**Aerococcus viridans** urinary tract infection in a pediatric patient with secondary pseudohypoaldosteronism

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**ABSTRACT**

*Aerococcus viridans* is a catalase-negative gram-positive bacterium rarely found as human pathogen. Some cases of urinary tract infection (UTI) have been described in immunocompromised adults. In this article we describe a UTI case caused by this agent in a child with severe obstructive uropathy, clinically presented with secondary pseudohypoaldosteronism (SPHA). Although *A. viridans* is rarely associated with child infection, it can be responsible for life threatening conditions/situations. To our knowledge, *A. viridans* UTI has never been reported in pediatric patients.

**Key words:** *Aerococcus viridans*, pediatric, urinary tract infection, secondary pseudohypoaldosteronism.

**Case Report**

A 28 day-old boy, with congenital bilateral vesico-urethral reflux grade V, was admitted to hospital presenting failure to thrive, vomiting and dehydration. He manifested severe hyponatraemia, hyperkalaemia and metabolic acidosis. Plasma aldosterone and renin concentration were markedly elevated featuring secondary pseudohypoaldosteronism (SPHA) syndrome. Later, *Enterococcus faecalis* was isolated from the urine culture. Clinical and laboratory features were normalized with antibiotics. After this episode, he received daily trimethoprim for prophylaxis and underwent vesicostomy to relieve the obstructive uropathy.

At 3 months old, he was readmitted in Pediatric Nephrology for vomiting and poor weight gain. Laboratory studies showed mild hyponatraemia (Na+ 125.9 mEq/l), hyperkalaemia (K+ 6.43 mEq/l), metabolic acidosis and slightly azotemia (BUN 45 mg/dl).

Plasma aldosterone (281.2 ng/dl) and renin (1679.0 pg/ml) were once again markedly elevated (normal ranges: aldosterone 5-90 ng/dl and renin 40-220 pg/ml).

Considering the possibility of another episode of SPHA triggered by UTI, fluid and antibiotic therapy (cefuroxime plus gentamicin i.v., according to our institutional guideline) was administered, with clinical improvement.

Meanwhile, the urine culture was worked up by a streaking technique on two plates with artificial media (Columbia colistin-nalidixic acid agar (Columbia CNA, at 35 °C with 5% CO₂) and cystine-lactose electrolyte deficient (CLED, at 35 °C) and a stained smear of a drop of urine was used for microscopy observation. After 24-48 hours of incubation, we obtained a heavy monomicrobial growth (that extends to the fourth quadrant) of small β-hemolytic colonies.
colonies on Columbia CNA agar with negative catalase reaction. The microscopic observation of the Gram stain smear showed leukocytes and pairs and tetrads of gram-positive cocci. The bacterial identification conducted by the automatic method: GPI-Vitek 2 (bioMérieux SA, France) and PostID-Walkaway (Dade-Behring, Germany) identified *A. viridans*.

On the 3rd day of treatment, the urine culture collected by pubic sterile aspiration was repeated. Unfortunately, no antimicrobial susceptibility test was done, but considering the clinical and analytic improvement with cefuroxime and gentamicin association, a ten day course of antibiotics was completed.

Later, on day 9, the internal aldosterone and renin levels showed lower values (190.9 ng/dl and 115.0 pg/ml, respectively)

SPHA is a rare syndrome occurring in early infancy, mainly characterized by very elevated concentrations of aldosterone and renin. It is transient and commonly associated with obstructive uropathy, urinary tract infection (UTI) or both (6).

UTI is a common infection in infancy, being *Escherichia coli* the most common responsible agent in all age groups. Immunocompromised hosts are at risk of infection with less typical agents like *Enterococcus, Pseudomonas aeruginosa* and *Candida albicans* (2).

In our report, the first episode of SPHA was triggered by an *E. faecalis* UTI. In the second SPHA episode, *A. viridans* was isolated from the urine culture. Aerococci are generally saprophytic and frequently considered contaminants in clinical cultures. However, there are some reports of human infection caused by this agent, such as bacteremia, septic arthritis, endocarditis, meningitis, osteomyelitis, empyema and urinary tract infection (7).

In the literature, there are some reports of UTI caused by this agent in adults, but we could not find any reports in children (3, 5). Aerococci appear to have low virulence, only becoming pathogenic in patients with vulnerable conditions, such as immunosupression, chronic disease, malnutrition, prolonged hospitalization, invasive procedures, urinary tract pathology and antibiotic treatment (8). *A. viridans* has indolent growth so its contribution to infectious states may be misdiagnosed (7).

Our patient, besides his early age, also presented a severe urinary tract malformation, a vesicostomy and had been exposed to a long course of antibiotic therapy (for prophylaxis), all potential risk factors for UTI caused by atypical agents.

There is limited data in the literature on the antimicrobial susceptibility of *A. viridans* because this organism has been infrequently associated with human infections and is usually susceptible to penicillin. However, susceptibility patterns have been changing from general susceptibility to the most commonly used antibiotic to recognize resistance not only to penicillin but also to chloramphenicol and quinolones (5). Augustine et al. reported a case of endocarditis caused by multidrug-resistant *A. viridans* (penicillin, ampicillin, cefotaxime, gentamicin, and intermediate resistant to ciprofloxacin) (1). Its susceptibility to second generation cephalosporin remains uncertain.

In our case, the fact of having sterile urine after three days of treatment with cefuroxime and gentamicin, shows good control of the infection; although the actual knowledge defends that with clinical improvement there is no benefit in doing a second urine culture (4). Unfortunately, in spite of the positive response to the treatment, the lack of a susceptibility test did not allow us to determine whether it was due to the use of cephalosporin, aminoglycoside or both.

In conclusion, even though *A. viridans* is rarely associated with child infection, it can be responsible for life threatening conditions, such as SPHA. If this agent is isolated in a culture, especially in immunocompromised or infant patients with obstructive uropathy, we must consider it a real pathogen and antibiotics should be promptly administered.

**REFERENCES**