Impact of Diabetes on Atrial Stiffness and Risk of Atrial Fibrillation: A Study of 200 Patients

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ABSTRACT

Background

Diabetes mellitus (DM) is a known risk factor for atrial fibrillation (AF), primarily due to its effects on atrial structural and electrical remodeling. Chronic hyperglycemia leads to atrial fibrosis, increased stiffness, and impaired conduction, all of which promote AF development. This study aimed to evaluate the impact of type 2 diabetes mellitus (T2DM) on atrial stiffness and AF risk using echocardiographic and electrocardiographic markers.

Methods

A retrospective study was conducted on 200 patients divided into two groups: 100 with T2DM and 100 non-diabetic controls. All patients underwent echocardiographic evaluation, including speckle-tracking echocardiography to assess reservoir strain, conduit strain, and Left Atrial Stiffness Index (LASI). Electrocardiographic analysis included P-wave dispersion (PWD) as a marker of conduction heterogeneity. Patients were followed for 12 months to assess AF incidence.

Results

Diabetic patients had significantly lower reservoir strain (-18% vs. -26%, $p = 0.01$) and conduit strain (-12% vs. -19%, $p = 0.02$), indicating increased atrial stiffness. LASI was higher in diabetics (0.82 vs. 0.65, $p = 0.02$), reflecting increased fibrosis. PWD was prolonged in diabetics (42 ms vs. 32 ms, $p = 0.03$), suggesting greater conduction heterogeneity. After 12 months, AF incidence was significantly higher in diabetics (22% vs. 9%, $p = 0.01$; OR = 2.87, 95% CI: 1.43–5.78).

Conclusion

T2DM is associated with increased atrial stiffness and higher AF risk due to structural and electrical remodeling. Early identification of atrial remodeling markers may help stratify AF risk in diabetics and guide preventive interventions.

Keywords: Diabetes mellitus, atrial stiffness, atrial fibrillation, P-wave dispersion, echocardiography

INTRODUCTION

Diabetes mellitus (DM) is a significant risk factor for cardiovascular diseases, including atrial fibrillation (AF) ([1]). Epidemiological studies suggest that diabetic patients have a 1.5- to 2.5-fold increased risk of developing AF compared to non-diabetic individuals ([2]). This increased risk is attributed to diabetes-induced structural and electrical remodeling of the atria ([3]).

Structurally, chronic hyperglycemia promotes atrial fibrosis through several mechanisms, including the accumulation of advanced glycation end-products (AGEs), oxidative stress, and inflammation ([4,5]). These processes increase atrial stiffness, impair atrial compliance, and elevate left atrial pressure ([6]).

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Electrically, diabetes alters atrial conduction through ion channel dysfunction and gap junction remodeling, leading to conduction heterogeneity and increased vulnerability to re-entry circuits ([7,8]). Prolonged P-wave dispersion (PWD) on electrocardiography is a marker of such conduction abnormalities and has been identified as an independent predictor of AF ([9]).

This study aims to assess the impact of T2DM on atrial stiffness and AF risk by evaluating echocardiographic and electrocardiographic markers in a cohort of 200 patients.

MATERIALS AND METHODS

Study Design and Population

A retrospective observational study was conducted on 200 patients who underwent routine cardiovascular evaluation. Patients were divided into two groups:

- Diabetes group (n = 100): Patients diagnosed with T2DM for ≥5 years.
- Control group (n = 100): Non-diabetic individuals with no history of cardiovascular disease.

Exclusion criteria included prior AF diagnosis, structural heart disease, significant valvular disease, and end-stage renal disease.

Echocardiographic Assessment

All patients underwent transthoracic echocardiography, including speckle-tracking echocardiography to assess:

- Reservoir strain: Measures left atrial expansion during ventricular systole.
- Conduit strain: Evaluates atrial emptying function.
- Left Atrial Stiffness Index (LASI): Calculated as the E/e' ratio divided by reservoir strain.

Electrocardiographic Assessment

P-wave dispersion (PWD) was measured as the difference between the longest and shortest P-wave duration in 12-lead ECGs.

Follow-Up and Outcome Measures

Patients were followed for 12 months to assess AF incidence. AF was defined as ECG-documented atrial fibrillation lasting >30 seconds.

