single-source exposure with C. botu*linum* type B in at least 3 IDUs; this implies that the heroin was obtained from a common source, where contamination with C. botulinum spores may have been introduced when mixed with adulterants or diluted with substances such as dextrose or dyed paper. Skin popping (subcutaneous and intramuscular injection), which may increase the odds of wound botulism by a factor >15 (9), was used by all patients for drug delivery. This study confirms previous observations that the duration of clinical symptoms before antitoxin administration affects the need for and duration of mechanical ventilation (10). Here, the time from hospital admission to antitoxin treatment ranged from several hours to 4 days and correlated with the mechanical ventilation interval ranging from 0 days to 11 weeks. In addition, the extent of abscesses, which ranged from no abscesses to multiple abscesses, seems to affect clinical outcome. As soon as an index case of wound botulism in IDUs is diagnosed, a coordinated public health case-management effort, including hospitals, outpatient clinics, and information centers for drug addicts, is mandatory to alert the medical community and the drug users to consider wound botulism if typical symptoms occur and to enable the prompt administration of antitoxin. Obtaining tissue samples or abscess fluid for culture and molecular epidemiologic studies of C. botulinum isolates is necessary to facilitate identification of the source of the contaminated heroin.

Acknowledgments

We thank our colleagues from Department of Neurology, University of Cologne Medical Center, and the Department of Neurology, Municipal Hospital of Cologne, for providing clinical information, and Danuta Stefanic for excellent technical assistance.

Wiltrud Maria Kalka-Moll,* Ute Aurbach,* Reiner Schaumann,† Rosemarie Schwarz,‡ and Harald Seifert*

*University of Cologne Medical Center, Cologne, Germany; †University of Leipzig, Leipzig, Germany; and ‡Municipal Hospital of Cologne, Cologne, Germany

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Address for correspondence: Wiltrud Maria Kalka-Moll, Institute of Medical Microbiology, Immunology and Hygiene, University of Cologne Medical Center, Cologne, Germany; email: wiltrud.kalka-moll@uk-koeln.de

Multidrug-Resistant Acinetobacter baumannii

To the Editor: In the January 2007 issue of Emerging Infectious Diseases, Sunenshine et al. (1) described their finding of an independent association between patients with multidrug-resistant (MDR) *Acinetobacter* infection and increased hospital and intensive care unit (ICU) length of stay compared with that for patients with antimicrobial drug–susceptible *Acinetobacter* infection. The authors did not, however, find a statistically significant difference in mortality rates between the 2 groups of patients.

Acinetobacter infections frequently occur in severely ill ICU patients with other chronic illnesses or prolonged hospitalizations. We analyzed data for 27 neutropenic cancer patients with A. baumannii-associated bacteremia (15 with MDR and 12 with drug-susceptible A. baumannii infections) but no other chronic illness. We considered A. baumannii strains to be MDR if they were resistant to amikacin, meropenem, and ciprofloxacin. Univariate analysis (Epi Info 2000; Centers for Disease Control and Prevention, Atlanta, GA, USA) showed that most of the bacteremic episodes were associated with certain risk factors, such as catheter insertion, neutropenia, acute leukemia, and previous prophylactic treatment with quinolones or therapeutic treatment with cephalosporins or carbapenems (meropenem or imipenem) (Table).

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Table. Risk factors and outcome for 27 neutropenic cancer patients with bacteremia due to multidrug-resistant (MDR) or drug-susceptible Acinetobacter baumannii infection

		Patients with drug-susceptible	Patients with MDR
	All patients,	A. baumannii,	A. baumannii,
	no. (%)	no. (%)*	no. (%)*
Characteristic	(N = 27)	(n = 12, 44%)	(n = 15, 56%)
Risk for bacteremia			
Central venous catheter	19 (70.4)	9 (75.0)	10 (66.7)
Acute leukemia	11 (40.7)	6 (50.0)	5 (33.3)
Previous prophylaxis with			
quinolones	14 (51.9)	8 (66.7)	6 (40.0)
Previous therapeutic treatment			
with cephalosporins	15 (55.6)	8 (66.7)	7 (46.7)
Previous therapeutic treatment			
with carbapenems	8 (29.6)	4 (33.3)	4 (26.7)
Outcome			
Septic shock	4 (14.8)	2 (16.7)	2 (13.3)
Death	2 (7.4)	1 (8.3)	1 (6.7)
*Insignificant difference between patients with drug-susceptible infection and those with MDR			

infection (p \leq 0.05 by univariate analysis).

