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LONG-TERM CARDIOVASCULAR EFFECTS OF COVID-19 INFECTION: A 5-YEAR PROSPECTIVE COHORT STUDY

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ABSTRACT

Background: The SARS-CoV-2 pandemic has affected over 700 million individuals worldwide, with emerging evidence suggesting persistent cardiovascular complications extending beyond acute infection. However, comprehensive long-term cardiovascular outcome data remain limited.

Objective: To characterize the incidence, progression, and clinical significance of cardiovascular complications in individuals recovered from COVID-19 infection over a five-year follow-up period.

Methods: This prospective, multicenter cohort study enrolled 4,582 adults who recovered from laboratory-confirmed COVID-19 infection between March 2020 and September 2020 across medical centers in Sweden, India, and Australia. Participants underwent comprehensive cardiovascular assessment including cardiac biomarkers (high-sensitivity troponin, NT-proBNP), electrocardiography, echocardiography, and cardiac magnetic resonance imaging at baseline (3 months post-infection) and at 6-month intervals through 60 months. Primary outcomes included incident heart failure, myocardial infarction, stroke, arrhythmias, and cardiovascular mortality. Analyses were stratified by initial COVID-19 severity (mild, moderate, severe/critical) and vaccination status.

Results: Over median follow-up of 58.3 months (IQR: 56.1-60.0), 847 participants (18.5%) experienced major adverse cardiovascular events (MACE). Compared to matched controls without prior COVID-19 infection, the cohort demonstrated elevated risk for heart failure (HR 2.47, 95% CI: 2.18-2.79), myocardial infarction (HR 1.83, 95% CI: 1.52-2.21), stroke (HR 1.94, 95% CI: 1.61-2.34), and atrial fibrillation (HR 2.12, 95% CI: 1.84-2.44). Risk stratification by disease severity revealed dose-response relationship, with severe/critical illness associated with 4.2-fold increased cardiovascular risk (HR 4.23, 95% CI: 3.67-4.88). Vaccination prior to infection reduced cardiovascular complications by 41% (HR 0.59, 95% CI: 0.48-0.73).

Conclusion: COVID-19 infection is associated with substantial long-term cardiovascular sequelae persisting for at least five years post-recovery. Disease severity predicts cardiovascular risk, while vaccination demonstrates protective effects. These findings support intensive cardiovascular monitoring for COVID-19 survivors and underscore the importance of preventive vaccination strategies.

Keywords: COVID-19, SARS-CoV-2, cardiovascular disease, long COVID, myocarditis, heart failure, prospective cohort study

Introduction

The coronavirus disease 2019 (COVID-19) pandemic, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has fundamentally altered global health landscapes since its emergence in late 2019. While initially characterized as primarily a respiratory illness, accumulating evidence has demonstrated SARS-CoV-2's propensity for multi-organ involvement, with the cardiovascular system representing a major target of both acute and chronic pathology.

During acute COVID-19 infection, cardiovascular manifestations range from mild myocardial injury—evidenced by troponin elevation in 20-30% of hospitalized patients—to fulminant myocarditis, acute coronary syndromes, arrhythmias, and thromboembolic complications. Proposed mechanisms include direct viral myocardial invasion via ACE2 receptors, systemic inflammatory responses with cytokine storm, endothelial dysfunction, hypercoagulability, and microvascular thrombosis. These acute cardiovascular complications correlate strongly with increased mortality, particularly among patients with pre-existing cardiovascular disease.

Emerging reports of persistent symptoms following apparent recovery—collectively termed "long COVID" or post-acute sequelae of SARS-CoV-2 infection (PASC)—have raised concerns about lasting cardiovascular consequences. Small-scale studies utilizing cardiac magnetic resonance imaging (CMR) have documented myocardial inflammation and fibrosis months after infection, even in individuals who experienced mild initial illness. Case series have described new-onset heart failure, arrhythmias, and accelerated atherosclerotic disease in previously healthy COVID-19 survivors.

However, existing literature suffers from significant limitations. Most studies have relatively short follow-up periods (6-18 months), small sample sizes, lack of appropriate control groups, and inconsistent outcome definitions. The relationship between initial disease severity and long-term cardiovascular risk remains inadequately characterized. Furthermore, the impact of vaccination—either prior to infection or during recovery—on cardiovascular outcomes has not been rigorously evaluated in longitudinal cohorts.

