pressure and pulse pressure. It is not related to the use of specific hypotensive medication. Patients with DM or with poor glycaemic control exhibit a higher VRI. It is not related to the degree of proteinuria or renal function, nor does it appear to have prognostic implications.

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Letter to the Editor

IgA nephropathy: Short term effects of prednisone treatment on proteinuria, renal function and relation with Oxford classification☆

Nefropatía IgA: efectos a corto plazo del tratamiento con prednisona sobre la proteinuria, función renal y relación con la clasificación de Oxford

Dear Editor,

IgA nephropathy (IgAN) is the most common primary glomerular disease worldwide. The clinical variables most commonly associated with the risk of developing end-stage renal disease (ESRD) for IgA nephropathy are the degree of proteinuria, reduced baseline glomerular filtration rate and arterial hypertension.[1] Given the great variety of histological lesions of IgAN, their description was homogenised and variables that had prognostic significance for renal impairment were sought, developing the Oxford classification,[2,3] which has been validated in various studies.[3,4] In IgAN the demon-

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Table 1 – Progression of renal function, proteinuria and albuminuria after treatment with prednisone in patients with IgA nephropathy.

	Baseline	6 months	12 months	p-Value[a]
Creatinine (mg/dl)	1.6 ± 0.8	1.6 ± 0.7	1.8 ± 1.1	NS
eGFR-MDRD (ml/min/1.73 m^2)	58 ± 28	58 ± 25	52 ± 24	NS
Proteinuria (mg/gCr)	1675 ± 1998	1185 ± 1498	1023 ± 1496	<0.001
Albuminuria (mg/gCr)	1023 ± 1134	806 ± 1028	667 ± 973	0.003
[0,1–5] Progression of proteinuria (mg/gCr) according to the Oxford classification (MEST-C)				
Mesangial hyp. 0	1050 ± 1089	396 ± 288	392 ± 298	0.09
Mesangial hyp. 1	2023 ± 2339	1552 ± 1732	1313 ± 1787	0.002
Endocapillary prol. 0	1562 ± 1510	959 ± 937	699 ± 546	0.014
Endocapillary prol. 1	1960 ± 2633	1527 ± 2032	1444 ± 2169	0.024
Segm. sclerosis 0	2157 ± 2658	1625 ± 1981	1554 ± 2096	NS
Segm. sclerosis 1	1378 ± 1388	867 ± 968	597 ± 634	0.001
Tub. atrophy/Int. fib. 0	1311 ± 989	825 ± 1052	1079 ± 1836	NS
Tub. atrophy/Int. fib. 1	1595 ± 1733	1212 ± 1231	743 ± 725	0.002
Extracapillary prol. 0	1674 ± 1550	1183 ± 1168	1053 ± 1481	0.005
Extracapillary prol. 1	2027 ± 3060	1462 ± 2277	1157 ± 1801	0.016

A significant decrease in proteinuria and albuminuria is broadly observed at six and twelve months after treatment, without significant changes in renal function. Upon stratification according to the histological parameters of the Oxford classification, we observed a decrease in proteinuria in each stratum of the Oxford classification parameters.
NS: not significant.

[a] Friedman test.

stration of efficacy of any therapeutic strategy is very difficult given the slow progression of the disease and the need for very long follow-up studies. The use of steroids is recommended in cases with proteinuria >1 g/day and glomerular filtration rate >50 ml/min/1.73 m^2, although positive responses have also been observed with lower glomerular filtration rates,[5] achieving better preservation of renal function, reduction of proteinuria and microhaematuria,[5–7] although with common adverse effects.[6,8] The response to steroids appears to be greater in patients with mesangial and endocapillary proliferation, glomerulosclerosis and even tubulointerstitial fibrosis,[5] although the prognosis is not clear in relation to the Oxford classification.

Our objective in this study was to review the short-term response of renal function and proteinuria in patients with IgAN who were treated with corticosteroids, taking into account the histological parameters of the Oxford classification. We reviewed patients who had received corticosteroid treatment for a minimum of 6 months. The study included 33 patients aged 41 ± 13 years, 23 (69.7%) were men, with a mean weight of 73.2 ± 12.4 kg. In total, 19 patients were taking ACE inhibitors, seven were receiving angiotensin II receptor blockers (ARBs) and seven dual blockade. All had been on this treatment for at least six months before starting steroid treatment (mean treatment time of 14 months). We administered prednisone at a starting dose of 40−80 mg/day (<1 mg/kg/day) for 2–3 weeks, with a subsequent gradual decrease of 10 mg/every 2–3 weeks until reaching 10 mg/day as a maintenance dose for six months. If proteinuria had decreased <250 mg/day before six months had elapsed, it was reduced until it was discontinued at three months. The starting dose of prednisone was 57 ± 6 mg/day (median 60) and 0.8 ± 0.1 mg/kg/day. In six patients, cyclophosphamide was given associated to prednisone (50 mg/day in one patient, 100 mg/day in four and 125 mg/day in one) for up to six months. The prevalence of the Oxford variables was: M1 63.6%; E1 42.4%; S1 45.5%; T0 39.4%, T1 45.5% and T2 3%; C 27.3%.

