

Pedro Pérez Díaz^{a,*},
 María Dolores Sánchez de la Nieta García^b,
 Jesús Piqueras Flores^a, Ramón Maseda Uriza^a,
 Juan Antonio Requena Ibáñez^a, Manuel Rayo Gutiérrez^a

^a Departamento de Cardiología, Hospital General Universitario de Ciudad Real (HGUCR), Ciudad Real, Spain

^b Departamento de Nefrología, Hospital General Universitario de Ciudad Real (HGUCR), Ciudad Real, Spain

*Corresponding author.

E-mail address: pedroperezdiaz61@gmail.com (P. Pérez Díaz).

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Obesity and kidney function; epidemiological study data: Prevalence of chronic kidney disease in Spain. EPIRCE study[☆]

Obesidad y función renal .datos del estudio epidemiológico: Prevalencia de la enfermedad renal crónica en España. Estudio EPIRCE

Dear Editor,

We would like to take advantage of World Kidney Day 2017 with the slogan “Obesity and Kidney Disease”, to share epidemiological data from the study into the epidemiology of chronic kidney disease in Spain (EPIRCE).¹

It is well known that obesity is a public health problem, and for several years different epidemiological studies have shown a clear relationship between obesity and the risk of developing chronic kidney disease (CKD).² The associated nephropathy is a result of hyperfiltration, glomerular hypertrophy and increased synthesis of vasoactive and fibrogenic substances and dyslipidaemia.³

The EPIRCE study is an observational study of a randomly-selected multistage sample in 42 sampling points (towns) stratified by habitat, age and gender, providing a representative cohort of the Spanish population (n: 2746). The prevalence of obesity (BMI >30 kg/m²) was 26.1% and the odds ratio (OR) of CKD development was 3.5 (95% confidence interval [CI]: 2.0–6.0) while the prevalence of another cardiovascular risk factor, such as arterial hypertension (HTN), was 42% and the OR for CKD development was 6.2 (95% CI: 4.0–9.6).

Table 1 shows that the obese population is significantly more hypertensive and dyslipidaemic, with a higher rate of insulin resistance, and the higher BMI is associated with

conventional risk factors (HTN, dyslipidaemia, HOMA) and with “worse” kidney function and higher proteinuria rate (Alb/creatinine). However, these changes are also seen in the “global” population, whether they are hypertensive or not.

The pathogenic mechanisms of nephropathy seem to be linked to: glomerular hyperfiltration and haemodynamic changes, the dyslipidaemia itself and a greater activation of the renin-angiotensin system, hyperinsulinaemia and a greater synthesis of leptin, oestrogen and TGF-β1.⁴ Regarding the therapeutic approach it is essentially to lose weight, and the progression of nephropathy is reduced through blood pressure control, improvement of insulin resistance and lipid profile, as well as reduction of leptin and RAAS.⁴ It should be noted that this association of HTN, obesity, dyslipidaemia or proteinuria is not a metabolic syndrome, a syndrome questioned not only by Reaven⁵ himself but by the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD)⁶ because the presence of a unique pathogenic substrate has not been proven. Many authors agree that the usefulness of the concept of metabolic syndrome is to highlight the association of multiple CVRFs when making clinical decisions.⁵ We also consider that CKD is the principal CVRF⁷ and the existence of a common pathogenic substrate which might explain the coexistence of obesity and CKD.⁴

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Table 1 – BMI and cardiovascular risk factors.

