The Egyptian Journal of Hospital Medicine (April 2025) Vol. 99, Page 2169-2172

# Cardiac Safety after AstraZeneca COVID-19 Vaccination: A Cohort Observational Study

**Khalid Karem *, Mina Barsoum, Sameh Sabet, Viola Willium**
Cardiology Department, Faculty of Medicine, Ain Shams University, Cairo, Egypt
*** Corresponding author:** Khalid Karem, **Email:** khalidkarem@med.asu.edu.eg, **Phone:** +20 100 549 9479

## ABSTRACT

**Background:** Myocarditis, an inflammatory myocardial disorder, is commonly associated with viral infections and has been reported following various vaccinations, particularly the smallpox vaccine. Recent concerns have emerged regarding the potential cardiac implications of COVID-19 mRNA vaccines. Nevertheless, evidence regarding the cardiovascular implications of AstraZeneca COVID-19 vaccine remains scarce.

**Aim:** This study aimed to compare left ventricular (LV) systolic function between AstraZeneca COVID-19 vaccine recipients and matched unvaccinated healthy controls.

**Patients and methods:** Over the course from June 2021 to February 2022, this prospective observational investigation was executed at Ain Shams University Hospital, enrolling 150 patients who were randomly and evenly split into two parallel arms: A vaccinated group (n=75) who received a complete two-dose regimen of AstraZeneca COVID-19 vaccine and a control group (n=75) who did not receive any COVID-19 vaccine. Subjects presenting with either established cardiac pathology or recent COVID-19 infection were excluded. Global longitudinal strain (GLS), mitral annular systolic velocity (S′), and left ventricular ejection fraction (LVEF) were assessed via transthoracic echocardiography, speckle-tracking analysis, and tissue Doppler imaging.

**Results:** No substantial differences were observed between the vaccinated and controls regarding LVEF (63.20 ± 4.09% vs. 62.99 ± 3.59%; p=0.735), GLS (-19 [-20 to -17] vs. -19 [-20 to -18]; p=0.365), or mitral annular S′ (9.56 ± 2.07 cm/sec vs. 9.99 ± 1.95 cm/sec; p=0.196).

**Conclusions:** Echocardiographic assessment revealed no evidence of myocardial dysfunction in relation to AstraZeneca COVID-19 vaccination, supporting its favorable cardiac safety.

**Keywords:** AstraZeneca vaccine, Myocarditis, Left ventricular function, Echocardiography, COVID-19 vaccination.

## INTRODUCTION

Myocarditis is most frequently attributed to viral infections, notably parvovirus B-19 and influenza. Among reported vaccine-related adverse events, this has been particularly linked to the smallpox vaccine, which demonstrates the highest incidence [1].

Evidence from the Israeli Ministry of Health highlights a limited number of myocarditis cases temporally associated with COVID-19 mRNA vaccination. Among approximately five million vaccinated individuals in Israel, only 62 cases of myocarditis were reported. The onset was primarily associated with the second vaccine dose, with young males under 30 years constituting the majority of reported cases. Two of these patients unfortunately succumbed to the condition [2].

In a report by the U.S. Department of Defense, 14 cases of myocarditis were identified in military members who had received mRNA-based COVID-19 vaccines. The entirety of reported cases was attributed to mRNA COVID-19 vaccines, with the Moderna vaccine accounting for the majority and a smaller subset linked to the Pfizer/BioNTech vaccine [2].

Currently, the scientific literature lacks documented evidence linking COVID-19 vaccination to the development of myocarditis [3, 4].

In light of these concerns, this study aimed to compare LV systolic function between AstraZeneca COVID-19 vaccine recipients and matched unvaccinated healthy controls. Among the various COVID-19 vaccines deployed in Egypt, the AstraZeneca vaccine ranks among the most extensively administered.

## PATIENTS AND METHODS

Spanning an 8-month period from June 2021 to February 2022, this prospective observational study was conducted at Ain Shams University Hospital.

A total of 150 participants were recruited and randomly allocated in a 1:1 ratio into 2 groups. Study group comprised 75 individuals aged 20 to 50 years who completed the full AstraZeneca vaccination protocol. Assessment was carried out within two weeks after the second vaccine dose. Control group comprised 75 individuals matched for age and sex, none of whom had received any form of COVID-19 vaccination.

**Exclusion criteria:** Individuals with a history of cardiac disease—including ischemic heart disease, valvular abnormalities, cardiomyopathies, or multiple cardiovascular risk factors—as well as those with a recent episode of COVID-19 infection.



Received: 11/01/2025
Accepted: 10/03/2025

All subjects underwent systematic history taking and physical examination. A complete transthoracic echocardiogram was performed using a Vivid E95 ultrasound system (GE Healthcare) with a 3.5 MHz transducer, adhering to the standardized criteria set by the American Society of Echocardiography [5].

