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FEDERACION ARGENTINA DE SOCIEDADES DE ENDOCRINOLOGIA

ARTÍCULO ORIGINAL / *Original*

ABDOMINAL AORTIC CALCIFICATION IN TYPE 2 DIABETES: ASSOCIATION WITH CORONARY HEART DISEASE AND PERFORMANCE OF A CLINICAL MODEL

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ARTICLE INFO

Keywords:
type 2 diabetes mellitus, vascular calcification, myocardial ischemia.

Received: September 21, 2025
Accepted: October 18, 2025

ABSTRACT

Type 2 diabetes mellitus (T2DM) is associated with an accelerated development of atherosclerotic vascular disease, which accounts for most of its cardiovascular complications. Abdominal aortic calcification (AAC) has been proposed as a marker of cardiovascular risk. The aim of this study was to describe the clinical and therapeutic profile of patients with T2DM and to evaluate the association between AAC and macrovascular complications. Materials and methods: Cross-sectional observational study of 209 adults with T2DM. Demographic, clinical, and biochemical variables were recorded, and micro- and macrovascular complications were documented. AAC was assessed on 118 interpretable lateral lumbar spine radiographs using the Kauppila score. The association between AAC and ischemic heart disease (IHD) was analyzed with logistic regression (age-adjusted model and parsimonious model). Results: Women comprised 56% of the cohort; median age was 62 years, T2DM duration 8 years, and median HbA1c 7.1%. Macrovascular complications occurred in 16.7%: IHD 9.6%, cerebrovascular disease 2.9%, and peripheral arterial disease 6.2%. AAC was present in 30.5% and was more frequent among those with IHD (66.7% vs. 26.4%; p=0.004). In the age-adjusted model, AAC showed a trend toward association with IHD (OR 3.44; 95% CI 0.883-13.40; p=0.075). In the parsimonious model, AAC remained an independent predictor (OR 5.25; 95% CI 1.42-19.40; p=0.0128). Model performance: AUC 0.81 (95% CI 0.71-0.91), good calibration (Hosmer-Lemeshow p=0.246), and Brier score 0.081. Discussion: In T2DM, AAC was associated with a higher probability of IHD and may serve as an accessible marker of subclinical cardiovascular risk. Prospective studies are warranted to confirm causality and define its prognostic utility.

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CALCIFICACIÓN DE AORTA ABDOMINAL EN DM2: ASOCIACIÓN CON CARDIOPATÍA ISQUÉMICA Y DESEMPEÑO DE UN MODELO CLÍNICO

INFO ARTÍCULO

Palabras clave:
diabetes mellitus tipo 2, calcificación vascular, isquemia miocárdica.

Recibido: 21 de septiembre 2025

Aceptado: 18 de octubre 2025

RESUMEN

La diabetes mellitus tipo 2 (DM2) se asocia con un desarrollo acelerado de enfermedad aterosclerótica, responsable de la mayoría de sus complicaciones cardiovasculares. La calcificación de la aorta abdominal (CAA) ha sido propuesta como marcador de riesgo cardiovascular. El objetivo del estudio fue describir el perfil clínico y terapéutico de pacientes con DM2 y evaluar la asociación entre CAA y complicaciones macrovasculares. Materiales y métodos: estudio observacional transversal en 209 pacientes con DM2. Se registraron variables demográficas, clínicas y bioquímicas, y se documentaron complicaciones microvasculaes y macrovasculares. La CAA se valoró en 118 radiografías interpretables mediante el índice de Kauppila. La asociación entre CAA y cardiopatía isquémica se analizó por regresión logística (modelo ajustado por edad y análisis parsimonioso). Resultados: 56% fueron mujeres; mediana de edad 62 años y de evolución de DM2 8 años; hemoglobina glucosilada (HbA_{1c}) 7,1%. Las complicaciones macrovasculares ocurrieron en 16,7%: cardiopatía isquémica 9,6%, cerebrovascular 2,9% y arterial periférica 6,2%. La CAA estuvo presente en 30,5% y se asoció con mayor frecuencia de IC (66,7% vs. 26,4%; p=0,004). En el modelo ajustado por edad, la CAA mostró una tendencia a la asociación con IC (OR 3,44; IC 95%: 0,883-13,40; p=0,075). En el modelo parsimonioso, la CAA permaneció como predictor independiente (OR 5,25; IC 95%: 1,42-19,40; p=0,0128). Desempeño del modelo: área bajo la curva (AUC) 0,81 (IC 95%: 0,71-0,91), buena calibración (Hosmer-Lemeshow p=0,246) y Brier 0,081. Discusión: en DM2, la CAA se relacionó con mayor probabilidad de IC y podría actuar como marcador de riesgo cardiovascular subclínico de fácil acceso. Se requieren estudios prospectivos para confirmar causalidad y utilidad pronóstica.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is associated with accelerated vascular aging and a high prevalence of atherosclerotic disease, which represents the main cause of morbidity and mortality in these patients [1-3]. Among structural vascular alterations, abdominal aortic calcification (AAC) is recognized as an early and powerful marker of subclinical cardiovascular disease (CVD) [4]. This process reflects the interplay of chronic inflammation, endothelial dysfunction, and vascular mineralization phenomena mediated by smooth muscle cells, which lead to arterial elasticity loss and increased vascular stiffness [5,6].

