MARMARA
MEDICAL JOURNAL

Original Article

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The effect of influenza vaccine on arterial stiffness in young healthy adults

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Submitted: 08.01.2025 **Accepted:** 11.07.2025

ABSTRACT

Objective: The arterial wall possesses a complex structure. Over time, arteries lose elasticity and become stiffer due to factors such as aging, chronic diseases, and increased inflammation. Pulse wave velocity (PWV) and augmentation index (AI) are widely recognized as direct markers of arterial stiffness. Previous studies have suggested a link between seasonal influenza infection and increased cardiovascular mortality. This study aimed to investigate the effects of influenza vaccination on arterial stiffness in healthy young adults.

Patients and Methods: The study enrolled participants who received the influenza vaccine according to the latest guidelines. Prior to vaccination, participants completed the Hospital Anxiety and Depression Scale (HADS) to assess baseline mood and anxiety levels. Arterial stiffness was measured using an oscillometric device. After a 15-day follow-up, HADS assessments and arterial stiffness measurements were repeated.

Results: A total of 48 participants were initially enrolled, and 33 who completed the follow-up were included in the final analysis. Statistically significant reductions were observed in systolic blood pressure ($p<0.001$), mean arterial pressure ($p=0.025$), pulse pressure ($p=0.001$), and PWV ($p<0.001$). No significant changes were found in HADS scores. No major adverse cardiovascular events occurred during the study.

Conclusion: Our findings suggest that influenza vaccination may lead to early hemodynamic changes in healthy young adults, independent of psychological stress. Although influenza vaccines have been shown to reduce cardiovascular risk, clinicians should be aware of potential early hemodynamic effects following vaccination.

Keywords: Arterial stiffness, PWV, AI, Influenza vaccine

1. INTRODUCTION

The arterial wall has a complex structure that allows central arteries to distend and store elastic energy during systole, which is released during diastole to promote blood flow (the Windkessel function) [1, 2]. Over time, arteries lose elasticity and stiffen. Since less blood can be stored due to reduced elasticity, more blood needs to be transported in the systole. This leads to increased systolic pressure in the arteries and the left ventricle, and ultimately to organ damage in the heart, kidneys, or brain [3].

Pulse wave velocity (PWV) is widely recognized as a direct marker of arterial stiffness. Augmentation and the augmentation index (AI) are being used ever more often in studies as parameters of wave reflection. PWV is correlated with very diverse familiar cardiovascular risk factors, e.g. age, systolic blood pressure (SBP), pulse pressure (PP), left ventricular hypertrophy and coronary heart disease. It has been proved that PWV is a strong vascular risk factor for the prediction of mortality in the elderly, and in patients with end-stage renal disease, diabetes mellitus, or hypertension, and in the general population [4]. Ideally, arterial stiffness should be measured invasively. But this is not always feasible; because of that, investigators developed non-invasive methods to measure arterial stiffness. One of the most widely used non-invasive methods is oscillometric method. This method uses a single cuff on the brachial artery. The device detects pressure changes in the artery, and these recordings are passed on to the computer and analyzed by the software to determine an accurate estimate of PWV and AI [4, 5].

How to cite this article: Ozturk U, Sunbul M, Erturk B, et al. The effect of influenza vaccine on arterial stiffness in young healthy adults. Marmara Med J 2025;38 (3): 204-208. doi: 10.5472/marumj.1798635

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ISSN: 1309-9469

http://doi.org/10.5472/marumj.1798635
Marmara Med J 2025;38(3): 204-208

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Although the impact of chronic inflammation on vascular function is well established, the effects of acute inflammation—such as that triggered by vaccination—remain less clear, particularly in healthy young adults. Acute systemic inflammation has been shown to transiently alter vascular tone and endothelial function, potentially leading to changes in arterial stiffness. However, the magnitude and direction of these effects appear to vary depending on age, baseline cardiovascular status, and the intensity of the inflammatory stimulus [6]. Most existing studies have focused on elderly or high-risk individuals, leaving a knowledge gap regarding how acute inflammation influences vascular function in younger, low-risk populations. This study aims to address this gap by evaluating the short-term effects of influenza vaccination, a standardized model of mild acute inflammation, on arterial stiffness parameters in healthy young adults.

