El género *Pseudomonas* se divide filogenéticamente en tres linajes y, al menos, 19 grupos y subgrupos de especies. Uno de los más relevantes es el grupo de especies relacionadas con *Pseudomonas putida*, que comprende hasta 15, y donde está incluida *P. monteilii*[3], descrita en 1997[4]. Fenotípicamente es fácil de encuadrar en este complejo con base en los esquemas de Pickett y Gilardi[5,6], incluyéndose dentro del grupo fluorescente de bacilos gramnegativos (BGN) no fermentadores, junto con *Pseudomonas aeruginosa* y *Pseudomonas fluorescens*. Esta orientación fenotípica tiene aún validez, a pesar de la reclasificación de *Pseudomonas* con base en estudios de homología ARN/ADN y a la caracterización basada en la secuenciación del ARNr 16S[7]. Además, en la práctica rutinaria del laboratorio clínico, *P. monteilii* puede ser identificada de forma rápida y segura mediante espectrometría de masas[8].

*P. monteilii* es un microorganismo ambiental en entornos sanitarios, aislándose con frecuencia en las superficies de los lavabos, grifos y duchas[9]. En este contexto, también debe considerarse un patógeno potencial y, como tal, se ha comunicado su aislamiento a partir de muestras clínicas como aspirados bronquiales, orina, heces, bilis y sangre[4]. Sin embargo, su papel como causante de infecciones en sistema nervioso central (SNC) ha sido escasamente publicado. Por otro lado, la evidencia de infección no está claramente demostrada en muchos de los casos descritos, y podría sugerir que *P. monteilii* podría tener baja patogenicidad, actuar como colonizador, y solo ser fuente de infección en pacientes gravemente enfermos, inmunodeprimidos o portadores de dispositivos biomédicos[10].

## Conclusiones

Con base en lo anterior, el aislamiento de *P. monteilii* estuvo relacionado con una infección de origen nosocomial de la válvula de derivación externa, bien por colonización a partir de la piel del paciente, bien por la manipulación del dispositivo por parte del personal sanitario, sirviendo de puerta de entrada de este microorganismo al líquido ventricular.

## Financiación

Guillermo Martín Gutiérrez disfruta de un contrato de investigación clínica Juan Rodés (JR19/00039) del Instituto de Salud Carlos III, Ministerio de Economía y Competitividad, así como de un contrato Acción B Clínicos Investigadores (B-0006-2019) para el refuerzo de la actividad investigadora en las Unidades de Gestión Clínica del Servicio Andaluz de Salud 2019, Consejería de Salud y Familias.

## Conflicto de intereses

Los autores declaran no tener ningún conflicto de intereses.

## Bibliografía

1. Micó M, Navarro F, de Miniac D, González Y, Brell A, López C, et al. Efficacy of the filmarray blood culture identification panel for direct molecular diagnosis of infectious diseases from samples other than blood. J Med Microbiol. 2015;64:1481–8, http://dx.doi.org/10.1099/jmm.0.000180.
2. EUCAST. Breakpoint tables for interpretation of MICs and zone diameters, Version 10.0, 2020. 2020, http://www.eucast.org.
3. Gomila M, Peña A, Mulet M, Lalucat J, García-Valdés E. Phylogenomics and systematics in Pseudomonas. Front Microbiol. 2015;6:214, http://dx.doi.org/10.3389/fmicb.2015.00214.
4. Elomari M, Coroler L, Verhille S, Izard D, Leclerc H. Pseudomonas monteilii sp. nov., isolated from clinical specimens. Int J Syst Bacteriol. 1997;47:846–52, http://dx.doi.org/10.1099/00207713-47-3-846.
5. Pickett MJ, Pedersen MM. Characterization of saccharolytic nonfermentative bacteria associated with man. Can J Microbiol. 1970;16:351–62, http://dx.doi.org/10.1139/m70-062.
6. Gilardi GL. Characterization of Pseudomonas species isolated from clinical specimens. Appl Microbiol. 1971;21:414–9, http://dx.doi.org/10.1128/aem.21.3.414-419.1971.
7. Ogura K, Shimada K, Miyoshi-Akiyama T. A multilocus sequence typing scheme of Pseudomonas putida for clinical and environmental isolates. Sci Rep. 2019;9, http://dx.doi.org/10.1038/s41598-019-50299-6.
8. Timperio AM, Gorrasi S, Zolla L, Fenice M. Evaluation of MALDI-TOF mass spectrometry and MALDI BioTyper in comparison to 16S rDNA sequencing for the identification of bacteria isolated from Arctic sea water. PLoS One. 2017;12, http://dx.doi.org/10.1371/journal.pone.0181860.
9. De Abreu PM, Farias PG, Paiva GS, Almeida AM, Morais PV. Persistence of microbial communities including Pseudomonas aeruginosa in a hospital environment: A potential health hazard. BMC Microbiol. 2014;14:118, http://dx.doi.org/10.1186/1471-2180-14-118.
10. Scotta C, Juan C, Cabot G, Oliver A, Lalucat J, Bennasar A, et al. Environmental microbiota represents a natural reservoir for dissemination of clinically relevant metallo-β-lactamases. Antimicrob Agents Chemother. 2011;55:5376–9, http://dx.doi.org/10.1128/AAC. 00716-11.