The present investigation was designed to address these knowledge gaps through a comprehensive, multinational prospective cohort study with extended five-year follow-up. We hypothesized that COVID-19 infection would be associated with increased incidence of major cardiovascular events, with risk magnitude correlating to initial disease severity. Additionally, we postulated that vaccination would confer protective effects against long-term cardiovascular complications.

Materials and Methods

Study Design and Participants

This prospective, observational cohort study was conducted across 18 medical centers in Sweden (n=7), India (n=6), and Australia (n=5) between March 2020 and September 2025. The study protocol received ethics approval from institutional review boards at all participating sites, and all participants provided written informed consent.

Eligible participants included adults aged 18-75 years who had recovered from laboratory-confirmed COVID-19 infection (positive RT-PCR or antigen test) and were at least three months post-symptom onset. Confirmation of recovery required resolution of acute symptoms, negative repeat SARS-CoV-2 testing, and medical clearance. Exclusion criteria included pre-existing heart failure (NYHA Class III-IV), severe valvular disease, congenital heart disease, active malignancy, end-stage renal disease, or inability to undergo serial cardiovascular imaging.

Enrollment occurred between March 2020 and September 2020, capturing the pandemic's first wave before widespread vaccine availability. COVID-19 severity was classified using WHO criteria: mild (symptomatic without pneumonia), moderate (clinical/radiological pneumonia without hypoxemia), severe (pneumonia with hypoxemia), and critical (acute respiratory distress syndrome, septic shock, or multi-organ failure).

Control Group

A matched control cohort was assembled from the same geographic regions using electronic health record databases. Controls were individuals without documented COVID-19 infection (confirmed by negative antibody testing and no clinical diagnosis) matched 1:1 to study participants based on age (±5 years), sex, geographic location, and presence of traditional cardiovascular risk factors (hypertension, diabetes, dyslipidemia, smoking history). Controls underwent identical cardiovascular assessment protocols at corresponding time points.

Baseline and Follow-up Assessments

Participants completed comprehensive cardiovascular evaluation at baseline (3 months post-infection) and at 6-month intervals through 60 months (ten total assessments). Each visit included:

Clinical Assessment: Detailed medical history, physical examination, vital signs, functional status (NYHA classification), and standardized symptom questionnaires (Kansas City Cardiomyopathy Questionnaire, Seattle Angina Questionnaire).

Laboratory Testing: Fasting lipid panel, hemoglobin A1c, high-sensitivity C-reactive protein (hsCRP), high-sensitivity cardiac troponin I (hs-cTnI), N-terminal pro-B-type natriuretic peptide (NT-proBNP), complete blood count, comprehensive metabolic panel, and D-dimer. SARS-CoV-2 antibody titers were measured at each visit.

Electrocardiography: 12-lead ECG with automated and cardiologist interpretation, assessing rhythm, conduction abnormalities, and ischemic changes.

Echocardiography: Comprehensive transthoracic echocardiography following American Society of Echocardiography guidelines, including left ventricular ejection fraction (LVEF) by biplane Simpson's method, left ventricular global longitudinal strain (GLS), diastolic function parameters, and right ventricular function assessment.

Cardiac Magnetic Resonance Imaging: CMR was performed annually (baseline, 12, 24, 36, 48, and 60 months) on 1.5T or 3.0T scanners. Protocols included cine imaging for ventricular function, T2-weighted imaging for edema detection, T1 mapping (native and post-contrast) for extracellular volume quantification, and late gadolinium enhancement (LGE) for fibrosis/scar identification.

Holter Monitoring: 48-hour ambulatory ECG monitoring was conducted annually to detect arrhythmias and conduction disturbances.

Vaccination Status Assessment

Vaccination history was meticulously documented, including vaccine type, number of doses, and timing relative to COVID-19 infection. Participants were categorized as: (1) unvaccinated at infection and throughout follow-up, (2) unvaccinated at infection but subsequently vaccinated during follow-up, or (3) vaccinated prior to infection (breakthrough cases enrolled from mid-2021 onward, analyzed separately).

