

with fusidic acid, as well as hygiene measures, with a favourable clinical course and the lesions disappeared at seven days.

Although globally the most common cause of genital lesions is herpes simplex virus infection, in children and non-sexually-active adolescents, other non-infectious and infectious aetiologies should be considered⁵. The importance of detecting any such process, and of ruling out sexual transmission, necessitates a precise aetiological diagnosis. *A. schaalii* is a microorganism occasionally isolated in elderly patients with underlying genitourinary disease, and can be responsible for invasive infections in immunosuppressed patients^{1,2,6}. Of the paediatric cases mentioned previously, five were urinary infections, with one case of intradural abscess and another of balanoposthitis⁴. As in adult patients, there is an association with a history of urogenital disease, which was present in five of the seven cases. Conditions of prolonged moisture in the genital area and anaerobiosis promote its growth. This was associated with four of the seven cases (enuresis, prolonged wearing of nappies or bathing suits). Likewise, our patient suffered from primary enuresis, with a worsening of the clinical picture in the context of prolonged lockdown due to the COVID-19 pandemic. The family described an increase in daytime wetting associated with the stressful situation, which could be considered an adverse effect of the lockdown.

The case presented is, to our knowledge, the first isolation of *A. schaalii* in genital ulcers in either children or adults. Although the presence of this genus has been demonstrated in the urogenital microbiota of asymptomatic patients², the species *A. schaalii* has shown the greatest pathogenic potential. During the patient's initial care in the paediatric emergency department, sending a urine sample for culture was not considered; the urinalysis using a reactive strip was normal, and the patient did not present signs or symptoms compatible with a urine infection other than his underlying enuresis, nor did he show any alterations of infection parameters in blood tests. Nevertheless, following the identification of the microorganism, on reviewing the patient, a microbiological study in urine was requested specifically to search for this microorganism, which was negative. Although in this case other causes of genital ulcers were plausibly ruled out, as the patient showed no other symptoms and presented an adequate response to antibiotic treatment, further case studies are required to fully explain this new association. For the treatment of infections caused by *A. schaalii*, amoxicillin or cephalosporins are recommended, as it is generally resistant to fluoroquinolones and cotrimoxazole⁴.

One limitation of this work is that identification was not performed using a molecular method, although the correct identification of this species with MALDI-TOF has previously been described⁷.

Utility of multiplex PCR syndromic panel for respiratory infections in the diagnosis of acute bacterial meningitis*

Utilidad de un panel sindrómico para infecciones respiratorias en el diagnóstico de meningitis aguda bacteriana

A five-year-old girl, up-to-date on vaccinations and with no history of interest, attended the emergency department due to a clinical picture over the previous 24 h of fever (39.2 °C),

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In conclusion, opportunistic microorganisms such as *A. schaalii* must be taken into account in the differential diagnosis of genital ulcers in patients with urological disease and, in particular, when there is prolonged exposure to moisture in the genital area.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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vomiting, hyporexia, lethargy and headache. She was admitted to paediatrics with progressive worsening at 24 h, left periorbital swelling, tendency to drowsiness and tripod position when attempting to sit. A lumbar puncture and CT were performed, being compatible with acute bacterial meningitis (polymorphonuclear pleocytosis, glucose 4 mg/dl, proteins 106 mg/dl). No microorganisms were visible in gram staining. She was admitted to the ICU, starting empirical antibiotic therapy with cefotaxime (300 mg/kg/day) and vancomycin (60 mg/kg/day), as well as dexamethasone (0.15 mg/kg/day).

A Filmaray® meningitis/encephalitis panel (BioMérieux, France) was conducted and was negative for the microorganisms that usually cause meningitis in our setting. Gram staining of the blood culture revealed clusters of gram-positive cocci. A

Filmarray® respiratory panel (RP2plus) was performed, amplifying *Streptococcus pyogenes* (SPY). The paediatrician was informed, but did not modify the treatment until SPY growth was obtained in cerebrospinal fluid (CSF). A coagulase-negative staphylococcus grew in the blood culture, but was considered a contaminant. Vancomycin was suspended and clindamycin added for nine days as toxic shock was suspected due to hypotension and erythrodermia in the hands and face. On the sixth day after admission, the patient returned to paediatrics, with progressive improvement of her general condition and resolution of the neurological symptoms, with the exception of right peripheral facial paralysis. She was diagnosed with acute otitis media, which gradually resolved after maintaining dexamethasone for four days (0.15 mg/kg/day) and left preseptal cellulitis, for which treatment was initiated with Tobradex® for 10 days.

On the seventh day, she was readmitted to the ICU due to a frontal headache (dexamethasone restarted for 48 h) and hyponatraemia, with findings compatible with cerebral salt wasting syndrome. After eight days of treatment, coinciding with the administration of cefotaxime, she presented an exanthematic pruriginous reaction on the face and trunk, which was alleviated with corticosteroids. Subsequently, she presented a similar reaction with vancomycin, and again with meropenem, likewise alleviated with intravenous corticosteroids. The allergology unit recommended administering vancomycin premedicated with ranitidine and polaramine and avoiding beta-lactams, and 20 days of treatment were completed without skin reactions. At discharge, the physical examination and audiology were normal, with minimal left ptosis. One year later, she had presented no sequelae.

SPY is a gram-positive bacteria that causes a wide spectrum of diseases, with acute pharyngotonsillitis being the most common.¹ It can be responsible for otitis media, sinusitis, and various invasive and immunological infections. On occasions, it can cause meningitis and other intracranial infections, either through direct extension of a contiguous focus or haematogenous dissemination.^{1,2} According to the Centers for Disease Control and Prevention, meningitis due to SPY has a prevalence of 0.06 cases per 100,000 children/year (<0.2% of bacterial meningitis cases)³ with a 43% mortality rate.

Acute bacterial meningitis is a medical emergency, and therefore early diagnosis and treatment are essential in order to avoid a fatal outcome. Given that SPY is a rare cause of pyogenic meningitis in comparison with *Streptococcus pneumoniae* and *Neisseria meningitidis*, empirical therapy with a third-generation cephalosporin plus vancomycin is recommended. Once SPY has been isolated in CSF, penicillin is the antibiotic of choice, although in some cases, such as ours, the cephalosporin is maintained, not always with good clinical progression.^{4,5}

In the last decade, hospitals have incorporated syndromic panels in their diagnostic arsenal, which in the case of acute meningitis detect 14 microorganisms in 70 min. However, when the aetiological agent is not observed in gram staining and is not one of the usual pathogens, it is imperative to resort to other alternatives, such as

a syndromic panel intended for the diagnosis of respiratory infections, in a similar manner to the use of the syndromic panel for sepsis to detect *S. pneumoniae* in pleural fluid⁶ or *N. meningitidis* in blood cultures and CSF.⁷ The meningoencephalitis panel⁸ has also been used to detect *Streptococcus agalactiae* in pleural fluid and *Listeria monocytogenes* in amniotic fluid.

In conclusion, in spite of multiple complications, the patient had a favourable long-term clinical course. Although microbiology used all the tools available to identify the aetiological agent within a few hours without considering the high cost, the early result did not alter the management of the patient. We believe that the barrier of convention needs to be overcome in order to adapt to the new technologies.

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