Under proper ECG-gated acquisition, echocardiographic imaging was performed from standard apical four-, two-, and three-chamber perspectives, with the patient maintained in the left lateral position.

**1)** Quantification of LV volumes was achieved by delineating the endocardial borders at end-systolic and end-diastolic phases in the apical four-chamber view, followed by EF calculation using the modified biplane Simpson's technique (**Figure 1).**

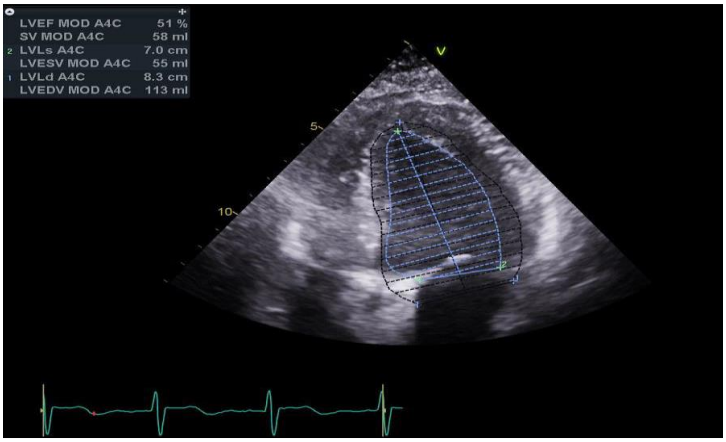


**Figure (1):** LV volumes from a study group person showing normal LV volumes.

**2)** Measurement of S′ wave velocities at the lateral and septal mitral annular sites was performed using tissue Doppler imaging, as depicted in figure (2).

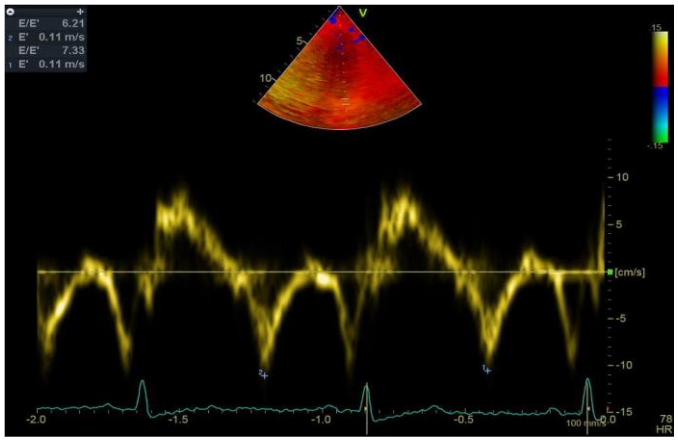


**Figure (2):** TDI from a study group.

**3)** 2D speckle tracking echocardiography was conducted using cine loops acquired from apical four-, three-, and two-chamber views. Offline analysis was subsequently performed using EchoPAC software to compute the GLS of LV, as illustrated in figure (3).

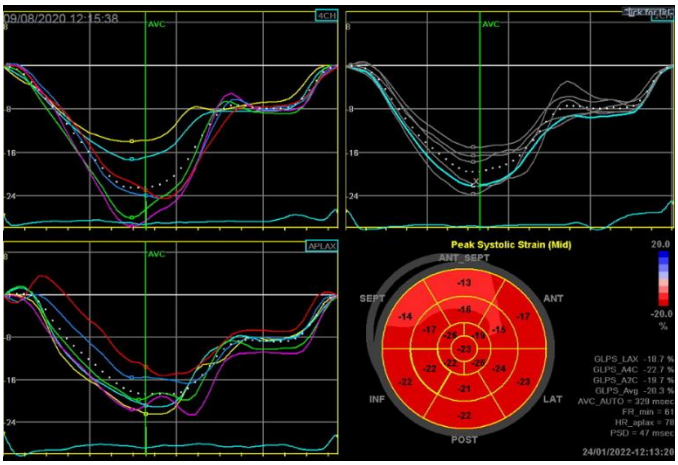


**Figure (3):** Global longitudinal strain (Bull's eye) of a normal control group.

**Ethical considerations: The study was done after being accepted by The Research Ethics Committee, Ain Shams University (Approval Code: FMASU MS 479/2021). All patients provided written informed consents prior to their enrolment. The consent form explicitly outlined their agreement to participate in the study and for the publication of data, ensuring protection of their confidentiality and privacy. This work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.**

***Statistical analysis***

Statistical analysis was performed using IBM SPSS Statistics software, version 27 (IBM Corp., Armonk, NY, USA). The distribution of quantitative data was evaluated using the Shapiro–Wilk test, supplemented by visual inspection of histograms and Q–Q plots. For continuous data, variables exhibiting normal distribution were summarized as mean ± standard deviation (SD), whereas non-normally distributed variables were reported as median and interquartile range (IQR). Categorical data were presented as absolute frequencies and percentages. Group comparisons for continuous variables were conducted using Mann–Whitney U test for non-parametric data and the Independent Samples t-test for normally distributed data. To explore associations between categorical data, the Chi-square test was employed; Fisher's exact test was substituted when expected cell frequencies were too low to satisfy test assumptions. Significance testing was based on a two-tailed p-value threshold of $\leq 0.05$.