There are several studies associating seasonal influenza infection and an increase in cardiovascular mortality [7]. It is hypothesized that influenza infection acts as an acute inflammatory trigger of cardiovascular events. Due to hypoxia and/or systemic inflammatory response syndrome, myocardial blood supply and demand balance is impaired. Furthermore, influenza infection can lead to plaque rupture and acute coronary syndrome [8]. Receiving influenza vaccine is associated with reduced major adverse cardiovascular events in patients with cardiovascular disease [9]. Although influenza vaccine results in decreased major adverse cardiovascular events, the vaccine itself also causes inflammation. This acute inflammation caused by the vaccine can also have adverse effects on cardiovascular system [10]. Studies on healthy adults have different results about the effects of systemic inflammation on arterial stiffness. Some studies suggest systemic inflammation results in increased arterial stiffness, while other studies suggest there is not any significant difference in arterial stiffness in healthy adults with systemic inflammation [6, 11, 12]. Aim of this study is to investigate the effects of influenza vaccine on arterial stiffness in healthy young adults.

2. PATIENTS and METHODS

Subject selection and study design

This was a prospective observational study. Ethical approval was obtained from the Marmara University, School of Medicine, Non-Interventional Ethics Committee (approval number: 03.12.2021.1371, date: 02.12.2021).

Subjects aged 18–40 years without a history of chronic systemic disease (e.g., diabetes mellitus, hypertension, chronic kidney disease, autoimmune disease, cardiovascular disease) were included. Those with a diagnosis of hypertension (defined as blood pressure ≥140/90 mmHg) or found to be hypertensive during the study were excluded. Individuals with a history of influenza infection within four weeks prior to vaccination or who developed influenza symptoms during the follow-up period were excluded from the study. Subjects who withdrew from the study at any stage of the study and subjects who did not participate in visits or interviews were excluded from the study.

All vaccinations were administered between October 1st and November 30th, 2023, during which a single trivalent/quadrivalent influenza vaccine formulation was used.

The demographic data of the subjects who met the inclusion criteria and consented to participate in the study were documented. Subjects who were planned to receive influenza vaccine in the Infectious Diseases Clinic were admitted to the Cardiology Outpatient Clinic and asked to fill out the Hospital Anxiety and Depression Scale (HADS) forms before receiving the vaccine to determine the baseline mood and anxiety levels. Since, psychological stress can affect vascular responses, we used HADS to assess emotional confounding factors. The HADS is a validated self-assessment questionnaire that does not require administration or interpretation by a medical doctor or psychiatrist. It is designed to be completed by patients independently, making it suitable for use in clinical settings without the need for specialist involvement [13].

After the HADS score assessment, arterial stiffness measurements were performed by the same investigator for all of the study subjects. After the measurements, subjects received the vaccine and then discharged from the hospital. After 15 days, subjects were admitted back for follow up. The study visits were completed at the same time of day to avoid diurnal variation. Subjects were asked to fill out the HADS form again and then arterial stiffness was measured using the same protocol as before.

Assessment of arterial stiffness parameters

Measurement of arterial stiffness can be performed by assessing PWV obtained from either a central or peripheral artery [14]. To measure regional PWV, mechanotransducers, high-fidelity applanation tonometers or Doppler probes can be used. By these devices, transit time of a pulse wave among two arterial sites such as carotid-femoral or carotid-brachial is obtained. For local PWV assessment, Doppler probes can be used on radial, brachial, femoral or carotid arteries. Then, the distance taken by pulse is divided by transit time between the two arteries. All of the aforementioned techniques require experienced operators and are time consuming. Also, their reproducibility is limited. Measurement of PWV using oscillometric method is more reliable in clinical practice compared with tonometric and piezo-electronic methods. [15]. A noninvasive device using oscillometric method to assess PWV was used in our study.