Héctor Toledo, Guillermo Martín-Gutiérrez * y José Antonio Lepe

*Unidad de Enfermedades Infecciosas, Microbiología y Medicina Preventiva, Hospital Universitario Virgen del Rocío, Sevilla, España*

* Autor para correspondencia.
*Correo electrónico:* gmartin-ibis@us.es
(G. Martín-Gutiérrez).

https://doi.org/10.1016/j.eimc.2021.01.003


## Varicella monoarthritis in an immunocompetent woman

### *Monoartritis varicelosa en una mujer inmunocompetente*

*Dear Editor,*

A woman in her early 30s with no relevant medical record came to A&E with a 24-h history of a fever (37.8 °C) and generalized pruritic cutaneous lesions. She presented with vesicles and erythematous papules, some scabby after recent scratching, distributed in torso and extremities. She also mentioned arthralgia in her right knee associated with a discrete swelling without erythema or ecchymosis, which caused her functional impotence. She had no history of previous traumatism or arthritis in the knee. Weight bearing X-ray of the knees revealed an increase in intra-articular fluid and volume of soft tissue.

Peripheral white blood count showed $6.9 \times 10^6/\mu L$ leukocytes [normal values: $3.9–10.2 \times 10^6/\mu L$], with $4.56 \times 10^6/\mu L$ neutrophils, $1.4 \times 10^6/\mu L$ lymphocytes, $12.4 \times 10^6/\mu L$ monocytes—slightly high—[$2.0–9.5 \times 10^6/\mu L$], $1.1 \times 10^6/\mu L$ eosinophils and $0.0 \times 10^6/\mu L$ basophiles. Hepatic enzymes were increased, with AST and ALT values of 121 IU/L [0–40 IU/L] and 187 IU/L [0–35 IU/L) respectively, and a GGT of 254 IU/L [0–38 IU/L] and LDH of 560 IU/L [100–190 UI/L]. RCP had risen to 35.1 mg/L [0–5 mg/L].

Enquiring about her epidemiological environment, her stepson had at that moment the chickenpox. She did not recall contracting it as a child. Suspecting a VZV primary infection with a post-exanthematous arthritis, she started treatment with acyclovir (800 mg IV/4 h) and NSAIDs (dexketoprofen and acetaminophen).

Serology for several microorganisms returned positive IgG for Parvovirus, measles and Herpes simplex virus (HSV) 1/2 whereas their respective IgM were all negative. Both IgM and IgG for HSV, rubella, syphilis and VVZ returned negative. Serology was also negative for HIV.

Before starting with acyclovir, a sample of joint fluid was taken. It was a transparent and yellowish liquid with no crystals but plenty of red blood cells and 4030/mm$^3$ nucleated cells. It had a glucose of 29 mg/dL, total proteins of 5.5 g/dL and 1854/mm$^3$ polymorphonuclear cells (46% neutrophils, 13% lymphocytes, 36% macrophages and 5% mesothelial cells). Bacterial culture after five days was sterile. However, the two different nucleic amplification tests (Clart® Entherpex, Genomica, and VHSI, VHSII and VZV Real Time Progenie Molecular) performed on the synovial fluid confirmed the VZV diagnosis.

After two doses of acyclovir IV and once confirmed the patient was clinically stable, she was discharged. She continued treatment with acyclovir 800 mg/4 h for 7 days. She had a follow-up serology repeated 10 days later: both VZV IgM and IgG returned distinctively positive (Varicella-Zoster ELISA IgM/IgG, Vircell). At the same time, a sample of the skin lesions was taken, and VZV was detected by PCR (Clart® Entherpex, Genomica).