## RESULTS

No substantial differences were identified in terms of demographic or clinical parameters, including age (P = 0.085), gender distribution (P = 1.000), and smoking status (P = 0.109).

None of the participants across both groups exhibited underlying conditions such as hypertension, diabetes mellitus, or dyslipidemia (**Table 1).**

**Table (1):** Baseline demographic and clinical characteristics of the study participants

| | | Cases (n = 75) | Controls (n =75) | P-value |
|---|---|---|---|---|
| **Age (years)** | Mean ±SD | 33.07 ± 7.52 | 35.19 ± 7.47 | 0.085 |
| **Gender** | | | | |
| Female | n (%) | 26 (34.7%) | 26 (34.7%) | 1 |
| Male | n (%) | 49 (65.3%) | 49 (65.3%) | |
| **Smoking** | n (%) | 18 (24.0%) | 27 (36.0%) | 0.109 |

**n:** number, **SD:** standard deviation.

All echocardiographic parameters showed no substantial variations between the vaccinated and control groups, including LVEF (P = 0.735), GLS (P = 0.365), and average S' velocity on the mitral valve (P = 0.196) (Table 2).

**Table (2):** Echocardiographic parameters between vaccinated and control groups

| | | Cases (n = 75) | Controls (n =75) | P-value |
|---|---|---|---|---|
| **EF (biplane) (%)** | Mean ±SD | 63.20 ±4.09 | 62.99 ±3.59 | 0.735 |
| **GLS (%)** | Median (IQR) | -19 (-20 – -17) | -19 (-20 – -18) | 0.365 |
| **Average S' on MV** | Mean ±SD | 9.56 ±2.07 | 9.99 ±1.95 | 0.196 |

**SD:** standard deviation, **GLS:** global longitudinal strain, **EF:** ejection fraction, **S′:** mitral annular systolic velocity, **MV:** mitral valve, **IQR:** interquartile range, **n:** number.

## DISCUSSION

This investigation was designed to explore any potential deterioration in LV systolic function following AstraZeneca COVID-19 vaccination, as measured by echocardiography. Results confirmed the preservation of systolic function, with no echocardiographic evidence of dysfunction post-vaccination.

Defined as inflammation of the myocardium, myocarditis is typically associated with viral etiologies, autoimmune disorders, or immune-modulating therapies. Reports highlighted numerous instances of myocardial injury among hospitalized COVID-19 patients, where elevated cardiac troponin levels have served as indicators of cardiac involvement and have been linked to worse prognoses [6]. Irrespective of its underlying cause, myocarditis accompanied by significant ventricular dysfunction increases the risk of cardiogenic shock and mortality, emphasizing the need to investigate potential temporal associations with COVID-19 vaccination [7]. Our findings are in harmonious agreement with those of **Aye *et al.*** [8] , who similarly found no evidence of myocarditis in AstraZeneca vaccine recipients. Our observations correspond to those reported by **Husby *et al.*** [9], who identified a non-significant rise in myocarditis or myopericarditis risk following COVID-19 vaccination. However, their study exclusively evaluated the Pfizer–BioNTech (BNT162b2) vaccine, not the AstraZeneca formulation explored in our research.

In the investigation by **Patone *et al.*** [10], an estimated two additional cases of myocarditis per one million vaccinations were observed subsequent to initial dose of the AstraZeneca vaccine, while no further incidents were recorded after the second dose. This figure contrasts with an estimated 40 additional myocarditis cases per one million individuals within 28 days of testing positive for COVID-19.

Numerous published reports have documented cases of myocarditis following COVID-19 vaccination, most frequently occurring after administration of the second dose of mRNA-based vaccines, including mRNA-1273 (Moderna) and BNT162b2 (Pfizer–BioNTech), with chest pain being the consistent presenting symptom. Echocardiographic abnormalities were observed in only 40% of cases, with a minority of patients exhibiting a LVEF below 50% at the time of presentation [11-13].

