

distintas enfermedades reumáticas tienen poco valor discriminatorio en las fases iniciales de evolución de la enfermedad, por lo que es necesario modificarlos para que así nos permitan diferenciar estas formas de artritis temprana^{3,6}. Dichos criterios deberían poder clasificar las artritis que permanecerán como indiferenciadas, en las que se ha demostrado que el tratamiento precoz es primordial, aunque no cumplan criterios de una enfermedad establecida^{7,8}. En la práctica, los nuevos criterios de AR y espondiloartritis ya han sido elaborados bajo esta óptica^{9,10}.

Bibliografía

- Guma M, Olive A, Holgado S, Casado E, Lafont A, Tena X. Un sistema de codificación en reumatología. Experiencia de 15 años. *Rev Esp Reumatol*. 2000;27:88-92.
- Decker JL. American Rheumatism Association nomenclature and classification of arthritis and rheumatism. *Arthritis Rheum*. 1983;26:1029-32.
- Narváez García F. Treating undifferentiated arthritis. What, when, how and how long? *Reumatol Clin*. 2009;5 Suppl 1:S31-9.
- Verpoort KN, van Dongen H, Allaart CF, Toes RE, Breedveld FC, Huizinga TW. Undifferentiated arthritis—disease course assessed in several inception cohorts. *Clin Exp Rheumatol*. 2004;5 Suppl 35:S12-7.
- Sampaio-Barros PD, Bortoluzzo AB, Conde RA, Costallat LT, Samara AM, Bértolo MB. Undifferentiated spondyloarthritis: A longterm followup. *J Rheumatol*. 2010;37:1195-9.
- Banal F, Dougados M, Combes C, Gossec L. Sensitivity and specificity of the American College of Rheumatology 1987 criteria for the diagnosis of rheumatoid arthritis according to disease duration: a systematic literature review and meta-analysis. *Ann Rheum Dis*. 2009;68:1184-91.
- Wiles NJ, Lunt M, Barrett EM, Bukhari M, Silman AJ, Symmons DP, et al. Reduced disability at five years with early treatment of inflammatory polyarthritis: Results from a large observational cohort, using propensity models to adjust for disease severity. *Arthritis Rheum*. 2001;44:1033-42.
- Wendling D, Claudepierre P, Prati C. Early diagnosis and management are crucial in spondyloarthritis. *Joint Bone Spine*. 2013;80:582-5.
- Aletaha D, Neogi T, Silman AJ, Funovits J, Felson DT, Bingham CO 3rd, et al. 2010 rheumatoid arthritis classification criteria: An American College of Rheumatology/European League Against Rheumatism collaborative initiative. *Ann Rheum Dis*. 2010;69:1580-8.
- Rudwaleit M, Landewé R, van der Heijde D, Listing J, Brandt J, Braun J, et al. The development of assessment of spondyloarthritis international society classification criteria for axial spondyloarthritis (part I): Classification of paper patients by expert opinion including uncertainty appraisal. *Ann Rheum Dis*. 2009;68:770-6.

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<http://dx.doi.org/10.1016/j.reuma.2014.05.006>

Parvovirus B19 chronic monoarthritis in a patient with common variable immunodeficiency



Monoartritis crónica por parvovirus B19 en un paciente con inmunodeficiencia común variable

Sir,

Human parvovirus B19 infection is mainly associated with erythema infectiosum or fifth disease in children and arthralgia/arthritis in healthy adults.

We describe the case of a patient who presented with chronic monoarthritis of the wrist due to parvovirus B19 and was found to have common variable immunodeficiency (CVID). A 48-year-old man was referred to the hospital because of right wrist monoarthritis lasting six months. Physical examination showed swelling of the right wrist not associated with erythema, warmth or skin lesions. Laboratory analyses showed a white blood cell count of 8300/mm³ (91% granulocytes, 4% lymphocytes), an erythrocyte sedimentation rate (ESR) of 67 mm/h and a C-reactive protein (CRP) of 9.4 mg/l. Serum electrophoresis demonstrated hypogammaglobulinemia: 0.06 g/dl (IgG < 8.6 mg/dl, IgA < 7.8 mg/dl, IgM < 29.8 mg/dl). Rheumatoid factor and antinuclear antibodies were negative. Low values of C3 and C4 were found (64 mg/l and 1.5 mg/l, respectively). Serologic tests for *Borrelia burgdorferi*, hepatitis B and C, rubella and mumps virus, enterovirus, cytomegalovirus, Epstein Barr virus, human immunodeficiency virus and parvovirus B19 were negative. A bone gammagraphy showed marked fixation of the tracer in the right carpus, suggestive of arthritis. A magnetic resonance displayed diffuse synovitis of the right distal radio-cubital joint, with no signs of necrosis or osteomyelitis. A synovial biopsy was performed; histopathological studies showed chronic nonspecific inflammatory changes and polymerase chain reaction (PCR) detected the presence of parvovirus B19 DNA in synovial tissue. PCR of parvovirus B19 DNA in blood was positive.

