

- Masoumi A, Reed-Gitomer B, Kelleher C, Bekheirnia MR, Schrier RW. Developments in the management of autosomal dominant polycystic kidney disease. *Ther Clin Risk Manag* 2008;4:393-407.
- Shiroyanagi Y, Suzuki M, Matsuno D, Mochizuki K, Kitagawa N, Tanaka M, et al. Asymmetric development of tumor-like cysts in a child with autosomal dominant polycystic kidney disease. *J Pediatr Surg* 2008;43:e21-3.
- Wilson RD, Baird PA. Renal agenesis in British Columbia. *Am J Med Genet* 1985;21:153-69.
- Costantini F. Renal branching morphogenesis: concepts, questions, and recent advances. *Differentiation* 2006;74:402-21.
- Dressler GR. Advances in early kidney specification, development and patterning. *Development* 2009;136:3863-74.
- Reidy KJ, Rosenblum ND. Cell and molecular biology of kidney development. *Semin Nephrol* 2009; 29:321-37.
- Yosypiv IV. Renin-angiotensin system in ureteric bud branching morphogenesis: insights into the mechanisms. *Pediatr Nephrol* 2011;26:1499-512.
- Bear RA. Solitary kidney affected with polycystic disease: A report of 2 cases. *J Urol* 1974;111:566-7.
- Todorov VV. The diagnostic dilemma of the unilateral cystic kidney-ADPKD with aplasia of one kidney. *Nephrol Dial Transplant* 1999;14:2775.
- Jeong GH, Park BS, Jeong TK, Ma SK, Yeum CH, Kim SW, et al. Unilateral autosomal dominant polycystic kidney disease with contralateral renal agenesis: Acase report. *J Korean Med Sci* 2003; 18:284-6.
- Sirvent AE, Enríquez R, Ardoy F, Amorós F, González C, Reyes A. Autosomal dominant polycystic kidney disease with congenital absence of contralateral kidney. *Int Urol Nephrol* 2006;38:773-4.
- Poster D, Kistler AD, Krauer F. Kidney function and volume progression in unilateral autosomal dominant polycystic kidney disease with contralateral renal agenesis or hypoplasia: a case series. *Am J Kidney Dis* 2009;54:450-8.
- Song R, Yosypiv IV. Genetics of congenital anomalies of the kidney and urinary tract. *Pediatr Nephrol* 2011;26:353-64.
- Skinner MA, Safford SD, Reeves JG, Jackson ME, Freermerman AJ. Renal aplasia in humans is associated with RET mutations. *Am J Hum Genet* 2008;82:344-51.
- Rozen EJ, Schmidt H, Dolcet X, Basson MA, Jain S, Encinas M. Loss of sprouty1 rescues renal agenesis caused by Ret mutation. *J Am Soc Nephrol* 2009;20:255-9.
- Saisawat P, Tasic V, Vega-Warner V, Kehinde EO, Günther B, Airik R, et al. Identification of two novel CAKUT-causing genes by massively parallel exon resequencing of candidate genes in patients with unilateral renal agenesis. *Kidney Int* 2012;81:196-200.
- Nakanishi K, Yoshikawa N. Genetic disorders of human congenital anomalies of the kidney and urinary tract (CAKUT). *Pediatr Int* 2003;45:610-6.
- Szmigielska A, Roszkowska-Blaim M, Werner B, Kamińska H, Brzewski M. Hypertension in a girl with severe coarctation of the aorta and renal agenesis. *J Pediatr* 2012;160:705-6.
- Schreuder MF. Unilateral anomalies of kidney development: why is left not right? *Kidney Int* 2011;80:740-5.
- Kerecuk L, Long DA, Ali Z, Anders C, Kolatsi-Joannou M, Scambler PJ, et al. Expression of Fraser syndrome genes in normal and polycystic murine kidneys. *Pediatr Nephrol* 2012; 27:991-8.

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Diabetic nephropathy confirmed by biopsy: on who and when do we have to perform a biopsy?
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To the Editor:

The prevalence of diabetes mellitus (DM) has increased globally, primarily type 2.¹ One of the main complications of this disease, diabetic nephropathy (DN), is the primary global cause of terminal chronic kidney disease (TCKD), and affects approximately one-third of all DM patients.² The diagnosis of this condition is usually established based on clinical criteria in diabetic patients with albuminuria and/or diabetic retinopathy.

However, it is also common to encounter non-diabetic kidney disease in diabetic patients, which necessitates the indication for renal biopsies in patients with DM and nephropathy, especially those with a rapid progression or atypical forms of disease.^{2,3}

In our study, we describe the characteristics of patients with DN confirmed by renal biopsy. We analysed the motives described for indicating the renal biopsy and the moment in the evolution of disease when the biopsy was taken.

During the study period of 2004-2011, a total of 156 native renal biopsies were performed at the Hospital General de Segovia. In 17 of these (10.9%), a final diagnosis of DN was made.

Table 1 describes the socio-demographic characteristics, pathological history, treatments given, and laboratory results for all of these patients prior to the renal biopsy.