The first studies exploring this relationship date back to the late 1980s. Siitonen et al. demonstrated that patients with non-insulin-dependent diabetes had a higher frequency of aortic calcifications compared with non-diabetic subjects, particularly at the level

of the abdominal aorta [7]. Subsequently, Niskanen et al. confirmed these findings in a five-year prospective study, showing a higher incidence of aortic and iliofemoral calcification in individuals with newly diagnosed T2DM -especially in women- and its association with intermittent claudication during follow-up [8].

More recent reviews suggest that T2DM is associated with both intimal (atherosclerotic) and medial calcification, and that AAC occurs more frequently and extensively in individuals with diabetes, even after adjusting for traditional risk factors such as hypertension and dyslipidemia [9,10]. Likewise, greater AAC severity is associated with a significant increase in major cardiovascular events -myocardial infarction, stroke, and cardiovascular death- reinforcing its value as an independent prognostic marker [11].

In this context, radiographic assessment of AAC, a simple, accessible, and reproducible tool, represents an opportunity for early detection of subclinical vascular disease in patients with T2DM, potentially useful for optimizing risk stratification and therapeutic strategies. This study described the clinical and therapeutic characteristics of a T2DM cohort and evaluated the association between AAC and ischemic heart disease; secondarily, it explored its relationship with other macrovascular complications.

MATERIALS AND METHODS

Study design and population

An observational, cross-sectional, and analytical study was conducted as part of a research project approved by the Institutional Ethics Committee. Patients over 18 years of age with a confirmed diagnosis of T2DM who provided written informed consent were included. Of the 209 patients with T2DM in the database, 118 had a lumbar spine radiograph suitable for AAC evaluation, constituting the final analysis sample.

Data collection

Demographic, clinical, and biochemical data were collected through structured interviews, medical record reviews, and laboratory analyses. The recorded variables included age, sex, duration of T2DM, type and number of hypoglycemic agents used (oral antidiabetic drugs and insulin), and glycated hemoglobin (HbA_{1c}) levels. Microvascular complications -diabetic retinopathy, nephropathy, and peripheral neuropathy- as well as macrovascular complications, including ischemic heart disease, cerebrovascular disease, and peripheral arterial disease, were also documented.

Evaluation of abdominal aortic calcification

In a subgroup of 118 patients, the presence of AAC was assessed using a lateral lumbar spine radiograph. Quantification was performed using the Kauppila index, which scores the extent of calcification in the aortic segments adjacent to vertebrae L1–L4, with total values ranging from 0 to 24 points [12]. All studies were interpreted by experienced observers blinded to the clinical variables.

Statistical analysis

Statistical analysis described continuous variables as median and interquartile range (IQR) and categorical variables as frequencies and percentages. Group comparisons were performed using the Mann–Whitney test for continuous variables and the chi-square test; when expected frequencies were <5, Fisher's exact test was applied.

