Aerococcus urinae spondylodiscitis: an increasingly described localization

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ABSTRACT

Aerococcus urinae is currently more frequently identified since the introduction of MALDI-TOF MS technique in routine laboratories. Serious infections such as endocarditis and spondylodiscitis are increasingly reported in the literature.

This is a case of septic spondylodiscitis and bacteremia due to Aerococcus urinae with a urinary starting point.
INTRODUCTION

*Aerococcus urinae* is a Gram-positive bacteria growing in clusters but in contrast to staphylococci they are catalase negative. Due to their similarities with staphylococci, streptococci and enterococci, correct species determination has been difficult in the past and aerococci have been thought to be rare causes of human infection until the recent introduction of matrix-assisted laser desorption ionization time-of-flight mass spectrometry (MALDI-TOF MS).

*Aerococcus urinae* is found in human skin and/or fecal flora and can cause urinary tract infections, or in some cases (elderly with comorbidities) it can cause invasive infections such as sepsis or infective endocarditis, or musculoskeletal infections. We report the case of spondylodiscitis caused by *Aerococcus urinae*.

PATIENT AND OBSERVATION

A 77 years old male presented to the ER with complaints of fever, hematuria and lower back pain. His medical history was significant for a benign prostatic hypertrophy and osteoarthritis; he had no history of smoking, alcohol or drug use and didn’t have any history of nephropathy or any other chronic diseases. He started having hematuria and nocturnal fever three days prior his admission; the back pain appeared only on the morning of his admission.

Upon initial examination the patient was awake and responsive; his vital signs included a temperature of 38.5 °C, heart rate of 94 bpm, oxygen saturation of 97% on room air, and blood pressure of 140/78 mm Hg.

Rheumatologic examination revealed lower lumbar para-vertebral pain, exaggerated by anterior and lateral flexion and spinal stiffness, neurological examination was unremarkable.

Urological examination found a hypertrophied homogenous prostate, urine dipstick was positive for nitrites (+), leukocyte esterase (+), and hemoglobin (++) and was negative for protein, ketone and bilirubin.

The reminder of the physical examination was unremarkable.

Biology showed a high CRP (=98 mg/L), hyperglycemia (6.9 mmol/L), leukocytosis (WBC= 14 300/mm³, PNN=10 000/mm³), urinalysis showed numerous white blood cells (=360/mL), red cells (>1000/mL) and gram-positive cocci arranged in clusters.

Thoracolumbar radiography showed spinal osteoarthritis with spondylolysis and ultrasound showed no abnormalities (no obstructive syndrome or renal mass syndrome). It was concluded that the patient has acute prostatitis, blood culture samples were taken then he was put under empiric antimicrobial therapy (Ceftriaxone) and antalgics (tramadol and paracetamol); he was then transferred to polyvalent medicine department for further investigation.

Urine and blood culture showed small alpha-hemolytic colonies which were identified by MALDI-TOF mass spectrometry system as *Aerococcus urinae*.

Given the persistence of lumbar pain of inflammatory nature, we decided to complete the investigation by performing a thoracolumbar MRI which showed L3/L4 and L5/S1 spondylodiscitis with para-vertebral soft tissue infiltration and epiduritis with no signs of fluid collection.

The patient underwent a percutaneous tomography-guided intervertebral disc biopsy which was performed after seven days of therapeutic window and it was also positive to *Aerococcus urinae*. Susceptibility testing was performed with the disk diffusion method; it came back susceptible to Penicillin 1U, Amoxicillin 20 µg, Ciprofloxacin 5 µg, Levofloxacin 5 µg, and Vancomycin 5 µg.
Endocarditis was ruled out after performing a transesophageal echocardiography.

The patient was diagnosed with *Aerococcus urinae* spondylodiscitis with urinary starting point. He was immediately put under monotherapy with intravenous Amoxicillin (200mg/Kg/day) for 2 weeks, then Amoxicillin (150mg/Kg/day) PO for 6 weeks.

The patient remained in strict decubitus in bed, with rehabilitation and muscle reinforcement until CRP was back to normal and the pain was resolved. Then the patient was discharged after two weeks of IV therapy and continued the oral treatment at a PM&R center.

**DISCUSSION**

*Aerococcus urinae* is a newcomer to clinical and microbiological practice. The first report on *A. urinae* was published in 1989 (1) and the name designated in 1992 (2). Isolates were originally recognized by the cell-morphology (Gram-positive cocci growing in clusters), a negative catalase reaction, Alpha-hemolytic colonies on blood-agar, and a consistent susceptibility to β-lactase and, resistance to sulfonamides and aminoglycosides.

