and draconian vaccination policies, limiting the freedoms of the unvaccinated across various parts of society.

Much of my social life in Vancouver has been restricted and my ability to maintain physical fitness — a preventative measure that reduces risk of serious Covid illness — has been radically hampered. With the rest of unvaccinated Canadians over 12 years of age, I am barred from exercising at a gym, going to nightclubs, bars, large gatherings, and weddings. Worst, I am now landlocked in Canada and unable to leave the country to do media appearances in the U.S and visit my family in India. I was recently planning to go to Florida to do Ben Shapiro's show, but the government won't even let me board a domestic flight. In what world is this fair?

Under governmental pressure, public organizations have also stepped up their efforts in mandating vaccination for the young. In Ontario, Canada the biggest youth hockey league (OMHA) recently mandated all players 12 and over to be vaccinated. OMHA President Bob Hill gave a statement on the league's decision:

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"We know that the environment around return to play is a real concern for a large proportion of hockey families....Our game is played in an indoor environment where there can be close contact, and we must do everything possible to reduce the risk of any transmission around the rink. It is the duty for our players, our officials and our communities."

Unless one is willing to give their child an insufficiently tested booster shot on a likely 6-month basis, such a rationale being used to push child vaccine mandates falls apart under closer scrutiny. Vaccine efficacy against infection significantly drops over time (an idea which up until last summer was considered right-wing conspiracy). A study published in The Lancet showed a 55% reduction in vaccine effectiveness against infection five months post-vaccination, a trend which spirals downward over time. Any public benefit that child vaccination would bring is temporary and short-lived.

I asked Dr. Mike Hart (known for his appearance on Joe Rogan's podcast), one of my consulting physicians who runs a top medical clinic in Ontario, what he thought about such a mandate:

"I don't think this is a good policy. For vulnerable populations, vaccines make sense; but for young healthy people, the risks of the vaccine may outweigh the benefits.

The risk of myocarditis from COVID is much higher than the risk of myocarditis from the vaccine in the general population, but in younger cohorts, the best available evidence suggests that's not true."

Unfortunately, medical experts such as Dr. Hart who consider both the costs and benefits of the vaccine have been marginalized by spokespeople of the medical establishment who are bizarrely devoted to vaccinating everyone regardless of their individual risk-benefit proposition.

When CNN's chief medical correspondent Dr. Sanjay Gupta appeared on Joe Rogan's podcast and was repeatedly asked about myocarditis risk in young males, he responded with the claim that most myocarditis patients experience mild symptoms and recover quickly. When celebrity physician Dr. Oz was asked the same question by FOX 29 Philadelphia earlier this year, he replied in nearly identical fashion: myocarditis is a mild, easily curable medical condition and shouldn't discourage healthy male teenagers from receiving the vaccine.

However, myocarditis has long been documented as a cause of chronic fatigue, shortness of breath and chest pain, leading to disruptions in physical activity. A number of top cardiologists across the country — such as Dr. John Mandrola, Dr. Amy Kontorovich, and Dr.Venk Murthy — have publicly spoken out against minimization of vaccine-induced myocarditis.

According to Dr. Kontorovich, professor of Medicine and Cardiology at the Icahn School of Medicine at Mount Sinai, "[M]any of those affected are young people who were previously healthy and are now on three or more heart medications and potentially out of work due to symptoms, even if their heart function is 'back to normal.'" University of Michigan cardiologist Dr.Venk Murthy has also noted,

"People with myocarditis are usually counseled to limit activity, placed on 1 or more meds and are at lifetime increased risk of cardiac complications. This can have profound consequences." "[They] are typically told to limit activity for several months, sometimes longer. This means no sports. Some kids are told not to carry books to school."

In attempts to downplay these real, quantifiable risks, those with the most powerful voices in the medical community perform glaringly disprovable sleight-of-hand distortions of the scientific research on mainstream networks. When discussing his viral JRE appearance on Erin Burnett's CNN program, Dr. Sanjay Gupta addressed the public concern of myocarditis for vaccinating teenagers by presenting a study finding infection-induced myocarditis poses a greater risk compared to the vaccine.

A cursory reading of the study reveals it is irrelevant to the cost-benefit analysis of vaccinating healthy young males. The post-vaccination myocarditis rate of 2.7 per 100,000 people is derived from a highly diverse population (in age and gender) with a median age of 38 years in the study. Moreover, the specific age group among the highest at risk of myocarditis — 12 to 15 year olds — was not included in the studied population. The alarming concern is with young males specifically, not the general population. And yet, the CNN segment closed with Erin Burnett summarizing this total falsehood based on Dr. Gupta's stunningly dishonest analysis of the issue:

"The number one [vaccine] risk you do hear about for young boys is myocarditis. You're saying you have about five times greater risk of getting that from Covid than the vaccine. I think that's an incredibly powerful, just basic statistic for people to know."

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Another viral clip of Joe Rogan talking about myocarditis has been exploited by the media to promote their universal vaccination agenda:

The study in the article Rogan looks at finding a higher risk of infection-induced myocarditis than from the vaccine is severely flawed. As practicing physician and epidemiologist Tracy Høeg has pointed out, the authors of the study vastly underestimate both the incidence of Covid infections (thereby exaggerating the infection risk) and post-vaccine myocarditis. The latter is underestimated by a factor of three or four at least.

