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Cardiovascular Risk Prediction in Patients with Type 2 Diabetes Using the DIAL Model: A Retrospective Study

Zakaria Laanibi[1]*, Asmaa Biaz[1], Samira El Machtani Idrissi[1], Ahmed Anas Guerboub[2], Sanae Bouhsain[1]

[1]Biochemistry Laboratory of the Mohammed V Military Teaching Hospital in Rabat, 10000, Morocco
[2]Endocrinology Department of the Mohammed V Military Teaching Hospital in Rabat, 10000, Morocco

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***Corresponding author:** Zakaria Laanibi
Biochemistry Laboratory of the Mohammed V Military Teaching Hospital in Rabat, 10000, Morocco

Abstract **Original Research Article**

Background and Aims: Type 2 diabetes mellitus (T2DM) confers a substantially elevated cardiovascular risk, yet personalised lifetime-risk estimation tools are rarely applied in routine clinical practice. The DIAL (Diabetes Lifetime-perspective Prediction) model, endorsed by the European Society of Cardiology (ESC 2021), simultaneously estimates 10-year cardiovascular risk, lifetime risk, and cardiovascular event (CVE)-free life expectancy. To quantify 10-year and lifetime cardiovascular risk and CVE-free life expectancy in a hospital cohort of T2DM patients, and to identify clinical and biochemical determinants of these DIAL outputs. ***Methods:*** Retrospective study, Endocrinology Department, Mohammed V Military Teaching Hospital, Rabat (January 2021 – December 2022). One hundred adults with T2DM and complete records were included. DIAL scores were calculated via www.U-Prevent.com. Associations between clinical variables and DIAL outputs were examined by simple linear regression and logistic regression (Jamovi 2.3.2); significance threshold $p < 0.05$. ***Conclusions:*** Mean age was 58.2 years; mean HbA1c 11.1%; 56% had a prior cardiovascular event; 41% had chronic kidney disease (CKD). CVE-free life expectancy was < 10 years in 19% of patients; 23% had a 10-year cardiovascular risk > 10%; and 21% had a lifetime risk > 70%. In multivariable-adjusted linear regression, CVE-free life expectancy was significantly reduced by CKD, prior CVD (–6.7 years), hypertension (–6.0 years), and cumulative diabetes duration (–0.03 years per year). Ten-year and lifetime cardiovascular risks increased progressively with albuminuria severity and CKD stage. Renal impairment, prior CVD, hypertension, and long diabetes duration are the dominant risk drivers.

Keywords: Type 2 diabetes mellitus; cardiovascular risk; DIAL model; lifetime risk; kidney disease; dyslipidaemia.

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) affects approximately 537 million adults worldwide and is projected to reach 783 million by 2045 [1]. In Morocco, the 2018 national STEPS survey identified 2.7 million people with diabetes (types 1 and 2), nearly half of whom were undiagnosed, alongside 2.2 million with prediabetes [2]. Beyond glycaemic dysregulation, T2DM is characterised by a pro-atherogenic lipid phenotype — hypertriglyceridaemia, low HDL-cholesterol, and small dense LDL particles — that compounds vascular risk [3,4].

Accurate cardiovascular risk stratification is central to T2DM management because it determines the intensity of preventive interventions. Most widely used tools — UKPDS Risk Engine, ADVANCE, Fremantle — provide only 5- to 10-year risk estimates, which may underestimate cumulative lifetime burden, particularly in younger patients [5,6]. The DIAL (Diabetes Lifetime-perspective Prediction) model, developed from pooled data of more than 500,000 patients with T2DM and formally recommended by the ESC in 2021 [7,8], addresses this gap by simultaneously estimating 10-year cardiovascular risk, lifetime risk, and CVE-free life expectancy, and by simulating the benefit of specific pharmacological strategies.

Despite its clinical relevance, DIAL has not yet been applied to North African or Sub-Saharan African populations. This study therefore aimed to (i) describe the distribution of DIAL-derived cardiovascular risk estimates in a Moroccan T2DM hospital cohort, and (ii) identify the clinical and biochemical predictors of elevated cardiovascular risk within this framework.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

We conducted a retrospective cross-sectional analysis in the Endocrinology Department of the Mohammed V Military Teaching Hospital (HMIMV), Rabat, Morocco. Consecutive patients with T2DM admitted between 1 January 2021 and 31 December 2022 and with complete clinical records were eligible.

