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Undiagnosed chronic kidney disease in people with type 2 diabetes in Spain: Prevalence, treatment patterns and associated factors in a multicentre cross-sectional study

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ABSTRACT

Background and aim: Chronic kidney disease (CKD) remains underdiagnosed in people with type 2 diabetes mellitus (T2DM), particularly in early stages, despite its strong association with renal disease progression, cardiovascular outcomes, and current guideline recommendations for early cardio-renal protective interventions. Contemporary, nationally representative primary care data from Spain evaluating CKD underdiagnosis in people with T2DM are limited. This study estimated the prevalence of diagnosed and undiagnosed CKD in adults with T2DM in Spain, described treatment patterns by CKD diagnosis status, and identified factors associated with undiagnosed CKD.

Methods: A planned secondary analysis of the DIAMOND2 multicentre cross-sectional study was performed in Spanish primary care. Data were retrospectively collected from electronic medical records during the calendar year 2022, and the analysis was performed between January and July 2023. Data from 5009 adults with T2DM randomly selected from 70 centres were analysed. CKD was defined according to KDIGO 2024 criteria as estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m^2 and/or urine albumin-to-creatinine ratio (uACR) ≥ 30 mg/g. Patients were classified as having diagnosed CKD (recorded), undiagnosed CKD (meeting criteria without record), or no CKD. Descriptive statistics were used, and multivariable Poisson regression models with robust variance were fitted to identify factors associated with undiagnosed CKD.

Results: True CKD prevalence was 32.0 %, with 54 % undiagnosed. Undiagnosed CKD was mainly in patients with isolated eGFR or uACR abnormalities and lower KDIGO risk. SGLT2 inhibitors were prescribed to 45.2 % of diagnosed and 40.0 % of undiagnosed CKD, versus 35.4 % without CKD ($p < 0.001$). In multivariable analysis, undiagnosed CKD was associated with metformin use and higher eGFR, and inversely with diabetes duration, heart failure, and proliferative retinopathy.

Abbreviations: ACR, albumin-to-creatinine ratio; BMI, body mass index; CI, confidence interval; CKD, chronic kidney disease; CKD-D, diagnosed chronic kidney disease; CKD-UD, undiagnosed chronic kidney disease; DPP-4i, dipeptidyl peptidase-4 inhibitor; EGFR, estimated glomerular filtration rate; GLP-1 RA, glucagon-like peptide-1 receptor agonist; HbA1c, glycated haemoglobin; KDIGO, Kidney Disease: Improving Global Outcomes; LRT, likelihood ratio test; NDM, non-diabetes mellitus; PR, prevalence ratio; ROC, receiver operating characteristic; SGLT2i, sodium-glucose co-transporter-2 inhibitor; SU, sulfonylurea; T2DM, type 2 diabetes mellitus; TZD, thiazolidinedione; UACR, urine albumin-to-creatinine ratio.

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Conclusions: Over half of CKD cases in Spanish adults with T2DM remain undiagnosed, particularly at early disease stages, limiting risk stratification and optimal cardio-renal management. These findings underscore the need for systematic CKD screening and improved recognition of early kidney disease in primary care.

1. Introduction

Type 2 diabetes mellitus (T2DM) is a major global public health concern. According to the World Health Organization (WHO), more than 500 million people are currently living with T2DM worldwide, and this number is expected to exceed 600 million by 2040 [1]. In Spain, the prevalence of T2DM among the adult population is estimated to be around 14 % [2]. This condition not only reduces patients' quality of life but also places a substantial burden on healthcare systems, particularly due to its chronic complications, among which chronic kidney disease (CKD) is one of the most prominent [3].

T2DM is a key contributor to the development and progression of CKD, due to persistent hyperglycaemia, the pro-inflammatory state associated with the disease, and the frequent coexistence of hypertension (HT). These factors contribute to glomerular damage and gradual loss of kidney function [4]. Beyond haemodynamic and metabolic disturbances, type 2 diabetes mellitus is characterised by a state of chronic low-grade inflammation, which plays a central role in the development of microvascular complications, including diabetic kidney disease. Inflammatory pathways and circulating inflammatory markers have been consistently associated with renal damage and disease progression in people with diabetes [5,6]. Diabetes is the leading cause of CKD worldwide, and up to 40 % of individuals with T2DM will develop some degree of CKD during their lifetime [7].

CKD itself represents a significant public health issue, affecting approximately 11 % of the global population, equivalent to more than 850 million people [8]. It is associated with a markedly increased risk of morbidity and mortality, including progression to end-stage kidney disease (ESKD), cardiovascular events, and all-cause death, with cardiovascular mortality being the leading cause of death in these patients [8]. In addition, CKD imposes a considerable economic burden: in Europe, the associated annual costs are estimated to exceed €140 billion [9]. In Spain, these costs were estimated at €4.29 billion in 2022 and are projected to reach €4.89 billion by 2027 [10].

Early detection of CKD in people with T2DM is crucial to slow disease progression and reduce complications. However, despite recommendations from clinical practice guidelines, underdiagnosis of CKD in people with T2DM, particularly in early stages (CKD 1–3), remains high [11,12]. One of the main limitations in routine clinical practice is the underutilisation of the urine albumin-to-creatinine ratio (uACR), one of the key diagnostic criteria for CKD. This is partly due to the low frequency of screening in at-risk individuals, under-recording of uACR in healthcare databases, and the limited clinical recognition of reduced estimated glomerular filtration rate (eGFR) as a marker of kidney disease [13].

In recent years, new therapeutic options with renal and cardiovascular benefits have been introduced for the management of T2DM and CKD. Notably, sodium-glucose co-transporter-2 inhibitors (SGLT2i) and glucagon-like peptide-1 receptor agonists (GLP-1 RA) have been shown to reduce CKD progression and improve cardiovascular outcomes in people with T2DM. However, data on their use in specific patient subgroups, particularly those with undiagnosed CKD, remain limited. In this secondary analysis of the DIAMOND2 study, we aimed to estimate the true prevalence of CKD among adults with T2DM in Spain, distinguishing between diagnosed and undiagnosed cases and describing CKD stages and risk categories among those with complete renal data. We further examined the use of antidiabetic agents according to CKD diagnosis status and identified factors associated with undiagnosed CKD.

