

among patients with persistent hematuria during the follow-up than in those who presented resolution of hematuria (28.2% versus 17.9% and 53.8% versus 0%; $P = .002$).

Furthermore, we found that patients who presented on the kidney biopsy a more intense immunofluorescence staining of C3 were more likely to evolve to ESRD or have greater loss of kidney function ($p = .001$).

Other variables, such as male gender, lower eGFR and presence of hypertension at diagnoses, also showed to be related to poor renal survival, although they did not reach statistical significance.

The treatment with renin-angiotensin-aldosterone system inhibitors or immunosuppressive agents did not show impact on renal survival.

CONCLUSION: The grade of proteinuria is a well-established predictor of worse outcomes in patients with IgAN, and its reduction is one of the main objectives in these patients. On the other hand, the role of hematuria in disease progression is still controversial and the relationship between the level of C3 deposition and the long-term prognosis has rarely been reported [1, 2, 3]. With our data, we reinforce the need to integrate all different pathophysiological mechanisms to establish the disease prognosis.

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MO205

THE OUTCOME OF PATIENTS WITHOUT NEPHROTIC SYNDROME (NS) IS NOT DIFFERENT FROM PATIENTS WITH NS, WHO WERE DIAGNOSED AS MINIMAL CHANGE LESION

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BACKGROUND AND AIMS: Minimal change lesion (MCD) accounts for 10–15% of adult patients with nephrotic syndrome and is characterized by edema, nephrotic-ranged proteinuria (NP) and pathologic findings of nearly normal appearance of glomeruli on light microscopy, minimal staining of immunoglobulins and extensive effacement of foot process of the podocyte. However, the fate of MCD with typical pathologic findings without nephrotic proteinuria (Non-NP) needs to be defined in more details.

METHOD: We enrolled 79 adult MCD patients with the first renal biopsy in a tertiary teaching hospital from May 2003 to June 2017. We did not include patients having any immunosuppressive treatment before renal biopsy, patients with inappropriate biopsy samples (<10 glomeruli/biopsy, any electron dense deposit, and light microscopic findings suggestive of secondary causes). Remission of proteinuria was defined as urine protein-to-creatinine ratio (UPCR) < 0.3 g/g creatinine and recurrence of proteinuria as UPCR ≥ 3.0 g/g creatinine. We compared the clinic-pathologic features of MCD with or without NP at admission for renal biopsy, the frequency of flare to nephrotic proteinuria and the renal failure which was defined as decrease of estimated glomerular filtration rate (GFR) more than 50% compared to GFR at renal biopsy, GFR less than 15 mL/min/1.73 m², or progressed to end-stage renal disease during follow-up period.

RESULTS: There were 3 patients with UPCR < 0.3 g/g creatinine, 17 patients with UPCR 0.3–2.9 g/g creatinine and 59 patients with UPCR ≥ 3.0 g/g creatinine at admission for renal biopsy. Mean age at renal biopsy was 53.7 ± 19.2 (range:18.5–99.0) years, and there were 38 male patients (48.1%). Non-NP group had lower UPCR (1.36 ± 0.99 versus 10.2 ± 6.21 g/g creatinine, $P < .001$) and higher GFR (98 ± 27 versus 75 ± 36 mL/min/1.73 m², $P = .012$) at renal biopsy, and lower frequency of acute kidney injury during follow-up period [1/20 (5.0%) versus 35/59 (59.3%) patients, $P < .001$]. NP group showed more severe foot process effacement and interstitial changes. Other clinic-pathologic findings were not different between groups. During follow-up period (63.2 ± 50.4 months), eight patients (40.0%) had recurrence of proteinuria and nine patients (45.0%) had steroid treatment in non-NP group. Among NP group, 55 patients (93.2%) had been treated by steroid. The peak dose of prednisolone for patients having steroid treatment was not different between groups (Non-NP versus NP: 0.92 ± 0.16 mg/kg/day versus 0.88 ± 0.17 mg/kg/day, $P = .491$). The remission rate by the first treatment of steroid was not different

between groups (non-NP versus NP: 9/9 versus 50/55, $P = 1.000$). The recurrence of proteinuria was developed in 8/9 non-NP patients (88.9%) and in 25/50 (50.0%) NP patients after the first remission of proteinuria by steroid treatment ($P = .282$). Recurrence of proteinuria was developed 0.23 ± 0.33 times/year in Non-NP group and 0.25 ± 0.41 times/year in NP group ($P = .830$). At the last follow-up period, the final remission rate of proteinuria was not different [65.0% of non-NP patients (17/20) versus 76.5% of NP patients (45/59), $P = .384$], and the rate of renal failure was not different, neither [15.0% of non-NP patients (3/20) versus 27.1% of NP patients (16/59), $P = .371$].

