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Obesity and chronic kidney disease: What specificity of this association and what epidemiological profile? A prospective study

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Abstract

Obesity is accompanied by chronic diseases including cardiometabolic diseases. Obesity is also an independent factor of renal risk that deserves to be taken into account. The aims of our work were to determine the prevalence of chronic kidney disease related to obesity independently of all other usual risk factors and to describe its epidemiological characteristics in our population.

We conducted a prospective descriptive and analytic study over a period of 12 months in 38 patients over 18 years of age, with a BMI> 30 Kg / m^2 and without diabetes, hypertension, or other apparent cause chronic kidney disease, collected at the consulting service of the Military Hospital Avicenna of Marrakech, Morocco.

The average age of our patients was 38 years with a female predominance (sex ratio (m/w) of 0.80). The average BMI was 35.35 kg / m^2. Dyslipidemia was found in 68.42% of patients, hyperuricemia in 18.42%, microalbuminuria in 13.15%, macroalbuminuria in 7.89% and hematuria in 2.6%. Glomerular hyperfiltration was present in 13.15% of cases and no patient had a GFR <60 ml / min / 1.73m^2. The prevalence of chronic kidney disease among obese people in our population was 10.5%. Older obese patients with high BMI, hypertriglyceridemia, hypercholesterolemia, and hyper LDLemia were the most affected, with statistically significant differences between the 2 groups of patients with and without chronic kidney disease (P <0.05).

Our study warn of the need for cost-effective and well-adapted prevention measures to reverse the growing epidemic of obesity in the world.

Keywords: Obesity; Chronic kidney disease; Epidemiology; Risk factors

1. Introduction

The ever-increasing prevalence of obesity, which initially affected developed countries, is now affecting a number of emerging countries and is currently considered a global public health problem [1]. The first non-infectious epidemic of modern times, obesity is the world's most important source of chronic diseases [2], such as hypertension, type 2 diabetes, respiratory diseases, joint diseases, as well as nephrological pathologies and other complications. Indeed, the renal complications of obesity have been the subject of numerous studies in recent years [3], several of which have established the link between obesity and the onset or worsening of chronic kidney disease independently of all other usual risk factors (type 2 diabetes, hypertension), with a risk in obese patients compared with the rest of the population [4,5].

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The aims of this study are:

- Main objective: To specify the prevalence of chronic kidney disease linked to obesity (independently of other usual risk factors).
- Secondary objective: To describe the epidemiological characteristics of chronic kidney disease in this population.

2. Methods

Our work reports the results of a prospective descriptive and analytical study carried out over a period of 12 months (from month 10/2017 to month 10/2018) concerning 34 patients collected at the Diabetology and Metabolic Diseases Endocrinology Department of the Avicenna Military Hospital in Marrakech.

The study population concerned subjects of 2 sexes meeting the following inclusion criteria: Age >18 years, BMI >30 kg/m^2. Exclusion criteria were: DT1 or 2, hypertension, urinary tract infection, lithiasis, history of nephropathy and history of systemic disease.

Patients included in our study underwent a detailed interview and clinical examination, including urinary dipstick. Selected patients underwent a detailed biological work-up (fasting glycemia, HbA1c, CBC, renal work-up, liver work-up, lipid work-up, etc.) and a radiological work-up including renal and abdominal ultrasound.

Data were entered into Microsoft Office Excel 2007. Statistical analysis was performed using SPSS software version 19.0. Descriptive analysis consisted in calculating absolute and relative frequencies for qualitative variables, and position and dispersion parameters for quantitative variables (mean, standard deviation). The normal distribution of the variables was studied using the Kolmogorov-Smirnov test. In bivariate analysis, qualitative variables were compared using Pearson's Chi2 statistical test, and Fisher's if necessary. Quantitative variables were compared using the Student's t test or the Mann Whitney test. The significance threshold was set at $p<0.05$.

3. Results

The mean age of patients in our study was 38±11.80 years, with extremes ranging from 20 to 64 years. Our population comprised 21 women and 17 men, giving a sex ratio (M/F) of 0.80. Eleven patients, i.e. 28.9% of cases, were inactive, and the majority of patients had an average socioeconomic level (76.3% of cases). A family history was found in 26 patients, or 68.4% of cases, including the notion of familial obesity in 39.5% of cases, familial diabetes in 34.2% of cases, and familial hypertension in 13.2% of cases. In our series, 39.5% of cases had been obese since childhood, while only 10.5% of cases were recently obese.

The mean BMI of patients in our series was 35.35±2.84 kg/m^2, with extremes ranging from 30.13 to 40.83 kg/m^2 and a median of 35.58 kg/m^2.