2. Methods

2.1. Study design and setting

We conducted a planned secondary analysis of the DIAMOND2 study, a multicentre, cross-sectional, observational study carried out in primary care centres throughout Spain. The DIAMOND2 study methodology has been described in detail previously [14]. In brief, the study was coordinated by the Spanish Diabetes Society (SED), which invited primary care centres nationwide to participate. A total of 70 centres from all Spanish regions took part. Data were retrospectively collected from the electronic medical records of participating centres for the calendar year 2022, and the analysis was conducted over a seven-month period (January–July 2023).

This secondary analysis was planned a priori and falls within the scope of the ethical approval granted by the San Juan de Alicante University Hospital Ethics Committee (approval code: 22/054). The study adhered to the principles of the Declaration of Helsinki and complied with the Spanish Organic Law 3/2018 on Personal Data Protection, and the European General Data Protection Regulation (EU) 2016/679.

2.2. Study population and sampling

The study population comprised adults with a diagnosis of T2DM recorded in their electronic medical records, identified through the presence of a recorded T2DM diagnostic code and/or active prescription of glucose-lowering medication, who had entries related to T2DM in 2022. T2DM-related entries included clinical visits coded for diabetes management, prescription records of antidiabetic therapies, and/or diabetes-related laboratory assessments. For each participant, data were extracted from the last diabetes-related visit recorded during 2022.

Detailed information on the sampling procedure and sample size calculation has been reported previously in the main DIAMOND2 publication [14]. In brief, the study applied a two-stage random sampling process across 70 primary care centres covering all Spanish regions. A randomly selected sample of 5262 adults with T2DM was drawn from a total population of 38,625 patients attended by 309 participating family doctors, ensuring national representativeness of people with T2DM managed in Spanish primary care. The DIAMOND2 dataset is particularly suitable for assessing CKD diagnosis status, as it integrates routinely collected electronic health record data, including both diagnostic coding and laboratory measurements of eGFR and uACR, allowing direct comparison between biological CKD criteria and recorded clinical diagnosis in real-world practice. The final analytical sample for this secondary analysis included 5009 participants, after excluding patients with missing values in key study variables.

2.3. Study variables

Sociodemographic and clinical variables included age, sex, duration of diabetes, comorbidities (hypertension, heart failure, cardiovascular disease, diabetic retinopathy, neuropathy), body mass index (BMI), blood pressure, and lifestyle factors. Laboratory variables comprised glycated haemoglobin (HbA1c), eGFR, and uACR. For each participant, all data corresponded to the last diabetes-related visit recorded in 2022. Antidiabetic treatment was classified by therapeutic group, including metformin, sulfonylureas, DPP-4 inhibitors, SGLT2 inhibitors, GLP-1 receptor agonists, and insulin. Data were extracted by the participating investigators and entered into a shared anonymised electronic registry, designed specifically for the DIAMOND2 study.

The main study variable was CKD status, defined according to the presence of diagnostic codes and/or laboratory criteria. Participants were classified into three mutually exclusive groups:

- Diagnosed CKD (CKD-D): patients with a CKD diagnosis recorded in the electronic medical record (ICD code or structured diagnostic entry), regardless of eGFR or uACR values at the index visit.
- Undiagnosed CKD (CKD-UD): patients without a recorded diagnosis but meeting laboratory criteria for CKD (eGFR < 60 mL/min/1.73 m^2 and/or uACR ≥ 30 mg/g), in accordance with the Kidney Disease: Improving Global Outcomes (KDIGO) 2024 Clinical Practice Guideline.
- No CKD: patients who did not meet either of the above criteria.

Patients with insufficient data to assess renal function (missing eGFR and/or uACR) were categorised separately as "missing data", and excluded from subgroup analyses.

CKD stages and risk categories were determined only among patients with both eGFR and uACR data available. Stages were defined according to KDIGO 2024 as G1 (eGFR $\geq$90 mL/min/1.73 m^2), G2 [60–89], G3a [45–59], G3b [30–44], G4 [15–29], and G5 (<15). Albuminuria was classified as A1 (<30 mg/g, normal to mildly increased), A2 (30–300 mg/g, moderately increased), or A3 (>300 mg/g, severely increased). By combining eGFR (G) and albuminuria (A) categories, patients were assigned to KDIGO risk strata (low, moderate, or high) exclusively based on the G–A heatmap. No additional KDIGO modifiers, such as CKD aetiology, rate of progression, or comorbid conditions, were incorporated into the risk classification.

The primary outcome was the prevalence of undiagnosed CKD among adults with T2DM in Spain. Secondary outcomes included the distribution of CKD stages and KDIGO risk categories among patients with complete renal data; analysis of prescribing patterns of antidiabetic therapies, particularly the use of SGLT2i and GLP-1 RA, and identification of clinical and demographic factors associated with undiagnosed CKD.

2.4. Statistical analysis

A descriptive analysis was performed, calculating frequencies for qualitative variables and means and standard deviations for quantitative variables. The association between qualitative variables and CKD was measured using two-way tables and the chi-square test. eGFR and uACR were categorised according to the KDIGO 2024 Clinical Practice Guideline [15] cut-off points for CKD staging and risk stratification. To analyse the association of explanatory variables with undiagnosed chronic kidney disease, multivariate Poisson models with robust variance were fitted to estimate prevalence ratios and their 95 % confidence intervals. Variable selection was performed using the stepwise procedure based on the Akaike Information Critierion (AIC), aiming to identify a parsimonious model with optimal explanatory performance [16]. Analyses were performed using SPSS v. 29 and R.

3. Results

Of the 5009 individuals with T2DM included in the analysis (mean age of 68 years-old), 3104 (62.0 %) did not present evidence of CKD, 736 (14.7 %) had a CKD diagnosis documented in their medical record (CKD-D), and 864 (17.2 %) met diagnostic criteria for CKD but lacked a

Table 1
Key baseline characteristics of patients according to Chronic Kidney Diseased status (n = 5009).

