To the Editor: A 64-year-old man was admitted to the urology department of Cochin Hospital in Paris, France, for acute urinary retention. He had a history of recurrent urolithiasis and undocumented urinary tract symptoms. At admission, a urethral catheter was inserted, and a plain radiograph showed 2 bladder stones and milk of calcium calcifications. Three days later, he underwent extracorporeal shock wave lithotripsy treatment, and empiric antimicrobial drug therapy with intravenous ceftriaxone, 1 g/day for 3 days, was administered. Three consecutive urinalyses showed a pH of 9, crystals of struvite, hematuria, and 10^5 leukocytes/mL. Gram-positive rods with rounded ends and nonhemolytic. This lipid-requiring strain was catalase positive and strongly urease positive. Testing with the API-Coryne strip (bioMérieux, Marcy l’Etoile, France) showed that the strain was nitrate-reduction positive and produced acid from glucose, ribose, sucrose, and maltose. However, this strain, which was designated CCH052683, did not hydrolyze gelatin or esculin. It was identified as Corynebacterium group F1 (the corresponding numeric profile of the gallery API-Coryne was 3001325).

The strain was correctly identified to the species level as Corynebacterium pseudogenitalium by using polymerase chain reaction and sequencing 16 rRNA as previously described (1,2). Comparison of 785 nucleotides (546–1,331) gave a 16S rDNA similarity value of 99.9% between the sequences of the isolated strain and C. pseudogenitalium ATCC 33039/NCTC11860 (European Molecular Biology Laboratory accession no. X81872).

The strain was sensitive to penicillin, ampicillin, gentamicin, rifampin, vancomycin, teicoplanin, tetracycline, sulfamethoxazole, trimethoprim, fusidic acid, ciprofloxacin, and norfloxacin and resistant to erythromycin, lincomycin, and nitrofurantoin. Ceftriaxone was replaced by norfloxacin (400 mg twice a day) for 1 month. The patient improved and remained healthy 6 months after therapy.

Nondiphtheric corynebacteria are of increasing importance. They have been observed in human specimens, and many new taxa of coryneform bacteria have been described (3). Interest in their taxonomy is increasing, and molecular, phenotypic, and biochemical analyses have resulted in the reclassification of this genus (3). C. pseudogenitalium was described in 1979 by Furness et al. (4) for lipophilic corynebacteria isolated from urinary tract and was not considered a pathogen, in contrast to C. genitalium. However, these 2 species were not included in the official list of recognized species.

C. pseudogenitalium was divided into 5 types based on biochemical patterns, and strains of the type C-5 were differentiated from other types on the basis of urease production. The biochemical and physiologic characteristics of this C-5 type were similar of those of the coryneform group F-1 described by the Centers for Disease Control and Prevention (CDC). In 1995, a comprehensive study on lipophilic corynebacteria demonstrated by DNA-DNA hybridization the similarity between a reference strain of C. pseudogenitalium type C-5 and reference strains of the CDC coryneform group F-1 (1). The CDC group F-1 make up 2 genomic groups at the species level. As shown by 16S rDNA gene comparisons, isolate CCH052683 belongs to the genomic group, including a reference strain of C. pseudogenitalium type C-5 and reference strains of the CDC coryneform group F-1 (1). The CDC group F-1 is represented by strain CDC G5911 (sequence X81904). The molecular genetic investigations identified our isolate as C. pseudogenitalium and placed it in 1 of the 2 genomic groups of CDC group F-1, which cannot be differentiated by biochemical tests (1).

The pathogenicity of this bacterium was associated with strong urease activity. This activity is similar to that of other urease-positive microorganisms, such as C. urealyticum and Proteus spp. (5,6), which infect the urinary tract. Unfortunately, the bladder stones were not analyzed after extracorporeal shock wave lithotripsy treatment. The C. pseudogenitalium
isolate was sensitive to most antimicrobial drugs, particularly β-lactams, aminoglycosides, and quinolones. Thus, urinary tract infections caused by this species of bacteria respond more readily to treatment than those caused by multidrug-resistant *C. urealyticum* (3).

In conclusion, we show that *C. pseudogenitalium* (CDC coryneform group F-1) can cause urinary tract infection (7) and produce urease, and like *C. urealyticum*, cause stone formation in humans. Thus, urease-positive microorganisms isolated by urinalysis that shows urinary alkalization and struvite and pyruvate crystallization should be considered pathogenic. Our results also confirm the difficulty in phenotypic identification of these strains and the need to use a molecular approach to identify coryneform bacteria with clinical relevance.

**References**


Address for correspondence: Gérard Vedel, Service de Bactériologie, Groupe Hospitalier Cochin Saint-Vincent-de-Paul La Roche-Guyon, 75679 Paris, France; fax: 33-1-58-41-15-48; email: gerard.vedel@cch.aphp.fr

---

**Puumala Virus RNA in Patient with Multiorgan Failure**

To the Editor: The hantaviruses (genus *Hantavirus*, family *Bunyaviridae*) include human pathogens and occur worldwide (1). In Western and Central Europe, the predominant serotype is Puumala virus (PUUV), which causes epidemic nephropathy. We report the first Austrian patient with reverse transcription–polymerase chain reaction (RT-PCR)–confirmed PUUV infection and, to our knowledge, the first detection of PUUV-specific RNA in bone marrow.

On April 27, 2004, a previously healthy 52-year-old bus driver stopped his bus because of visual disturbance, dizziness, headache, and weakness in his legs; he then lost consciousness for a few minutes. He was seen at the neurology emergency service and subsequently admitted to the university hospital in Graz. He smoked tobacco, drank beer on the weekends, and cleaned his bus in the garage daily. The patient showed slight paresis of the right leg, nystagmus, cognitive deficit, and retrograde amnesia. Laboratory tests showed increases in (normal values are shown in parentheses) C-reactive protein (CRP) 40 mg/L (<9), creatine kinase (CK) 224 U/L (<170), lactate dehydrogenase (LDH) 244 U/L (<240), and myoglobin 416 ng/mL (<90). Cerebrospinal fluid showed elevated protein of 60 mg/dL (<45) but no other abnormalities. Results of computed tomographic scan of the brain and chest radiograph were normal. Because of increasing CRP (115 mg/L), empiric antimicrobial therapy with pipera-cillin/tazobactam was started. During an electroencephalogram on April 29, the patient deteriorated and was admitted to the intensive care unit for respiratory failure with a partial oxygen pressure of 40 mm Hg; he required intubation and...