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Commentary

From Silent Rhythm Disturbance to System-Level Cost: Cardiac Arrhythmias as a Modifiable Burden in Type 2 Diabetes

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An under-recognized clinical intersection

Type 2 diabetes mellitus (T2D) is most commonly managed through the lens of glycemic control. However, cardiac rhythm disturbances represent a clinically consequential and comparatively under-monitored dimension of diabetic cardiovascular disease. The American Diabetes Association estimated the total annual cost of diagnosed diabetes in the United States at $412.9 billion in its most recent report, including $306.6 billion in direct medical expenditures and $106.3 billion in indirect costs related to lost productivity, premature mortality, and other economic consequences **[1].**

Despite well-established mechanistic links between dysglycemia and arrhythmogenesis, including autonomic dysfunction, electrical remodeling, oxidative stress, inflammation, and structural myocardial change, **[2]** systematic arrhythmia surveillance is not routinely embedded in contemporary diabetes care pathways. This creates a clinically important detection gap: many arrhythmias, particularly atrial fibrillation, may remain asymptomatic or unrecognized until they present through stroke, heart failure decompensation, syncope, or sudden cardiac death **[3]**.

Two complementary real-world analyses recently conducted by our group help quantify the clinical and economic relevance of this gap and suggest that it may be modifiable.

The first, a population-based retrospective cohort study published in the Journal of Health and Medical Economics, characterized predictors, incremental healthcare resource utilization, and costs associated with arrhythmias among patients with T2D, and evaluated utilization patterns among patients undergoing ambulatory electrocardiographic monitoring **[4]**. The second, presented at the 2025 American Diabetes Association Scientific Sessions, examined the incidence and temporal relationship of arrhythmias to T2D diagnosis and major adverse cardiovascular events **[5]**. Considered together, these analyses support a reframing of arrhythmia in T2D: not as an incidental comorbidity, but as a measurable, costly, and potentially actionable component of diabetic cardiovascular risk.

Incidence, timing, and clinical context

The first key observation is scale. In the ADA analysis, conducted using the Symphony Integrated Dataverse, 30,323,803 adults met criteria for T2D. Among the 8,807,487 patients

with at least 12 months of available lookback data, 1,143,374 had a diagnosis of major arrhythmia **[5]**. The population was older and approximately balanced by sex, with a median age of 60 years and a distribution of 46% male and 54% female.

The temporal relationship between T2D and arrhythmia was notably heterogeneous. Arrhythmia preceded the T2D diagnosis in 43% of patients, occurred on the same day in 10%, and followed the diabetes diagnosis in 47%. Among patients in whom arrhythmia developed after T2D, the median time to arrhythmia diagnosis was 496 days. This interval is clinically relevant because it suggests a window during which proactive rhythm surveillance may be feasible, particularly in patients with elevated cardiometabolic risk.

Atrial fibrillation was the predominant arrhythmia phenotype across timing groups, identified in approximately 80% of patients. Other arrhythmias, including atrial flutter, atrioventricular block, supraventricular tachycardia, and ventricular tachycardia, occurred at substantially lower frequencies. The cardiometabolic risk profile was dense: among patients who developed arrhythmia after T2D,

hypertension was common, and 38% had more than one additional metabolic risk factor, including chronic kidney disease, dyslipidemia, liver dysfunction, or obesity.

The association with major adverse cardiovascular events was also substantial. A major adverse cardiovascular event occurred before arrhythmia diagnosis in 45% of patients, with a median interval of 542 days prior to arrhythmia diagnosis. An additional 25% experienced a major adverse cardiovascular event at the time of, or following, arrhythmia diagnosis, with a median interval of 1 day. These findings suggest that arrhythmia in T2D may function both as a marker of accumulated cardiovascular injury and as a signal of imminent or ongoing clinical instability. This pattern supports a strategy of targeted surveillance rather than reliance on symptom-triggered detection alone.

Biological plausibility: why diabetes destabilizes cardiac rhythm

The epidemiologic findings are biologically plausible. T2D has been associated with an approximately 40% increased risk of atrial fibrillation **[6]**. This increased risk likely reflects a convergence of structural, electrical, autonomic, and metabolic mechanisms.

Hyperglycemia, insulin resistance, and associated inflammatory pathways may contribute to atrial fibrosis and remodeling **[2]**. Electrical remodeling, including QTc prolongation and increased dispersion of repolarization, may further increase susceptibility to atrial and ventricular arrhythmias.

Autonomic dysfunction is particularly relevant in diabetes. Patients with diabetic autonomic neuropathy may exhibit impaired circadian variation in heart rate and QTc interval, potentially increasing vulnerability during periods of heightened autonomic instability, including nighttime and early morning hours. Reduced heart rate variability, a validated marker of autonomic dysfunction, declines progressively across the diabetes continuum and has been associated with increased arrhythmia risk.