		No CKD n = 3104		CKD-D n = 736		CKD-UD n = 864		Missing n = 305		p-value
		n	%	n	%	n	%	n	%	
Sex	Male	1760	56.7	428	58.2	466	53.9	161	52.8	0.195
	Female	1344	43.3	308	41.8	398	46.1	144	47.2	
Age (years)	< 55	497	16.0	25	3.4	69	8.0	53	17.4	< 0.001
	55–64	857	27.6	91	12.4	147	17.0	103	33.8	
	65–74	932	30.0	181	24.6	0	26.7	82	26.9	
	75–84	641	20.7	281	38.2	253	29.3	52	17.0	
	> 84	177	5.7	158	21.5	164	19.0	15	4.9	
Duration of diabetes (years)	< 5	798	25.7	67	9.1	180	20.8	102	33.4	< 0.001
	5–9	681	21.9	118	16.0	153	17.7	56	18.4	
	10–14	702	22.6	190	25.8	201	23.3	63	20.7	
	> 14	766	24.7	326	44.3	282	32.6	49	16.1	
	Missing	157	5.1	35	4.8	48	5.6	35	11.5	
Hypertension	No	1020	32.9	62	8.4	146	16.9	101	33.1	< 0.001
	Yes	2067	66.6	667	90.6	708	81.9	183	60.0	
	Missing	17	0.5	7	1.0	10	1.2	21	6.9	
Dyslipemia	No	902	29.1	126	17.1	208	24.1	92	30.2	< 0.001
	Yes	2174	70.0	602	81.8	643	74.4	191	62.6	
	Missing	28	0.9	8	1.1	13	1.5	22	7.2	
Cardiovascular event	No	2312	74.5	399	54.2	566	65.5	169	55.4	< 0.001
	Yes	661	21.3	295	40.1	247	28.6	54	17.7	
	Missing	131	4.2	42	5.7	51	5.9	82	26.9	
GFR (mL/min)	< 60	0	0.0	503	68.3	416	48.1	0	0.0	< 0.001
	≥ 60	3036	97.8	214	29.1	416	48.1	100	32.8	
	Missing	68	2.2	19	2.6	32	3.7	205	67.2	
ACR (mg/g)	< 30	2060	66.4	225	30.6	199	23.0	0	0.0	< 0.001
	≥ 30	0	0.0	343	46.6	533	61.7	0	0.0	
	Missing	1044	33.6	168	22.8	132	15.3	305	100.0	
HbA1c Control	Control < 7 %	1852	59.7	377	51.2	485	56.1	178	58.4	< 0.001
	Uncontrolled ≥ 7 %	1252	40.3	359	48.8	379	43.9	127	41.6	
HbA1c Control according to individualized target	Poor control	1136	39.2	226	34.2	226	34.2	226	34.2	0.019
	Good control	1759	60.8	435	65.8	435	65.8	435	65.8	
BMI (kg/m^2)	BMI < 25	491	18.1	118	18.4	153	21.0	64	26.6	0.029
	BMI 25–29.9	1092	40.1	247	38.4	269	37.0	77	32.0	
	BMI 30–34.9	712	26.2	176	27.4	204	28.0	59	24.5	
	BMI 35–39.9	310	11.4	69	10.7	69	9.5	24	10.0	
	BMI ≥ 40	115	4.2	33	5.1	33	4.5	17	7.1	

Abbreviations: ACR, albumin-to-creatinine ratio; BMI, body mass index; CKD, chronic kidney disease; GFR, glomerular filtration rate;

recorded diagnosis (CKD-UD). In 305 patients (6.1 %), data for eGFR and/or uACR were missing, precluding CKD classification. Overall, 1600 patients fulfilled diagnostic criteria for CKD, of whom 54 % were undiagnosed, yielding a true CKD prevalence of 32 % in the study population.

Key demographic and clinical characteristics of the study population according to CKD status are summarised in Table 1, while the full set of baseline variables is provided in Supplementary Table S1. Of the total population, 2307 (46.0 %) were women and 2702 (54.0 %) were men, with no significant differences in sex distribution observed between the diagnostic groups. Patients with CKD-D had a longer duration of diabetes compared to those with CKD-UD and those without CKD ($p < 0.001$). The prevalence of cardiovascular comorbidities, such as hypertension, dyslipidaemia, heart failure, and established cardiovascular disease, was highest in the CKD-D group ($p < 0.001$). Similarly, diabetic complications, including proliferative retinopathy and diabetic foot, were more common among patients with CKD-D ($p < 0.001$). In contrast, individuals with CKD-UD were more similar to those without CKD in terms of age, comorbidity burden, and diabetes-related complications, but shared similar renal function profiles with the CKD-D group by definition.

3.1. CKD stages and KDIGO risk categories

Table 2 shows the percentage distribution of patients across CKD stages and KDIGO 2024 risk categories among those with available data for both eGFR and albuminuria (n = 3227). Overall, approximately 63 % of participants were located in the upper left area of the KDIGO heatmap, corresponding to preserved eGFR (G1–G2) and normal or mildly increased albuminuria (A1). According to KDIGO risk stratification, 22.6 % of patients were classified as having low risk, 8.1 % as moderate risk, and 5.9 % were considered to have high risk of kidney disease progression or cardiovascular events.

Fig. 1 compares the KDIGO risk levels between patients with diagnosed and undiagnosed CKD. Patients with CKD-D were predominantly classified within the *high* and *moderate* KDIGO risk strata, while those with CKD-UD were mostly distributed within the *low* category.

