Weight Reduction in Massive Obesity Associated with Focal Segmental Glomerulosclerosis: Another Evidence for Hyperfiltration?

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Dear Sir,

Focal segmental glomerulosclerosis has been described associated to massive obesity [1]. Although its pathogenesis is not clear, hyperfiltration has been advocated as a plausible explanation. We describe a case in which proteinuria was clearly reduced after weight loss without any significant variation in the serum total protein or lipid value.

Case Description

A 39-year-old woman was admitted to our hospital because of respiratory failure. She was a morbid obese woman (150 cm, 117 kg) with a 4-year history of hypertension and noninsulin-dependent diabetes mellitus who showed symptoms of exertional dyspnea, diurnal hypersomnia and obstructive sleep apnea. Relevant laboratory data were: serum creatinine 1.7 mg/dl (150 μmol/l), creatinine clearance 33 ml/min (corrected for lean body weight), triglycerides 84 mg/dl (0.92 mmol/l), cholesterol 284 mg/dl (7.38 mmol/l), proteinuria 9 g/24 h and total protein 64 g/l. Polysomnography was performed and a definitive diagnosis of sleep apnea syndrome was established. The patient was began on a hypocaloric diet and on captopril 25 mg/day. She lost 13 kg and proteinuria was reduced to 7 g/day.

Captopril was discontinued because of intolerance. Evolution in the next few months showed proteinuria in the nephrotic range which worsened proportionally to weight gain (fig. 1) as the patient did not follow the diet at home. Percutaneous renal biopsy under ultrasound control was performed showing focal segmental glomerulosclerosis without immune deposits. The patient was discharged on a treatment with theophylline, and calcium channel blockers for hypertension, and a hypocaloric diet. After a 20-kg weight reduction proteinuria was less than 1 g/24 h, total protein was 7.1 g/l and serum creatinine 1.7 mg/dl.

1	1	1	1	1	1	1
0	4	8	12	16	20	24
Weeks						

Fig. 1. Evolution of different analytical parameters with weight variation.

Discussion

Focal segmental glomerulosclerosis has been described associated to sleep apnea syndrome and massive obesity [2]. Its pathogenesis may be related to glomerular hyperfunction with maladaptive changes leading to glomerular hypertension and finally focal glomerulo-

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sclerosis [3,4], whereas the role played by hypoxia subsequent to sleep apnea is still unclear. What was striking about our case was the close relationship between proteinuria and weight without substantial modifications of serum proteins or lipids. Renal function as measured by creatinine clearance or serum creatinine remained unchanged. We think that although ECA inhibition has proved of some benefit in the treatment of proteinuria of diverse origin [5, 6] and was prescribed for that reason in our patient, it was not the cause of proteinuria reduction as it was interrupted long before the latter took place. We wonder if weight reduction was crucial for hyperfunctioning nephrons so as to allow them reversal of basement membrane structural or functional lesions. If this may represent a living model for hyperfiltration/glomerular hypertension and its consequences, the possibility exists that after reducing the hemodynamic burden some of the lesions may improve. Weight reduction seems therefore not only a logical measure but a definitive goal to pursue in nephrotic syndrome associated with massive obesity.

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