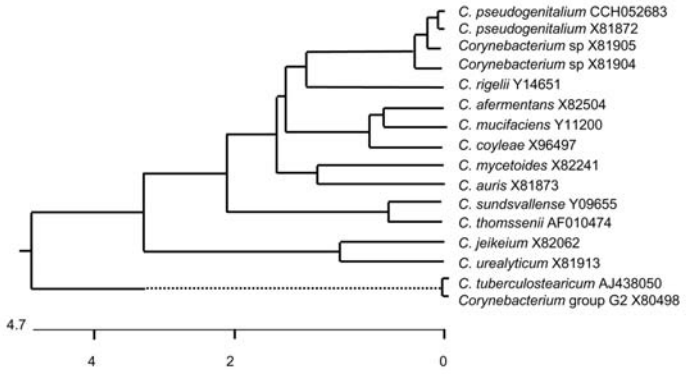

Figure. Unrooted tree showing phylogenetic relationships of *Corynebacterium pseudogenitalium* CCH052683 and other members of the genus *Corynebacterium*. The tree was constructed by using the DNAstar program (DNAstar Inc., Madison, WI, USA) (Clustal method) and based on a comparison of 785 (546–1,331) nucleotides. European Molecular Biology Laboratory sequence accession numbers are shown. The scale bar shows the percentage sequence divergence. Dotted line indicates a distant phylogenetic group for which the scale is not applicable.

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 alkalinization and struvite and pyuria 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.

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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

mechanical ventilation. A chest radiograph showed diffuse pulmonary infiltration and slight bilateral pleural effusion. Laboratory examination showed CRP 265 mg/L, CK 42,570 U/L, LDH 1,235 U/L, myoglobin >3,000 ng/mL, aspartate aminotransferase 368 U/L (<35), alanine aminotransferase 96 U/L (<45), γ-glutamyl transpeptidase 182 U/L (<55), erythrocytes 3.76×10^9/mL, leukocytes 9.09×10^6/mL, thrombocytopenia of 9.2×10^4 platelets/mL, and lymphoplasmacytoid cells on peripheral blood smear. Serum electrophoresis and immunofixation showed an increased γ-globulin fraction with oligoclonal immunoglobulin G (IgG) λ and IgGκ components. A bone marrow biopsy showed hypercellularity and 15% lymphoid cells with plasmocytoid features. Fluorescence-activated cell sorter testing showed 3% reactive B- and T-cell blasts but no signs of a malignant hematologic disease. Culture of bronchoalveolar lavage for bacteria and fungi was negative. Urinary antigen tests for *Legionella* spp. and pneumococci were negative. Serum antibody tests for *Leptospira* spp. were negative, but IgM against PUUV was detected by POC Puumala rapid test (Erilab Ltd, Kuopio, Finland) and recomLine Bunyavirus IgG/IgM test (Mikrogen, Martinsried, Germany). PUUV RNA was detectable in serum and in bone marrow by RT-PCR (*2*). PUUV was confirmed with a bootstrap probability of 99% on phylogenetic analysis (*2*). On May 1, status epilepticus developed and was treated with clonazepam. On May 2, renal function deteriorated and progressed to a maximum serum creatinine concentration of 4 mg/dL (0.6–1.3) and urea of 244 mg/dL (10–45), which required hemodialysis. CRP increased to 360 mg/L, and blood pressure decreased to 95/65 mm Hg. The patient received intensive supportive care including dopamine and norepinephrine. After improvement, the patient was extubated on May 9. Eight days later, fever (temperature up to 40°C), *Enterococcus faecalis* bacteremia, nosocomial pneumonia from methicillin-resistant *Staphylococcus aureus*, respiratory failure requiring mechanic ventilation, and renal failure developed in the patient. Despite antimicrobial drug therapy with linezolid, the patient died 19 days after reintubation.

In Austria, before this case, PUUV RNA had only been detected by RT-PCR in rodents (*2*). We report the first Austrian patient with RT-PCR–confirmed PUUV infection. Furthermore, PUUV-specific RNA had never been detected in bone marrow. In animal studies, PUUV induces production of proinflammatory cytokines, such as interleukin (IL)-6 and IL-10 (*3*). IL-6 constitutes a major growth factor for myeloma and plasma cells, induces immunoglobulin production, and is an active factor in B-cell differentiation (*4*,*5*). IL-10 is a differentiation factor for plasma cell formation and immunoglobulin secretion. Since we detected a clear increase of IL-6, IL-10, and tumor necrosis factor α (TNFα) during the acute phase of infection (IL-6 133.0 pg/dL, IL-10 218.0 U/mL, and TNFα 29.7 pg/mL), we assume that lymphoplasmacytoid cells in bone marrow and peripheral blood of our patient and his production of oligoclonal γ-globulins were due to PUUV-induced cytokine release. Epidemic nephropathy usually takes a benign course, but multiorgan failure with cerebral involvement developed in our patient. Whereas neurologic symptoms such as headache (97% of patients), blurred vision (40%), and vomiting (31%) are common in patients infected with PUUV, only a few cases have been reported with severe central nervous system involvement (i.e., meningitis, epileptiform seizures) (*6*,*7*). Our patient had visual disturbances, slight paresis of the right leg, nystagmus, cognitive deficit, retrograde amnesia, and status epilepticus. We want to draw attention to the severe course PUUV infections can rarely take. The presence of PUUV in bone marrow explains the marked hematologic changes with lymphoplasmacytoid cells in marrow and peripheral blood.

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