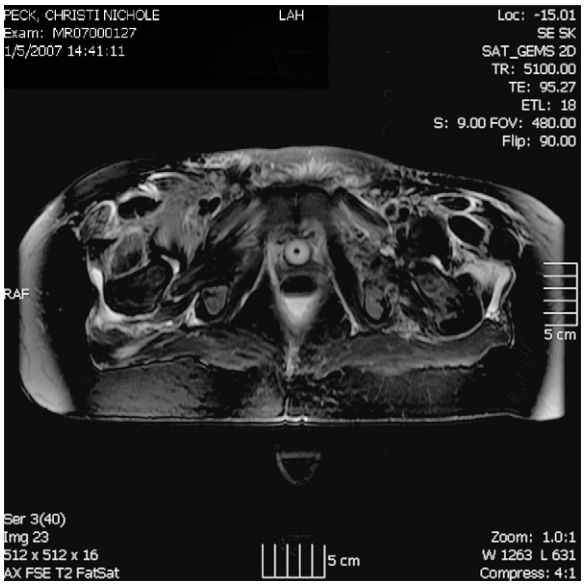

Figure 1 MRI of the pelvis showing pelvic fascial fluid and edema with a hyperintense signal of the fascia surrounding the muscles in the pelvis, suggestive of myofasciitis.

hard to initially evaluate and treat. Necrotizing fasciitis should be considered in the differential diagnosis of severe abdominal pain.

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Hamed A. Shabaneh Al-Tamimi*
Lee Thomas
Department of Internal Medicine,
Division of Gastroenterology,
Texas Tech University Health Sciences Center,
3601 4th Street, Lubbock, Texas 79430, USA

*Corresponding author. Tel.: +1 806 743 3155;
fax: +1 806 743 3148
E-mail address: hamed.shabanehaltamimi@ttuhsc.edu
(H.A.S. Al-Tamimi)

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Septicemia caused by *Corynebacterium macginleyi*: a rare form of extraocular infection

Non-diphtheria *Corynebacterium* species have emerged as nosocomial pathogens and cause diseases in risk populations, such as the immunocompromised and patients with indwelling medical devices.[1] Recognized cases of bacteremia with non-diphtheria *Corynebacterium spp* have become more prevalent.[1] We report herein the first case of *Corynebacterium macginleyi* septicemia in an immunocompromised adult patient, which resulted in death.

A 73-year old male was hospitalized with headaches, disorientation, instability, and vomiting. The patient had previously been diagnosed with non-Hodgkin lymphoma, hypertension, and type 2 diabetes mellitus. Physical examination showed partial disorientation and globally diminished osteomuscular reflexes. The patient had no motor or sensory deficits and had no signs of meningococcal irritation.