3.2. Use of antidiabetic medications

Table 3 summarises the use of glucose-lowering therapies by CKD diagnosis status. Overall, patients with CKD-UD received fewer antidiabetic medications compared to those with CKD-D, with the exception of metformin, which was more frequently prescribed in the CKD-UD group. Use of agents with proven cardio-renal-metabolic benefits, such as SGLT2i and GLP-1 RA, was generally low, with less than half of the total study population receiving these treatments. Furthermore, both SGLT2i and GLP-1 RA were prescribed less frequently in the CKD-UD group than in patients with CKD-D. These descriptive differences should be interpreted with caution, as they may be influenced by underlying clinical and demographic characteristics. To account for potential confounding, adjusted analyses of glucose-lowering therapy use according to CKD status are shown in Supplementary Table S2. Use of SGLT2 inhibitors remained higher among both patients with diagnosed CKD (PR 1.28; 95 % CI 1.09–1.50) and those with undiagnosed CKD (PR 1.30; 95 % CI 1.10–1.53), compared with patients without CKD. In contrast, the higher crude use of GLP-1 receptor agonists among patients with diagnosed CKD was no longer significant after adjustment. Metformin use showed a distinct pattern, with a higher prevalence among patients with undiagnosed CKD compared with those without CKD (PR 1.20; 95 % CI 1.09–1.33).

Table 2
Distribution of patients across Chronic Kidney Diseased stages and KDIGO 2024 risk categories among those with available Glomerular filtration Rate and albuminuria data (n = 3227).

KDIGO 2024 Glomerular filtration rate Categories, description, and ranges (mL/min/1.73m²)			**Albuminuria: categories, description, and ranges (mg/g)**		
			A1	**A2**	**A3**
			Normal to mildly increased	Moderately increased	Severely increased
			< 30 mg/g* < 3mg/nmol	**30-300 mg/g* 3-30 mg/nmol**	**> 300 mg/g* > 30 mg/nmol**
G1	Normal or high	**≥ 90**	24.7	5.3	0.6
G2	Mildly decreased	**60-89**	38.0	9.7	1.2
G3a	Mildly to moderately decreased	**45-59**	7.6	3.9	0.5
G3b	Moderately to severely decreased	**30-44**	3.0	2.0	1.0
G4	Severely decreased	**15-29**	0.8	0.9	0.4
G5	Kidney failure	**< 15**	0.1	0.1	0.1

(*) Albuminuria expressed as albumin-to-creatinine ratio (ACR). Percentages indicate the expected distribution of patients in each cell. Colors denote prognosis: no risk (green), low risk (yellow), moderate risk (orange), and high risk (red). Percentages are based on the 3227 patients (64 %) with both eGFR and ACR data available out of the total sample (n = 5009).
Abbreviations: ACR, albumin-to-creatinine ratio; eGFR, estimated glomerular filtration rate

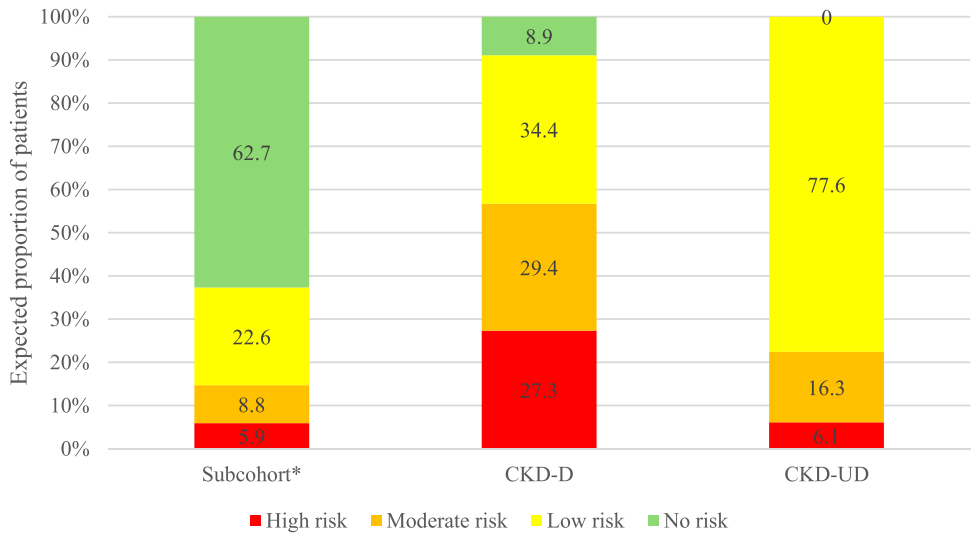

Fig. 1. Distribution of patients with diagnosed and undiagnosed chronic kidney disease across KDIGO 2024 risk categories (n = 3227). * Subcohort includes patients with available albuminuria and glomerular filtration rate (eGFR) data. Abbreviations: CKD-D, diagnosed chronic kidney disease; CKD-UD, undiagnosed chronic kidney disease.

Table 3
Use of medications according to the presence of diagnosed or undiagnosed chronic kidney disease.

		NO CKD		CKD-D		CKD-UD		MISSING		p-value
		n	%	n	%	n	%	n	%	
DPP-4i	No	2081	67.0 %	437	59.4 %	528	61.1 %	174	57.0 %	< 0.001
	Si	953	30.7 %	279	37.9 %	306	35.4 %	77	25.2 %	
	Missing	70	2.3 %	20	2.7 %	30	3.5 %	54	17.7 %	
SGLT2i	No	1934	62.3 %	383	52.0 %	497	57.5 %	153	50.2 %	< 0.001
	Si	1099	35.4 %	333	45.2 %	346	40.0 %	104	34.1 %	
	Missing	71	2.3 %	20	2.7 %	21	2.4 %	48	15.7 %	
SU	No	2794	90.0 %	671	91.2 %	787	91.1 %	220	72.1 %	< 0.001
	Si	224	7.2 %	33	4.5 %	42	4.9 %	29	9.5 %	
	Missing	86	2.8 %	32	4.3 %	35	4.1 %	56	18.4 %	
Glinides	No	2903	93.5 %	655	89.0 %	790	91.4 %	234	76.7 %	< 0.001
	Si	100	3.2 %	57	7.7 %	32	3.7 %	8	2.6 %	
	Missing	101	3.3 %	24	3.3 %	42	4.9 %	63	20.7 %	
Basal insulin	No	2541	81.9 %	490	66.6 %	652	75.5 %	198	64.9 %	< 0.001
	Si	488	15.7 %	226	30.7 %	186	21.5 %	48	15.7 %	
	Missing	75	2.4 %	20	2.7 %	26	3.0 %	59	19.3 %	
Metformin	No	584	18.8 %	288	39.1 %	214	24.8 %	42	13.8 %	< 0.001
	Si	2500	80.5 %	440	59.8 %	638	73.8 %	253	83.0 %	
	Missing	20	0.6 %	8	1.1 %	12	1.4 %	10	3.3 %	
GLP-1 RA	No	2660	85.7 %	585	79.5 %	741	85.8 %	207	67.9 %	< 0.001
	Si	358	11.5 %	126	17.1 %	90	10.4 %	37	12.1 %	
	Missing	86	2.8 %	25	3.4 %	33	3.8 %	61	20.0 %	
TZD	No	2967	95.6 %	692	94.0 %	812	94.0 %	234	76.7 %	< 0.001
	Si	38	1.2 %	14	1.9 %	9	1.0 %	5	1.6 %	
	Missing	99	3.2 %	30	4.1 %	43	5.0 %	66	21.6 %	