2.2 Inclusion and Exclusion Criteria

Patients were included if they had a confirmed diagnosis of T2DM and complete data for all DIAL input variables. Patients were excluded if they had (i) incomplete medical records, (ii) any active malignancy (ICD-10 codes C00–C97), or (iii) advanced CKD (stages 4–5, eGFR < 30 ml/min/1.73 m²), in keeping with DIAL model exclusion criteria [8].

2.3 Data Collection

Demographic, clinical, and laboratory data were extracted from paper and electronic medical records into a 817tandardized Microsoft Excel 2016 spreadsheet. Variables collected included: age, sex, smoking status (current/ex/never), diabetes duration, systolic and diastolic blood pressure, BMI, insulin therapy, lipid-lowering therapy, and antithrombotic treatment; total cholesterol, triglycerides, HDL-C, LDL-C (Friedewald), HbA1c (IFCC), eGFR (CKD-EPI 2021 equation), and albumin-to-creatinine ratio (ACR). Cardiovascular history (prior myocardial infarction, stroke, heart failure, peripheral artery disease) was recorded from physician notes and discharge summaries.

2.4 DIAL Model Application

Individual DIAL scores were calculated by entering each patient's data into the freely accessible web calculator (www.U-Prevent.com). Outputs were: (i) CVE-free life expectancy (years to first non-fatal myocardial infarction or stroke), (ii) 10-year cardiovascular risk (%), and (iii) lifetime cardiovascular risk (%). The calculator also generated estimated absolute risk reductions associated with statin, antihypertensive, and glucose-lowering therapy intensification.

2.5 Statistical Analysis

Descriptive statistics are reported as mean ± SD for continuous variables and as frequency (%) for categorical variables. Associations between independent clinical variables and each continuous DIAL output were explored by simple linear regression (coefficients reported as β with 95% CI). Binary logistic regression was used for dichotomised outcomes (e.g., 10-year CVR > 10% vs. ≤ 10%). Categorical predictors were entered as dummy variables. All analyses were performed in Jamovi 2.3.2. Statistical significance was set at $p < 0.05$.

This study was conducted in accordance with the Declaration of Helsinki. Institutional review board approval was obtained from the HMIMV ethics committee. Given the retrospective design, individual informed consent was waived.

3. RESULTS

3.1 Patient Characteristics

100 patients were included (58 men, 42 women; M:F ratio 1.38:1). Key baseline characteristics are presented in Table 1. The cohort was middle-aged (mean 58.2 ± 10.7 years), with long-standing, poorly controlled diabetes (mean duration 13.0 ± 9.8 years; mean HbA1c 11.1%); 92.3% did not meet their individualised HbA1c target.

Table 1: Baseline patient characteristics (n = 100)

Characteristic	Value / n (%)	Reference / Notes
Total patients	100	
Sex (M/F)	58/42 (ratio 1.38)	
Mean age (years)	58.2 ± 10.7 (19–79)	
Mean diabetes duration (years)	13.0 ± 9.8 (0–40)	
Mean HbA1c (%)	11.1	IFCC standard
Uncontrolled glycaemia (HbA1c above target)	92.3%	Per HAS individualized targets
Mean BMI (kg/m²)	28.4	
Overweight (BMI 25–29.9)	31%	
Obesity (BMI ≥ 30)	30%	
Hypertension	44%	
History of cardiovascular disease	56%	Includes HF, IHD, stroke
Active smoking	16%	
Insulin therapy	65%	
Statin therapy	32%	
Low HDL-C	54%	
Hypertriglyceridaemia	38%	
Elevated LDL-C	15–17%	
Elevated total cholesterol	10%	
CKD (any stage)	41%	eGFR CKD-EPI
Severely elevated albuminuria	14%	ACR

3.2 Lipid Profile

The lipid phenotype was consistent with the classic T2DM dyslipidaemia triad: low HDL-C (54%), hypertriglyceridaemia (38%), and elevated LDL-C (15–17%). Mixed dyslipidaemia was present in 38% of patients. Total hypercholesterolaemia was observed in only 10%, underscoring those standard lipid thresholds alone may underestimate atherogenic risk in this population.

