

multidrug-resistant gram-negative pathogens, and enhanced measures are needed to prevent spread of these organisms. A greater understanding of the modes of spread and acquisition of these organisms is essential for effective control of this problem. We have reported just 1 case of infection with an almost completely resistant gram-negative organism. This case expands the known geographic spread of organisms with this resistance problem. This case also underscores the importance of studying the epidemiology of antimicrobial drug resistance in gram-negative organisms in the rural setting as well as in large metropolitan centers. Dissemination of knowledge regarding appropriate antimicrobial drug susceptibility testing for resistant organisms is also needed.

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## Severe Pneumonia and Human Bocavirus in Adult

**To the Editor:** The newly identified human bocavirus (hBoV), a member of the *Parvovirus* family, is suspected to infect the cells of the respiratory tract and thus may be an etiologic agent of respiratory disease in humans (1). Although Koch postulates have not been fulfilled for hBoV, it appears likely to cause a substantial number of respiratory tract infections, at least in children (2,3). We describe a case of severe atypical pneumonia associated with hBoV DNA in a bronchoalveolar lavage (BAL) sample from an adult.

The patient was a 28-year-old Caucasian woman with an angioimmunoblastic T–non-Hodgkin lymphoma (NHL) that changed into a highly malignant blastic B-cell lymphoma (T-cell-rich B-NHL state I with 70% CD20+ cells, initial stage IIIB). The patient was previously treated with vincristine and prednisone, followed by chemotherapy according to the R-CHOEP-14 protocol (3 cycles) (November 2003 through January 2004). From January through February 2004, chemotherapy was combined with antimicrobial drug therapy according to the R-DHAP protocol (which includes dexamethasone, the chemotherapy drugs cytarabine and cisplatin, and the monoclonal antibody drug rituximab) for persisting symptoms from the B-cell lymphoma. This regimen was followed by a therapy switch to alemtuzumab with ifosfamid, carboplatin, and etoposide (March 2004), which led to a therapy-induced leukopenia, thrombocytopenia, and high fever >40°C by the end of March and the beginning of April 2004. In May 2004, a second round of alemtuzumab with ifosfamid, carboplatin, and etoposide chemotherapy was initiated. In June 2004, a therapy-induced

hemorrhagic cystitis occurred. During July 2004, the patient had ongoing high fever and aplasia of bone marrow with unclear etiology. On July 22, hospital treatment was initiated; it consisted of antimicrobial drug treatment with ceftriaxone (1,000 mg once daily) and gentamicin (320 mg once daily), and antimycotic therapy was started with caspofungin (50 mg once daily).

Since cytomegalovirus (CMV) infection was suspected, ganciclovir (250 mg twice daily) was administered IV for 2 weeks. Although the patient reported an ongoing cough and pneumonialike symptoms, a severe atypical pneumonia that was refractive to antibacterial and antimycotic treatment was diagnosed for the first time during this hospital treatment. Computed tomography scan showed bilateral atypical reticulonodular infiltrations predominant in the lower zones of the lungs (Figure).

The BAL obtained during exacerbation of the pulmonary symptoms was tested for *Mycobacterium tuberculosis*, *Chlamydia pneumoniae*, *Pneumocystis jirovecii*, *Aspergillus* sp., *Candida* sp., *Cryptococcus neoformans*, CMV, Epstein-Barr virus, hepatitis B virus, hepatitis C virus, HIV, herpes simplex virus, and vari-

cella-zoster virus by PCR and culture cultivation. Results were negative, except for a temporarily weak reactivity for *Aspergillus* antigen in serum and for CMV DNA in peripheral blood lymphocytes, which was positive before and became negative after ganciclovir therapy. An archived portion of the BAL was assayed retrospectively by PCR/reverse transcriptase-PCR for human bocavirus, respiratory syncytial virus, human coronaviruses including severe acute respiratory syndrome-associated coronavirus, influenza virus, and human metapneumovirus (hMPV). The only positive result was obtained for human bocavirus, which was confirmed by sequence analysis of the PCR product.

Within a few days, the patient's symptoms decreased, and she was discharged from hospital on day 41, despite ongoing bone marrow aplasia with antimicrobial and antimycotic prophylaxis, including trimethoprim/sulfamethoxazole (160 mg/800 mg once daily) and (voriconazole 200 mg twice daily). Clinical observations led to the primary assumption that the fever, cough, and pulmonary symptoms were likely caused by the postchemotherapeutic extended bone marrow aplasia and CMV infection

accompanied by an unclear bacterial but fungus-typical infection. Retrospectively, however, human bocavirus DNA in the archived BAL strongly suggests that pulmonary symptoms were caused by this agent rather than by a yet unknown bacterial or fungal infection. Thus, in the clinical episode described here, the likely causative agent responsible for the severe pneumonia was the recently described bocavirus.