	BMI <25 (kg/m ²)	BMI 25–30 (kg/m ²)	BMI >30 (kg/m ²)	p value (BMI)
<i>Age</i>				
HTN	56.45	60.65	59.61	0.0016
NHTN	39.01	44.39	46.19	0.0000
Global	42.84	51.59	54.53	0.0000
<i>SBP; mmHg</i>				
HTN	149.63	151.3	150.25	0.4889
NHTN	114.58	120.29	123.23	0.0000
Global	122.27	133.97	140.01	0.0000
<i>DBP; mmHg</i>				
HTN	85.93	86.6	87.86	0.0606
NHTN	71.24	74.08	76.83	0.0000
Global	74.45	79.6	83.68	0.0000
<i>Ct; mg/dl</i>				
HTN	211.02	209.64	210.64	0.0000
NHTN	192.16	205.64	205.91	0.0000
Global	196.41	207.39	208.85	0.0000
<i>Tg; mg/dl</i>				
HTN	96.11	126.44	136.07	0.0000
NHTN	79.41	102.45	124.57	0.0000
Global	82.99	112.97	131.72	0.0000
<i>HDL-C; mg/dl</i>				
HTN	77.86	69.65	68.22	0.0000
NHTN	78.69	70.64	66.52	0.0000
Global	78.56	70.22	67.58	0.0000
<i>LDL-C; mg/dl</i>				
HTN	130.08	130.94	129.85	0.8895
NHTN	114.42	129.01	128.91	0.0000
Global	117.94	129.84	129.49	0.0000
<i>eGFR; ml/min</i>				
HTN	83.25	79.22	79.27	0.0203
NHTN	88.88	87.11	85.24	0.0049
Global	87.64	83.65	81.54	0.0000
<i>HOMA</i>				
HTN	1.52	2.04	2.52	0.0000
NHTN	1.54	1.88	2.53	0.0000
Global	1.54	1.95	2.52	0.0000
<i>Alb/Cr; mg/g</i>				
HTN	10.22	9.99	16.23	0.0007
NHTN	7.29	6.69	10.16	0.0077
Global	7.89	8.17	13.93	0.0000

Alb/Cr: albumin/creatinine; Ct: total cholesterol; DBP: diastolic blood pressure; eGFR: estimated glomerular filtration rate; Global: general study population; HDL-C: HDL cholesterol; HOMA: homeostatic model assessment; HTN: hypertension; LDL-C: LDL cholesterol; NHTA: normotensive; SBP: systolic blood pressure; Tg: triglycerides.
ANOVA “t” test.

REFERENCES

- Otero A, de Francisco ALM, Gayoso P, Garcia F, EPIRCE Study Group. Prevalence of chronic renal disease in Spain: results of the EPIRCE study. *Nefrologia*. 2010;30:78–86.
- Fox CS, Larson MG, Leip EP, Culleton B, Wilson PW, Levy D. Predictors of new-onset kidney disease in a community based population. *JAMA*. 2004;291:844–50.
- Praga M, Morales E. Obesity, proteinuria and progression of renal failure. *Curr Opin Nephrol Hypertens*. 2006;15:481–6.
- Morales Ruiz E, Praga Terente M. Relación entre obesidad y desarrollo de insuficiencia renal. *Hipertensión*. 2008;25:61–9.
- Reaven GM. The metabolic syndrome: is this diagnosis necessary? *Am J Clin Nutr*. 2006;83:1237–47.
- Kahn R, Buse J, Ferrannini E, Stern M. The metabolic syndrome: time for a critical appraisal Joint Statement from the American Diabetes Association and the European Association for the Study of Diabetes. *Diabetologia*. 2005;48:1684–99.
- Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med*. 2004;351:1296–305.

Alfonso Otero González^{a,*}, A.L.M. de Francisco^b, P. Gayoso^c, F. Garcia López^d

^a Servicio de Nefrología, Complejo Hospitalario Universitario de Ourense, Ourense, Spain

^b Servicio de Nefrología, Hospital Marqués de Valdecilla, Santander, Cantabria, Spain

^c Centro de Salud Oroso-XXI de Santiago, Instituto de Investigación Sanitaria de Santiago de Compostela, Santiago de Compostela, La Coruña, Spain

^d Instituto de Salud Carlos III, Centro Nacional de Epidemiología, Madrid, Spain

* Corresponding author.

E-mail address: alfonso.otero.gonzalez@sergas.es (A. Otero González).

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