Subjects were asked to refrain from eating and drinking alcohol, coffee, or tea for at least 12 hours prior to the study. The test of arterial stiffness was performed in the supine position in a quiet, temperature-controlled room (22–24 °C) in the early morning hours. Measurements were carried out by using a Mobil-O-Graph arteriograph system (Mobil-O-Graph NG, Stolberg, Germany). This system detects signals from the brachial artery even though cuff pressure is 35 mmHg higher than systolic pressure in the brachial artery. This technique is based on the fact that the contraction of the myocardium initiates a pulse

http://doi.org/10.5472/marumj.1798635
Marmara Med J 2025;38(3): 204-208

wave (early systolic peak) running down in the aorta. This first wave is reflected from the aortic wall at the distal branching point and causes a reflected second wave (late systolic peak). The morphology of this second reflected wave depends on the stiffness of the large artery. By using amplitude and time difference of first and second wave, AI (AI adjusted for heart rate 75 bpm) and PWV are calculated according to guidelines [16].

Statistical Analysis

Statistical analyses were performed using IBM® SPSS® Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Normality of continuous variables was determined by the Shapiro–Wilk test. The demographic and clinical characteristics of the subjects were expressed as numbers and percentages for categorical variables and as mean and standard deviation (SD) for normally distributed continuous variables. Values not displaying normal distribution were expressed as median (interquartile range). Statistical comparison of quantitative data was performed with paired sample t-test for continuous variables displaying normal distribution. Wilcoxon signed-rank test was used for continuous variables not displaying normal distribution. A p-value of less than 0.05 was considered statistically significant.

3. RESULTS

A total of 48 subjects were included in the study. 33 subjects who completed the follow up period were included in the statistical analysis. 15 subjects who did not complete the follow up period were excluded from the study. The mean age of the subjects included in the study was 28.9 ± 9.9 years. Of the total number of subjects, 20 (60.6%) were female and 13 (39.4%) were male. The mean height of the study group was 169.8 ± 8.3 centimeters. The mean weight of the study group was 67.8 ± 12.8 kilograms. 10 subjects (30.3%) in the study group were active smokers. The baseline characteristics of the study group is shown in Table I.

Table I. *Baseline characteristics of the study group (n=33)*

Age (years)	28.9 ± 9.9*
Sex (n-female)	20 (60.6%)*
Height (cm)	169.8 ± 8.3*
Weight (kg)	67.8 ± 12.8*
Smoking (n)	10 (30.3%)*

**Values are presented as mean ± standard deviation or number (percentage) as appropriate.*

Mean SBP at the baseline was 123.2± 12.3 mmHg. At the follow-up, mean SBP was 115.2 ± 8.9. Reduction in the SBP was statistically significant (p <0.001). Mean diastolic blood pressure (DBP) at the baseline was 75.7 ± 8.1 mmHg. At the follow-up, mean DBP was 75.3 ± 6.7. There was not a statistically significant change in the DBP at the follow-up (p=0.766). Mean value of mean arterial pressure (MAP) at the baseline was 96.9 ± 9.5. Mean MAP at the follow-up was 93.5 ± 6.6. There was a statistically significant reduction in MAP (p=0.025). Mean heart rate (HR) was 81.4 ± 14.6 bpm. Mean HR at the follow-up was 82.8 ± 14.1 bpm. There was not a statistically significant change in the HR at the follow-up (p=0.383). Mean PP at baseline was 47.5 ± 10.0 mmHg. Mean PP at the follow-up was 39.9 ± 8.1 mmHg. Reduction in the PP was statistically significant (p <0.001). Mean PWV was 5.5 ± 0.9 m/s at baseline. Mean PWV was 5.2 ± 0.9 m/s at the follow-up. There was a statistically significant reduction in PWV at the follow-up (p=0.001). Mean value of AI was 21.2 ± 10.0 % at the baseline. Mean AI at the follow-up was 21.1 ± 10.9 %. There was not a statistically significant difference for AI at the follow-up (p=0.990). The comparison of hemodynamic parameters and arterial stiffness parameters at the baseline and at the follow-up is summarized in Table II respectively.