Among the 72 patients with dyslipidaemia, 64% were not receiving statin therapy. Of those with elevated LDL-C specifically, 81% were untreated with statins, representing a major treatment gap.

3.3 DIAL Model Outputs

The distribution of DIAL model outputs is summarised in Table 2. Nineteen percent of patients had a CVE-free life expectancy < 10 years; 23% had a 10-year CVR > 10%; and 21% had a lifetime CVR > 70%. The majority (62%) had a CVE-free life expectancy between 10 and 30 years, and 64% had a lifetime CVR between 5% and 20%.

Table 2. Distribution of DIAL model outputs (n = 100)

DIAL Outcome	Category	Patients (%)
CVE-free life expectancy	< 10 years	19%
	10–30 years	62%
10-year CVR	> 10%	23%
	1–5%	52%
Lifetime CVR	> 70%	21%
	5–20%	64%

3.4 Determinants of DIAL Outputs (Regression Analysis)

Regression findings are summarised in Table 3. In brief:

CVE-free life expectancy was significantly reduced by CKD (β = –9.31 years for mild, –19.18 years for moderate CKD; $p < 0.001$), prior CVD (β = –6.69 years; $p < 0.05$), hypertension (β = –6.00 years; $p < 0.05$), and diabetes duration (β = –0.028 years per year; $p < 0.05$). Albuminuria showed a progressive, graded inverse association with CVE-free life expectancy.

10-year cardiovascular risk was significantly increased by prior CVD (β = +13.21%; $p < 0.05$) and by increasing CKD stage and albuminuria severity (graded positive β; $p < 0.05$ at each level).

Lifetime cardiovascular risk was most strongly increased by prior CVD (β = +24.38%; $p < 0.001$) and by progressive albuminuria and CKD (graded positive β; $p < 0.05$).

Total cholesterol, HDL-C, and dyslipidaemia per se did not reach statistical significance in simple linear regression for any DIAL output, likely reflecting the already-captured effects of these variables within the composite risk score itself.

Table 3: Summary of regression associations between clinical variables and DIAL outputs

Variable	Outcome	β coefficient	p-value
Hypertension	CVE-free LE	–6.00 years	< 0.05
History of CVD	CVE-free LE	–6.69 years	< 0.05
Diabetes duration (per year)	CVE-free LE	–0.028 years	< 0.05
CKD (moderate)	CVE-free LE	–19.18 years	< 0.001
CKD (mild)	CVE-free LE	–9.31 years	< 0.001
History of CVD	10-yr CVR	+13.21%	< 0.05
CKD / albuminuria (progressive)	10-yr CVR	Positive, graded	< 0.05
History of CVD	Lifetime CVR	+24.38%	< 0.001
Albuminuria severity	Lifetime CVR	Positive, graded	< 0.05

4. DISCUSSION

To our knowledge, this is the first study to apply the DIAL model to a North African or Sub-Saharan cohort of T2DM patients. Our data show that despite a high prevalence of classical cardiovascular risk factors — poorly controlled diabetes (HbA1c 11.1%), widespread dyslipidaemia (72%), and a 56% prevalence of prior CVD — only 21% of patients had a lifetime CVR > 70% and 23% had a 10-year CVR > 10%. This apparently paradoxical finding warrants careful interpretation.

The lower-than-expected rate of high 10-year risk may partly reflect the relatively young mean age (58 years) of our cohort, since DIAL assigns greater absolute weight to age in short-horizon estimation. By contrast, lifetime risk estimates may more faithfully capture cumulative burden. Compared with Amellouk *et al.,*[9], who reported 40% high CVR using a factor-summing approach in a Moroccan T2DM population, our DIAL-based figure of 21% reflects the more conservative, model-based quantification of absolute risk rather than a categorical risk-factor count.

