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Social disparities, risk factors and chronic kidney disease☆

Disparidad social, factores de riesgo y enfermedad renal crónica

Dear Editor,

The term "health disparities" refers to those differences in health status experienced by different demographic groups that occur in the context of social or economic inequality. Health disparities affect access to services and quality of care, which is reflected by the increase in the morbidity and mortality of chronic diseases.[1]

In countries where medical care for chronic kidney disease (CKD) is not universal, treatment for this disease represents a devastating medical, social and economic problem for patients and their families; thus, the costs of treating this disease are considered as "catastrophic health expenditures." A catastrophic health expenditure can be defined as one where the whole family spends more than 30% of their income to pay for the family's healthcare.[2]

In industrialised countries, CKD disproportionately affects socially disadvantaged groups, such as ethnic minorities and people with a low socioeconomic income.[3] Multiple studies conducted in the United States and Canada have shown a strong association between low socioeconomic status and higher incidence and prevalence and more complications related to CKD. Crews et al.[4] showed that people with a lower socioeconomic status had a 59% greater risk of developing CKD. This association was higher in the black population. Also, residence in poor neighbourhoods was found to be strongly associated with an increased prevalence of CKD.

In Europe, the relationship between socioeconomic status and CKD has been less studied; however, studies in Sweden, the UK and France have also found this association.[5,6]

Unfortunately, there are few studies in industrialising countries like India and Mexico. In these countries, there is a high prevalence of the disease in the socioeconomically disadvantaged population.[7] In Central America, particularly in Nicaragua and El Salvador, there have been reports of a new kidney condition called Mesoamerican nephropathy, which occurs mainly in poor workers who toil in suboptimal working conditions at extreme ambient temperatures and experience long periods of dehydration.[8]

Poverty also adversely affects some of the most important social determinants of health, such as developing healthy habits, getting healthcare in a timely manner and suffering environmental exposure to nephrotoxic agents such as lead, cadmium and arsenic (Table 1).

A higher prevalence of births with low birth weight promotes not only less development in terms of renal mass but also an increased risk of hypertension and CKD; the association of post-streptococcal GN with CKD has also been reported as a risk factor in some populations. Depression, anxiety and increased exposure to addictions also promote the activation of the sympathetic nervous system and an increased release of cytokines that can directly influence the pathogenesis of kidney damage (Fig. 1).[9]

An increased intake of sodium, sweetened beverages and foods with phosphorus has also been reported in this population. In addition, the chances of receiving proper treatment to slow the progression of kidney damage are lower in this population.[10]

A clearer understanding of the situations of vulnerable populations and risk factors in people in the lower socioeconomic strata might allow for designing better public health measures to reduce the burden of kidney disease in this population, since growth of national income per capita does not necessarily mean that the poorest members of society get better access to quality health services.

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Table 1 – Main mechanisms by which poverty leads to the development of chronic kidney disease.

Health behaviour	Access to healthcare	Biological factors	Environmental factors
Lack of information on preventive measures	Lack of access	Low birth weight	Exposure to nephrotoxic agents, e.g. Pb, As and Cd
Lack of knowledge of how to deal with the disease	Geographic distance to health centres	Genetic predisposition	Increased exposure to infectious diseases
Unhealthy habits	Catastrophic health expenditure ("out of pocket")	Inadequate nutrition	Lack of safe drinking water and proper sanitation
Unhealthy work, long hours in the sun and low hydration levels		Less control of chronic diseases	

Source: Adapted from García-García and Jha.[11]

