
Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

illustrate that PHPT is a possible contributor to high CAC score and PHPT should be considered as a contributing factor of high CAC scores even when there is a possible alternative explanation, such as long-term endurance exercise. The first case illustrates "normohormonal PHPT", where an elevated calcium level is associated with a transiently normal but high for the Ca and PTH levels.[4] The reverse or "normocalcemic PHPT" is illustrated by the second case in which the Ca level is normal but the PTH level is elevated. Both obscure the diagnosis of PHPT and are due to variability in PTH levels.[4] The current evidence about the association between PHPT and high CAC score is limited and controversial. Both of our patients were endurance athletes, which probably reflects the bias of patients referred to our practice, but it is possible that exercise plus PHPT produced the higher-than-expected CAC score. Additional studies are needed to investigate the frequency of PHPT in patients with high CAC scores and to examine the relation between physical activity, PHPT, and CAC.

## Disclosures

The authors have no conflict of interest to declare.

Mohammed Elsadany, MD[a]
Paul D. Thompson, MD[b]
[a] Geriatric medicine department, University of Connecticut, Connecticut
[b] Cardiology department, Hartford Hospital, Hartford, Connecticut
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## Myopericarditis After COVID-19 Booster Dose Vaccination


COVID-19 vaccine boosters were recommended by the Centers for Disease Control (CDC) for all populations ≥18 years to provide better protection against circulating variants. The risk of myopericarditis after sequential COVID-19 vaccination needs to be evaluated. We have demonstrated that the Vaccine Safety Datalink rapid cycle analysis method identified a lower incidence of myopericarditis after COVID-19 mRNA vaccine, in part because their search for hospital discharge claims omitted International Classification of Diseases, Tenth Revision codes, and because insurance claims from community hospitals may be delayed by weeks.[1] We provide a more timely and complete case ascertainment of myopericarditis after COVID-19 booster vaccine in populations aged 18 to 39 years.

We studied a cohort of 65,785 Kaiser Permanente Northwest Health Plan members aged 18 to 39 years who received a COVID-19 vaccine booster at least 5 months after completion of the primary series. We identified cases of myopericarditis by searching the electronic health record for the National Center for Health Statistics text label for "myocarditis" or "pericarditis" diagnosis codes in all inpatient and outpatient encounters through January 18, 2022. The cohort was followed for 21 days after their booster. We excluded anyone with a documented diagnosis of myocarditis or pericarditis before their first COVID-19 vaccination. Two physicians independently reviewed the identified patient records and applied the CDC myocarditis and pericarditis surveillance case definition to classify records as confirmed, probable, or excluded on the basis of the previous published definition.[2] Kaiser Permanente's institutional review board approved the study.

Our method identified 6 patients who met the confirmed or probable CDC case definition for acute myocarditis or pericarditis within 21 days of receiving the COVID-19 booster dose among 65,785 eligible members (Table 1). A total of 4 cases occurred in 27,253 men. All 6 patients received a Pfizer vaccine as their booster dose. A total of 5 of 6 patients reported chest pain within 4 days of vaccination, although 1 patient waited until Day 8 to present for her chest pain. Patient number 6 developed chest pain, myocarditis, and cardiogenic shock after his booster dose. Though the illness was attributed to the booster dose, his clinicians are concerned that the dose unmasked an underlying autoimmune condition. Additionally, patient number 5 presented with mild

Table 1
Summary of myopericarditis cases following COVID-19 booster dose

| Case | Age (years) | Sex | Primary series | Boost vaccine | Dose | Interval: Prime series to boost (days) | Chest pain onset (days) | EKG: ST elevation | Trop peak (mcg/L) | LVEF on echo | LOS (days) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 18-24 | M | Pfizer | Pfizer | 3 | 204 | 3 | + | 2.93 | 55-60% | 2 |
| 2 | 18-24 | M | Pfizer | Pfizer | 3 | 260 | 3 | + | Trop I 10.4 | 55% | 1 |
| 3 | 25-29 | M | Pfizer | Pfizer | 3 | 210 | 3 | + | 10.4 | 60% | 1 |
| 4 | 30-39 | F | Pfizer | Pfizer | 3 | 239 | 2 | + | < 0.01 | 55% | 1 |
| 5* | 18-24 | F | J&J | Pfizer | 2 | 196 | 4 | + | .04 | 60-65% | ED |
| 6* | 30-39 | M | Pfizer | Pfizer | 3 | 183 | 19 | − | 17.8 | 35-40% | 4 |

LVEF = left-ventricular end-systolic function; LOS = length of stay; ED = emergency department; Pfizer = Pfizer BioNTech; J&J = Johnson and Johnson. Normal troponin range: <=.03 mcg/L.
* Met CDC case definition, atypical presentation.

myocarditis after a heterologous series of Johnson & Johnson vaccine followed by a booster dose of Pfizer vaccine.