Evidence from prior reports in both Israel and the United States has pointed to an elevated incidence of myocarditis after receiving mRNA COVID-19 vaccines [14-16]. A possible explanation for the divergence between our findings in Egypt and those reported in Israel and the United States may lie in the predominant use of the AstraZeneca vaccine in Egypt, whereas the studies conducted in Israel and the U.S. primarily involved mRNA-based vaccines, such as mRNA-1273 (Moderna) and BNT162b2 (Pfizer–BioNTech).

The majority of myocarditis cases attributed to mRNA COVID-19 vaccines necessitate hospitalization. However, most patients exhibit a favorable response to conventional therapies, with the condition typically being mild and self-limiting [17]. The underlying pathophysiological mechanism is not yet fully understood. However, it is hypothesized that myocarditis may arise due to an autoimmune reaction, either through structural similarities between vaccine antigens and

myocardial proteins or via a generalized inflammatory response induced by vaccination [18].

A key limitation of the present study is its focus solely on the AstraZeneca COVID-19 vaccine, without including or comparing other vaccine types. Furthermore, the absence of cardiac biomarker evaluation—such as serum troponin levels—restricted the ability to detect subclinical myocardial injury.

## CONCLUSION

The present study provides reassuring evidence that AstraZeneca COVID-19 vaccination did not compromise LV systolic function. This was confirmed through detailed echocardiographic analysis employing 2D, speckle tracking, and tissue Doppler modalities.

**Financial support and sponsorship:** Nil.
**Conflict of Interest:** Nil.

## REFERENCES

1. **Chao M, Menon C, Elgendi M (2022):** Effect of COVID-19 vaccination on the menstrual cycle. Front Med (Lausanne), 9: 1065421.
2. **Singh V, Pir M, Buch T *et al.* (2021):** Myocarditis linked to Pfizer-Biontech COVID-19 vaccine. Chest, 160: A444.
3. **Fung G, Luo H, Qiu Y *et al.* (2016):** Myocarditis. Circulation Research, 118: 496-514.
4. **Keinath K, Church T, Kurth B *et al.* (2018)**: Myocarditis secondary to smallpox vaccination. BMJ Case Rep., 2018:bcr2017223523.
5. **Lang R, Badano L, Mor-Avi V *et al.* (2015):** Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr., 28: 1-39.e14.
6. **Chapman A, Bularga A, Mills N (2020):** High-Sensitivity Cardiac Troponin Can Be an Ally in the Fight Against COVID-19. Circulation, 141: 1733-5.
7. **Kociol R, Cooper L, Fang J *et al.* (2020):** Recognition and Initial Management of Fulminant Myocarditis: A Scientific Statement From the American Heart Association. Circulation, 141: e69-e92.
8. **Aye Y, Mai A, Zhang A *et al.* (2023):** Acute myocardial infarction and myocarditis following COVID-19 vaccination. QJM., 116: 279-83.
9. **Husby A, Hansen J, Fosbøl E *et al.* (2021):** SARS-CoV-2 vaccination and myocarditis or myopericarditis: population based cohort study. BMJ., 375: e068665.
10. **Patone M, Mei W, Handunnetthi L *et al.* (2022):** Risks of myocarditis, pericarditis, and cardiac arrhythmias associated with COVID-19 vaccination or SARS-CoV-2 infection. Nature Medicine, 28: 410-22.
11. **Rosner C, Genovese L, Tehrani B *et al.* (2021):** Myocarditis Temporally Associated With COVID-19 Vaccination. Circulation, 144: 502-5.
12. **Abu Mouch S, Roguin A, Hellou E *et al.* (2021):** Myocarditis following COVID-19 mRNA vaccination. Vaccine, 39: 3790-3.
13. **Larson K, Ammirati E, Adler E *et al.* (2021):** Myocarditis After BNT162b2 and mRNA-1273 Vaccination. Circulation, 144: 506-8.
14. **Barda N, Dagan N, Ben-Shlomo Y *et al.* (2021):** Safety of the BNT162b2 mRNA Covid-19 Vaccine in a Nationwide Setting. N Engl J Med., 385: 1078-90.
15. **Klein N, Lewis N, Goddard K *et al.* (2021):** Surveillance for Adverse Events After COVID-19 mRNA Vaccination. JAMA., 326: 1390-9.
16. **Witberg G, Barda N, Hoss S *et al.* (2021):** Myocarditis after Covid-19 Vaccination in a Large Health Care Organization. N Engl J Med., 385: 2132-9.
17. **Montgomery J, Ryan M, Engler R *et al.* (2021):** Myocarditis Following Immunization With mRNA COVID-19 Vaccines in Members of the US Military. JAMA Cardiol., 6: 1202-6.
18. **Segal Y, Shoenfeld Y (2018):** Vaccine-induced autoimmunity: the role of molecular mimicry and immune crossreaction. Cell Mol Immunol., 15: 586-94.