Analysis of the chart representing the numbers of patients by BMI stage shows a predominance of obesity stage II: 52.6% of cases, the mean waist circumference of patients in our series was 98.61±11.07 cm with extremes ranging from 70 to 125 cm and a median of 98 cm.

88.2% of men and 85.7% of women were android obese. In our population, only 12 patients, i.e. 31.6% of cases, were on a strict diet, and only 5.26% of cases had sufficient regular physical activity. Thus, no patient in our series was on medical treatment, and no patient was scheduled for surgical treatment. One patient had a positive hematuria, ten patients had a PH<6 and two patients had traces of protein on urine dipstick (all patients had fresh urine samples examined with multi-parameter urine dipsticks).

Fasting blood glucose and HbA1c were normal in all our patients, dyslipidemia was found in 26 patients, or 68.42% of cases, with triglyceride levels higher in women than in men ($p<0.05$). Liver function tests were normal in all our patients. Hyperuricemia was found in 7 patients (6 men and 1 woman), i.e. 18.42% of cases, and CRP was unremarkable in all our patients. Blood urea and creatinine were normal in all our patients, glomerular filtration rate (GFR) was underestimated in 1 patient (GFR at 86 ml/min/1.73m2) and was elevated in 5 patients, or 13.15% of cases (GFR>130 ml/min/1.73m2). No patient in our series had a GFR<60 ml/min/1.73m2. Proteinemia and albuminemia were normal in all our patients, with albuminemia values higher in men than in women, $p<0.05$.

The albumin/creatinine ratio (ACR) was elevated (above 2mg/mmol) in 8 patients, or 21.05% of cases, of whom 5 patients, or 13.15% of cases, had microalbuminuria (2<ACR<30 mg/mmol) and 3 patients, or 7.89% of cases, had macroalbuminuria (ACR>30 mg/mmol). Microcytic hypochromic anemia was found in 1 woman, or 2.6% of cases.

One case of hematuria was found on ECBU, and the rest of the patients had a normal ECBU.

Renal ultrasound was normal in all patients. Hepatic steatosis was found on abdominal ultrasound in 17 patients, i.e. 44.73% of cases.

In our population, the number of obese patients with CKD, independently of all other usual risk factors, was 4:

- Three patients, or 7.89% of cases, with clinical albuminuria (RAC>30 mg/mmol) persisting for more than 3 months. Determinations were respectively at initial assessment: 35.7mg/mmol, 43mg/mmol and 31.2mg/mmol and at follow-up (after 3 months): 39.4mg/mmol, 64.5mg/mmol and 44.7mg/mmol.
- One patient, or 2.6% of cases with hematuria (RBC>10^4/ml) persistent for more than 3 months, in the absence of infection and after ruling out a urological cause.

The prevalence therefore of CKD in healthy obese patients in our population was 10.5%. All these patients were at stage 1 CKD (GFR ≥ 90 ml/min/1.73m2 + markers of persistent renal impairment for more than 3 months).

To determine the risk factors associated with CKD in our patient population (obese patients without diabetes, hypertension or other apparent cause of chronic kidney disease), we compared groups of obese patients with and without CKD in bivariate analysis (Tables 1 and 2).

Analysis of the data revealed a statistically significant difference between the 2 groups of patients (with and without CKD) with regard to: age (p=0.001), BMI (p=0.046), triglycerides (p=0.002), total cholesterol (p=0.008), LDL cholesterol (p=0.004), CRP (p=0.015), WBC (p=0.035). No other significant differences were found for the other factors tested, notably gender, chronic smoking, familial diabetes, familial hypertension, physical activity and waist circumference.

Table 1 Risk factors associated with chronic kidney disease in the obese population in our bivariate analysis (categorical variables)

Parameters		**Chronic kidney disease (CKD)**		**p**
		Yes	**No**	
Gender	M	0% (0 case)	100% (17 cases)	NS
	F	19% (4 cases)	81% (17 cases)	
Origin	Rural	0% (0 case)	100% (7 cases)	NS
	Urban	13% (4 cases)	87% (27 cases)	
Profession	Assets	15% (4 cases)	85% (23 cases)	NS
	Sedentary	0% (0 case)	100% (11 cases)	
Socio-economic level	Low	33.33% (2 cases)	66.66% (4 cases)	NS
	Medium or high	6.25% (2 cases)	93.75% (30 cases)	
Chronic smoking	Yes	0% (0 case)	100% (4 cases)	NS
	No	11.76% (4 cases)	88.24% (30 cases)	
Family diabetes	Yes	13.33% (2 cases)	86.66% (13cases)	NS
	No	8.69% (2 cases)	91.30% (21cases)	
Family Arterial hypertension	Yes	0% (0 case)	100% (8 cases)	NS
	No	13.33% (4 cases)	86.66% (26cases)	
Age of obesity	<10 years	16.66% (1case)	83.33% (5 cases)	NS