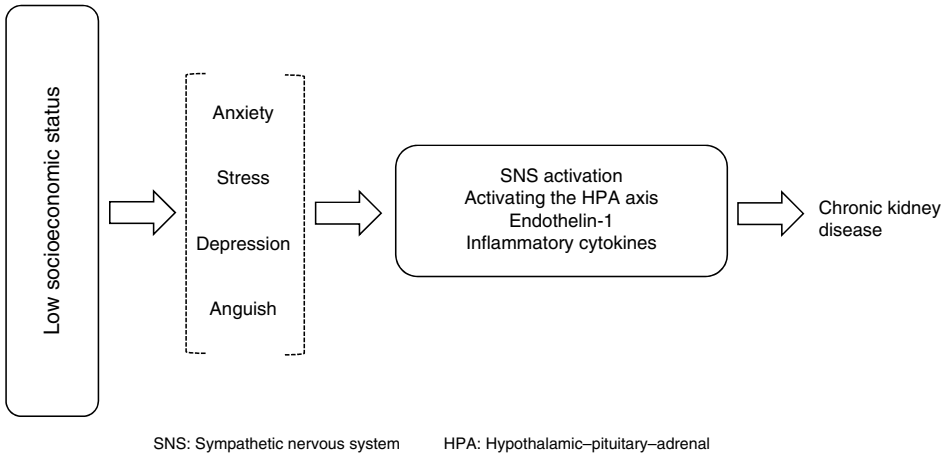

Fig. 1 – Pathophysiological mechanisms linking low socioeconomic status, psychosocial factors and the development of CKD.

Further studies in industrialising countries and studies that provide more information about the pathophysiological mechanisms by which poverty is associated with a higher prevalence of CKD are needed.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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Treatment with sodium thiosulfate in calciphylaxis of topical active renal transplant patient☆

Tratamiento con tiosulfato de sodio tópico en calcifilaxia de paciente con trasplante renal activo

Dear Editor,

Calciphylaxis is a clinical problem with increasing incidence in elderly patients, patients with vascular disease and haemodialysis patients. The treatment of choice should be multidisciplinary.[1,2] The main agent is intravenous sodium thiosulfate (ST); however, it may be poorly tolerated. Here we discuss the possibility of using ST adjuvant treatment.

73-Year-old woman on chronic haemodialysis for end-stage renal failure (diabetic and chronic interstitial nephropathy). Hypertension, hypertensive heart disease, type 2 diabetes mellitus, diabetic neuropathy, chronic revascularised ischaemic heart disease, severe atheromatous of the aorta and visceral branches with mesenteric angina and microscopic colitis, transient ischaemic attack, peripheral vascular disease and severe hyperparathyroidism. Treated with carvedilol, clopidogrel, simvastatin, ezetimibe, omega 3 fatty acids, paricalcitol, cinacalcet, omeprazole, insulin, repaglinide, lanthanum carbonate, transdermal nitroglycerin, oral budesonide, calcifediol, erythropoietin and intravenous iron and carnitine.

Dialysed through a outer humeral gore-tex prosthesis with *online haemodiafiltration*, for 3 h, 4 times a week, with high-permeability polysulfone, anticoagulation with enoxaparin, and calcium bath 1.5 mEq/L.

At 18 months she had a painful macular lesion in the distal third of the right leg, which became ulcerated. Diagnostic biopsy revealed atherosclerotic vascular disease, treated conservatively and stabilised. Five months later she received a kidney transplant hat was technically difficult due to severe calcification. Immunosuppression was induced with basilixiimab, steroids, tacrolimus, and mycophenolic acid, and kidney function stabilised at Cr 1.2 mg/dL (MDRD-IDMS 55 ml/min). In the following weeks the ulcer worsened and 3 more lesions appeared on the legs, which were very painful, necrotic that rapidly worsened. A second biopsy showed calciphylaxis, and so oral bisphosphonate and intravenous ST at doses of 7.5 g/session/2 sessions per week for 4 h were started. She developed metabolic acidosis, severe gastrointestinal intolerance, low blood pressure and general deterioration. The dose of ST was reduced and bed-rest was indicated after each session. No improvement with bicarbonate or ondansetron supplements; so after 50 days of treatment, during which she received 135 g for 18 sessions, the ST iv was suspended and topical ST started (magistral formula: 25%, based on Beeler). Occlusive dressings every 12 h, alternating with debriding ointment (Dertrase®) for 90 days. Now the patient is doing very well, with less pain and with the lesions resolving without significant complications (Figs. 1 and 2).

Calciphylaxis, or uraemic gangrene or calcific uraemic arteriolopathy,[3] is a necrosis of fat caused by hypoperfusion that primarily affects the proximal areas of the legs. The diagnosis was made by biopsy, in which we found necrosis of the epidermis, dermis and hypodermis, intimate fibrodysplasia, micro-thrombosis and calcium deposition in the small arterioles and panniculitis.[2] She presented with livedo reticularis, subcutaneous nodules and plaques that lead to necrosis and cause deep necrotic ulcers.

The following are risk factors for their development[1,2]: kidney failure (1–4% incidence in haemodialysis and 1.3–4.5%

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