"laboratory reagents" in order to meet the needs of groups that carry out clinical research. There are many more examples of this concern for clinical research in the hospital setting but presenting an exhaustive list does not seem necessary.

A brief final reference to the dichotomy of clinical and basic investigation: we have to forget about it. We must stop systematically talking about this antagonism. Those researchers who identify themselves as basic researchers should start interiorizing a profound and convinced feeling of respect for clinical research. Clinical researchers should understand that some of their future activities are going to be conditioned by the work of basic researchers.

They should speak amongst themselves in order to understand each other and, despite the difficulty, speak less and less about this antagonism thus making it disappear.

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Membranoproliferative Glomerulonephritis and Monoclonal Gammopathy of Uncertain Significance

Nefrología 2009;29**(2)**:175-176.

Dear Editor,

A series of patients with kidney problems secondary to monoclonal gammopathies without malignant criteria was recently published in Nephrology.[1] We present the case of a patient with membrane-proliferative glomerulonephritis of a non-filiated aetiology where a monoclonal gammopathy of undetermined significance was detected.

A 60 year old woman presented with a history of high blood pressure, heterozigous beta thalasaemia and plantar psoriasis treated topically. She sought a consultation for leg oedema of one week duration. Impairment of kidney function was detected in the blood tests. Cr 1.9mg/dl, urea 85mg/dl. The full blood count showed anaemia with the rest of the parameters and coagulation normal. There was nephrotic-range proteinuria with normal total serum protein and albumin levels. The urinary sediment contained 35 red blood cells/field. The immunological study showed hypocomplementaemia with a decrease in the C3, C4 and CH50 levels. Two cryoglobulin tests were carried out and both were negative. The electrophoretic blood study was normal while the electrophoretic urine study presented a Bence Jones Lambda band (200 mg/24 h). In the bone marrow biopsy, the percentage of plasma cells was 2%. The serology for HBV and HCV did not indicate active infections.

All of the glomeruli in the kidney biopsy presented a similar histological aspect with a diffuse proliferation of the matrix and the mesangial cellularity adopting a lobulated pattern. The interstitial area had a slight non-specific infiltration of lymphocytes without fibrosis. The vascular and tubular components showed no lesions. The immunoflourescence showed granular deposits of IgG and C3 (figures 1 and 2).

This patient presented with monoclonal gammapthy of unknown significance and membranoproliferative glomerulonephritis for which no underlying cause was found an aetiological factor.

The glomerular involvement in the monoclonal gammopathies generally occurs in the malignant forms and is usually associated with deposition of light chains.[2] The presence of renal involvement is less frequent in patients with benign monoclonal gammopathies. However, the benign character does not exclude the existence of secondary renal involvement.[3,4] Recently, a series of ten patients with glomerular involvement associated with benign monoclonal gammopathies has been published.

There is however a few reported cases in the literature.[1]

In 2004, Nasr *et al*. published a series of ten patients with idiopathic proliferative glomerulonephritis associated with a deposition of monoclonal immunoglobulins (IgG).[5] Half of the patients presented monoclonal bands on blood and urine electrophoresis, none of them meeting criteria for myeloma in bone marrow biopsies. Four of the patients presented hypocomplementaemia but the cryoglobulin determinations were negative for all of them. Samuh and coworkers suggested that the deposit of monoclonal immunoglobulins might cause a proliferative glomerulonephritis mimicking the immune complex-mediated glomerulonephritis. We report this new case of renal involvement in monoclonal gammopathies of undetermined significance that we believe could be similar to those published in the series of Nasr *et al*.[1,5]

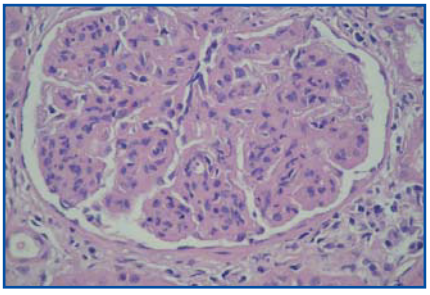

Figure 1. Kidney biopsy: hematoxylin-eosin: glomerulus of lobulated appearance with diffuse matrix expansion and increased megangial cellularity.

