

Estos microorganismos son difíciles de identificar con los métodos convencionales ya que son fácilmente confundibles con *Enterococcus* o *Streptococcus viridans*, *Abiotrophia defectiva*, *Lactococcus*, *Leuconostoc* y *Pediococcus*. Además, frecuentemente los urocultivos son falsamente negativos ya que son bacterias de lento crecimiento, nutricionalmente exigentes y anaerobias facultativas, que suelen crecer con CO<sub>2</sub>, y que requieren la experiencia del microbiólogo<sup>1,2</sup>. Actualmente se está usando la espectrometría de masas MALDI-TOF, que ayuda a identificar estos patógenos.

En conclusión, *A. urinae* y *A. sanguinicola* son microorganismos infrecuentes y difíciles de evidenciar, causantes de infecciones genitourinarias en el paciente pediátrico, y probablemente infra diagnosticado. El estudio de su epidemiología, manifestaciones y asociación con patología de base en la población pediátrica permitirá establecer relación con el pronóstico y seleccionar tratamientos adecuados.

## Bibliografía

- Jiménez-Guerra G, Lara-Oya A, Martínez-Egea I, Navarro-Marí J, Gutiérrez-Fernández J. Urinary tract infection by *Aerococcus sanguinicola*. An emerging opportunistic pathogen. *Rev Clin Esp*. 2018;218:351–5.
- Gómez-Luque JM, Foronda-García-Hidalgo C, Gutiérrez-Fernández J. Balanopostitis por *Facklamia hominis* en Pediatría. *Rev Esp Quimioter*. 2019;32:278–80.
- Sous N, Piwoz J, Baer A, Bhavsar S. Subacute *Aerococcus urinae* infective endocarditis with mycotic aneurysms in a pediatric patient: case report and literature review. *J Ped Infect Dis Soc*. 2019;8:492–4.
- Qureshi N, Patel E. *Aerococcus urinae* as the causative agent in infective endocarditis of the aortic valve in a pediatric patient. *Ped Infect Dis J*. 2018;37:1065–6.
- Skalidis T, Papaparaskevas J, Konstantinou D, Kapolou E, Falagas M, Legakis N. *Aerococcus urinae*, a cause of cystitis with malodorous urine in a

child: clinical and microbiological challenges. *JMM Case Rep*. 2017;4:e005083. <http://dx.doi.org/10.1099/jmmcr.0.005083>.

- Lenherr N, Berndt A, Ritz N, Rudin C. *Aerococcus urinae*: a possible reason for malodorous urine in otherwise healthy children. *Eur J Ped*. 2014;173:1115–7.
- Gibb A, Sivaraman B. A second case of foul smelling urine in a boy caused by *Aerococcus urinae*. *Ped Infect Dis J*. 2013;32:1300–1.
- De Vries T, Brandenburg A. Foul smelling urine in a 7-year-old boy caused by *Aerococcus urinae*. *Ped Infect Dis J*. 2012;31:1316–7.
- Murray T, Muldrew K, Finkelstein R, Hampton L, Edberg S, Cappello M. Acute pyelonephritis caused by *Aerococcus urinae* in a 12-year-old boy. *Ped Infect Dis J*. 2008;27:760–2.
- Colakoglu S, Turunc T, Taskoparan M, Aliskan H, Kizilkilic E, Demiroglu Y, et al. Three cases of serious infection caused by *Aerococcus urinae*: a patient with spontaneous bacterial peritonitis and two patients with bacteremia. *Infection*. 2008;36:288–90.

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## Molecular characterization of four Group A *Streptococcus* causing invasive infection in a short time



### Caracterización molecular de cuatro estreptococos del grupo A que causan infección invasiva en un corto periodo de tiempo

Group A *Streptococcus* [GAS] is a community-acquired pathogen causing non-invasive and invasive infections, which are a major cause of morbidity and mortality worldwide.<sup>1</sup> Healthcare-associated infections due to GAS and nosocomial transmission have been well described.<sup>2–4</sup>