Abbreviations: CKD, chronic kidney disease; CKD-D, diagnosed chronic kidney disease; CKD-UD, undiagnosed chronic kidney disease; DPP-4i, DPP-4 inhibitors; GLP-1 RA, GLP-1 receptor agonists; SU, sulfonylureas; SGLT2i, SGLT2 inhibitors; TZD, thiazolidinediones. Green indicates medications with lower use in patients with undiagnosed chronic kidney disease (CKD-UD), and blue indicates medications with higher use in CKD-UD patients.

3.3. Factors associated with undiagnosed CKD

Table 4 presents the results of a multivariable model adjusted for age, sex, comorbidities, complications, and antidiabetic treatments, assessing the prevalence ratio of factors associated with CKD-UD. The factors associated with undiagnosed CKD included a higher likelihood of metformin prescription, shorter duration of T2DM, lower prevalence of heart failure and proliferative retinopathy, and higher eGFR. The moderate discriminative ability of the model suggests that additional unmeasured clinical, organisational, or behavioural factors may also contribute to CKD underdiagnosis.

4. Discussion

This study provides new evidence on the magnitude and characteristics of CKD underdiagnosis among adults with T2DM in Spain. Our findings demonstrate that a substantial proportion of patients with T2DM meet diagnostic criteria for CKD without a recorded diagnosis in primary care electronic health records. Furthermore, these undiagnosed cases are primarily concentrated in the early stages of kidney disease and in the lower KDIGO risk categories, highlighting insufficient recognition of mild renal impairment in real-world clinical practice.

The true prevalence of CKD in our study (32 %) is consistent with prior national estimates [17], but the proportion of patients with

Table 4
Multivariable regression analysis of factors associated with undiagnosed chronic kidney disease (CKD-UD) among patients meeting chronic kidney disease (CKD) diagnostic criteria.

		PR	95 % CI	p-value
Metformin	No	1		
	Yes	1.217	(1.090–1.360)	0.001
	Missing	1.312	(0.921–1.869)	0.132
Duration of diabetes (years)	< 5	1		
	5–9	0.803	(0.708–0.912)	0.001
	10–14	0.755	(0.669–0.851)	< 0.001
	> 14	0.722	(0.645–0.807)	< 0.001
	Missing	0.843	(0.694–1.025)	0.087
Heart failure	No	1		
	Yes	0.701	(0.587–0.837)	< 0.001
	Missing	1.086	(0.904–1.306)	0.377
Proliferative retinopathy	No	1		
	Yes	0.548	(0.428–0.701)	< 0.001
	Missing	0.958	(0.825–1.112)	0.572
eGFR	< 60	1		
	≥ 60	1.317	(1.201–1.444)	< 0.001
	Missing	1.264	(1.015–1.573)	0.036

n = 1600; undiagnosed chronic kidney disease (CKD-UD) = 864.
Likelihood ratio test (LRT) = 84.2 (p < 0.001).
Area under the ROC curve = 0.683 (95 % CI, 0.657–0.709).
Abbreviations: CI, confidence interval; eGFR, estimated glomerular filtration rate; PR, prevalence ratio.

clinically recorded CKD (14.7 %) confirms a persistent gap between biological evidence and diagnostic coding. These results are in line with earlier Spanish and international studies that reported high levels of unrecognised CKD among patients with diabetes [13,18–20]. The ONDAAS study, also conducted in the primary care setting in Spain, demonstrated that the lack of systematic albuminuria testing is a key barrier to early CKD detection, particularly in the initial disease stages [13]. Likewise, large-scale registries such as NHANES (United States) and CKDopps (Europe) have estimated that between 40 % and 60 % of individuals meeting CKD criteria lack a documented diagnosis, limiting appropriate risk stratification and access to evidence-based therapies [11,21,22]. Importantly, among patients classified as having undiagnosed CKD, renal function and/or albuminuria measurements were available in routine clinical care, but CKD had not been formally recognised or coded in the medical record, suggesting that underdiagnosis primarily reflects lack of diagnostic recognition rather than absence of testing.

Our analysis of KDIGO risk categories showed that undiagnosed CKD cases were predominantly classified within low-risk strata, whereas patients with documented CKD were more often moderate or high risk. This indicates that underdiagnosis occurs mainly at earlier stages, when abnormalities in eGFR or albuminuria may go unnoticed. Importantly, albuminuria has independent prognostic value even in patients with preserved eGFR. Not to recognise early CKD, particularly when driven by isolated albuminuria, may delay risk stratification and the timely initiation of cardio-renal protective interventions, potentially affecting long-term renal and cardiovascular outcomes. Identifying these patients early is essential, as interventions aimed at slowing CKD progression are most effective in these stages [23–25]. From a system-level perspective, underrecognition of early CKD in primary care is less likely to reflect isolated clinician oversight and more commonly arises from organisational and workflow constraints. These include limited consultation time, fragmented access to laboratory data, insufficient integration of uACR results into electronic health records, weak clinical decision-support tools, and the absence of standardised CKD screening and referral pathways. In addition, uncertainty around guideline implementation and competing clinical priorities may further hinder timely CKD recognition. Addressing these challenges will likely require system-level strategies, such as redesigned EHR prompts and registries, clearer CKD care pathways, protected time and quality metrics for kidney health, and targeted training and communication models embedded within routine primary care practice [26,27].