Table II. *The comparison of hemodynamic and arterial stiffness parameters*

	Baseline	Follow-up	p*
Systolic Blood Pressure (mmHg)	123.2 ± 12.3	115.2 ± 8.9	**< 0.001**
Diastolic Blood Pressure (mmHg)	75.7 ± 8.1	75.3 ± 6.7	0.766
Mean Arterial Pressure (mmHg)	96.9 ± 9.5	93.5 ± 6.6	0.025
Heart Rate (beats/min)	81.4 ± 14.6	82.8 ± 14.1	0.383
Pulse Pressure (mmHg)	47.5 ± 10.0	39.9 ± 8.1	**< 0.001**
Pulse Wave Velocity (m/s)	5.5 ± 0.9	5.2 ± 0.9	**0.001**
Augmentation Index (%)	21.2 ± 10.0	21.1 ± 10.9	0.990

**Data are presented as mean ± standard deviation. Comparisons between baseline and follow-up were performed using paired sample t-tests.*

The HADS scores were recorded at the baseline and at the follow-up. Anxiety score at the baseline was 7.0 ± 4.3 and 7.3 ± 3.6 at the follow-up (p=0.379). Depression score at the baseline was 5.5 ± 3.4 and 5.2 ± 3.5 at the follow-up (p=0.429). There was not a statistically significant change in the study populations' HADS score at the follow-up. The comparison of the HADS scores at the baseline and at the follow-up is summarized in Table III respectively.

Table III. *Comparison of the hospital anxiety and depression scale (hads) scores*

	Baseline	Follow-up	p*
Anxiety Score	7.0 ± 4.3	7.3 ± 3.6	0.379
Depression Score	5.5 ± 3.4	5.2 ± 3.5	0.429

**Data are presented as mean ± standard deviation. Comparisons between baseline and follow-up were performed using paired sample t-tests.*

4. DISCUSSION

In this prospective study involving healthy young adults, we observed significant reductions in SBP, MAP, PP, and PWV 15 days after influenza vaccination. No significant changes were

http://doi.org/10.5472/marumj.1798635
Marmara Med J 2025;38(3): 204-208

detected in DBP, AI, or HADS scores. These findings suggest that influenza vaccination may induce favorable short-term hemodynamic effects independent of psychological stress. While prior studies have primarily focused on cardiovascular outcomes in older or high-risk populations, our results contribute to a growing body of evidence indicating that acute inflammation from vaccination may influence vascular function even in younger, low-risk individuals [17].

The effectiveness and benefit of the influenza vaccine in elderly patients and in patients with cardiovascular disease are well documented [18, 19]. In young adults, influenza vaccine can reduce flu symptoms, hospital visits and missed work or school. Cardiovascular benefits of vaccination are not as clear in young adults contrary to the elderly population. Our study showed reduction in SBP, MAP, PP and PWV in young adults after influenza vaccination without a significant change in their HADS scores. These findings suggest that influenza vaccination may confer cardiovascular benefits even in healthy young adults.

One plausible mechanism for the observed decrease in PWV following influenza vaccination involves transient endothelial activation mediated by inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α). These mediators can promote the release of nitric oxide (NO) and prostaglandins, leading to short-term vasodilation and reduced vascular tone. In young, healthy individuals with preserved arterial elasticity, this vasodilatory response may translate into measurable reductions in central arterial stiffness.

While the observed reductions in SBP, PP, and PWV were modest, they may still hold clinical relevance, particularly in the context of early vascular aging and cardiovascular risk stratification. Even small improvements in arterial stiffness, as reflected by decreased PWV, have been associated with reduced long-term cardiovascular risk in population-based studies [20]. Similarly, reductions in SBP and PP can lessen the mechanical load on the heart and arterial system, which may have preventive implications if sustained. Although our findings are limited to a short follow-up period, they raise important questions about whether influenza vaccination could transiently modulate cardiovascular function, even in low-risk populations.