Hypoglycemia may also contribute to arrhythmogenesis. Episodes of hypoglycemia can provoke a biphasic autonomic response characterized by sympathetic activation followed by parasympathetic rebound. This sequence may be especially hazardous in patients with established cardiovascular disease, autonomic impairment, or underlying conduction abnormalities. Taken together, these mechanisms provide a strong biological rationale for ambulatory rhythm assessment in selected high-risk patients with T2D.

Quantifying the clinical and economic burden

The companion economic analysis translated this epidemiologic burden into healthcare utilization and cost. From an open-claims population of approximately 32 million patients with T2D, 1.19 million developed arrhythmia and 12.3 million did not **[4]**. Patients who developed arrhythmia were older and more often male, with a median age of 71 years and 57% male, compared with a median age of 62 years and 48% male among those without arrhythmia. Median arrhythmia onset occurred 18 months after the T2D index date.

Patients who developed arrhythmia also had substantially greater baseline disease burden. An Elixhauser Comorbidity Index greater than 5 was present in 61% of patients with arrhythmia compared with 12% of those without arrhythmia. A Diabetes Complication Severity Index score of 2 or higher was present in 28% versus 10%, respectively. These differences underscore that arrhythmia in T2D occurs within a clinically complex population, rather than as an isolated rhythm disorder.

To estimate the incremental burden associated with arrhythmia, the analysis used the closed-claims Merative Market Scan database and exact 1:1 matching by age, sex, region, insurance type, comorbidity index, and diabetes severity. This yielded 213,226 matched pairs. Even after matching, healthcare utilization was substantially higher among patients with arrhythmia. Hospitalizations occurred at a rate of 395 versus 139 per 1,000 patients per year, corresponding to a risk ratio of 2.85. Thirty-day readmissions occurred at 95 versus 51 per 1,000 patients per year, with a risk ratio of 1.86. Emergency department visit-days occurred at 985 versus 468 per 1,000 patients per year, with a risk ratio of 2.10. All comparisons were statistically significant at $p < 0.001$.

Cost differences were similarly pronounced. Total annual cost of care was $34,171 per patient with arrhythmia compared with $18,687 per patient without arrhythmia, corresponding to a risk ratio of 1.83. The difference was observed across care settings. Among patients using inpatient services, costs were $24,908 versus $15,611; among those using outpatient services, costs were $7,660 versus $5,152. The temporal cost pattern was particularly informative: expenditures increased sharply around the arrhythmia index date, reaching $17,692 per patient per month in the arrhythmia group compared with $2,087 in the comparator group, before declining toward a persistently elevated post-index baseline. This suggests that the economic burden is concentrated around episodes of clinical decompensation, a period during which earlier detection and intervention may plausibly alter downstream utilization.

Predictive modeling and social-risk context

Identifying patients most likely to develop arrhythmia is essential for implementing a targeted surveillance strategy. In the economic analysis, mutual information methods were used to identify statistically meaningful differentiators, followed by an XG Boost machine-learning model with SHAP-based variable importance to predict arrhythmia onset within a six-month window. The strongest predictors included age greater than 65 years, hypertension, elevated Diabetes Complication Severity Index, and food insecurity.

The clinical trajectory of patients with arrhythmia was also characterized by greater use of diagnostic cardiac testing, more frequent cardiology visits, and higher prescription rates for beta-blockers, antiarrhythmic agents, and anticoagulants. These patterns are consistent with increased cardiovascular complexity and subsequent treatment intensification after arrhythmia recognition.

The unmet social needs findings require cautious interpretation. Differences in food insecurity, financial strain, and transportation barriers reached statistical significance, likely reflecting the very large sample size. However, Cramer's V values indicated that the magnitude of association was limited. Therefore, the most defensible interpretation is not that social adversity independently causes arrhythmia, but rather that unmet social needs are highly prevalent in this population and may complicate access to timely diagnosis, longitudinal monitoring, and treatment adherence **[7]**. Where food insecurity contributed to a predictive signal in the multivariable model, it should be interpreted as a marker of vulnerability that may compound clinical risk, rather than as an isolated causal driver.