4.1 Renal Impairment as the Dominant Risk Driver

The most clinically striking finding was the magnitude of risk attributable to CKD. Moderate CKD was associated with a reduction of 19.18 years in CVE-free life expectancy, substantially greater than the effect of prior CVD (–6.69 years) or hypertension (–6.00 years). These effects are biologically plausible: CKD accelerates vascular ageing through uraemic toxin accumulation, endothelial dysfunction, and dysregulation of mineral metabolism. Our findings are directionally consistent with data from the CKD Prognosis Consortium, which demonstrated that each 10 ml/min/1.73 m² reduction in eGFR independently increases major cardiovascular event risk [10]. The graded association between albuminuria severity and all three DIAL outputs further supports glomerular filtration and tubular integrity as major determinants of residual cardiovascular risk in T2DM.

4.2 Prior Cardiovascular Disease

A history of CVD was the strongest single predictor of both 10-year (β = +13.21%) and lifetime (β = +24.38%) cardiovascular risk, and reduced CVE-free life expectancy by nearly 7 years. The high prevalence of prior CVD in our cohort (56%) — comparable to Amellouk *et al.,* (59%) [9] and likely reflecting referral bias in a tertiary military hospital — emphasises the secondary prevention imperative. Yet only 32% of our patients were on statins, a stark treatment gap given current ESC guideline recommending high-intensity statin therapy for all T2DM patients with established CVD [7].

4.3 Hypertension and Diabetes Duration

Hypertension reduced CVE-free life expectancy by 6 years in our cohort, corroborating the UKPDS finding that tight blood pressure control reduces diabetes-related mortality by 32% [11]. The effect of diabetes duration (–0.028 years of CVE-free life expectancy per year of diabetes) is modest in absolute terms but cumulative: a patient with 30 years of diabetes duration would lose nearly one additional year of event-free life solely attributable to disease chronicity, consistent with Framingham data showing a 38% increase in CHD risk per additional 10 years of T2DM [12].

4.4 Dyslipidaemia and Treatment Gaps

Despite the predominance of the atherogenic lipid triad (low HDL-C 54%, hypertriglyceridaemia 38%, elevated LDL-C 17%), individual lipid variables did not achieve significance in simple regression — most likely because the DIAL model internally captures their composite contribution. Clinically, however, the finding that 64% of dyslipidaemic patients and 81% of those with elevated LDL-C were not on statins represents a critical gap. Several factors may explain this undertreatment: limited awareness of cardiovascular risk among patients and practitioners, cost constraints, and insufficient implementation of risk stratification tools. Our findings underscore the need for systematic lipid-lowering therapy initiation guided by formal risk quantification tools such as DIAL.

4.5 Limitations

Several limitations deserve acknowledgement. First, the retrospective single-centre design introduces selection bias: patients at a military tertiary hospital may differ systematically from the general Moroccan diabetic population in terms of age, comorbidity, and treatment access. Second, with 100 patients, this study was insufficiently powered for multivariable-adjusted models or subgroup analyses; regression findings should therefore be considered exploratory. Third, because DIAL was developed and validated on predominantly European cohorts, its calibration may not be optimal in our population; external validation studies are needed. Finally, we did not assess the impact of DIAL-guided therapeutic adjustments on subsequent outcomes — a prospective interventional study would be required to demonstrate that the tool improves clinical endpoints beyond risk stratification alone.

5. CONCLUSION

In this first application of the DIAL model to a Moroccan T2DM cohort, we observed a meaningful proportion of patients with high lifetime cardiovascular risk. Renal impairment, prior cardiovascular disease, hypertension, and long diabetes duration emerged as the primary modifiable and non-modifiable determinants of elevated cardiovascular risk. Our data expose a substantial statin treatment gap: the majority of dyslipidaemic patients, including those with elevated LDL-C, were not receiving lipid-lowering therapy at study enrolment.

The DIAL calculator offers a practical, freely accessible tool for personalised risk communication and therapeutic decision-making in T2DM. Its integration into routine outpatient diabetes care in Morocco and the broader North African region requires prospective validation in larger, population-representative cohorts. Such studies should also evaluate whether DIAL-guided treatment intensification translates into measurable improvements in cardiovascular outcomes.

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