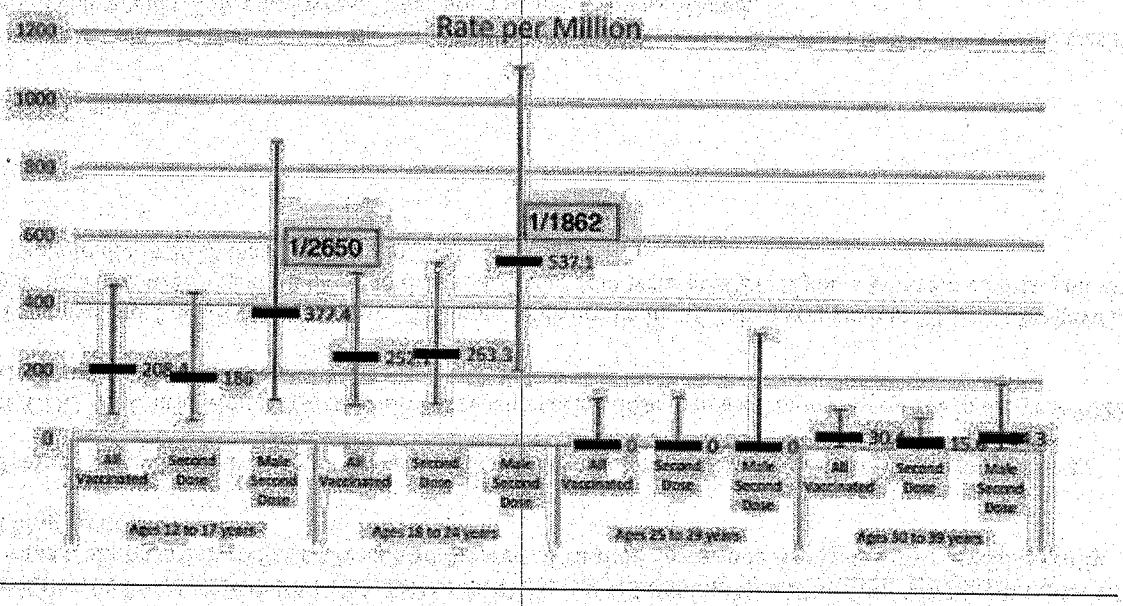
As a result, the authors fallaciously conclude post-infection myocarditis poses a higher risk than post-vaccine myocarditis in young males.

The aforementioned pre-print by Oxford researchers published last month is the most comprehensive, robust, and rigorous analysis of relative myocarditis risk.

Tracy Høeg, MD, PhD @TracyBethHoeg

The 2nd study also said myocarditis occurs after vax at a rate of 66/million in this age group which is not correct. Two studies now, one out of Hong Kong and one out of Kaiser Permanente put that risk at about 380/million for boys 12-17 after dose 2. References in next tweet.

Figure 1: Rate per Million



(Note: Dr. Høeg was the lead researcher in the compelling study finding a four to six times higher incidence of vaccine-related myocarditis than any form of Covid hospitalization in 12 - 15-year-old boys. Read The Guardian's coverage here.)

Similar to our conversations surrounding climate change, criminal justice, and racism, the topic of vaccination has become painfully tribalized along Manichean lines. Any deviation from support of universally mandated vaccination prompts indefensible accusations of being an 'anti-vaxxer' — a reality that came crashing down on 23-year-old unvaccinated NBA player Jonathan Isaac (who has natural immunity) in a misleading Rolling Stone story.

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Neither the risk of Covid or vaccine side effects is equally distributed across the population. While the general risk is minuscule, the individual risk of vaccine-induced myocarditis in young males between the ages of 18 and 24 is roughly 1 in 2,000 according to a recent study by top infectious disease physician Dr. Katie A Sharff. According to this calculation, one million administrations of the vaccine in this age group would yield 500 cases of heart inflammation in kids who were otherwise at near-zero risk of Covid.

The implications of this data are devastating if public health authorities continue to encourage, and worse, mandate boosters for young males as is being done at Princeton, NYU, Stanford, UMass Amherst, Dartmouth, and other major American universities (more on that soon).

Many in the media and medical establishment rightfully promote vaccination to prevent serious illness or death, but react to any information that delegitimizes or questions the safety and efficacy of vaccination in the slightest with a kind of strict religious opposition. "Safe and effective" has become a mantra used to shut down opposition to universal vaccination. Supporting the vaccine means honestly discussing the real risks of vaccination in specific demographics — without either agenda-driven minimization or exaggeration. Obfuscating, downplaying, and misleading the public, on the other hand, undermines trust in the vaccine — a miraculous scientific innovation that has transformed the course of the pandemic by preventing millions of deaths and cases of severe disease.

Honesty, nuance, and compassion are especially needed when it comes to personal health choices. We are only born with one body and we must make medically informed decisions at our own volition without governmental