

ESBLs among *Klebsiella* spp. ranged between 0.6% and 1.6%. In 2005 and 2006, the rate of ESBL-producing *Klebsiella* spp. increased to 3.8% (44 isolates) and 4.5% (55 isolates), respectively, and originated mainly from intensive care units (Figure, panel B). In 2005, a single *Klebsiella pneumoniae* isolate showed reduced susceptibility to imipenem (MIC 2 µg/mL) and to meropenem (MIC 4 µg/mL) and resistance to ertapenem (MIC >16 µg/mL). Nevertheless, production of ESBL by *Enterobacteriaceae* organisms is still rare in southeast Austria compared with other European countries (6). However, a dramatic increase of ESBL-producing *E. coli* and *Klebsiella* spp. has been observed during recent years.

The increase of ESBL-producing *E. coli* isolates in outpatients with urinary tract infections leads to serious treatment problems. Results from a recent study indicate that the increase of ESBL-producers in southeast Austria is caused mainly by the emergence of CTX-M-type ESBLs, which are increasingly being isolated from outpatients (7). The *K. pneumoniae* isolate found in 2005 represents the first ESBL-producing isolate not susceptible to carbapenems reported from Austria. Development of resistance to carbapenems in *Enterobacteriaceae* organisms has been reported increasingly, which substantially limits treatment options for persons with multidrug-resistant gram-negative infections (8).

Our data show insignificant changes in prevalence of MRSA and vancomycin-resistant enterococci in southeast Austria during the past decade but an alarming increase of multidrug-resistant ESBL-producing *E. coli* and *Klebsiella* spp. isolates in recent years. Detection of an ESBL-producing *K. pneumoniae* isolate with reduced susceptibility to carbapenems shows that pathogens with new mechanisms of resistance are emerging in this region.

**Alexandra Badura,\*  
Gebhard Feierl,\*  
Harald H. Kessler,\*  
Andrea Grisold,\*  
Lilian Masoud,\*  
Ute Wagner-Eibel,\*  
and Egon Marth\***

\*Medical University of Graz, Graz, Austria

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Address for correspondence: Alexandra Badura, Institute of Hygiene, Medical University of Graz, Universitaetsplatz 4, 8010 Graz, Austria; email: [alexandra.badura@meduni-graz.at](mailto:alexandra.badura@meduni-graz.at)

## Osteomyelitis of Parietal Bone in Melioidosis

**To the Editor:** In Europe and the United States, melioidosis is a rare disease, with no cases reported thus far from Slovenia. However, it is a relatively common disease in certain areas of Southeast Asia and northern Australia. Potentially fatal, this disease is caused by the gram-negative bacillus *Burkholderia pseudomallei*, an environmental organism found in the soil and water of disease-endemic areas. Human infections are mostly acquired through percutaneous inoculation during contact with contaminated water and soil, although inhalation is also a recognized route of acquisition (1). Heavy monsoon rain is associated with severe disease course (2). Melioidosis was reported in some persons injured in the Tsunami in 2004 (3). The disease has a wide spectrum of signs and symptoms (4). Osteomyelitis is a rare manifestation. It occurs in <5% of cases and is a clinical challenge to diagnose and treat (1,4,5).

We describe a case of melioidosis in a previously healthy, 40-year-old Slovenian man. The patient had been working as a basketball trainer in Jordan for the previous 12 months and was traveling to Brunei in mid-summer 2006, 14 days before the illness started. While visiting Brunei, he sustained a minor head trauma when he hit his head on a night table at the hotel. Ten days later, high-grade fever up to 40°C developed, without any other signs or symptoms of disease. After returning to Jordan, the patient was admitted to a local hospital and received different antimicrobial agents without any improvement of his medical condition. After 6 weeks of unsuccessful treatment, he decided to continue medical treatment in Slovenia.

On admission to our hospital, he reported headache and persistent high fever of 6 weeks' duration. Physi-

cal examination indicated high fever (39.5°C) and occipital swelling without any neurologic deficits or other abnormal findings. Initial complete blood cell count, liver function test results, blood urea nitrogen levels, and creatinin levels were normal. C-reactive protein was 60 mg/L, and erythrocyte sedimentation rate was 47 mm/h. Results of chest radiograph and abdominal ultrasound were normal. Results of repeated blood cultures and urinalysis were negative.

