Obesity and Chronic Kidney Disease in Diabetic and/or Hypertensive Patients in the Laghouat District, Algeria: A Cross-Sectional Study

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Abstract

Background: Obesity is a recognised contributor to chronic kidney disease (CKD) through glomerular hyperfiltration, obesity-related glomerulopathy and adipokine-mediated inflammation, but its relationship with CKD in diabetic and hypertensive patients from North Africa is poorly documented.

Objective: To describe overweight and obesity and their association with CKD (KDIGO 2012 definition) in diabetic and/or hypertensive patients of the Laghouat district, as a focused secondary analysis of the cohort whose overall CKD prevalence we reported previously.

Methods: Cross-sectional prevalence study with prospective random recruitment (Diabetes Care Centre, Public Hospital Establishment of Laghouat, January 2018–January 2019); 302 of 420 screened patients aged 18–70 years were analysed. CKD: eGFR (MDRD) < 60 mL/min/1.73 m² and/or abnormal urinary albumin- or protein-to-creatinine ratio, confirmed at three months. BMI was classified into four categories as recorded in the source dataset. Means were compared with Student's t-test; BMI categories were compared by χ^2 and trend tests, with crude odds ratios computed from the cross-tabulated counts; independent predictors were sought by multivariable logistic regression. Reporting follows STROBE.

Results: Mean BMI was 28.52 kg/m²; 215/302 patients (71.2%) were overweight or obese, including 124 (41.1%) with obesity. Overweight or obesity affected 88/121 patients with CKD (72.7%) and 127/181 without CKD (70.2%). Mean BMI was 28.80 kg/m² (95% CI 27.85–29.75) with CKD and 28.33 kg/m² (27.63–29.03) without (difference 0.47 kg/m², 95% CI −0.68 to 1.62; p = 0.417). BMI category was not associated with CKD (χ^2 p = 0.97; trend p = 0.64); crude OR for obesity was 1.08 (95% CI 0.68–1.72). Fasting glucose, HbA1c and blood pressure were significantly higher in CKD. BMI was entered in the initial multivariable model but was not retained; the final model retained diabetic retinopathy (OR 13.05; 6.84–24.88), anaemia (2.23), positive urine dipstick (2.18) and antihypertensive treatment (1.52).

Conclusion: Overweight and obesity were very frequent irrespective of CKD status, and BMI was not associated with CKD in this high-risk sample; the confidence intervals exclude large crude associations (OR > ~1.7 for obesity) but not modest ones. This

cross-sectional analysis cannot establish or exclude causal or mediating pathways; we propose, as hypotheses for longitudinal studies, that adiposity acts through glycaemic and haemodynamic correlates and that measures of visceral adiposity would be more informative than BMI.

Keywords: obesity; body mass index; chronic kidney disease; diabetes mellitus; hypertension; Algeria; cross-sectional study.

1. Introduction

Obesity is one of the defining non-communicable epidemics of this century, affecting an estimated 604 million adults worldwide [1]. Chronic kidney disease (CKD) affects 10–14% of adults in Western populations and is associated with excess cardiovascular mortality even at early stages [2,3]. The 2012 Kidney Disease: Improving Global Outcomes (KDIGO) classification, which stratifies CKD by estimated glomerular filtration rate (eGFR) and albuminuria category [4], has been retained in the 2024 update [5].

Beyond its role in type 2 diabetes and hypertension, obesity is increasingly regarded as an independent contributor to CKD. Excess adiposity induces glomerular hyperfiltration that may progress to obesity-related glomerulopathy [6], and adipose tissue secretes adipokines that promote inflammation and fibrosis [7]. Epidemiological evidence includes a systematic review and meta-analysis linking obesity to CKD [8], a graded association between body mass index (BMI) and end-stage renal disease in a large US cohort [9], and associations of obesity with prevalent and incident CKD in prospective cohorts [10,11]. The Canadian Society of Nephrology advises aiming for a healthy weight (BMI 18.5–24.9 kg/m²) to reduce the risk of end-stage kidney disease [12].

Algeria, notably its southern High Plateaus, is undergoing a rapid epidemiological transition in which non-communicable diseases predominate. In the same cohort as the present report, we previously documented an overall CKD prevalence of 40% among diabetic and/or hypertensive patients [13]. Obesity is a frequent comorbidity in this population, but its relationship with CKD has not been characterised locally.

