is notable for being the first reported case of balanitis caused by *A. urinae*.

These micro-organisms are difficult to identify by conventional methods since they are easily mistaken for *Enterococcus* or *Streptococcus viridans*, *Abiotrophia defectiva*, *Lactococcus*, *Leuconostoc*, or *Pediococcus*. Furthermore, urine cultures often yield false negatives since these are slow-growing, nutritionally demanding, facultative anaerobic bacteria that usually grow with CO_2. Proper identification requires an experienced microbiologist.[1,2] At present, MALDI-TOF mass spectrometry is being used to help identify these pathogens.

In conclusion, *A. urinae* and *A. sanguinicola* are uncommon, difficult-to-identify micro-organisms that cause genitourinary infections in paediatric patients and are probably underdiagnosed. Studying their epidemiology, signs and association with underlying disease in the paediatric population will enable the relationship to the prognosis to be established and suitable treatments to be selected.

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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.[1] Healthcare-associated infections due to GAS and nosocomial transmission have been well described.[2–4]

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.[5] At least 200 *emm* types have been defined (https://www.cdc.gov/streplab/groupa-strep/index.html) being *emm*1 and *emm*89 the most frequently associated with severe infections in Europe.[6] 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.[7]

We report four invasive GAS infection cases in the Hospital Universitari 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.[8]

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.[7]

The four GAS strains were susceptible to penicillin, clindamycin, erythromycin, tetracycline and vancomycine. 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.[9]

An identical clone *emm*1 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

Table 1
Toxin-gene profiling and *emm* type of the four invasive GAS strains isolated.

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

Note. +: present; –: absent.

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 *emm*89. The emergence of *emm*89 strains has been described in several geographic locations.[6] 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 *emm*1 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 *emm*89 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.

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Nocardia: the great simulator☆

Nocardia*: la gran simuladora*

Nocardia infection may manifest in multiple ways, representing a significant challenge when it comes to detecting it.

With signs ranging from pulmonary to intracranial lesions, it may require an extensive differential diagnosis with other diseases such as metastatic pulmonary neoplasia, lymphoma, Wegener's syndrome, sarcoidosis, pulmonary aspergillosis and tuberculosis.

A 50-year-old patient with hypertension, diabetes and dyslipidaemia visited the emergency department due to complete amaurosis fugax with a duration of 10 min, followed by full recovery and blurred vision as well as a sensation of loss of vision in the left hemifield, associated with a headache lasting a week. Two weeks earlier, he had presented respiratory infection with left perihilar consolidation for which he had received antibiotic treatment.

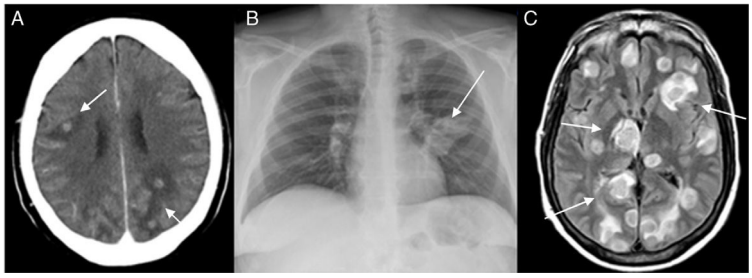

Fig. 1. A) Brain CT scan showing juxtacortical lesions (arrows), in both cerebral hemispheres, with perilesional oedema. B) Chest X-ray showing left perihilar opacity (arrow) with probable associated lymphadenopathy. C) Brain MRI scan, FLAIR sequence, showing numerous ring-shaped hyperintense intraparenchymal lesions (arrows) in both cerebral hemispheres, with a hypointense peripheral ring and surrounding oedema.

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