Overall, we estimated 9.1 cases (exact 95% confidence interval [CI] 3.4 to 19.9) of post-booster myopericarditis per 100,000 booster doses given. In men, we estimated 14.7 cases (exact 95% CI 4.0 to 37.6) per 100,000 booster doses given.

We identified a risk of 9.1 cases per 100,000 booster doses. Our small sample size limits the precision of our estimate. This risk is higher than previous estimates reported by Vaccine Adverse Event Reporting System which identified 54 preliminary reports of vaccine-related myopericarditis; 12 confirmed and 38 under review, after 26.3 million booster doses administered across all ages, with an unadjusted estimate of 0.21 cases per 100,000 doses (95% CI 0.15 to 0.27).[3] Vaccine Adverse Event Reporting System is passive system relying on patients or providers to report; but limitations include both over- and under-reporting.[4] Active surveillance by the Vaccine Safety Datalink has not yet reported a risk of myopericarditis after booster vaccinations, although we would anticipate underestimation because of limitations in their methods.[1] Israel reported the risk of myopericarditis after booster dose as 4.7 cases per 100,000 in men aged 20 to 24.[5]

Myopericarditis occurs after booster doses and may be underreported by current surveillance methods. Completeness or high sensitivity of these case estimates are essential when modeling risk and benefit for wide-scale vaccine implementation and sequential COVID-19 vaccinations for the general population.

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The authors have no conflicts of interest to declare.

Katie A. Sharff, MD[a]
David M. Dancoes, BS[b]
Jodi L. Longueil, PharmD[c]
Paul F. Lewis, MD, MPH[d]
Eric S. Johnson, PhD[b]

[a] Department of Infectious Diseases, Kaiser Permanente Northwest, Portland, Oregon
[b] Department of Analytics, Kaiser Permanente Northwest, Portland, Oregon
[c] Division of Pharmacy, Kaiser Permanente Northwest, Portland, Oregon
[d] Department of Pediatrics, Kaiser Permanente Northwest, Portland Oregon

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## Transcatheter Aortic Valve Implantation in Patients With Previous Coronary Artery Bypass Grafting

Several mechanisms that drive coronary artery disease (CAD) and severe aortic stenosis (AS) are similar, with a substantial portion of patients referred for transcatheter aortic valve implantation (TAVI) in pivotal trials having a history of coronary artery bypass grafting (CABG).[1] TAVI has emerged as an alternative treatment strategy to treat AS compared with surgical aortic valve replacement patients with previous CABG to avoid the need for redo sternotomy, with similar in-hospital and short-term outcomes.[2,3] We aimed to evaluate intermediate-term outcomes in patients with and without a history of CABG who underwent TAVI.

We performed a single-center retrospective analysis of patients with severe AS who underwent TAVI from January 1, 2012, to July 31, 2020, and had follow-up through August 31, 2020. Patients with a history of CABG were identified before the index TAVI procedure through chart review. The primary outcome was all-cause mortality. Secondary outcomes included 30-day permanent pacemaker rate, overall stroke and myocardial infarction, and all-cause bleeding and heart failure (HF) hospitalizations. Descriptive analysis, baseline differences, and Kaplan−Meier survival analysis were performed between patients with and without CABG.

A total of 915 patients met the inclusion criteria. The average age of the cohort was 78.5 ± 9.8 years, with no significant differences in the groups. Patients who underwent TAVI with a history of CAD comprised 63% of the cohort, with 21% of patients having a history of CABG and 29% with a history of percutaneous coronary intervention (Figure 1). Patients with a history of CABG were predominantly male (83.5% vs 52.3%, p <00001) and had significantly higher Society of Thoracic Surgeon scores (6.5% vs 5.5%, p <0.0001).

At a mean follow-up of 2.4 years, all-cause rate of mortality was 18.5% versus 17.5% (p = 0.75) in patients with versus without CABG. Unadjusted Kaplan-Meier curves for all-cause mortality are presented in Figure 1. Clinical outcomes are presented in Figure 1. Rates of 30-day permanent pacemaker were similar between the 2 groups (3.1% vs 2.6%, p = 0.7). Overall stroke rate was also similar in the 2 groups (2.2 vs 1.5%, p = 0.58). There were similar rates of overall myocardial infarction post-TAVI (1.5% vs 1.6%, p = 0.93), bleeding related hospitalizations (5.7% vs 5.2%, p = 0.78) and HF hospitalizations (10.5% vs 10.4%, p = 0.96) in patients with versus without previous CABG.

Our study demonstrates several key findings to help understand outcomes in patients with previous CABG who underwent TAVI. We demonstrate similar rates of all-cause mortality and statistically insignificant differences in intermediate-term outcomes of overall stroke, bleeding, or HF-related