Cardiopulmonary and abdominal examinations were normal. An electrocardiogram showed signs of earlier myocardial ischemia. Laboratory studies showed a hemoglobin level of 7.9 g/dl, hematocrit value of 23.6%, and normal platelet and white blood cell counts. Pancreatic and liver function tests were normal. Cerebrospinal fluid (CSF) analysis revealed 10×10^6 cells/l (mononuclear), glycorrhachia 94 mg/l (glycemia 154 mg/dl), and a total protein count of 1.1 g/l. Treponemal tests, *Borrelia burgdorferi*, and *Brucella* serology in samples from serum and CSF were also found to be negative. The results of serological tests for the human immunodeficiency virus were all negative. Herpes simplex virus IgM, cytomegalovirus IgM and IgG, and measles IgM and IgG were negative. However, herpes simplex virus IgG was positive. Computed tomography showed bilateral hypodensity and periventricular lesions. Multiple high-density signals in both cerebral hemispheres, the cerebellum, and pons were enhanced after endovascular contrasts. Urine culture was negative. On the fifth day of hospitalization, blood cultures (three out of three aerobic and anaerobic sets) were found to be positive. Small colonies were observed on the Columbia agar plate supplemented with 5% sheep blood. Colonies consisted of Gram-positive pleomorphic rods. The isolate was sub-cultured again on the same medium with and without 1% (vol/vol) of Tween 80 added at 37 °C in a 5% CO_2 enriched atmosphere. Optimal growth of the strain was shown after 72 hours with Tween 80. Identification was performed with the API Coryne system (bioMérieux, France) together with API Coryne database 2.0 following the manufacturer's instructions. The results identified *Corynebacterium macginleyi* with a 0.87 T value. Culture of the CSF for bacteria was negative. The antimicrobial susceptibility patterns were determined by agar diffusion tests,[2] and the minimal inhibitory concentrations (MIC) were determined by E-test (AB Biodisk, Sweden) on Mueller—Hinton blood agar supplemented with 5% sheep blood (Becton Dickinson). The isolate was sensitive to β-lactams (except penicillin G), glycopeptides, tetracycline, rifampin, and gentamicin but resistant to co-trimoxazole, quinolones, clindamycin, tobramycin, and erythromycin. A corticosteroid, ceftazidime, and clindamycin high-dose treatment was established on the fourth day of admission due to the onset of fever and leukocytosis. The patient deteriorated, showing persistent fever, a diminished level of consciousness, and septic shock. Antimicrobial susceptibility data were not available before the patient's death and therefore effective treatment could not be applied. Necropsy showed an intravascular non-Hodgkin lymphoma with central nervous system and systemic involvement. Septic shock, acute respiratory distress syndrome, and acute myocardial disease (with less than a 48-hour evolution, probably related to hemodynamic deterioration due to previous heart disease) were the final causes of death.

In 1995, Riegel et al.[3] were the first to establish the existence of *Corynebacterium macginleyi*. When this microorganism was first isolated it was defined as an exclusively conjunctival pathogen.[4] However, in 2002, Villanueva et al. published the first case of infection in the urinary tract of a patient with a permanent bladder catheter.[5] In 2003, two more cases of extraconjunctival infection with *C. macginleyi* were documented: one of them was an intravenous catheter-related infection[6] and the other was the causal agent of infectious endocarditis.[7] The number of isolates from conjunctiva swabs ($n = 28$, 90%) suggests that the ocular region is the main habitat for this microorganism, although there has been no systematic examination of this bacterium in any parts of the body other than the throat and conjunctiva.[8,9]

A variety of antibiotic regimens have been used successfully in the management of extraconjunctival cases (glycopeptides,[5] beta-lactams,[6] beta-lactams with aminoglycosides,[7] and beta-lactams with clindamycin in the present report). The susceptibility of the analyzed strains appears to be different. The strain isolated in the catheter-related infection[6] was resistant to a greater number of antimicrobials (beta-lactams, beta-lactams with inhibitors, quinolones, monobactams, gentamicin, macrolides, co-trimoxazole, and lincosamides) and it was only sensitive to vancomycin, netilmicin, and tetracycline. The other three reported strains (one from a urinary tract infection,[5] one the agent of infectious endocarditis,[7] and the one found in the present report) were sensitive to beta-lactams (except for penicillin, in this case report), rifampin, and glycopeptides and they were resistant to quinolones, lincosamides, and co-trimoxazole. With respect to the aminoglycosides, two out of the three cases were resistant to tobramycin and susceptible to gentamicin, while only one case was susceptible to erythromycin.[5] The susceptibility patterns of strains isolated in eye swabs bear a greater resemblance to each other. In a study by Joussen et al.,[10] 66% of the strains were sensitive to chloramphenicol. Giammanco et al.[4] described an isolate that was susceptible to a group of antimicrobials: beta-lactams with and without inhibitors, macrolides, aminoglycosides, glycopeptides, quinolones, lincosamides, rifampin, tetracycline, and chloramphenicol.

Even though the patient described in our case most probably died of intravascular non-Hodgkin lymphoma and acute myocardial diseases, the potential virulence of *C. macginleyi* must be considered when it is isolated in pure culture.

