of HIV-infected patients. Palliative approaches are appropriate in dual-management strategies, alongside new therapies, and should be introduced and withdrawn as necessary.

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Richard Harding,1 Philippa Easterbrook,2 Natalla Dinat,3 and Irene J. Higginson1

1Department of Palliative Care and Policy and 2Academic Department of HIV/ST Medicine, King’s College London, London, United Kingdom; and 3Perinatal HIV Research Unit, University of the Witwatersrand, South Africa

Influenza Deaths in Spite of Immunization and Prophylaxis

Sir—We would like to add evidence for the multimodal influenza prevention strategies so ably advanced by Monto et al. [1] and by Bridges and Harper [2]. In a long-term care facility (Laguna Honda Hospital, San Francisco, California) with a census of 1055, we documented 29 patients with cases of influenza A by rapid EIA during the 2001–2002 influenza season. Of these 29 patients, 26 received timely influenza immunizations, as did 92% of all facility residents. Four patients died, all of whom had received timely influenza vaccinations and had received at least 5 days of prophylaxis with rimantadine before the appearance of their first symptoms. All 4 patients had their treatment switched to oseltamivir after the onset of symptoms—3 patients within 24 h after the onset of symptoms and 1 patient within 2 days after the onset of symptoms. One of the deaths occurred within 24 h after the onset of symptoms. The rapid course of the illness and the autopsy findings of hemorrhagic tracheitis and bronchopneumonia were consistent with primary viral pneumonia. In addition to those 4 deaths, another death occurred in a patient who had negative EIA results but whose illness was clinically consistent with influenza. This brought our total number of cases to 30, for an attack rate of 5%. The average age of the 30 patients involved in the cases was 72 years; the average age at death for the 5 patients was 80 years. We checked one specimen for virus subtype and found that it was H2N3. Two nursing facilities in our area experienced >50% attack rates with subtype H2N3.

These deaths did not diminish our enthusiasm for immunization or antiviral prophylaxis; the benefit of both is well demonstrated. Rather, with this reminder of the lethal potential of influenza, we redoubled our efforts in all components of influenza prevention, including patient and staff immunization, aggressive surveillance with rapid-testing followed by more-sensitive testing for specimens with negative results, oseltamivir treatment within 24 h after the onset of symptoms for patients with documented infection, prophylaxis with antivirals (rimantadine, in our case), and implementation of infection control measures. The staff immunization rate increased from 26% in 2001–2002 to 37% in 2002–2003 and 48% in 2003–2004. Improvements in tracking and follow-up will likely yield better rates during the 2004–2005 season.

For most nursing facilities, the most difficult prevention component will be to perform rapid tests and to implement treatment and prophylaxis protocols within 24–36 h after the onset of symptoms. Even partial implementation of laboratory testing has payoffs, however, because laboratory-confirmed diagnoses can galvanize nurses, administrators, and physicians into action. Aggressive viral testing of appropriate nursing facility patients in hospital emergency departments could improve identification of influenza outbreaks and help to leverage system changes. Testing also reveals the presence of other lethal viruses. In particular, the impact of respiratory syncytial virus infection in our facility has again motivated us to strengthen our infection control procedures.

As Bridges and Harper [2] suggest, it is unrealistic to think that most nursing facilities can plan and implement all of these components without outside assistance.

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Reprints or correspondence: Dr. Richard Harding, Dept. of Palliative Care and Policy, Guy’s King’s and St. Thomas’ Medical School, King’s College London, Outcome Rd., London SE5 9RJ, United Kingdom (Richard.harding@kcl.ac.uk).

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We support their call for all stakeholders to move to a new level of collaboration.

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Terry Hill,1,3 Angela Plazter,2 and Cristina Reyes1
1Department of Medicine, University of California–San Francisco, 2Laguna Honda Hospital, and 3Lumetra, San Francisco, California

References

Reprints and correspondence: Dr. Terry Hill, Lumetra, One Sansome St., Ste. 600, San Francisco, CA 94104 (thill@lumetra.com).

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Multidrug-Resistant Malaria from South Africa

Sr—A 28-year-old white woman who had not previously travelled to an area where malaria is endemic spent 2 weeks on holiday in Zimbabwe, Botswana, and South Africa. She took regular antimalarial prophylaxis with weekly dosages of chloroquine (300 mg) and daily dosages of proguanil (200 mg). The patient remained healthy while she was away. Four days after returning to London, she developed headache, generalized musculoskeletal pain, and an intermittent fever. Three days later, she presented to our hospital (Hospital for Tropical Diseases, London, United Kingdom) with a fever (temperature, 40°C) and sinus tachycardia (heart rate, 120 beats/min). There were no localized signs of infection. A blood film confirmed the diagnosis of Plasmodium falciparum malaria and peripheral-blood parasitemia (parasite percentage, 0.1%). The patient was treated with 9 oral doses of quinine at dosages of 600 mg t.i.d., which cleared the parasitemia; she then received a single dose of 3 tablets of sulfadoxine-pyrimethamine, according to standard practice at the hospital, and was discharged.

One month later, the patient presented to the hospital again, reporting that she had had general malaise and fever for the previous 3 days. In the interim, she had not left the United Kingdom. A blood film confirmed P. falciparum malaria with a parasite percentage of 0.1%. The patient was readmitted to the hospital and was treated with 4 tablets of atovaquone-proguanil (Malarone; GlaxoSmithKline) daily for 3 days. She made a full recovery and, 12 months later, remained healthy.

Pretreatment blood samples from both episodes were obtained from the parasitology laboratory of the Hospital for Tropical Diseases, and parasite DNA was extracted. The multiplicity of infection and the presence of point mutations at the Pfcrt-76 and Pfdmrd-86 loci, which mediate resistance to chloroquine, and the Pfδhps and Pfδhfr loci, which mediate resistance to sulfadoxine-pyrimethamine, were determined by standard methods [1–3]. The isolates from the 2 episodes were indistinguishable from each other, containing a single mad20 type for the msp1 locus and a single ic type for the msp2 locus [1]. This finding strongly suggested that the treatment had failed and that the infection had recrudesced with the same clone. Furthermore, these isolates were found to be carrying the following markers associated with resistance to chloroquine and sulfadoxine-pyrimethamine: pfcr-76T, pfmdr-1-86Y and pfmdr-1-184F, pfδhfr-511 and pfδhfr-108N, and pfδhps-437G and pfδhps-581G. Although no genetic marker for quinine resistance has yet been identified, there is an association between clinical failure after quinine treatment and the development of mutations in the Pfmdr1 locus [2]. Resistance of P. falciparum to chloroquine has been reported widely from sub-Saharan Africa, and resistance to sulfadoxine-pyrimethamine is becoming increasingly common [2]. Although multidrug-resistant malaria has been reported from southeast Asia, this is the first such report from southern Africa. Several authors have suggested recently that artemisinin-based combination treatments should now be the first-line treatment for malaria in Africa [4]. The finding of this multidrug-resistant strain lends support to that view.

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U. Schwab,1 A. Allouche,2 and J. F. Doherty1,2
1Hospital for Tropical Diseases, and 2Clinical Research Unit, London School of Hygiene & Tropical Medicine, London, United Kingdom

References

Reprints or correspondence: Dr. Tom Doherty, Hospital for Tropical Diseases, Mortimer Market Centre, Capper St., University College of London Hospitals, London, WC1E 6AU, United Kingdom (tom.doherty@uclh.org).

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