The use of antidiabetic agents with proven cardio-renal-metabolic benefits was suboptimal across all groups. The use of SGLT2 inhibitors was more common among patients with diagnosed CKD than among those without a recorded diagnosis, consistent with guideline recommendations [11,19–23]. Similarly, GLP-1 receptor agonists were prescribed more frequently in patients with CKD-D, in line with current evidence and recent clinical trials—such as FLOW—demonstrating their renal and cardiovascular benefits [25,28–31]. These findings highlight a potential therapeutic gap in patients with undiagnosed CKD, who may not benefit from guideline-recommended treatments due to the absence of formal CKD recognition in their medical records. This underuse may also reflect organisational and workflow-related barriers in primary care, including competing clinical priorities, limited consultation time, fragmented access to laboratory results, and lack of standardised CKD screening pathways. The use of SGLT2 inhibitors and GLP-1 receptor agonists remains suboptimal worldwide despite robust evidence supporting their cardiovascular and renal benefits. Previous real-world studies have reported that this underuse is influenced by multiple factors, including limited prescriber familiarity or experience with newer therapies, concerns regarding safety or contraindications, patient-related factors such as comorbidity burden or perceived adherence, and broader health system–level constraints [32]. Overcoming these barriers is likely to require multifaceted strategies, including targeted education for prescribers, simplification of coverage and reimbursement pathways, and interventions aimed at supporting medication adherence and equitable access, particularly in vulnerable populations [33,34]. After multivariable adjustment, several associations observed in crude analyses were attenuated, indicating that differences in the use of some drug classes (e.g. DPP-4 inhibitors) were largely explained by patient characteristics rather than CKD status itself. In contrast, use of SGLT2 inhibitors remained independently higher among both patients with diagnosed and undiagnosed CKD compared with those without CKD.

Conversely, metformin use was appropriately lower in patients with diagnosed CKD, consistent with guideline recommendations to limit its use in moderate-to-severe renal impairment [30]. The greater use of insulin among CKD-D patients likely reflects the need for tighter glycaemic control in the context of declining renal function [35]. In contrast, no differences were observed in sulfonylurea use between groups, although these agents should be discontinued when eGFR falls below 50 mL/min/1.73 m^2. The low but higher use of glinides among CKD patients is consistent with their hepatic metabolism, which made repaglinide a preferred option before the introduction of SGLT2 inhibitors and GLP-1 receptor agonists.

The multivariable analysis identified several factors associated with undiagnosed CKD, including a higher likelihood of metformin prescription, shorter duration of diabetes, lower prevalence of heart failure and proliferative retinopathy, and higher eGFR values. These results suggest that undiagnosed CKD is more frequent among patients with fewer diabetes-related complications and preserved renal function, reflecting milder disease stages. This analysis seeks to provide evidence that may support improvements in CKD detection and management in people with T2DM, particularly in the context of primary care.

This study has several limitations. CKD diagnosis relied on documentation in electronic medical records, and some cases of clinically recognised CKD may not have been recorded in structured diagnostic fields. CKD classification relied on single-point eGFR and/or uACR measurements obtained from routine clinical practice, without confirmation of persistence over at least three months, which may have led to some misclassification due to transient abnormalities and potential overestimation of CKD prevalence. In addition, the inclusion of patients attending primary care visits during the study period may have introduced selection bias. Its cross-sectional design precludes causal inference and does not allow evaluation of CKD progression or long-term

outcomes. Accordingly, longitudinal studies are needed to assess the impact of early CKD detection on renal and cardiovascular prognosis. Although serum creatinine and urine albumin testing are universally available in Spanish primary care, variability in test ordering practices across centres and clinicians cannot be excluded and could have influenced CKD detection. Nevertheless, incomplete adherence to albuminuria screening recommendations may have led to additional undetected CKD cases, particularly in patients with preserved eGFR. Finally, the multivariable model showed a moderate discriminatory capacity, and unmeasured factors associated with CKD may still exist. Residual confounding cannot be excluded, as detailed data on nephrology follow-up, adherence to albuminuria screening recommendations, and certain socioeconomic or educational factors were not systematically available and could influence both CKD diagnosis and management. The possible autocorrelation of some variables due to clustering at the level of primary care centres or individual physicians was not accounted for in the analyses, which may have influenced variance estimates, although we believe that its impact on the main findings is likely to be limited.

The reliance on data extracted from electronic medical records implies potential variability in data completeness and accuracy, particularly regarding coding practices and laboratory data recording. Approximately 6 % of patients had missing eGFR and/or uACR data, which may have led to some underestimation of CKD prevalence or misclassification of risk categories; however, given the limited magnitude of missing data and the absence of imputation procedures, the overall impact on prevalence estimates is likely small. In addition, our analysis reflects prescribing behaviour rather than actual medication exposure, as data on treatment adherence and the clinical or patient-related reasons for non-prescription are not systematically captured in electronic medical records. Finally, the influence of socioeconomic and sociocultural factors on CKD diagnosis and treatment could not be explored in depth and may partly explain the observed underdiagnosis, particularly in the early stages of disease. Future studies should specifically address these determinants to better understand their impact on early CKD recognition and management.

Despite these limitations, our findings have strong practical implications. Beyond decision-support tools, strategies to improve early CKD detection in primary care may include the standardisation of annual CKD screening workflows, the integration of automatic eGFR and uACR reflex testing within electronic health records, and targeted educational interventions for primary care clinicians to enhance awareness, registration, and prognostic understanding of early CKD. They underscore the need for systematic CKD screening in patients with T2DM in primary care, including annual assessment of both eGFR and albuminuria. Implementing automated alerts or decision-support tools within electronic health records could facilitate earlier recognition and coding of CKD, enabling timely initiation of cardio-renal-protective therapies. Future studies should evaluate the effectiveness of such interventions and explore clinician and system-level barriers to improving CKD detection, risk stratification, and optimal therapeutic management.