The primary outcome was ischemic heart disease, and the main exposure was AAC, assessed on lumbar radiographs using the Kauppila index and modeled dichotomously (present/absent). A binary logistic regression model was fitted with AAC as the predictor, and age and duration of diabetes were included as a priori covariates; additionally, a parsimonious model using backward elimination (Wald criterion) was explored as a sensitivity analysis. Results were expressed as odds ratios (OR) with 95% confidence intervals (95% CI) and two-sided p-values, with statistical significance set at $p<0.05$.

Model performance was evaluated using

the area under the ROC curve (AUC) with 95% CI calculated by the DeLong method, calibration with the Hosmer–Lemeshow test, the Brier score, and the calibration curve (intercept and slope), as well as the Nagelkerke pseudo-R^2 for explained variance. Collinearity among predictors was checked using the variance inflation factor (VIF). All analyses were performed using R version 4.3.0® (R Foundation for Statistical Computing, Vienna, Austria) with the package's *stats*, *pROC*, *DescTools*, *ResourceSelection*, *rms*, *car*, and *ggplot2*, as appropriate. Since AAC could only be assessed in patients with interpretable radiographs, analyses involving this variable were conducted using a complete-case approach (n=118).

RESULTS

General characteristics of the population

A total of 209 patients with a diagnosis of T2DM were included, of whom 55.9% were women. The median age was 62 years (IQR 56-67), and the median disease duration was 8 years (IQR 1-15). The median HbA_{1c} was 7.1% (IQR 6.4-8.2), with 69.1% of patients showing values >6.5%.

Overall, 87.1% were treated with oral antidiabetic drugs (OAD), and 28.2% received insulin. The most frequently used medication was metformin (83.3%), followed by DPP-4 inhibitors (30.1%) and sulfonylureas (20.1%). Only 1.9% of patients were treated with pioglitazone, and 7.2% received sodium-glucose cotransporter 2 inhibitors (SGLT2i) (Table 1).

Patients treated with insulin had a longer duration of T2DM (15 years; IQR 8–20) compared with those not receiving insulin (5 years; IQR 2-10), p<0.0001, and exhibited higher HbA_{1c} levels (7.9% vs. 6.8%; p<0.0001).

Microvascular and macrovascular complications

A total of 23.6% of patients presented microvascular complications, including diabetic retinopathy (8.7%), nephropathy (9.1%), and peripheral neuropathy (10.1%). Patients with microangiopathy showed higher HbA_{1c} levels (7.6% vs. 7.0%; *p*=0.0375).

Macrovascular complications were recorded in 16.7% of patients, corresponding to ischemic heart disease (9.6%), peripheral arterial disease (6.2%), and cerebrovascular disease (2.9%). The duration of T2DM was significantly longer in those with macrovascular complications, 13 years (IQR 8-20) vs. 6 years (IQR 2.5-14) in those without (p=0.0013).

Median HbA_{1c} did not differ between patients with and without cardiovascular disease: 7.55% (IQR 6.28–8.28) vs. 6.80% (IQR 6.20-8.00), respectively (p=0.43). Patients with ischemic heart disease were older (65 years; IQR 62.8-67) compared with those without the condition (61 years; IQR 55-67), a statistically significant difference (p=0.03). The duration of

Table 1. Distribution of antihyperglycemic treatment in the total cohort

Antihyperglycemic treatment	Frequency (n)	Percentage (%)
No pharmacological treatment	10	4,8
1 OAD	71	34,0
2 OAD	48	23,0
3 OAD	18	8,6
4 OAD	3	1,4
Insulin monotherapy	14	6.7
OAD + insulina	45	21,5

Abbreviations: OAD, oral antidiabetic drugs.

T2DM was also significantly longer in those with ischemic heart disease -15 years (IQR 7.5-20.0) vs. 7 years (IQR 3.0-14.25) in those without (p=0.036)-.

Abdominal aortic calcification

Of the 132 lumbar spine radiographs obtained, 118 were technically evaluable and constituted the analytical cohort. In this subgroup, 36 patients (30.5%) presented AAC, with a median Kauppila index of 8 points (IQR 4-12.25). Patients with AAC were older (65 years; IQR 63-68) than those without AAC (59 years; IQR 51.25-65.75), a statistically significant difference (p=0.0002). The duration of T2DM was longer in patients with AAC compared to those without [10 years (IQR 5.0-20.0) vs. 6.0 years (IQR 2.25-14.0); p=0.0246].