	>10 years	9.37% (3 cases)	90.62% (29 cases)	
Android Obesity android	Yes	22.22% (4 cases)	77.77% (14 cases)	NS
	No	0% (0 case)	100% (3 cases)	
Regular physical activity	Yes	0% (0 case)	100% (2cases)	NS
	No	11.11% (4 cases)	88.88% (32cases)	
Fatty liver	Yes	17.64% (3 cases)	82.35% (14 cases)	NS
	No	4.76% (1case)	95.23% (20 cases)	

(NS: not significant)

Table 2 Risk factors associated with CKD in our obese population in bivariate analysis (quantitative variables)

Parameters	**Chronic kidney disease (CKD)**		**p**
	Yes	**No**	
Age (yeas)	57.00±1.82	36.81±10.72	0.001
BMI (kg/m^2)	37.38±2.21	34.43±2.64	0.046
Waist circumference (cm)	98.25±4.71	93.50±11.82	NS
HbA1c test (%)	5.45±0.20	5.43±0.37	NS
Fasting blood glucose (mmol/l)	5.32±0.16	5.13±0.34	NS
Triglycerides (mmol/l)	2.05±0.28	1.43±0.35	0.002
total cholesterol (mmol/l)	5.57±0.58	4.56±0.66	0.008
LDL c (mmol/l)	4.12±0.36	3.17±0.58	0.004
HDLc (mmol/l)	1.27±0.02	1.28±0.10	NS
Alanine transaminase (U/l)	27.00±11.69	25.92±14.95	NS
Aspartate aminotransferase (U/l)	26.75±4.27	24.81±7.29	NS
Alkaline phosphatase (U/l)	75.00±9.09	72.51±16.11	NS
Gamma-glutamyl transferase(U/l)	33.50±10.37	34.00±13.88	NS
Uric acid (µmol/l)	273.75±27.98	27.76±43.94	NS
C-reactive protein (mg/l)	4.84±1.26	3.51±0.92	0.015
Glomerular filtration rate (ml /min/1.73m^2)	107.75±5.85	115.41±16.87	NS
Hemoglobin (g/dL)	13.92±0.38	14.67±1.78	NS
White cells (*10^3/µl)	7.28±0.29	6.8±1.42	0.035
Platelets (*10^3/µl)	297.25±35.95	227.88±50.1	0.013

(NS: not significant)

4. Discussion

The assessment of renal complications of obesity has been the subject of numerous studies in recent years, carried out by researchers from a variety of specialties in different parts of the world. However, most of these studies have focused on renal impairment in obese patients, irrespective of the underlying condition, notably diabetes and/or hypertension.

However, in our study, which is the first to be carried out at national level, we were interested in obesity-induced renal complications independently of all other usual risk factors, taking as our study base a non-hypertensive, non-diabetic obese population with no history of nephropathy or urological disease or other cause of chronic kidney disease. We

tried to compare the epidemiological, clinical and biological data of our patients with those of the closest studies to give more comparative value (Table 3). The number of patients included differs from one study to another. This can be explained by the time taken to recruit patients, which differs from one study to another (from a few months to several years), but also by the difference in prevalence of obesity from one country to another.

The mean age of patients in our series was 38 years, closer to that of the American study by Patel et al (37.2 years) [6] and the Spanish study by Serra et al (41.8 years) [7]. However, it is higher than that of the Iranian study by Minoo et al (30.4 years) [8] and lower than that of the Japanese study by Yano et al (59.4 years) [9]. With regard to gender, all studies noted a clear predominance of females, with a sex ratio (m/f) in the Japanese study by Yano et al [9] similar to that in our study (0.80). In our series, the majority of patients (84.2%) had been obese for more than 10 years, in line with the results of the Spanish study by Serra et al, where the mean duration of obesity was 236±105 months [7].

