Letters

Preventing end-stage kidney disease: a personal opinion from the Third World

Sir,

I read with great interest the editorial, by Schieppati, Perico and Remuzzi, which was recently published in NDT [1]. I also enjoyed the special note, written by the editors in chief of both NDT and Kidney International, about the importance of the prior scientific paper [2].

As a Latin American nephrologist, I know the socioeconomic and sanitary status of this region. In recent years, an increase in the prevalence and incidence of end-stage kidney disease (ESKD) has been observed in Latin America and around the world. At the same time, we have witnessed the arrival of more expensive new technologies for renal replacement treatment. In consequence, I have become aware of the necessity for a social projection of nephrology [3]. To achieve this it is vital to establish health programmes to prevent or at least lessen the impact of ESKD in our communities, by screening the general population for renal diseases and risk factors for renal function (principally, diabetes and hypertension) [3,4].

It is, indeed, very satisfying to confirm that we are neither alone nor forgotten in this world and that colleagues of regions with elevated standards of development and better sanitary structures than ours, have perceived the magnitude of the problem and have proposed possible solutions. One of the most relevant ideas expressed in the paper was that ‘the magic bullet that is indispensable to cure most of the problem is money’ [1].

I firmly support the creation of a global fund to fight renal diseases. But, as was stated in July 2002 in the Lancet [5], ‘without supporting biomedical and health-sciences research on the problems facing the world’s poor, the long-term value of these global funds will be severely compromised.’ The approach is not easy, it will be large and with a lot of stumbling blocks. However, with determination and tenacity, working together and aiming at the same objective, we can succeed.

I offer my gratitude as a physician, a nephrologist and a researcher in renal epidemiology to the authors of the editorial for their noble purpose. I also thank them as a Latin American and a human being.

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Systemic AA amyloidosis and nephrotic syndrome associated with small cell carcinoma of the bladder

Sir,

Amyloidosis is a well-known cause of nephrotic syndrome and renal failure, which is usually associated with chronic inflammatory disease (e.g. rheumatoid arthritis), chronic infections (e.g. tuberculosis and bronchiectasis) or familial Mediterranean fever (FMF). All of these conditions are associated with elevated circulating levels of serum amyloid A (SAA). SAA is one of the acute phase proteins in inflammation and is a precursor of AA amyloid. AA amyloidosis has also been described in association with some tumours [1–3]. We report here the first case of small cell carcinoma (SCC) of the bladder complicated with nephrotic syndrome and renal failure due to systemic AA amyloidosis.

Case. A 57-year-old male was referred to our hospital because of generalized oedema and intermittent macroscopic haematuria of 2 months duration. He had no history of fever, recurrent infections or chronic inflammatory disease. He had neither a family history nor a clinical presentation compatible with FMF or familial amyloidosis. Systemic physical examination was found to be normal except for marked, pitting oedema on both legs.

The haemoglobin level was 11 g/dl (normochromic normocytic anaemia), the white blood cell count was 6120 mm⁻³ and the platelet count was 254 000 mm⁻³. The erythrocyte sedimentation rate (ESR) (120 mm/h) and C-reactive protein (CRP) (6.93 mg/dl) were elevated, but no source of infection was found. Blood chemistry was as follows: urea 90 mg/dl, creatinine 6 mg/dl, total serum protein 4.2 g/dl, albumin 1.4 g/dl, triglycerides 355 mg/dl, total cholesterol 300 mg/dl, glucose 85 mg/dl, AST 20 U/l and ALT 25 U/l, with normal serum electrolyte levels. Serum protein and immunoelectrophoresis revealed hypoalbuminaemia with no monoclonal gammopathy.

Serum IgA was 349 mg/dl (normal 93.2–445), IgG 1177 mg/dl (normal 802–1760), IgM 125 mg/dl (normal 65–280), C3 89.1 mg/dl (normal 52.6–120) and C4 37.9 mg/dl (normal 20.5–49). The urine was 4⁺ for protein and 10–15 red blood cells were observed per high power field. Twenty-four hour urinary protein excretion was 12 g; Bence-Jones proteinuria was not present. Serological tests for ANA, HBsAg, anti-HBsAg, anti-HCV, anti-HIV, VDRL, p-ANCA, c-ANCA, cryoglobulins, rheumatoid factor as well as tuberculin skin test were all negative.

A computed tomography (CT) of the abdomen and pelvis showed a 4 × 3 cm solid mass on the left lateral bladder wall with no evidence of lymphadenopathy or metastasis. Cystoscopy revealed a solitary friable tumour. Transurethral resection was performed. The histopathological examination

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