



Letters to the Editor

Serum Cystatin C as a Marker of Renal Function in Patients With Systemic Lupus Erythematosus[☆]

Cistatina C sérica como marcador de función renal en pacientes con lupus eritematoso sistémico

To the Editor:

Kidney damage occurs in more than half of patients with systemic lupus erythematosus (SLE).¹ The glomerular filtration rate (GFR) is the most important marker of renal function and crucial for diagnosis, stratification and response to treatment.² The ideal marker of GFR should be constantly produced, be freely filtered, not reabsorbed or secreted by the renal tubules or metabolized or eliminated by extrarenal mechanisms.² Inulin meets these characteristics, however, the measurement of GFR by this method is very complex, expensive and difficult to perform.³ The surrogate marker employed is endogenous creatinine, which does not complete the requirements of an ideal marker because it is subjected to tubular secretion as well as being influenced by the muscle mass of the patient.² Cystatin C is a protein produced by all nucleated cells, freely filtered by the glomerulus, not returned to the bloodstream and not secreted by the renal tubules.^{2,4} The above features make it a better marker of renal function than creatinine,⁵ however, we recognize that there are different factors influencing the levels of cystatin C to be taken into account when performing this test, such as levels of C reactive protein, smoking, obesity, gender, glucocorticoids, age and diabetes.^{6,7} Based on the above, this test could be useful to assess renal function in patients with lupus nephritis (LN) in whom various factors associated with both disease (lupus) and the drugs used could modify the levels of cystatin C.⁸ We included 60 consecutive patients with SLE in the order in which the blood sample was taken to determine levels of creatinine (creatinine standardized by the Roche enzymatic method) and cystatin C (immunonephelometric test particles). Table 1 shows the mean±standard deviation (SD) of the data evaluated in patients, as well as the correlation (Pearson's rho) that existed between cystatin C and the different factors.

We included 5 pediatric patients and 55 (91.7%) were women, 63.3% used prednisone at the time of determination, 36.7% azathioprine, 28.3% mycophenolate mofetil, 53.3% methotrexate, 58.3% chloroquine and 73.3% statins. The correlations that showed statistical significance were creatinine and creatinine clearance. The importance of negative results (no correlation) implies that cystatin C may be a good marker of renal function in patients with lupus

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Table 1

Characteristics of 60 Evaluated Patients.

	Mean±SD	Correlation (r)
Cistatin C	1.16 ± 1.0	NA
Age, years	36.8 ± 15.1	-0.172
Time since onset of SLE, months	85.3 ± 84.6	0.107
MEX-SLEDAI	2.9 ± 3.0	0.237
SCr, mg/dl	1.1 ± 1.5	0.778*
Prednisone, mg/d	8.2 ± 8.8	0.049
Azathioprin, mg/d	32.5 ± 45.5	0.152
Mycophenolate mofetil, mg/d	353.8 ± 630.6	0.239
Methotrexate, mg/week	6.7 ± 6.4	-0.224
Cloroquine, mg/week	562.5 ± 455.2	-0.182
CrC	76.9 ± 27.2	-0.649*

SD, standard deviation; SCr, serum creatinine; CrC, creatinine clearance; MEX-SLEDAI, Mexican Systemic Lupus Erythematosus Disease Activity Index.

* $P < 0.05$.

nephritis, since factors such as disease activity or use of drugs such as glucocorticoids do not modify the renal levels of this marker. On the other hand, it is necessary that studies determine the best equation to determine GFR in patients with LN, as there are different equations for patients with diabetes or chronic kidney disease, but not for rheumatic disease, which have different implications for renal⁹ damage¹⁰. Another important point to consider when estimating renal function is the cost of the test in our country (Mexico), which is on average \$30 (U.S.), a cost 2.5 times greater than creatinine clearance and 8.5 times the creatinine determination only.

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Carpal Arthritis as the Initial Manifestation of Gitelman's Syndrome[☆]

Artritis de carpos como debut en un síndrome de Gitelman

To the Editor:

Gitelman's syndrome is a renal tubular defect and an autosomal recessive disease with metabolic alkalosis, hypokalemia, increased aldosterone and plasma renin with blood pressure in normal range. It maintains a close similarity with Bartter syndrome but unlike the latter hypomagnesemia and hypocalciuria are also persistent. The fundamental change lies in the Na⁺/Cl⁻ cotransporter sensitive to thiazides in the distal tubule.^{1,2}

We report the case of a 43-year-old male with no personal or family history, referred to the outpatient clinics from the hospital emergency room after presenting an acute episode of arthritis of both wrists treated for a few days with low dose corticosteroids. The patient had no previous injuries or infectious symptomatology.

The study of rheumatoid factor and autoantibodies (ANA, ENA, antiCCP) and the serology for human immunodeficiency virus and hepatitis B and C were negative. The acute phase reactants were in the normal range. Radiological examination showed mild degenerative signs of the right third metacarpophalangeal joint with no detectable calcification in the carpal triangular ligament, the symphysis pubis or knees. Incidental findings were a low serum potassium (2.1 mEq/l) and magnesium (1.4 mg/dl).

After study for an electrolyte disorder by the nephrology department, he was diagnosed with Gitelman's syndrome. He was prescribed chronic oral supplements of potassium and magnesium and potassium-sparing diuretics, spironolactone initially, which had to be replaced by epleronone after developing gynecomastia.

It is well known that magnesium is a cofactor of many pyrophosphatases and plasma levels have been associated with calcium pyrophosphate crystal arthropathy. In cases of hypomagnesemia,

it alters the solubility of calcium pyrophosphatase leading to precipitation of crystals at the joint level and producing pseudogout.³

In Gitelman's syndrome there is an increased elimination of magnesium via the kidneys which is not easy to correct. This is because high oral doses usually result in episodes of diarrhea that favor its loss via the gut. In addition, higher intake also correlate with greater urinary losses.³

Our patient had several episodes of arthritis of the wrists despite optimal medical therapy, usually coinciding with periods when plasma magnesium levels were lower. This, together with the absence of other causes explaining the presence of self-limited arthritis, suggests that we find ourselves with a rare form of calcium pyrophosphate crystal arthritis. Although we must emphasize that in this case the diagnosis of the disease is not definitive, we did not analyze the joint fluid for crystals and have not found characteristic⁴ radiological signs.

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