1.1 Relationship to prior publication

This manuscript is a focused secondary analysis of the same cohort (n = 302) reported in our earlier publication on the prevalence and predictors of CKD [13]. That publication reported overall CKD prevalence (40%), the KDIGO stage distribution and the full multivariable model (diabetic retinopathy, anaemia, urine dipstick positivity, antihypertensive treatment); these results are cited but not re-presented here, except Table 3, which reproduces the final model solely to show how BMI behaved relative to the retained variables. New to the present manuscript are: the distribution of BMI categories by CKD status (Table 1); BMI and metabolic parameters with 95% confidence intervals (Table 2); BMI-specific tests of association and crude odds ratios (Table 4); and the discussion of the obesity–CKD relationship.

The specific aim was to describe overweight and obesity in this population and to evaluate their association with CKD relative to the other metabolic determinants measured in the same cohort.

2. Materials and Methods

2.1 Study design and setting

This is a secondary analysis of a descriptive, cross-sectional prevalence study with prospective random recruitment, conducted from January 2018 to January 2019 in a primary-care clinic of the Diabetes Care Centre, Public Hospital Establishment of Laghouat, Algeria. The parent study is reported separately [13]. Reporting follows the STROBE statement [14].

2.2 Participants

Of 420 diabetic and/or hypertensive patients randomly screened, 302 aged 18–70 years, resident in the district and giving informed consent were analysed (249 type 2 diabetes, 44 hypertension, 9 type 1 diabetes). Exclusion criteria were acute kidney injury, dialysis or transplantation, pregnancy and minor age.

2.3 Anthropometry

Weight and height were measured and BMI was calculated as weight (kg)/height² (m²). Four categories were used exactly as recorded in the source dataset: < 18, 18–25, 26–30 and > 30 kg/m². These cut-offs approximate but do not reproduce the standard World Health Organization categories (< 18.5, 18.5–24.9, 25.0–29.9, ≥ 30.0 kg/m²) [15]; as recorded, they leave values between 25 and 26 kg/m² unassigned unless BMI was recorded as whole numbers [author to confirm]. We did not reclassify patients post hoc. Waist circumference and body composition were not measured.

2.4 Definition of CKD

CKD followed the KDIGO 2012 criteria [4]: abnormalities of kidney structure or function persisting > 3 months, including eGFR < 60 mL/min/1.73 m² or abnormal albuminuria/proteinuria. eGFR was calculated with the four-variable MDRD equation, the formula recommended nationally at the time; CKD-EPI is now considered more accurate near normal eGFR [16]. A positive urine dipstick prompted quantification of the protein-to-creatinine ratio; a negative dipstick prompted immunoturbidimetric albumin-to-creatinine ratio (ACR). Patients with eGFR < 60 mL/min or ACR > 30 mg/g were retested at three months.

2.5 Statistical analysis

Analyses used SPSS version 19. Continuous variables are presented as means with 95% confidence intervals and compared with Student's t-test; categorical variables as counts and percentages. For this manuscript, BMI category was compared between groups with the χ^2 test and a Cochran–Armitage trend test, and crude odds ratios with Wald 95% confidence intervals were computed from the counts in Table 1 (post hoc; unadjusted). Independent predictors of CKD were sought by multivariable logistic regression. The initial model included sex, age, BMI, diabetes treatment, antihypertensive treatment, lipid-lowering treatment, nephroprotective treatment, urine dipstick result, duration of diabetes or hypertension, smoking, HbA1c, dyslipidaemia, haemoglobin, diabetic retinopathy, hypertensive retinopathy, renal ultrasound findings and left ventricular hypertrophy; the final model retained four variables (Table 3). [Variable coding, variable-selection method and handling of missing data to be confirmed by the author from the SPSS output.] An odds ratio was considered significant when its 95% confidence interval excluded 1; two-sided $p < 0.05$ defined statistical significance.

2.6 Ethics

Written informed consent was obtained from each participant, in accordance with the Declaration of Helsinki. As an observational, non-interventional study, trial registration was not required.

3. Results

3.1 Body mass index in the cohort

Mean BMI of the 302 patients was 28.52 kg/m² (range 15–42). Overweight or obesity (categories 26–30 and > 30 kg/m²) was present in 215 patients (71.2%): 124 (41.1%) obese and 91 (30.1%) overweight; 3 patients (1.0%) were underweight.

Table 1. Distribution of BMI categories by CKD status (row percentages).

