Preventing Drug-resistant Infections in Health Care
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Health care-acquired infections (HAIs) affect millions of health care workers and patients every year and are becoming increasingly resistant to common treatments. These dangerous infections are largely preventable, but are created primarily by the inappropriate administration of antibiotics and spread by inadequate infection control practices by health care workers. This article introduces contamination types and transmission facets of the HAI problem. Physical reservoirs in the health care environment are identified and the epidemiology, surveillance and control of emerging multidrug-resistant bacteria are reviewed.

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Health care-acquired infection (HAI) is a leading cause of death in the United States. Every year, 1.7 million Americans contract HAIs, which results in additional costs that exceed an estimated $30 billion. One study reported that average costs for patients with HAIs are more than 600% higher than the costs for patients who do not contract similar infections. In 2002, the Centers for Disease Control and Prevention (CDC) estimated that more than 98,900 Americans died of HAIs, including nearly 36,000 suffering from HAI pneumonia and another 30,000 with bloodstream infections (bacteremia). Overall, the CDC reports that 32% of all HAIs are urinary tract infections (UTIs), 22% involve surgical sites, 15% are lung infections and 14% involve bacteremia.

With rapidly evolving medical technology and expertise designed to save more patients, it is disheartening to lose lives to largely preventable infections. HAIs are caused by inappropriate antibiotic usage and ineffective infection control practices. The CDC identifies hand hygiene by health care personnel as "the single most important factor" to prevent infection spread, but numerous studies reveal dismal compliance with effective hand washing guidelines. In May 2008, for example, CDC investigators reported that at least 85 patients at an endoscopy clinic in Las Vegas, Nevada, had become infected with the hepatitis C virus because of lapses in clinic workers' hand hygiene and the inappropriate reuse of single-use syringes and sedative vials.

Increasingly, HAIs involve multidrug-resistant organisms (MDROs), reflecting a global trend of antibiotic ineffectiveness. As many as 70% of HAIs may involve MDROs, although exact proportions vary between facilities and over time. Because of their resilience, MDRO infections can be difficult to resolve. At greatest risk are elderly patients, newborns, intensive care unit patients and those with immune system deficiencies, cancer or burns. Although some multidrug-resistant HAIs are familiar in the health care setting, emerging HAIs present new and complex challenges for infection control efforts at hospitals.

Because Medicare pays for more than one-third of U.S. hospital costs and HAIs are preventable, Congress mandated in the Deficit Reduction Act of 2005 that, as of October 2008, hospitals are no longer to be
reimbursed for additional costs caused by certain HAIs, including infections caused by vascular catheters, UTIs caused by catheters and infections following coronary artery bypass surgery. Likewise, in July 2011 the Patient Protection and Affordable Care Act ruled that health care institutions could not receive Medicaid reimbursement for certain health care-acquired conditions.

An increasing number of states are requiring health care facilities to report certain HAIs to the National Healthcare Safety Network (NHSN), a public health surveillance system maintained by the CDC. Hospitals participating in the Centers for Medicare and Medicaid Services (CMS) quality reporting program also are required to use NHSN for public reporting of HAIs. The assumption is that once hospitals are forced to disclose infection rates, they will be more likely to make infection control a high priority.

History

In retrospect, it is surprising and tragic that antibiotics were not discovered decades earlier than they were. In the late 1880s, scientists discovered that pyocyanase, a pigment released by the bacterium now known as Pseudomonas aeruginosa, slowed the growth of other bacteria, including those causing typhoid, anthrax and plague. However, pyocyanase was too toxic for human patients, and scientific excitement over the discovery quickly waned. Few researchers continued to search for a safe bacterial disease cure.

In the 1890s, researchers at Johns Hopkins University noticed that molds on agar plates stopped the growth of bacterial colonies — the same observation that led to the discovery of penicillin in 1929. At the time, however, the implications of the discovery were missed. Also in the 1890s, German scientist Robert Koch noted that common soil killed pathogenic bacteria, but he did not realize that he was witnessing the effects of antibiotics produced by soil bacteria. Nearly 50 years later, soil microbiologists rediscovered what Koch had observed and systematically searched for antibiotics produced by soil microbes. In 1939, soil-dwelling Bacillus brevis was isolated and shown to produce an antibiotic called gramicidin, which killed Staphylococcus bacteria in the lab. Because of its severe toxicity, however, gramicidin’s clinical use was limited to the treatment of skin infections. By this time, German scientists already had synthesized the first sulfonylurea antimicrobial.