5. Conclusions

This study demonstrates that more than half of CKD cases in adults with T2DM remain undiagnosed in Spanish primary care. Underdiagnosis primarily affects patients in early stages and low KDIGO risk categories, which may limit appropriate risk stratification and the use of guideline-recommended cardio-renal protective therapies, such as SGLT2 inhibitors and GLP-1 receptor agonists. Enhancing systematic testing of eGFR and albuminuria, together with improved diagnostic coding and adherence to clinical practice guidelines, represents a practical opportunity to strengthen early CKD recognition and management in routine primary care practice. Given the cross-sectional design and reliance on routinely collected electronic health record data, these findings should be interpreted as associative and within the context of real-world clinical practice. Overall, these results highlight the importance of prioritising early detection strategies and integrated care models to reduce the burden of diabetic kidney disease and its cardiovascular consequences in real-world primary care settings.

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Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors used ChatGPT (OpenAI, San Francisco, CA, USA) to support language editing, translation from Spanish to English, and style refinement to meet scientific writing standards. After using this tool, the authors reviewed, verified, and edited the content as needed and take full responsibility for the accuracy and integrity of the final manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.pcd.2026.02.007.

References

[1] E.T. Fenta, H.B. Eshetu, N. Kebede, et al., Prevalence and predictors of chronic kidney disease among type 2 diabetic patients worldwide: systematic review and meta-analysis, Diabetol. Metab. Syndr. 15 (1) (2023) 245, https://doi.org/10.1186/s13098-023-01202-x.

[2] F. Soriguer, A. Goday, A. Bosch-Comas, et al., Prevalence of diabetes mellitus and impaired glucose regulation in Spain: the Di@bet.es Study, Diabetologia 55 (1) (2012) 88–93, https://doi.org/10.1007/s00125-011-2336-9.

[3] M. Kumar, S. Dev, M.U. Khalid, et al., The bidirectional link between diabetes and kidney disease: mechanisms and management, Cureus 15 (9) (2023) e45615, https://doi.org/10.7759/cureus.45615.

[4] R. García-Maset, J. Bover, J. Segura de la Morena, et al., Documento de información y consenso para la detección y manejo de la enfermedad renal crónica, Nefrologia 42 (3) (2023) 233–264, https://doi.org/10.1016/j.nefro.2021.07.010.

[5] F. Bassami, M. Yavari, A. Feizi, M. Siavash, M. Akbari, M. Karimifar, Association between high-sensitivity C-reactive protein and diabetic nephropathy: a systematic review and meta-analysis, BMC Nephrol. 26 (2025) 418, https://doi.org/10.1186/s12882-025-04358-y.

[6] S.K. Shaw, S. Sengupta, R. Jha, C. Pattanaik, H. Behera, P.K. Barik, D. Meher, R. Sarangi, S. Devadas, Meta-inflammation in type 2 diabetes mellitus: unveiling the role of aberrant CD4+ T cells and pro-inflammatory cytokine networks, Front Immunol. 16 (2025) 1603484, https://doi.org/10.3389/fimmu.2025.1603484.

[7] American diabetes association professional practice committee. Chronic kidney disease and risk management: standards of care in diabetes—2025, Diabetes Care 48 (1) (2025) S239–S251, https://doi.org/10.2337/dc25-S011.

[8] K.J. Jager, C. Kovesdy, R. Langham, et al., A single number for advocacy and communication: worldwide more than 850 million individuals have kidney diseases, Kidney Int 96 (5) (2019 Nov) 1048–1050, https://doi.org/10.1016/j.kint.2019.07.012.

[9] A. Ortiz, (A.I.R.G.-E.) Asociación Información Enfermedades Renales Genéticas, (E. K.P.F.) European Kidney Patients' Federation, et al., RICORS2040: the need for