However, the Aerococcus species shared these characteristics with other Gram positive cocci, including Streptococci, Staphylococci, and Enterococci and was therefore often mistaken for these, many studies showed that the true incidence of Aerococci infections is underestimated due to this confusion (3,4,5). Since the emergence of new sophisticated identification systems such as mass spectrometry and genomic sequencing, the incidence of Aerococcus haven’t ceased growing.

The three Aerococcus species which are mostly responsible for human infections are *A. viridans, A. urinae, and A. sanguinocola*. Other Aerococcus species, such as *A. christensenii* and *A. urinae hominis*, have also been described but their pathogenic role in humans still remains uncertain.

*A. urinae* can cause invasive infections, mostly in older males (>65 years old) with underlying urinary tract disease, particularly those with a urinary tract catheter; the most frequently found are bacteriemia followed by infectious endocarditis. Other less common invasive infections have been described; among them, only a few musculoskeletal infections have been documented (6). In a majority of cases, *A. urinae* urinary tract infection was suspected, and although the prognosis is relatively favorable, some fatal cases of infectious endocarditis described have been recorded (7).

To date, six cases of spondylodiscitis due to *Aerococcus urinae* were found in the literature based on a PubMed® research using “Aerococcus” and “Discitis” MeSH.

One case was excluded for the lack of a reliable identification technique (8).

Among these cases, five were men; most of these patients were over 65 years and had comorbidities and almost all of them presented an underlying urinary tract disease.

Spondylodiscitis was always located in the lumbar spine. Regarding the antimicrobial approach, the previous cases were treated mostly by beta-lactams for very different treatment durations from one case to another; ranging from 6 weeks, in our case, to 28 weeks.

The outcome was generally good, the resolution of back pain and the normalization of inflammatory parameters took from 2 weeks to 2 months with a mean of 4.1 weeks. (Table 1)

Reviews about other *A. urinae* invasive infections reported good sensitivity to beta-lactams. However, the beta-lactam-aminoglycoside combination is not entirely clear because, although this combination has been shown to be synergistic in vitro for *A. urinae* isolates,
Rasmussen et al. could only demonstrate this synergy in a few cases (7).

In this way, we treated our case with intravenous Amoxicillin for 2 weeks then switched to oral treatment for 6 weeks. The choice of antibiotic was made based on its side effects and its level of bone penetration.

**CONCLUSION**

In conclusion, spondylodiscitis seems to have become a recurrent localization of *A. urinae* infections in the literature, which suggests that spondylodiscitis should be suspected, especially in older males with previous history of urinary tract disease.

<table>
<thead>
<tr>
<th>Case</th>
<th>Treatment</th>
<th>Total duration</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Astudillo et al. 2003</td>
<td>Amoxicillin + Clindamycin IV/1 month Amoxicillin + Clindamycin PO/1 month Amoxicillin PO /5 months</td>
<td>28 weeks</td>
<td>Resolution of back pain and normalization of inflammatory parameters after 2 months.</td>
</tr>
<tr>
<td>Tekin et al. 2007</td>
<td>Gentamycin IV+ Penicillin G/4 weeks PO antibiotic (no data)/4 weeks</td>
<td>8 weeks</td>
<td>Resolution of back pain after 3 weeks of therapy.</td>
</tr>
<tr>
<td>Torres-Mortos et al. 2017</td>
<td>Ampicillin IV/3 weeks Amoxicillin/5 months</td>
<td>23 weeks</td>
<td>Resolution of back pain after 3 weeks of therapy.</td>
</tr>
<tr>
<td>Degroote et al. 2017</td>
<td>Penicillin G IV/2 weeks Ciprofloxacin + Clindamycin PO/8 weeks</td>
<td>10 weeks</td>
<td>Resolution of back pain after 3 weeks of therapy.</td>
</tr>
<tr>
<td>Rougier et al. 2018</td>
<td>Amoxicillin + Clindamycin IV Levofoxacin + Clindamycin PO</td>
<td>6 weeks</td>
<td>Resolution of back pain and normalization of inflammatory parameters.</td>
</tr>
<tr>
<td>Our case</td>
<td>Amoxicillin IV/2 weeks Amoxicillin PO/6 weeks</td>
<td>8 weeks</td>
<td>Resolution of back pain and normalization of inflammatory parameters after 2 weeks.</td>
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</tbody>
</table>
This emphasizes the importance of a reliable identification technique and calls for a standardization of antibiotherapy and its duration.

Authors’ contributions

Dr. Amina LYAGOUBI: conception, data collection, literature review and writing.
Dr. Chahrazad SOUFFI: supervision and critical review.
Dr. Victoria BAROILLER: data collection and critical review.
Dr. Eric VALLEE: critical review.

REFERENCES