



Figure 1. Computed tomograph of the lungs revealing a dense right upper and middle lobe infiltrates.

bined cadaveric liver and kidney transplantation in October 2008 and developed catheter-related vancomycin-resistant enterococcal bloodstream infection 2 weeks after transplantation. He was initially treated with linezolid (600 mg every 12 h) but remained bacteremic after 9 days of therapy. A transesophageal echocardiogram showed no valvular vegetations. His regimen was transitioned from linezolid to daptomycin at 10 mg/kg per day. The patient's blood cultures sterilized, and he was discharged to a skilled nursing facility on day 40 after the operation.

Five days after discharge, while receiving daptomycin, the patient developed fever and dyspnea. He was transferred back to our facility for further evaluation. Blood culture results were negative, but a chest computed tomograph revealed new dense right upper and middle lobe consolidations (Figure 1). Daptomycin therapy was discontinued at admission, and bronchoalveolar lavage was performed. The lavage fluid grew moderate MRSA in 2 separate cultures, and the minimum inhibitory concentration (MIC) of daptomycin for the MRSA isolate was 0.5 $\mu\text{g}/\text{mL}$. The patient commenced treatment with linezolid at 600 mg twice per day (the

MIC of linezolid for the MRSA isolate was 1 $\mu\text{g}/\text{mL}$). His fever and hypoxia improved, and the patient ultimately received 4 weeks of linezolid. The patient has been without relapse during 3 months of follow-up.

To our knowledge, this is the first reported case of a patient developing bronchoalveolar MRSA pneumonia while receiving high-dose daptomycin for another infection. We speculate that the patient's lower respiratory tract was inoculated with MRSA from the nasopharynx and not hematogenously, given the chest computed tomography findings and negative blood culture results. This case supports laboratory findings that even a small amount of surfactant (1%) results in a 16–32-fold loss of potency of daptomycin [4]. This case reinforces that daptomycin is inappropriate treatment for bronchoalveolar pneumonia. Moreover, the lower respiratory tract may be susceptible to infection during daptomycin therapy because of surfactant-mediated daptomycin inactivation.

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Time for a Worldwide Shift from Oral Polio Vaccine to Inactivated Polio Vaccine

TO THE EDITOR—Poliomyelitis (often called *polio*) is an acute viral infectious disease caused by poliovirus. Polio was one of the most lethal childhood diseases of the 20th century [1].

Two polio vaccines are commonly used throughout the world for poliomyelitis. The first was developed by Jonas Salk in 1952; the second was an oral vaccine developed by Albert Sabin. These 2 vaccines have eradicated polio from most countries and have reduced the worldwide incidence of polio from 350,000 cases in 1988 to just 1300 cases in 2007 [1, 2].

The Salk vaccine, or inactivated poliovirus vaccine (IPV), is based on 3 virulent reference strains—Mahoney, MEF-1, and Saukett. The Salk vaccine provides immunoglobulin G-mediated immunity in the bloodstream, which prevents infection from progressing to viremia and protects the neurons. The Salk vaccine is 60%–70%

effective against poliovirus 1 and is 90% effective against both poliovirus 2 and 3 [1].

Oral polio vaccine (OPV) is a live attenuated vaccine: it is produced by passage of poliovirus through nonhuman cells at a subphysiological temperature, which causes spontaneous mutations in the viral genome. OPV is superior to IPV in ease of administration, and there is no need for sterile syringes, as with IPV. OPV also provides longer immunity than does the Salk vaccine. However, OPV has strict requirements for transport and storage, and this is a big problem in some hot or remote areas [2, 3]. Table 1 summarizes the key differences between OPV and IPV.

A major concern about OPV is its ability to revert to a form that can cause paralysis. Outbreaks of vaccine-associated paralytic poliomyelitis (VAPP) have been reported in many countries of the world [2, 4].

In 2005, it was reported that children in a small village in the United States had contracted vaccine-derived polio. In Nigeria, >70 cases have been reported. In 2006, ~1600 cases of vaccine-induced polio occurred in India, according to the Indian Medical Association Sub-Committee on Immunisation's report on the Polio Eradication Initiative [3]. The point to be noted is that these cases were reported *during* repeated mass-immunization campaigns in which repeated doses of OPV were administered. In 2008, many cases of polio were reported in all provinces of Pakistan, where OPV is used for repeated mass-immunization campaigns. These vaccine-related cases are big challenge for the scientific community if the polio-eradication goal is to be achieved, and there is a need for prompt action to combat the issue [1–5].

According to the World Health Organization, routine immunization with OPV must cease after the eradication of poliovirus because of the danger of outbreaks of circulating vaccine-derived poliovirus and the risk of VAPP. In the regions of the world in which wild-type poliovirus has

Table 1. Comparison of Oral Polio Vaccine (OPV) and Inactivated Poliovirus Vaccine (IPV)

Property	OPV	IPV
Mode of administration	By mouth	Injectable
Type	Live attenuated	Inactivated
Gastrointestinal tract immunity	Yes	No
Virus shed in feces	Yes	No
Requirements for transport and storage	Strict	Not strict
Ability to revert	Yes	No

been eliminated, moving to an IPV or IPV/OPV sequential schedule will reduce or eliminate the risk of VAPP and outbreaks of circulating vaccine-derived poliovirus, as well as increase the likelihood of countries agreeing to stop administering OPV after eradication is achieved. IPV could also be used with OPV in routine schedules to increase immune responses and to decrease the circulation of wild-type poliovirus in countries in which transmission has not been stopped. IPV alone was very successful in eliminating wild-type poliovirus in many European countries and has been used exclusively in the United States since January 2000.

The above observations suggest that OPV has lost its effectiveness in providing herd immunity. It seems that children are getting polio from OPV, and it also seems that OPV is proving to be ineffective in stopping polio transmission from another source. Therefore, the whole world—and especially developing countries—should shift from OPV to IPV, in my opinion. There is still a need for active research in exploring various vaccine strategies for polio and to combat adverse effects associated with polio vaccination; otherwise, the dream of polio eradication will never come true [2–4].

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Surveillance Definitions for Urinary Tract Infections

TO THE EDITOR—We would like to provide a correction to the article on inappropriate treatment of catheter-associated asymptomatic bacteriuria in the 1 May 2009 issue of *Clinical Infectious Diseases* [1]. The article makes reference to previous Centers for Disease Control and Prevention surveillance definitions for urinary tract infection (UTI), which included both symptomatic UTI and asymptomatic bacteriuria [2]. We recently revised the UTI surveillance criteria, in consultation with outside experts, to improve the accuracy of UTI surveillance and to discourage inappropriate screening and treatment for asymptomatic bacteriuria. Specifically, we removed the asymptom-