The mean BMI of our population was 35.35 kg/m2, similar to that of the Australian study by Stevens et al [10], and that of the Iranian study by Minoo et al [8], and lower than that of the American study by Patel et al [6], and that of the Spanish study by Serra et al [7]. This can be explained by the fact that these latter studies took as their populations; patients scheduled for bariatric surgery and therefore they were particularly patients with severe or morbid obesity. The mean waist circumference of patients in our series was 98.61cm, close to that of the Australian study by Stevens et al (105.9cm) [10]. However, the percentage of patients with android obesity was higher in our series than in the Australian study (86% vs. 69%). In our population, 31.6% of cases were on dietary restriction and only 5.26% of cases were sufficiently physically active, a percentage too low compared with that found in the Korean study by Chang et al [11]. This was to be expected, as the study of physical activity in Marrakech found a high level of sedentariness, especially among women (1 in 3), most of whom (85.4%) were housewives and therefore did little high-intensity activity [2].

No patient in our series was under medical treatment or scheduled for surgery. Dyslipidemia is a frequent metabolic complication of obesity [12]. In our series, dyslipidemia was found in 68.42% of cases, a figure similar to that found in the series by Serra et al (69.50%) [7], although it was predominantly higher than in the study by Pinto et al (32.63%) [13].

Hyper-uricemia is another metabolic complication of obesity, with a prevalence of 17.95%. In our study, we also found that 18.42% of cases had hyper-uricemia (6 men and 1 woman). With regard to renal function, all our patients had normal serum urea and creatinine levels, in line with the results of Spanish and Iranian studies [7,8]. However, 13.15% of our patients, compared with 32.63% of patients in the Spanish series [7], had an elevated GFR. Several studies have demonstrated the presence of glomerular hyper-filtration in obese patients [13,14,15,16,17], and its expression in absolute values (ml/min) in most subjects gives values 50% higher than those observed in normo-weight subjects. It should also be noted that none of the patients in our series had a GFR < 60 ml/min/1.73m2, as was the case in the Spanish series by Serra et al [7], while in another Dutch series, 3.64% of cases had a decreased GFR compared with 6.57% of cases with glomerular hyper-filtration [13]. Microalbuminuria in non-diabetic subjects is a marker of CV risk, diabetes risk, risk of impaired renal function and total mortality. It is also a risk marker for the development of hypertension in normotensive subjects [18]. In our series, micro-albuminuria was found in 13.15% of cases, a percentage close to that of the Iranian study by Minoo et al (11.8%) [8] and that of the American study by Patel et al (11%) [6]. However, it is higher than that of the French study by Cassuto et (7.1%) [19] and lower than that of the Spanish study by Serra et al (41%) [7]. Macro-albuminuria, or clinical proteinuria, is therefore no longer a risk marker but a true marker of renal damage, and its persistence for more than three months defines CKD. It was found in 7.89% of cases in our series, compared with 13.3% in the American series [6], 4% in the Spanish series [7] and 3% in the French series [18]. Numerous studies have assessed the suitability of renal ultrasound as a screening test for alterations in both renal morphology and renal function, and several have demonstrated that it has very poor sensitivity as an indirect indicator of renal function and should continue to be combined with an examination estimating or measuring renal function [19]. In our study, renal ultrasonography was normal in all patients, while abdominal ultrasonography showed hepatic steatosis in 44.73% of cases; a significant percentage, but still moderate compared with the prevalence of hepatic steatosis in obese patients, which is between 70 and 90% [20-22].

The prevalence of CKD in the obese in our population was 10.5%, compared with 2.9% in the general Moroccan population [23,24], while a Spanish study, "The FATH Study", reported that the prevalence of chronic renal failure in obese hypertensive patients was 23%, and in obese diabetic patients, 30.1% [25]; So while it's clear that concomitant diabetes or hypertension significantly increases the risk of chronic kidney disease, it's also clear from our study that obesity itself more than doubles the prevalence of CKD compared with the general population. The role of obesity is also clear when comparing the prevalence of macro albuminuria in our population (7.89%) with that found by NHANES III "The Third National Health and Nutrition Examination" in a non-diabetic, non-hypertensive population of any weight

(0.3%) [26]. As for similar studies investigating renal impairment in healthy obese people (non-diabetic, non-hypertensive and with no other apparent cause of renal disease), they have all demonstrated that obesity is associated with incipient nephropathy independently of all other usual risk factors. However, to the best of our knowledge, there are no studies that have indicated the prevalence of CKD in this type of population in its entirety, i.e., taking into account GFR and all markers of renal impairment; the available studies were aimed solely at investigating alterations in GFR and/or any albuminuria or proteinuria in this population. In the French study by Cassuto et al, the prevalence of clinical proteinuria in healthy obese patients was 3% [19], whereas the American study by Patel et al found a much higher prevalence of 13.3% [6], similar to that found in another Korean study (13.1%), which also included overweight patients with abdominal obesity [27].