The suspected clinical diagnosis was brucellosis (the patient had eaten unpasteurized soft cheese during his stay in Jordan, and brucellosis is endemic in the Middle East). While waiting for *Brucella* spp. tests, we began empirical antimicrobial drug treatment with doxycycline. The patient's condition improved promptly. He became afebrile after 4 days of therapy. In the following week, ultrasound of occipital area soft tissue was performed, and posttraumatic seroma was diagnosed. *B. pseudomallei* was isolated from the seroma on sheep blood agar and identified with VITEK 2 gram-negative identification card (bioMérieux, Mar-

cy l'Etoile, France). The isolate was sensitive to piperacillin, piperacillin-tazobactam, ceftazidime, imipenem, meropenem, and chloramphenicol. It was resistant to aminoglycosides (gentamicin, tobramycin, amikacin, netilmicin), colistin, and polymyxin B. Etest MIC showed susceptibility to doxycycline (MIC 2 µg/mL) and trimethoprim/sulfamethoxazole (TMP/SMX) (MIC 1/19 µg/mL). Susceptibility of *B. pseudomallei* to TMP/SMX was tested with Etest because the disc-diffusion method is inappropriate and can overestimate the extent of resistance (6).

The patient later recalled going on a jungle trip in Brunei the day after his accident. During the trip, he scratched his head, and the skin started to bleed. Thus, he likely inoculated bacteria into the subcutaneous tissue of the head. Fever developed 10 days later.

Computed tomography of the scalp was performed (Figure), and osteomyelitis of the right parietal bone was detected. Magnetic resonance imaging (MRI) excluded involvement of intracranial tissues. Doxycycline was stopped and, as recommended, treat-

ment with ceftazidime and oral TMP/SMX was started. The patient received 8 weeks of intensive parenteral therapy. Once he was discharged, he received 4 months of oral eradication therapy with TMP/SMX and doxycycline. The outcome was excellent. He is now without signs and symptoms of disease, has normal laboratory test results, and has no signs of inflammation on MRI.

Involvement of the skin and soft tissue is common in melioidosis (7). Osteomyelitis is a rare manifestation, usually part of a disseminated infection involving metaphyseal regions of long bones and vertebral bodies. Localized bone involvement is very rare (8). In a Thailand group of 21 patients with musculoskeletal melioidosis, all were initially treated with surgical debridement, followed by long course of antimicrobial therapy (9). A single report of parietal bone osteomyelitis was found in the literature; it was connected to a cerebral abscess due to hematogenous dissemination (10). Because of the specific location of the osteomyelitis (close to the leptomeninges), nonextensive bone damage, and good initial response to antimicrobial therapy, we decided on conservative therapy only.

Melioidosis, although a rare disease, should be considered in the differential diagnosis of any febrile illness in patients returning from disease-endemic regions, especially Thailand and northern Australia. Without special awareness of this possibility, microbiologic laboratories in nonendemic regions could likely misidentify the bacteria and consequently misdiagnose the organism.

**Nina G. Miksić,\*  
Nadja Alikadić,† Tatjana Z. Lejko,†  
Alenka Andlovic,‡  
Jernej Knific,† and Janez Tomažič†**

\*General Hospital Maribor, Maribor, Slovenia; †University Medical Center Ljubljana, Ljubljana, Slovenia; and ‡University Ljubljana, Ljubljana, Slovenia