Macrovascular complications were observed in 47.6% of patients with AAC (*p*=0.06), with a significantly higher prevalence of ischemic heart disease compared with those without AAC (66.7% vs. 26.4%; p=0.004).

No differences were found in 25(OH)D levels, serum calcium, phosphate, alkaline phosphatase, or calcium–phosphate product between patients with and without AAC. HbA_{1c} levels also did not differ according to the presence of calcification.

Logistic modeling of predictors of ischemic heart disease

In the logistic regression analysis with ischemic heart disease as the outcome, AAC showed a positive association that was attenuated after adjustment for age (full model – AAC, years of T2DM, and age: OR 3.14; 95% CI: 0.79-12.40; *p*=0.103; age-adjusted model: OR 3.44; 95% CI: 0.883-13.40; *p*=0.075). Neither years of T2DM nor age reached statistical significance (p=0.285 and p=0.083, respectively).

In a parsimonious model (stepwise selection), AAC remained the only predictor and showed a significant association (OR 5.25; 95% CI: 1.42-19.40; p=0.0128). Given the biological plausibility of age as a confounder, the age-adjusted model is presented as the primary analysis, with the parsimonious model considered a sensitivity analysis.

The discriminative ability was good: AUC (C-statistic) = 0.81 (Figure 1). Calibration was adequate: Hosmer–Lemeshow χ^2=10.29 (df=8), p=0.246; Brier score = 0.081; calibration curve with intercept ≈ 0 and slope ≈ 1 (Eavg=0.027). The explained variance (Nagelkerke R^2) was 0.195. No relevant collinearity was detected (VIF: AAC 1.06; years of T2DM 1.01; age 1.05) (Table 2).

DISCUSSION

In our cohort, AAC detected on lateral lumbar radiography was a common finding and was consistently associated with a higher likelihood of ischemic heart disease. Beyond its simplicity and low cost, AAC behaved as a "fingerprint" of cumulative vascular damage, appearing more frequently in older individuals and those with a longer duration of diabetes. This pattern, consistent with the pathophysiology of vascular mineralization in T2DM, suggests that incorporating AAC assessment could facilitate the detection of subclinical cardiovascular risk and the prioritization of cardioprotective interventions in daily clinical practice.

Our results expand upon both historical observations and contemporary evidence. As early as the 1980s–1990s, Siitonen et al. and Niskanen et al. documented a higher frequency of aortic calcification in diabetes and its relationship with claudication and cerebrovascular events during follow-up [7,8]. More recent reviews suggest that T2DM is associated with both intimal (atherosclerotic) and medial calcification, and that AAC is more prevalent and extensive in diabetes even after adjustment for traditional risk factors [9,10]. Likewise, a higher AAC burden has been linked to major cardiovascular events and mortality, supporting its role as an independent prognostic marker [11].

In the Framingham Heart Study, AAC was independently associated with coronary and cerebrovascular disease, even after adjusting for traditional risk factors [13]. Similarly, a recent