Currently the most widely used method for GAS typing is 5' *emm* gene sequencing which encodes the hypervariable region of the M protein, a major virulence determinant of GAS.<sup>5</sup> At least 200 *emm* types have been defined (<https://www.cdc.gov/streplab/groupa-strep/index.html>) being *emm1* and *emm89* the most frequently associated with severe infections in Europe.<sup>6</sup> Several streptococcal pyrogenic exotoxins (Spe) are implicated in the aggressiveness of GAS diseases. *Spe* genes (*speA*, *speB*, *speC*, *speF*, *speG*, *speH*, *speJ*, *speK*, *ssa* and *smeZ*) encode a group of mitogenic proteins secreted by many GAS strains, most of them showing very potent superantigen activity. Toxin-gene profiling is commonly used for GAS characterization.<sup>7</sup>

We report four invasive GAS infection cases in the Hospital Universitario Arnau de Vilanova (Lleida, Spain) in a short time: Patient A was a 65-year-old woman who required ICU admission on April 13, 2019 with septic shock; Patient B was a 67-year-old woman admitted in the ICU on April 26, 2019 with septic shock and pneumonia; Patient C was a 40-year-old immunosuppressed man who were admitted to the Emergency setting on April 28, 2019 with fever;

Patient D was a 82-year-old woman admitted to the Emergency setting on May 28, 2019 with fever and surgical wound infection.

Three strains were isolated from blood samples (strains A, C and D) and one from bronchoalveolar lavage (BAL) (strain B).

GAS antibiotic susceptibility testing was performed with Panel Type 33 of Microscan WalkAway microdilution system using the breakpoints recommended by The European Committee on Antimicrobial Susceptibility Testing.<sup>8</sup>

Because the four invasive GAS cases [three bacteremias (patient A, patient C and patient D) and one pneumonia (patient B)] occurred so closely in time, the isolates were sent to the Centro Nacional de Microbiología, Instituto de Salud Carlos III for M protein gene (*emm*) typing and toxin-gene profiling as previously described.<sup>7</sup>

The four GAS strains were susceptible to penicillin, clindamycin, erythromycin, tetracycline and vancomycin. Toxin-gene profiles and *emm* typing of the four GAS strains are shown in Table 1.

All strains harbored the 3 superantigenic toxin genes: *speB*, *speF* and *speG* regardless of *emm* type. GAS strains with the same *emm* type have shown the same toxin profile (Table 1) coinciding with that described by Bencardino D et al.<sup>9</sup>

**Table 1**

Toxin-gene profiling and *emm* type of the four invasive GAS strains isolated.

Strain	<i>emm</i> type	<i>speA</i>	<i>speB</i>	<i>speC</i>	<i>speF</i>	<i>speG</i>	<i>speH</i>	<i>speJ</i>	<i>ssa</i>	<i>smeZ</i>
A	1	+	+	–	+	+	–	+	–	+
B	1	+	+	–	+	+	–	+	–	+
C	89	–	+	+	+	+	–	–	–	–
D	89	–	+	+	+	+	–	–	–	–

Note. +: present; –: absent.

An identical clone *emm1* was isolated from two of the patients (A and B) coinciding in time in ICU setting (April 26–29, 2019). Strain A was recovered from blood sample obtained in the Emergency Service previously ICU admission. Strain B was isolated from a BAL obtained upon ICU admission. Both patients developed a serious invasive infection with multiorgan failure and were treated with penicillin and clindamycin after GAS isolation. Patient A was discharged from ICU setting after 16 days of admission (April 29, 2019) and patient B died after 4 days of admission (April 30, 2019). A healthcare associated GAS infection has been discarded due to BAL sample of patient B was obtained in the first day of ICU admission.

Strains from patients C and D were an identical clone *emm89*. The emergence of *emm89* strains has been described in several geographic locations.<sup>5</sup> Blood samples of both patients were obtained in the Emergency setting upon admission and patients did not coincide in time in our hospital. Both patients were treated with penicillin and clindamycin after GAS isolation and the evolution of both patients was favorable.

### Conclusions

In our study the GAS clone *emm1* has been related with severe invasive infections and was isolated from two patients who were coinciding in time in ICU setting but nosocomial transmission was discarded, while the GAS clone *emm89* was isolated from two patients with a lighter invasive GAS infection that did not coincide in time in hospital. More molecular studies are needed to describe the molecular epidemiology of GAS infection in our area and the most prevalent GAS circulating clones.

### Conflict of interest

The authors declare no conflict of interest.