Although the reductions in arterial stiffness and systolic parameters were statistically significant, DBP and AI remained unchanged. This contrast is noteworthy. DBP is predominantly influenced by peripheral vascular resistance and diastolic runoff, whereas SBP is more sensitive to central arterial stiffness and stroke volume. It is plausible that the mild inflammatory response induced by vaccination resulted in vasodilation primarily within central arteries, reflected by reductions in SBP and PWV, while peripheral vascular tone remained largely unaffected, maintaining stable DBP levels. It is also possible that the degree of peripheral vasodilation was not sufficient to significantly impact DBP measurements, despite transient changes occurring in central arteries. Similarly, the lack of change in AI may be attributed to its dependence on wave reflection timing, which may not be significantly altered by short-term systemic inflammation.

The absence of significant changes in HADS scores before and after vaccination indicates that the reductions observed in arterial stiffness and blood pressure were not influenced by psychological stress or mood-related factors. This supports the interpretation that the hemodynamic effects, including decreases in PWV, SBP, MAP, and PP, were likely driven by physiological mechanisms—such as mild, vaccine-induced inflammation—rather than by emotional or cognitive responses. Given that all participants were healthy young adults, received a routine vaccination, and were not subjected to additional clinical stressors, it is reasonable that HADS scores remained stable. These findings strengthen the validity of the arterial stiffness measurements as true physiological outcomes, independent of subjective psychological influence.

Arterial compliance decreases with age, thereby limiting the capacity for vasodilation. In contrast, young adults possess more elastic arterial systems that may respond more readily to transient inflammatory stimuli. Prior studies examining the vascular effects of acute inflammation in young individuals have reported mixed results [6, 11, 12]; while some observed increased arterial stiffness, others found no significant change. Our findings are consistent with the hypothesis that a moderate, controlled inflammatory response – such as that triggered by vaccination – may lead to short-term peripheral vasodilation and reduced arterial stiffness in healthy young adults. Importantly, no participants experienced adverse cardiovascular symptoms, supporting the short-term safety of influenza vaccination in this population.

Limitations

Our study is not without limitations. First, the number of participants was relatively small, which may limit the generalizability of the findings. Second, the study did not include a control group, sham intervention group, or a comparison group receiving a different vaccine. The absence of these comparative groups limits the ability to isolate the specific effects of the influenza vaccine from other potential contributing factors, such as time or measurement variability. Additionally, the follow-up period was limited to 15 days, preventing assessment of long-term hemodynamic effects. Another important limitation is the lack of biochemical assessment of acute inflammation markers, such as C-reactive protein (CRP) or IL-6, which could have helped to support the hypothesized inflammatory mechanism behind the observed vascular changes. Future studies with larger sample sizes, extended follow-up durations, and comparative groups are needed to validate and expand upon our findings.

Conclusion

In this study, we observed short-term reductions in blood pressure and arterial stiffness parameters following influenza vaccination in healthy young adults. These changes were not associated with alterations in psychological stress levels, as reflected by stable HADS scores. While these preliminary findings suggest that influenza vaccination may have transient hemodynamic effects even in low-risk populations, the limited sample size and short follow-up period preclude drawing

http://doi.org/10.5472/marumj.1798635
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definitive conclusions. Further large-scale, controlled studies with extended observation periods are needed to clarify the potential cardiovascular effects of influenza vaccination in healthy young individuals.

Compliance with Ethical Standards

Ethical approval: Ethical approval for this research was obtained from the Marmara University, School of Medicine, Non-Interventional Ethics Committee (approval number : 03.12.2021.1371, date: 02.12.2021). This study was performed in line with the principles of the Declaration of Helsinki.

Conflict of interest: The authors declare that there is no conflict of interest.

Financial support: This study received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Author contributions: MS and ETT: Study design, ED, DP, BK, ZEA and BE: Data acquisition, BO, MKT, AAC, EG, NS and UO: Data analysis and interpretation, UO and MS: Drafting the manuscript. All authors approved the final version of the manuscript.

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