VZV arthritis has a low incidence even in children.[1] In adults, it is usually seen in patients diagnosed with autoimmune diseases and subsequently treated with immunosuppressive therapy.[2] It is not known if the increased risk of VZV complications is due to the immunosuppression, the underlying disease or a combination of both.[3] It is extremely rare for an immunocompetent adult to develop this presentation.[4]

Detection of the virus has been reported before the exanthem but is usually seen at the onset or within the first 72 h.[5] It usually presents with a monoarticular pattern, being the knee the most commonly affected joint,[1] where it produces pain, swelling and limitation of motion.

The pathogenic path of the joint infection is not clear. Several hypotheses have been postulated, including direct invasion of the joint, immune complex formation or immune dysregulation.[6] Isolation of the DNA virus had only been achieved a few times before in the literature.[7]

Our patient presented an IgM rise and subsequent seroconversion. Clinical symptoms were compatible with chickenpox and VZV DNA was detected in the skin lesions and the joint fluid. We can safely assume that the arthritis was reactive to a primary infection by VZV.

Bacterial exclusion must always be performed in VZV acute arthritis since it mimics a septic joint. Septic arthritis is provoked by the haematogenous spread of the bacteria from infected lesions, so it occurs after the onset of the exanthem.[8] Detection of VZV DNA by PCR reduces the time of diagnosis and allows us to rule out septic arthritis. VZV arthritis usually has a short duration and it generally do not persist or recur. However, providing prompt acyclovir treatment should be assessed to see if it reduces the duration of the symptoms.

## ICMJE statement

All authors meet the ICMJE authorship criteria.

## Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

## Conflict of interest

The authors declare that there are no conflicts of interest.

## Bibliografía

1. Bevilacqua S, Poircuitte JM, Boyer L, May T, Lascombes P, Venard V. Varicella arthritis in childhood: a case report and review of the literature. Pediatr Infect Dis J. 2011;30:980–2.
2. Senlis M, Dieudé P, Ottaviani S, Gardette A, Palazzo E, Coustet B. Varicella-Zoster virus hip arthritis in a rheumatoid arthritis patient. Jt Bone Spine. 2016;84:241–2, http://dx.doi.org/10.1016/j.jbspin.2016.02.030.
3. Chakravarty EF. Viral infection and reactivation in autoimmune disease. Arthritis Rheum. 2008;58:2949–57.
4. Evans E, Dawes PT, Mattey DL. An unusual case of adult varicella-associated arthritis [3]. Rheumatology. 2000;39:806–8.
5. Fink CG, Read SJ, Giddins G, Eglin RP. Chicken pox infection (varicella zoster virus) and acute monoarthritis: evidence against a direct viral mechanism. J Clin Pathol. 1992;45:267–9.
6. Moore TL, Schur PH, Romain PL. Viral arthritis: approach to evaluation and management [Internet]. UpToDate Inc.; 2019. Available from: https://www.uptodate.com/contents/viral-arthritis-approach-to-evaluation-and-management?search=pathogenes and diagnosis of viral arthritis&source=search_result&selectedTitle=1~41&usage_type=default&display_rank=1.
7. Stebbings S, Highton J, Croxson M, Powell K, McKay J, Rietveld J. Chickenpox monoarthritis: demonstration of varicella-zoster virus in joint fluid by polymerase chain reaction. Br J Rheumatol. 1998;37:311–3. Available from: https://www.ncbi.nlm.nih.gov/pubmed/?term=(Chichenpox+monoarthritis%3A+demonstration+of+varicella-zoster+virus+in+joint+fluid+by+polymerase+chain+reaction
8. Lobera Gutiérrez de Pando E, García García JJ, González Pascual E. Artritis varicelosa preexantemática. Descripción de un caso. An Esp Pediatr. 1997;46:511–2. Available from: https://www.aeped.es/sites/default/files/anales/46-5-21.pdf

Laura Alonso Acero[a,*], Eloy José Tarín Vicente[b], Elena Sendagorta Cudos[b], María Pilar Romero Gómez[a]

[a] *Microbiology and Parasitology Department, Hospital Universitario La Paz, Madrid, Spain*
[b] *Dermatology Department, Hospital Universitario La Paz, Madrid, Spain*

* Corresponding author.
*E-mail address:* laura.a.acero@gmail.com (L. Alonso Acero).

https://doi.org/10.1016/j.eimc.2020.12.018