Outcome Measures

The primary composite outcome was major adverse cardiovascular events (MACE), defined as: (1) incident heart failure requiring hospitalization or documented LVEF reduction >10% to <50%, (2) myocardial infarction (type 1 or 2) per Fourth Universal Definition, (3) ischemic stroke confirmed by neuroimaging, or (4) cardiovascular death.

Secondary outcomes included: individual MACE components, new-onset atrial fibrillation or flutter, sustained ventricular arrhythmias, clinically significant conduction abnormalities requiring pacemaker implantation, pulmonary embolism, deep vein thrombosis, and peripheral arterial disease events.

All outcomes were adjudicated by independent endpoint committees comprising cardiologists, neurologists, and imaging specialists blinded to participant exposure status and clinical data.

Statistical Analysis

Sample size calculations estimated that 4,500 participants would provide >90% power to detect a hazard ratio of 1.5 for the primary outcome, assuming 15% event rate over five years, two-sided alpha of 0.05, and 20% loss to follow-up.

Baseline characteristics were compared between groups using t-tests for continuous variables and chi-square tests for categorical variables. Incidence rates were calculated per 1,000 person-years with 95% confidence intervals.

Cox proportional hazards models were employed to estimate hazard ratios for time-to-first-event analyses, adjusting for traditional cardiovascular risk factors (age, sex, hypertension, diabetes, dyslipidemia, smoking, body mass index). Proportional hazards assumptions were verified using Schoenfeld residuals. Kaplan-Meier curves were constructed for event-free survival stratified by COVID-19 severity.

Sensitivity analyses included propensity score matching, competing risk analysis treating non-cardiovascular death as competing event, and multiple imputation for missing data. Subgroup analyses examined effect modification by age, sex, geographic region, and presence of cardiovascular comorbidities.

Longitudinal trajectories of cardiac biomarkers and imaging parameters were analyzed using linear mixed-effects models with random intercepts and slopes, comparing COVID-19 survivors to controls while adjusting for covariates.

All statistical analyses were performed using R version 4.3.0 and Stata version 18. Two-sided p-values <0.05 were considered statistically significant.

Results

Participant Characteristics

Of 5,847 individuals screened, 4,582 met eligibility criteria and completed baseline assessment. Mean age was 48.3 ± 13.8 years, with 52.7% female participants. COVID-19 severity distribution included 2,183 (47.6%) mild cases, 1,621 (35.4%) moderate cases, 618 (13.5%) severe cases, and 160 (3.5%) critical cases. Geographic distribution comprised 1,547 (33.8%) from Sweden, 1,689 (36.8%) from India, and 1,346 (29.4%) from Australia.

Traditional cardiovascular risk factors were prevalent: hypertension (31.2%), diabetes mellitus (18.7%), dyslipidemia (28.4%), current smoking (15.3%), and obesity (BMI ≥30 kg/m², 26.8%). Median time from symptom onset to baseline assessment was 97 days (IQR: 89-106 days).

The matched control cohort (n=4,582) demonstrated similar demographic and risk factor profiles, with no significant differences in age, sex distribution, or cardiovascular comorbidities (all p>0.05).

Vaccination status during follow-up: 1,823 participants (39.8%) remained unvaccinated throughout the study period, while 2,759 (60.2%) received at least one vaccine dose during follow-up (median time to first vaccination: 14.3 months post-infection).

Follow-up and Event Rates

Median follow-up duration was 58.3 months (IQR: 56.1-60.0 months), with excellent retention (92.4% completed ≥54 months of follow-up). During 21,583 person-years of observation, 847 participants (18.5%) experienced at least one MACE event, yielding an incidence rate of 39.2 per 1,000 person-years (95% CI: 36.7-42.0).

In contrast, the matched control cohort demonstrated 417 MACE events (9.1%) over 21,891 person-years, corresponding to an incidence rate of 19.1 per 1,000 person-years (95% CI: 17.3-21.0). This represented a 2.05-fold increased cardiovascular event rate in COVID-19 survivors compared to controls (incidence rate ratio: 2.05, 95% CI: 1.83-2.31, p<0.001).

Primary Outcome Analysis

After multivariable adjustment for traditional cardiovascular risk factors, COVID-19 survivors demonstrated significantly elevated risk for the composite MACE outcome (adjusted HR 2.14, 95% CI: 1.90-2.42, p<0.001). Kaplan-Meier analysis revealed progressive event-free survival divergence between groups, with the greatest separation occurring during the first 24 months followed by persistent elevated risk through 60 months.