As regards the motive for indicating the renal biopsy, 82.4% of cases were due to nephrotic range proteinuria or nephrotic syndrome, 5.9% were due to acute renal failure, another 5.9% were due to persistent urinary alterations, and the final 5.9% were due to other indications. In 16 patients, the diagnosis was established based on the first renal biopsy, and the other patient required a second biopsy to confirm the diagnosis.

In two patients, the diagnosis of DN was confirmed when the patient had TCKD, while on a dialysis programme.

Table 1. Sociodemographic characteristics, pathological history, laboratory results, and treatments given prior to renal biopsy.

Age (years)	63.41±12 (39-83)
Sex (male/female)	58.8 %/41.2 %
Arterial hypertension	94.1 %
Known diabetes mellitus	94.1 %
Baseline serum creatinine (mg/dl)	1.31±0.45 (0.6-2.50)
Serum creatinine at diagnosis (mg/dl)	1.96±1.19 (0.7-5)
Creatinine clearance (ml/min)	49.15±36 (0-137)
Serum albumin (g/dl)	3.28±0.58 (1.7-4.40)
24 hour-urine proteinuria (g/24h)	7.01±5.82 (1.56-26)
Haemoglobin A1c (%)	7.52±1 (6-9.6)
Years evolution of diabetes mellitus	9.92±6.47 (1-25)
ACE inhibitors	64.7 %
ARB	64.7 %
ACE inhibitors and ARB	47.1 %
Oral anti-diabetics	41.2 %
Insulin	58.8 %
Oral anti-diabetics + insulin	17.6 %
Number of glomeruli biopsied	11±5.7 (5-23)

ARB: angiotensin receptor blocker; ACE: angiotensin-converting enzyme.

Lin et al. performed a retrospective analysis of 50 renal biopsies in patients with type 2 DM, showing that in patients with type 2 DM of at least 10 years evolution and retinopathy, the presence of non-diabetic kidney disease cannot be ruled out. In their study, elevated serum albumin levels and low urinary protein losses served as indications for renal biopsies in order to exclude the possibility of non-diabetic kidney disease.⁴ In our study however, the primary motive for indicating renal biopsy was severe, persistent, or increasing proteinuria in patients that had already been treated with anti-proteinuric drugs, in which the diagnosis of DN was confirmed.

To conclude, our patients with DN as confirmed by renal biopsy had nephrotic range nephropathy or severe nephrotic syndrome, with DM of long evolution and associated with poor metabolic control.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

1. Ritz E, Zeng XX, Rychlik I. Clinical manifestation and natural history of diabetic nephropathy. *Contrib Nephrol* 2011;170:19-27.

2. Chong YB, Keng TC, Tan LP, Ng KP, Kong WY, Wong CM, et al. Clinical predictors of non diabetic renal disease and role of renal biopsy in diabetic patients with renal involvement: a single centre review. *Ren Fail* 2012;34(3):323-8.

3. Haider DG, Peric S, Friedl A, Fuhrmann V, Wolzt M, Hörl WH, et al. Kidney biopsy in patients with diabetes mellitus. *Clin Nephrol* 2011;76(3):180-5.

4. Lin YL, Peng SJ, Ferng SH, Tzen CY, Yang CS. Clinical indicators which necessitate renal biopsy in type 2 diabetes mellitus with renal disease. *Int J Clin Pract* 2009;63(8):1167-76.

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Results of renal transplant with multiple renal arteries in Veracruz, Mexico

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To the Editor:

Many of the challenges presented by the surgical procedure of renal transplants are the result of anatomical variations, such as multiple renal arteries (MRA), which are present in 12%-30% of all transplanted kidneys.¹⁻⁵ The surgical evaluation of live donors facilitates a determination of kidney anatomy in order to establish the safety of the nephrectomy, the most appropriate surgical technique to use, and the length of the blood vessels that will be used. Complex renal vascularisation continues to present a problem that can affect the prognosis of the transplant.² The first studies involving this issue considered kidneys with MRA to be a contraindication due to the possible increase in vascular complications (stenosis of the renal artery or thrombosis and bleeding),^{4,5} although currently, the use of these kidneys is more widely accepted.³

We performed a retrospective analysis of 216 cases recorded over the course of 7 years; of these, 23 patients (10.6%) had MRA as compared to a control group (n=23) with single renal arteries (SRA). The mean patient age in the SRA group was 34±10.3 years (range: 18-52 years), whereas the mean age in the MRA group was 35±10.7 years (range: 17-51 years). The majority of patients were male in both groups (SRA: 82.6%, n=19; MRA: 82.6%; n=19). Mean body mass index (BMI) in the SRA group was 25.08±3.85kg/m² (range: 19.74-36.94kg/m²), and the mean BMI in the MRA group was 25.05±4.34kg/m² (range: 19.07±36.7kg/m²). The mean time on dialysis in the SRA group was 24±13.56 months (range: 3-78 months), and the mean time on dialy-