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Acute renal failure in a case of nephrotic syndrome secondary to focal and segmental glomerulosclerosis

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SUMMARY

Nephrotic syndrome is infrequently complicated with appearance of acute renal failure and minimal change disease is the glomerulopathy more usually involved. Pathogenesis is unclear and three possible mechanisms it has been proposed to explain the decrease of glomerular filtration rate: a severe reduction of glomerular permeability, the presence of acute tubular necrosis or an increased intrarrenal pressure related with interstitial oedema. Here we present a 36 years-old-male with a nephrotic syndrome caused by focal and segmental glomerulosclerosis who developed an anuric acute renal failure. Renal function did not change despite oedema removal with haemodialysis and only after corticosteroid and cyclophosphamide therapy introduction we observed a rapid recovery of urinary output and resolution of acute renal failure. Renal biopsy did not show signs of tubular damage or obstruction with proteins nor significant interstitial oedema. Therefore, in this case we think acute renal failure was caused by a severe reduction in glomerular ultrafiltration rate and steroids were the effective treatment that allowed recovery of renal function.

Key words: *Nephrotic syndrome. Acute renal failure. Focal and segmental glomeruloesclerosis.*

RESUMEN

El fracaso renal agudo en el síndrome nefrótico es poco frecuente y suele asociarse con una nefropatía de cambios mínimos. Su etiopatogenia es oscura y se relaciona con una reducción de la permeabilidad glomerular, con necrosis tubular aguda o con un incremento de la presión intrarrenal debido al edema intersticial. Presentamos un varón de 36 años con un síndrome nefrótico por glomeruloesclerosis focal y segmentaria que desarrolló un fracaso renal agudo anúrico. A pesar de reducir el edema con hemodiálisis fue tras iniciar tratamiento con esteroides e inmunosupresores cuando la diuresis se restableció y mejoró rápidamente la función renal. En la biopsia renal no se observaron datos de necrosis u obstrucción tubular ni de edema intersticial, por lo que atribuimos el fracaso renal agudo a una severa reducción del coeficiente de ultrafiltración glomerular.

Palabras clave: Síndome nefrótico. Fracaso renal agudo. Glomeruloesclerosis focal y segmentaria.

INTRODUCTION

The development of acute renal failure (ARF) in patients with nephrotic syndrome is rare and usually associated to minimal changes nephropathy.[1] The pathogenesis is not clear and several mechanisms have been implicated.[2,3] The increase in the pressure within the tubules and the Bowman's space can play a role. This is due both to interstitial edema and to intratubular obstruction because of accumulation of proteins[4] and cellular detritus.[5] Hypovolemia is another mechanism implicated and is related to hypoalbuminemia and to the excessive use of diuretics.

CASE REPORT

A 36 year-old male is presented with a history of nephrotic syndrome in complete remission since 2000.

In 2004 he was evaluated in the Emergency Room because of edemas. The laboratory findings revealed mild renal function impairment and mild proteinuria. Diuretics were prescribed and the patient was referred to the outpatient clinic. Eight days later he was again evaluated because of increasing edemas and he was admitted to the hospital. The following laboratory findings were obtained: urea 71, Cr 1.15, cholesterol 423, triglycerides 254 mg/dL; total proteins 4.32, albumin 2.22g/dL; proteinuria 3.1 g/12-hours. A new renal biopsy was performed and the patient was discharged.

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A week later the patients was again admitted with anasarca and oligo-anuria. Serum level of urea was 287 and creatinine was 4.1 mg/dL. Severe hypoalbuminemia was detected that prompted the administration of albumin. The urine analysis revealed Na < 10 mEq/L and Cr > 130 mg/dL, which supported the diagnosis of prerrenal renal failure. Diuretics were withdrawn. The patient developed anuria and a central catheter was placed to initiate hemodialysis. Renal biopsy (fig. 1) showed 8 glomeruli with mild sclerosis and capillary collapse within the vascular pole and others with capsular adhesions or capillary lumen collapse. The interstitium and the tubules were normal. The immunofluorescence pattern was granular mesangial IgM+ and C3 ++. A diagnosis of focal and segmentary glomerulosclerosis (FSGS) was made and three boluses of steroids were administrated followed by oral prednisone and cyclophosphamide. When the patient was discharged he was in anuria and required periodic hemodialysis.

Two days later the patient was again admitted to the hospital to evaluate renal function because he referred increasing diuresis that reached 7 L/day. On day 9 of hospital stay the laboratory parameters were as follows: urea 32, Cr 0.8 mg/dL; proteinuria 2.5 g/24h; CrCl 156 mL/min.

After 10 months of periodic follow up, the proteinuria was negative and therefore treatment with steroids and cyclophosphamide could be discontinued. The patient remained asymptomatic, with no signs of nephropathy relapse.

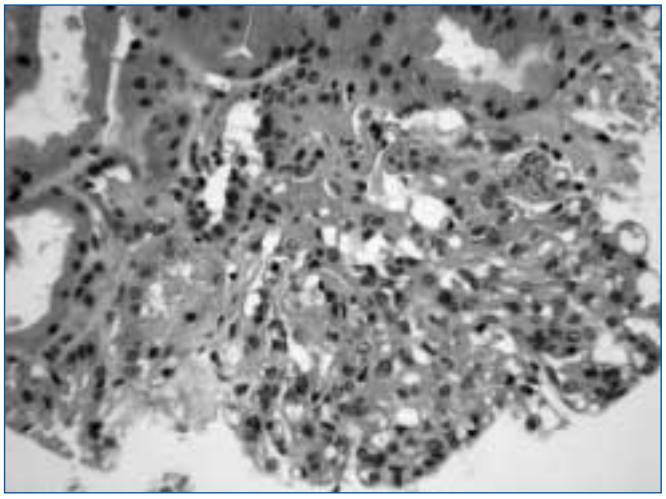

Figure 1. Glomerule with mild mesangial hypercellularity, and hyaline material condensation within the afferent arteriole (H&E 20x10).

DISCUSSION

Some cases of ARF remission without steroidal administration have been reported.[7] However the treatment with high doses of prednisone (1 mg/kg/day) is considered necessary in case of nephrotic syndrome associated to FSGS and can induce complete remission in 35-45% of the patients.[8] The effect of steroids on glomerular filtrate in ARF associated to nephrotic syndrome is unknown, but they may prevent the tubular reabsorption of sodium and allow the complete recovery of filtration fraction[9,10] as proteinuria decreases. The judicious use of diuretics should maintain the diuresis, avoiding a misbalance of pressures within the glomeruli and the potential progression to anuria. Ultrafiltration hemodialysis is indicated in case of nephrotic syndrome due to FSGS with ARF, which presents with oligo-anuria and accumulation of nitrogen products. At the same time, steroidal treatment should be initiated or their dose increased. The resolution of ARF is in this way to be expected, which may occur in a few weeks or months[5,11] or dramatically, as it happened in the reported case.

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