Septic shock developed in 4 (14.8%) of the 27 neutropenic patients with A. baumannii-associated bacteremia, and 2 (7.4%) of the 27 died (Table). However, we did not find a statistically significant association between death among patients with bacteremia caused by MDR A. baumanni (1 death) compared with death among those with bacteremia caused by A. baumannii strains susceptible to the carbapenems, ciprofloxacin, and amikacin (1 death) (Table). This finding is similar to that described by Sunenshine et al. (1) in the general ICU population and in neutropenic cancer patients with bacteremia; however, multivariate analysis was not conducted to control for severity of illness and coexisting illness. In conclusion, neutropenic cancer patients with bacteremia due to MDR A. baumannii infection do not appear to be at increased risk for death compared with patients with bacteremia due to antimicrobial drug-susceptible A. baumannii.

This work was supported by grant 06/07/VEGA of the Ministry of Education of Slovak Republic and approved by the Ethics Committee of St Elizabeth Cancer Institute in Bratislava.

Vladimir Krcmery* and Erich Kalavsky†

*St Elizabeth School of Health and Social Sciences, Bratislava, Slovakia; and †Trnava University, Trnava, Slovakia

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Address for correspondence: Vladimir Krcmery, St Elizabeth School of Health and Social Sciences, Department of Oncology, Heydukova 10, 812 50 Bratislava, Slovakia; email: vladimir. krcmery@szu.sk

Letters

Letters commenting on recent articles as well as letters reporting cases, outbreaks, or original research are welcome. Letters commenting on articles should contain no more than 300 words and 5 references; they are more likely to be published if submitted within 4 weeks of the original article's publication. Letters reporting cases, outbreaks, or original research should contain no more than 800 words and 10 references. They may have one Figure or Table and should not be divided into sections. All letters should contain material not previously published and include a word count.

Serogroup X in Meningococcal Disease, Western Kenya

To the Editor: Although >12 different serogroups of Neisseria meningitidis exist, most disease outbreaks across the African meningitis epidemic belt are caused by serogroup A and, less frequently, by serogroups C and W135 (1). N. meningitidis serogroup X was first described in the 1960s and has been found to cause a few cases of invasive disease across North America, Europe, and Africa (2). In Africa, small serogroup X outbreaks have been described in Ghana (9 cases over a 2-year period) and in Niger (134 cases between 1995 and 2000) (3,4). In 2006, however, 51% of 1,139 confirmed cases of meningococcal meningitis in Niger were found to be caused by serogroup X (5). Before the 2005-06 meningococcal epidemic season, no published reports had described serogroup X isolates in East Africa. We report the involvement of N. meningitidis serogroup X in an outbreak of meningococcal disease in Western Kenya.

In January 2006, the Ministry of Health of Kenya and Médecins sans Frontières were notified of a suspected meningococcal disease outbreak in West Pokot District, bordering Uganda, in Western Kenya. On the basis of the initial outbreak investigation, the outbreak was assessed to have begun in late December 2005. Subsequent active surveillance, using the same clinical case definition of sudden fever onset with stiff neck, altered mental status, or both, showed 74 suspected cases through mid-March 2006, with a case-fatality rate of 20%. No cases were reported after March 2006.

Over the course of the outbreak, cerebrospinal fluid samples were obtained from 18 patients. Due to low population density, poor access to seminomadic populations, and the