### Bibliografía

- Bergin SM, Periaswamy B, Barkham T, Chua HC, Mok YM, Fung DSS, et al. An outbreak of *Streptococcus pyogenes* in a mental health facility: advantage of well-timed whole-genome sequencing over *emm* typing. *Infect Control Hosp Epidemiol.* 2018;39:852–60.

- Cabal A, Schmid D, Lepuschitz S, Stöger A, Blaschitz M, Allerberger F, et al. Nosocomial outbreak of *Streptococcus pyogenes* puerperal sepsis. *Clin Microbiol Infect.* 2019;25:521–3.
- Plainvert C, Longo M, Seringe E, Saintpierre B, Sauvage E, Ma L, et al. A clone of the emergent *Streptococcus pyogenes emm89* clade responsible for a large outbreak in a post-surgery oncology unit in France. *Med Microbiol Immunol.* 2018;207:287–96.
- Lacy MD, Horn K. Nosocomial transmission of invasive group A *Streptococcus* from patient to health care worker. *Clin Infect Dis.* 2009;49:354–7.
- Steer AC, Law I, Matatolu L, Beall BW, Carapetis JR. Global *emm* type distribution of group A streptococci: systematic review and implications for vaccine development. *Lancet Infect Dis.* 2009;9:611–6.
- Friães A, Machado MP, Pato C, Carriço J, Melo-Cristino J, Ramirez M. Emergence of the same successful clade among distinct populations of *emm89 Streptococcus pyogenes* in multiple geographic regions. *mBio.* 2015;6:e01780–1815.
- Schmitz FJ, Beyer A, Charpentier E, Normark BH, Schade M, Fluit AC, et al. Toxin-gene profile heterogeneity among endemic invasive European group A streptococcal isolates. *J Infect Dis.* 2003;188:1578–86.
- The European Committee on Antimicrobial Susceptibility Testing, Breakpoint tables for interpretation of MICs and zone diameters. Version 9.0.2019. <http://eucast.org>
- Bencardino D, Di Luca MC, Petrelli D, Prenna M, Vitali LA. High virulence gene diversity in. *PeerJ.* 2019;7:e6613.

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## Nocardia: la gran simuladora

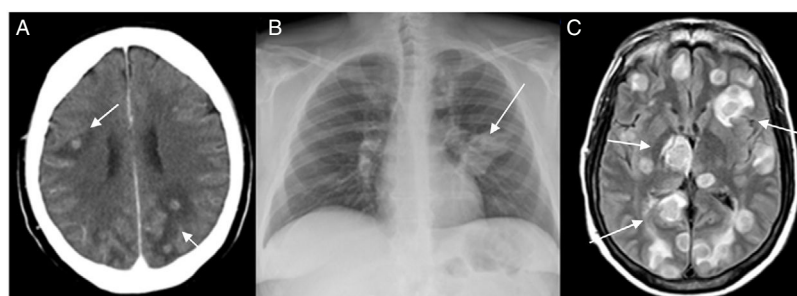


### Nocardia: the great simulator

La infección por *Nocardia* puede manifestarse de múltiples formas, suponiendo un importante reto a la hora de su detección.

Desde lesiones pulmonares hasta intracraneales, puede requerir un diagnóstico diferencial extenso con otras entidades como neoplasia pulmonar metastásica, linfoma, síndrome de Wegener, sarcoidosis, aspergilosis pulmonar o tuberculosis, entre otras.

Paciente de 50 años, hipertenso, diabético y dislipidémico, que acudió a urgencias por amaurosis fugax completa de 10 min de duración, con recuperación posterior con visión borrosa y sensación de pérdida de visión en el hemisferio izquierdo, asociado a cefalea de una semana de evolución. Quince días antes había presentado infección respiratoria con consolidado parahiliar izquierdo por la que había recibido antibioterapia.



**Figura 1.** A) TC craneal donde se objetivan lesiones yuxtacorticales (flechas), en ambos hemisferios cerebrales, con edema perilesional. B) Radiografía de tórax que muestra opacidad parahiliar izquierda (flecha) con probables adenopatías asociadas. C) Resonancia magnética cerebral, secuencia FLAIR, en la que se detectan numerosas lesiones intraparenquimatosas (flechas) hiperintensas en ambos hemisferios cerebrales, de morfología anular, con anillo periférico hipointenso y edema circundante.