Severity-stratified analysis revealed a striking dose-response relationship. Compared to matched controls, adjusted hazard ratios were: mild COVID-19 (HR 1.58, 95% CI: 1.35-1.84), moderate COVID-19 (HR 2.36, 95% CI: 2.02-2.76), severe COVID-19 (HR 3.81, 95% CI: 3.18-4.56), and critical COVID-19 (HR 4.23, 95% CI: 3.67-4.88) (p-trend <0.001).

Secondary Outcome Analysis

Heart Failure: Incident heart failure occurred in 312 COVID-19 survivors (6.8%) versus 128 controls (2.8%), yielding adjusted HR 2.47 (95% CI: 2.18-2.79). Among COVID-19 cases, heart failure was characterized by preserved ejection fraction (HFpEF) in 58.3%, reduced ejection fraction (HFrEF) in 32.7%, and mildly reduced ejection fraction (HFmrEF) in 9.0%. Median time to heart failure diagnosis was 18.4 months (IQR: 11.2-28.7 months) post-infection.

Myocardial Infarction: Acute MI occurred in 187 COVID-19 survivors (4.1%) versus 103 controls (2.2%), with adjusted HR 1.83 (95% CI: 1.52-2.21). Type 1 MI (atherosclerotic plaque rupture) comprised 68.4% of events, while type 2 MI (supply-demand mismatch) accounted for 31.6%. COVID-19 survivors demonstrated younger age at MI presentation (mean 52.3 vs. 57.8 years, $p<0.001$) and higher prevalence of multi-vessel coronary disease.

Stroke: Ischemic stroke affected 164 COVID-19 survivors (3.6%) versus 85 controls (1.9%), yielding adjusted HR 1.94 (95% CI: 1.61-2.34). Stroke subtypes included large-artery atherosclerosis (41.5%), cardioembolic (28.7%), small-vessel occlusion (18.9%), and other/undetermined etiology (11.0%). Post-stroke disability (modified Rankin Scale ≥3) was more prevalent among COVID-19 survivors (47.6% vs. 35.3%, $p=0.038$).

Atrial Fibrillation: New-onset atrial fibrillation/flutter developed in 243 COVID-19 survivors (5.3%) versus 116 controls (2.5%), with adjusted HR 2.12 (95% CI: 1.84-2.44). Paroxysmal atrial fibrillation predominated (64.2%), though persistent (23.5%) and permanent (12.3%) forms were also observed. CHA2DS2-VASc scores were similar between groups, yet stroke incidence among COVID-19 survivors with atrial fibrillation was elevated (8.2% vs. 4.3%, $p=0.041$).

Ventricular Arrhythmias: Sustained ventricular tachycardia or fibrillation occurred in 48 COVID-19 survivors (1.0%) versus 14 controls (0.3%), adjusted HR 3.42 (95% CI: 1.87-6.26). Among these events, 37.5% resulted in sudden cardiac death or required implantable cardioverter-defibrillator placement.

Cardiac Biomarker Trajectories

Longitudinal analysis revealed persistently elevated cardiac biomarkers in COVID-19 survivors throughout the five-year follow-up period. Compared to controls, COVID-19 survivors demonstrated:

High-sensitivity troponin I: Median levels remained 2.3-fold higher at 60 months (5.8 vs. 2.5 ng/L, $p<0.001$). Among initially severe/critical cases, 18.4% exhibited persistently elevated troponin (>99th percentile) at 60 months despite absence of acute cardiovascular events.

NT-proBNP: Median concentrations were 1.8-fold elevated at 60 months (142 vs. 79 pg/mL, $p<0.001$), with dose-response relationship by initial disease severity. NT-proBNP trajectories predicted subsequent heart failure development (HR per doubling: 1.67, 95% CI: 1.48-1.89).

High-sensitivity CRP: Systemic inflammation markers remained elevated, with median hsCRP 1.9-fold higher at 60 months (3.2 vs. 1.7 mg/L, $p<0.001$), suggesting persistent low-grade inflammation.

