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Case report

Foul smelling urine in an adult caused by *Aerococcus urinae*

Felix Geeraedts [a,*], Carmen Stoffers [a], Henk Smidt [b], Maarten Schijffelen [a]

[a] *Laboratory for Medical Microbiology and Public Health, Hengelo, the Netherlands*
[b] *General Practice de Es, Hengelo, the Netherlands*

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ABSTRACT

We describe the first adult case with *Aerococcus urinae* positive urine cultures as the proven cause of recurrent socially disabling malodorous urine. Bacterial strain specific factors as well as host factors are shown to play a role. The condition can be resolved with proper antibiotics.

Foul smelling urine in adults can have many causes including infection, and is seldom a reason to start treatment with antibiotics. Non-infectious causes may be metabolic disease as for instance trimethylaminuria (fish odor syndrome), ingestion of certain food (asparagus) or drugs, or bad hygiene [1]. Only few cases of foul smelling urine associated with the presence of *Aerococcus urinae* in the patient's urine have been described, either representing colonization [1–3] or urinary tract infection [4], and these were all children. In one previous adult case description *A. urinae* infection, causing carpal and ankle arthritis and bacteremia, was associated with foul smelling urine, but *A. urinae* could not be detected in urine by culture [5]. *A. urinae* is part of the indigenous flora of the genitourinary tract. Infections with *A. urinae* have been described, including urinary tract infection, endocarditis, and bloodstream infection; sporadically peritonitis in patients receiving peritoneal dialysis, vertebral osteomyelitis, soft-tissue infection in the genital area, postpartum infection, and joint infection [6].

Our patient, a 61-year-old male, consulted the general practitioner in February 2019 because of foul smelling urine, which affected his social life and psychological wellbeing. The smell was so penetrating that his wife did not allow him to use the toilet downstairs when having visitors. There were no underlying diseases, or use of medication or specific diets associated with odorous urine (e.g. asparagus). Although there were no symptoms of urinary tract infection, urine was sent to the microbiology laboratory for culture, in which grew *A. urinae*, as was determined by matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (MALDI-TOF MS, Bruker Daltonics GmbH, Bremen, Germany; *A. urinae* highest score of 2.16), in high density (>10^5 c.f.u./ml), susceptible to nitrofurantoin, ciprofloxacin and amoxicillin. No leukocytes were seen in the gram stain. Because *A. urinae* had been associated with foul smelling urine in children previously, the patient was pragmatically treated with nitrofurantoin after which he remained free of complaints for a short time. In May 2019 he returned with the old complaints of malodorous urine. A urine culture again revealed a monoculture of *A. urinae* (>10^5 c.f.u./ml), with the same antibiotic susceptibility pattern. This time both the patient and his wife were treated with amoxicillin for 10 days, because we could not exclude possible recolonization through partner contact. After treatment the patient was free of complaints and control culture of his urine only grew *Enterococcus faecalis* (>10^5 c.f.u./ml). To further assess the role of the *A. urinae* strain of the patient, we performed the following experiment.

Urine samples (3 ml) free of *A. urinae*, proven by culture on solid agar plates, of two control patients (1 and 2), and of our patient, were inoculated with 30 μl of a 0.5 McFarland suspension of one of two control *A. urinae* strains (A and B), or the *A. urinae* strain derived from our patient. Urine samples were then incubated at 37 °C temperature overnight. Hereafter the sample tubes were randomized and blinded for the 3 observers, who scored independently for a malodorous fragrant, as experienced by olfactory perception.

The urine sample with the strongest smell, appointed as such by all 3 observers, was the urine of the patient incubated with the patient derived strain of *A. urinae* (Fig. 1). This smell was according to one of the observers reminiscent of the foul odor of lactic acid. A less strong smell was noted by all 3 observers for the urine samples of the patient incubated with either one of the control *A. urinae* strains (1 and 2). The urine sample of the patient without *A. urinae* was found to produce some smell as compared to both control urines (1 and 2) with or without any of the

* Correspondence to: Laboratorium Microbiologie Twente Achterhoek (LabMicTA), Boerhaavelaan 59, 7555 BB Hengelo, Overijssel, the Netherlands.
E-mail address: f.geeraedts@labmicta.nl (F. Geeraedts).