Respiratory viruses such as respiratory syncytial virus, hMPV, and hBoV seem to be the most prevalent etiologic agents of acute lower respiratory tract infection in children. Recently, evidence of human bocavirus infection was reported for 3.1% to 5.7% of children <3 years of age (1–3). Previously, only limited data on adults, including immunocompromised patients, were available, but the case we describe supports the hypothesis proposed for other newly identified respiratory viruses, namely, that these pathogens also contribute to severe infections in adult patients at high risk. For example, hMPV was found in 3% of stem-cell transplant recipients who underwent BAL because of lower respiratory tract infection (4). In those high-risk patients, hMPV also induced fatal infections (4). This finding led to the conclusion that a “new” virus that induces the identical clinical symptoms, like the human bocavirus, may also contribute to severe respiratory infections. In summary, this first report of a respiratory tract infection with hBoV in an adult immunocompromised patient strongly supports the assumption that hBoV is an emerging pathogen that requires our attention, even for adult patients (1–3).

#### Acknowledgments

We thank Tobias Allander and colleagues for the bocavirus control plasmid and Carola Maiworm for assistance in grammatical revision.



Figure. Computed tomography scan showing reticulonodular infiltrations of both lungs in the lower zones.

This work was partially supported by grants from the Else Kröner-Fresenius-Stiftung (grant no. A 01/05/ /F 00), Herbert-Reeck-Stiftung (Bonn, Germany), and the BONFOR program of the Medical Faculty of the University of Bonn (grant no. O-151.0028).

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## Leishmaniasis in Ancient Egypt and Upper Nubia

**To the Editor:** Leishmaniasis is a disease caused by parasites of the genus *Leishmania*. The infection is transmitted to humans through the bites of female sandflies and manifests mainly in 3 forms: visceral, cutaneous, and mucocutaneous. Visceral leishmaniasis or kala-azar, the often fatal form of the disease, is caused by species of the *Leishmania donovani* complex. These parasites were responsible for severe recent outbreaks in Sudan and other countries and are thought to originate in East Africa (1–4).

In this report, we describe the successful amplification of *L. donovani* DNA in ancient Egyptian and Christian Nubian mummies dating back 4,000 years. Besides the first proof for visceral leishmaniasis in paleopathology, we provide evidence that leishmaniasis was present in Nubia in the early Christian period and that the organism also infected ancient Egyptians, probably because of close trading contacts to Nubia, during the Middle Kingdom. We analyzed 91 bone tissue samples from ancient Egyptian mummies and skeletons and 70 bone marrow samples from naturally mummified human remains from Upper Nubia. The Egyptian material derived from the Pre- to Early Dynastic site of Abydos (n = 7; 3500–2800 BC), a Middle Kingdom tomb in Thebes West (42; 2050–1650 BC), and different tomb complexes in Thebes West, which were built and used between the Middle and New Kingdom until the Late Period (42; c. 2050–500 BC). The Nubian samples were taken before the flooding caused by the Aswan Dam from 2 early Christian burial sites at Kulubnarti, between the second and third cataracts of the Nile River in northern Sudan. One site was on an

island in the Nile and dated from 550 to 750 AD. The other was on the western bank of the Nile and was in use from c.750 to 1500 AD. All samples were tested for *Leishmania* spp. DNA and further characterized by direct sequencing.

In 4 of the 91 Egyptian and 9 of the 70 Nubian samples, a 120-bp fragment of a conserved region of the minicircle molecule of kinetoplastid mitochondrial DNA of the parasite (5,6) could be successfully amplified and, with the first primer pair, unambiguously related to *L. donovani* species after sequencing (Figure). The positive samples from ancient Egypt exclusively originated from the Middle Kingdom tomb, while no molecular evidence for ancient *Leishmania* DNA was found in the Pre- to Early Dynastic and the New Kingdom to Late Period specimens.

In the Middle Kingdom, the Egyptians extended trade relationships and military expeditions to Nubia, the modern Sudan, with particular interest in the gold resources of the country and in obtaining slaves to serve as servants or soldiers in the pharaoh's army. Today, the Sudan is one of the highly endemic countries for visceral leishmaniasis or kala-azar, which is thought to have originated in East Africa and later spread to the Indian subcontinent and the New World (4). Therefore, the high incidence of *Leishmania* DNA in the Middle Kingdom samples (4 [9.5%] of 42) and the lack of findings in earlier or later time periods, may indicate that leishmaniasis was introduced into Egypt at this time. Leishmaniasis did not likely become endemic in the Egyptian Nile Valley because the disease is closely linked to its vector, the phlebotomine sandfly, and the distribution of Acacia-Balanites woodland (7). That ancient Egyptians became infected because of close trade contacts and associated travel with Nubia during the Middle Kingdom seems more plausible. The high frequency of