In our study, patients with CKD were older, with a highly significant difference (p=0.001) between the 2 groups of patients (with and without CKD), which is consistent with data from the Korean study by Chang et al, where differences in CKD risk among BMI categories were greater in participants aged 40 and over than in younger participants with p=0.02 [11]. None of the series in the literature found a statistically significant difference in CKD risk between male and female sex, and in our series, too, we noted no significant difference for sex (p>0.05) even though all CKD cases in our population were women. Several studies have shown that chronic smoking contributes to the risk of CKD [28-30], whereas in our study and in the corneal study by Chang et al [11] there was no link between smoking and the occurrence of CKD.

Table 3 Comparison of our socio-demographic data with the literature

Series	**Chagnac [55] Israel**	**Stevens [56] Australia**	**Yano [57] Japan**	**Serra [58] Spain**	**Minoo [59] Iran**	**Patel [60] United States**	**Our study**
Year	2000	2001	2007	2008	2015	2018	2018
Number of patients	12	48	63	95	186	101	38
Average age (years)	35.2	47.2	59.4	41.8	30.4	37.2	38
Sex ratio (m/f)	0.33	0.45	0.80	0.61	0.06	0.42	0.80
Chronic smokers (%)	-	-	16	50.5	-	-	10.4

Familial hypertension: In our study, we found no significant difference between the two groups of patients with and without CKD for familial hypertension. However, a French study [31] evaluated the prevalence in obese patients of increased urinary albumin excretion rate (UAER) and the factors involved in this parameter, and found that UAER was higher in patients with familial hypertension than in those without (with a highly significant difference p = 0.002). In our population, patients with CKD were more obese than others, with a fairly significant difference (37.38 vs. 34.43 kg/m2, p=0.046), in line with data from the Korean study by Chang et al [11], which showed that the cumulative incidence of CKD in metabolically healthy obese people was systematically higher in those with a high BMI throughout follow-up. While high waist circumference, in our study, tends to favor CKD without reaching the significance threshold, but in the Australian study by Stevens et al [10], a significant difference in the risk of renal dysfunction was observed in individuals with the same BMI, but differing in fat mass distribution, with a profile of central obesity being of greater risk. dyslipidemia: In our study, triglyceride, total cholesterol and LDL cholesterol levels were higher in patients with CKD with statistically significant differences (p=0.002, p=0.008 and p=0.004, respectively), while HDL cholesterol levels did not differ significantly between those with and without CKD (p>0.05), while the French series by Cassuto et al [19], whose objectives included studying the relationship between urinary albumin excretion rate (UAER) and metabolic parameters, found that only total cholesterol had a statistically significant positive correlation with UAER (P<0.05).

In addition to all the above factors, we also found that patients with CKD had higher CRP, platelet and white blood cell values than those without, with statistically significant differences between the two groups of patients; for CRP (p=0.015), WBC (p=0.035) and Plq (p=0.013). This result may be explained by the presence of an inflammatory syndrome, often associated with obesity on the one hand and CKD on the other. A Korean study [29], for example, evaluated the relationship between CRP, metabolic syndrome and CKD, and showed that the probability of CKD in adults with metabolic syndrome, without diabetes or hypertension, increased in the presence of elevated CRP. As for uric acid, it had no significant effect on the occurrence of CKD in our patients (p>0.05), however several recent studies have spoken of the likelihood of it being another risk factor for CKD often ignored. Similarly, non-alcoholic fatty liver disease (NAFLD) was not significantly linked to CKD risk in our study, but one series in the literature found that NAFLD with

elevated GGT concentration was associated with an increased risk of CKD in non-diabetic, non- hypertensive Korean patients.

5. Conclusion

Obesity is a factor in chronic kidney disease, not only because of the conditions with which it is associated (hypertension, diabetes), but also on its own, with a specific type of damage. This renal risk is exerted through interactions between hemodynamic factors at the glomerular level, hyperactivity of the renin-angiotensin-aldosterone system and the autonomic nervous system, and the involvement of pro-inflammatory and pro fibrosing mediators. Our study showed that CKD was present in 10.5% of obese patients in our population, independently of all other usual risk factors, notably diabetes and hypertension. Older obese patients with high BMI, hypertriglyceridemia, hypercholesterolemia and hyperLDLemia were most affected. Further studies would be desirable to better describe the renal lesions specifically associated with obesity, to enable their early detection and management. The results of our study, like those of series in the literature, sound an alarm about the crucial need to reverse the growing epidemic of obesity worldwide.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare that the research was conducted without any commercial or financial relationships that could be construed as potential of interest.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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