Statistical Analysis

Continuous variables were analyzed using Student's t-test, while categorical variables were compared using the chi-square test. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated for AF incidence. A p-value <0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

Diabetic patients had significantly higher BMI, blood pressure, and HbA1c levels compared to controls.

Variable	Diabetes Group (n=100)	Control Group (n=100)	p-value
Age (years)	63 ± 8	61 ± 7	0.15

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BMI (kg/m²)	30.2 ± 3.5	26.8 ± 3.1	0.01
HbA1c (%)	7.8 ± 1.2	5.4 ± 0.8	<0.001
SBP (mmHg)	136 ± 12	124 ± 10	0.02

Echocardiographic and Electrocardiographic Findings

Parameter	**Diabetes Group**	**Control Group**	**p-value**
Reservoir strain (%)	-18	-26	0.01
Conduit strain (%)	-12	-19	0.02
LASI	0.82	0.65	0.02
PWD (ms)	42	32	0.03

Incidence of Atrial Fibrillation

AF incidence was significantly higher in the diabetes group (22% vs. 9%, OR = 2.87, 95% CI: 1.43–5.78, p = 0.01).

DISCUSSION

Our study confirms that type 2 diabetes mellitus (T2DM) significantly increases atrial stiffness and the risk of atrial fibrillation (AF) through a combination of structural and electrical remodeling mechanisms ([10,11]). The markedly higher Left Atrial Stiffness Index (LASI) observed in diabetic patients compared to non-diabetic controls suggests a more advanced state of atrial fibrosis in this population. These findings align with prior research indicating that chronic hyperglycemia promotes extracellular matrix deposition, collagen cross-linking, and reduced atrial compliance, all of which contribute to increased atrial stiffness ([12]). The association between LASI and AF incidence in diabetics highlights the potential utility of LASI as a predictive marker for AF risk stratification in clinical practice.

In addition to structural remodeling, our study demonstrates that electrical remodeling is also a key contributor to AF susceptibility in diabetics. The significant prolongation of P-wave dispersion (PWD) in diabetic patients indicates increased atrial conduction heterogeneity, a well-established electrophysiological substrate for AF development ([13]). Similar results have been reported in previous studies, which have shown that diabetes is associated with slowed atrial conduction, impaired gap junction function (notably through connexin-43 dysregulation), and the facilitation of micro-reentrant circuits ([14,15]). These electrical abnormalities predispose diabetic patients to atrial arrhythmias by creating areas of conduction delay and unidirectional block, further increasing the likelihood of AF initiation and maintenance.

Furthermore, the 12-month follow-up data reveal a significantly higher incidence of AF in diabetic patients compared to controls (22% vs. 9%). This supports the hypothesis that progressive atrial remodeling due to diabetes creates an arrhythmogenic substrate that facilitates AF onset ([16]). The increased AF incidence in our diabetic cohort is consistent with previous large-scale studies that have reported a 1.5- to 2.5-fold increased risk of AF in diabetic individuals compared to the general population. These findings underscore the need for early identification of high-risk diabetic patients who may benefit from targeted AF prevention strategies.

From a clinical perspective, our results emphasize the importance of integrating echocardiographic and electrocardiographic parameters, such as LASI and PWD, into routine cardiovascular assessments for diabetic

patients. The ability to detect early signs of atrial remodeling could help refine risk stratification and allow for the timely implementation of preventive measures. Potential interventions include optimizing glycemic control, as studies have suggested that tighter glucose regulation can mitigate atrial fibrosis and reduce AF risk ([17]). Additionally, emerging antifibrotic therapies, such as sodium-glucose cotransporter-2 (SGLT2)

inhibitors, have shown promise in reducing cardiac fibrosis and may play a role in AF prevention among diabetic patients ([17]).

Overall, our study reinforces the growing body of evidence linking diabetes to AF pathogenesis and highlights the need for proactive monitoring and management strategies to mitigate arrhythmia risk in this vulnerable population. Future research should explore whether targeted therapies aimed at reducing atrial fibrosis and conduction abnormalities can effectively lower AF incidence in diabetic individuals.

CONCLUSION

Diabetes mellitus significantly increases atrial stiffness and AF risk due to structural and electrical remodeling. Early identification of atrial dysfunction could aid in risk stratification and targeted interventions.

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Conflict of Interest

The authors declare no conflicts of interest.

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Informed Consent Statement

Informed consent was obtained from all participants, and the study was approved by the institutional ethics committee.

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