diverse setting. Additionally, although we completed a thorough review of inpatient and outpatient records from several healthcare facilities in Maryland, data may have been missing. However, this factor is expected to have been similar for both cases and controls.

As the incidence of ESBLs increases and they contribute to considerable morbidity and mortality, it is imperative to develop systems to identify children who are most at risk of infections caused by ESBL-producing organisms. Our findings suggest that in addition to reviewing prior culture histories to identify children with previous ESBL colonization or infection, targeted screening of children who received medical care abroad in high-risk countries, who recently received chemotherapy, or who recently underwent hematopoietic stem cell transplantations may be another strategy to help identify those most at risk for ESBL colonization.

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Colistin-Resistant *Klebsiella* Infections Among Pediatric Oncology and Hematopoietic Stem Cell Transplantation Patients in Eastern India

To the Editor—The epidemiology and clinical outcome of extremely drug-resistant (XDR) gram-negative bacterial (GNB) infections in the hemato-oncologic pediatric population is not well documented. Colistin-resistant *Klebsiella* (CRK) is one such XDR infection; it is resistant to most classes of antibiotics except 1 or 2 classes, and it is one of the most difficult pathogens to treat. These infections are increasingly common in settings with high XDR prevalence, and they are increasing globally because of travel, movement of food items, use in livestock, and widespread use of antibiotics (in patients and food animals).[1–5] In the last 3 years, the trend in colistin resistance has increased among the commonly encountered GNB in our hospital (ie, for adults and pediatrics combined). From 2014 to 2016, the resistance to colistin in *Klebsiella* increased from 1.98% to 3.12%; in *Escherichia coli,* colistin resistance increased from 0.14% to 0.24%; in *Pseudomonas,* colistin resistance increased from 0% to 0.87%; and in *Acinetobacter,* colistin resistance increased from 0% to 4.49% (see the Figure online). In this retrospective study, we investigated the incidence, clinical presentation, and outcomes of CRK infection among pediatric oncology/transplantation patients (0–18 years old) in a cancer hospital in India between May 2011 and April 2017.

TABLE 1. Profiles of Patients Infected With Colistin-Resistant *Klebsiella* Infections

Time of Occurrence	Background and Presentation	Antimicrobial Therapy	Other Microbiology	Outcome
Case 1: Jul 2013	5.6 yo/male; β-thalassemia; allogeneic HSCT; perianal abscess/pus; ANC-0/mm^3	Meropenem + teicoplanin + colistin + tigecycline >> surgical drainage with povidone iodine dressing	ESBL Escherichia coli	Survived
Case 2: Aug 2013	4.8 yo/ male; relapsed AML; RTI/sputum; ANC-48/mm^3	Meropenem + colistin + teicoplanin	*Elizabethkingia meningoseptica* and carbapenem-resistant *Klebsiella* in blood culture	Died
Case 3: Oct 2014	12.2 yo/male; ALL with HSCT/sepsis/blood culture; ANC-30/mm^3	Meropenem + colistin + teicoplanin + chloramphenicol	ESBL *Escherichia coli*	Died
Case 4: Jan 2016	7.8 yo/male; β-thalassemia with HSCT; sepsis/ blood culture; ANC-19/mm^3	Meropenem + teicoplanin + fosfomycin	Positive for CRK in stool surveillance 3 weeks before CRK infection	Survived
Case 5: Jan 2016	12.1 yo/female; AML with intra-abdominla infection/pus; ANC-10/mm^3	Meropenem + colistin >> chloramphenicol + tigecycline; splenectomy	ESBL *Escherichia coli*; carbapenem-resistant Klebsiella; carbapenem-resistant *Proteus*	Survived

NOTE. yo, years old; HSCT, hematopoietic stem cell transplant; ANC, absolute neutrophil count; ESBL, extended-spectrum β-lactamases; AML, acute myeloid leukemia; RTI, response to intervention; ALL, acute lymphoblastic leukemia; CRK, colistin-resistant *Klebsiella*.