A diagnosis of parvovirus B19 monoarthritis in the setting of CVID was made, and prompt treatment with intravenous immunoglobulin (0.4 g/kg every 4 weeks) was administered with complete resolution of articular symptoms after the second infusion. Because of hypogammaglobulinemia, treatment with intravenous immunoglobulin was continued indefinitely. Human parvovirus B19 infection is detected in 3.3% of patients examined for acute reactive arthritis.¹ Joint symptoms usually resolve within two weeks; however, 0-17% of patients has chronic arthritis,² generally a symmetric polyarthritis that can resemble rheumatoid arthritis. Chronic monoarthritis is much less frequent. To our knowledge, five cases of parvovirus B19 chronic monoarthritis have been described: three children^{3,4} and two adults.⁵ Diagnosis of B19 infection in immunocompetent individuals is made by detection of B19 specific antibodies in blood. Caution should be made when interpreting serology for parvovirus B19 in immunodeficient patients and pregnant women because of their decreased capacity to mount an antibody response. In these patients, serology should be complemented by PCR analyses of B19 DNA.

In our patient, serologic tests for parvovirus B19 were negative and diagnosis was made by PCR of blood serum and synovial tissue. Even though the presence of parvovirus B19 DNA in synovial tissue does not allow a definite diagnosis, the absence of other causes and the rapid response to intravenous immunoglobulin, led us to assume the viral aetiology of the arthritis.⁶

CVID is a primary immune deficiency characterized by reduced levels of immunoglobulins of all classes despite normal numbers of circulating B cells. The deficiency in IgG production may lead to recurrent infections with encapsulated organisms, such as *Streptococcus pneumoniae* and *Haemophilus influenzae*, causing otitis, sinusitis, bronchitis and bronchiectasis. Septic arthritis with usual bacterial pathogens such as *Staphylococcus aureus*, *S. pneumoniae* and *H. influenzae* and unusual bacteria such as mycoplasma and ureaplasma species, have been described in these patients. Viral arthritis are uncommon; the main causes are adenovirus type I and echovirus 11 and a few cases related to parvovirus B19 have

been reported.⁷ An aseptic non-erosive polyarticular arthritis that resembles rheumatoid arthritis is seen in 10–30% of hypogammaglobulinemic patients. These types of arthritis respond well to immunoglobulin replacement treatment.^{8–10} To our knowledge, only one other case of persistent parvovirus infection leading to a diagnosis of CVID has been reported; it is the case of a 6-year-old boy who presented with a 1-month history of fever and polyarticular arthritis in whom laboratory investigation showed hypogammaglobulinemia. Intravenous immunoglobulin was started 2 weeks into the illness, resulting in prompt recovery.⁷

This case highlights the importance of considering the diagnosis of parvovirus B19 infection not only in cases of polyarthritis but also in any case of unexplained chronic monoarthritis and the special situation of immunodeficient individuals, in whom serologic tests can be negative.

Bibliografía

1. Zerrak A, Bour JB, Tavernier C, Dougados M, Maillefert JF. Usefulness of routine hepatitis C virus, hepatitis B virus, and parvovirus B19 serology in the diagnosis of recent-onset inflammatory arthritides. *Arthritis Rheum.* 2005;53:477–8.
2. Colmegna I, Alberts-Grill N. Parvovirus B19: its role in chronic arthritis. *Rheum Dis Clin N Am.* 2009;35:95–110.
3. Nocton JJ, Miller LC, Tucker LB, Schaller JG. Human parvovirus B19-associated arthritis in children. *J Pediatr.* 1993;122:186–90.
4. Rivier G, Gerster JC, Terrier P, Cheseaux JJ. Parvovirus B19 associated monoarthritis in a 5-year-old boy. *J Rheumatol.* 1995;22:766–7.
5. Lennerz C, Madry H, Ehlhardt S, Venzke T, Zand KD, Mehraein Y. Parvovirus B19-related chronic monoarthritis: immunohistochemical detection of virus-positive lymphocytes within the synovial tissue compartment: two reported cases. *Clin Rheumatol.* 2004;23:59–62.
6. Schmid S, Bossart W, Michel BA, Brühlmann P. Outcome of patients with arthritis and parvovirus B19 DNA in synovial membranes. *Rheumatol Int.* 2007;27:747–51.
7. Adams STM, Schmidt KM, Cost KM, Marshall GS. Common variable immunodeficiency presenting with persistent parvovirus B19 infection. *Pediatrics.* 2012;130:1711–5.
8. Lee AH, Levinson AI, Schumacher HR Jr. Hypogammaglobulinemia and rheumatic disease. *Semin Arthritis Rheum.* 1993;22:252–64.
9. Sordet C, Catagrel A, Schaefferbeke T, Sibilia T. Bone and joint disease associated with primary immune deficiencies. *Joint Bone Spine.* 2005;72:503–14.
10. Samson M, Audia S, Lakomy D, Bonnotte B, Tavernier C, Ornetti P. Diagnostic strategy for patients with hypogammaglobulinemia in rheumatology. *Joint Bone Spine.* 2011;241–5.

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<http://dx.doi.org/10.1016/j.reuma.2014.07.001>