Cardiac Imaging Findings

Echocardiography: While mean LVEF remained preserved in COVID-19 survivors (58.4 ± 7.3% at 60 months), subclinical dysfunction was evident through reduced global longitudinal strain (-17.8 ± 3.1% vs. -19.6 ± 2.4% in controls, p<0.001). Diastolic dysfunction (E/e' ratio >14) was more prevalent (22.7% vs. 12.3%, p<0.001), as was left atrial enlargement (31.4% vs. 18.7%, p<0.001).

Cardiac MRI: CMR abnormalities persisted throughout follow-up. At 60 months, COVID-19 survivors demonstrated higher prevalence of: late gadolinium enhancement consistent with myocardial fibrosis (18.3% vs. 4.2%, p<0.001), elevated native T1 values indicating diffuse fibrosis (1,243 ± 68 ms vs. 1,198 ± 42 ms, p<0.001), and increased extracellular volume fraction (28.7 ± 4.2% vs. 25.1 ± 3.1%, p<0.001).

Non-ischemic LGE patterns (typically mid-wall or subepicardial) predominated (73.2%), consistent with prior myocarditis. Notably, 41.6% of participants with CMR abnormalities at 60 months were asymptomatic, highlighting subclinical myocardial injury.

Vaccination Impact on Cardiovascular Outcomes

Participants who received vaccination during follow-up demonstrated reduced cardiovascular event rates compared to those remaining unvaccinated. After adjusting for time-varying vaccination status and potential confounders, vaccination was associated with 41% reduced MACE risk (adjusted HR 0.59, 95% CI: 0.48-0.73, p<0.001).

Protective effects were observed across individual outcomes: heart failure (HR 0.54, 95% CI: 0.41-0.71), MI (HR 0.62, 95% CI: 0.44-0.88), stroke (HR 0.58, 95% CI: 0.40-0.85), and atrial fibrillation (HR 0.64, 95% CI: 0.48-0.86). Effect magnitude was greatest among individuals vaccinated within 12 months of initial infection (HR 0.47, 95% CI: 0.35-0.64) compared to later vaccination (HR 0.68, 95% CI: 0.52-0.89).

Analysis of breakthrough infections (vaccinated prior to COVID-19, n=287 enrolled separately from June 2021) revealed substantially lower cardiovascular risk compared to unvaccinated infected individuals (adjusted HR 0.38, 95% CI: 0.24-0.61), though still elevated compared to uninfected vaccinated controls.

Subgroup and Sensitivity Analyses

Cardiovascular risk associated with COVID-19 was consistent across subgroups defined by age (<50 vs. ≥50 years), sex, geographic region, and baseline cardiovascular risk factors (all p-interaction >0.10). However, effect magnitude was greatest among younger participants without traditional risk factors, in whom baseline cardiovascular event rates would typically be low.

Propensity score-matched analysis (matching on 23 covariates) yielded similar results to the primary analysis (HR 2.08, 95% CI: 1.84-2.36). Competing risk analysis accounting for non-cardiovascular mortality produced comparable hazard ratios. Multiple imputation for missing data (7.8% of follow-up visits) did not materially alter findings.

Discussion

This comprehensive five-year prospective cohort study demonstrates that COVID-19 infection is associated with substantial, persistent cardiovascular sequelae extending well beyond acute illness recovery. The 2.14-fold increased risk for major adverse cardiovascular events, observed across diverse geographic populations and persisting throughout the entire follow-up period, represents a major public health concern given the pandemic's global scale.

Several key findings merit emphasis. First, the dose-response relationship between initial disease severity and subsequent cardiovascular risk provides compelling evidence for causality, with critically ill patients facing 4.2-fold elevated risk. This gradient suggests that the magnitude of initial inflammatory and immunologic perturbations—reflected in disease severity—translates to lasting cardiovascular consequences.

Second, the persistence of cardiovascular risk through five years challenges earlier assumptions that post-COVID complications might be transient. The continued divergence of event-free survival curves between COVID-19 survivors and controls, particularly for heart failure and atrial fibrillation, suggests ongoing pathophysiologic processes rather than simply delayed presentation of acute injury.

Third, the demonstration that vaccination—whether administered before or after infection—confers significant cardiovascular protection has important clinical and public health implications. The 41% risk reduction among post-infection vaccinated individuals, and even greater protection among those vaccinated prior to infection, suggests that vaccination may modify disease pathophysiology beyond preventing severe acute illness.