Over the 6-year study period, 8 pediatric patients were identified to be infected or colonized with CRK. The ratio between patients infected or colonized with colistin-sensitive *Klebsiella* and CRK during this period was 19 to 1. Of these 8 patients, 5 had clinically significant CRK infections and 3 were colonized with CRK detected during stool surveillance cultures (Table 1). The CRK-infected patients (4 males and 1 female) had a median age of 7.8 years (range, 4.8–12.2 years), and their median absolute neutrophil count (ANC) at the time of CRK infection was 19 cells/mm^3 (range, 0–48 cells/mm^3). Among the CRK-infected patients, 3 had hematological malignancy (2 with acute myeloid leukemia, 1 with acute lymphoblastic leukemia) and 2 had β-thalassemia with hematopoietic stem cell transplantation (HSCT). Their clinical presentations included disseminated infection in 4 patients and localized infection in 1 patient. All of the patients received systemic antibiotics. Surgical intervention (ie, splenectomy and abscess drainage) was performed on 2 of these 5 patients even though they had very low ANCs, and both patients survived. Multidrug-resistant (MDR) GNB were detected in microbiological samples prior to the detection of CRK in all patients. Tigecycline, chloramphenicol, fosfomycin, and cotrimoxazole were a few antibiotics that showed in vitro susceptibility. Fosfomycin was sensitive in 3 of 5 isolates tested; chloramphenicol was sensitive in 3 of 3 isolates tested; tigecycline was intermediately susceptible in 4 of 5 isolates tested (the other being sensitive); co-trimoxazole was sensitive in 2 of 5 isolates tested; and amikacin was intermediately susceptible in 1 of 5 isolates tested (the others were resistant). The 30-day all-cause mortality from the time of microbiological confirmation of CRK infection was 40% (2 of 5 patients).

Colistin-resistant *Klebsiella* infections are potentially fatal, and antibiotic treatment options are limited. Surgical intervention for source control is an important treatment modality wherever feasible; however, the risk of post-surgical sepsis is high in immunocompromised and neutropenic patients. In this study, all patients with CRK were found to be colonized with MDR-GNB (2 with carbapenem resistant *Klebsiella*, 1 with CRK, and 2 with ESBL *E. coli*) in other samples, including stool. This finding suggests that antibiotic-induced selection pressure could have played a part in selecting out these organisms as a cause of infection in neutropenic patients.

Unanswered questions arising from this study include mode of acquisition of these infections (community or healthcare associated). Plasmid-mediated genes for colistin resistance (mcr-1) have been identified in China (in pigs), and the possibility of horizontal spread exists. Colistin is widely used as a growth promoter in livestock farming. At least 1 of the top 10 producers of colistin for agricultural use is in India. The Center for Disease Dynamics Economics and Policy previously reported that India is one of the top consumers of agricultural antibiotics worldwide, accounting for 3% of their global consumption, and this figure is estimated to double by the year 2030.[5–7] This situation has threatened the position of colistin as 1 of the last-resort antibiotics for the treatment of carbapenem-resistant GNB infections.

The exact proportion of colistin resistance in clinical settings may be underreported. Unlike many other antibiotics, the cheaper and relatively easier disc diffusion technique cannot be used for the detection of colistin susceptibility, and the more laborious, time-consuming, and expensive broth-dilution technique must be used (Clinical Laboratory Standards Institute, Wayne, PA). In 1 such method used globally (Vitek, BioMerieux, France, communicated on May 24, 2017), the system had shown high rate of very major errors (ie, resistance was reported as sensitive). Molecular tests for the detection of colistin resistance are not easy to implement due to the higher cost, the need for infrastructure, and the multiplicity of genetic changes involved (eg, *mgrB, phoP/phoQ, pmrA, pmrB, pmrC*, and *crrABC*).[8]

To conclude, CRK represents a new threat in the antibiotic-resistance landscape. Although our sample size was much smaller, the mortality rate seen in our CRK cohort was similar that documented in a study of Italian allogeneic stem-cell transplant recipients where the 30-day crude mortality was 24% for single-GNB bloodstream infection but reached 39% for *Pseudomonas aeruginosa*.[9] Controlling the threat requires a multifaceted strategy involving the restriction of colistin use in the agricultural sector, prudent use in human health care, and accurate laboratory diagnosis. The inclusion of this emerging pathogen on the World Health Organization's Priority Pathogen List may help facilitate the development of new antibiotics.[10]

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SUPPLEMENTARY MATERIAL

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Coordination of Infection Control Activities at the Healthcare System Level: Survey Results

To the Editor—Approximately 66% of community hospitals are part of a healthcare system (multihospital or a diversified single hospital system).[1] We sent a 10-question survey electronically to 96 hospital epidemiologists on September 9, 2016, to determine how they organize infection control activities across healthcare system hospitals. Of 22 respondents, 21 were working in a facility that was part of a hospital system. Most respondents noted that infection control activities were coordinated across the healthcare system, with a system-wide hospital epidemiologist alone having ultimate authority in nearly half of the healthcare systems (Table 1). One-third of system-wide infection control leadership reported to a system-wide Chief Medical Officer, and another third reported to a system Chief Quality Officer. Most of these systems reported having standardization of infection control policies and procedures, and two-thirds reported having a system-wide infection control committee.

Although greater numbers of US hospitals are part of nongovernmental healthcare systems, the best model to coordinate infection control policies, procedures, and activities remain unknown.[2] Our survey was not a random sample and likely suffers from ascertainment bias. Nevertheless, we hope that the data presented will stimulate greater discussion and investigation by members of the infection control community so that we can chart a course forward regarding this important and understudied issue. If not, we must assume that administrators will determine the structure of system-wide infection control activities, whether or not we agree with it.

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