BMI category (as recorded)	No CKD n = 181 (%)	CKD n = 121 (%)	Total n = 302 (%)
Underweight (< 18 kg/m²)	2 (66.7)	1 (33.3)	3 (100)
Normal weight (18–25 kg/m²)	52 (61.9)	32 (38.1)	84 (100)
Overweight (26–30 kg/m²)	54 (59.3)	37 (40.7)	91 (100)
Obesity (> 30 kg/m²)	73 (58.9)	51 (41.1)	124 (100)
Overweight or obesity (≥ 26 kg/m²)	127 (59.1)	88 (40.9)	215 (100)
Total population	181 (59.9)	121 (40.1)	302 (100)

3.2 BMI and CKD status

Overweight or obesity affected 88 of 121 patients with CKD (72.7%; 51 obese [42.1%] and 37 overweight [30.6%]) and 127 of 181 patients without CKD (70.2%; 73 obese and 54 overweight) (Table 1). Mean BMI was 28.80 kg/m² (95% CI 27.85–29.75) with CKD and 28.33 kg/m² (27.63–29.03) without; the difference was 0.47 kg/m² (95% CI −0.68 to 1.62; Student's t-test, p = 0.417) (Table 2). Recalculation of the t-test from the published group means and confidence intervals gives p ≈ 0.42–0.43, consistent with the reported value.

BMI category was not associated with CKD (χ^2 = 0.26, 3 df, p = 0.97; χ^2 with underweight and normal weight pooled = 0.24, 2 df, p = 0.89; Cochran–Armitage trend z = 0.47, p = 0.64). Crude odds ratios were 1.08 (95% CI 0.68–1.72) for obesity versus non-obesity and 1.13 (0.68–1.89) for overweight or obesity versus underweight/normal weight (Table 4). In contrast, fasting glucose, HbA1c and systolic and diastolic blood pressure were significantly higher in patients with CKD (Table 2).

Table 2. Age, BMI and metabolic parameters by CKD status (Student's t-test).

Parameter	CKD (n = 121) mean (95% CI)	No CKD (n = 181) mean (95% CI)	p
Age (years)	58.53 (56.81–60.25)	56.50 (55.08–57.92)	0.07
BMI (kg/m²)	28.80 (27.85–29.75)	28.33 (27.63–29.03)	0.417
Fasting glucose (g/L)	1.85 (1.72–1.98)	1.57 (1.48–1.66)	0.001
HbA1c (%)	8.22 (7.85–8.58)	7.56 (7.27–7.85)	0.005
Systolic BP (mmHg)	147.14 (142.60–151.68)	134.17 (131.14–137.20)	0.001
Diastolic BP (mmHg)	85.25 (82.54–87.96)	79.39 (77.50–81.28)	0.001

p values are reproduced as printed in the source SPSS output (0.001 denotes p ≤ 0.001).

Table 4. Crude (unadjusted) odds ratios for CKD by BMI category, computed from the counts in Table 1.

Comparison (unadjusted)	Crude OR	95% CI	p
Obesity (> 30) vs non-obesity	1.08	0.68–1.72	0.75
Overweight or obesity (≥ 26) vs underweight/normal	1.13	0.68–1.89	0.63
Overweight (26–30) vs underweight/normal	1.12	0.61–2.05	—
Obesity (> 30) vs underweight/normal	1.14	0.65–2.01	—

Computed post hoc from cross-tabulated counts (Wald confidence intervals); p values from χ² tests; unadjusted for age, diabetes, blood pressure or other covariates.

3.3 BMI in the multivariable model

BMI was included among the candidate variables of the initial multivariable model but was not retained in the final model, which retained four variables: diabetic retinopathy, anaemia, a positive urine dipstick and antihypertensive treatment (Table 3). The adjusted estimate for BMI at entry into the initial model is not reported in the source tables [to be inserted from the SPSS output]; until it is available, the unadjusted estimates in Table 4 are the only quantitative estimates of the BMI–CKD association we can report.

Table 3. Final multivariable model for CKD, and position of BMI.

Variable	Adjusted OR	95% CI	p
Antihypertensive treatment	1.52	1.17–1.98	0.002
Positive urine dipstick	2.18	1.20–3.97	0.010
Anaemia (haemoglobin)	2.23	1.19–4.19	0.013
Diabetic retinopathy	13.05	6.84–24.88	<0.001
BMI — entered in initial model, not retained‡	[from SPSS step 1]	[from SPSS step 1]	[from SPSS step 1]

‡ BMI was a candidate variable in the initial model and was not retained in the final model. The final-model estimates are those reported in the parent publication [13].