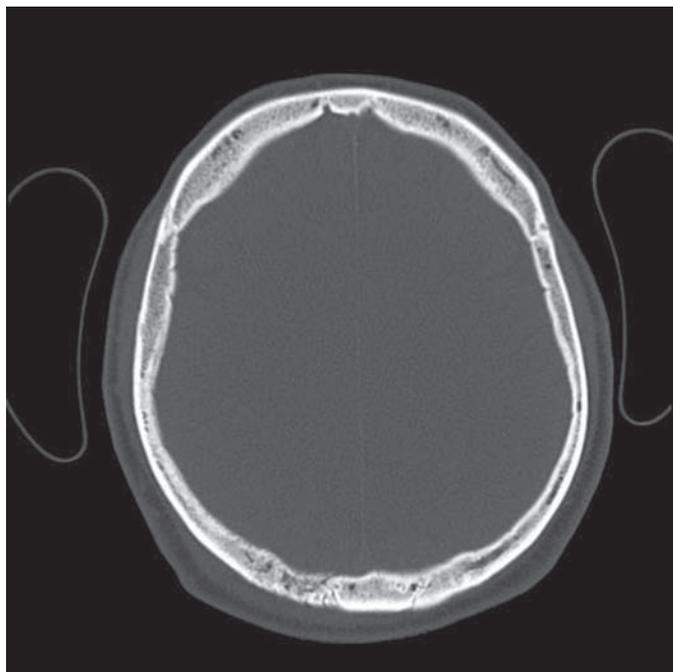


Figure. Axial nonenhanced computed tomographic scan showing moth-eaten appearance of right parietal bone characteristic of osteomyelitis.

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Address for correspondence: Nina Miksić, General Hospital Maribor Ljubljanska 5, 2000 Maribor, Slovenia; email: nina.gorisek@guest.arnes.si



## Chikungunya Fever, Andaman and Nicobar Islands, India

**To the Editor:** The outbreak of chikungunya fever that started in the Indian Ocean Islands in early 2005 (1) spread through adjoining islands and appeared in peninsular India by late 2005 (2). It was first noticed in the southern state of Andhra Pradesh in February 2006; it spread to Tamil Nadu in April 2006 and to Karnataka and Kerala in May. The western state of Gujarat also reported cases in April, but no cases were reported in May and June. The disease again reappeared in July and reached a peak in August. Later it affected the central Indian states of Maharashtra and Madhya Pradesh. In most states, the outbreak declined by October 2006 (3,4).

Andaman and Nicobar Islands, a union territory of India, is an archipelago of >500 islands and islets situated in the Bay of Bengal, 1,200 km from peninsular India. People are constantly moving between mainland India and these islands. Chikungunya fever has previously not been reported from these islands.

During July and August 2006, medical professionals noticed an increase in the number of cases of febrile illness in Port Blair, the headquarters of the union territory and the only urban area in the islands. The total number of patients with fever who visited the 5 urban health centers (UHC) in the town went up from the baseline of 300–450 per day to 550–900 per day in July and August 2006. Most of the patients had associated joint pain. In view of the clinical features suggestive of chikungunya fever, the ongoing epidemic on mainland India, and the widespread presence of the vector, *Aedes aegypti*, within the urban area of Port Blair (5), chikungunya fever was suspected. To confirm this hypothesis, 17 persons who fulfilled the case defi-

inition of having an acute febrile illness associated with severe pain in multiple joints were selected from among the initial patients who went to the UHCs and the referral hospital in Port Blair. Among these study participants, 15 were adults and 2 were adolescents 15 years of age; 6 were female and 11 male. Four adults had febrile illness associated with joint pain; in these patients, weakness of all 4 limbs developed 3–15 days after onset of illness. All of the 4 patients with weakness had areflexic quadriplegia; 1 required ventilatory support. The patients with areflexic quadriplegia were treated with injections of methylprednisolone; all recovered within a week.

Blood samples were collected from these study participants. Serum samples were separated and sent to the National Institute of Virology, Pune, for detection of anti-chikungunya virus (CHIKV) immunoglobulin M (IgM) antibodies. Samples were collected from 12 patients >4 days after the onset of symptoms. In the remaining patients, the interval between onset of symptoms and collection of blood samples was <4 days. Of the 17 study participants, 13 were positive for anti-CHIKV IgM antibodies. Three of 4 samples that were negative for IgM antibodies to CHIKV were collected <3 days after the onset of symptoms. Among these, 2 samples were subjected to reverse transcriptase-PCR by using the primers CHIKV/E1S (5'-TAC CCA TTC ATG TGG GGC-3') and CHIKV/E1C (5'-GCC TTT GTA CAC CAC GAT T-3'), as described by Hasebe et al. (6); both were positive for CHIKV RNA. All these samples were tested for dengue IgM antibodies by using SD Bioline Dengue IgM Rapid Test (Standard Diagnostics Inc., Kyonggi-Do, South Korea), which uses a mixture of dengue recombinant envelop proteins and can detect all of the 4 dengue serotypes. None of the samples tested positive for dengue antibodies. Hence, CHIKV infection was confirmed in 15 of 17 patients.