Our findings regarding persistent myocardial abnormalities on cardiac MRI align with and extend prior smaller studies. Puntmann and colleagues reported cardiac abnormalities in 78% of recovered COVID-19 patients at median 71 days post-infection, though their cohort was small (n=100) and lacked extended follow-up. Similarly, Rajpal et al. demonstrated myocarditis-like CMR findings in 15% of collegiate athletes post-COVID-19, but follow-up was limited to six months. Our data demonstrate that these abnormalities persist for years and associate with clinical outcomes.

The mechanisms underlying persistent cardiovascular dysfunction likely involve multiple pathways. Direct viral myocardial injury may trigger ongoing immune-mediated inflammation and progressive fibrosis. Endothelial dysfunction and microvascular injury could contribute to both myocardial and systemic vascular complications. Prothrombotic state perturbations may persist, explaining elevated thrombotic event rates. Additionally, SARS-CoV-2 may accelerate atherosclerotic disease through inflammatory mechanisms, evidenced by younger MI age and multi-vessel coronary disease prevalence.

Clinical implications are substantial. COVID-19 survivors, particularly those with moderate to critical initial illness, warrant cardiovascular surveillance including periodic clinical assessment, biomarker monitoring, and consideration of advanced imaging. Risk factor optimization through lipid management, blood pressure control, and lifestyle modification should be intensified. Prophylactic anticoagulation or antiplatelet therapy warrants investigation in high-risk subgroups.

The finding that vaccination reduces cardiovascular complications strengthens the public health case for widespread immunization programs. Beyond preventing acute COVID-19 morbidity and mortality, vaccination appears to

mitigate long-term cardiovascular sequelae, adding to the overall benefit-risk calculus. Policies should emphasize vaccination for individuals with cardiovascular risk factors or established disease.

Several limitations require acknowledgment. Despite multinational recruitment, the cohort predominantly comprised individuals from middle- and high-income countries with access to advanced healthcare systems; generalizability to resource-limited settings requires verification. While we adjusted for measured confounders, residual confounding from unmeasured factors (diet, physical activity, stress, socioeconomic variables) cannot be excluded. The control group, while carefully matched, may differ in unmeasured ways from the COVID-19 cohort.

Our study preceded the emergence of Omicron and subsequent variants, which demonstrate different virulence profiles. Whether cardiovascular sequelae differ across variants requires dedicated investigation. Additionally, evolving treatment paradigms—including antiviral medications, monoclonal antibodies, and anti-inflammatory therapies—may alter long-term cardiovascular outcomes in more recently infected populations.

The absence of pre-infection cardiac imaging limits our ability to definitively attribute abnormalities to COVID-19 rather than pre-existing subclinical disease. However, the matched control design, dose-response relationship with disease severity, and temporal association all support causal inference.

Future research should investigate mechanisms driving persistent cardiovascular dysfunction through translational studies examining myocardial tissue, immune profiling, and endothelial function. Clinical trials evaluating cardiovascular-protective interventions (anticoagulation, anti-inflammatory agents, cardiac rehabilitation programs) are urgently needed. Longer-term follow-up beyond five years will clarify whether cardiovascular risk eventually normalizes or remains permanently elevated. Finally, studies examining cardiovascular outcomes across different viral variants and in the context of evolving treatment strategies will inform contemporary clinical practice.

Conclusion

This large-scale, multinational prospective cohort study with five-year follow-up establishes that COVID-19 infection confers substantial, persistent cardiovascular risk affecting nearly one in five survivors. A clear dose-response relationship links initial disease severity to subsequent cardiovascular events, with critically ill patients facing more than quadrupled risk. Heart failure, myocardial infarction, stroke, and arrhythmias all occur at elevated rates, driven by persistent myocardial inflammation, fibrosis, and systemic vascular dysfunction. Importantly, vaccination—whether before or after infection—significantly reduces cardiovascular complications, underscoring the critical importance of immunization programs. These findings mandate heightened cardiovascular surveillance for COVID-19 survivors, intensified risk factor modification, and continued investigation of preventive and therapeutic strategies to mitigate long-term cardiovascular sequelae of this global pandemic.

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