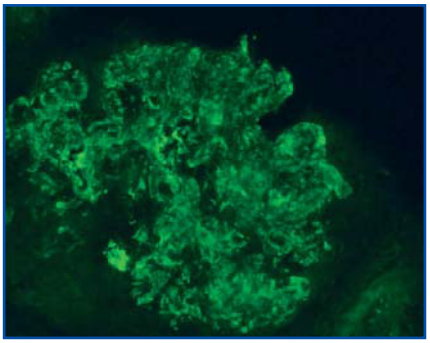

Figure 2. Kidney biopsy: immunofluorescence: deposits of IgG in the glomerular capillary wall.

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B) BRIEF INFORMATION ON RESEARCH AND CLINICAL REPORTS

Infection in Haemodyalisis Catheters: a Retrospective Examination

Nefrología 2009;29**(2)**:176-177.

Dear Editor,
Central venous catheter infections are an important cause of morbidity and mortality in patients on haemodialysis. The KDOQI describes an infection frequency of between 3.8 and 6.6 episodes/1,000 days of catheter for non-tunnelled catheters and between 1.6 to 5.5/1,000 days of catheter for tunnelled catheters.[1] The frequency of bacteraemia varies in various studies between 1.6 and 7.7/1,000 days of catheter for non-tunnelled catheters and between 0.2 and 0.5/1,000 days of catheter for tunnelled catheters.[2-6]

Our goal was to review the catheter infections in our Haemodialysis Unit. Patients and methods: we examined the catheter infections over a period of six years (1 January 2001 to 31 December 2006). We have implanted 168 catheters, 90 of which were non-tunnelled and 78 tunnelled, in 70 patients. Mean age at the time of catheter implantation was 70 ± 14 years old and the average time on haemodialysis 10 months (1 day-17 years). Approximately 25.7% of the patients suffered from kidney disease of unknown aetiology and 22.8% had diabetic nephropathy. Cephazolin was administered before the implantation of tunnelled catheters in all the cases.

Results: The tunnelled catheters were left in place for a median time of 4 (0-45) months and the non-tunnelled catheters were left for 1 (1-6) months. Diabetic patients did not experience more infections (32.4 vs. 34.2%; p = NS) or bacteraemia (2.6 vs. 3.8%; p = NS) than the rest of the patients. The incidence of infections was 2.33/1,000 days of catheter for non-tunnelled catheters and 3.10/1,000 days of catheter for tunnelled catheters (p = NS). The most frequent type of infection was that of the exit site both in tunnelled catheters (44/57 [77.2%]) and non-tunnelled catheters (7/9 (77.8%]); p = NS. The incidence of bacteraemia of the non-tunnelled catheters was not greater than that of the tunnelled catheters (0.78/1.000 versus 0.22/1,000 days of catheter; p = 0.08). Twenty-one tunnelled catheters (26.9%) were implanted over a guidewire in non-tunnelled catheters but these did not suffer more infections (8/29 versus 13/49, p = NS).

Cephazolin (55.3%) was the empirically-used antibiotic in the majority of the infections.

Staphylococcus was the predominant type, identified in blood cultures in 100% of the cases and in 79.2% of the exit site swabs. The prevalence of Methicillin-resistant species was 60%.

After the microbiological results, the initial antibiotic was changed in 22.7% of the cases (N = 15). In almost half of them (46.7%) the antibiotic was switched to Vancomycin.

All patients with sepsis (N = 9) were hospitalized. One died of septic shock and the rest recovered fully.

Conclusions: The non-tunnelled catheters were used for one month without any negative impact on the number of infections. In our opinion, they are safe as a temporary access site for patients that are waiting for the construction or maturing of a definitive access.

Due to the high prevalence of Methicillin-resistant species in our centre led to the subsequent inclusion of Vancomycin in our protocol for the management of catheter infections.

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