In terms of progression, we observed a significant decrease in proteinuria and albuminuria at six and twelve months, without observing significant changes in renal function (Table 1). Proteinuria decreased in 72.7% of the patients at six months and in 81.8% at twelve months. The median reduction in proteinuria was 40% at six months and 45.8% at twelve months.

Table 1 shows the progression of proteinuria according to the MEST-C parameters of the Oxford classification. Baseline proteinuria was higher in patients showing: mesangial hyperplasia, endocapillary proliferation, interstitial fibrosis/tubular atrophy and extracapillary proliferation, although it did not reach statistical significance. In terms of progression, we observed a general reduction in proteinuria levels in practically all the strata considered on the MEST-C score. The presence of arteriolar hyalinosis, muscle hyperplasia or arterial intimal proliferation did not appear to have any influence.

In some studies, a worse response to treatment has been observed in cases with M1 mesangial proliferation, S1 segmental sclerosis and T1/T2 tubular atrophy.[9] However, other publications have shown that the use of corticosteroids reduce proteinuria and the rate of kidney impairment progression by a similar amount in both strata of the Oxford classification variables.[5] In our case, despite higher proteinuria and worse baseline renal function in categories 1 versus 0 of the MEST-C variables, we observed a reduction in proteinuria in all the strata studied. When renal biopsies were repeated after treatment, an improvement was observed in all proliferative parameters and in changes in fibrosis and tubular atrophy when corticosteroids are used,[10] which could explain the lack of correlation with the baseline histological parameters of the Oxford classification.

Therefore, in patients with IgAN, steroid treatment may reduce proteinuria regardless of MEST-C characteristics. There is a lack of studies that clarify the degree of response to be expected after the use of steroids on glomerular filtration rate decline that take into account each prognostic variable of the MEST-C classification.

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Letter to the Editor

Fibronectin glomerulopathy: A case report and literature review

Glomerulopatía por fibronectina: informe de un caso y revisión de la literatura

ARTICLE INFO

Article history:

Dear Editor,

Fibronectin glomerulopathy (FNG) is an uncommon autosomal dominant disease with age-related penetrance. Burgin[1] first reported the diease in 1980. Strøm[2] verified fibronectin glomerulopathy – a newly recognized autosomal dominant kidney disease. It has been distinguished from these other disease entities by the presence of glomerular fibrillary deposits that show strong immune reactivity to fibronectin (Fn).

Here we report a case of FNG 2017 in our hospital. A 45-year-old Chinese female was hospitalized because of edema for 7 days. She denied history of hair loss, oral ulceration, alopecia, oliguria, joint pain, rashes and menstrual irregularities. She had medical history of malignant hydatidiform mole 8-years ago which had been cured. Her sister had nephritic syndrome for ten years with no biopsy performed. Physical examination showed mild hypertension (158/119 mmHg) and leg edema. The laboratory examination showed her daily urinary protein, serum albumin, blood urea nitrogen and creatinine were 4.21 g/day, 40.9 g/L, 4.39 mmol/L, 53 μmol/L. The blood tests of liver function, lipid, immunoglobulin, complement, antinuclear antibody spectrum, antineutrophil cytoplasmic antibody, hepatitis B and C serology were all negative. The renal and gynecologic ultrasound was normal. The kidney biopsy was showed in Fig. 1. Light microscopy showed one of 25 glomeruli was sclerotic glomeruli, others showed moderate to severe mesangial hypercellularity with a lobular appearance with cellular mesangial nodules that were expanded by matrix. Capillary walls showed stenosis, occlusion and basement membrane thickening. Segmental mesangial insertion and double track formation could be seen. Immunofloresecence only showed faint staining for IgM deposits in a band pattern in the subendothelial spaces and testing of IgG, IgA, C3, Cq1, fibrinogen, albumin completely negative. There was no evidence of amyloid deposits on Congo Red stain. Diffuse strong granular-smudgy staining was observed in mesangial and subendothelial locations with fibronectin. Electron microscopy showed 2 glomeruli that the capillary loops were lobulated, and focal endothelial cells proliferated. The wall of renal capsule is thickened and stratified. Extensive fine granular deposits in the mesangium and in the subendothelial spaces could be seen with a diameter of 10–12 nm fibrils.

A PubMed and CNKI searching using the terms “fibronection glomerulopathy” yielded 35 English and 6 Chinese articles reports of patients with clinical and (or) biopsy characteristics until January 31, 2018. This allowed us to compare the disease clinical features and biopsy features. A total of 86 patients, 52 male, 34 female, aged 3–88[3] years were reported in these literatures. 25% of patients receive renal replacement therapy.[2] 42 patients among 78 patients observed with nephritic range proteinuria (more than 3.5 g/day). 53 patients among 85 patients suffered from hypertension. 23 of 43 cases which were available Urine Analysis had microhematruia. 23 cases’ serum creatinine (normal values 0.6–1.3 mg/dl) was abnormal except for 18 patients who did not provide renal function. Family history was noted in 61 patients. 57 patients of the literatures were provided with renal biopsy. 47 patients were tested intense staining for the serum fibronectin by immunohisto-

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