collaborative research in chronic kidney disease, Clin. Kidney J. 15 (3) (2021) 372–387, https://doi.org/10.1093/ckj/sfab170.
[10] J.F. Navarro González, A. Ortiz, A. Cebrián Cuenca, et al., Projection of the clinical and economic burden of chronic kidney disease between 2022 and 2027 in Spain: results of the Inside CKD project, Nefrologia 44 (6) (2024) 807–817, https://doi.org/10.1016/j.nefroe.2024.11.009.
[11] C.J. Diamantidis, S.L. Hale, V. Wang, et al., Lab-based and diagnosis-based chronic kidney disease recognition and staging concordance, BMC Nephrol. 20 (1) (2019) 357, https://doi.org/10.1186/s12882-019-1551-3.
[12] N. Tangri, E.J. Peach, S. Franzén, et al., Patient management and clinical outcomes associated with a recorded diagnosis of stage 3 chronic kidney disease: the REVEAL-CKD study, Adv. Ther. 40 (6) (2023) 2869–2885, https://doi.org/10.1007/s12325-023-02482-5.
[13] D. Sánchez-Ospina, S. Mas-Fontao, M.M. Palencia, et al., Impact of albuminuria screening in primary care on the detection and management of chronic kidney disease: findings from the ONDAAS study, Clin. Kidney J. 18 (5) (2025) sfaf123, https://doi.org/10.1093/ckj/sfaf123.
[14] D. Orozco-Beltran, M. Mata-Cases, S. Artola-Menéndez, et al., Glycemic and weight control in people with type 2 diabetes: a real-world observational study in primary care, Prim. Care Diabetes 19 (1) (2025) 7–14, https://doi.org/10.1016/j.pcd.2024.12.002.
[15] Kidney Disease: Improving Global Outcomes (KDIGO) CKD work group. KDIGO 2024 clinical practice guideline for the evaluation and management of chronic kidney disease, Kidney Int 105 (4) (2024) S117–S314, https://doi.org/10.1016/j.kint.2023.10.018.
[16] Harrell F.E.Jr Regression Modeling Strategies. Cham, Switzerland: Springer International Publishing; 2016.
[17] M. Mata-Cases, B. Vlacho, J. Real, et al., Trends in the degree of control and treatment of cardiovascular risk factors in people with type 2 diabetes in a primary care setting in Catalonia during 2007–2018, Front Endocrinol. (Lausanne) 12 (2022) 810757, https://doi.org/10.3389/fendo.2021.810757.
[18] M. Afkarian, L.R. Zelnick, Y.N. Hall, et al., Clinical manifestations of kidney disease among US adults with diabetes, 1988-2014, JAMA 316 (6) (2016 Aug 9) 602–610, https://doi.org/10.1001/jama.2016.10924.
[19] M.C. Thomas, M. Brownlee, K. Susztak, et al., Diabetic kidney disease, Nat. Rev. Dis. Prim. 1 (2015 Jul 30) 15018, https://doi.org/10.1038/nrdp.2015.18.
[20] N. Montero, L. Oliveras, A. Martínez-Castelao, et al., Clinical practice guideline for detection and management of diabetic kidney disease: a consensus report by the Spanish society of nephrology, Nefrologia 45 (1) (2025) 1–26, https://doi.org/10.1016/j.nefroe.2025.04.005.
[21] B. Neal, V. Perkovic, K.W. Mahaffey, et al., Canagliflozin and cardiovascular and renal events in type 2 diabetes, N. Engl. J. Med 377 (7) (2017) 644–657, https://doi.org/10.1056/NEJMoa1611925.
[22] V. Perkovic, M.J. Jardine, B. Neal, et al., Canagliflozin and renal outcomes in type 2 diabetes and nephropathy, N. Engl. J. Med 380 (24) (2019) 2295–2306, https://doi.org/10.1056/NEJMoa1811744.
[23] C. Wanner, S.E. Inzucchi, J.M. Lachin, et al., Empagliflozin and progression of kidney disease in type 2 diabetes, N. Engl. J. Med 375 (4) (2016) 323–334, https://doi.org/10.1056/NEJMoa1515920.
[24] H.J.L. Heerspink, B.V. Stefánsson, R. Correa-Rotter, et al., Dapagliflozin in patients with chronic kidney disease, N. Engl. J. Med 383 (15) (2020 Oct 8) 1436–1446, https://doi.org/10.1056/NEJMoa2024816.
[25] M.J. Davies, V.R. Aroda, B.S. Collins, et al., Management of hyperglycemia in type 2 diabetes, 2022: a consensus report by the ADA and the, Easd. Diabetes Care. 45 (1) (2022) S125–S143, https://doi.org/10.2337/dci22-0034.
[26] E. Neale, J. Middleton, K. Lambert, Barriers and enablers to detection and management of chronic kidney disease in primary healthcare: a systematic review, BMC Nephrol. 21 (2020) 83, https://doi.org/10.1186/s12882-020-01731-x.
[27] P. Kushner, K. Khunti, A. Cebrián, G. Deed, Early identification and management of chronic kidney disease: a narrative review of the crucial role of primary care practitioners, Adv. Ther. 41 (2024) 3757–3770, https://doi.org/10.1007/s12325-024-02957-z.
[28] H.C. Gerstein, H.M. Colhoun, G.R. Dagenais, et al., Dulaglutide and renal outcomes in type 2 diabetes: an exploratory analysis of the REWIND randomised, placebo-controlled trial, Lancet 394 (10206) (2019) 121–130, https://doi.org/10.1016/S0140-6736(19)31150-X.
[29] V. Perkovic, K.R. Tuttle, P. Rossing, et al., FLOW trial committees and investigators. Effects of semaglutide on chronic kidney disease in patients with type 2 diabetes, N. Engl. J. Med 391 (2) (2024) 109–121, https://doi.org/10.1056/NEJMoa2403347.
[30] Y. Zheng, J. Sun, Long-term effect of sodium-glucose cotransporter 2 inhibitors on kidney function: a systematic review and meta-analysis, Med. (Baltim.) 104 (7) (2025) e41422, https://doi.org/10.1097/MD.0000000000041422.
[31] J.F.E. Mann, D.D. Ørsted, K. Brown-Frandsen, et al., LEADER steering committee and investigators. Liraglutide and renal outcomes in type 2 diabetes, N. Engl. J. Med 377 (9) (2017) 839–848, https://doi.org/10.1056/NEJMoa1616011.
[32] T. Kurevija, D. Šojat, I. Bilić-Ćurčić, S. Canecki-Varžić, L. Trtica-Majnaric, Barriers in prescribing antidiabetic medications with cardiovascular benefits: practice, experience, and attitudes of GPs in Croatia, BMC Prim. Care 26 (2025) 88, https://doi.org/10.1186/s12875-025-02837-7.
[33] I. Alhomoud, S. Aldakhil, R. Alhosan, H. Alrashidi, N. Alsaqabi, A. Aldugishem, A. Alsuhibani, A. Alrasheedy, Knowledge, counseling practice, perceived barriers, and clinical decision-making of community pharmacists in Saudi Arabia regarding glucagon-like peptide-1 receptor agonists and sodium-glucose cotransporter 2 inhibitors, Diabetes Metab. Syndr. Obes. 18 (2025) 3093–3107, https://doi.org/10.2147/DMSO.S552287.
[34] J. Moore, N. Iheme, N. Rebold, H. Kusi, C. Mere, U. Nwaogwugwu, E. Ettienne, W. Chaijamorn, D. Rungkitwattanakul, Factors and disparities influencing sodium-glucose cotransporter 2 inhibitors and glucagon-like peptide 1 receptor agonists initiation in the United States: a scoping review of evidence, Pharm. (Basel) 13 (2025) 46, https://doi.org/10.3390/pharmacy13020046.
[35] C.S. Fox, K. Matsushita, M. Woodward, et al., Chronic kidney disease prognosis consortium. Associations of kidney disease measures with mortality and end-stage renal disease in individuals with and without diabetes: a meta-analysis, Lancet 380 (9854) (2012) 1662–1673, https://doi.org/10.1016/S0140-6736(12)61350-6.