Early detection as a potential intervention point

One of the most clinically provocative findings was that utilization appeared lower among patients who underwent ambulatory ECG monitoring. In a secondary matched analysis comparing patients monitored with an ambulatory Zio cardiac monitor with patients who had arrhythmia but were not monitored, groups were matched 1:1 by age, sex, region, insurance type, comorbidity index, and diabetes severity. The monitored group had lower healthcare utilization, including hospitalizations of 64 versus 215 per 1,000 patients per month, corresponding to a risk ratio of 0.30; 30-day readmissions of 31 versus 56 per 1,000 patients per month, corresponding to a risk ratio of 0.56; and emergency department visit-days of 103 versus 202 per 1,000 patients per month, corresponding to a risk ratio of 0.51.4

Among monitored patients, 18% received an arrhythmia diagnosis within four weeks of monitoring. This diagnostic yield suggests that a meaningful proportion of clinically relevant arrhythmias may be detectable through ambulatory monitoring but remain unidentified under usual care. This is particularly important in T2D, where silent or minimally symptomatic atrial fibrillation may be under-recognized and where delayed diagnosis may defer initiation of anticoagulation, rate or rhythm control, or risk-factor modification **[3]**.

These findings should not be interpreted as evidence of causality. The monitoring analysis was observational, and patients selected for ambulatory ECG monitoring may have differed systematically from those who were not monitored. Confounding by indication, differences in clinician behavior, and unmeasured differences in baseline risk cannot be excluded. Nevertheless, the direction, consistency, and magnitude of the observed associations, combined with a plausible clinical mechanism, support prospective evaluation of ambulatory rhythm monitoring as a risk-modification strategy in selected patients with T2D.

Implications for clinical practice and health policy

As health systems increasingly emphasize value-based care, arrhythmia in T2D represents a compelling target for proactive management. The burden is large, clinically meaningful, concentrated around identifiable episodes of decompensation, and associated with risk factors that can be incorporated into pragmatic surveillance algorithms.

Two practical implications follow. First, patients with T2D who have higher-risk clinical profiles, including older age, hypertension, established diabetes complications, chronic kidney disease, autonomic dysfunction, or prior cardiovascular events, may warrant a lower threshold for extended ambulatory ECG monitoring. This approach is especially relevant because symptom-based detection may be insufficient in patients with diabetes, who may have autonomic impairment or atypical symptom perception.

Second, integrating ambulatory ECG monitoring with continuous glucose monitoring may provide a more complete view of the interaction between metabolic instability and cardiac electrophysiology. Such an approach could help identify temporal relationships among glycemic variability, hypoglycemia, autonomic perturbation, and arrhythmic events. At present, these domains are often managed in separate clinical silos; integrating them may improve risk stratification and guide more individualized intervention.

The limitations of the available evidence are important. Both analyses were observational and subject to residual confounding. Administrative claims data capture billed diagnoses and procedures rather than adjudicated clinical events, and they may lack granularity regarding arrhythmia burden, rhythm duration, glycemic patterns, medication adherence, and ECG-confirmed disease. The absence of consistent high-resolution ECG and glucose data limited more detailed mechanistic and temporal analyses.

These limitations do not negate the observed signal, but they define the next research step: prospective, longitudinal studies that jointly assess arrhythmia burden and glucose dynamics and evaluate whether targeted monitoring improves clinical outcomes, reduces avoidable utilization, and lowers total cost of care.

The convergence of findings across large independent datasets and the consistency of the clinical, utilization, and cost signals support further investigation.

Conclusion

Cardiac arrhythmia in T2D is common, costly, frequently silent, and closely linked to the cardiovascular events that diabetes care seeks to prevent. Across more than 30 million patients in one analysis and approximately 32 million in another, arrhythmia was associated with greater comorbidity burden, substantially higher healthcare utilization, higher costs, and frequent major adverse cardiovascular events both before and after arrhythmia diagnosis. Ambulatory ECG monitoring was associated with lower subsequent utilization and identified arrhythmia in a meaningful proportion of monitored patients within four weeks.

The central implication is that comprehensive diabetes care should extend beyond glucose control to include cardiovascular rhythm risk. Treating arrhythmia detection as an integrated component of value-based diabetes care, rather than as a reactive step after an acute event,

may offer a path toward improved outcomes and more efficient resource use. For clinicians, the practical message is to maintain a lower threshold for rhythm evaluation in older patients with T2D, particularly those with hypertension, diabetes complications, autonomic dysfunction, unexplained palpitations, syncope, cryptogenic stroke, or other signs of cardiovascular instability. For payers and policymakers, the concentration of cost around arrhythmia-related decompensation identifies a measurable point in the patient journey where investment in earlier detection may plausibly avert more expensive downstream events.

The next step is to move from real-world association to prospective evidence. Carefully designed studies are needed to determine whether targeted ambulatory rhythm monitoring in high-risk patients with T2D can reduce stroke, heart failure events, hospitalizations, emergency department use, and total cost of care. Until such evidence is available, the current data provide a strong rationale for heightened clinical vigilance and for integrating rhythm assessment more deliberately into the broader management of diabetic cardiovascular risk.

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