CONCLUSION: The adult MCD patients with non-nephrotic-ranged proteinuria at renal biopsy showed similar clinical outcomes compared with patients with nephrotic-ranged proteinuria. Therefore, the MCD pathology should be paid more attention regardless of the amount of proteinuria at renal biopsy.

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HLA FREQUENCY IN A SPANISH TRANSPLANT PATIENTS COHORT WITH ANCA-ASSOCIATED VASCULITIS

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BACKGROUND AND AIMS: ANCA-associated vasculitis (AAV) is an autoimmune disease that causes end-stage renal disease in ~20%–25% of patients who develop kidney failure with progression to end-stage kidney disease with requirement of renal replacement therapy. Previous studies have found a genetic association between some major histocompatibility complex (MHC) loci, like the locus DPB1*0401, and AAV. We conducted a retrospective observational uncentric study with the objective to identify other possible loci related to AAV.

METHOD: As MHC determination is not usually done in patients with AAV, we looked for patients with AAV included in the kidney transplant list with a complete classic HLA allele determination registered (A, B, C, DR, and DQ). Only 14 patients with diagnosis of AAV included in the kidney transplant list from August 1993 to December 2020 in our hospital centre fulfilled this inclusion criterion. To the statistical analysis, we compared the observed proportion to the theoretical one using a normal test, since the groups in our sample included at least five subjects. We also use a continuity correction factor to approach to a binomial test.

RESULTS: The highest allele frequency in our cohort were the HLA B*51 and the haplotype HLA DRB1*04-DQB1*03:02. We identified a B*51 frequency of 42.8% (6/14 patients) in our AAV register, while its population frequency in Spain is 15%, difference = 27.86% [Confidence interval (CI) 1.93%–53.78%] $P = .01$. We detected a DRB1*04 frequency of 57.1% (8/14 patients) in our cohort, while the population frequency of DRB1*04 and its associated loci DQB1*03:02 is 22%, difference = 35.14% (CI 9.22%–61.07%) $P < .05$.

CONCLUSION: HLA B*51 and the haplotype DRB1*04-DQB1*03:02 are alleles more frequent in patients with AAV than general population. This could translate a genetic predisposition of people with these alleles to develop AAV.

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A PHASE 1/2, MULTICENTER STUDY TO INVESTIGATE THE SAFETY, TOLERABILITY, PHARMACOKINETICS AND PHARMACODYNAMICS OF BION-1301 IN HEALTHY VOLUNTEERS AND ADULTS WITH IGA NEPHROPATHY

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BACKGROUND AND AIMS: Immunoglobulin A nephropathy (IgAN) is the leading cause of primary glomerulonephritis worldwide with limited treatment options, especially for high-risk patients [1]. BION-1301 is a novel humanized monoclonal antibody that blocks a proliferation-inducing ligand (APRIL), a soluble factor that has been shown to be elevated in patients with IgAN and is correlated with poorer outcomes, including increased proteinuria and decreased eGFR [2, 3]. APRIL promotes IgA class switching, the survival of IgA-secreting plasma cells and the excess production of a galactose-deficient variant form of IgA1 (Gd-IgA1), which is an initiating step in the multi-hit pathogenesis of IgAN. This leads to the generation of anti-Gd-IgA1 autoantibodies and the formation of nephritogenic immune complexes that deposit in the kidney, resulting in inflammation and damage [2–4]. Blocking APRIL with BION-1301 is a novel approach to address the underlying pathogenesis of IgAN by reducing circulating levels of Gd-IgA1 and preventing the formation of pathogenic immune complexes. The primary objective of this Phase 1/2 study is to assess the safety and tolerability of BION-1301 in healthy volunteers (HV) and patients with IgAN, and secondarily to assess the PK, PD, immunogenicity and preliminary clinical activity.