Cases of ocular infections related to *Corynebacterium spp* are probably underreported in the literature. This may be due to underestimation of the possible association between ocular infections and coryneform bacteria. Isolations in pure culture samples, its discovery in patients with indwelling catheters, its presence in sterile tissues and/or urine, and certain clinical signs in immunocompromised patients, should not be underestimated. The increasing number of reported infections with *C. macginleyi* and the continued growth in the number of immunocompromised patients suggests that infection with this pathogen is likely to become more widespread. More information is needed on the factors that predispose patients to systemic infection and on the primary site of infection, as well as other pathogenic properties of *C. macginleyi*. Despite the limited number of isolates reported and the incomplete available data, the literature suggests that vancomycin should be the preferred treatment for non-ocular *C. macginleyi* infections. If glycopeptides cannot be used, the results of susceptibility testing should be used to determine another treatment option.

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Iago Villamil-Cajoto
Department of Internal Medicine, Hospital Clínico Universitario Santiago Compostela, Choupana s/n, Santiago de Compostela, Spain

Luís Rodríguez-Otero*
María Angeles García-Zabarte
Antonio Aguilera-Guirao
Carlos García-Riestra
Benito José Regueiro
Department of Microbiology, Hospital Clínico Universitario Santiago Compostela, Choupana s/n, 15706 Santiago de Compostela, Spain

María José Villacián-Vicedo
Primary Attention of Pediatrics, Hospital Clínico Universitario Santiago Compostela, Choupana s/n, Santiago de Compostela, Spain

*Corresponding author. Tel.: +34 981 950350; fax: +34 981 950369
E-mail address: luis.rodriguez.otero@sergas.es
(L. Rodríguez-Otero)

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Nocardia cyriacigeorgica: pulmonary infection in a patient with Basedow—Graves disease and a short review of reported cases

Nocardia species are aerobic and saprophytic actinomycetes found all around the world. They invade the human body from the environment via trauma and the respiratory tract, and cause cutaneous, pulmonary, and systemic nocardiosis in humans. There is no age, ethnic group, or geographic variation in nocardiosis caused by the different *Nocardia* species, and the male to female ratio for infection is 3 to 1.[1] Nocardiosis is a rare infection and almost half of all cases are immunocompromised subjects.[2—7]

The taxonomy of *Nocardia* species is a complex issue and more than 30 different species have been reported to date.[8] The morphologic and phenotypic characterization of these species is difficult and time-consuming, hence they have been grouped mainly on the basis of their pattern of drug resistance (types I—VI). *Nocardia asteroides* drug pattern type VI has recently been identified as *Nocardia cyriacigeorgica* by use of the 16S rRNA gene amplification method. Therefore, *N. cyriacigeorgica* is not really a new species, but a new name given to a relatively common species.[9,10] This bacterium was originally described by Yassin et al. with the name *N. cyriacigeorgici*;[11] its name was subsequently changed to *N. cyriacigeorgica*.

Few invasive forms of *Nocardia* species have been reported in the literature.[12—22] The case described herein is the first in the literature involving pulmonary *N. cyriacigeorgica* infection in an immunocompromised patient with Basedow—Graves disease. We also review the previously reported cases of *N. cyriacigeorgica* infections in humans.

The case was a 37-year-old man who was started on propylthiouracil treatment, 450 mg daily, for treatment of Basedow—Graves disease. After approximately one year, an orbital computerized tomography (CT) scan was performed, and findings resembling a thyroid orbitopathy were found. He was started on methylprednisolone treatment, 96 mg daily, which was gradually tapered and ceased a month later; he developed diabetes mellitus secondary to corticosteroid treatment, which was controlled with appropriate diet and gliclazide treatment.

In July 2003 following consultation at the ophthalmology department, methylprednisolone treatment, 48 mg daily, was started once more. In September 2003, while under the corticosteroid treatment, he was admitted to