Penicillin, the most famous antibiotic, was discovered not through the diligence of chemists, but rather sheer luck. In 1928, English microbiologist Alexander Fleming returned from vacation to find that atop a stack of agar plates sitting in a lab sink to be cleaned, 1 plate had not been submerged in the soapy water. On that plate, Fleming noticed that growth of a Staphylococcus colony had been halted by an adjacent mold. From this mold, Fleming isolated enough penicillin to describe it scientifically, but not enough to conduct clinical studies. Oxford researchers were able to do so, however, and by the outbreak of World War II, their animal experiments had convinced American pharmaceutical firms, with backing from the U.S. government, to mass-produce the drug. Early in World War II, penicillin was a secret and supplies were controlled by the military. It was largely unknown to the public until the November 1942 Cocoanut Grove nightclub fire in Boston, Massachusetts, when the Merck Company released 32 liters of the antibiotic to Massachusetts General Hospital for the hundreds of burn victims. Three years later, penicillin was known and readily available to most Americans — and very much in demand. Ironically, the ready availability of these drugs rapidly eroded their usefulness.

The Antibiotic Paradox

The central paradox of the antibiotic era is that over-reliance on antimicrobial drugs erodes their efficacy. Antibiotic drugs have saved tens of millions of lives since World War II, but decades of indiscriminate, ubiquitous and prophylactic use have driven the evolution of drug-resistant organisms. In a 1945 interview, Fleming warned that inappropriate use of his “wonder drug” would yield drug-resistant infections. But it was not until the mid-1950s that over-the-counter sales were curtailed and prescriptions were required to purchase penicillin. By the mid-1970s, 2 infants had died of a drug-resistant strain of Hemophilus at the Bethesda Naval Hospital. In addition, penicillin-resistant gonorrhea appeared among U.S. servicemen and spread from Asian brothels to the United States. To this day, military medical officials are caught in an epidemiological Catch-22, with recruits in basic training requiring prophylactic antibiotic therapy to avoid otherwise nearly inevitable disease outbreaks, but prophylaxis facilitating the emergence of MDROs.

Inappropriate antibiotic prescribing is the root cause of the MDRO epidemic, but it is not the only factor. Patient demographics and changes in the hospital environment also have contributed. There are more elderly and immunocompromised patients today than in the mid-20th century, and they undergo more invasive procedures involving a greater diversity of medical devices. The body of knowledge is ever expanding, so it is crucial...
of a host often remains asymptomatic until the host becomes immune-challenged or the microbes gain entry to the bloodstream. Pathogenic bacteria have several mechanisms to escape from circulating immune lymphocytes, including a smooth protein coat to which it is difficult for lymphocytes to attach (see Figure 1). Some bacteria, such as *Legionella*, survive ingestion by lymphocytes and replicate within host cells. Many pathogenic bacteria release toxins that ward off lymphocytes or help the bacteria to digest host cells.

**Evolution of Bacterial Drug Resistance**

Antibiotics help resolve infections that have circumvented or overwhelmed a patient’s immune defenses. However, pathogens can become adept at circumventing mechanisms of drug action. Common mechanisms that have evolved among MDROs include:

- Decreased absorption of the drug through the bacterial cell wall or increased rates of pumping drugs back out of a microbe before they can harm it.
- Deactivation of the drug by microbial enzymes (eg, beta-lactamases, which changes the structure of penicillin).
- Changes in the protein-binding sites of bacterial cell walls, denying antimicrobial drugs the binding targets through which they originally attached to and destroyed the microbe.
- New microbial metabolic pathways to circumvent the pathways once disrupted by antimicrobial drugs.

Some degree of drug resistance among bacterial colonization probably is inevitable whenever antibiotics are prescribed. Because the lifespan of individual bacteria is so short, the evolution of drug resistance can occur with astonishing speed — in some cases, a few days. Because an administered antimicrobial kills both harmful and helpful bacteria in a patient, rare problems can emerge. For example, within a day of exposure...