Conflict of interest

The authors declare that they have no conflicts of interest.

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2173-5743/

Response to: Tropical Arthritogenic Alphaviruses☆

Respuesta a: Alfavirus tropicales artritogénicos

To the Editor,

We read the complete review recently reported by Mejía and López-Vélez on tropical arthritogenic alphaviruses with interest.[1] However, based on our experience in Colombia, a country that is significantly affected by arboviruses, such as Chikungunya virus and Zika virus infections, there are certain aspects of the review that should have been mentioned.[2]

Latin America is seeing the emergence of new tropical viral agents, transmitted by arthropod vectors (arboviruses). They have been classified into 4 groups, A, B, C and D, the first 2 being of greater clinical importance.[2] Group A is comprised of the genus alphavirus, involving Chikungunya virus and Mayaro virus, both arthritogenic; whereas group B includes other tropical arthritogenic viruses of other genera and families, also of considerable clinical importance, including their rheumatological manifestations, such is the case of Zika and dengue viruses.[2] The title of the article makes one think that it refers only to arthritogenic alphaviruses, but that does not occur in Table 1, where there is a broad spectrum of viruses that cause musculoskeletal manifestations, which makes one think that the authors are dealing in general with viruses of rheumatological importance. However, in this case, they should have mentioned, for example, Zika virus, which not only provokes arthralgia and other rheumatological manifestations, but has recently been detected directly in synovial fluid, where it can even remain for several weeks.[3]

Speaking specifically of Chikungunya virus, one of the most important arthritogenic alphaviruses in terms of its acute and chronic morbidity and its persistence for long periods of time, in Colombia, between 2014 and 2015, there were more than 3 million new cases, and a proportion of nearly 50% of those patients developed chronic post-Chikungunya inflammatory rheumatism (pCHIK-CIR). This has been documented in a number of cohorts in the departments of Sucre,[4] Tolima[5] and Risaralda,[6] since the beginning of 2016, following along the lines of estimates[7] and meta-analyses of observational studies conducted in other countries.[8] Thus it is surprising that, when discussing pCHIK-CIR, the authors refer only to studies dealing with Reunion Island in France, but there is absolutely no mention of Latin America.

It is important to call attention to the relatively high frequency observed in certain reports, with ranges from 14.4% to 87.2%, as well as a mean persistence of 20.12 months in 47.57% of the patients (95% confidence interval: 45.08–50.13), and a duration of even more than 5 years.[7,9] Thus, pCHIK-CIR has been established as a challenge for Latin American rheumatology.

Finally, it is also surprising that there is such a limited reference to Mayaro virus, especially since, after Chikungunya, this arthritogenic alphavirus could be important not only because of its acute morbidity, but also chronic as well, in many Latin American countries. New outbreaks have recently been reported in Venezuela and in Haiti, among others. Thus, this virus should also be considered in the differential diagnosis.[10]

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☆ Please cite this article as: Rodríguez-Morales AJ, Sánchez-Duque JA, Anaya J-M. Respuesta a: Alfavirus Tropicales Artritogénicos. Reumatol Clin. 2018;14:245–246.

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2173-5743/

Lupus Nephritis Associated With Cytoplasmic Anti-neutrophil Cytoplasmic Antibodies☆

Nefritis lúpica asociada con c-ANCA

To the Editor,

Systemic lupus erythematosus (SLE) and the vasculitides associated with antineutrophil cytoplasmic antibodies (ANCA) are well-differentiated diseases; although approximately a third of the patients with SLE are ANCA-positive,[1] this is rarely associated with vasculitis. The presence of ANCA in patients with SLE seems to predispose them to lupus nephritis (LN), low levels of complement C3 and a higher rate of complications.[2]

