Nephrotic Syndrome after Extracorporeal Shock Wave Lithotripsy

A.		Araque
C.		Alamo
B.		Fraile
R.		Díaz-Gonzalez
M.		Praga

Departments of Nephrology and Urology, Hospital 12 de Octubre, Madrid, Spain

M. Praga, Servicio Nefrología, Hospital 12 de Octubre, Ctra Andalucía km 5.400, E-28041 Madrid (Spain)

Dear Sir,

Extracorporeal shock wave lithotripsy (ESWL) is a noninvasive therapy used worldwide for upper tract renal stone disease. Several studies have shown that ESWL is frequently followed by anatomical (perirenal fluid collections, loss of corticomedullary junction) and functional (decrease in renal blood flow, hypertension) complications [1, 2]. In addition, another study [3] showed that proteinuria, reaching the nephrotic range in some cases, appeared immediately after ESWL, without changes in glomerular filtration rate; proteinuria disappeared 3-6 months after ESWL. However, it was not described in this study whether these proteinuria excretions were accompanied or not by the development of complete nephrotic syndrome (hypoproteinemia and hypoalbuminemia, hyperlipidemia, edema).

We report on a 62-year-old woman that developed a severe nephrotic syndrome after ESWL for a single stone in the left kidney. Serum creatinine before ESWL was 0.9 mg/dl (79 $\mu\text{mol/l}$) and proteinuria negative. Her blood pressure was normal, and she did not report previous nephrourological diseases, with the exception of left renal colics. ESWL was technically successful, and an abdominal radiography obtained 7 days after ESWL did not show renal stones. On the 15th day after ESWL the patient noted pedal edema that rapidly increased in the following days. On admission, massive edema with ascites and pleural effusion was found. Serum creatinine was 1.4 mg/dl (123 $\mu\text{mol/l}$), total proteins 4.8 g/dl (48 g/l), albumin 1.4 mg/dl (14 g/l) and cholesterol 578 mg/dl (14.9 mmol/l). Proteinuria was 12.5 g/24 h. Antinuclear antibodies and circulating antibodies against glomerular basement membrane (GBM) were negative, and serum complement fractions were normal. An abdominal echography ruled out urological abnormalities.

A renal biopsy was performed on the 25th day after ESWL. Optical and electron microscopy were normal, with the exception of effacement of visceral epithelial cell foot processes. Immunofluorescence was negative. With these histological data, a diagnosis of adult minimal-change nephrotic syndrome was suspected. However, in the following days proteinuria started to decrease spontaneously, with progressive weight loss and disappearance of edema. Eleven days after renal biopsy (36 days after ESWL) proteinuria was negative. After 10 months of follow-up, the patient is asymptomatic, and proteinuria has remained negative.

Although a fortuitous coincidence between ESWL and nephrotic syndrome cannot be excluded, it should be considered that proteinuria spontaneously disappeared 21 days after the onset of nephrotic syndrome. Spontaneous remissions, without steroid treatment, are exceptional in minimal-change nephrotic syndrome. This evolution, together with the clear chronologic relationship, strongly suggests that ESWL was the precipitating cause of the transient nephrotic syndrome. ESWL might theoretically disturb the permselectivity properties of glomerular capillary walls, leading to an abnormal filtration of macromolecules. Interestingly, 2 cases of anti-GBM glomerulonephritis following ESWL have recently been described [4, 5]. Alteration of the normal GBM structure by ESWL, followed by the formation of anti-GBM antibodies, was suspected as the pathogenic mechanism in these cases. Nephrologists and urologists should be aware of these possible complications of ESWL. In addition, further studies are required to establish the precise incidence and clinical consequences of ESWL-related proteinuria.

References

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