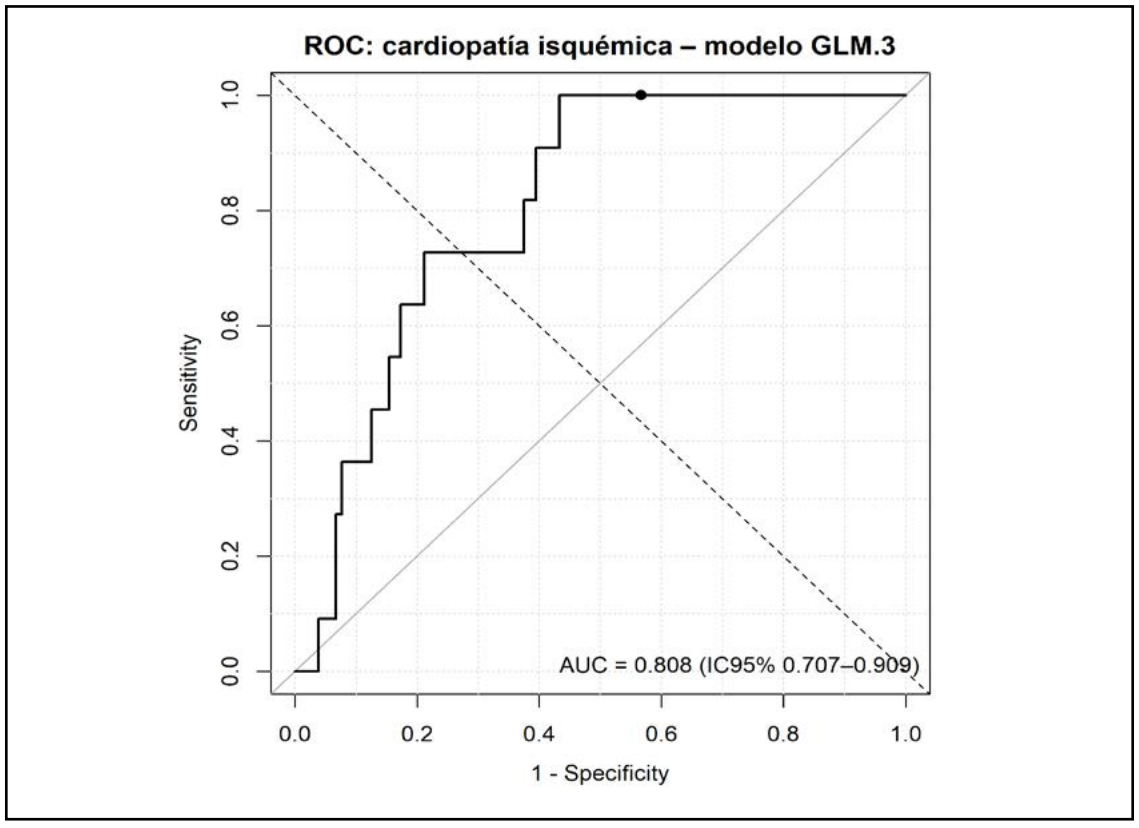

Figure 1. ROC curve of the model for ischemic heart disease.
ROC curve of the logistic regression model predicting ischemic heart disease in patients with T2DM. The area under the curve (AUC) was 0.808 (95% CI: 0.707–0.909), indicating good discriminative ability. The solid line represents sensitivity versus 1–specificity across all cutoff points, while the diagonal line represents random classification (AUC=0.5).

Table 2. Association between abdominal aortic calcification (AAC) and ischemic heart disease (IHD) in patients with T2DM

Model	Predictor	OR	95% CI	p
Full (AAC + years of T2DM + age)	AAC (yes vs. no)	3,14	0,79-12,40	0,103
	Years of T2DM (per year)	1,04	0,97-1,12	0,285
	Age (per year)	1,08	0,98-1,19	0,107
Age-adjusted	AAC (yes vs. no)	3,44	0,883-13,40	0,075
	Age (per year)	1,08	0,989-1,19	0,083
Parsimonious	AAC (yes vs. no)	**5,25**	**1,42-19,40**	**0,0128**

Logistic regression models. Outcome: ischemic heart disease (1=present; 0=absent). Predictors: AAC (yes vs. no); age and years of T2DM modeled per 1-year increase. Samples: full and age-adjusted models calculated in *n*=118 patients with evaluable radiographs. The "age-adjusted" model includes AAC and age; the "parsimonious" model was derived through stepwise selection (Wald criterion) and retained only AAC. Model performance metrics (age-adjusted model): AUC=0.81; Nagelkerke R^2=0.195; Brier=0.081; Hosmer–Lemeshow χ^2=10.29 (df=8), p=0.246.
Abbreviations: T2DM, type 2 diabetes mellitus; OR, odds ratio; 95% CI, 95% confidence interval. Bold values indicate $p<0.05$.

meta-analysis confirmed that AAC predicts cardiovascular events and mortality both in the general population and in individuals with chronic comorbidities [14]. Among people with diabetes, Echouffo-Tcheugui et al. showed that T2DM is associated with greater arterial calcification burden and higher cardiovascular risk, highlighting the value of assessing calcification across different vascular beds [10]. Reaven and Sacks also reported that coronary and abdominal aortic calcifications are independently associated with CVD in T2DM, reinforcing the role of AAC as a clinically relevant risk marker in this population [15]. In our cohort, the strongest association was observed with ischemic heart disease, consistent with these previous findings.

