the forearm RC-AVF. After surgical closure of the AVF the blood flow will return to normal values. This relatively low flow may result in stasis and clot formation in the aneurysm sac and, subsequently, thrombosis with distal perfusion impairment and peripheral ischaemia.

Treatment by means of ligation of the aneurysm and creation of an arterial bypass have been shown effective in this case and other reports [5].

In summary, late aneurysm formation, after surgical closure of a dialysis AVF, may occur. Although a high blood flow, shear stress and immunosuppressive therapy may play a major role in the aneurysm formation, the exact mechanism is not clear.

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Dual blockade of renin–angiotensin system in primary focal segmental glomerulosclerosis

Sir,

I encountered an interesting case of primary focal segmental glomerulosclerosis (FSGS) undergoing complete remission of nephrotic-range proteinuria in response to combination therapy with angiotensin-converting enzyme inhibitor (ACEI) and angiotensin-receptor blocker (ARB).

A 65-year-old Caucasian woman was referred for evaluation of new-onset nephrotic syndrome for over a period of >2 months since diagnosis. Her initial blood pressure was 160/90 mmHg and pulse rate was 86/min while physical examination was notable only for 3+ pedal oedema. Her past medical history was notable for essential hypertension, right breast carcinoma, status post-lumpectomy and osteoporosis. Her medications included alendronate for 3 years, atenolol 50 mg daily, nifedipine XR 90 mg daily, clonidine 0.2 mg three times daily, furosemide 40 mg daily and irbesartan 150 mg daily. Laboratory results were as follows: blood urea nitrogen 30 mg/dl, serum creatinine 1.6 mg/dl, serum albumin 2.2 g/dl, total cholesterol 269 mg/dl, LDL cholesterol 138 mg/dl while the rest of the electrolyte values, liver function test and coagulation profile were normal. Hepatitis B surface antigen, hepatitis C antibody, antinuclear antigen, rheumatoid factor, cytoplasmic antinuclear cytoplasmic antibody,
perinuclear antinuclear cytoplasmic antibody and C3 and C4 complement levels were normal. Initial 24 h urine protein was 7.4 g. Random urinalysis was notable for small blood, 2–5 red blood cells per high power field, and 100 mg/dl protein. However, no casts were noted. Serum and urine immunofixation studies were negative for light chains. A sonogram of the kidneys was unremarkable. In view of elevated blood pressure and significant proteinuria, irbesartan dose was increased to 300 mg daily. At this time, the patient underwent kidney biopsy for persistent proteinuria and haematuria. Renal biopsy showed idiopathic focal and segmental glomerulosclerosis with mild mesangial proliferation, without any significant interstitial fibrosis or tubular atrophy.

Two weeks later, in view of persistent proteinuria, she was started on fosinopril, initially at 20 mg daily followed by further increase to 40 mg. Over the following period of 2 months, her proteinuria gradually decreased to 1.5 g while serum albumin stabilized at 3 g/dl. Repeat serum creatinine was 0.9 mg/dl. At the time of her last clinic visit, which was 3 months since her first evaluation, her proteinuria had undergone complete remission while her blood pressure remained well controlled at 110/70 mmHg. She continued to have normal renal function along with normalization of serum albumin and lipid profile. Her current blood pressure medications included only irbesartan 300 mg daily and fosinopril 40 mg daily.

It is well known that combination therapy with ACEI and ARB reduces proteinuria significantly beyond their individual blood pressure-lowering effect in non-diabetic glomerular diseases [1]. Many recent studies have demonstrated a significant effect of dual blockade of the renin–angiotensin system upon proteinuria and lowering of blood pressure in chronic kidney disease [1–4].

However, primary FSGS may continue to have marked proteinuria and progressive renal disease despite usage of ACEI. Hence, steroids and immunosuppressive agents are always warranted in nephrotic primary FSGS with reasonably well-preserved renal function in order to prevent progressive glomerulopathy [5].

Experience with usage of ACEI and/or ARB alone in primary FSGS is limited, possibly because of insignificant effects on the progression of primary FSGS in few studies with a limited number of patients [6,7]. This case represents a rare example of a significant effect of aggressive dual blockade of the renin–angiotensin system on proteinuria and hypertension in primary FSGS in a short period of 3 months. Though it offers a novel approach, at least in patients who have contraindication to usage of steroid and immunosuppressive agents, its long-term effect in this patient remains to be seen.

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