4. Discussion

Overweight and obesity were very frequent in this high-risk population (71.2% and 41.1%), and similarly frequent whether or not CKD was present (72.7% versus 70.2%). These figures exceed those reported in an Algerian study of hypertension and diabetic nephropathy, in which overweight was found in 27.7% of one sex and 23.3% of the other and obesity in 14.7% and 9.7% [17], and are consistent with the burden of obesity, diabetes and hypertension described in Moroccan adults [18]. Differences in recruitment (a diabetes referral centre, enriched for metabolic risk) and in BMI categorisation probably contribute.

Unlike several studies identifying obesity as an independent risk factor for CKD and proteinuria [8,19], we found no association between BMI and CKD: the means were similar, BMI category was unrelated to CKD, and BMI was not retained in the multivariable model. Interpretation requires attention to precision. The 95% confidence interval for the crude odds ratio of obesity (0.68–1.72) excludes large associations but not modest ones, and the confidence interval for the mean BMI difference (−0.68 to 1.62 kg/m²) is compatible with small differences in either direction; our data therefore show no evidence of an association, not evidence of its absence.

Several non-exclusive explanations are offered as hypotheses, none testable with this cross-sectional design. First, the cohort was selected on diabetes and/or hypertension, in which excess weight is nearly universal; this restricted range of exposure limits the ability to detect an additional association. Second, BMI does not distinguish visceral from subcutaneous fat, the former being more closely linked to renal lipotoxicity and hyperfiltration in experimental and clinical physiology [6,20]; waist circumference and body composition were not measured. Third, we hypothesise that part of the renal impact of adiposity may be conveyed by glycaemic and haemodynamic correlates: glycaemia, HbA1c and blood pressure were significantly higher in CKD in our data and are plausible intermediates. However, no formal mediation analysis was performed, no collinearity diagnostics (such as variance inflation factors) were examined, and these variables may equally be correlated with, rather than downstream of, adiposity. Testing these hypotheses requires longitudinal data with formal mediation analysis.

These findings do not argue against weight management in diabetic and hypertensive patients. Severe obesity is associated with glomerular hyperfiltration and increased renal plasma flow [20], and weight loss reduces proteinuria and systolic blood pressure in obesity-related kidney damage [21].

Limitations. The design precludes causal inference and the observation of weight trajectories. Single-centre recruitment in a diabetes referral facility may have introduced selection bias. Adiposity was assessed by BMI only. BMI categories, as recorded, deviate from WHO thresholds, which limits comparability with studies using strict categories. The adjusted estimate for BMI in the initial multivariable model is not available in the source tables, and the crude odds ratios in Table 4 are unadjusted and were computed post hoc. The sample size limits precision, as reflected in the confidence intervals above. Finally, the MDRD equation rather than CKD-EPI may have misclassified eGFR near the 60 mL/min/1.73 m² threshold [16].

Strengths include the community-based, prospective and random recruitment, standardised BMI measurement in all participants, and the simultaneous assessment of adiposity alongside the other metabolic determinants of CKD in the same cohort. To our knowledge, few studies have examined obesity and CKD in a Saharan Algerian primary-care population.

5. Conclusion

In diabetic and/or hypertensive patients of the Laghouat district, overweight and obesity were highly prevalent, including among patients with CKD, but BMI was not associated with CKD status: means, category distribution and crude odds ratios were similar, and BMI was not retained in the multivariable model. The data exclude large crude associations but cannot exclude modest ones, and the cross-sectional design cannot establish or rule out causal or mediating mechanisms. Longitudinal studies with waist circumference or body-composition measures and formal mediation analysis are needed. These results do not challenge recommendations to pursue a healthy weight in diabetic and hypertensive patients as part of nephroprotection.

Declarations

Funding

No specific funding was received for this work.

Conflicts of interest

The authors declare no conflicts of interest.

Ethics approval and consent

Written informed consent was obtained from each participant in accordance with the Declaration of Helsinki. No trial registration was required for this observational study.

Relationship to prior publication

This is a secondary analysis of the cohort reported in [13]; see Section 1.1.

Data availability

The datasets analysed are available from the corresponding author on reasonable request.

Author contributions

A.D.: conception, data collection and analysis, drafting. F.H.: scientific supervision, critical revision. Both authors approved the final manuscript.

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