In this single-center cohort of individuals with T2DM, AAC -quantified by the Kauppila index on lumbar radiography- was frequent (30.5%) and associated with a higher likelihood of ischemic heart disease. The strength of the association was robust in the bivariate analysis and remained as a trend after adjustment for age (OR 3.44; 95% CI: 0.883–13.40), reaching statistical significance in the parsimonious model (OR 5.25; 95% CI: 1.42-19.40). The model showed good discrimination (AUC 0.81) and adequate calibration, suggesting that -even within a cross-sectional design and a limited set of covariates- the prognostic signal of AAC for ischemic heart disease is consistent.

Moreover, AAC was observed in older individuals and those with longer diabetes duration, consistent with its nature as a cumulative marker of vascular injury. AAC reflects the interplay of chronic inflammation, endothelial dysfunction, and osteogenic transdifferentiation of smooth muscle cells-processes promoted by sustained hyperglycemia, oxidative stress, advanced glycation end products, and disturbances in phosphocalcic metabolism [5,6]. These mechanisms lead to arterial stiffness and increased hemodynamic load, facilitating myocardial ischemia even in the absence of critical coronary stenosis.

The lack of association between HbA_{1c} and AAC/CVD in our study likely reflects the cross-sectional design (a "snapshot" of glycemic control) and the fact that vascular mineralization integrates long-term exposures (diabetes duration, age) and other determinants (blood pressure, lipids, smoking, renal function) not fully captured in the model.

Assessment of AAC using lateral lumbar radiography -an inexpensive, low-radiation, and widely available test- may add value to risk stratification in T2DM by identifying patients at elevated subclinical risk who could benefit from intensive treatment optimization (lipid and blood pressure control, smoking cessation, and the use of cardioprotective drugs such as SGLT2 inhibitors or glucagon-like peptide-1 receptor agonists [GLP-1 RA], and, in selected cases, aspirin for primary prevention according to the individual risk profile) [12]. The good performance of the model suggests that AAC could be integrated into existing prediction models, although this requires validation.

Among the strengths of this study are the use of a standardized method (Kauppila index) and the formal assessment of model performance (discrimination, calibration, and R^2). Although computed tomography allows a more sensitive quantification of atherosclerotic burden, lateral radiography offers advantages in cost and accessibility, particularly in resource-limited settings [16].

The main limitations include the cross-sectional design (which precludes causal or temporal inference), the complete-case analysis for AAC (118 evaluable radiographs) with potential selection bias, and the limited sample size for the ischemic heart disease outcome, reflected in wide confidence intervals and *p*-values near the threshold of significance. In addition, not all potential confounders were adjusted for (e.g., smoking, detailed lipid profile, renal function, physical activity), so residual confounding cannot be ruled out.

Prospective studies are needed to assess the incremental predictive value of AAC over

traditional atherosclerotic cardiovascular disease risk scores using reclassification metrics -net reclassification improvement and integrated discrimination improvement- and decision curve analysis, as well as to evaluate its temporal evolution and response to interventions (intensive blood pressure and lipid control, and antidiabetic agents with cardiovascular benefit). It will also be useful to compare AAC with other measures of calcification (computed tomography coronary calcium score) and to explore specific subgroups (age, sex, renal function). Pragmatic trials could test AAC screening strategies to guide therapeutic intensity.

In conclusion, in individuals with T2DM, AAC detected on lumbar radiography was associated with a higher likelihood of ischemic heart disease and showed good discriminative performance, supporting its role as an easily accessible marker of subclinical cardiovascular risk. Although age attenuated the association in the adjusted model, the signal persisted and remained significant in the parsimonious analysis. Prospective validation and assessment of its clinical impact will be key for its routine adoption.

Conflicts of interest

The authors declare no conflicts of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author contributions

All authors participated in the conception of the study and critically reviewed the content. All contributed to the drafting of the manuscript and approved the final version for publication.

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