We present the case of a 55-year-old man from Mexico City, who presented with a 5-month history of arthralgia in shoulders, wrists and ankles, that was bilateral and symmetrical; moreover, he had episodes of recurrent sinusitis, weight loss and edema arising from the lower limbs; there were no lesions affecting the skin. In the initial examination he underwent urinalysis which showed proteinuria of 200 mg/dL, innumerable erythrocytes, leukocytes 20 to 25 per field, red blood cell and leukocyte casts; creatinine: 1.66 mg/dL (reference range 0.6–1.3 mg/dL). Blood tests revealed hemoglobin concentration of 9.1 g/dL (reference range 13–17 g/dL), normocytic and normochromic; the remaining cell lines were normal; erythrocyte sedimentation rate (ESR): 55 mm/h (reference range 0–15 mm/h); C-reactive protein (CRP): 6.24 mg/dL (reference range 0–3 mg/dL). Immunological profile with a 1:160 titer of antinuclear antibodies (ANA), which had a homogeneous/fine speckled pattern by immunofluorescence and 78.0 IU/mL in enzyme-linked immunosorbent assay (ELISA) (reference range for high positive >60 IU/mL), anti-double stranded DNA (anti-ds DNA) by chemiluminescence: 1.4 IU/mL (reference range for negative <20 IU/mL), antiproteinase 3 (cytoplasmic [c-ANCA]) positive with titers reaching 1:320, granular pattern by immunofluorescence and higher than 100 IU/mL by ELISA (reference range 0–3.5 IU/mL), myeloperoxidase (perinuclear [p-ANCA]) were negative using the same technique, complement C3 was 71.7 mg/dL (reference range 90–180 mg/dL) and complement C4 was 17.6 mg/dL (reference range 10–40 mg/dL); human immunodeficiency virus (HIV) antibodies, cryoglobulins and hepatitis B and C negative; subsequently, 24 h urine was collected and revealed a proteinuria of 1.77 g.

Given the presence of data compatible with nephritic syndrome, the patient underwent renal biopsy. The result was membranoproliferative and active diffuse extracapillary glomerulonephritis (crescents 80%) due to immune complexes, interstitial fibrosis, tubulointerstitial nephritis with mononuclear infiltrate, with no evidence of vasculitis; direct immunofluorescence with deposits of immunoglobulin G (IgG), immunoglobulin A (IgA), immunoglobulin M (IgM), complement C1q, complement C3 with a focal granular pattern (Fig. 1). With these data associated with the clinical presentation, the diagnosis was LN class IV-S (A), with an activity score of 18 and chronicity of 7.

Treatment was begun with 1 g intravenous cyclophosphamide every 30 days for a total of 6 doses, 1 g intravenous methylprednisolone every 24 h the first 3 days, followed by 60 mg of oral prednisone every 24 h for 4 weeks, 24-hour monitoring of proteinuria 1 month after treatment of 870 mg, creatinine of 1.54 mg/dL, at which time a trend toward a partial response was established.[3] The patient continued with cyclophosphamide and steroids were tapered. During follow-up, there was an increase in proteinuria, persistence of dysmorphic erythrocytes at 0–5 per field, monitoring of positive c-ANCA at a titer of 1:160 in immunofluorescence, aside from a reduction in the glomerular filtration rate, with an increase >1 mg/dL in creatinine, leading to renal relapse.[3] Table 1 summarizes the 6-month follow-up. In line with the diagnosis of LN, given the symptoms of chronic sinusitis and positive c-ANCA, we studied a possible associated vasculitis. We performed computed tomography of the paranasal sinuses 20 days after the initiation of treatment (Fig. 2), which revealed swelling of the nasal mucosa, with slight bone erosion in left maxillary antral wall and turbinates justified by the chronic sinusitis process; chest radiograph was normal (Fig. 2C), and a biopsy of the nasal mucosa showed the absence of granulomas or other data suggesting a process of vasculitis.

This case corresponds to LN with failure of the first line of treatment, associated with high titers of positive c-ANCA, with no evidence of solid elements enabling the parallel diagnosis of vasculitis. A number of authors have observed that SLE patients can present these antibodies; that is the case of Galeazzi et al.,[4] who evaluated 566 patients with SLE in 11 European centers, presenting a prevalence of 16.4% (15.4% with p-ANCA and 1% with c-ANCA) in individuals in whom that relationship was detected; other reports demonstrate a highly variable relationship with prevalence of up to 37.3%,[5,6] predominantly with p-ANCA positivity.

We recommend that all the patients with LN should undergo an intentional search for ANCA, because they have a higher positivity rate than SLE patients with no renal involvement; Pradhan et al.[7] recorded a prevalence of 54.5%, and all of these individuals were positive for p-ANCA; subsequently, Pan et al.[2] evaluated 60

☆ Please cite this article as: Tobar-Marcillo M, Destruge-Molina I, Torres-Orozco L, Santiago-Ramirez R. Nefritis lúpica asociada con c-ANCA. Reumatol Clin. 2018;14:246–248.