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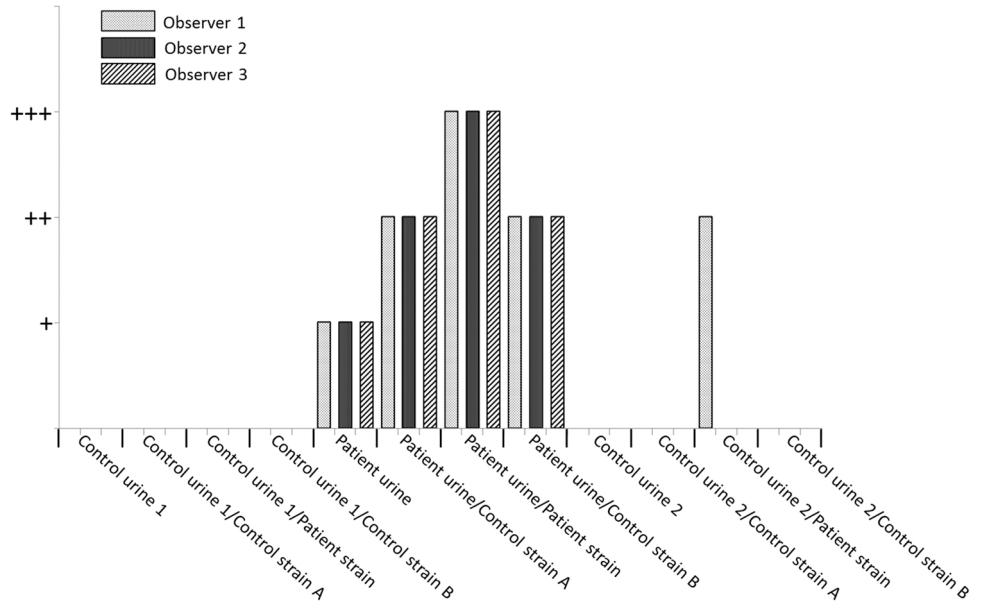

Fig. 1. Olfactory perception of malodorous fragrance produced by urine inoculated with *A. urinae*. Urine of the patient was incubated with either control strains (A or B) or the patient's own strain of *A. urinae*. The same was done for urine of control patients (1 and 2). The scores by each independent observer are given as follows: no smell, some smell (+), smell (++), strong smell (+++).

two control *A. urinae* strains (A and B) or the patient *A. urinae* strain. Only one observer signaled a smell for one of the control urines (2) incubated with the patient *A. urinae* strain.

Our case strongly resembles the two described cases in children, who also had no signs of infection, and where *A. urinae* was the only pathogen isolated from urine [2,3]. In these cases, complaints of malodorous urine completely resolved after a single treatment course of either co-trimoxazole or oral penicillin. Our patient experienced recurrent colonization of the urine with *A. urinae* and concomitant foul smell after treatment with nitrofurantoin. Nitrofurantoin has poor tissue penetration capacity, and may not be the best option for eradication of *A. urinae*. On the other hand, recolonization may have occurred through an outside source**.** We did not provide evidence that his wife was a potential source of recolonization, because she was not tested for the presence of *A. urinae*. Also, the fact that new recurrences were not seen after both were treated with amoxicillin merely leaves open the possibility of recolonization, and does not prove the benefit of partner treatment.

The in vitro experiment clearly shows that *A. urinae* causes malodorous urine in combination with specific components of the patient's urine, and that strain specific properties of *A. urinae* increases the smell. Multiple bacterial pathogens are known to be able to produce volatile metabolites leading to a specific smell, including trimethylamine (TMA) which is the metabolite to produce malodorous urine in the genetic disorder trimethylaminuria (fish odor syndrome) [7]. However, in one of the children's cases TMA levels in urine were not elevated, making trimethylaminuria highly unlikely the cause of the foul smelling urine [1]. Which metabolic pathway is possibly involved in the foul smell produced by *A. urinae*, and why some strains produce a stronger smell than others remains to be resolved. We did not check for differences in growth between the *A. urinae* strains, and therefore cannot exclude that the *A. urinae* strain of the patient produces an increased foul smell by mere multiplicity. It is however recognized that genetic variations within a species may result in different enzymatic efficacy within a specific metabolic pathway, which could also explain the differences in outcome of the strains used in the in vitro experiment [7].

In conclusion, colonization of the urinary tract with *A. urinae* in certain patients can be the cause of foul smelling urine, a condition which may affect the patient's social life but which can be treated effectively with antibiotics. Interaction between patient specific features and bacterial strain specific properties plays a role, which needs further elucidation.

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Ethical approval

Not applicable.

CRediT authorship contribution statement

Felix Geeraedts: Conceptualization, Investigation, Writing – original draft. **Carmen Stoffers:** Investigation, Writing – review & editing. **Henk Smidt:** Investigation, Resources, Writing – review & editing. **Maarten Schijffelen:** Validation, Writing – review & editing.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Declarations of interest

None.

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